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Spontaneous Respiration in Neonates and its Clinical Implications

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Commentary

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DESCRIPTION

The spontaneous respiration a pressure gradient develops between the mouth and alveoli. This is achieved by the contraction of the respiratory muscles, which increases the intrathoracic volume and lowers the pleural pressure to sub-atmospheric levels. Compliance is the volume change per unit of pressure. Total lung compliance is a function of the lung tissue, the surface characteristics and volume. The chest wall of the infant is cartilaginous soft and passage and this means that it is very compliant compared to adult lungs, resulting in a low functional residual capacity. With increasing age the thorax becomes relatively stiffer and gains a greater amount of outward recoil, the thoracic compliance decreases.

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This is due to increasing calcification of the ribs, connective tissue changes and growth of the chest wall relative to the lungs. Once the upright pressure has been assumed and the abdomen has grown abdominal contents shift away from the upper abdomen thus creating a more negative pressure under the diaphragm favouring outward recoil of the chest wall. The chest wall of the adult has a similar compliance to that of the lungs and so it better able to oppose the action of the diaphragm. An adult diaphragm has 55% of slow twitch, high oxidative fibres, where as a term infant has only 25% and a preterm may only have 0% of these fatigue resistant fibres.

In neonates nearly all the volume change is abdominal and the circular rather than oblique lie of the ribs in the horizontal plane does not allow such increases in lung volume. The accessory muscles of respiration are not used during quiet tidal volume breathing but are recruited during times of respiratory embarrassment. Expiration is passive and is facilitated by the elastic properties of the lungs.

There many clinical implications in the mechanical differences between developing and mature chest walls. In the range of normal breathing the thorax of the infant is highly complicated. This becomes functionally significant in the presence of lung disease, when the greater negative pressure and therefore respiratory effort required to inflate the lungs in the chest wall. This results in less effective gas exchange and further leads to increase in the work of breathing. The very low elastic recoil pressure of the new born chest wall is one of the factors which predisposes the infant to lung collapse. In order to achieve an adequate tidal volume the infant has to generate comparatively greater pressure than an adult, because the contraction of the diaphragm produces a negative pleural pressure which tends to distort the compliant rib cage. In times of respiratory distress the infant may show signs of intercostal, subcostal and sternal recession. With severe distress the rib cage will actually move inward as the abdomen moves outward during inspiration, causing a see saw effect. The infant muscles of respiration will fatigue at a relatively quicker rate than adults due to the difference in fibre composition, when confronted with the need to increase their work of breathing to maintain ventilation the muscles can fatigue to such an extent that the infant may become apnoeic. During rapid eye movement sleep it is believed that tonic inhibition of intercostal muscles occurs which may allow rib cage distortion during inspiration. This may have serious consequences as the premature infant spends as much as 50% and sleeping time in the REM sleep state and may during this time be less able to defend against an increase in inspiratory load.