Pediatrics Made Simple

For Final Year MBBS Students and Doctors

1st EDITION

By

Dr. Ahmad Hassan

Batch 39

Rawalpindi Medical College

Rawalpindi

Zubair Book Depot

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Title: Pediatrics Made Simple – The Study Mate Series

Author: DR. AHMAD HASSAN

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This Book is

Dedicated

to

My Grandmothers

Mrs. Imam Bibi (Late)

Mrs. Surriya Akhtar (Late)

Mrs. Hashmat Bibi (Late)

Preface to the First Edition

With the first edition of **The Study Mate Series' Pediatrics Made Simple –For the Students of MBBS** I, Dr. Ahmad Hassan hereby continue my commitment to provide students with the **most useful and easy to use preparation guide for Undergraduate exams** in the subject stated.

When I completed my MBBS and studied pediatrics in final year the major problem I encountered was to manage time for pediatrics since there are 3 other major subjects to study and retain.

It was an uphill task to take time out for each subject equally and make a study plan to score well so I resorted to making notes for each subject. The First Read is always the hardest for every subject since it requires you to read through an entirely new subject, using multiple books so I highlighted stuff, made small foot notes and my own study plans
Once I passed the phase of FIRST READ all that was left was to cram and ingest the stratified and systematic information that I had written/highlighted.

At that time I used those notes as a study guide for me so that I could go through the topics multiple times with good understanding in an exam-oriented manner. I was not the only one who used those notes. I uploaded them on various platforms and thousands of aspiring students used those notes to ace their professional exams as well and till date thousands of students are using them. Alhamdulillah I have received an amazing response from people all across Pakistan and even at an international level.

I was requested multiple times by the students of final year to write out a book for pediatrics since the books they have to follow are not exam oriented with extensive details that are not required for an undergraduate student. The first read takes a lot of time and they are not able to manage time and prepare the subject/revise the important things in a maximum of 3 days time during their professional examinations.

This book will make your life easier since it contains only the important and to the point information that a final year student needs to ace the pediatrics professional examination. Multiple topics are in a tabulated form making it easier to understand and revise and reproduce it on paper during the real exam along with mnemonics and past MCQs in statements form this will be your go-to book for everything be it your written examination, OSPE/TOACS or Viva. This book when used with the Book **"The Wardmate"** and the videos uploaded on our YouTube channel / Facebook page will

serve as an excellent source to ace your short and long cases as well Insha'Allah. It also contains the study plans, Table of specifications and the list of important topics that you need to study.

One thing that I am a firm believer of is that Allah Subhan A Taala's help descends only to those who have a sincere heart and a strong will to pull others up once they pass through the phase and help them get through that phase as well. This book is a testament to what I believe in and I really hope it helps you people out to memorize the basics and the most repeated things that you encounter in the exam in the first read itself. With the pattern it is written in you can revise it in 2-3 days before the exam and you can ace the exam with your short-term memory and concepts using this very book Insha'Allah.

Your feedback, Suggestions and comments matter a lot and I would appreciate anyone who reads this book to come up with improvements and helpful criticism so that others can be helped too by making improvements in the next editions ☺

May This Book be a source of Relief for you people Aameen
Remember me in your prayers
JazzakAllah ul Khair

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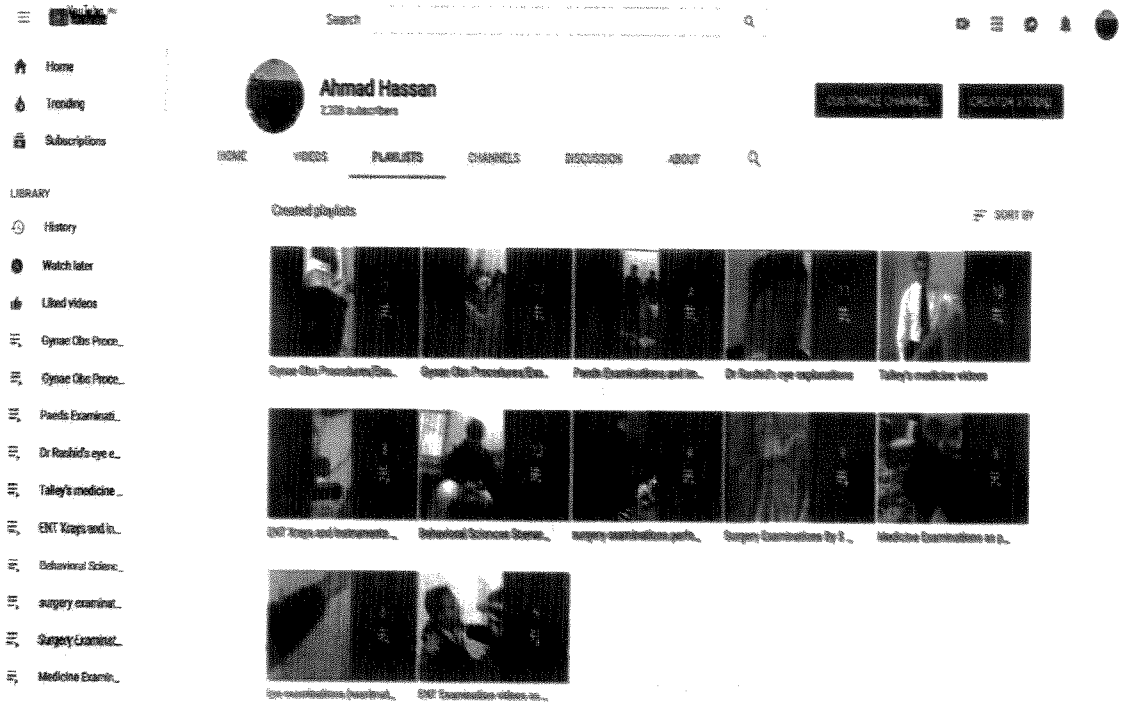
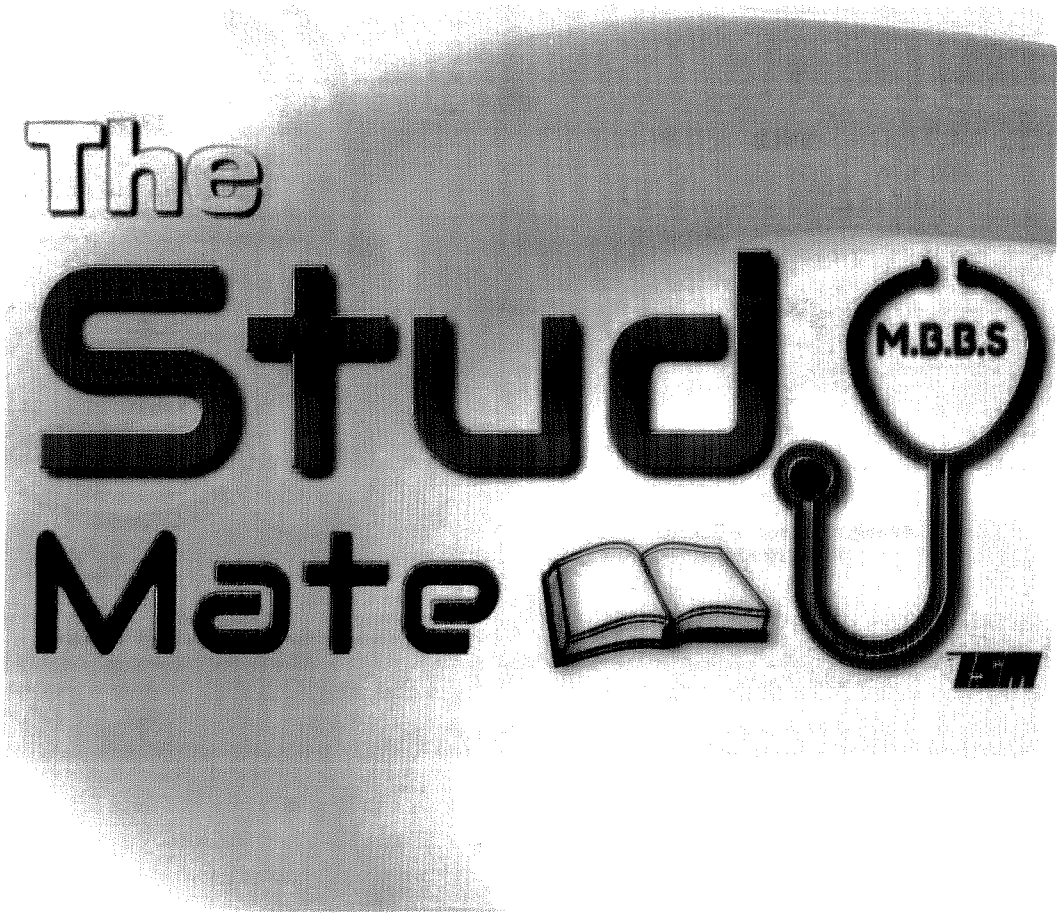
There is a platform by the name of

The STUDY MATE MBBS & Links to Wardmate Videos-Dr. Ahmad Hassan

on facebook in which you can find the links to all the notes, past MCQs, STUDY GUIDELINES, OSPES, Time tables, important topics and much more For MBBS (All years) and even clinical videos

You Can also access the videos of examinations exactly according to the ones written in this book WARDMATE by following the youtube channel for free
https://www.youtube.com/channel/UC17uGzhIaxrdac8EgpuyneA
Here is the screenshot of the youtube channel playlists :

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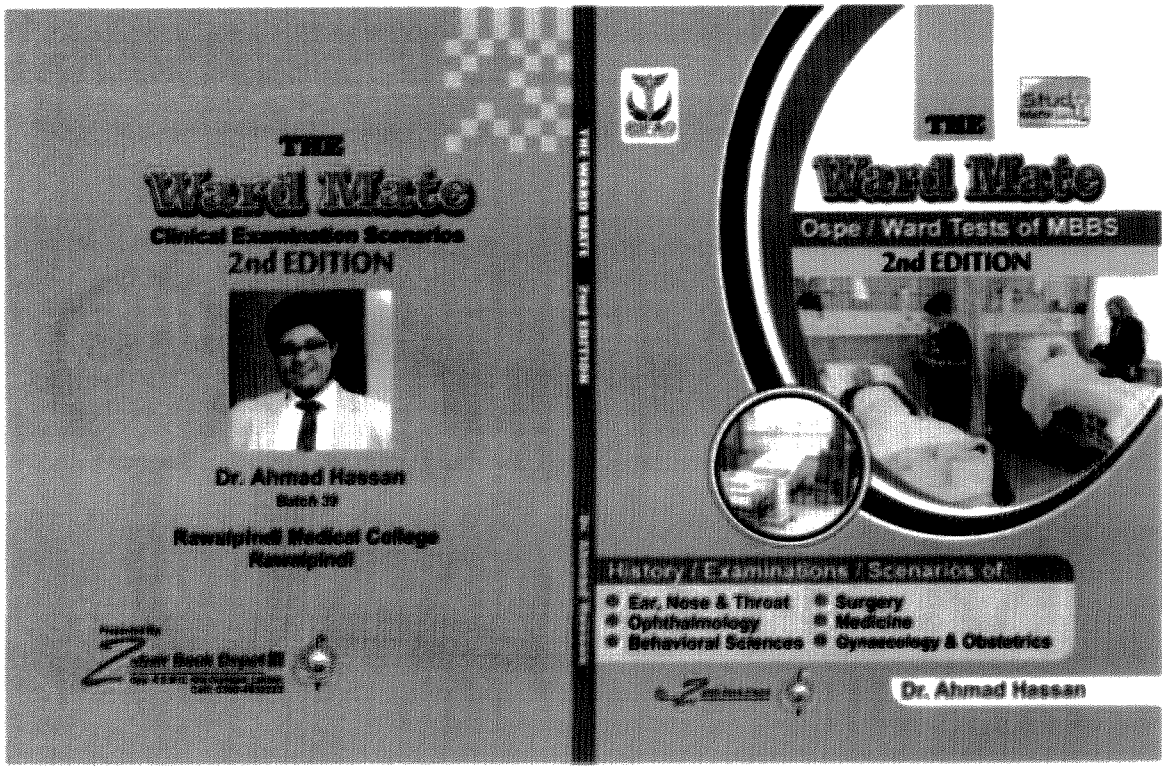

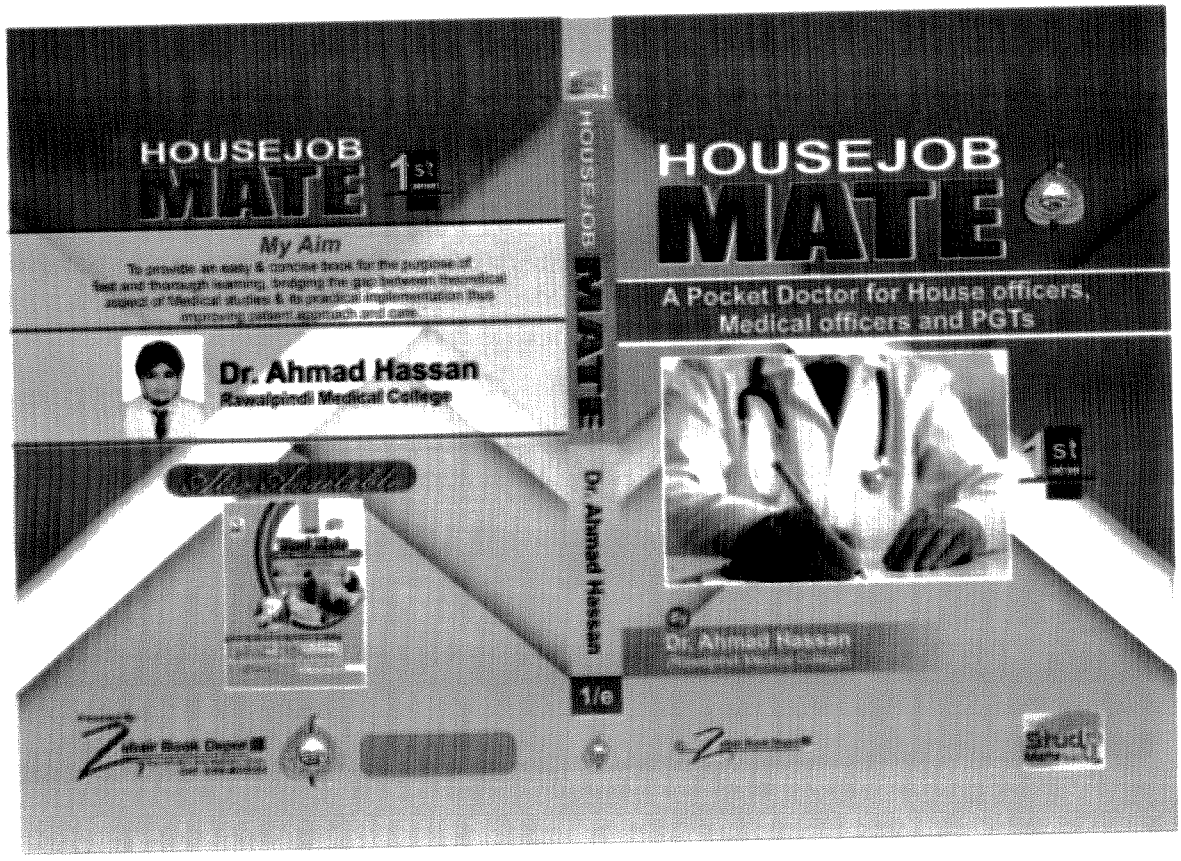
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My Aim
To provide an easy & concise book for the purpose of fast and thorough learning, bridging the gap between theoretical aspect of Medical studies & its practical implementation thus improving patient approach and care.
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A Pocket Doctor for House officers, Medical officers and PGTs
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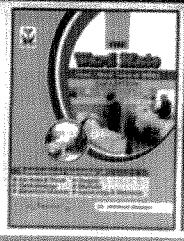
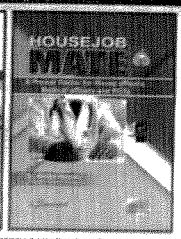
Dr. Ahmad Hassan

My Aim

To provide an easy yet comprehensive book for quick and thorough learning of the facts that are most tested in Postgraduate exams in Pakistan thus making it easier for students to look to a single book for revision. Even if a person starts studying the book just before exam, it is easy to retain and recall during the real examination.

Other Publications





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- All MCQs present in form of statements for easy revision.
- High yield and difficult to memorize things highlighted and separated for systematic study.
- Arranged in chapters that helps to read through all important stuff that could appear from the topic you have just read from theory.
- Topic wise and Chapter wise division of all possible stems/answers that a certain topic can have.
- Summary of Latest Golden files in points form.

By Dr Ahmad Hassan

Acknowledgements

Writing a book is an uphill task and this book would not have been a reality without the support system I had. My Parents, My teachers, My friends, My Colleagues, My seniors, My juniors and My Siblings.

I would like to Thank **My Father Dr. Ghazanfar Ali khan** for being there for me always and bucking me up to achieve new heights in my career.

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I would Also Like to thank **The Whole Batch 39**, Rawalpindi Medical College for their continuous support throughout.

JazzakAllah Ul Khair Everyone for being there and Helping me Finish this task.

Index/Contents

1 Growth & Development

- Growth and development are so closely related that they are usually assessed simultaneously.
- Diseases tend to have more impairment when they occur during period of rapid growth

Growth

- Change in size due to increase in number and size of cells
- Quantitatively measured (in centimeters and kilograms)

Development

- Functional maturation of organ systems
- Acquisition of skills and ability to adapt to new situations as the nervous system matures

Growth

- Rate of growth is more important than actual size
- Influenced by nutritional status, climate, season, illness and activity
- Serial measurements of growth are best indicators of health
- Measurements are plotted on centile charts and compared with normal standards

Weight

- Best index of nutrition and growth
- Average weight at birth = 3.2kg (7 pounds)
- Initially Newborn loses 10% weight (due to meconium,urine,less intake,edema)
- Regains weight by 10th day of life
- Increase in weight in first 3 months = 30g/day or 200g/week
- 3 months → 1 year = 150g/week
- Doubled at 6 months ,Trippled at 1 year,4 times at 2 years and then 5 pounds/year
- During puberty → rapid growth and weight gain

Age	Weight(pounds)	Weight(kg)	
At birth	7	3.5	
6 Months	14	7.5	2x
1 Year	21	10	3x
2 Years	28	12	4x
3.5 years	35	15	
7 years	49	22	
10 years	70	32.5	

Height (Length)

- Average at birth → 50cm
- 25 cm increase in 1st year
- 3 years → 3 feet (90cm)
- 4 years → 40 inches (100cm)
- Adult height is likely to be Twice the height at age 2 years
- After 4 years increase by 5cm/year till growth spurt
- From growth spurt to 2-3 years height increase by 9-10 cm/year

Age	Height (cm)	
Birth	50	
1 year	75	
2 Years	85	Multiply by 2 to get adult height
3 Years	95	
4 Years	100	

Head circumference

- Helps estimating brain growth
- Increases rapidly during infancy
- Sometimes head remains small due to premature union of skull sutures (Craniosynostosis)

Age	
Birth	Head circumference > Chest Circumference at birth
1 Year	Head circumference = Chest Circumference at birth
Later	Chest circumference > Head Circumference at birth

Age	Head Circumference (cm)
Birth	35
3 Months	41
6 Months	44
9 Months	46
1 Year	47
2 Years	49
3 Years	50
5 Years	51
5→12 years of age	0.5 cm increase/year

Dentition

- Not a dependable milestone since it is highly variable
- On average first deciduous tooth appears at 6 months
- Eruption of deciduous teeth is complete by 2.5 years (20 teeth)
- Shedding of deciduous teeth starts at 6 years
- Shedding of deciduous teeth complete by 12 years

Development

Summary

Fields of development with limit ages

Gross motor development

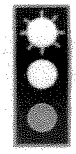
- Acquisition of tone and head control
- Primitive reflexes disappear
- Sitting
- Locomotor patterns
- Standing, walking, running
- Hopping, jumping, peddling

Gross motor	Limit ages
Head control	4 months
Sits unsupported	9 months
Stands independently	12 months
Walks independently	18 months

Vision and fine motor development

- Visual alertness, fixing and following
- Grasp reflex, hand regard
- Voluntary grasping, pincer, points
- Handles objects with both hands, transfers from hand to hand
- Writing, cutting, dressing

Vision and fine motor	Limit ages
Fixes and follows visually	3 months
Reaches for objects	6 months
Transfers	9 months
Pincer grip	12 months



Hearing, speech and language development

- Sound recognition, vocalisation
- Babbling
- Single words, understands simple requests
- Joining words, phrases
- Simple and complex conversation

Hearing, speech and language	Limit ages
Polysyllabic babble	7 months
Consonant babble	10 months
Saying 6 words with meaning	18 months
Joins words	2 years
3-word sentences	2.5 years

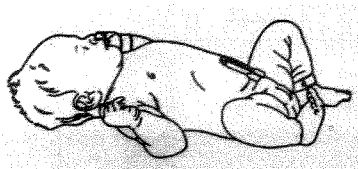
Social, emotional, behaviour development

- Smiling, socially responsive
- Separation anxiety
- Self-help skills, feeding, dressing, toileting
- Peer group relationships
- Symbolic play
- Social/communication behaviour

Social behaviour	Limit ages
Smiles	8 weeks
Fear of strangers	10 months
Feeds self/spoon	18 months
Symbolic play	2–2.5 years
Interactive play	3–3.5 years

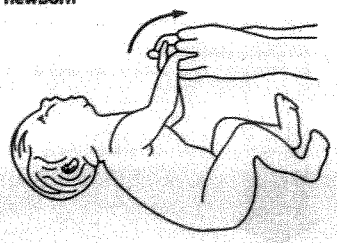
Gross motor development (median ages)

newborn



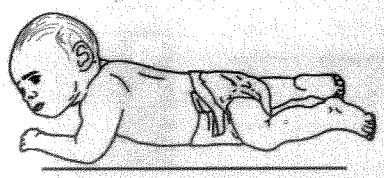
Limbs flexed, symmetrical posture

newborn



Marked head lag on pulling up

6–8 weeks

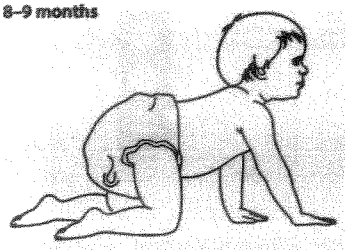


Raises head to 45° in prone

6–8 months

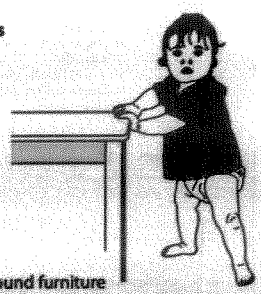
Sits without support
– at 6 months: with round back
– at 8 months: with straight back (shown)

8–9 months



Crawling

10 months

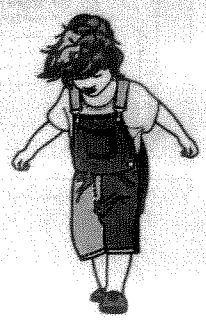


Cruises around furniture

12 months

Walks unsteadily,
broad gait, hands apart

15 months



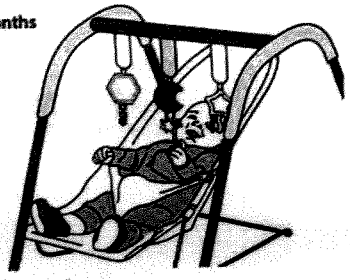
Walks steadily

Vision and fine motor (median ages)

6 weeks

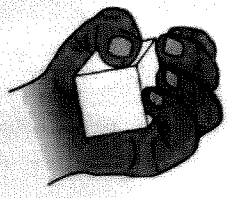
Follows moving object or face by turning the head (illustrated).

4 months



Reaches out for toys

4–6 months

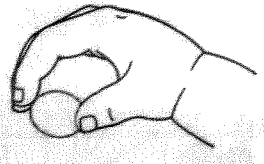


Palmar grasp

7 months

Transfers toys from one hand to another

10 months

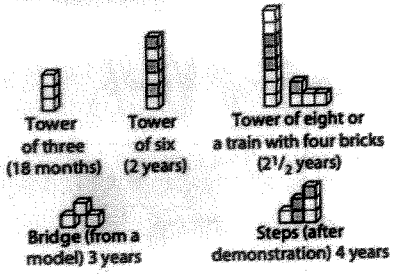


Mature pincer grip

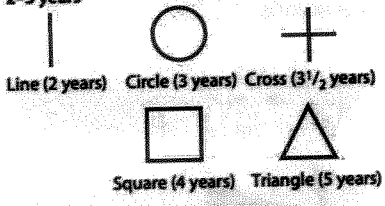
16–18 months

Makes marks with a crayon

14 months–4 years



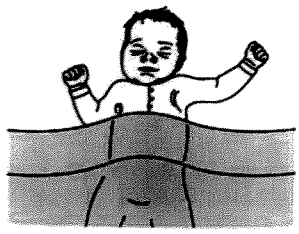
2–5 years



Ability to draw without seeing how it is done. Can copy (draw after seeing it done) 6 months earlier.

