Baby’s First Breath — Consideration of Some Physiological Aspects

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The Onset of Respirations

Baby’s first breath is probably the most important event in the delivery room for everybody concerned. The factors which cause this to happen have always fascinated doctors and scientists for a long time and it is through extensive research with animal newborns and later with humans that today we are perhaps beginning to understand the complex factors which help the baby in the initiation of the first breath. However, the factors discussed below are not necessarily all the factors, because, there are definitely many more factors which we are not fully aware of as yet.

The First Breath

With the clamping of the cord, the newborn establishes and sustains a pattern of regular respirations within about 60 seconds.

The various factors involved are:

1. Physical Stimuli: (a) Influence of gravity. (b) Lowering of the skin temperature. (c) Sensation and stimuli from the skin, e.g. tapping etc.
2. Chemoreceptors — Responding to the changes in PaO₂ and PaCO₂.
3. The Respiratory Centre: C.N.S. Activity.
4. Aeration of the Lungs: Normal inspiratory pressure is about 70 cm of H₂O in the mature newborns. On radiological studies, the lungs show evidence of aeration within 3 seconds of the onset of breathing.
5. Clearance of Lung Liquid: Lungs contain approx. 3 ml/Kg of fluid at birth — a volume equal to the Functional Residual Capacity (FRC). Up to 35 ml of fluid drains from the infant’s mouth during a normal vaginal delivery.
6. Pulmonary Perfusion.
7. Lung Compliance: Changes in the volume in ml for unit change in pressure in cm H₂O. Normally — 5 cm H₂O⁻¹.
8. Airways Resistance.

What is Surfactant?

1. It is a layer of lipoprotein, which keeps the pressure constant within the alveoli irrespective of their diameter.
2. Lowers surface tension in vitro to less than 10—15 dyn cm⁻¹.
3. Synthesised in the Type II or Granular Pneumocytes in the alveolar epithelium.
4. Stored in the Lamellar bodies of the cells.
5. Released by fusion of the Lamellar body membrane with the cell wall.
6. Complex biochemistry — but basically it has:
   (A) Lipids (85%) —
       (a) Phospholipids,
       (b) Neutral lipids,
       (c) Cholesterol,
       (d) Sphingomyelin.
   (B) Proteins (15%).

How Does Surfactant Work?

1. All the components of surfactant must be present and must interact in order to achieve its striking surface tension lowering action.
2. The enzymatic pathways for lecithin synthesis are sensitive to cold, hypoxia and acidemia.
3. Postnatal exposure to temperatures less than 35°C and pH less than 7.5 causes a rapid fall in the amount of available surfactant.
(4) Surfactant and Type II Pneumonocytes appear in the human lungs at about 20 weeks gestation. The amount increases slowly until a surge occurs at about 30—34 weeks when a large amount of surfactant suddenly becomes available.

(5) Detection of this surge prenatally indicates pulmonary maturity and means that the infant should not develop Hyaline Membrane Disease when delivered.

(6) Prenatally the amount of surfactant present in lungs can be assessed by analysis of liquor amnii, since surfactant is constantly being washed up to the fetal airways with the fetal lung fluid.

(7) Infants with HMD have lower levels of T3 and T4 in their blood than gestation-matched control babies. Congenital hypothyroid babies have an increased incidence of Hyaline Membrane Disease.

(8) A normal thyroid function is required for a normal surfactant development.

(9) At birth the surfactant has to be released and spread out on the alveolar surface. This is primarily dependent upon ventilation and distension of alveoli.

(10) Surfactant, once released, has a half-life of 10—14 hours. The rate of breakdown is increased by breathing pure O₂ and over-ventilation and using inflation pressures of 40 cm H₂O.

How Do You Measure Surfactant?

The amount of Surfactant present in various fluids has been measured in four ways:-

(1) By comparing the ratio of Lecithin to Sphingomyelin (L/S Ratio).

(2) By measuring the absolute amount of Lecithin present.

(3) Measuring the amount of Palmitate (from DPL).

(4) Assessing the surface tension lowering properties of the fluid in the 'Shake test'.

Physiological Changes in H.M.D.

(A) Changes due to Deficiency of Surfactant:

1. Lung compliance falls to about 25% of normal. Decreased FRC (Functional Residual Capacity) and TGV (Thoracic Gas Volume). Increased Dead Space.

2. Greater effort is required to achieve alveolar ventilation — with increase in the work of breathing.

3. Intrapulmonary shunting of blood past completely or partially collapsed alveoli causes hypoxaemia.

(B) Changes due to Hypoxaemia

1. C.V.S.: Pulmonary artery pressure remains at fetal level leading to Right to left Shunt aggravating the hypoxaemia. Also ductus falls to close.

2. Vascular drainage causing transudation of fluid and peripheral oedema.

3. Anaerobic metabolism — leads to accumulation of lactic acid and severe acidemia. Also acid-oemia and hypoxaemia are aggravated by low blood volume and hypotension.

4. Diminished organ perfusion leads to acute renal failure, necrotising enterocolitis and possibly brain damage.

Prevention of H.M.D.:

1. This can be attempted by minimising those conditions which, during labour, or in the first few minutes inhibit surfactant synthesis.

2. Induce surfactant synthesis in the fetal lungs prior to delivery.

This can be done by:-

(1) Giving narcotics (opiates etc.) to the mothers.

(2) Use of glucocorticoids.

Physiological Principles of H.M.D. Treatment

1. Once HMD has developed, surfactant cannot as yet be replaced in the lung.

2. Attempts using purified DPL aerosols have failed.

3. The aim of treatment is to keep the baby alive in good condition so that endogenous synthesis of surfactant may take place.

4. Avoidance of hypoxaemia, acidemia, and hypothermia which causes 'Consumption' of surfactant.

5. Continuous Positive Airways Pressure (CPAP).

6. Intermittent Positive Pressure Ventilation (IPPV).

References:


