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Chest Wall Physiology : Implications for the Neonate

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To survive without mechanical assist devices two pumps are essential 24 hours a day : the heart and the chest wall. Surprisingly, relatively little attention has been paid to the respiratory pump until the last few decades. In 1958 Campbell published his first book on the respiratory muscles and stimulated intense investigation into adult chest wall physiology as reflected in the second edition of 1979.[1] In the 1970's advances in less invasive technologies made it possible to take many of the concepts discovered in adults and examine their roles in the newborn. For example, we learned first in adults that functional residual capacity (FRC) falls when anaesthesia decreases muscle tone, and then investigators studied neonates and found that their FRCs also decrease substantially when rapid eye movement (REM) sleep decreases their muscle tone. In this article we will review some of these discoveries, with particular emphasis on the contribution of chest wall physiology to both the neonate's vulnerability to respiratory dysfunction and its therapeutic management. We will touch on the potential contributions of all three components of the chest wall, namely the rib cage, the diaphragm, and the abdomen.

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Functional Residual Capacity

The neonate fights a continuous battle to maintain an optimal lung volume at end-expiration or FRC. First the newborn has to work extremely hard to establish it, generating pleural pressures in the order of −60 to −80 cm H_2O during its first breath to draw air into fluid-filled alveoli.[2] Then the newborn has to work actively to maintain that lung volume. If there is adequate surfactant in the Type II cells at birth this pours out onto the alveolar surface with the first breath and creates a low-tension interface such that more air is retained after each expiration and subsequent breaths require less effort. If surfactant is deficient, surface forces remain high and the lung tends to shrink down to airlessness with each expiration. This is the essential problem of respiratory distress syndrome (RDS) with its low lung volume, poor gas exchange and low compliance. Even if surfactant is normal, however, lung volume is vulnerable in the infant. At total lung capacity (TLC) both lung and chest wall tend to recoil inward when one relaxes (**Figure 1**). Therefore TLC volume must be actively sustained with muscular effort in both adult and neonate. As the adult exhales from TLC a point is reached where the tendency of the lung to shrink is exactly balanced by the outward pull of the chest wall. This FRC position is a static (i.e. no

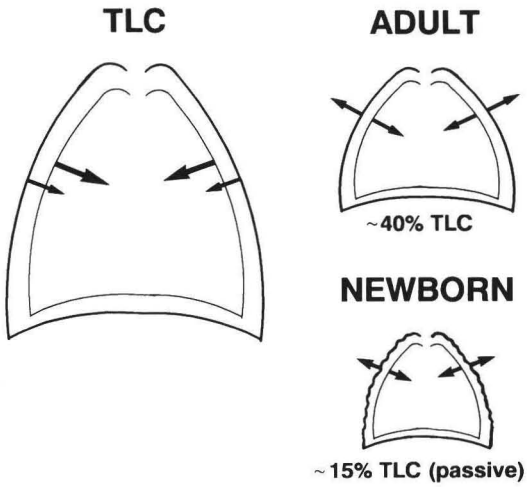

Fig. 1 Schematic diagram of the passive balance of elastic forces at total lung capacity (TLC) and functional residual capacity (FRC) in adult and newborn. See text for discussion.

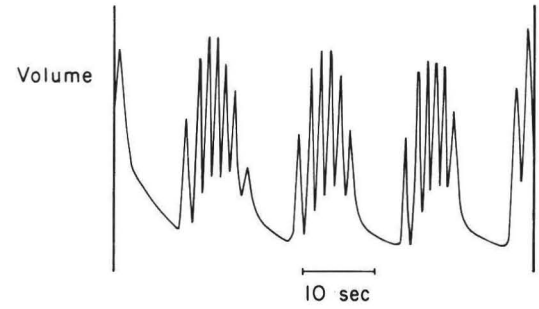

Fig. 2 Tracing of lung volume changes during breathing in a neonate with periodic respiratory pauses. Note that the end-expiratory volume is higher when the infant breathes rapidly, but decreases exponentially towards the true passive FRC volume whenever a pause prolongs the time available for expiration.