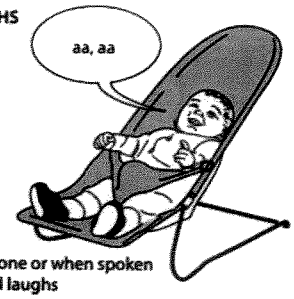
Hearing, speech and language (median ages)

NEWBORN



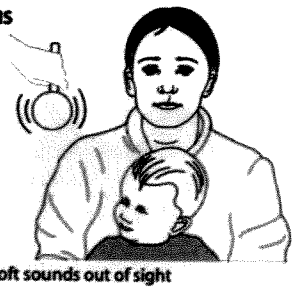
(a) Startles to loud noises

3–4 MONTHS



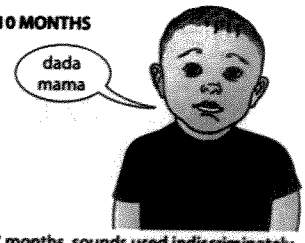
(b) Vocalises alone or when spoken to, coos and laughs

7 MONTHS



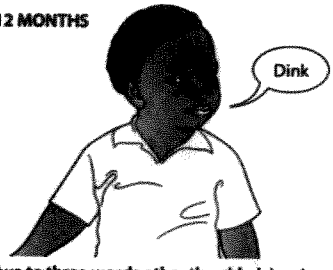
(c) Turns to soft sounds out of sight

7–10 MONTHS



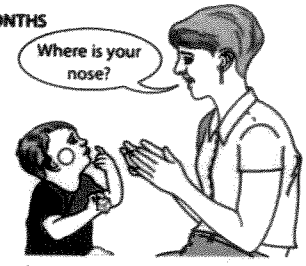
(d) At 7 months, sounds used indiscriminately. At 10 months, sounds used discriminately to parents

12 MONTHS



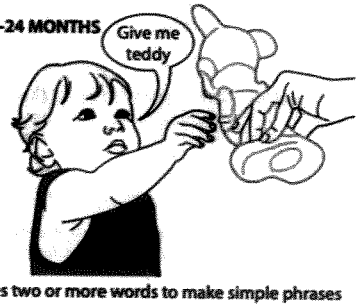
(e) Two to three words other than 'dada' or 'mama'

18 MONTHS



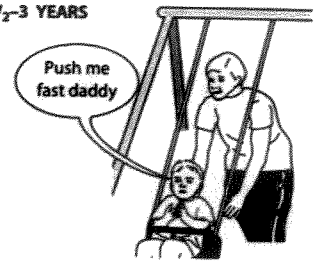
(f) 6–10 words. Shows two parts of the body

20–24 MONTHS



(g) Uses two or more words to make simple phrases

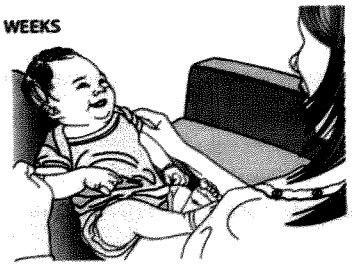
$2^1/_2$–3 YEARS



(h) Talks constantly in 3–4 word sentences

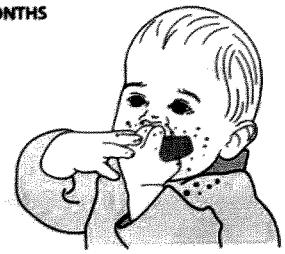
Social, emotional and behavioural development (median ages)

6 WEEKS



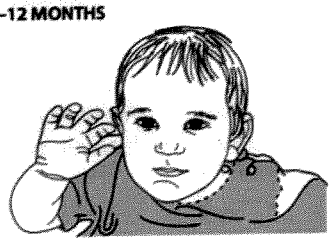
(a) Smiles responsively

6–8 MONTHS



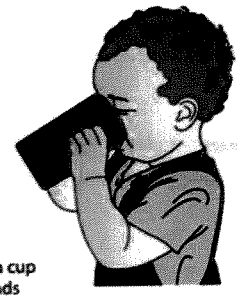
(b) Puts food in mouth

10–12 MONTHS



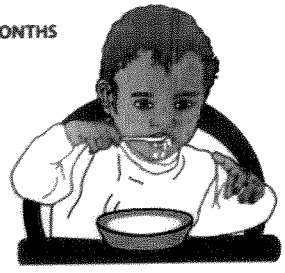
(c) Waves bye-bye, plays peek-a-boo

12 MONTHS



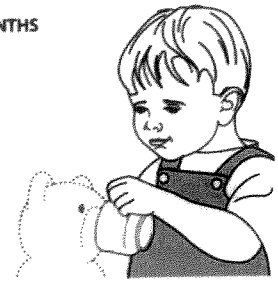
(d) Drinks from a cup with two hands

18 MONTHS



(e) Holds spoon and gets food safely to mouth

18–24 MONTHS



(f) Symbolic play

2 YEARS

(g) Dry by day. Pulls off some clothing

2.5–3 YEARS

(h) Parallel play. Interactive play evolving. Takes turn

Summary

Developmental milestones by median age

Age	Gross motor	Vision and fine motor	Hearing, speech and language	Social, emotional and behavioural
Newborn	Flexed posture	Fixes and follows face	Stills to voice Startles to loud noise	Smiles – by 6 weeks
7 months	Sits without support	Transfers objects from hand to hand	Turns to voice Polysyllabic babble	Finger feeds Fears strangers
1 year	Stands independently	Pincer grip (10 months) Points	1–2 words Understands name	Drinks from cup Waves
15–18 months	Walks independently	Immature grip of pencil Random scribble	6–10 words Points to four body parts	Feeds self with spoon Beginning to help with dressing
2½ years	Runs and jumps	Draws	3–4 word sentences Understands two joined commands	Parallel play Clean and dry

2 Immunization and Centile Charts

Definitions

Active Immunity

- Long term/Slow in onset
- Administration of all or part of microorganism or a modified product of the organism to evoke an immunological response

Passive Immunity

- Short term/Rapid in onset
- Administration of preformed antibody to a recipient

Herd Immunity

- If Number of people in a community have active immunity against an infection exceeds a critical level
- If this level is achieved then even non-vaccinated people are protected from getting the disease

Vaccine

- Protein similar to part of virulent organism that can be recognized by individual's immune system which then produces antibodies or cell mediated immunity against the antigen in the vaccine.

Live attenuated Vaccine

- Weakened virulent organism producing antigenic response by causing a MILD infection.
- Produces active immunity
- Example : BCG,OPV,MMR,Yellow fever

Killed or inactivated vaccine

- Prepared from virulent organisms or preformed antigen inactivated by heat,phenol,formaldehyde or any other means.
- Example : IPV,Pertussis,Cholera,Influenza,Rabies

Conjugated vaccines

- When polysaccharide vaccines are conjugated with other antigens in an attempt to improve the immunological response.
- Example : Linkage of H.Influenza and DPT

Toxoid

- Toxins that have been rendered non-toxic by treatment with formaldehyde but their ANTIGENICITY IS MAINTAINED
- Example : Diphtheria,Tetanus

BCG vaccine

Facts	• Bacille Calmette Guerin • **Most widely used vaccination in the world** • Live Attenuated vaccine of mycobacterium bovis • Effective in reducing the likelihood and severity of TB in infants and young children
Administration	• **Ideal age is < 1 week of age** • **If older child reports – Do Mantoux test → if negative → vaccinate** • **0.05 ml for newborn and 0.1 ml for other children Intradermal**
Normal Course	• Wheal disappears in 30 minutes • Nodule forms in 2-3 weeks • Nodule indurates and forms superficial abcess • Abcess heals in 4-6 weeks • **Scar left in a total period of 2 months**
Complications	• **Koch's Phenomenon** → accelerated reaction that completes in 10 days • Erethyma nodosum • Deep abcess and ulceration • Lymphadenopathy of supra-clavicular or axillary nodes • Generalized Tuberculosis

Rabies

Active(Human Diploid cell vaccine-HDCV)	
Pre Exposure	• 1ml S/C or I/M • 2 doses at 4 week interval • Booster after 1 year • Then booster 3-4 yearly
Post exposure	• 1 ml S/C or I/M • **0,3,7,14,28 days**

Passive Immunization	
Human Rabies Immunoglobulin • If HDCV not available • 20 IU/kg I/M , half given at wound site	**Rabies Anti-serum** • If Human Rabies Immunoglobulin not available • 40 IU/kg I/M , half given at wound site

Poliomyelitis

- **Live Attenuated polio virus I,II and III Oral Polio Vaccine (OPV) – Sabin** (Remember Soap-Sabin in the mouth)
- **Injectable Polio Vaccine (IPV) – Salk** → Inactivated polio virus
- **Remember IPV is not capable of causing poliomyelitis but OPV rarely does cause poliomyelitis**
- Even if child has suffered from poliomyelitis he should be vaccinated so as to protect him against other two types of polio viruses
- It leads to lifelong immunity if boosted by wild virus

Oral Polio Vaccine (OPV) – Sabin	
Advantages	• Easy to administer • Superior antibody response • Rapid immunity within 1 week • Provides HERD immunity
Side effects	• Paralytic polio in immunized child • Paralytic polio in close contact of immunized child
Contraindications	• HIV • Any household contact with HIV • Known Immunodeficiency • Immuno-deficient household contact
OPV not contraindicated	• Breast feeding • Current antimicrobial therapy • Mild diarrhea

Factors reducing immune response in developing countries

- Loss of cold chain
- Interference from other enteroviruses

Cold Chain

- A system of storing and transporting the vaccine, at a low temperature from the place of manufacture to the actual vaccination site is called cold chain

Diphtheria vaccine

Active immunization	
Facts	• Toxoid • Formaldehyde inactivation of diphtheria toxin adsorbed into alluminium salts to increase antigenicity • Even immunized persons can be infected by toxin producing strains of bacteria but systemic manifestations donot occur • **Almost always administered as a part of DPT** • **0.5 ml Intramuscular** • No significant adverse effect
Preparations available	• **Diphtheria toxoid** → used alone only when pertussis and tetanus toxoid are contraindicated • **DT(Diphtheria-tetanus)** →When pertussis vaccine is contraindicated (Only used in children) • **Td(Tetanus-diphtheria)** →Used in persons 7 years of age or older • **DPT (Diphtheria-Pertussis-tetanus)** →Standard immunizing agent • **Pertussis vaccine**

Passive Immunization	
Anti-diphtheric Antibody • Prophylactic : 300 IU by I/M • Therapeutic : 1200-20,000 IU by I/M	Anti-Diphtheric Serum • Horse serum for passive immunization used when Anti-diphtheric Antibody is not available • Test dose is necessary • Prophylactic dose 10,000 IU by I/M • Therapeutic dose : 40,000-120,000 IU by I/M depending on severity

Typhoid Vaccine (Typhium Vi)

- Purified Vi antigen
- One dose injectable vaccine
- Single I/M dose of 0.5ml with boosters every 2 years

Cholera vaccine

- Killed whole cell vaccine
- Not too much practical value
- Doesnot confer immunity against serotype O139

Tetanus

Active Immunization (Tetanus Toxoid)

- Toxoid → inactivated toxin with formaldehyde
- Stable and can withstand exposure to room temperature months without significant loss to potency (can withstand 37C for few weeks)
- Even if a person is previously diseased still he should be vaccinated since previous disease doesnot confer immunity
- Booster every 5-10 years
- Toxoid → induces formation of anti-toxin → neutralizes the toxin
- Newborns can be protected if mother during pregnancy is given 2 injections of toxoid at 6 week interval (2^{nd} injection atleast 4-6 weeks before delivery to provide adequate time for antibody production)
- Dose 0.5ml I/M

Side effects

- Rare since it is really safe
- Anaphylaxis , GBS,Brachial neuritis

Passive Immunization (tetanus Immunoglobulin and tetanus Anti-toxin)

Tetanus Immunoglobulin (TIG)	Tetanus anti-toxin (ATS)
• For prophylaxis and therapy • Provides Protection for 30 days • Administered without test dose • Prophylactic dose 250 IU I/M • Therapeutic dose 100-10,000 IU I/M	• Provides protection for 7-15 days • Test dose is a must before administration • Prophylactic dose 1500-3000 IU I/M • Therapeutic dose • Neonates : 10,000 IU S/C around umbilicus,10,000 IU I/M , 10,000 IU IV • Children : 40,000-60,000 IU (Half I/M and half I/V)

Pneumococcal vaccine

Facts	• **Capsular antigens to 7,9 or 23 serotypes** • Polysaccharide vaccine • Immunogenic in children over 2 years of age
High risk	• **Sickle cell disease** • **Chronic renal failure** • **Immunosupression from organ transplantation** • **Leaks of CSF** • **HIV infection**
Dose	• 0.5ml I/M or S/C

Pertussis Vaccine

Facts	• Killed/inactivated vaccine • Used as a component of DPT vaccine • 0.5 ml Intradermal • Antibodies are produced against different components of pertussis bacteria
Precautions/Contraindications (TCS-CENA)	• Temperature >40C • Collapse/Shock like state • Seizures • Crying > 3 hours • Encephalopathy • Neurological Sequelae • Anaphylaxis
Serious side effects	• Encephalopathy • Neurological Sequelae
No Contraindications	• Family history of convulsions • Family history of Sudden infant death syndrome • Family history of an adverse event following DPT administration

- If Convulsions occur within 72 hours of DPT injection ,further administration of pertussis vaccine is contraindicated .Give DT alone in this case
- After 2 years of age children should not receive Pertussis vaccine
- Children with brain damage or previous history of convulsions should not receive pertussis vaccine

Contraindications & precautions to diphtheria-, tetanus-, &/or pertussis-containing immunizations		
Vaccine component	**Contraindications**	**Precautions**
Diphtheria/ tetanus	• Anaphylaxis to vaccine ingredients	• Moderate or severe acute illness +/- fever • Guillain-Barré syndrome within 6 weeks of tetanus toxoid-containing vaccine • Arthus-type hypersensitivity reaction following diphtheria- or tetanus toxoid-containing vaccine

Pertussis	• Anaphylaxis to vaccine ingredients
	• Progressive neurologic disorder (eg, uncontrolled epilepsy, infantile spasms)
	• Encephalopathy within a week of previous vaccine dose
		• Moderate or severe acute illness +/- fever
		• Reactions to previous doses:
		  • Seizure within 3 days
		  • Temperature ≥40.5 C (105 F) within 2 days
		  • Hypotonic-hyporesponsive episode within 2 days
		  • Inconsolable, persistent crying within 2 days

Measles

Facts	• Live attenuated vaccine • Produces neutralizing antibody • Immunity is life-long if boosted by wild virus • Immunity is shorter when no wild virus is circulating • **Mostly as a component of MMR (Measles-Mumps-Rubella)**
Dose	• 0.5ml Subcutaneously
Contraindications (MMR)	• **Anaphylaxis to neomycin** • **Anaphylaxis to gelatin** • **Pregnancy** • **Immunodeficiency** • **Long term immunosuppressant therapy**
No Contraindications	• TB or positive PPD • Simultaneous TB skin testing • Breast feeding • Pregnancy of MOTHER
Side effects	• Mild febrile illness • Morbiliform rash • Febrile convuslions • Encephalitis
Post-exposure prophylaxis	• If within 72 hours of exposure → give **vaccine** immediately • If more than 3 days but less than 6 days of exposure → Immunoglobulin given I/M

Hepatitis B Vaccine

Facts	• Given to all infants,children and adolescents • Not contraindicated in immunosuppressed or in pregnant women
Administration	• 3 I/M doses • Increasing the interval between 1st & 2nd doses has little effect on immunogenicity • **Longer the interval b/w 2nd and 3rd dose higher the titers of anti-HBs** • **0.5 ml if <19 years** • **1ml if > 19 years**
Schedule	• **0,1 and 6 months**
Infants born to HBsAg +ve mothers	• **0.5ml HBIG within 12 hours after birth and Hepatitis B vaccine at another site** • **2nd dose of vaccine = 1 month** • **3rd dose of vaccine = 6 months** • **12-15 months → tested for antibody to HBsAg → if positive means immunization has failed and infant is a chronic carrier** • **12-15 months → tested for antibody to HBsAg → if Negative→means vaccination is effective**
Infants born to mothers of unknown HBsAg status	• **Schedule of 0,1,6 months and then test (Same as previous) but withhold HBIG until HBsAg status is known and if found positive give HBIG within 7 days of life**

Post Exposure prophylaxis	• **Unvaccinated → HBIG within 1 day and Vaccine within 7 days** • **Previously vaccinated → retested for anti-HBs → if levels are adequate (>10mIU/ml) then no treatment but if levels are less than 10mIU/ml then booster dose is required** • **Individuals who cannot be tested → give HBIG** • **Chronic carrier patient → all household contacts should receive vaccination**

EPI

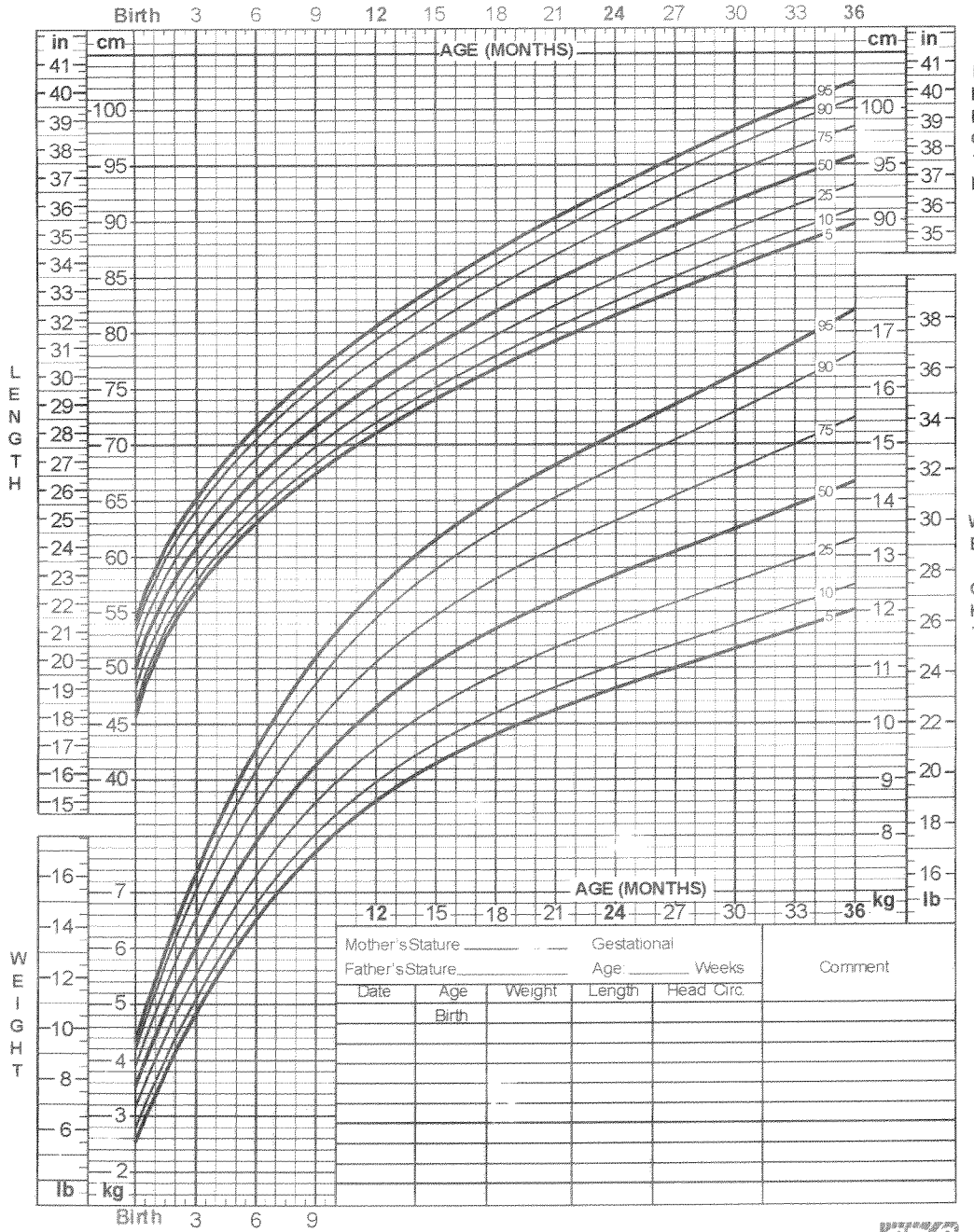
EPI (Expanded Programme of Immunization)

Age	Vaccines	Dose	Route
At Birth	OPV-0	2 drops	Oral
	BCG	0.05ml	I/D
	Hepatitis B	0.5ml	I/M
6 Weeks (ORPP)	OPV-1	2 drops	Oral
	Rota-1	1.5 ml	Oral
	Pentavalent-1	0.5 ml	I/M
	PCV-1		
10 Weeks (ORPP)	OPV-2	2 drops	Oral
	Rota-2	1.5 ml	Oral
	Pentavalent-2	0.5 ml	I/M
	PCV-2		
14 Weeks (ORPI)	OPV-3	2 drops	Oral
	Rota-3	1.5 ml	Oral
	Pentavalent-3	0.5 ml	I/M
	IPV		I/M
9 Months	Measles-1	0.5 ml	S/C
15 Months	Measles-2	0.5 ml	S/C
20-23 Months	DPT Booster (If >24 Months =DT Booster)	0.5 ml	I/M
	OPV Booster	2 drops	Oral
5 Years	DT Booster	0.5 ml	I/M
	OPV Booster	2 drops	
10 Years	Td Booster	0.5 ml	I/M

Birth to 36 months: Boys
Length-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____



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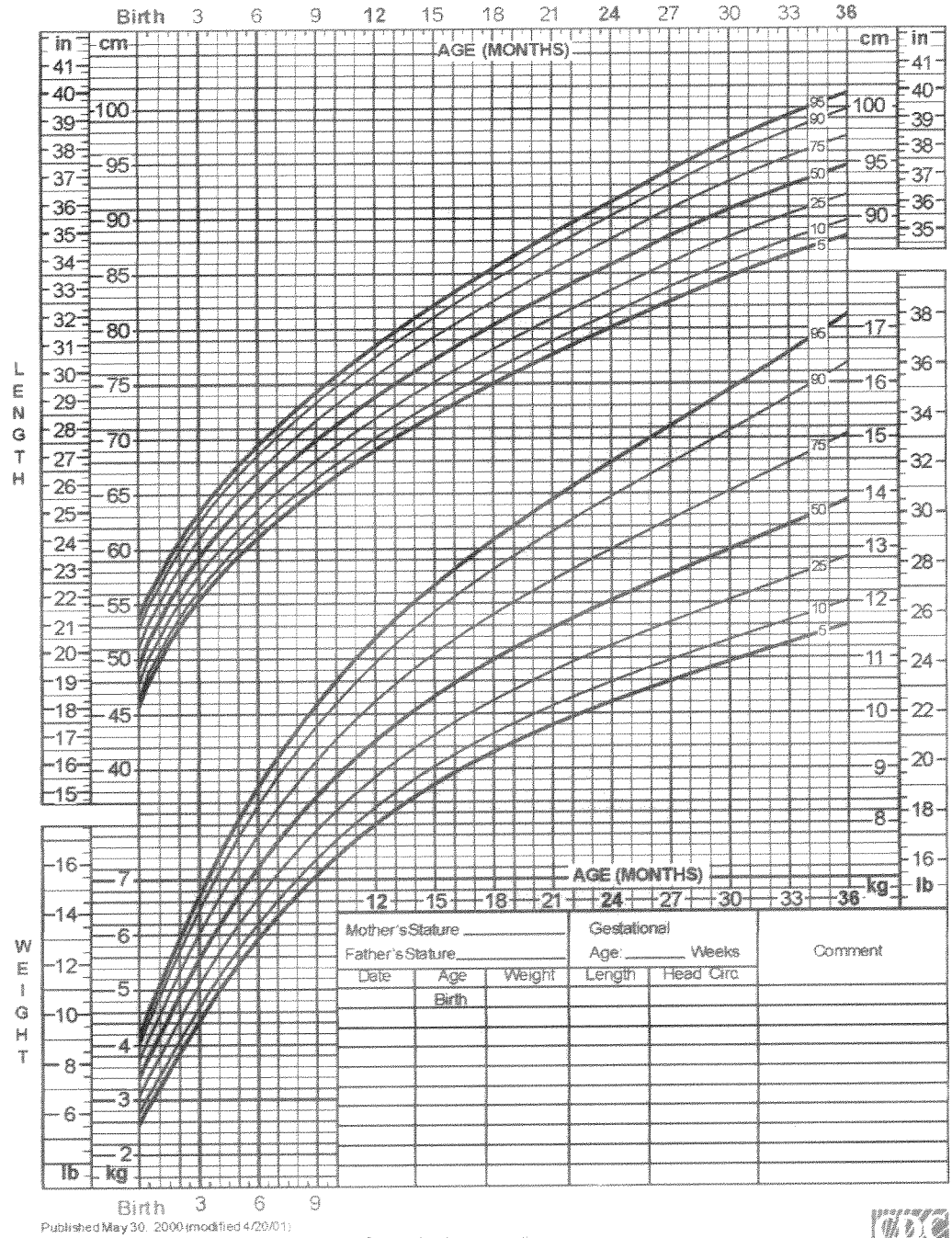
Birth to 36 months: Girls
Length-for-age and Weight-for-age percentiles
NAME
RECORD #
Birth 3 6 9 12 15 18 21 24 27 30 33 36
AGE (MONTHS)
LENGTH
WEIGHT
in
cm
lb
kg
95
90
75
50
25
10
5
Mother's Stature
Father's Stature
Gestational Age: Weeks
Date
Age
Weight
Length
Head Circ.
Comment
Birth
Published May 30, 2000 (modified 4/20/01)
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000)
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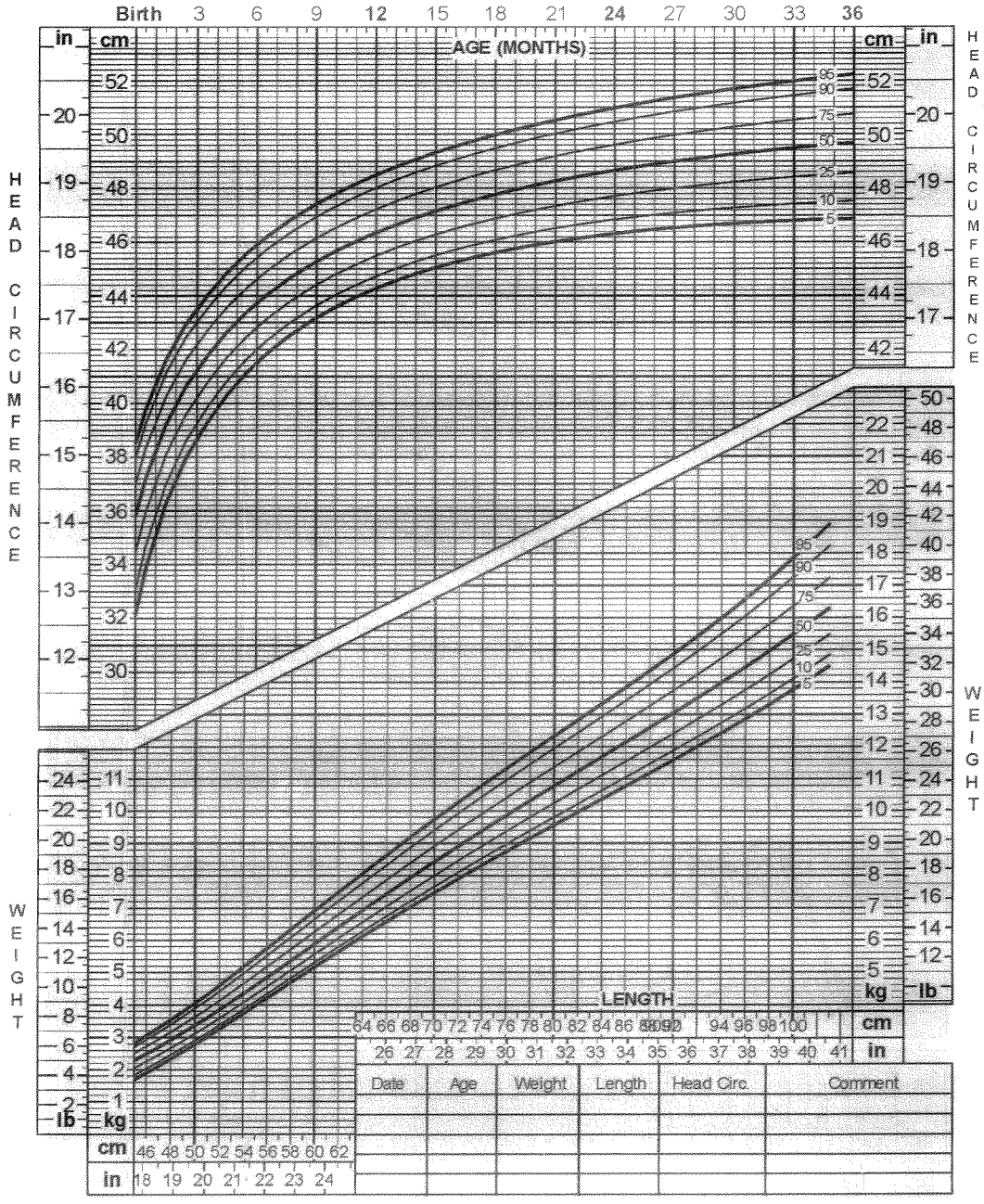
Birth to 36 months: Boys
Head circumference-for-age and Weight-for-length percentiles

NAME _____

RECORD # _____



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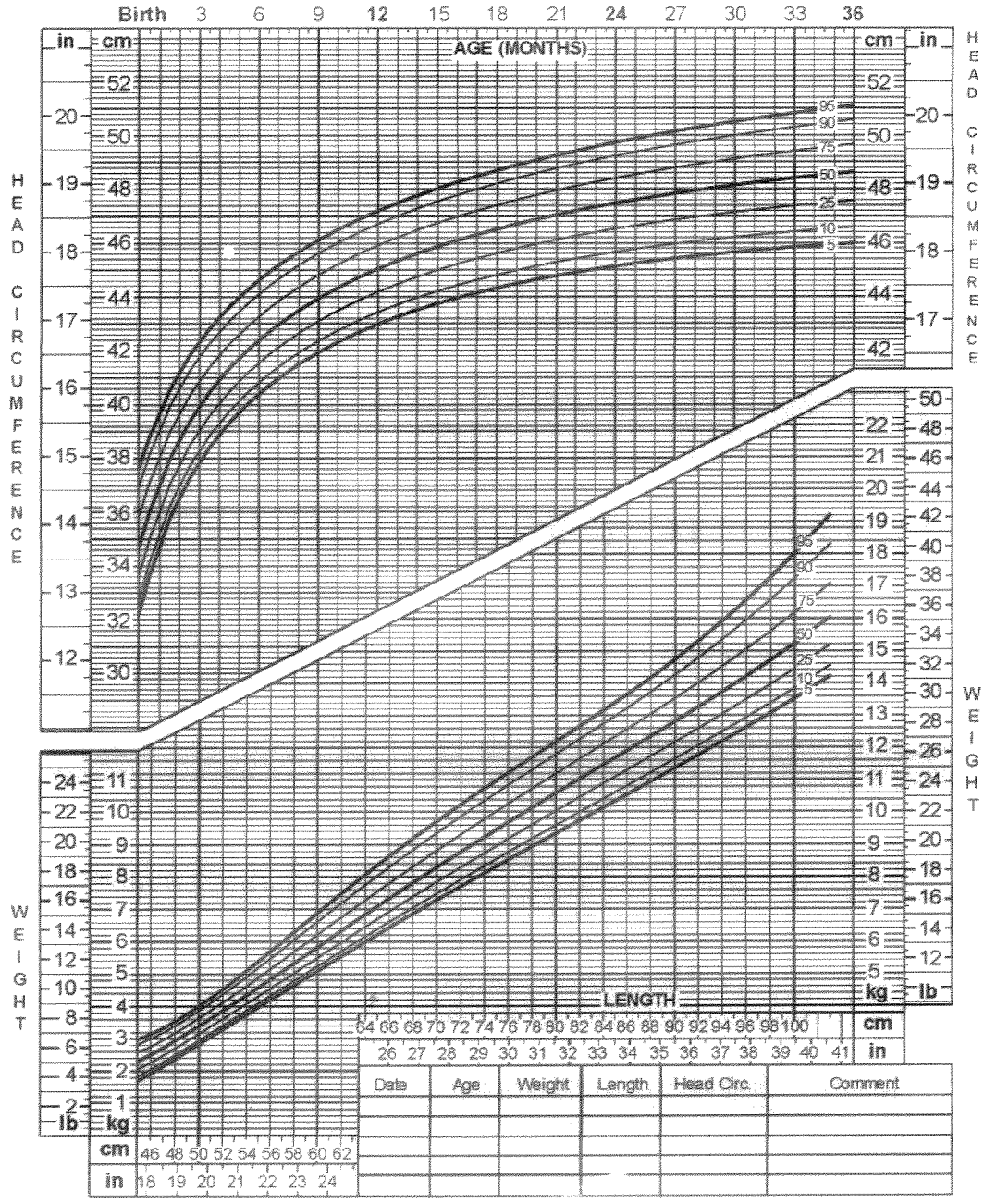
Birth to 36 months: Girls
Head circumference-for-age and Weight-for-length percentiles

NAME _____

RECORD # _____



Date	Age	Weight	Length	Head Circ.	Comment

Published May 30, 2000 (modified 10/16/00).
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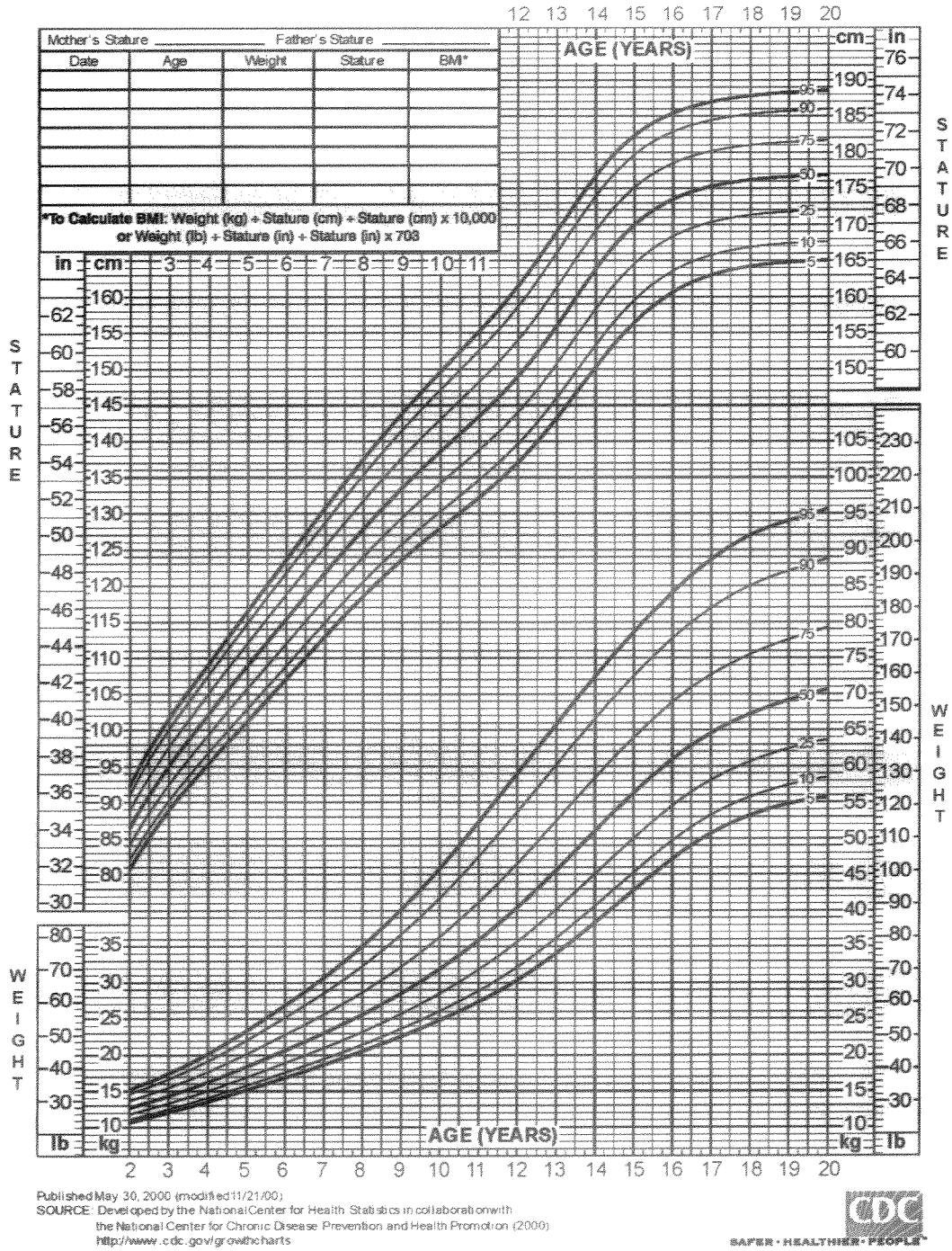
2 to 20 years: Boys
Stature-for-age and Weight-for-age percentiles
NAME
RECORD #
Mother's Stature
Father's Stature
Date
Age
Weight
Stature
BMI*
*To Calculate BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703
AGE (YEARS)
STATURE
WEIGHT
in
cm
lb
kg
95
90
75
50
25
10
5
Published May 30, 2000 (modified 11/21/00)
SOURCE: Developed by the National Center for Health Statistics in collaboration with
the National Center for Chronic Disease Prevention and Health Promotion (2000)
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CDC
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2 to 20 years: Girls
Stature-for-age and Weight-for-age percentiles

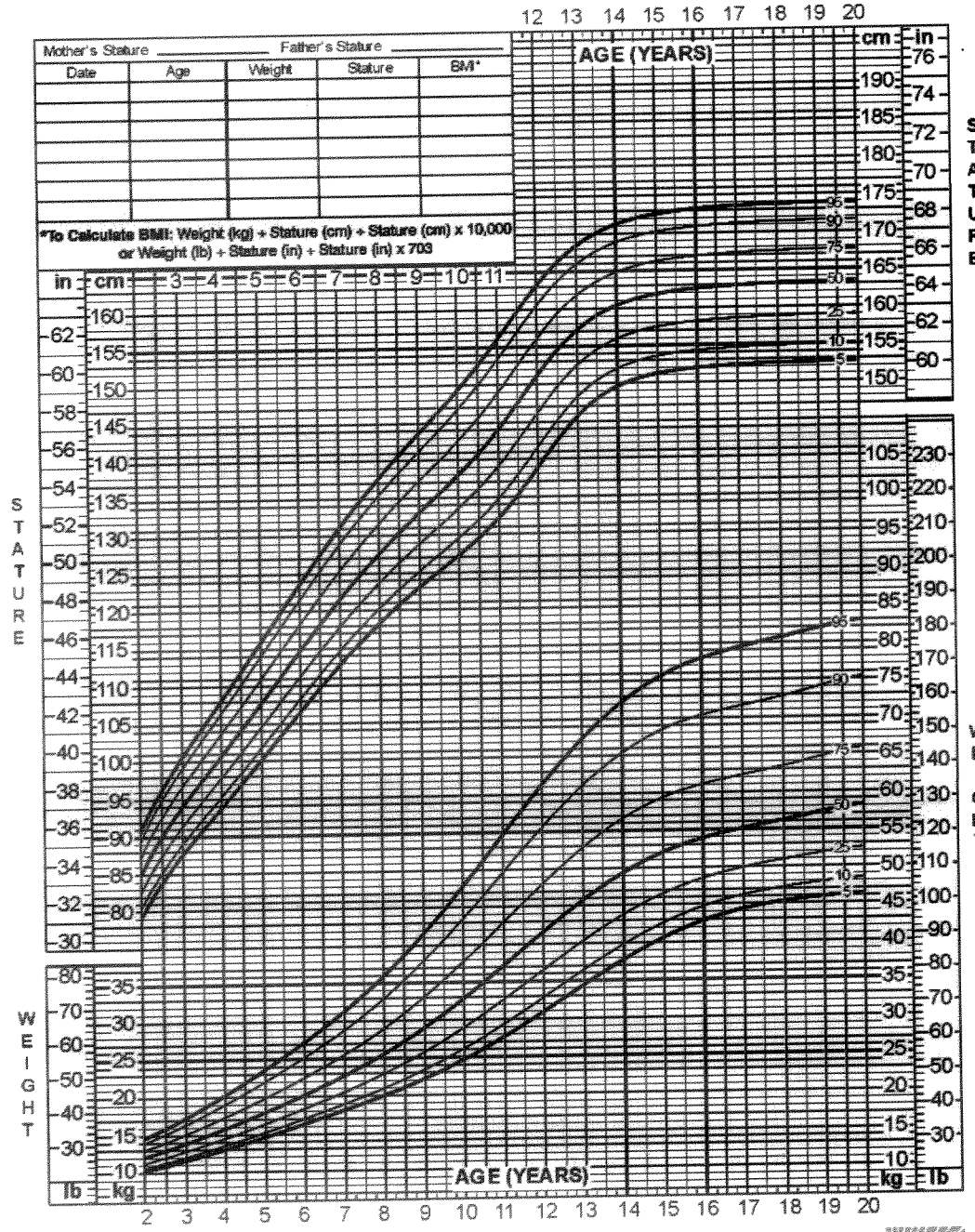
NAME _____

RECORD # _____

Mother's Stature _____ Father's Stature _____

Date	Age	Weight	Stature	BMI*

*To Calculate BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



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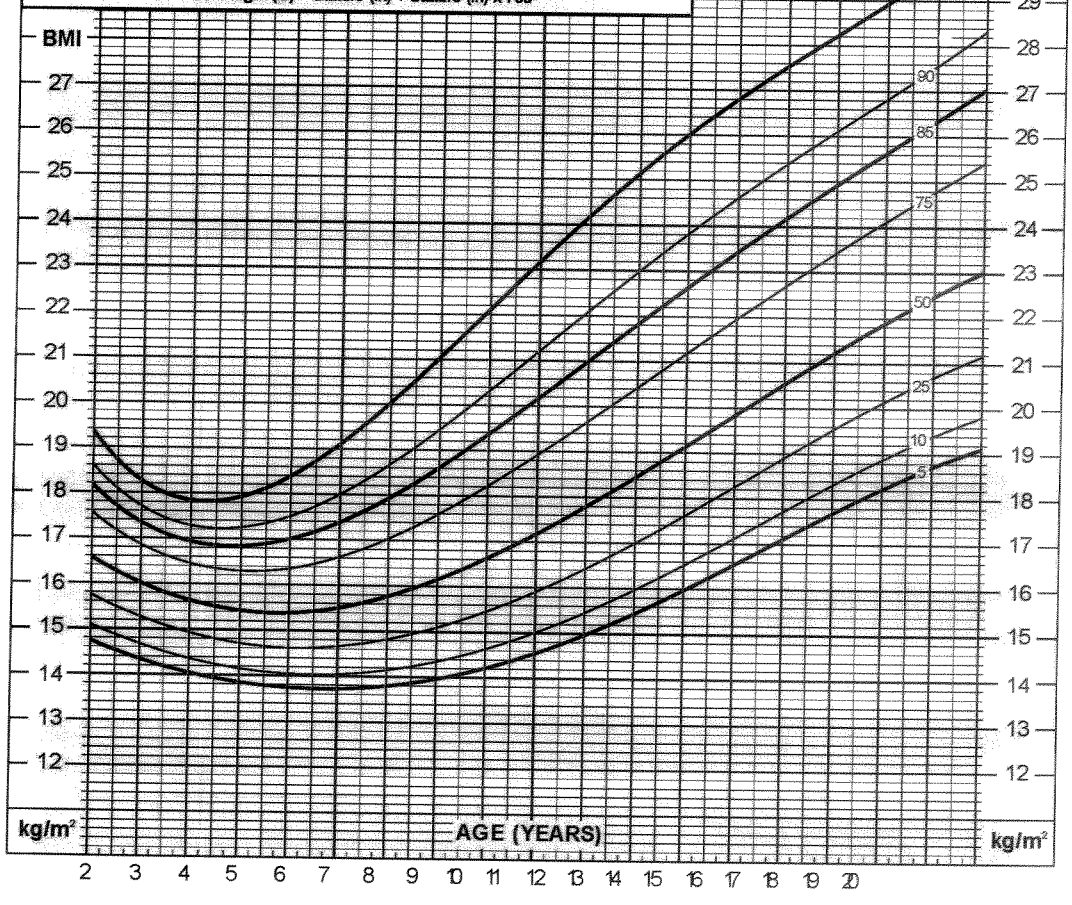
2 to 20 years: Boys
Body mass index-for-age percentiles

NAME _____

RECORD # _____

Date	Age	Weight	Stature	BMI*	Comments

*To Calculate BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



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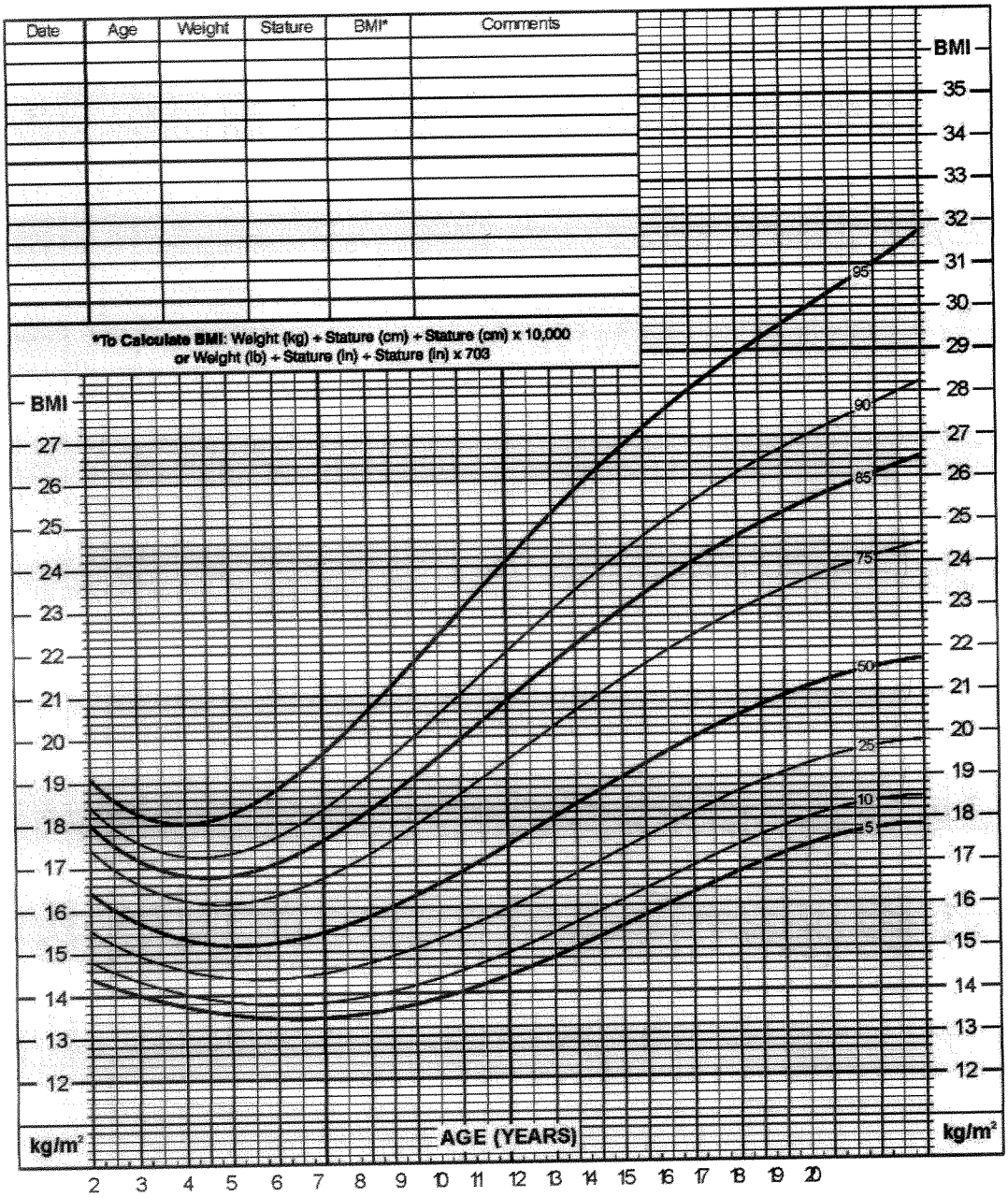
2 to 20 years: Girls
Body mass index-for-age percentiles

NAME _____

RECORD # _____

Date	Age	Weight	Stature	BMI*	Comments

*To Calculate BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



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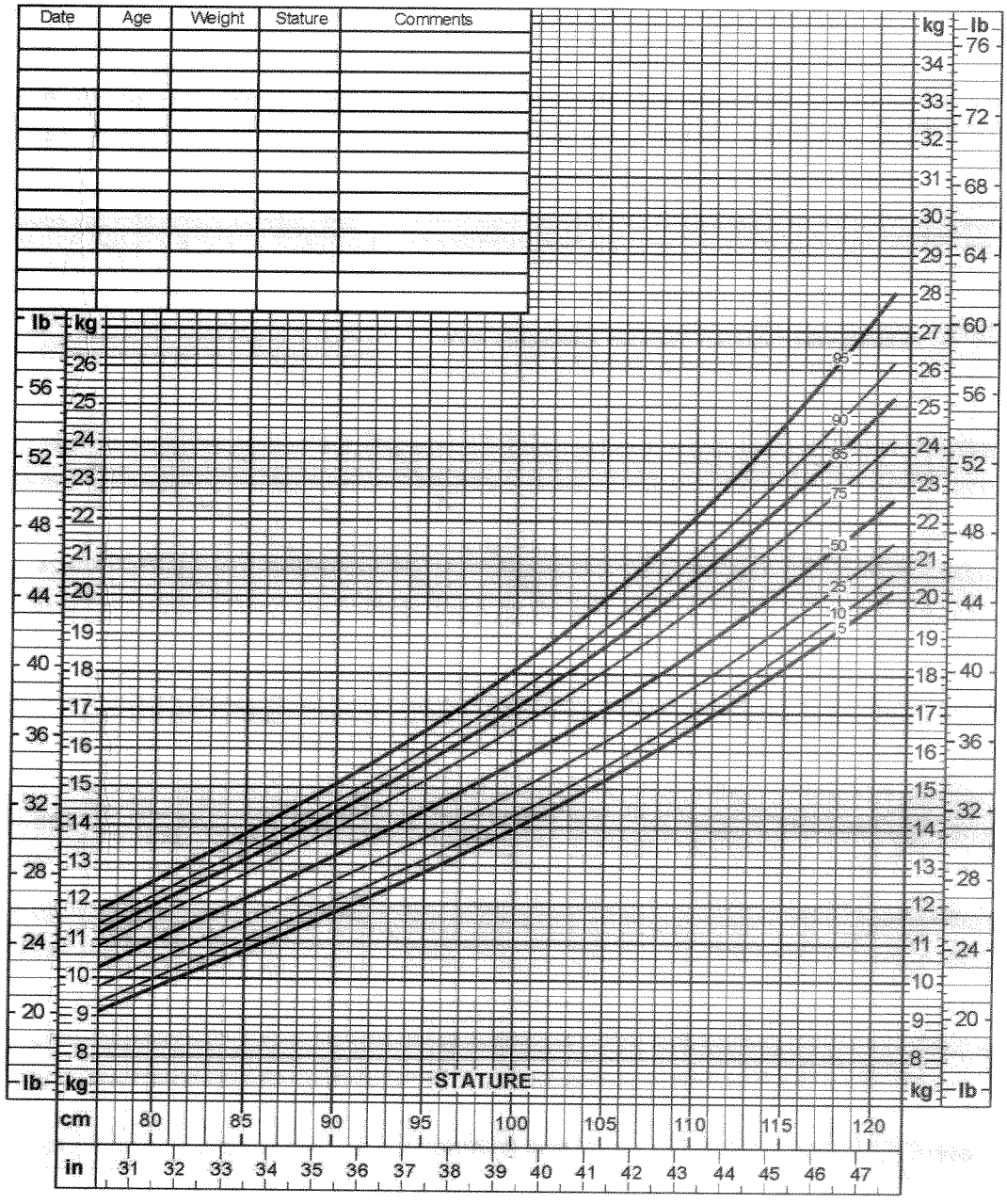
NAME _____

RECORD # _____

Weight-for-stature percentiles: Boys

Date	Age	Weight	Stature	Comments



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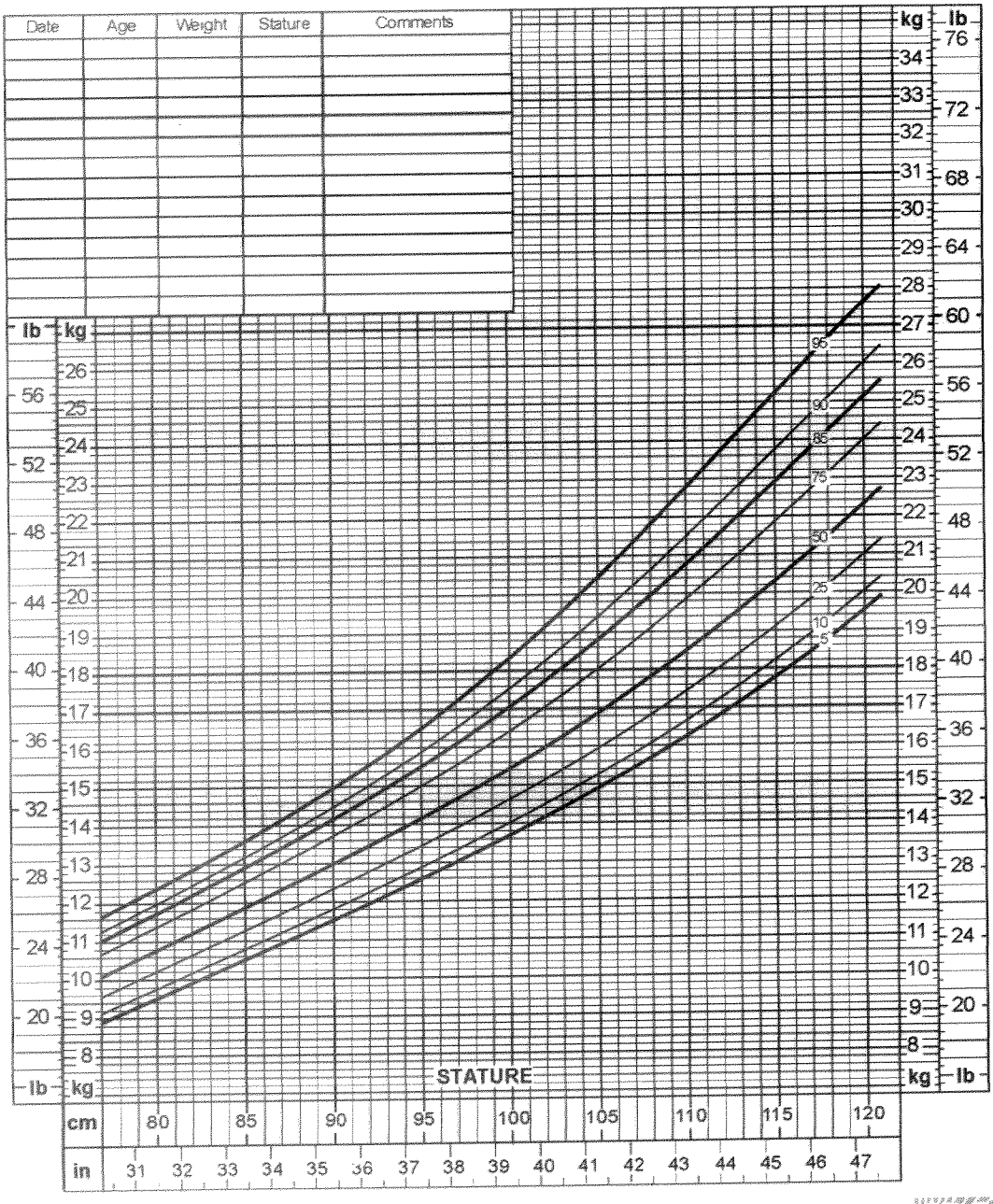
NAME _____

RECORD # _____

Weight-for-stature percentiles: Girls

Date	Age	Weight	Stature	Comments



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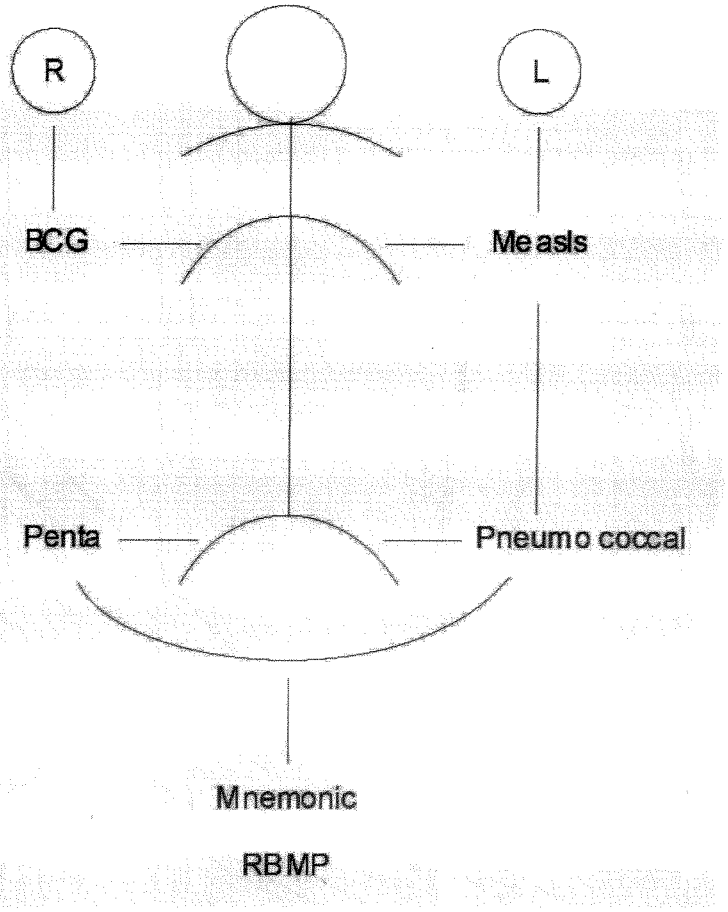
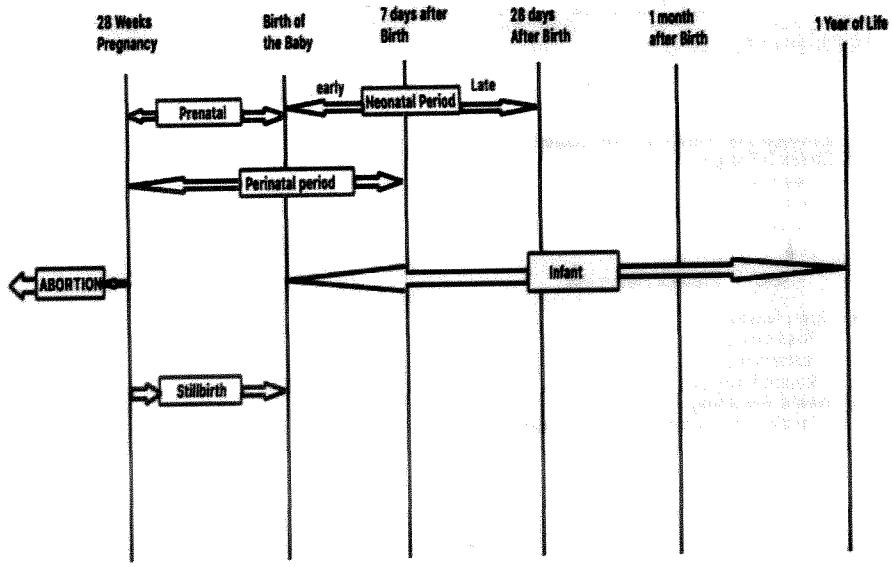

R
L
BCG
Measls
Penta
Pneumo coccal
Mnemonic
RBMP

7 Neonatology

DEFINITIONS



NEONATAL Period	• First 28 days of Life • First 7 days = Early Neonatal Period • 8-28th day of Life = late Neonatal Period
Perinatal Period	• From 28th Week of Gestation to 7 days after Birth
Prenatal Period	• From 28th Week of Gestation to the Birth of the baby
Post Natal Period	• After Birth
Infant	• 1st Year of Life (first 365 days)
Abortion	• Expulsion of dead fetus prior to 28 weeks pregnancy (Pakistan) and 24 Weeks gestation (developed countries)
Still birth	• Expulsion of dead fetus after 28 weeks of Pregnancy
Perinatal Mortality	• Number of Stillbirths and early neonatal deaths (upto 7 days) per 1000 TOTAL births
Neonatal Mortality	• Number of deaths in first 28days of Life per 1000 LIVE births
Infant Mortality Rate	• Number of deaths in first 365 days of life per 1000 LIVE births
Newborn Classification by Weight	**Incredibly Low Birth Weight** <750g **Extremely Low Birth Weight** <1000g/1Kg **Very Low Birth Weight** <1500g/1.5kg **Low Birth Weight** <2500g/2.5kg

Newborn Classification by Gestation	**Preterm**	Baby Born less than 37 completed weeks of Gestation
	Full term	Baby born b/w 37-42 weeks gestation
	Post term	Baby born after 42 weeks of Gestation
Newborn Classification by Weight and Gestation	**Small for gestational Age**	Less Than 10th centile for weight expected for gestation
	Appropriate for Gestational age	b/w 10th and 90th centile for weight expected for gestation
	Large for Gestational Age	More than 90th centile for weight expected for gestation

Neonatal Resuscitation

Neonatal Resuscitation Steps

1. Evaluate the infant (APGAR Score)
2. Check 3 Things:
   - Respiration
   - Heart Rate
   - Colour

   ↓

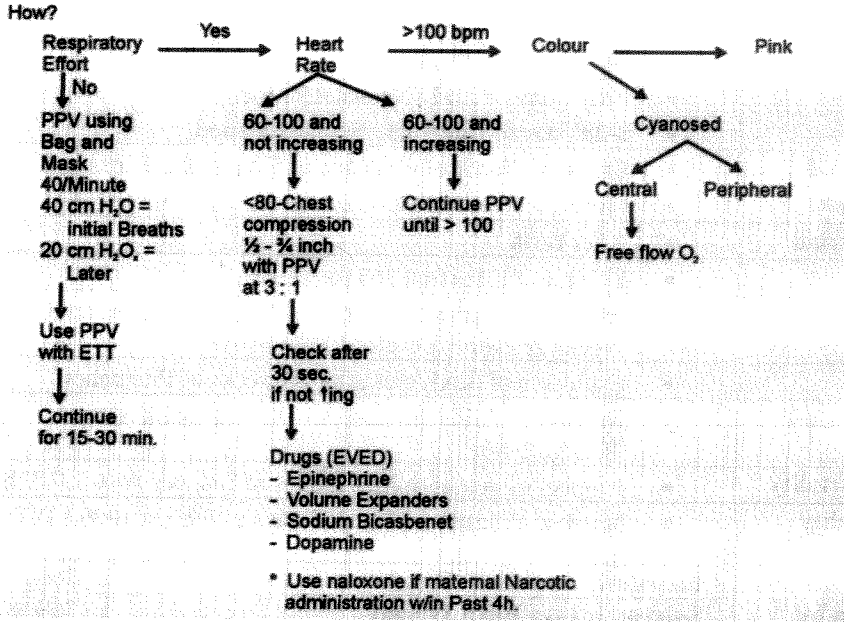
   - ABC + Prevent Heat Loss

a) Open Airway
   - Positioning
   - Suctioning
   - Check Patency

b) Initiate Breathing
   - Tactile Stimulation (Slap, Flick Sole / Rub Back)
   - PPV using bag + Mask or Bag + ETT

c) Maintain Circulation
   - Chest Compressions
   - Medications

How?



* Discontinue resuscitation if no RR or inaudible heart or pupils dilated and fixed after 20 Minutes

Care of the Newborn

The **BV** works in **PCB To Wash** and **M**easure **B**aby's **G**lucose

1)**B**reast feeding early (2-4 hours of life)
2)**V**itamin K Injections(prevents HDN)
3)**P**ositioning of the baby to avoid aspiration(supine/right side)
4)Coomb's testing
5)hepatitis **B** Vaccine
6)**T**emprature regulation(Thermoneutral environment)
7)**W**ash hands to handle baby ,Wash Baby's umblical stump with antibiotic cream or spirit 3-4 times daily
8)**M**easure: Weight,Length,Head circumferrence
9)**B**athing delayed-since causes hypothermia (Most common cause)
10)**G**lucose testing

Temperature Regulation	**Hypothermia**	**Hyperthermia**
Defintition	**Core temperature <35C**	**Core temperature >37.5**
Causes Risk factors And Mechanism	Heat lost via conduction,convection,radiation and evaporation **Risk factors** • **Preterm infants** -Little Subcutaneous fat -High ratio of Surface area : Body weight -Reduced glycogen and brown fat stores Immature shivering and vasoconstriction mechanisms • **IUGR** -Little Subcutaneous fat -High ratio of Surface area : Body weight • **Birth Asphyxia** Poor peripheral circulation	**Causes** • Infections • Dehydration • Over-wrapping • Increased environmental temperature • Maternal fever during delivery
Clinical Findings	• Cold to touch infant • Weak suck and cry • Decreased activity • Slow irregular breathing • Slow heart rate (Bradycardia) • Cyanosis • Generalized bleeding (DIC)	- • Increased Metabolic rate • Increased O2 consumption) • Tachycardia(increased HR) • Tachypnea(Increased RR) • Irritability • Dehydration • Acidosis • Brain damage • Death
Compx	I remember these by knowing that everything decreases with decreased temperature • Glucose(Hypoglycemia) • pH(Acidosis) • O2(Hypoxia) • Decreased perfusion • Decreased HR(Sinus bradycardia) • Apnea	

Temperature Regulation	Hypothermia	Hyperthermia
	And there are 3 Blood issues issues • DIC • Intraventricular hemorrhage • Pulmonary hemorrhage •	
Mx	• Prevention (best management) • Remove precipitating issue • Donot bathe the infant • Dry the infant • Place in radiant warmer • Make him wear a hat • Ensure 23-24C temperature of room • 26-28C temperature of room if high risk infant • Slow rewarming • Full term – radiant warmer • Preterm-incubator • Manage for sepsis	• Remove the causative factor • Treat infection if any • Run sepsis screening • Turn off any heat source • Remove excessive clothing • Tepid water sponge bath • Paracetamol
Thermoneutral Environment	It is the environmental temperature at which the heat production and O2 consumption is minimal yet the core temperature is maintained within the normal range	

Nutritional Management of Newborn

- Breast Milk is the best and most easily tolerated food

Why Calories are required in a newborn

- Maintain Weight 50-60Kcal/Kg/day
- Induce Weigh gain 100-120 Kcal/Kg/day

What source of calories should comprise of

Lipids	35% (4-6g/kg/day)
Proteins	15% (2.25-4 g/kg/day)
Carbohydrates	50%(11-15g/kg/day)

Nutritional Considerations

- Term infant is fed on demand
- 34-38 Weeks(healthy preterm) → Bottle,breast or gavage
- Less than 34 Weeks (preterm) → Feeding tube
- Weight < 1Kg(1000g) →Continuos gastric feeding
- Vitamin supplementation to all babies once feeding is established till 2 years age
- Iron supplementation to babies less than 2.5 kg birth weight or <36 weeks gestation till solid food intake starts
- Foilc acid supplementation to all babies with hemolytic anemia till 8 weeks age
- Fluid therapy for an infant

Age	Full term (ml/mg/day)	Preterm/SGA (ml/mg/day)
Day 1	40	60
Day 2	60	80
Day 3	80	100
Day 4	100	120
Day 5	120	150
> Day 5	180-200	200-220
Add 30ml/day for phototherapy		
Add 20-30 ml/day for radiant heater		

Total Parenteral Nutrition

- Total parenteral nutrition Intravenous administration of all nutrients (Fats, protein, carbohydrates, vitamins and minerals)
- Parenteral nutrition is supplemental intravenous administration of nutrients
- Enteral nutrition is oral or gavage feeding

Indications

- NPO x 2 weeks
- Periods of poor intake for 2-3 days to 2 weeks

Newborn	Older children
• LBW <1000g • Severe respiratory problem All other indications are related to GIT • Intestinal failure (sepsis,NEC) • Diaphragmatic hernia • TEF • Gastroschisis • Congenital GIT malformations • Meconeum ileus	• Intestinal failure • Malabsorption • Short gut syndrome • Intestinal obstruction • Paralytic ileus • Diarrhea/Vomiting • Hypermetabolic states

Monitoring TPN	Complications TPN
• Daily Weight • Strict intake/output record • Start with peripheral vein and then replace with central vein if prolonged TPN is required • Vital sign monitoring • Avoid infection by meticulous asepsis • Fat solution must be given by a different line or by side channel • All vitamins,trace elements,electrolytes added to glucose infusion • No Fats if on phototherapy • No Aminoacids if has hyperbilirubinemia • Control hyperglycemia • Send tip of catheter for culture • Check blood and urine glucose daily	(THIS-MAT) • **T**race elements deficiency (Cu,Zn) • **H**epatic issues (Cirrhosis,Hepatomegaly) • **H**ydropneumothorax • **I**nfections • **S**kin Sloughing • **M**etabolic ➢ Hyperglycemia ➢ Hypoglycemia ➢ Hypomagnesemia ➢ Hypocalcemia ➢ Hyperlipidemia

Monitoring TPN	Complications TPN
• CBC,S/E.LFTs.RFTs,Urea/Cr checked weekly • Ca,PO4,Mg checked 2 weekly	➢ Hyperaminoacidemia • Acidosis • Arrhythmias • Thrombosis

Birth Asphyxia

Definition	Lack of oxygen due to failure of initiation of breathing		
Risk Factors	**Maternal**	**Placental**	**Fetal**
	• Hypertension • Hypotension • DM • Cardiac or pulmonary disease • Pelvic abnormality • Nephritis • Infections • Uterine tetany	• Abruption • Placental insufficiency (toxemia,postmaturity)	• Cord prolapse or compression • Abnormal Lie/presentation • Postmaturtity • Anemia • Infections • Cerebral abnormalities • Pulmonary and Cardiac abnormalities
Patho	• CNS → Fluid leaks leading to cerebral edema and death • Increased Lactate and decreased phosphate levels • Failure of energy dependent ionic pumps → membrane damage and thus neuronal damage • Full term infant → cortical necrosis and parasaggital ischemic injury → focal seizures and hemiplegia • Preterm infants → Periventricular leukomalacia (White matter necrosis) ,Basal ganglia damage and IVH		
Diagnosis	**Before Birth** • Identify Risk factors • Signs of Fetal distress (Bradycardia,Late decelerations,USG showing decreased fetal activity , Thick meconium in liquor) **At Birth** • Depression of APGAR (</= 3 for more than 5 minutes)		
Compx (HAM-DRUGS)	• Hypoxic Ischemic Encephalopathy • Acute Tubular Necrosis • Myocardial Ischemia • DIC • Respiratory distress syndrome,Meconeum aspiration • Ulcerations in GIT • Glucose (hypoglycemia) • Shock Liver (Raised LFTs)		

Mx	**Supportive** • Nurse in thermoneutral environment • Record vitals and take input/output record • Pass NG and aspirate the stomach • Monitor glucose • Measure ABGs • Treat hypoxia with O2 and IPPV • Treat acidosis with NaHCO3 • Maintain fluid and electrolyte balance • Review infection risks and use antibiotics **CNS** • Treat seizures → phenobarbitone • Cerebral edema → fluid restriction and Mannitol **CVS** • Hypotension → plasma and inotropic support • Treat Cardiac failure → digoxin and inotropic support **Renal** • Monitor Urine output • Treat acute renal failure if present **Pulmonary** • Meconium aspiration → tracheal suctioning and respiratory support • Apnea and CO2 retention → IPPV • Pulmonary hypertension → Inhaled NO **GIT** • NEC → antibiotics and avoid enteral feeding • Correct metabolic abnormalities

APGAR Score

- Simple, painless and effective check used by midwives and doctors to assess your newborn's health
- **At one minute after birth, and again at five minutes after birth.**
- Score out of 10
- This Apgar score will help to decide if your baby needs any immediate treatment during the first moments of his life.
- Each factor in the table below is given a score between zero and two, which are then added up to give the Apgar score for each check
- **APGAR score at 15 and 20 Minutes are more strongly correlated with asphyxia**
- Some conditions that may keep scores artificially low
  - Maternal anesthesia

- Trauma
- Some Neuromuscular disorders
- Metabolic or Infectious insults to CNS
- Cardiac or pulmonary malformations

