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SERIES 'UPDATE ON RESPIRATORY MUSCLES'
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Respiratory muscle function in infants

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ABSTRACT: In newborns and infants a variety of respiratory disorders lead to ventilatory failure. In early life the ventilatory response to loaded breathing is limited. The risk factors of ventilatory failure are related to the developing respiratory pump because of the immaturity of the chest wall, respiratory muscles and coupling between thoracic and abdominal movements. Assessment of respiratory muscle function in infants is limited, due to the objections to using invasive techniques. However, measurement of airway pressures during crying may provide an index of respiratory muscle strength in infants. Real-time ultrasonography allows investigation of diaphragmatic movements. Pattern of thoracoabdominal motion can be assessed using uncalibrated respiratory inductive plethysmography. Finally, electromyographic recording of respiratory muscles by surface electrodes is of clinical usefulness during sleep studies.
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In newborns and infants, a variety of diseases can rapidly lead to ventilatory failure: neonatal distress syndrome, bronchopulmonary dysplasia, bronchiolitis, upper airway obstruction and abdominal wall defects. In early life, the ventilatory response to respiratory disease is limited and risk factors for ventilatory failure are related, in part, to the immaturity of the developing respiratory pump. However, despite its potential clinical usefulness, assessment of respiratory muscle function has some limitations in infants as compared to adults.

The developing respiratory pump

Respiratory muscle recruitment strategies used by an individual in response to increased mechanical respiratory loads depend upon thoracoabdominal wall mechanics and state of wakefulness.

Mechanical properties of the chest wall

During development, the shape of the thorax changes significantly. At birth, the ribs are composed mainly of cartilage and extend almost at right angles from the vertebral column. As a result, the rib cage is more circular than in adults [1] and, consequently, lacks mechanical efficiency. In adults, the volume of the rib cage can be increased by elevating the ribs. In infants, the ribs are already elevated, and this may be one reason why motion of the rib cage during room air breathing contributes little to tidal volume [2]. The diaphragm appears flattened. The angle of insertion of the diaphragm on the rib cage is very wide, resulting in the absence of the area of apposition. The orientation of the ribs does not change substantially until the infant assumes the upright posture. Concurrently, there is progressive mineralization of the ribs. These changes in shape and structure play a central role in stiffening the rib cage.

A high chest wall compliance (Cw) relative to lung compliance is an inherent characteristic of the newborn mammal [3]. Measurements of passive Cw in human infants are scanty [4]. There are still uncertainties about age-related Cw changes [5].

Chest wall compliance has a major influence on maintenance of lung volume. Functional residual capacity (FRC) is defined as the static passive balance of forces between the lung and the chest wall. In infants, outward recoil of the chest wall is very small [3]. Consequently, the static passive balance of forces would dictate a very small FRC. There are compelling reasons to believe that dynamic end-expiratory lung volume in newborns and infants is substantially above the passively determined FRC. It has been shown that in newborns, in contrast to adults, expiration is terminated at substantial flow rates [6]. This suggests active interruption of a relaxed expiration. Newborns may use two mechanisms to actively slow expiration, *i.e.* post-inspiratory activity of the diaphragm [7], and laryngeal narrowing during expiration [8].

Respiratory muscles

With growth, there is a progressive increase in the bulk of respiratory muscles. There are also important

changes in the fibre composition, fibre size, and oxidative capacity. In preterm infants, the diaphragm is composed of less than 10% type I fibre [9] and low percentages of type II fibre, particularly fibre type IIc [10–12]. Mean cross-sectional area of all fibre types increases postnatally [12]. The total oxidative capacity of the diaphragm, defined as succinyl dehydrogenase activity, is low at birth [11, 12].

Maximum pressures exerted by infants are surprisingly high compared to adults. This is probably related to the small radius of curvature of the rib cage, diaphragm, and abdomen, which, according to the Laplace relationship, converts small tensions into relatively high pressures. Oesophageal pressures of up to -70 cmH_2O have been recorded in infants during the first breath [13]. Inspiratory and expiratory pressures of about 120 cmH_2O have been recorded during crying in normal infants [14]. However, despite relatively high maximal static inspiratory pressure, the inspiratory force reserve of respiratory muscles is reduced in infants with respect to adults because inspiratory pressure demand at rest is greater [15]. High pressure demand in infants is related to high minute ventilation and to high metabolic rate [16].