movement), passive (i.e. no effort) lung volume that in the adult occurs at~40 percent TLC. Because it depends only on passive elastic forces and requires no muscular effort, FRC is a fairly stable volume in the adult. In contrast, in the neonate the outward recoil of the chest wall is much less, such that a static balance of elastic forces does not occur until it reaches a lung volume of 10-15 percent TLC[3]. This feature is valuable in utero, otherwise the lung would fill up with fluid. However--it would be disastrous to have such a low FRC in vivo. A low FRC volume introduces problems of low oxygen stores, increased airway closure, impaired gas exchange, low lung compliance and high airway resistance, and a resultant increased work of breathing, such as are seen in low lung volume syndromes such as RDS. In actual fact, however, the FRC of the normal neonate is 25-30 ml/kg, which is similar to adult values. This suggests that the infant is maintaining his FRC dynamically, rather than statically. This is very significant since the static FRC of adult is a very stable volume, but dynamically determined things can be very unstable.

Mechanisms That Increase FRC

1. High Respiratory Rate

There are several ways the infant increases his operative FRC. The passive time constant for lung emptying is somewhat greater than one second in the infant. Therefore only 63 percent of a breath can exit passively in one second. When the infant breathes at rapid raies of 50 or 60, incomplete emptying will occur with a resultant increase in end-expiratory lung volume. This mechanism gives rise to the classic trace of lung volume changes seen in infants with apnea (**Figure 2**). Whenever apnea occurs and the expiratory period is prolonged, the lung passively empties down to a lower volume.

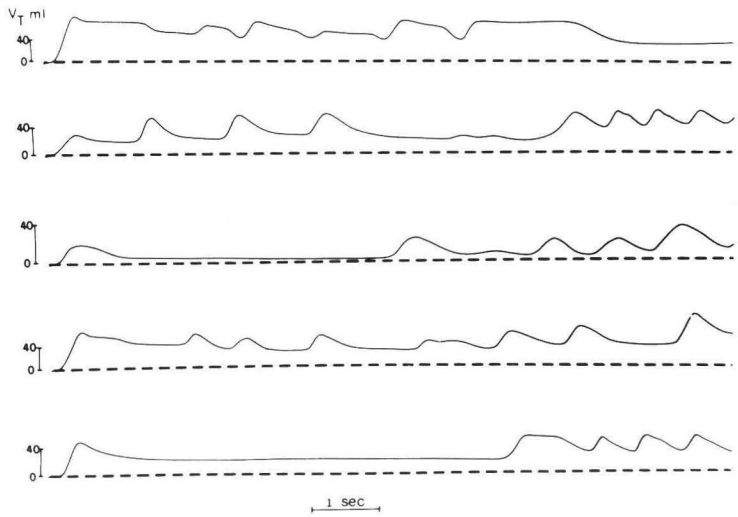

Fig. 3 Spirometric records obtained in five infants immediately after delivery by cesarian section, beginning with the first breath after birth. Note the great variability in breathing pattern, with several records (1, 4 and 5) demonstrating prolonged pauses in an inspiratory position, suggestive of laryngeal braking. Note also the progressive development of the functional residual capacity. VT ; tidal volume. (Reprinted with permission from Mortolo et al. : J Appl Physiol 52 : 716-724, 1982)

2. Active Stiffening of Chest Wall

The infant can also stiffen his chest wall by maintaining inspiratory tone in the intercostal muscles and diaphragm throughout expiration.[4] This in effect will shift the balance of lung and chest wall recoil to a higher volume. This mechanism is very effective, but again introduces the potential for lung volume instability because the amount of this tone has been found to vary substantially with both sleep and anaesthesia/paralysis. For example, Henderson-Smart et al.[5] documented a 30 percent decrease in the thoracic gas volume when neonates shifted from quiet sleep, in which inspiratory muscle tone is preserved, to rapid eye movement (REM) sleep during which muscles such as the intercostals are strongly inhibited.

3. Active Increases in Upper Airway Resistance

Active narrowing of the larynx during expiration is a third mechanism used by neonates to elevate their end-expiratory lung volume above passive FRC. This mechanism is most obvious in the audible expiratory grunt often heard in the minutes immediately post-delivery and in infants developing RDS. Laryngeal braking is most important in the first few hours after birth.[6] During this time frequent pauses can be seen in an inspiratory position (**Figure 3**). Even in older preterms and full-terms this mechanism remains important, however, as evidenced from the work of Berman et al.[7] who found that when intubation interrupted this laryngeal braking function, a positive end-expiratory pressure (PEEP) of 2 cm

H_2O was needed to maintain the same FRC. We conclude that FRC is dynamically determined in the neonate and therefore potentially unstable. It is probably because the infant's lung volume is so vulnerable that such phenomenal progress in care of neonates has been made since continuous positive airway pressure was introduced into the fight to maintain lung volume.

The Chest Wall and Tidal Ventilation

Chest wall characteristics also introduce significant problems for the neonate with respect to generating an adequate tidal volume. These problems arise from maturational differences in muscle type, chest wall anatomy, chest wall compliance, and neuromuscular response to chest wall distortion.

Chest Wall Muscle

Neonates, particularly preterms, have a rather small mass of muscles attached to the rib cage. This muscle is also of the wrong type. Keens et al. performed fiber typing on samples of diaphragm and intercostal muscle from fetuses of various gestation as well as infant and adult specimens.[8] They found that the percentage of high oxidative slow twitch muscle fibers was only about 10 percent at less than 34 weeks gestation, 25 percent at term, and 40 percent by three months of age. By seven to eight months the adult pattern of fiber type was achieved with 50-55 percent slow twitch, high oxidative fibers. These data suggest that the neonate may be deficient in the high-endurance fibers needed to sustain breathing 24 hours a day. Somewhat contradictory fiber type patterns were reported in the premature baboon,[9] but again some functional deficit was demonstrated in electrophysiological properties in the neonatal baboon respiratory muscles, as compared to the adult.

Chest Wall Anatomy

The chest wall characteristics of the neonate facilitate passage through the birth canal, but leave the neonate in need of some postnatal structural remodelling to optimize respiratory function. An adult's chest wall configuration is such that contraction of the diaphragm tends to push outwards both the abdomen and rib cage as it raises abdominal pressure.[1] This coupling of diaphragm contraction to rib cage expansion depends on the area of apposition of the diaphragm where the fibers run cephalad along the inner surface of the ribs before curving medially to form its characteristic dome shape. Efficient rib cage/diaphragm coupling also requires that the rib cage be stiff enough to move as a unit so that an expanding force applied to the lower rib cage (through this area of apposition) will cause expansion of the upper ribs as well. Even in the adult this coupling is not assured, since it varies with the degree of intercostal muscle tone and therefore can be impaired by REM sleep[10] or certain anaesthetic agents.

Neonatal chest wall anatomy differs markedly from that of the adult (**Figure 4**). The chest is essentially a cylinder rather than an ellipse. In the neonate, insertion of the ribs on the spine in a frontal plane is virtually perpendicular from the second to eighth ribs, and the anteroposterior and lateral diameters are about equal.[1] In other words, the rib cage of the newborn infant resembles a pile of rings positioned perpendicularly to the spine. This configuration reduces both the "pump handle" and

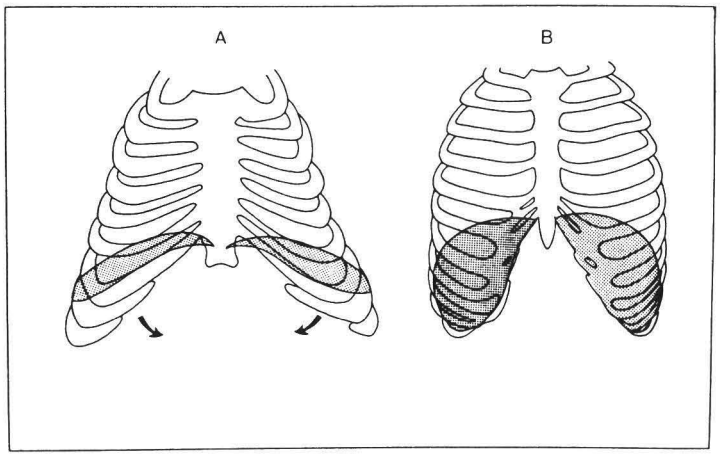

Fig. 4 Outline of the rib cage of the neonate (A) and adult (B). The stippled areas represent the anterior projection of the diaphragm in both cases. Note marked developmental differences in shape of the thorax and relationship of diaphragm and anterior rib cage. In the adult, diaphragm fibers attach to the lowest margin of the ribs anteriorly and run cephalad along the inner surface before curving into the dome. This area of apposition is mechanically important in coupling diaphragm contraction to rib cage expansion. In the neonate, the diaphragm is flatter, the area of apposition minimal, and the direct action of diaphragmatic contraction is to pull the lower ribs inwards. (Reprinted with permission from a thesis by H. Devlieger, Katholieke Universiteit Leuven, 1987)

"bucket-handle" components of chest wall expansion that are classically described in the adult.[1] The attachments of the diaphragm also differ markedly. The neonatal diaphragm has no area of apposition ventrally or laterally ; instead the diaphragm inserts into the internal side of the rib cage in these regions and then runs obliquely back to extend over the whole width of the lumbar muscles without clearly defined crural segments.[11]

There are several important functional consequences to this anatomical arrangement. First of all, the direct effect of diaphragm contraction is to pull the ribs in rather than push them out, while moving more like a bellows than a piston (**Figure 5**). Frequently, motion of the upper (above diaphragm insertion) and lower rib cage (area of diaphragm insertion) will be totally opposite in direction, more often in REM than quiet sleep and more often in the supine than prone position. Also since the ribs of the neonate—especially the preterm—have little ossification they are easily distorted, leading to the classic funnel chest deformity seen in infants with chronic lung disease.

Chest Wall Compliance

We noted earlier that the compliant chest wall of the neonate creates problems with respect to maintenance of FRC. It also influences the ability of the neonate to generate an adequate tidal volume. Any force gener-

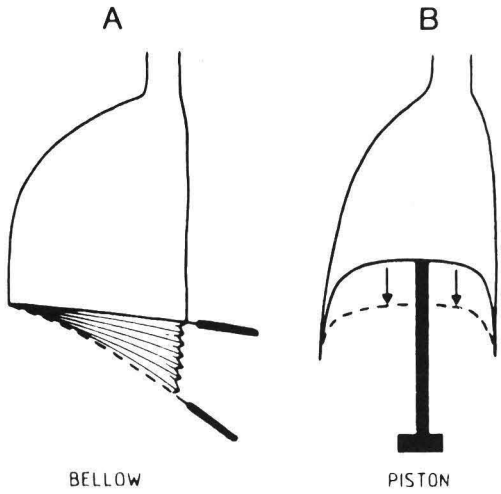

Fig. 5 Schematic representation of displacement of the neonatal and adult diaphragms. The adult diaphragm moves like a piston while the neonatal diaphragm exhibits a more bellows-like action. (Reprinted with permission from a thesis by H. Devlieger, 1987)

ated by the muscles of respiration (i.e. a drop in pleural pressure) will act in parallel on the lung and the chest wall, dividing its displacement between them.[12] In the adult, the compliance of the lung and chest wall are similar, and about half the force generated by the respiratory muscles is used to drive the chest wall and half to expand the lung. In the full term the ratio of chest wall and lung compliance is 4 : 1, in the preterm it approximates 6 : 1. Superficially it may seem advantageous to have a high chest wall compliance so that little force need be wasted to displace it. In practice any such theoretical benefit is overshadowed by the problem of chest wall distortion : for any given change in pleural pressure, the premature has six times the change of sucking in ribs rather than fresh air if motion is determined solely by the passive properties of the lung and chest wall!

Chest Wall Distortion

Both the anatomical configuration and compliance of the chest wall make the neonate vulnerable to rib cage distortion. The consequences of such distortion have been recognized increasingly over the past decade. We now realize that rib cage distortion can influence respiratory timing, increase the work of breathing, lead to respiratory mucle fatigue, and contribute to growth retardation.