	Sign	0 points	1 point	2 points
A	Appearance/ color	Completely blue/pale	Body pink, extremities blue	Completely pink
P	Pulse	Absent	<100/min	>100/min
G	Grimace/ reaction	Absent	Grimace/ whimper	Cough/ sneeze/cry
A	Activity/ muscle tone	Limp	Some flexion	Active/ spontaneous
R	Respiratory effort	Absent	Slow, weak cry	Regular, good cry

- **Score 10 is rare**
- **Score** 7-9 → mostly, require no further intervention
- **Score** <7 → require further evaluation and resuscitation,pulse oximetry monitoring and positive pressure ventilation.
- If heart rate <60 → chest compression may be given
- APGAR score at 5 min is useful in assessing response to preliminary intervention
- Most concerning factors are heart rate and respiration.
- Extremity cyanosis is common and may resolve in 1-2 days. Central body cyanosis raises concern for respiratory or cardiac problems
- Maternal factors that ↑ risk for resuscitation
- very young maternal age
- H/o DM
- H/o HTN
- H/o Substance abuse

Prematurity

Definition

- Live born infant delivered before 37 weeks from the first day of Last menstrual period

Etiology

Maternal	Uterine	Fetal	Others
• Teenage pregnancy • Twin pregnancy • Malnutrition • Anemia • Smoking • Drug abuse • Chronic illness • (DM,HTN) • Infection • Illegitimate birth	• Bicornuate uterus • Incompetent cervix • Placenta previa • Abruptio placenta • Placental dysfunction	• Fetal distress • Multiple gestation • Chromosomal issues • Intrauterine infections • Erythroblastosis	• Polyhydramnios • PROM • Trauma • Iatrogenic

Problems/Complications of prematurity

Short term (Immediate/Acute)

- Hypothermia
- Hypoglycemia → lack of glycogen stores
- Hypocalcemia → immaturity of hormonal control system
- Intraventricular hemorrhage→ immature vasculature and clotting factor deficiency (treatment = vitamin K)
- Retinopathy of prematurity→ Retrolental fibroplasia
- Feeding issues→uncoordinated sucking and swallowing,GERD
- Necrotising enterocolitis →immature gut and enzyme deficiencies
- PDA
- Respiratory issues → IRDS and Apneic spells
- Liver immaturity→ leads to prolonged physiological jaundice
- Anemia of prematurity →decreased iron stores , Vitamin E deficiency , physiological anemia
- Metabolic bone disease → Rickets (due to calcium and vitamin D deficiency)

Long-Term (LPC)

- Lung → Bronchopulmonary dysplasia
- Poor growth →due to vitamin and iron deficiency
- CNS dysfunction →CP,learining issues ,deafness,mental subnormality ,hydrocephalus

Assessment of gestational age (Ballard Score)

MATURATIONAL ASSESSMENT OF GESTATIONAL AGE (New Ballard Score)

NAME _____ SEX _____

HOSPITAL NO. _____ BIRTH WEIGHT _____

RACE _____ LENGTH _____

DATE/TIME OF BIRTH _____ HEAD CIRC. _____

DATE/TIME OF EXAM _____ EXAMINER _____

AGE WHEN EXAMINED _____

APGAR SCORE: 1 MINUTE _____ 5 MINUTES _____ 10 MINUTES _____

NEUROMUSCULAR MATURITY

NEUROMUSCULAR MATURITY SIGN	SCORE							RECORD SCORE HERE
	-1	0	1	2	3	4	5	
POSTURE								
SQUARE WINDOW (Wrist)	 >90°	 90°	 60°	 45°	 30°	 0°		
ARM RECOIL		 180°	 140 - 180°	 110 - 140°	 90 - 110°	 <90°		
POPLITEAL ANGLE	 180°	 160°	 140°	 120°	 100°	 90°	 <90°	
SCARF SIGN								
HEEL TO EAR								
							TOTAL NEUROMUSCULAR MATURITY SCORE	

SCORE
Neuromuscular _____
Physical _____
Total _____

MATURITY RATING

SCORE	WEEKS
-10	20
-5	22
0	24
5	26
10	28
15	30
20	32
25	34
30	36
35	38
40	40
45	42
50	44

GESTATIONAL AGE (weeks)
By dates _____
By ultrasound _____
By exam _____

PHYSICAL MATURITY

PHYSICAL MATURITY SIGN	SCORE							RECORD SCORE HERE
	-1	0	1	2	3	4	5	
SKIN	sticky friable transparent	gelatinous red translucent	smooth pink visible veins	superficial peeling & / or rash, few veins	cracking pale areas rare veins	parchment deep cracking no vessels	leathery cracked wrinkled	
LANUGO	none	sparse	abundant	thinning	bald areas	mostly bald		
PLANTAR SURFACE	heel-toe 40–50 mm: -1 < 40 mm: -2	>50 mm no crease	faint red marks	anterior transverse crease only	creases ant. 2/3	creases over entire sole		
BREAST	imperceptible	barely perceptible	flat areola no bud	stippled areola 1–2 mm bud	raised areola 3–4 mm bud	full areola 5–10 mm bud		
EYE / EAR	lids fused loosely: -1 tightly: -2	lids open pinna flat stays folded	sl. curved pinna; soft; slow recoil	well-curved pinna; soft but ready recoil	formed & firm instant recoil	thick cartilage ear stiff		
GENITALS (Male)	scrotum flat, smooth	scrotum empty faint rugae	testes in upper canal rare rugae	testes descending few rugae	testes down good rugae	testes pendulous deep rugae		
GENITALS (Female)	clitoris prominent & labia flat	prominent clitoris & small labia minora	prominent clitoris & enlarging minora	majora & minora equally prominent	majora large minora small	majora cover clitoris & minora		
							TOTAL PHYSICAL MATURITY SCORE	

Reference
Ballard JL, Khoury JC, Wedig K, et al. New Ballard Score, expanded to include extremely premature infants. J Pediatr 1991; 119:417–423. Reprinted by permission of Dr Ballard and Mosby—Year Book, Inc.

Management

Delivery Room Care	• Attended by pediatrician • Proper resuscitation • Early stabilization of vital signs • Prevent hypothermia and hypoglycemia • If weight is low (<1kg) → intubate and shift to NICU for ventilator care • If weight is 1-1.5kg → Shifted to NICU for observation and management of potential issues • Baby is of good size →clear airways,wrap baby,shift to nursery and monitor
After Birth care	• Maintain thermoneutral environment • Set temperature of incubator and keep humidity at 70%

>2kg	31-33 C
1.5-2 kg	32-34 C
1-1.5 kg	32-35 C
<1 kg	35-37 C

• Use radiant heaters
• Wrap properly(mitten and socks + cap)
• Monitor for hypoglycemia,hyponatremia,hypernatreia,hyperkalemia
• O2 administration (but not more than 40% since increases risk of bronchopulmonary dysplasia and retrolental fibroplasia)
• Fluids (see table earlier in chapter)
• Feed the baby (oral or tube)
• Supplementation of vitamins (A,B,C,D,E,K)
• Protection from infections (antiseptic measures)
• Early detection and management of complications of prematurity
• Immaturity of drug metabolism

Discharge criteria	• The infant should be feeding by nipple • Baby should be gaining weight properly (10-30g/day) • Temperature should be stabilized in open cot • No recent episode of bradycardia or apnea • No parenteral drug administration (oral only)

Respiratory distress syndrome/Hyaline Membrane Disease

Respiratory distress syndrome/Hyaline Membrane Disease	
Risk factors	• Prematurity • Infant of diabetic mother • Caesarean section without labour • H/O prior infant effected • Male gender • Chorioamnionitis • Hydrops foetalis • Cold sucss • Perinatal asphyxia

Definition	• Preterm newborn with • RR >60/minute (Tachypnea) • Chest retractions(sternal and intercostal) • Cyanosis • CXR = whiteout/Uniform reticulo granular pattern and air bronchogram • Delayed onset of respiration in very premature baby
Pathophysiology	• Surfactant is produced by type 2 alveolar cells • Surfactant production begins at 24-28 weeks gestation • It reduces surface tension of alveoli • So it prevents alveolar collapse during expiration • Lecithin/Sphingomyelin ratio of 2:1 indicates pulmonary maturity • If there is decreased surfactant it leads to atelectasis and V/Q mismatch
What increases surfactant	• Pregnancy induced hypertension • IUGR • Twin gestation • Cortisol released during labour
What decreases surfactant	• Hyperinsulinism
Complications	• PDA • Intraventricular hemorrhage • Pneumothorax • Bronchopulmonary dysplasia • Pneumonia • Complications of mechanical ventilation
Investigations	• CXR → whiteout/Uniform reticulo granular pattern /mottling and air bronchogram • Septic screen • ABGs • Pulse oximetry • Hyperoxia test • Blood sugar and calcium levels • ECG
Management	• Maintain thermoneutral environment • Monitor fluid and electrolytes • Minimal handling of baby • Intake/output record • Monitor O2 saturation • Treat metabolic acidosis • Warm humidified O2 • CPAP(continuos positive airway pressure) • IPPV (Intermittent positive pressure ventilation) • Surfactant replacement therapy
Prevention	• Avoid C-sections • Appropriate management of high risk pregnancy and labor • Administration of dexamethasone

Necrotizing Enterocolitis (NEC)

Necrotizing Enterocolitis(NEC)	
Definition	• Acquired neonatal disorder representing an end expression of serious intestinal injury following combination of vascular,mucosal and toxic insults to a relatively **immature** gut
Risk factors	• Prematurity • Exchange transfusion • Polycythemia • Hyperviscocity syndromes • Enteral feeding • Hyperosmolar formula feeding • Asphyxia
Patho	• Bowel ischemia secondary to asphyxia • Milk provides substrate for bacterial growth • Bacterial invasion of gut → gas produced (pneumatosis intestinalis) → gut necrosis and perforation
Clinical Findings	• During 1st two weeks of life shortly after starting enteral feeding • Triad of feeding intolerance,abdominal distension and grossly bloody stools • Apnea,Lethargy,Abdominla wall discoloration • Unstable temperature • Hyperglycemia • Metabolic acidosis
Diagnosis	• Clinical triad • CBC : Leukocytosis or neutropenia • X-ray : Dilated thickened bowel loops,Pneumatosis intestinalis,Perforation with free abdominal air and portal vein air • Blood culture • Stool screening for occult blood • ABGs • Serum Electrolytes • Coagulation profile
Management	• Discontinue enteric feeding (NPO x 2 weeks and give TPN) • Gastric drainage (NG tube) • Monitor vital signs • Monitor abdominal circumference • Metronidazole • Antibiotics • Surgical resection of necrotic bowel segment

Intraventricular Hemmorrhage

Intraventricular Hemmorrhage	
Definition	• Hemmorrhage originating in periventricular sub-ependymal germinal matrix with subsequent entry of blood into ventricular system • Early → within 72 hours after birth • Late → After 72 hours after birth
Incidence	• Inversly propotional to gestational age
Patho	• Watershed zone b/w caudate nucleus and thalamus →prone to hypoxic injury • Vessels around this zone are prone to rupture in babies <34 weeks gestation or <1500g birth-weight • Fluctuations in cerebral blood flow lead to rupture
Risk factors (remember it as a story)	• Aik **Premature** bacha ,jo bht dair aur mushkil se paida hua **(Prolonged and difficult labour)** , ab us se saans nai lia ja raha **(asphyxia)** is lie resuscitate karna para **(resuscitation at birth)** aur ventilate karna para **(Ventilated infant)** jiski waja se **pneumothorax** hu gai aur jhatkay **(seizures)** lagnay lag gaye aur ultimately usay **acidosis** aur **hypothermia** hu gya
Clinical findings	• Bulging fontanelle • Sudden drop in HCt • Apnea • Bradycardia • Acidosis • Cutaneous mottling • Seizures • Change in muscle tone/consciousness
Complications	• Hydrocephalus • Epilepsy • Microcephaly • Irreversible brain damage • Shock and death
Diagnosis	• USG • CBC Septic screen • Bleeding profile
Prevention	• Avoid preterm delivery • Manage CPD • Vitamin K to all women before delivery • C section in high risk deliveries • Avoid birth asphyxia
Management	• General supportive care • Ventilation for apnea • Transfusion for shock • Anticonvulsants for seizures • Serial USG/CT to manage hydrocephalus

Neonatal Sepsis

Neonatal Sepsis (Investigations and Treatment made Easy)

Investigations (Start with CXR,then 2 aspirates,3 cultures ,4 for blood)

- CXR
- Gastric aspirate smears,Tracheal aspirate
- Culture of Urine,Culture of Blood,Culture of CSF
- FBC,TLC,DLC
- ESR,CRP

Treatment (start with general and then think of TASKIN - a cricketer from bangladesh , looks like he has sepsis wese:P)

General

- Antibiotics,Temperature control
- IV Fluids,Monitor Vitals
- Intake output record
- Respiratory support
- Blood transfusion

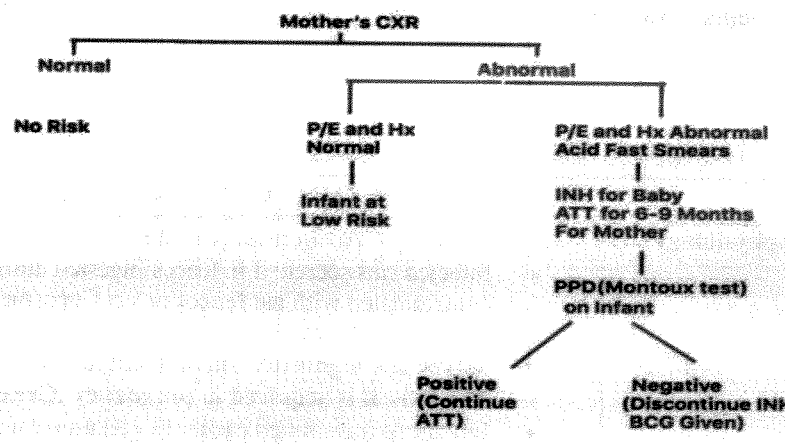
TASK(e)IN

- Transfusion of blood
- acidosis-Sodium bicarbonate
- Septic Shock-Steroids
- Circulatory support-Dopamine
- Immunoglobulins
- Neutropenia → GM-CSF

TORCH INFECTIONS

TOXOPLASMOSIS	
Pathophysiology	• Caused By **Toxoplasma Gondii** • Infected cats secrete it in feaces Ingested during contact with soil contaminated with **cat feaces** or via Unpastuerized milk or raw or undercooked meat • Maybe due to primary acquired infrection in mother or reactivation • **The later it is acquired in pregnancy ,Greater is the transmissibility** • Infections transmitted earlier in gestation causes SEVERE FETAL EFFECTS (Abortion,Stillbirth,Teratogenesis) • Infections transmitted later in pregnancy = Sub Clinical infection
Clinical Findings	• **Triad of Obstructive Hydrocephalus,Chorioretinitis and Intracranial calcifications** • Microcephaly,Microphthalmia • Seizures • Jaundice,Rash,Hepatospleenomegaly • Thrombocytopenia and Petichiae • Fever • Lymphadenopathy
Diagnosis	• Isolation of organism from body fluids and tissues • Serologic tests (4 fold rise in antibody titer) • Perinatal diagnosis

TOXOPLASMOSIS	
	➔Detect parasite in fetal blood/amniotic fluid ➔Document IgM or IgA in fetal blood ➔PCR • CSF Examination ➔Xanthochromia ➔Mononuclear pleocytosis ➔Increased Protein • Skull Xray/CT Scan (intracranial calcifications) • Ophthalmologic Exam (Chorioretinitis)
Treatment	• Pyrimethamine and Sulfadiazine + Folic acid • Spiramycin • If Acute CNS/Ocular infection = corticosteroids

Others	
Hepatitis B	• Transmitted Via Blood • All Pregnant women are screened for HBsAg • If Positive ➔ Infants given HBIG and Hep B Vaccine
Tuberculosis	Rare ! 

RUBELLA	
Pathophysiology	• **Acquired from infected Respiratory secretions** • Maternal viremia leads to fetal infection • Maternal Antibody to previous infection is protective to fetus • **Chances of FETAL infection is greatest in 1st Trimester**
Clinical Findings	• **Sensorineural Hearing Loss** • **Metaphysial Lucencies** • **B and T Cell Deficiencies** • Blueberry Muffin Rash • Virus maybe present in infant's throat for 1 year

	• IUGR,Microcephaly,Microphthalmia • Cataract,Glaucoma,Chorioretinitis • Jaundice,Hepatospleenomegaly • PDA,Pulmonary artery stenosis • Anemia,Leukopenia,Thrombocytopenia
Diagnosis	• Culture(Nasal Swab,Conjuctival scraping,Urine,CSF) • CSF Exam (Encephalitis with increased Protein:Cellular ratio) • Positive IgM in Newborn • **Metaphyseal lucencies due to osteoporosis in Long Bone films**
Prevention	• Vaccinate susceptible population (Contraindicated in pregnancy) • Infants with congenital rubella should be considered contagious until they are atleast 1 year old
Treatment	• No Specific treatment

Cytomegallovirus (CMV)

Pathophysiology ***Most Common Human Virus that is transmitted vertically to the fetus**	• **Transmitted via secretions : Blood,Urine,Sexual contact** • **>90% are asymptomatic and 10% have Mononucleosis like illness** • Can penetrate placental barrier and Blood Brain Barrier • Period of Greatest Fetal Risk for disease and subsequent neurologic impairment is first 22 Weeks of Gestation
Clinical Findings	• IUGR,Chorioretinitis,Microcephaly • Anemia,Leukopenia,Thrombocytopenia • Jaundice,Hepatospleenomegaly,Abnormal LFTs • Blueberry Muffin Rash • Periventricular calcifications • Deafness and Pneumonia
Diagnosis	• Urine or Saliva culture (Gold Standard) • Serology • Skull Xray/CT = Intracranial calcifications
Prevention	• Check blood before transfusion
Treatment	• Gancyclovir

HSV(Herpes Simplex Virus)

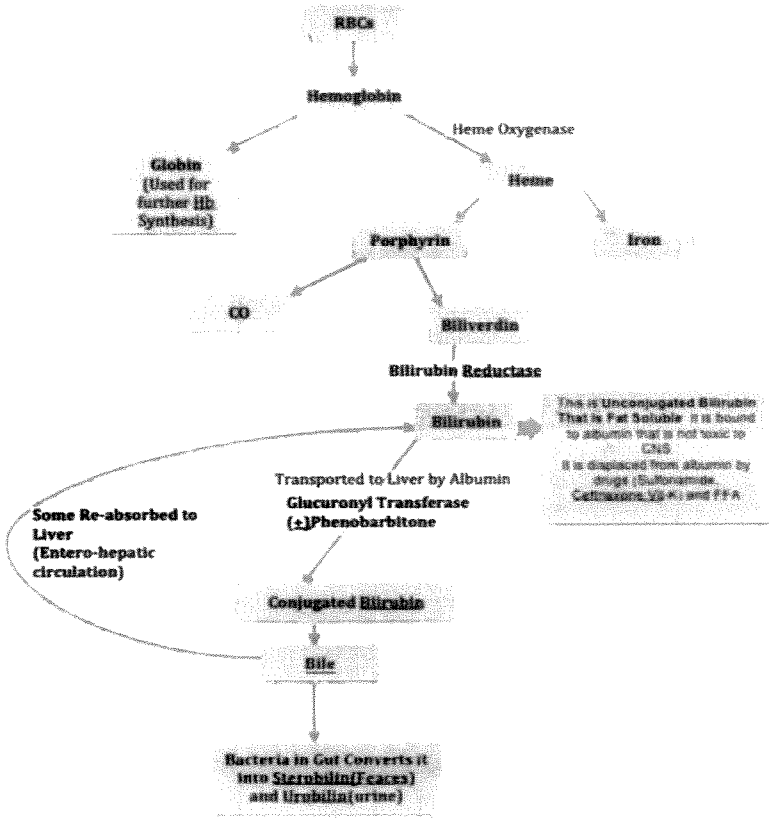
Pathophysiology	• ¼ = HSV-1 and ¾=HSV-2 • 80% acquired intrapartum via ascending infection due to ruptured membranes or via delivery through infected cervix/vagina • 3 Patterns **1)Skin,Eyes,Mouth** **2)CNS** **3)Disseminated disease** • Maternal Ig is not neccesarily protective in fetus
Clinical findings	• Intrauterine • Chorioretinitis • Skin Lesions • Microcephaly • Post-Natal

	• Encephalitis • Disseminated disease • Skin Vesicles • Keratoconjuctivitis
Diagnosis	• Viral Cultures • Immunologic Assays (ELISA) • **Tzank Smear (Giant cells + Eosinophilic intranuclear inclusions)** • PCR (Very sensitive) • Lumbar Puncture • CT/MRI of Head
Prevention	• **Deliver By C-Section**
Treatment	• **1st line = Acyclovir** • **2nd Line = Vidarabine**

Jaundice Neonatorum

- Excessive Bilirubin in the blood
- Can be unconjugated(Indirect) ➔ Physiological/Pathological or Conjugated(Direct) ➔ Always Pathological
- Neurologic concentrations of Unconjugated Bilirubin ➔ Kernicterus

Normal Bilirubin Metabolism



Causes

- Increased UCB only = Pre-Hepatic cause
- Increased CB only = Post-Hepatic cause (Interference with excretion)
- Increased Both UCB and CB = Problem with Liver or it's enzymes

Why Jaundice is More common IN Newborns

- High Hemoglobin mass 18-22g/dL
- Fetal Hemoglobin is unstable
- Fetal Hb has short lifespan (60-70 days)

Unconjugated Hyperbilirubinemia

Etiology

Due to

- Increased Bilirubin production (Hemolysis)
- Defective Bilirubin Conjugation
- Defective bilirubin Clearance from Blood

Causes

- Physiologic (Most Common)
- Hemolytic Anemias
  - ABO or Rh Incompatibility
  - G6PD deficiency
  - Sepsis
  - Drugs
  - Spherocytosis
- Polycythemia
  - Infant of Diabetic Mother
  - Postmaturity,SGA
  - Delayed cord clamping
  - Fetomaternal ot Fetofetal transmission
- Blood Extravasation (Cephalhematoma,Bruises)
- Breast Milk
- Glucuronyl transferase defect (Criggler Najar)
- Metabolic (Glactosemia,Hypothyroidism)
- Increased Enterohepatic circulation
  - Paralytic ileus
  - Intestinal obstruction
  - Pyloric Stenosis

Physiologic jaundice

Definition

- Diagnosis of exclusion
- Occurs in apparent healthy infants
- Clinical jaundice appears after 24 hours of age
- Total Bilirubin rises less than 5mg/dL/day
- Peak bilirubin occurs at 3-5 days of age with total bilirubin of <12.9(term) and >15 (preterm)
- Resolves in 1 week (term) and in 2 weeks (preterm infant)

Mechanism

1)Increased Bilirubin load
2)Defective uptake by liver

3)Defective Coagulation
4)Impaired excretion into Bile

Hemolytic disease of the Newborn

Rh-incompatibility

- Incompatibility of Maternal and fetal Rhesus groups
- Rh +ve child to Rh –ve Mother
- Fetal cells enter maternal circulation thus mother forms Anti-D IgM and IgG
- IgG requires amplified response on 2nd exposure therefore significant disease is Uncommon in 1st pregnancy, The risk increases with increased Parity(conceptions)
- IgG crosses placenta and forms complex with fetal RBCs
- Hemolysis occurs in spleen leading to anemia and jaundice in the baby
- Earlier the IgG crosses the placenta , severe is the disease
- If it crosses at term the anemia will be moderate
- If it crosses during 2nd trimester there will be hepatospleenomegaly,liver disease,cardiac failure,hypoproteinemia,ascites(hydrops),edema and ultimately Death

→Major Blood group (ABO) incompatibility protects against Rh-incompatibility since Anti-A and Anti-B antibodies are destroying fetal cells in Maternal circulation before they sensitize the mother's immune system to produce antibodies ←

ABO incompatibility

- Can be seen in 1st pregnancy
- When Mothers Blood group is O, baby's Blood group is A/B
- Severe hemolysis is rare
- Naturally occurring antibodies are IgM that DONOT CROSS PLACENTA
- Fetal A/B antigens produce a WEAK maternal Immunoglobulin response
- Antibodies produced by mother are NEUTRALISED by A/B antigens
- Coombs test may be –ve (Since it is positive due to IgG)

Clinical Features/Evaluation

Family History	• Ask for any H/O previous sibling with jaundice
Maternal Hx	• DM, Infection, Trauma • Birth trauma, Asphyxia, Delayed cord clamping, Prematurity • Use of Oxytocin, Sulfonamides, Antimalarials, Nitrofurantoins
Infant Hx	• Poor breast feeding (Increases enterohepatic circulation) • Vomiting (Obstruction, Sepsis, Metabolic disorders) • Delayed Meconium passage /infrequent stools
S/S	• Jaundice (Check in daylight) • Color of Urine and Stool • Clinical if 5-7 mg/dL → appears first on Nose → trunk → Legs • Areas of bleeding (cephalhematoma, Petichiae, Ecchymosis) • Hepatospleenomegaly (hemolysis, liver disease, infection) • Plethora (Polycythemia) • Pallor(Hemolysis) • Large infants (Maternal DM) • Prematurity ,IUGR,Post maturity • Omphalitis,Chorioretinitis,Microcephaly (infections)