Fatiguability of neonatal respiratory muscles as compared to adults remains a controversial issue. The paucity of fatigue resistant type I fibre, high proportion of fatigue-susceptible type IIc fibre and low oxidative capacity of the neonatal diaphragm suggest that the muscle may be relatively prone to fatigue. However, *in vitro* [10–12, 17] and *in vivo* [12] studies of the neonatal diaphragm have shown the opposite. In contrast, an *in vivo* study in rabbits found that fatigue occurred more quickly in neonatal than adult animals [18].

Thoracoabdominal coupling: relationship with states of wakefulness

Chest wall muscle contraction helps stabilize the compliant infant rib cage; minimizing inward displacement of the rib cage by diaphragmatic contraction. However, when the stabilizing effect of intercostal muscles is inhibited, such as during rapid eye movement (REM) sleep, paradoxical inward motion of the rib cage occurs during inspiration [19]. It must be emphasized that fullterm newborns spend more than 50% of their total sleep time in REM sleep, and that REM sleep is even more prominent in premature infants.

Asynchronous chest wall movements during REM sleep have been shown to be associated with several mechanical derangements in newborns: 1) decrease in FRC; 2) decrease in transcutaneous partial pressure of oxygen [20]; and increase in diaphragmatic work of breathing [21]. During REM sleep, the diaphragm dissipates a large fraction of its force in distorting the rib cage rather than effecting volume exchange. Furthermore, infants can use their abdominal muscles to optimize diaphragmatic length, and this abdominal muscle activity is inhibited in REM sleep [22]. The increase in diaphragmatic work of breathing may represent a significant expenditure of calories, and may contribute to the development of diaphragmatic fatigue and ventilatory failure. Furthermore, acidosis and hypoxia, both of which increase muscle fatigability, are not uncommon in sick premature infants.

Assessment of respiratory muscle function

In contrast to other pulmonary function tests, assessment of respiratory muscle function is not performed in clinical practice, in the majority of neonatal and paediatric departments. This is due to difficulties raised by use in infants of respiratory muscle function testing methods developed in adults.

Pressure measurements

Maximal static inspiratory (Pimax) and expiratory (Pemax) airway pressure provide estimates of maximal force generated by the respiratory muscles. Airway occlusion performed during crying allows measurements of maximal static pressures in infants. Pimax and Pemax are the most negative and most positive pressures generated during crying against an occluded airway at a volume approaching residual volume and total lung capacity [14]. The measurements have been shown to be reproducible [14]. Normative data have been established in healthy infants in the 0.06–3.76 yrs age range [14]. Maximal inspiratory pressure is independent of age, sex, and anthropometrics, whilst maximal expiratory pressure shows a weak but significant positive correlation with body weight [14]. Measurements of airway pressures during crying may provide an index of respiratory muscle strength in infants with generalized muscle weakness [23]. Inclusion of maximal inspiratory pressures among extubation criteria in infants has been proposed [24, 25].

Reports on transdiaphragmatic pressure measurements are scanty in infants [21, 26]. Attention should be given to the influence of chest wall distortion on oesophageal pressure measurements [27]. Accuracy of oesophageal pressure measurements must be confirmed by comparison of oesophageal pressure with mouth pressure during airway occlusion [28]. Scott *et al.* [26] reported transdiaphragmatic pressures measured during crying in healthy awake infants during the first year of life. Guslits *et al.* [21] have developed a technique for assessing diaphragmatic work of breathing utilizing transdiaphragmatic pressure and abdominal volume displacement.

Diaphragmatic movements

Axial movement of the right hemidiaphragm during tidal breathing has been recorded using real-time ultrasonography [29, 30]. Normative data have been collected in healthy newborns [30]. This investigation may be of clinical value in the assessment of diaphragm, rib cage, or abdominal defects in newborns, as well as of neuromuscular disorders.

Phrenic nerve stimulation

Phrenic nerve stimulation in the neck is one of the techniques used to investigate diaphragmatic contractility. Bilateral supramaximal phrenic stimulation with needle electrodes has been used in adults to detect and quantify peripheral diaphragmatic fatigue [31]. Needle stimulation is potentially dangerous, especially in infants. Therefore only transcutaneous phrenic stimulation has been performed in infants. Transcutaneous stimulation requires use of a high stimulus voltage, needed to overcome the resistance of the skin, and can therefore be painful. This technique has been used in infants to measure phrenic nerve latency [32–34]. Diaphragmatic signals are recorded using surface electrodes. Normative phrenic nerve latency data are available for healthy infants and children [32]. Latency is longer in the left phrenic nerve than in the right [32]. This investigation represents a useful tool in the investigation of infants at particular risk for postoperative phrenic nerve damage [34]. This technique is also clinically useful to estimate diaphragmatic dysfunction during repair of diaphragmatic hernia or abdominal wall defects [35].

