CYANOSIS

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CYANOSIS

- Cyanosis is a blue-purple discoloration of the skin and mucous membranes
- Cyanosis is not a disease
- Cyanosis is a physical sign
- Many causes

CYANOSIS

Cyanosis is better perceived in natural light

And;

- **Nail beds**
- **The Lips**
- **The Tongue**
- **The Mucous membranes**
- **The conjunctiva**
- **Natural light**

CYANOSIS

- **Acrocyanosis; bluish color of the fingers and less commonly the toes**
- **Circumoralcyanosis; bluish color of the skin around the mouth**
- **Differential cyanosis; color differences from upper to lower body**

CYANOSIS

- Visible cyanosis is dependent on the concentration of reduced Hb in blood rather then O_2 saturation
- Clinical cyanosis occurs when the amount of reduced Hb in the capillaries reaches a critical level, about 5 gr/dl
- Two major mechanism
  - Desaturation of arterial blood : CENTRAL CYANOSİS
  - Cyanosis associated by normal SaO_2 (increased extraction of O_2 by peripheral tissue) : PERİPHERAL CYANOSİS

OXYGEN IN BLOOD

Oxygen is carried in the blood;

- **Dissolved in plasma**
  - **The amount is quite small and usually ignored (at $37^{0=}$ 0,03 ml/mmHg-Lt)**
- **Oxygen bound to Hb**

  Influenced by;
  - **Type of Hb (fetal Hb binds more O_2)**
  - **Partial pressure of O_2**
  - **Temperature**
  - **pH**
  - **$PaCO_2$**
  - **2,3-DPG** *** Lowers the affinity of Hb**
- **O_2 capacity of 1 gr Hb=1,36 ml**
- **The amount of reduced Hb in the blood is dependent on its ability to bind O_2**

OXYGEN IN BLOOD

- Hipoxemia is a state of abnormally decreased arterial blood O2 concentration. It is recognized by measurement of;
  - PaO_2 (partial pressure of O_2)
  - SaO_2 (arterial blood O_2 saturation)
- The relationship between the PaO_2 - SaO_2 is important in understanding the determinants of cyanosis, hipoxemia and tissue oxygenation
  - When SaO_2= % 85-88, (cyanosis is apparent to almost observers) PaO_2 can range from30-85 mmHg

    (pH, Temperature, Fetal Hb, 2,3-DPG)

Factors Effecting the Deoxygenated Hb in Capillary Bed

- Deoxygenated Hb concentration in afferent arterioles
- Deoxygenated Hb concentration in efferent venula
- Blood flow velocity
- Hb concentration
- Hb-O_2 dissociation curve

ARTERIAL BED

- Saturated Hb level is never 100 % in arterial blood
- Normal O_2 saturation is 95-98 %in arterial blood (19,5-20 ml %)
  - Bronchial venous blood drains into the pulmonary veins
  - A little amount of CO and CO_2 are bounded to Hb

ARTERIAL BED-2

- Normal arterial O_2 saturation is 95-98 %
- If arterial saturation level is 75-92 % there is arterial desaturation
- If arterial saturation level is 50-75 % there is severe arterial saturation
- If arterial saturation level is <50 % there is very severe arterial desaturation
- If arterial saturation level is <30 % there is limit of viability

VENOUS

- Venous O_2 saturation is depend on;
  - The organ
  - The metabolism
  - The blood flow velocity

Arterio-venous difference

- Average arterio-venous difference is 4,5 ml %
- O_2 binding capacity of 100 ml blood is (Hb;15 gr/dl)= 20 ml
- Consequently, O_2 in venous blood is 15-16 ml %, saturation level is 72-76 %

LUNDSGAARD FORMULA

- Normal arterial saturation is 97%, desaturation 3%
- Normal venous saturation is 73%, desaturation 27%
- Capillary saturation= (arterial+venous saturation) / 2
- Capillary saturation = (97+73) / 2 = 85 %
- Capillary desaturation = 15 %

Hb CONCENTRATION

- The level of the Hb greatly influences the occurrence of cyanosis
- Normally about 2 gr/dl reduced Hb present in the venules
- Additional 3 gr/dl reduced Hb need for presence of cyanosis

Hb CONCENTRATION-2

Case No	[Hb]	Hb sat	deoxy. Hb	deoxy. [Hb]	Reduced [Hb] ın venul	Total reduced [Hb]	Clinical findings
1	15 gr/dl	% 80	% 20	3 gr/dl	2 gr/dl	5 gr/ dl	CYANOSİS
2	6 gr/dl	% 60	% 40	2,4 gr/dl	2 gr/dl	4,4 gr/dl	Pink mucosa
3	6 gr/dl	% 50	% 50	3 gr/dl	2 gr/dl	5 gr/dl	CYANOSİS
4	20 gr/dl	% 85	% 15	3 gr/dl	2 gr/dl	5 gr/dl	CYANOSİS

CAUSES OF CYANOSIS

- Reduced arterial oxygen saturation (central)
  - Inadequate alveolar ventilation
  - Desaturated blood bypassing the effective alveolar units
- Abnormal Hb
- Increased deoxygenation in the capillaries (peripheral)