Investigations

Basic	• Serum Bilirubin (Total,direct and indirect) • CBC and Reticulocyte count • Maternal and fetal blood groups • Direct Coombs test in infants
Secondary	• TFTs • Complete urine exam • LFTs • TORCH antibody titer • G6PD Enzyme Assay • Abdominal USG • HIDA scan • Urine exam for reducing substances • Liver Biopsy

Management

3 main methods
1- Phototherapy
2-Exchange Transfusion
3-Pharmacologic therapy

1)Phototherapy

Indications

- When Bilirubin is 5mg below transfusion level
- When serum Bilirubin is unconjugated
- In Hemolytic disease of Newborn,waiting for exchange transfusion
- Following exchange transfusion
- Prophylactic

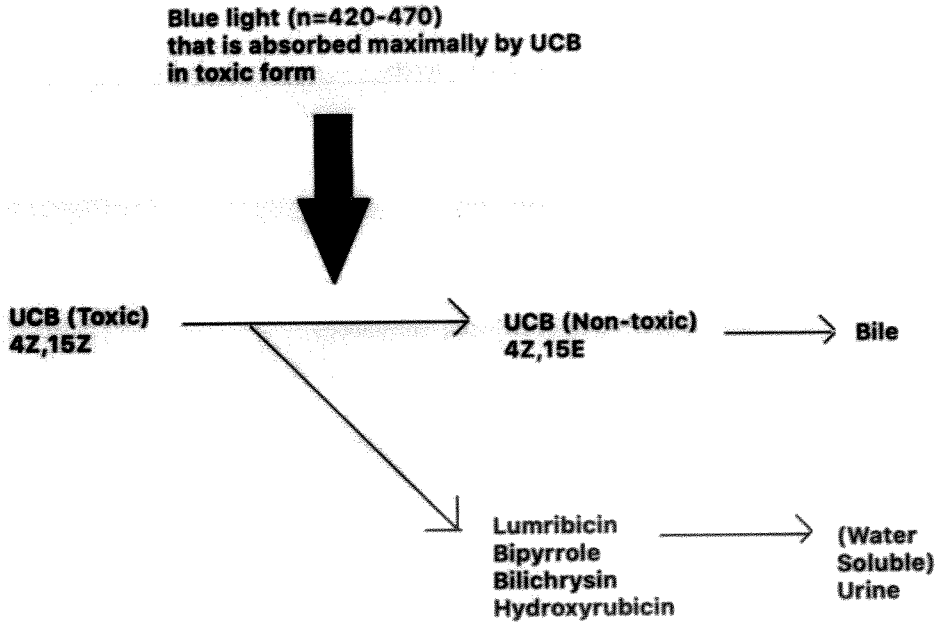
Duration

Stopped when

- Bilirubin is low enough to eliminate kernicterus risk
- Risk factors have resolved
- Infant is old enough to handle bilirubin load

Precautions

- Phototherapy given continuously
- Infant`s eyes are covered by a patch
- Infant is turned frequently for maximum skin exposure
- Increased Fluid intake by 30% to meet losses
- Allow infants to be removed for feeding
- Monitor bilirubin levels every 12-24 hours



Complications

- Overheating and dehydration
- Hypothermia and Chilling in winter months
- Skin rashes
- Loose stools (Increased salts and Unconjugated bilirubin in stool)
- Eye injury (Retinal damage,Corneal abrasion,Conjuctivitis)
- **Bronze baby syndrome** → When infants with conjugated bilirubinemia (mixed jaundice) develop dark brown discolouration that persists for months

2) Exchange transfusion

- Done through umbilical vein

Aims

- Remove sensitized RBCs
- Remove circulating antibodies
- Remove Circulating Bilirubin
- Improve anemia

Indications

At Birth

- Hb < 12g/dL
- Bilirubin >5 mg/dL
- Coombs test positive
- Reticulocytes > 10%

First Week

- Total Bilirubin 20mg/dL
- Bilirubin rise > 1mg/dL/hour or 10 mg/day
- History of same disease and treatment in sibling
- Premature LBW
- Phototreatment fails to prevent bilirubin rise
- Progression of anemia

Equipment and Procedure

- Wet Umbilical stump,catheter passed in umbilical vein but should not reach liver
- Blood required = 2 x Infant`s blood volume (160 ml/kg)
- Blood agitated periodically to maintain constant Hematocrit
- Blood warmed to 37C
- Blood withdrawn 10-20ml , discarded using 3-way catheter,10-20 ml drawn from blood bag and infused
- 1-2 ml calcium gluconate may be infused during or after exchange transfusion

Site

- Umbilical vein
- Umbilical artery
- Large Peripheral vein (Jugular/saphenous)

Blood Type

- Use fresh blood stored in citrated phosphate dextrose
- Hemolytic disease → Blood group O –ve that is cross matched

Infant preparation

- Drape the baby,empty stomach by NG aspiration
- Maintain temperature,Respiratory rate and Heart rate
- Cut umbilical cord near the stump
- Identify Umbilical vein,insert catheter under strict asepsis
- Flush catheters and Syringes with heparin

Complications

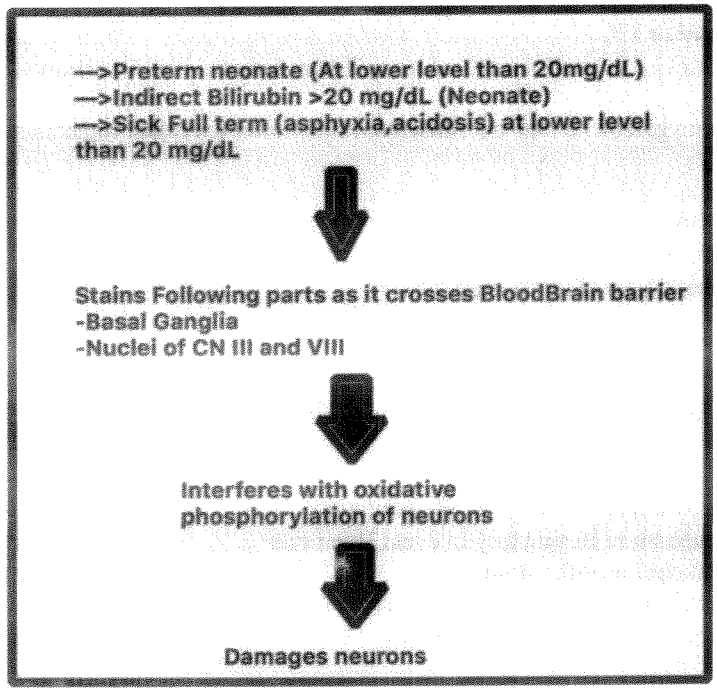
- Hypovolemia
- Hypothermia
- Incompatibility reaction
- Vomiting and aspiration if stomach is not empty
- Cardia arrhythmias and arrest
- Hypocalcemia
- Hyperkalemia (if blood is >5 days old)
- Infections
- Air embolism
- Portal vein thrombosis
- Necrotizing enterocolitis
- Anemia → if blood with low haemoglobin is transfused

3)Pharmacologic Treatment

- Give antibiotics if septicemia is present
- Adequate feeding
- Rhogam to mother within 72 hours of delivery
- Phenobarbitone
- Metalloporphyrins (Hemeoxygenase inhibitors)
- IVIG
- Albumin transfusions if albumin < 3g/dL

Kernicterus

- Neurological syndrome due to Unconjugated bilirubin deposition in brain cells



Clinical findings

- Majority infants die
- Neurological signs predominant
- Sudden lethargy and poor feeding
- Weak/absent Moro's reflex
- High pitched cry
- Head retracted and increased muscle tone = Opisthotonus
- Apnea,respiratory irregularity,convulsions
- Uprolling of eyeballs and tongue protrusion
- 2^{nd} or 3^{rd} year of life → neurological syndrome (Bilateral choreoatheosis ,extra-pyrimidal signs,High tone deafness,Squint,Seizure,Mental retardation)
- Coma
- Death

Prevention

- Intensive physiotherapy
- Exchange transfusion
- Albumin infusion + IVIG
- Follow-up

Breast Milk Jaundice

- Breast Milk has **pregnanediol,FFA,steroids,Beta-glucuronidase** that inhibits bilirubin conjugation
- Late onset → peaks in 10-15 days → returns to normal by 4-12 weeks

Persistent Jaundice

- Jaundice that remains for >2 weeks

Causes

- Prematurity
- Breast Milk
- Hypothyroidism
- Downs syndrome
- Galactosemia
- Criggler-Najjar Syndrome
- Neonatal hepatitis
- Biliary atresia

Pathologic Jaundice

- Evident on 1st day of life
- Bilirubin increases more than 0.5 mg/dL/hour
- Hepatospleenomegaly + anemia
- Clinical jaundice for > 1 week in full-term and >2 weeks in preterm infants

Conjugated Hyperbilirubinemia

- Direct Bilirubin > 1.5-2 mg/dL

Considerations

- Sign of hepatobiliary dysfunction
- Defect in bile secretion,flow or both
- Appears in newborn after 1st week of life
- May be associated with hepatomegaly,Spleenomegaly,Pale stools and dark urine

Causes

- Bile Flow obstruction
- Biliary atresia
- Choledochal cyst
- Bile duct stenosis
- Cystic fibrosis
- Liver cell injury
- Infections
- Metabolic(Galactosemia,Fructosemia,A1AT deficiency)
- Toxic(TPN induced cholestasis)
- Genetic (Duboin jhonson,Rotor syndrome)

Management

- Depends on cause
- Sepsis = Antibiotics
- TPN induced → stop TPN
- Cholestatic → Phenobarbitone + Cholestyramine
- Biliary atresia → Kasai procedure within first 6-8 weeks
- If no treatment is available/successful go for liver transplant

Post-term infant

Post-term infant	
Definition	• Born after 42 weeks of gestation calculated from LMP
Etiology	• Idiopathic (most common) • Anencephaly • Trisomies 16 and 18 • Seckel's syndrome
Clinical findings	• Absent lanugo (body hair) • Decreased vernix caseosa(waxy or cheese-like white substance found coating the skin of newborn) • Long nails • Abundant scalp hair • White scaly loose wrinkled skin • Increased alertness • If placental insufficiency → retarded growth
Complications	• Meconeum aspiration • Hypoglycemia • Hypocalcemia • Asphyxia • Polycythemia
Mx	• Careful Obstetric monitoring to avoid post-term infants • Early feeding for proper nutritional support
Prognosis	• Significant risk of mortality if delivery is delayed 3 weeks or more beyond the term

Small for Gestational Age and Large for Gestational age infants

	Small For Gestational Age Infant (SGA)	**Large for Gestational Age Infant (LGA)**
Definition	Birth weight **less than 10th centile** for his gestation or **More than 2 SD below the mean** for gestation	Birth weight **above the 90th centile** for his gestation or **More than 2 SD above the mean** for gestation
Etiology	**Maternal Causes** 1. Chronic illness (HTN) 2. Young maternal Age (<18) 3. Prepregnancy weight <50kg 4. Poor Maternal Weight gain during pregnancy (<0.9 kg/Month) 5. Multiple pregnancy	1. Constitutional in large parents 2. Diabetes in Mothers 3. Beckwith-Wiedmann Syndrome 4. Post-term infants 5. TGA 6. Erythrobalstosis foetalis

	6. Poor socioeconomic status 7. Malnutrition 8. Smoking 9. Short stature 10. Anemia 11. Fundal Lag (<4 cm for gestational age) 12. Drugs(Phenytoin,Valproate) **Fetal causes** 1. Chromosomal disorders (Trisomies 13,18,21,Turner) 2. TORCH infections 3. Congenital malformations(Potter) 4. Congenital Heart disease **Placental** 1. Decreased placental weight 2. Decreased Placental Surface area 3. Placental separation 4. Tumor 5. Twin-Twin transfusion syndrome	
Complications	**(First 6 Deals with decrease of things since it is small for Gestational age)** 1)Glucose(Hypoglycemia) 2)Temperature (Hypothermia) 3)Neutrophils(Neutropenia) 4)Platelets(Thrombocytopenia) 5)Calcium (Hypocalcemia) 6)Oxygen(Perinatal asphyxia) Then There is **MNOP** 7)**M**econium aspiration 8)**N**ecrotising Enterocolitis and other infections 9)c**O**ngenital abnormalities 10)**P**olycythemia 11)**P**ulmonary hemorrhage	1. Birth Asphyxia 2. Birth Trauma(Fractured clavicle/Erb's Palsy) 3. Hypoglycemia 4. Polycythemia
Management	• Diagnose it early prenatally • Paediatrician should be present to deal with birth asphyxia and meconium aspiration • Temperature maintained to prevent hypothermia • Monitored for Hypoglycemia and Polycythemia • Early feeding (They require more calories than usual Newborns) • Evaluate for any congenital infections and chromosomal abnormalities	• Evaluate for the complications • Feed Early since they are prone to hypoglycemia (due to hyperinsulinism)

Classification	**Asymmetric Growth retardation** When Weight is more effected than Length/Head Circumferrence Mainly due to Placental insufficiency or maternal malnutrition **Symmetrical Growth retardation** Head size is also small in comparison to weight and length of the baby Congenital infections and chromosomal abnormalities are the major cause **Term SGA** When gestation (37 weeks) is complete but Weight is less for gestation **Preterm SGA** When gestation is less (<37 weeks) AND weight is also less

IUGR INFANTS with Chromosomal Abnormalities have 100% Handicap rate and with congenital infections have 50% Handicap rate

Meconium Aspiration syndrome

Meconium Aspiration syndrome	
Intro	• Meconium is first intestinal discharge of newborn • Composed of Epithelial cells,Fetal hair,mucus and Bile • Intrauterine stress /Asphyxia leads to increased intestinal peristalsis with relaxation of internal anal sphincter and passage of meconium into amniotic fluid • This meconium stained amniotic fluid (MSAF) may be aspirated by fetus in-utero or by newborn during labor and delivery • It leads to inflammation of lung parenchyma (interstitial pneumonitis-small airway narrowing) and total (leading to atelectasis) or partial (ball-valve phenomena leading to air trapping) obstruction of air passages thus resulting in respiratory distress • CO_2 retention and hypoxemia results • Leads to asphyxia → multiple organs involved
Risk factors	• Post-term pregnancy • Preeclampsia or eclampsia • Maternal hypertension • Maternal DM • Abnormal fetal Heart rate • SGA • Biophysical profile < 6 • Maternal smoking or respiratory or cardiovascular disease

C/F	• Mostly post-maturity → SGA with long nails and peeling skin stained with yellow or green pigment • Respiratory depression at birth with poor effort and decreased muscle tone • Heavily stained thick **Pea soup like meconium** is associated with higher mortality as compared to lighltly stained green meconium • Tachypnea,nasal flaring etc • May deteriorate later as chemical pneumonitis develops
Dx	• Meconium in tracheal or amniotic fluid along with respiratory distress • CXR → hyperinflated lungs,flattened diaphragm • ABGs → Hypoxemia
Mx	• Non vigorous (HR<100 with poor effort) → Endotracheal suctioning • Vigorous (HR >100 with good resp effort) → no need for endotracheal suctioning • Sidenafil (Vascular Smooth muscle relaxant) • High frequency ventilation and Inhaled NO

Transient Tachypnea of Newborn

Transient Tachypnea of Newborn	
Definition	• Newborn with respiratory distress shortly after delivery that resolves within 3-5 days
Risk Factors	• Elective C-section • Male Sex • Excessive maternal sedation • Macrosomia • Birth asphyxia • Prolonged labor • Breech delivery • Fetal Polycythemia • IDM • Delayed clamping of Umbilical cord (>45s) • Fluid overload to mother • Negative amniotic fluid phosphatidyl-glycerol
Clinical Findings	• Tachypnea (RR >60/min) shortly after birth • Grunting and nasal flaring • Subcostal and/or intercostal recessions • Cyanosis relieved by minimal O2 • Good air entry without crepts or ronchi
Patho	• Delayed resorption of fetal lung fluid from pulmonary lymphatic system • Increased fluid in lungs →decreases lung compliance and increased airway resistance • C-section → lack of normal vaginal thoracic squeeze that forces lung fluid out • Pulmonary immaturity and mild surfactant deficiency
Dx	• ABGs → Mild respiratory acidosis • CBC • Blood culture • CXR → cardiomegaly and flattened diaphragm • Hyperoxia test rules out Congenital heart disease

Mx	• Adequate oxygenation • NPO if RR > 60/min • RR >80 = IV-nutrition • RR 60-80/min = feed by NG tube • RR <60/min = oral feeding • Antibiotics till blood culture is negative

Hypoglycemia

Hypoglycemia	
Definition	• Blood Glucose level of • <40 mg/dL in term infants • <30 mg/dL in preterm infants
Transient Hypoglycemia	• Lasts < 7 days
Persistent Hypoglycemia	• Lasts >7 days
Etiology	• Decreased Glucose stores/production • Pre or post maturity • SGA • IUGR • Inadequate feeding or caloric intake • Increased Glucose utilization • IDM • Beckwith-Weidman syndrome • Insulin producing tumors • Others • Sepsis • Asphyxia • Hypothermia • Polycythemia • After exchange transfusion • LGA or SGA
Facts	• After cutting umbilical cord newborn's glucose falls in first 1-2 hours of life • Then levels increase and stabilize at 65-71mg/dL in 3-4 hours of age • Normal Glucose requirement : 6mg/kg/min
Diagnosis	• Measure blood glucose • Should be measured at 1,2,4,6,12,24 hours of age in high risk infants
Compx	• CNS damage • IVH
Mx	• IV Glucose • Bolus • Drugs (Hydrocortisone,Prednisolone,Glucagon,GH,Diazoxide) • Surgery (in case of insulin producing tumors)

Hypocalcemia

Hypocalcemia	
Definition	• Total calcium level of <7mg/dL or ionized calcium level of <3mg/dL
Early onset	• During the first 3 days of life
Late Onset	• After the first 3 days of life
Etiology	• **50% LBW and almost 100% VLBW infants have hypocalcemia** Early Onset • PRETERM (decreased response to PTH) • IDM(Increased Calcium demand) • Asphyxia • Poor enteral intake • Stressed during perinatal life • Infants receiving blood transfusions • Infants receiving diuretics • Meconeum aspiration • RDS • Bicarbonate Therapy,alkalosis Late Onset • Hyperphosphatemia • Shock,Sepsis • HypomagnesemiaHypoparathyroidism (congenital,Digeorge) • Vitamin D deficiency
Clinical Findings	• Increased extensor tone,clonus,jitteriness,lethargy & hyperreflexia • Early onset → usually asymptomatic • Late onset → seizures
Investigations	• Serum Calcium Levels • Serum Phosphate,Magnesium and ALP • ECG : prolonged QT (>0.4s)
Mx	• Anticipate and prevent hypocalcemia • Asymptomatic → no treatment • Symptomatic → Slow IV bolus of Ca-gluconate • If unresponsive to Ca-gluconate → add MgSO4 (since may be due to hypomagnesemia) • Treat specific cause too

Infant of Diabetic Mother (IDM)

- Baby born to a mother in whom adequate control of blood sugar has not been accomplished during pregnancy
- Maternal Hyperglycemia →Fetal Hyperglycemia →Fetal insulin production → during birth there is interruption of glucose infusion to the fetus but insulin production remains high →Hypoglycemia

Clinical Findings

- Large and Plump baby (Macrosomia) due to increased body fat
- Puffy plethoric facies
- Enlarged Viscera
- Signs of irritability , hyperexcitability and later hypotonia,lethargy and poor sucking
- Signs of respiratory distress (immature lungs and decreased surfactant)
- Congenital anomalies
- Cardiac issues

Complications

- Macrosomia
- SGA
- Hypoglycemia
- Hypocalcemia
- Hypomagnesemia
- Birth asphyxia
- Birth trauma (Klumpke's or Erb's palsy,Clavicle fracture,Cephalhematoma)
- RDS
- Hypertrophic cardiomyopathy
- Hyperbilirubinemia
- Polycythemia and hyperviscocity
- Renal Venous thrombosis
- Cardiac → TGA, VSD,ASD
- Renal → Agenesis
- GIT → Situs inversus,Small left colon syndrome
- Neuro → Anencephaly,Meningocele
- Skeletal → Hemivertebrea

Investigations

- CBC
- Serum Calcium
- Serum Glucose levels
- Serum Bilirubin levels
- ABGs
- Blood culture and Gram staining
- ECG and Echocardiography

Management

- NORMAL newborn evaluation
- Check Blood glucose and Hematocrit
- Physical examination
- Hypoglycemia → infuse glucose solution
- Manage other complications
- Start feeding when baby is stable and is able to suck
- Encourage breast feeding

Neonatal Seizures

Definition	• Paroxysmal burst of electrical activity within the central nervous system • Early onset → 0-3 days of age • Late onset → after 3 days of age
Etiology	• Hypoxic ischemic encephalopathy (60%) • Intracranial hemorrhage (15%) • Infections –Meningitis,sepsis,TORCH(12%) • Idiopathic(10%) • Metabolic causes (glucose,sodium,magnesium) • Pyridoxine deficiency • Drug withdrawal (Narcotics,BZs,Barbiturates) • Trauma • Kernicterus • Familial
Types	Subtle(50%) • Preterm > Full-term • Tonic horizontal deviation of the eyes, jerking blinking of eyes.,Sucking, drooling lip smacking, yawning • Swimming, rowing or pedaling movements Clonic • Focal → well localized clonic jerking • Multi-focal→Several body parts involved Tonic • Premature infants • Focal or generalized Myoclonic • Both in full-term and preterm • Focal,Multifocal or generalized
Evaluation	• Age • Shortly after birth → pyridoxine deficiency • 1st day of life (< 3 days) → Asphyxia and birth trauma,Early onset hypocalcemia • 5-7 days of life → late onset hypocalcemia • Clinical status • Preceeded by lethargy → asphyxia or birth trauma • Maternal drug history +ve → drug withdrawal • Family history +ve → Familial • Bulging fontanelle → Meningitis
Investigations	• CBC with ESR and CRP • Serum electrolytes including calcium • BUN/Creatinine • LFTs,RFTs • CSF examination • Cranial USG/CT • EEG

Management	General • Keep airway clean • Support ventilation by giving free flow O2 • Set up IV line • Keep baby warm • NG tube to prevent aspiration and vomiting & choking • Keep NPO • Send Labs • Monitor vitals Specific • Give 10% glucose IV to correct hypolglycemia • If not controlled → give 10% calcium gluconate to correct hypocalecemia • If not controlled → start anticonvulsant therapy (Phenobarbitone,Diazepam) • Still not controlled →Pyridoxine

Hemorrhagic disease of Newborn

Pathogenesis and Findings

- Vitamin K deficiency → Spontaneous hemorrhage

Why decreased Vitamin K in a new-born

- Lack of free vitamin K in Mother
- Immaturity of New born's Liver
- Absence of bacterial intestinal flora
- Decreased Vitamin K → Deficiency of factors 2,7,9,10
- Thus decreased coagulation and leads to hemorrhage
- Bleeding from gut (hematemesis)
- Bleeding from GUS (hematuria)
- Bleeding from umblical cord
- Bleeding from circumcision site/injection site
- Scalp (Hematoma)
- Intracranail hemmorrhae (**FATAL**)

Diagnosis

- PT,APTT,CT →prolonged
- Factor 2,7,9,10 significantly reduced
- BT,Fibrinogen,Platelets and Factor 5 → NORMAL
- Confirmed by improvement following vitamin K administration OR demonstrating PIVKA in plasma