Electromyographic (EMG) recording of respiratory muscles

EMG activity of the diaphragm can be recorded *via* surface electrodes and/or oesophageal electrodes. Surface electrodes are usually placed in the right 7th and 8th interspaces between the mid-clavicular and mid-axillary lines. With surface electrodes, contamination of diaphragmatic signals with EMG activity from other muscles cannot be excluded [36]. Several parameters have been calculated from surface diaphragmatic EMG signals, such as peak amplitude [37, 38] high-to-low frequency ratio [37, 38], and centroid frequency [39] of the power spectrum. Early studies suggested that an increase in low frequency and decrease in high frequency power on the surface diaphragmatic EMG in preterm infants was associated with diaphragmatic fatigue [37, 38]. A recent study compared diaphragmatic EMG in preterm infants using both surface and oesophageal electrodes during the various sleep states [36]. This study showed that diaphragmatic EMG obtained *via* oesophageal electrodes showed shorter phasic activity of the diaphragm and negligible tonic activity compared with the surface EMG.

EMG activity of respiratory muscles other than the diaphragm, such as the intercostal [37] and abdominal muscles, has been recorded using surface electrodes in infants [22, 40]. Surface electrode recordings of respiratory muscle EMG activity are of clinical usefulness during sleep studies [40].

Thoracoabdominal motion measurements

Indices of thoracoabdominal motion have been employed to indirectly assess respiratory muscle function [41, 42].

The pattern of thoracoabdominal motion can be assessed using uncalibrated respiratory inductive plethysmography. Signals are used solely as indices of relative timing and magnitude of rib cage (RC) and abdominal (ABD) motion, rather than of volumetric contribution to tidal volume. RC and ABD signals are displayed on an X-Y recording system to form a "Lissajous figure". From the Lissajous figure, a "phase angle" can be calculated as an index of thoracoabdominal asynchrony [43–46]. The degree of thoracoabdominal asynchrony has been shown to be related to the degree of lung disease [43]. Furthermore, developmental changes in thoracic properties with advancing age in early childhood influence the pattern of thoracoabdominal asynchrony when mechanical respiratory loads are increased [45]. Prone posture [47], continuous (nasal) positive airway pressure [48], and abdominal loading [49] have been shown to improve thoracoabdominal motion synchrony in preterm infants and therefore to reduce diaphragmatic work of breathing.

References

1. Openshaw P, Edwards S, Helms P. Changes in rib cage geometry during childhood. *Thorax* 1984; 39: 624–627.
2. Hershenson, Colin AA, Wohl MEB, Stark AR. Change in the contribution of the rib cage to total breathing during infancy. *Am Rev respir Dis* 1990; 141: 922–925.
3. Agostoni E. Volume-pressure relationships to the thorax and lung in the newborn. *J. Appl Physiol* 1959; 14: 909–913.
4. Gerhard T, Bancalari E. Chest wall compliance in fullterm and premature infants. *Acta Paediatr Scand* 1980; 69: 349–364.
5. Sharp M, Druz W, Balgot R, Bandelin V, Damon J. Total respiratory compliance in infants and children. *J Appl Physiol* 1970; 2: 775–779.
6. Kosch PC, Stark AR. Dynamic maintenance of end-expiratory lung volume in full-term infants. *J Appl Physiol: Respirat Environ Exercise Physiol* 1984; 57: 1126–1133.
7. Kosch PC, Hutchison AA, Wozniak JA, Carlo WA, Stark AR. Posterior cricoarytenoid and diaphragm activities during tidal breathing in neonates. *J Appl Physiol* 1978; 64: 1968–1978.
8. Harding R, Johnson P, McClelland ME. Respiratory function of the larynx in developing sheep and the influence of sleep state. *Respir Physiol* 1980; 40: 165–179
9. Keens TG, Bryan AC, Levison H, Ianuzzo CD. Developmental pattern of muscle fiber types in human ventilatory muscle. *J Appl Physiol: Respirat Environ Exercise Physiol* 1978; 44: 909–913.
10. Maxwell LC, McCarter JM, Keuhl TJ, Robotham JL. Development of histochemical and functional properties of baboon respiratory muscles. *J Appl Physiol: Respirat Environ Exercise Physiol* 1983; 54: 551–561.
11. Sieck GC, Fournier M. Developmental aspects of diaphragm muscle cells, structural and functional organization. *In*: GG. Haddad, JP. Farber, eds. Developmental Neurobiology of Breathing. New York, Marcel Dekker, pp. 1991 375–428.
12. Sieck GC, Fournier M, Blanco CE. Diaphragm muscle fatigue resistance during postnatal development. *J Appl Physiol* 1991; 71: 458–464.
13. Karlberg P, Koch G. Respiratory studies in newborn infants. *Acta Paediatr Scand* 1962; 105 (Suppl.): 439–448.