CAUSES OF CENTRAL CYANOSIS

- **Inadequate alveolar ventilation**
  - **Central nervous system depression**
    - **Decreased alveolar ventilation**
    - **Decreased alveolar partial O_2 pressure**
    - **Increased alveolar partial CO_2 pressure**
  - **Inadequate ventilatory drive**
    - **Obesity, Pickwickian syndrome**
  - **Obstruction of airway**
    - **Congenital**
    - **Acquired (foreign badies, croup)**

Inadequate alveolar ventilation (continued)

- Structural changes in the lungs and/or ventilation-perfusion mismatch
  - Pneumonia, atelectasia
  - Cystic fibrosis
  - Hyaline membrane disease
  - Pulmonary edema
  - CHF
  - Other
- Weakness of the respiratory muscles
- Decreased partial air O_2 pressure: high altitude

CAUSES OF CENTRAL CYANOSIS (continued)

- **Desaturated blood bypassing effective alveolar unite**
  - **Intra cardiac right to left shunt**
  - **Intrapulmonary shunt**
    - **Pulmonary AV fistula**
    - **Chronic hepatic disease**
    - **Obesity, Pickwickian syndrome**
  - **Pulmonary hypertension with resulting right to left shunt at any level**
    - **Eisenmenger syndrome**
    - **PPHN**

ABNORMAL Hb

- Right shifted Hb-O_2 dissociation curve: Low affinity Hb
  - Hb Kansas
  - Hb Beth Israel
- Hb M: Chronic hemolytic anemia: Fe^{+3} form in Hb
- Methemoglobinemia: Cytochrom B5 reductase deficiency
- Carbon monoxide poisoning
- Well water ingestion

CAUSES OF PERIPHERAL CYANOSIS

- Local venous stasis
  - VCS syndrome
  - Catheter obstruction
  - External mass
- Inflammatory edema: thrombophlebitis
- Circulatory collapse
  - CHF
  - Shock
- Hypotonia of venous capillaries
- Vasoconstriction: Reynaud phenomen
- Acrocyanosis of newborn

MANAGEMENT OF CYANOTIC NEWBORN AND INFANT

- History
- Physical examination
- Evaluation of respiratory pattern
- Hiperoxia test: inhalation of 100% O_2, if Pa O_2 >150 mmHg, not cyanotic congenital heart disease
- Thorax graphy
- Echocardiography

GENERAL APPEARANCE OF CYANOTIC PATIENT

- Failure to thrive
- Decreased activity
- Failure to neurological development
- Conjuctival hyperemia: capillary distention and proliferation
- Gingival hypertrophy
- Clubbing: capillary proliferation and periosteal reaction

ADAPTATION AND COMPLICATION OF CYANOSIS

- Polyglobuly
- Capillary distention and proliferation
- Stasis due to hyper viscosity
- Acidosis due to tissue anoxia

CLUBBING

- Very rare before 6 months of age
- Early stage: shininess and redness of finger tips
- Finger and toes become thick and wide and have convex nail beds.
- It may be associated by other system disease
  - Chronic lung disease
  - Bronchopulmonary cancer
  - Endocarditis
  - Hepatic disease
  - Chronic inflammatory bowel disease

HEMATOLOGICAL CONSEQUENCES OF CYANOSIS

- **Increased eritrositosis**
- **Increased Hct (plasma volume not increased)**
- **If Hct > 60-65 %**
  - **Increased viscosity of blood**
  - **Difficulty in heart contraction**
  - **Increased cyanosis due to stasis**
- **Increased Fe^{+2} consumption**
  - **Tendency to Fe deficiency**
  - **Increase in blood viscosity secondary to hypochromy**
- **Bleeding disorders**
  - **Thrombocytopenia, defective platelet aggregation**
  - **prolonged PTT**

NEUROLOGICAL CONSEQUENCES OF CYANOSIS

- Subnormal IQ
- Retardation of motor development
- Cerebro-vascular accident between 6 months-2 years
  - Anoxia -- Dehidratation
  - Polyglobuly -- Fever
  - Hyperviscosity
- Cerebral abscess after 2 years of age
  - Anoxia
  - micro thrombosis
  - Right to Left shunt; escape pulmonary filtration function

CONGENITAL CYANOTIC HEART DISEASE

- Intracardiac right to left shunt with right ventricular outflow tract obstruction
- Common mixing diseases: single ventricle, TAPVR
- Transposition of the great arteries
- Left to right shunt with pulmonary congestion
- Persistent fetal circulation

CONGENITAL CYANOTIC HEART DISEASE

- Tetralogy of Fallot
- Transposition of the great arteries
- VSD+Pulmonary atresia
- Pulmonary atresia with intact ventricular septum
- Double outlet right ventricle
- Single ventricle
- Tricuspid atresia
- Truncus arteriosus
- Total anomalous pulmonary venous return

PERSISTANT FETAL CIRCULATION

- High pulmonary vascular resistance, right to left shunt through ductus arteriosus and/or foramen ovale, severe hypoxia discordant to thorax graphy
- Maladaptation: insufficient vasodilatation in pulmonary bed
- Abnormal muscular structure in pulmonary vascular bed due to chronic fetal hypoxia
- Pulmonary hypoplasia: diaphragmatic hernia, Potter syndrome
- Obstruction of pulmonary veins: polycytemia, TAPVR
- Alveoli capillar dysplasia: Lethal familiar disorders