Prevention

- 1g IM vitamin K to all newborns at birth

Treatment

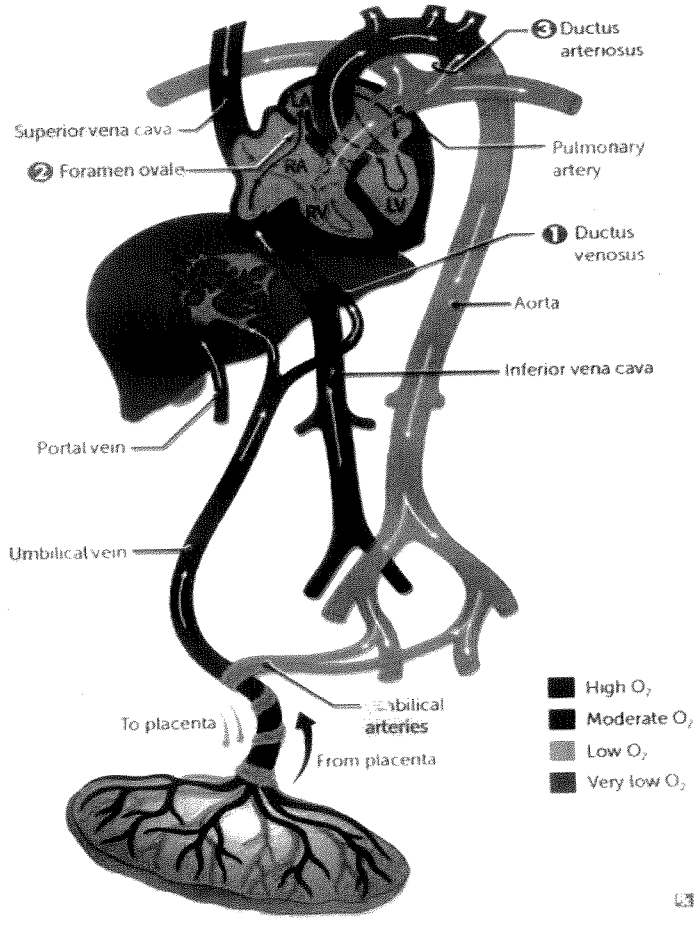
- Vitamin K given parenterally
- Serious bleeding → transfusion may be required

11 Cardiovascular Disorders

Fetal and Neonatal Circulation

- Fetal blood is oxygenated in the placenta
- Then Enters umblical vein
- After that :
- One portion enters Liver ➔ Hepatic portal vein ➔ Inferior Vena cava ➔ Right atrium
- Other portion enterductus venosus ➔Inferior vena cava ➔ Right Atrium
- From Right atrium
- 1/3rd blood of Right atrium ➔foramen ovale➔Left atrium ➔Left Ventricle➔Ascending aorta
- 2/3rd Blood Right atrium ➔Right Ventricle ➔Pulmonary artery ➔Ductus arteriosus ➔Descending aorta (The blood doesnot enter lungs and then pulmonary veins because there is increased pulmonary vascular resistance)

Fetal circulation



Transition to Neonatal circulation

- First breath increases arterial O2
- This leads to vasodilation in pulmonary circulation thus decreasingg pulmonary resistance and increasing pulmonary blood flow
- Increased pulmonary venous return to the Left atrium increases LA pressure thus causing functional closure of foramen ovale
- Then there is closure of ductus arteriosus as a result of decrease in circulating prostaglandin E2 and fibrosis (Remember PgE2 KEEPS the ductus arteriosus open) and Ligamentum arteriosum is formed
- Normal circulation is started

Congenital heart disease

Cyanotic heart diseases (6 Ts) - eaRLy cyanosis → Right to Left Shunt

- **Truncus arteriosus** (Single trunk , no demarcation of aorta and pulmonary artery,1 vessel)
- **Transposition of great vessels** (Pulmonary artery arising from Left atrium and Aorta arising from Right atrium – 2 switched vessels) –MOST SERIOUS CYANOTIC LESION
- **Tricuspid atresia** (3=Tri)
- **Tetralogy of Fallot** (Pulmonary stenosis, RVH, VSD and Overriding of aorta) (Tetra=4) – Most common cyanotic congenital heart lesion (10-15%) which is compatible with life
- **TAPVR** (5 letters) (Total anamolous pulmonary venous return)

Truncus arteriosus

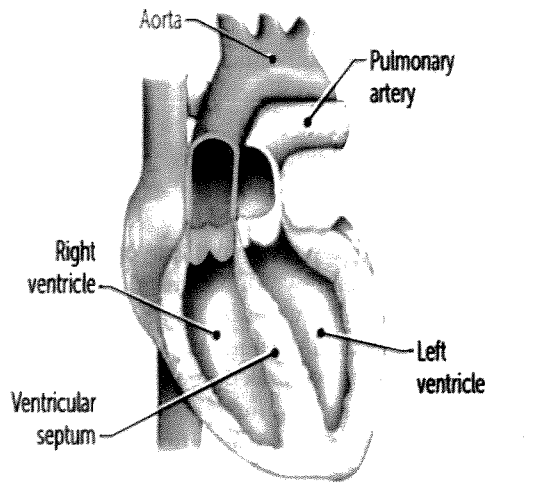
- Truncus arteriosus fails to divide
- There is no demarcation between pulmonary trunk and aorta due to lack of Aorticopulmonary septum formation
- Most patients have accompanying VSD

Transposition of Great vessels

- More common in infants of DIABETIC mothers
- More common in Males 3:1
- Most serious cyanotic lesion
- Rarely survive (Without surgical correction most die within first months of life)

Pathophysiology

- Aorta arises from RV and Pulmonary artery arises from LV
- So there is separation of systemic and pulmonary circulation thus 2 paralell circuits present
- Not compatible with life unless there is a shunt present to allow mixing of blood (VSD,PDA or patent foramen ovale)
- Assocaiated abnormalities include VSD,PDA and/or Pulmonary Stenosis
- Congestive heart failure may develop with VSD because of High cardiac output



Clinical Features

TGA with intact Ventricular septum

- Needs early diagnosis and prompt management
- Before birth oxygenation is normal but once the baby is born and truncus arteriosus closes then there is minimal mixing of blood b/w systemic and pulmonary circulation (only via foramen ovale) that is insufficient and ultimately hypoxemia occurs that is severe (Congestive heart failure is less common since Cardiac output is not high)
- Cyanosis and Tachypnea in 1st hours of life

TGA with VSD

- There is good mixing of blood even after the baby is born due to presence of VSD
- In this case there is MILD cyanosis
- Due to VSD → High cardiac output → Cardiac failure is common
- Murmur → Pansystolic and indistinguishable from VSD murmur
- Large neonates weighing >4Kg (due to heart failure and most being infants of Diabetic mothers)
- There is also retardation of growth and development after neonatal period

Diagnosis

CXR	• Cardiomegaly • Narrow base produced by Anteroposterior arrangement of great arteries give appearance of a narrow pedicle and heart shape looks like an egg placed on the side • Pulmonary vascularity maybe increased or normal
ECG	• Normal Neonatal Right sided dominant pattern • Later → RVH or combined Ventricular hypertrophy
Echocardiogram	• Anteroposterior arrangement of great vessels visible
Arterial Blood Gasses	• Hypoxemia • Arterial PO2 doesnot rise even after 100% O2 administration (hyperoxia test)

Management

Medical

- PGE1 infusion ➔ Keeps ductus arteriosus open(patent)
- Correct hypothermia
- Correct Hypoglycemia
- Correct Acidosis
- If Heart Failure develops ➔ Digoxin,diuretics,increased caloric density of the formula and afterload reduction

Surgical management (TRAM)

- Corrective surgery by the age of 2 Weeks
- **T**otal repair by arterial switch technique ➔ treatment of choice in neonates having TGA with intact ventricular septum (survival rate of 90-95%)
- **R**ashkind procedure or **A**trial balloon septostomy ➔ initial palliative therapy in which atrial septum is perforated(hole is formed) through fossa ovalis using balloon catheter
- **M**ustard procedure ➔ The procedure employs a baffle (artificial tunnel) to redirect vena caval blood flow to the left atrium which then pumps blood to the left ventricle which then pumps the deoxygenated blood to the lungs.

Prognosis

If left untreated 30% die in 1st week and 50% die in 1st month and more than 90% in 1st year due to

- Hypoxia
- Pulmonary hypertension
- Congestive heart failure

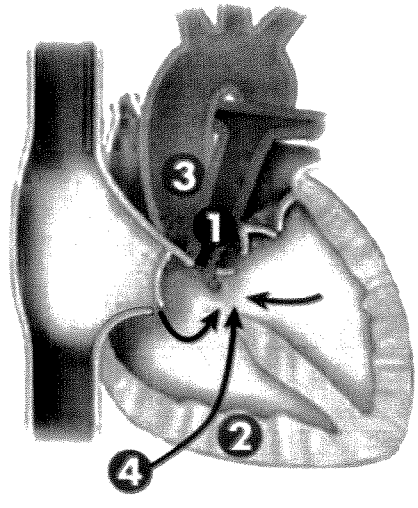
Tricuspid atresia

Absence of tricuspid valve and hypoplastic RV
Requires both ASD and VSD for viability

Tetralogy of Fallot

Pathophysiology

4 components
1= Pulmonary stenosis – mainly infundibular but may involve valve(Most important determinant for prognosis) – determines Right to Left shunt and cyanosis
2=Right Ventricular hypertrophy (Secondary to Right ventricular outflow obstruction)
3=Over-riding of the aorta (over the ventricular septum)
4=VSD (Also important along with PS for prognosis)
(Right to left shunt depends on size of VSD and systemic vascular resistance + BP)



Clinical Manifestations

- Degree of RV outflow obstruction determines the timing of onset of symptoms
- Baby may not be cyanosed at birth
- Right ventricular outflow tract obstruction is progressive resulting in increasing hypoxemia and cyanosis over the first few months/weeks
- Retarded growth and development
- Digital clubbing and Dyspnea → related to degree of cyanosis
- Infants and toddlers play for a short time and then sit or lie down, mostly assume a SQUATTING position
- Squatting → increases systemic vascular resistance and decreases Venous return →increases Left heart pressure as compared to right heart → decreases right to left shunt thus improves cyanosis
- Pulse is usually normal
- Left anterior hemithorax may bulge anteriorly and there maybe left parasternal heave (Both due to Right Ventricular hypertrophy)
- Heart is usually normal in size
- Heart Failure DOESNOT occur

Physical Exam findings

- Systolic thrill along Left sternal border and 3rd and 4th Left parasternal spaces
- S2 is single or pulmonic component is soft
- Harsh ejection systolic murmur along left sternal border in 3rd intercostal space (length and loudness are inversely propotional to the degree of outflow obstruction)

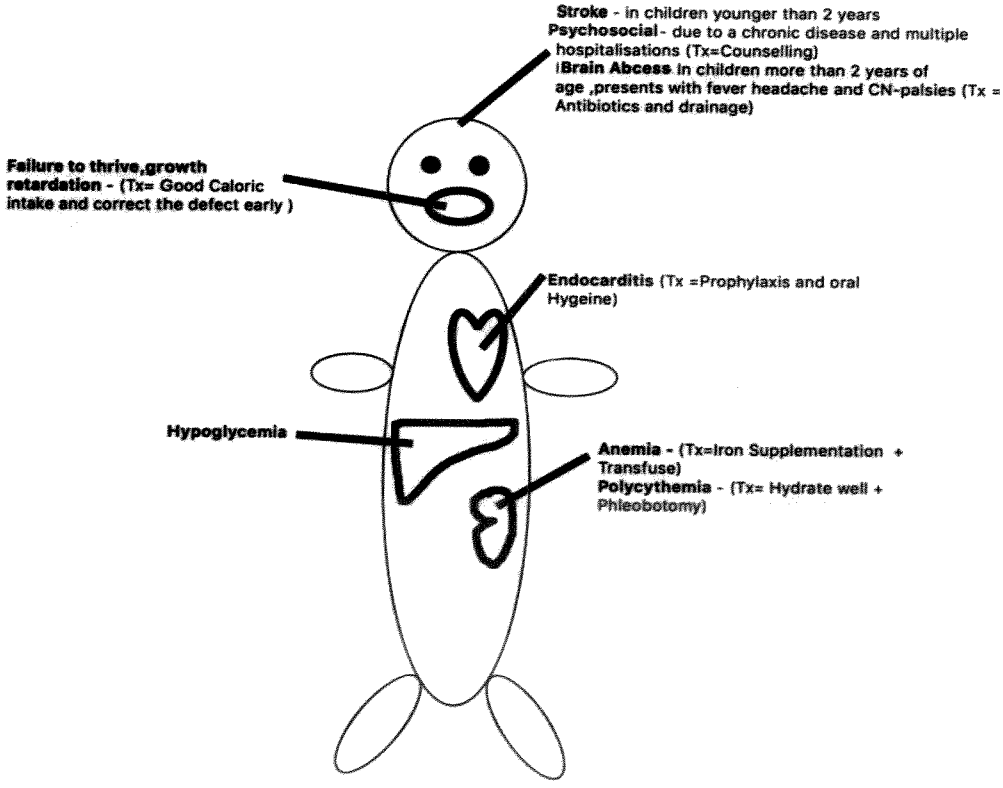
Tet Spell (Paroxysmal hyper-cyanotic attacks/Hypoxic or Blue spell)

- During first 2 years of life
- Infant becomes hyperpneic and restless
- Cyanosis increases
- Gasping respiration and ultimately syncope may result
- More common in the morning or after vigorous crying
- During a spell there is decrease in intensity or disappearance of systolic murmur as flow across RV outflow tract diminishes

Diagnosis

CXR	Size of heart is normal but apex is lifted Boot Shaped Heart Diminished pulmonary vascular markings (oligemic lung fields)
ECG	Right axis deviation and RVH
Echocardiogram	RVH,PS,VSD and overriding of aorta visible
Blood counts	Hb,HCt,RBC counts raised depending on degree of arterial oxygen saturation
Cathetrization	Measuring the degree of desaturation and pressures in ventricles and aorta

Complications with Treatment



Management

Medical management

- Treat the Complications + Maintain HT
- Maintain **h**ydration to avoid hemoconcentration and possible thrombotic episodes
- Maintain **H**emoglobin and **H**ematocrit in normal range
- Maintain **t**emperature to limit oxygen consumption

Managing Tet Spell (OKMAB)

- **O**xygen inhalation
- **K**nee-chest position
- **M**orphine given to decrease anxiety and prevent sympathetic overdrive
- **A**cidosis corrected using $NaHCO_3$
- **B**eta Blockers (Propanolol) – Spells can be prevented by propranolol

Surgical Management

Palliative Surgery (BPW)	Complications of Palliative surgery
1. **B**lalock-Taussig shunt (Right subclavian → Pulmonary artery)	• Diminished radial pulse

2. Pott's Shunt (Descending aorta → Left pulmonary artery) 3. Waterson Shunt (Ascending aorta → Right pulmonary artery)	• Arm-length discrepancy • Horner syndrome • Diaphragmatic paralysis • Chylothorax
Corrective Surgery • b/w 3 months to 2 years of age • Close the VSD and resect RV outflow obstruction	Complications of Corrective surgery • RBBB(Right bundle branch block) • PVBs(premature ventricular beats)

Prognosis

- Death may occur in tet spell
- If person survive the firdst year of life , he improves and may survive the first decade without surgical correction since collaterals form between systemic and pulmonary circulation
- Complete repair before school age has good survival (death may result from dysarrhythmias)

A notic Heart diseases (LaTer Cyanosis – Left to Right Shunt)

1. **Ventricular Septal defect** – Most Common congenital cardiac Defect
2. **Atrial Septal Defect**
3. **Patent Ductus arteriosus**

V ricular Septal Defect

- Most common congenital cardiac defect
- Defect may be in the Membranous part (Most common) or the Muscular part(Less common) of the ventricular septum
- Size can vary from Small/Restrictive (<0.5cm2), Moderate(0.5-1cm2), Large/Nonrestrictive(> 1.0cm2)
- There is Left to Right shunt depending on the size of defect and ratio of pulmonary to systemic blood pressure and vascular resistance
- Biventricular hypertrophy since RVH since it has to deal with increased blood volume (normal VR and blood from LV) and LVH (due to increased pulmonary blood flow thus increased preload)

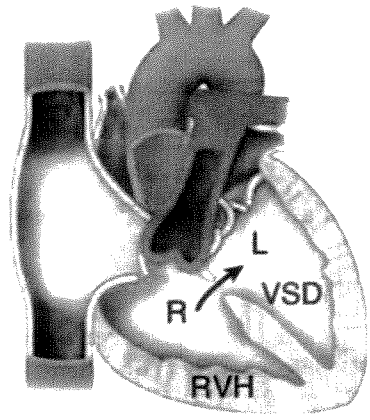
Clinical findings

Small VSD	• Asymptomatic • Both Heart sounds normal • Pansystolic murmur best heard at Left Lower Sternal border + Thrill
Moderate VSD	• Moderate Left to Right shunt • Tachypnea,Dyspnea,Feeding difficulties,Recurrent respiratory infections and growth retardation • Congestive cardiac failure results • Normal S1,Split S2 with loud P2,Loud Pansystolic murmur best heard at Left Lower Sternal border + Thrill
Large VSD	• Excessive Pulmonary blood flow and pulmonary hypertension • Dyspnea,Feeding difficulties,Sweating,Poor growth and recurrent pulmonary infections

	• Congestive cardiac failure • Cyanosis may occur during crying • Loud P2 , closely split S2.Pansystolic murmur (less harsh and more blowing) at left sternal edge.Mid diastolic rumbling murmur at apex.Ejection systolic murmur at pulmonary area
Complications Of VSD	• Congestive Heart Failure • Repeated Respiratory infections • Growth failure • Infective Endocarditis • Pulmonary Hypertension • Eissenmenger Syndrome (Reversal of shunt)

Eissenmenger Syndrome

Uncorrected Left to Right Shunt (VSD,ASD,PDA) → increased Pulmonary blood flow → REMODELLING OF PULMONARY VASCULATURE → Pulmonary hypertension →RVH to compensate →Shunt reverses and becomes Right to Left → Causes Cyanosis



Diagnosis

CXR	• Normal in Small defects • Large defect : Cardiomegaly,increased Pulmonary vascularity and enlargement of LA and LV
ECG	• Normal in Small defect • Large defect : P waves notched or peaked,Biventricular hypertrophy • Left axis deviation if VSD in endocardial cushion region (Speciually in Down's syndrome)
Echocardiogram	• Shows size and position of VSD
Cathetrization	• Demonstrate pressure and O2 saturation in different chambers • (O2 saturation is more in RV and Pulmonary artery)

Management

Medical management

- Small defects close spontaneously by 1 year of age
- Large defects are treated symptomatically

- Controlling Congestive heart failure : Digoxin,Diuretics and afterload reduction medications
- Preventing the development of pulmonary vasculature disease

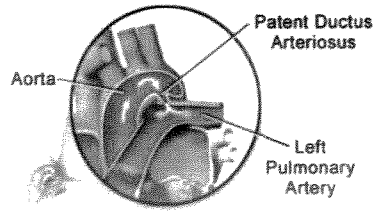
Surgical Management

Procedure	Indications
• Done before 2 years of Age • Primary closure with prosthetic patch	• Inability to control Cardiac failure despite medications • Failure to thrive • Rising pulmonary hypertension • Associated pulmonary stenosis • Development of Aortic regurgitation

Patent Ductus Arteriosus (PDA)

Epidemiology and Pathophysiology

- Ductus arteriosus connects pulmonary artery ➔ descending aorta
- Normally it closes after birth
- In some cases it doesnot close (associated with Very low birth weight babies,Congenital rubella,More common in FEMALES 2:1)
- If it persists after 1st year of life it closes in premature babies spontaneously but in term infants it RARELY closes spontaneously and pharmacologic or surgical interventions are required



High Yield Points
- "Machine-like mummur"
-Patency maintained by PGE and low oxygen tension.
-Close with Indomethacin (Endomethacin)

- If Ductus arteriosus persists
- Blood flows from Aorta ➔ Pulmonary artery
- More blood returns to Left side of heart
- Workload of LA and LV increases and later Pulmonary hypertension occurs
- There is Left to Right shunt depending on the size of defect and ratio of pulmonary to systemic blood pressure and vascular resistance

Clinical Findings

Small PDA	• No Symptoms • Pulse is normal • Heart size is normal
Large PDA	• Congestive cardiac failure • Endocarditis • Breathlessness while feeding • Slowed growth • Repeated LRTIs • Bounding pulses with wide pulse pressure

	• Moderately or grossly enlarged heart • Apical impulse is prominent • Systolic/Diastolic theill present in 2nd Left intercostal space • Systolic/Diastolic Machine-like murmur in Pulmonary area (2nd left intercostal space) • Diastolic component disappears when there is pulmonary hypertension
Complications	• Small PDA : Endocarditis • Large PDA : CHF,Pulmonary vascular disease

Diagnosis

CXR	• Small PDA : Xray is normal • Large PDA : Cardiomegaly and left heart enlargement , LEFT ATRIAL ENLARGEMENT SEEN AS Double contour of Right heart border
ECG	• SMALL : normal • Large : Left Verntricular or Biventricular hypertrophy
Echocardiogram	• Small : Normal • Large : Left atrial and Ventricular enlargement • Ductus visualized form suprasternal notch

Management

Medical	• Indomethacin or Ibuprofen - usually closes after the first dose • Contraindications of indomethacin = Cr >1.7 mg/dL,Thrombocytopenia,Renal or GI bleeding,NEC,Sepsis
Surgical	• Term or post-term → surgical closure necessary • Ligation and division of ductus before 1 year of age

Atrial Septal Defect

High in the septum	• Sinus venosus defect
Mid-portion (Ostium secundum defect)	• **MC type of ASD** • Mostly asymptomatic • In Older children there maybe exercise intolerance • Pulse is normal • Normal AV-Valves • Soft Systolic murmur at upper left sternal border • Fixed splitting of 2nd heart sound
Low in Septum primum (Ostium primum defect)	• Mostly Asymptomatic • History of effort intolerance,easy fatiguability and recurrent pneumonia • Soft Systolic murmur at upper left sternal border • Apical Pansystolic murmur (AV-Valve regurgitation • Fixed Wide splitting of 2nd heart sound

Pathophysiology

- O2 saturation increase in RA,RV and Pulmonary artery
- May lead to paradoxical emboli (Systemic venous emboli use ASD to bypass lungs and become systemic arterial emboli)

Diagnosis

CXR	• Cardiomegaly • Increased Pulmonary vascular markings
ECG	• Partial RBBB • RVH • Superior axis in primum ASD
Echocardiogram	• RV enlarged • ASD visualized

Management

Medical	• Endocarditis prophylaxis (in primum defects and if there is mitral regurgitation)
Surgical **(For all symptomatic and asymptomatic patients in which shunt ratio is 2:1)**	• Done prior to entry to school • Mortality rate = < 1%

Some basic Things That A Final Year student should know

Surgical Management Time	• TOF : 3 Months → 2 Years • TGA Before 2 Weeks • VSD Before 2 Years • PDA Before 1 Year • ASD Before School Entry
Associations With History	• TGA : Diabetic Mother • PDA : VLBW babies with pulmonary disease and Maternal Rubella infection • VSD : Down's Syndrome
CXR Findings (Heart)	• Boot Shaped = TOF • Pedicle with egg on its side = TGA • Cardiomegaly = VSD • Double contour of Right heart border = PDA • Cardiomegaly and increased pulmonary markings = ASD
Incidence	• Most common congenital heart disease = VSD • Most common cyanotic heart disease = TOF • Most serious cyanotic heart disease = TGA
Other Important points	• No Heart failure in TOF • VSD is most common in membranous part • ASD is most common in mid portion (ostium seccundum defect)

Congestive cardiac failure

- Heart is unable to meet the circulatory and metabolic needs of the body or heart is unable to maintain peripheral perfusion despite normal filling pressure
- **Compensated heart failure** : If the dilated ventricle is able to maintain CO at a level that meets the needs of the body
- **Decompensated Heart Failure** : When ventricular dilation no longer results in increased contractility but instead leads to progressive decrease in myocardial contractility and a decline in Cardiac output

Etiology

Fetus	Neonates/Infants	Children
• Severe Anemia(Hemolysis,Fetal maternal transfusion) • Supraventricular tachycardia • Complete Heart block	• Fluid overload • PDA VSD • SVT • Metabolic causes • Viral myocarditis • Left sided obstructive lesion s	• Rheumatic fever • Hypertension (glomerulonephritis) • Viral Myocarditis • Cardiomyopathy

Clinical Findings

- Tachypnea
- Tachycardia
- Tender Hepatomegaly (Normally 2cm below palpable before 2 years of age)
- Basal Crepts
- Increased JVP
- Dependant edema
- Cardiomegaly
- Sweating
- Orthopnea
- Growth failure

Diagnosis

CXR	• Cardiomegaly • Increased pulmonary vascularity depending on the etiology of the cardiac failure • Acute pulmonary edema
ECG	• Ventricular hypertrophy • Helps evaluating rhythm disorders • Low Voltage QRS with diffuse ST elevation suggest myocardial inflammatory disease or pericarditis
ABGs	• Indicates metabolic acidosis
Echocardiogram	• Assess the cause