14. Shardonofsky FR, Perez-Chada D, Carmuega E, Milic-Emili J. Airway pressures during crying in healthy infants. *Pediatr Pulmonol* 1989; 6: 14–18.
15. Milic-Emili J. Respiratory muscle fatigue and its implications in RDS. *In*: Cosmi EV, Scarpelli EM, eds. Pulmonary Surfactant System. Elsevier Science Publishers B.V. 1983; pp. 135–141.
16. Gaultier C, Perret L, Boule M, Buvrey A, Girard F. Occlusion pressure and breathing pattern in healthy children. *Respir Physiol* 1981; 46: 71–80.
17. Trang TTH, Viires N, Aubier M. *In vitro* function of the rat diaphragm during postnatal development. *J Dev Physiol* 1992; 17: 1–6.
18. Lesouef PM, England SJ, Stogryn HAF, Bryan AC. Comparison of diaphragmatic fatigue in newborn and older rabbits. *J Appl Physiol* 1988; 65: 1040–1044.
19. Bryan AC, Gaultier CI. The thorax in children *In:* Macklem PT, Roussos H, eds. The Thorax. Part B. New York, Marcel Dekker, 1985 pp. 871–888.
20. Martin RJ, Okkern A, Rubin D. Arterial oxygen tension during active and quiet sleep. *J Pediatr* 1979; 94: 271–274.
21. Guslits BG, Gaston SE, Bryan MH, England SJ, Bryan AC. Diaphragmatic work of breathing in premature human infants. *J Appl Physiol* 1987; 62: 1410–1415.
22. Praud JP, Egreteau L, Benlabed M, Curzi-Dascalova L, Nedelcoux H, Gaultier C. Abdominal muscle activity during CO_2 rebreathing in sleeping neonates. *J Appl Physiol* 1991; 70: 1344–1350.
23. Shardonofsky FR, Perez-Chada D, Milic-Emili J. Airway pressure during crying: an index of respiratory muscle strength in infants with neuromuscular disease. *Pediatr Pulmonol* 1991; 10: 172–177.
24. Malsch E. Maximal inspiratory force in infants and children *S Med J* 1978; 71: 428–429.
25. Shoults D, Clarke TA, Benumof JL, Mannino FL. Maximum inspiratory force in predicting successful neonate tracheal extubation. *Crit Care Med* 1979; 7: 485–486.
26. Scott CB, Nickerson BG, Sargent CW, Platzker ACG, Warburton D, Keens TG. Developmental pattern of maximal transdiaphragmatic pressure during crying. *Pediatr Res* 1983; 17: 707–709.
27. Lesouef PN, Lopes JM, England SJ, Bryan MH, Bryan AC. Influence of chest wall distortion on esophageal pressure. *J Appl Physiol: Respirat Environ Exercise Physiol* 1983; 55: 353–358.
28. Beadsmore C, Helms P, Stocks J, Hatch DJ, Silverman M. Improved oesophageal balloon technique for use in infants. *J Appl Physiol: Respirat Environ Exercise Physiol* 1980; 49: 735–742.
29. Devlieger H, Daniel H, Marchal G, Moerman Ph, Casaer P, Eggermont E. The diaphragm of the newborn infant: anatomic and ultrasonographic studies. *J Dev Physiol* 1991; 16: 321–329.
30. Laing IA, Teele RL, Stark AR. Diaphragmatic movement in newborn infants. *J Pediatr* 1988; 112: 638–643.
31. Aubier M, Murciano D, Lecogguic Y, Viires N, Pariente R. Bilateral phrenic stimulation: a simple technique to assess diaphragmatic fatigue in humans. *J Appl Physiol* 1985; 58: 58–64.
32. Raimbault J, Renault F, Laget P. Technique et resultats de l'exploration électromyographique du diaphragme chez le nourrisson et le jeune enfant. *Rev EEG Neurophysiol* 1983; 13: 306–311.
33. Ross Russell RI, Helps BA, Elliot MJ, Helms PJ. Phrenic nerve stimulation at the bedside in children; equipment and validation *Eur Respir J* 1993; 6: 1332–1325.
34. Ross Russell RI, Helps BA, Dicks-Mireaux CM, Helms PJ. Early assessment of diaphragmatic dysfunction in children in the ICU; chest radiology and phrenic nerve stimulation. *Eur Respir J* 1993; 6: 1336–1339.
35. D'Allest AM, Praud JP, Delaperche MF, Trang TTH, Gaultier Cl. Que peut apporter l'EMG du diaphragme en pathologie malformative de l'enfant (omphalocèle et hernie diaphragmatique). *In:* Beaufils F, ed. Défauts congénitaux de la paroi abdominale: omphalocèle laparoschisis, hernies diaphragmatiques. Paris, Arnette, 1989; pp. 190–191.
36. Reis FJC, Cates DB, Landriault LV, Rigatto H. Diaphragmatic activity and ventilation in preterm infants. I. The effects of sleep state. *Biol Neonate* 1994; 65: 16–24.
37. Lopez JM, Muller NL, Bryan MH, Bryan AC. Synergistic behavior of inspiratory muscles after diaphragmatic fatigue in the newborn. *J Appl Physiol: Respirat Environ Exercise Physiol* 1981; 51: 547–551.
38. Muller NL, Gulston G, Cade D, *et al.* Diaphragmatic muscle fatigue in the newborn. *J Appl Physiol: Respirat Environ Exercise Physiol* 1979; 46: 688–695.
39. Chambille B, Vardon G, Monrigal JP, Dehan M, Gaultier Cl. Technique of on-line analysis of diaphragmatic electromyogram activity in the newborn. *Eur Respir J* 1989; 2: 883–886.
40. Praud JP, D'Allest AM, Nedelcoux H, Curzi-Dascalova L, Guilleminault C, Gaultier C. Sleep-related abdominal muscle behavior during partial or complete obstructed breathing in prepubertal children. *Pediatr Res* 1989; 26: 347–350.
41. Sackner MA, Gonzalez H, Rodriguez M, Belisto A, Sackner DR, Grenvik S. Assessment of asynchronous and paradoxic motion between rib cage and abdomen in normal subjects and in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1984; 130: 588–593.
42. Tobin MJ, Perez W, Guenther SM, Lodato RF, Dantzker DR. Does rib cage-abdominal paradox signify respiratory muscle fatigue? *J Appl Physiol* 1987; 63: 851–860.
43. Allen JL, Wolfson MR, McDowell K, Shaffer TH. Thoracoabdominal asynchrony in infants with airflow obstruction. *Am Respir Dis* 1990; 1941: 337–342.
44 . Allen JL, Greenspan JS, Deoras KS, Keklikian E, Wolfson MR, Shaffer TH. Interaction between chest motion and lung mechanics in normal infants and infants with bronchopulmonary dysplasia. *Pediatr Pulmonol* 1991; 11: 37–43.
45. Goldman MD, Pagani M, Trang HTT, Praud JP, Sartene R, Gaultier C. Asynchronous chest wall movements during non-rapid eye movement and rapid eye movement sleep in children with bronchopulmonary dysplasia. *Am Rev Respir Dis* 1993; 147: 1175–1184.
46. Sivan Y, Deakers TW, Newth CJL. Thoracoabdominal asynchrony in acute upper airway obstruction in small children. *Am Rev Respir Dis* 1990; 142: 540–544.
47. Wolfson MR, Greenspan JS, Deoras KS, Allen JL, Shaffer TH. Effect of position on the mechanical interaction between the rib cage and abdomen in preterm infants. *J Appl Physiol* 1992; 72: 1032–1038.
48. Locke R, Greenspan JS, Shaffer TH, Rubenstein SD, Wolfson MR. Effect of nasal CPAP on thoracoabdominal motion in neonates with respiratory insufficiency. *Pediatr Pulmonol* 1991; 11: 259–264.
49. Fleming PJ, Muller NL, Bryan H, Bryan AC. The effects of abdominal loading on rib cage distortion in premature infants. *Pediatrics* 1979; 64: 425–428.