Management

1. Complete Bed rest
   Prop up patient at 20-30 degrees (reduces VR)
   Increasing daily caloric intake and restricting Salt
2. Oxygen inhalation
3. Diuretics (Furosemide 1mg/kg/day)
   Digoxin (1/3 stat,1/3 after 8 hours and 1/3 after 24 hours)
   ACEi (Captopril)
4. If the patient is in shock add dopamine/Dobutamine
5. Treat the cause

Infective Endocarditis

- Inflammatory disorder of endocardial surface mainly cardiac valves that result from infection

Etiology

- Streptococcus viridans = 50% cases
- Staphylococcus aureus and Staphylococcus Epidermidis = 30% Cases
- Enterococcus,H. Influenza and Psuedomonas = 20% Cases

Microbs	Clinical Association/Predisposing Condition
Staphylococcus aureus	• Prosthetic valves • Intravascular catheters • Implanted devices (pacemaker, defibrillator) • Injection drug users
Viridans group Streptococci	• Dental procedures • Respiratory procedures
Coagulase Negative streptococci	• Intravascular catheters • Prosthetic valves • Pacemaker/Defibrilators
Enterococci	• Nosocomial UTIs
Strep Bovis	• Colon Carcinoma • Inflammatory Bowel disease
Fungi	• Immunocompromised • Chronic indwelling catheters • Prolonged antibiotic therapy

Pathogenesis

- Blood turbulence or trauma leads to damage of cardiac endothelium
- This damaged endothelium serves as a nidus for infection
- Bacteria invade and infect the damaged epithelium
- Vegetations (fibrin,platelets,bacterial masses) form on valve leaflet
- Vegetations may break away and cause embolization (Splinter hemorrhages,Roth spots)

Risk Factors	• Poor dentition • Cardiac causes (Congenital heart disease) • Intravascular catheters (eg : hemodialysis) • IV drug use
Physical Examination	• **Skin** : Osler's nodes(pea-sized painful nodules on fingers),Subungal hemorrhages,Janeway lesions (Flat painless hemorrhagic macules on hands or feet),Petichiae • **Ocular** : Roth Spots (Retinal hemorrhages with clear centers) • **Cardiac** : Heart murmurs • **GI** : Spleenomegaly,Spleenic infarction,Spleenic abcess • **Neurologic** : Symptoms related to stroke,meningitis or brain abcess,Mycotic aneurysm • **Other** : Clubbing of fingers
Labs/Imaging/Diagnosis	• Positive Blood cultures • Increased WBC count or normal WBC count (Subacute endocarditis) • Raised ESR • Hematuria/Proteinuria • Septic Emboli • Transesophageal Echocardigram – Gold standard for diagnosis and localizes vegetations
Treatment	• Remove the soure of infection • Antibiotics (remember by **P**ersitant **V**e**G**etations- P for Penicillin , V for Vancomycin,G for Gentamycin) • Empiric treatment for all : P+V+G • Staphylococcus : V + G • Streptococcus : P + G • Surgical debridement if required
Prophylaxis	• Dental or respiratory procedure : Oral Amoxicillin • GI or Genitourinary procedure : Oral Amoxicillin or parenteral ampicillin and gentamycin • If Allergic to penicillin : Clindamycin
Complications	• Congestive Cardiac Failure • Myocardial Abcess • Pulmonary Emboli • Aneurysm formation

Infective endocarditis – modified Duke criteria	
Diagnostic criteria for IE	**Major criteria** • Blood culture positive for typical microorganism (eg, *Streptococcus viridans*, *Staphylococcus aureus*, *Enterococcus*) • Echocardiogram showing valvular vegetation **Minor criteria** • Predisposing cardiac lesion • Intravenous drug use • Temperature >38 C • Embolic phenomena • Immunologic phenomena (eg, glomerulonephritis) • Positive blood culture not meeting above criteria **Definite IE** 2 major OR 1 major + 3 minor criteria **Possible IE** 1 major + 1 minor OR 3 minor criteria
Clinical findings (frequency)	• Fever (>90%) • Heart murmur (85%) • Petechiae (≤50%) • Subungual splinter hemorrhages (<50%) • Osler nodes, Janeway lesions (<50%) • Neurologic phenomena (embolic) (≤40%) • Splenomegaly (≤30%) • Roth spots (retinal hemorrhage) (<5%)

19 Pediatric Surgery

Cleft Lip/Palate

- Cleft Lip : Failure of medial nasal and maxillary prominences to join
- Cleft palate : Failure of Palate shelves to approximate and fuse
- Cleft Lip alone or with cleft palate is twice as common in boys
- Cleft palate alone is more common in girls

Etiology

- Maternal drug exposure
- As a part of any syndrome
- Genetic causes (More important in cleft lip than isolated cleft palate)
- Idiopathic

Clinical Manifestations/Complications

- Cleft maybe unilateral (more often on the left side) or Bilateral
- Deformed supernumerary or absent teeth are associated
- Difficulty to Eat
- Difficulty to Speak (hypernasality and articulation issues)
- Hearing and Language delays
- Dental and orthodontic complications
- Cosmetic issues and psychological problems
- Recurrent serous otitis media

Recurrence

- 4% for a couple with 1 effected child
- 9% for a couple with 2 effected children

Pierre-Robin sequence

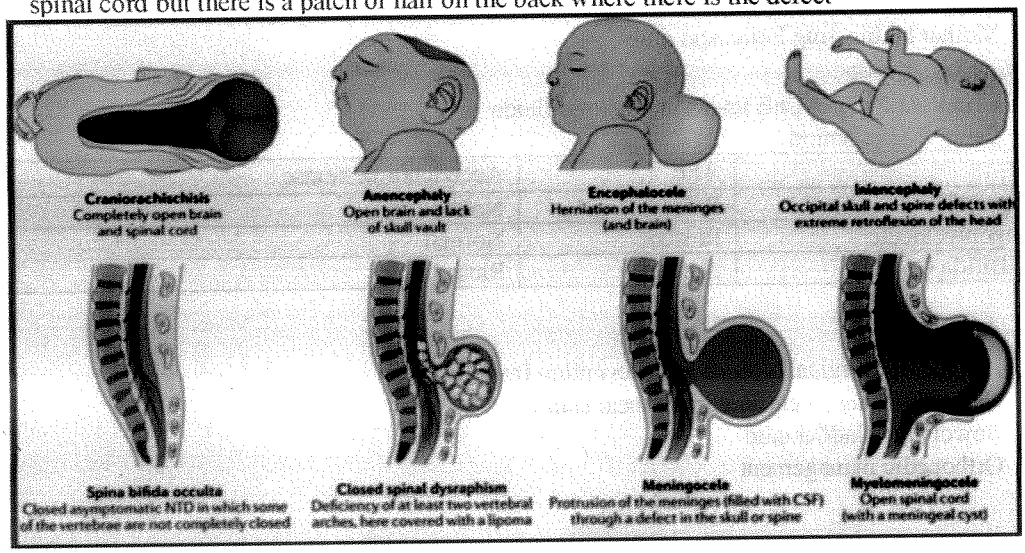
Cleft palate associated with micrognathia and projection of tongue posteriorly during development preventing closure of the palate

Treatment

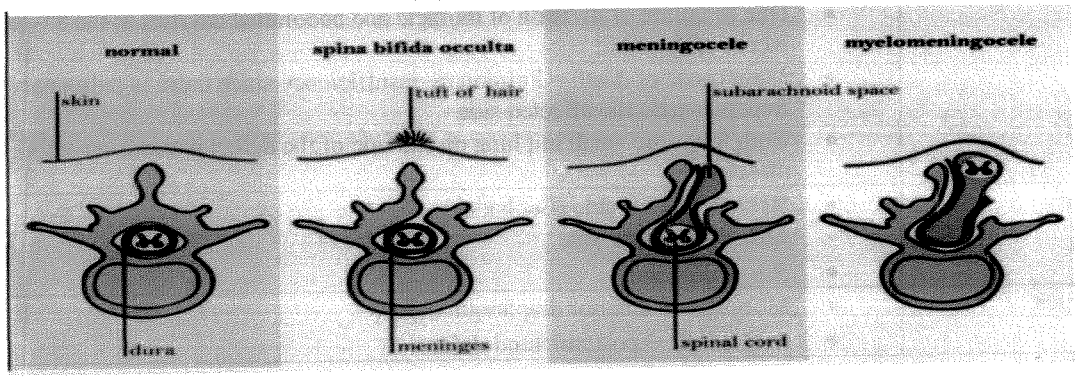
- Feeding by using a soft and long teat with enlarged hole
- Tube feeding in erect position or breast feeding in supine position
- Newborn hearing screening test and then audiometry
- Speech therapy
- Surgical correction (Lip at 3 Palate at 6) – Cleft lip at 3 months or more and Cleft palate at 6 Months or more of age

Neural tube defects

- 1-2/1000 newborns
- Due to failure of neural groove to fuse completely into a tube by 28th day of pregnancy
- **Anencephaly** : Failure of cranial neuropore to close thus there is no cranium and no brain formation (except the most basal portion) leading to neurological abnormalities- It is incompatible with life
- **Meningomyelocele** : Failure of closure of caudal neuropore leading to herniation of Meninges(meningocele) or both meninges and spinal cord (Meningomyelocele) – Most commonly occurs in the lumbar region
- **Spina bifida occulta** – When there is a bony defect but there is neither protrusion of meninges or spinal cord but there is a patch of hair on the back where there is the defect



Neural Tube Defects



Risk factors

- Maternal Age (teenage and Older mothers)
- Prenatal exposure to Valproic acid
- Maternal Diabetes
- Folic acid deficiency

Clinical findings

- Hydrocephalus
- Absent Bladder control leading to recurrent UTIs
- Loss of Bowel function thus severe chronic constipation
- Clubfoot below the lesion
- Mental retardation,Seizures

Recurrence

- 1 child effected : 2-3%
- 2 children effected : 10-12 %
- 1 parent effected : 2-3 %

Prevention

- Mother taking 4mg Folic acid daily

Prenatal diagnosis

- Check AFP and AChE levels in amniotic fluids
- Level II Ultrasound

	AFP	Acetylcholinesterase
Meningocele	Raised	Normal
Meningomyelocele	Raised	Normal
Spina Bifida Occulta	Normal	Raised

Management

- If Open → Surgical closure and prevention from infection
- Hydrocephalus : Ventriculoperitoneal shunt
- Bowel and Bladder care
- Orthopedic management

Congenital diaphragmatic hernia

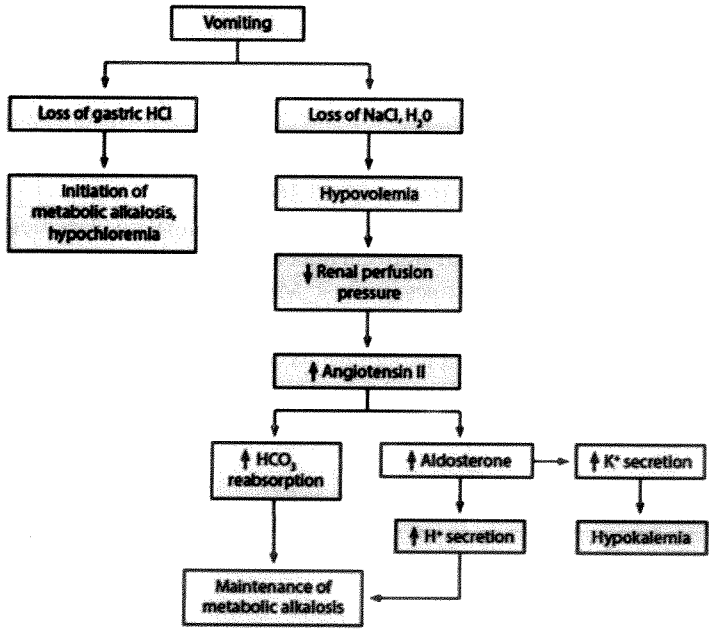
Facts	• Herniation of abdominal contents into thoracic cavity • Due to failure of division of thoracic and abdominal cavities at the 8^{th}-10^{th} weeks of fetal life • Main cause of death = Pulmonary insufficiency since there is pulmonary hypoplasia on the affected side • Both lungs are small but lung on the side of the defect is more severely affected
Site	• 80% → on Left side (Bochadek) • 15% → on Right side • 5%-->Bilateral
Types	• Morgagni →anterior diaphragm defect • Bochadeck →posterolateral defect
Clinical findings	• Respiratory distress • Displacement of abdominal viscera thus scaphoid abdomen • Breath sounds absent on affected side • Bowel sounds maybe audible in chest • Associated vomiting due to intestinal obstruction
Complications	• Pulmonary hypoplasia • Mediastinal shift

	• Pulmonary infection • Prematurity • Cardiac anomalies • Intestinal malformation • CNS lesions • Esophageal atresia • Omphalocele • Syndromes (Trisomy 21,13,18)
Diagnosis	• Antenatal → Polyhydramnios,Mediastinal displacement,Absence of intraabdominal stomach bubble • CXR → intestines in chest ,Mediastinal shift • Contrast study
Management	• Extracorporeal membrane oxygenation • Immediate NG suction • Baby nursed in head-up position

Congenital Hypertrophic pyloric stenosis

Congenital Hypertrophic pyloric stenosis	
Risk Factors	• First Born Baby • Positive family history • Predominant in Males • Erythromycin • Formula Feeding • Blood groups B and O
Clinical Findings	• Present at age 1-2 months (3-5 weeks) • Projectile non-bilious vomiting • Poor Weight Gain • Dehydration • **Olive shaped abdominal Mass**(It is best felt when the patient is calm after emesis since distended stomach can obscure the mass and abdominal musculature is more relaxed after vomiting) • Patient is hungry even immediately after vomiting • Baby accepts feed eagerly
Association	• Other congenital abnormalities like Tracheo-esophageal fistula
Investigations	• Hypokalemic Hypochloremic Metabolic alkalosis • Delayed gastric emptying • Barium swallow → SHOULDER SIGN(bulge of pylorus into antrum) and DOUBLE TRACT SIGN(barium seen in narrowed channel) • USG (90% sensitive) → elongated thickened pylorus
Mx	• IV rehydration • Pyloromyotomy (**Ransted**)

Laboratory derangements in pyloric stenosis



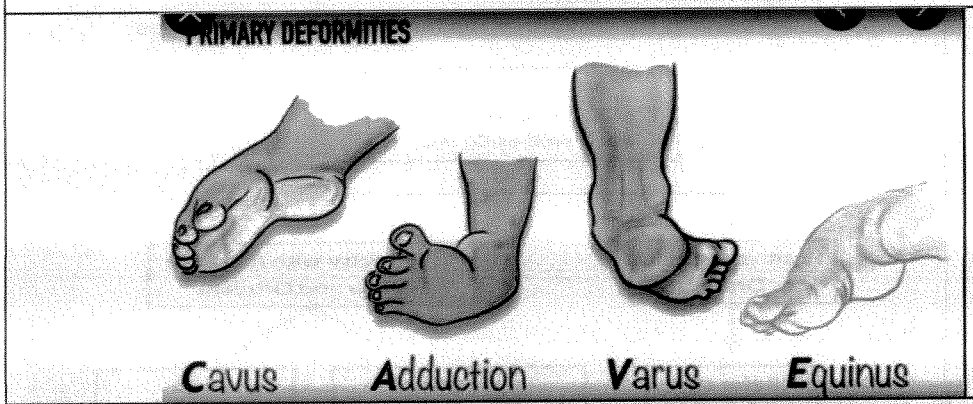
Hirschprung's disease

Facts	• Congenital absence of ganglion cells both in Myenteric and Aurebach's plexus • Due to failure of Neural crest cells to migrate • Aganglionic segment is narrowed • Most commonly involved area = Rectosigmoid
Risk factors	• Boys • Down's syndrome
Clinical Findings	• Failure of Newborn to pass meconium • Vomiting,abdominal distension and reluctance to feed • Enterocolitis → may lead to sepsis and colonic perforation • **Ribbon-like stools** • Peristaltic waves visible • On Palpation → fecal masses felt • Per rectal exam → anal canal and rectum empty
Investigations	• Xray = dilated proximal colon and absence of gas in pelvic colon • Anorectal manometry → failure of sphincter to relax • Rectal biopsy → ganglion cells absent in both submucosal and muscular layers
MANAGEMENT	• Surgical resection of aganglionic segment (**Soave,Svenson**) • Colostomy

Difference b/w Hirschprung and Meconium Ileus

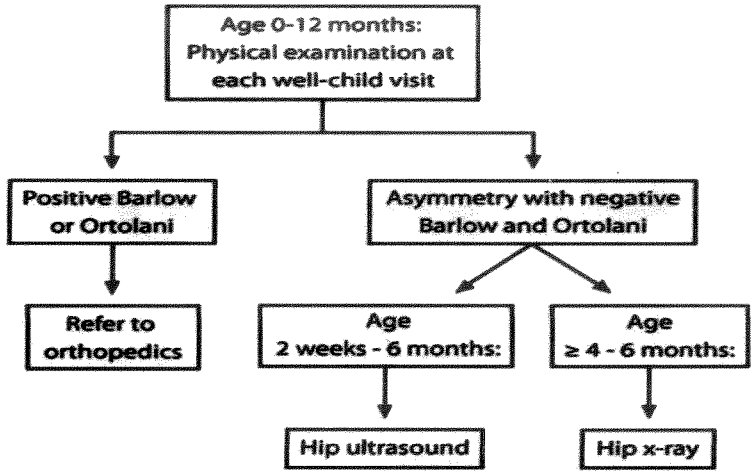
Differentiating features of Hirschsprung disease and meconium ileus		
	Hirschsprung disease	**Meconium ileus**
Associated disorder	Down syndrome	Cystic fibrosis
Typical level of obstruction	Rectosigmoid	Ileum
Meconium consistency	Normal	Inspissated
"Squirt sign"	Positive	Negative

Club foot –Talipes Equinovarus

Club foot –Talipes Equinovarus	
	
Definition	• Equinus(plantar flexion of foot at ankle) and varus (inversion of heel) of calcaneum and talus, varus of midfoot and adduction of forefoot • **Navicular bone is primary site of deformity**
Etiology	• Idiopathic • Congenital—usually isolated and idiopathic • Teratogenic—associated with neuromuscular disorder or a complex syndrome • Positional—abnormal positioning of affected foot in utero
Risk factors	• More common in males • 50% bilateral
Mx	• Immediate non-surgical methods like stretching and manipulation of foot, followed by serial plaster casts, malleable splints or taping →unsatisfactory response→surgical repair **preferably between 3-**

	• **6months of age but always before 12 months (in mcq chose 6-12 months of age if that is the option)** • Majority cases respond to conservative management without surgical repair • Resistant cases → surgical release and correction • Untreated cases → further deformation, gait abnormality and development of ulceration

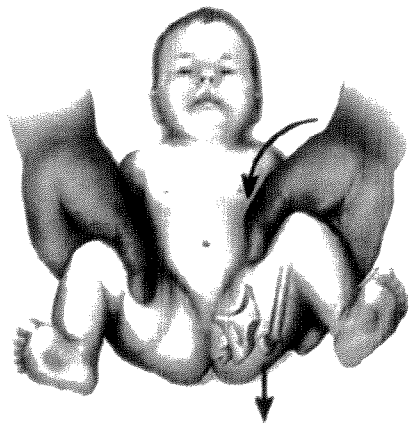
Congenital Dislocation of Hip

Congenital Dislocation of Hip	
Details	• dislocation of femoral head from the acetabulum. • Early diagnosis is critical as treatment initiation before the age six months proves favourable
Risk Factors	• breech presentation • female sex • white ethnicity • family history of DDH • *all infants must have serial hip examinations from birth until they are walking (~1year) since most have no Risk factors associated
Screening/ Imaging	**Screening for developmental dysplasia of the hips**  • Developmental dysplasia of hip is bilateral in approximately 20% of • patients and thus, both sides should be imaged. • X-ray is not helpful until Age 4 to 6 months because the femoral head and acetabulum are not yet ossified. After ossification, x-ray is better at showing acetabular development and positioning.
Diagnosis	• Barlow and Ortolani maneuvers should be performed to assess joint stability. This consists of placing the infant supine with each hip flexed to 90 degrees followed by abduction to feel for dislocatability and adduction to feel for reducibility of an unstable joint. A palpable clunk with either maneuver is

	alarming sign of hip dislocation and should prompt referral to an orthopedic surgeon. • Equivocal signs such as a soft click, leg length discrepancy, or asymmetric inguinal skin fold suggest possible hip laxity.
Tx	• < 6 Months → Pavlik harness /Plaster cast is a splint that holds the hip in flexion and adduction while preventing extension and abduction, which can exacerbate dislocation • > 6 Months → Reduction under anesthesia
Prognosis	• Delayed diagnosis is one of the most common reasons for malpractice suits against pediatricians due to potential complications such as limp (Trendelenburg gait), scoliosis, arthritis, and avascular necrosis.

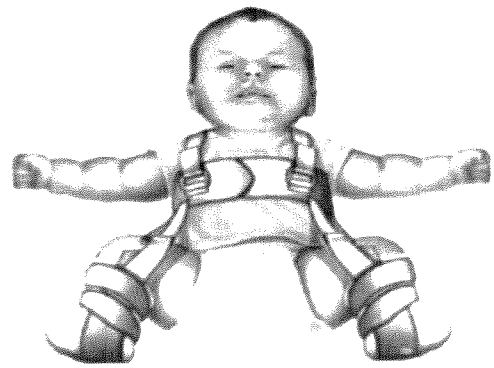
Barlow & Ortolani maneuvers

Ortolani Maneuver:
Abduction with anterior lifting of the hip

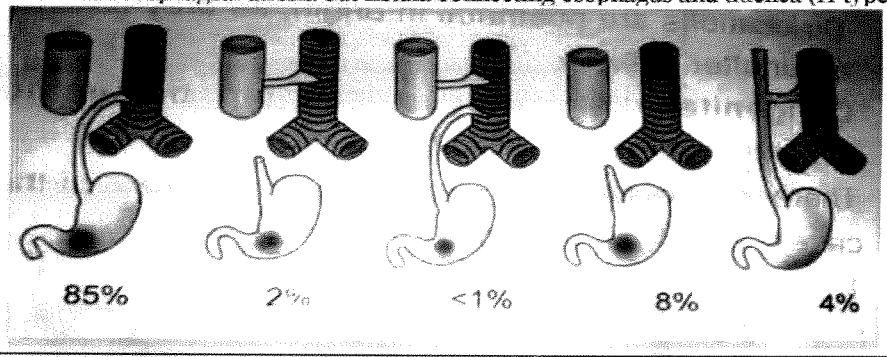


Barlow Maneuver:
Adduction with posterior pressure on the hip

The Pavlik Harness



Esophageal atresia and TEF

Esophageal atresia and TEF	
Types	• Blind esophageal pouch and lower pouch communicating with trachea • Esophageal atresia with fistula from upper pouch • Both pouches of esophagus communicating with trachea • Esophageal atresia with no tracheal communication • No esophageal atresia but fistula connecting esophagus and trachea (H type) 
C/F	• Polyhydramnios due to inability to swallow amniotic fluid • Excessive drooling, choking, coughing and regurgitation with initial feeding attempts immediately after • Birth • Inability to pass naso or orogastric tubes into stomach • Presence of fistula□ air entry into stomach and intestines with each breath□ abdominal distention • Gastric fluid can reflux in to esophagus and through fistula to trachea and lungs→ aspiration pneumonia→respiratory distress, crackles and infiltrates in lungs • 50% pts with trach.. nd esophageal anomalies have additional anomalies→ workup for VACTERI ..rtebral, anal atresia, cardiac, tracheoesophageal fistula, renal and ....b anomalies) should be considered
Mx	• Stop feeding • IV fluids • NG tube in proximal pouch • Elevate head of bed to prevent reflux and aspiration • Surgery is definitive treatment

Biliary atresia

Biliary atresia	
C/F	• Complete persistent cholestasis (acholic stools) in first 2 months of life • Lack of patency of extrahepatic biliary tree • Firm to hard hepatomegaly later splenomegaly • Typical histological features on liver biopsy • Jaundice in Newborn

	• Urine deep yellow but stool is pale (acholic) • Pruritis,digital clubbing,xanthoma • Poor weight gain,ascites,bleeding
Investigations	• HIDA scan • Elevated GGT or ALP • High cholesterol • Prolonged PT • USG → choledochal cyst and other intra-abdominal anomalies
Compx	• Failure to thrive • Pruritis • Portal Hypertension • Hypersplenism • Bleeding diathesis • Rickets • Ascites • Hepatic failure • Death (almost always by age 18-24 months)
Mx	• Surgical (ideal age 6-10 weeks) • Kasai procedure (Hepato-porto-enterostomy) • Gall Bladder Kasai procedure (Porto-cholecystostomy) • Liver transplant • Vitamin A,D,E,K and caloric support • Choleretics and Bile acid binding products • Antibiotics to treat ascending cholangitis