Hagan et al. studied 16 sleeping newborns free from cardiopulmonary disease and examined the relationship between the rate of distortion of the rib cage (assessed by magnetometers), inspiratory time (Ti) and the resultant tidal volume (VT)[13]. They found a strong negative correlation between Ti and the rate of rib cage distortion (**Figure 6**). Breaths with the highest rate of distortion achieved the lowest inspiratory times and tidal volumes, suggesting that the infant rib cage can actually distort enough to inhibit its own breathing efforts. The pattern of response to vibratory stimulation of the intercostal muscles suggested that this inhibition occurs via inhibition of phrenic motor neuron discharge by chest wall afferent stimuli.

A number of studies have also attempted to quantitate the mechanical cost of rib cage distortion by measuring the work of breathing. Such studies are particularly difficult in the neonate because one of the measurements needed for the usual calculations of work of breathing is the change in pleural pressure for a given tidal volume. In adults this is usually estimated satisfactorily from changes in esophageal pressure. However, LeSouef et al. have demonstrated

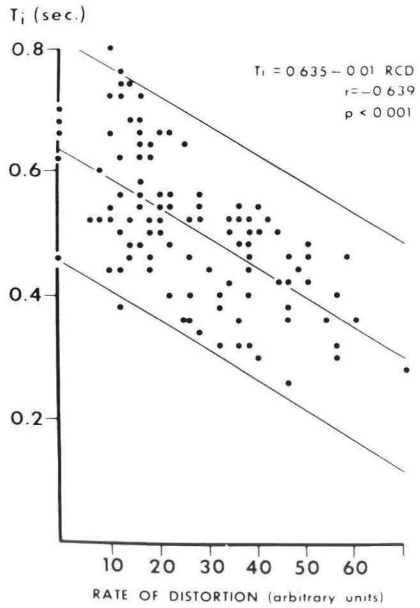

Fig. 6 Relationship between the rate of distortion and inspiratory time (Ti) in an infant during REM sleep. The regression line is shown with 95% confidence limits. Note that rapid rib cage distortion appears to inhibit inspiration. (Reprinted with permission from Hagan et al.: J Appl Physiol 42: 362-367, 1977)

that this standard approach isn't valid in a distorting system.[14] Guslits et al[15]. circumvented this problem by utilizing transdiaphragmatic pressure and abdominal volume displacements to calculate the diaphragmatic component of the work of breathing in preterm infants who had either recovered from RDS without sequelae or had never had a respiratory illness. They calculated diaphragmatic work for breaths with equal tidal volumes but extremes of rib cage distortion. Diaphragmatic work was approximately doubled ($5.9\ g \cdot cm \cdot ml^{-1}$ to $12.4\ g \cdot cm \cdot ml^{-1}$) during periods of paradoxical rib cage motion (**Figure 7**). Howard extended this assessment to infants with bronchopulmonary dysplasia (BPD)[16]. Again, diaphragmatic work was greater during periods of greatest rib cage distortion. Of special significance, however, is the observation that the increase in diaphragmatic work was of substantially greater magnitude in the group of infants with abnormal lungs (**Figure 8**). Howard went on to estimate the caloric cost of this distortion for both normal infants and those with BPD. She concluded that the diaphragmatic work of breathing could go as high as 20 percent of the basal metabolic rate during rib cage distortion in infants with BPD as compared to three percent of BMR during the least distortion. These calculations yielded an overall estimate that ten percent of the daily BMR is required simply to supply adequate energy for diaphragmatic contraction in the infant with BPD. Howard also found a correlation between diaphragmatic work and growth retardation in the infants with BPD. The higher the diaphragmatic work, the greater was the growth retardation. This finding suggests that measures taken to minimize rib cage distortion might improve growth in these infants.

Recognition of high levels of work of breathing inevitably leads one to consider the problem of respiratory muscle fatigue. We noted earlier that the muscle fiber type of the neonate is not ideal. Add to that the cost of a distorting rib cage and the stage is set for fatigue.

In 1979 Muller et al. reported evidence of diaphragmatic fatigue in newborn infants, using surface EMG signals processed to remove interference from the EKG.[17] They found the frequency spectrum was identical to that of adult skeletal muscle, and that under conditions such as rib cage distortion

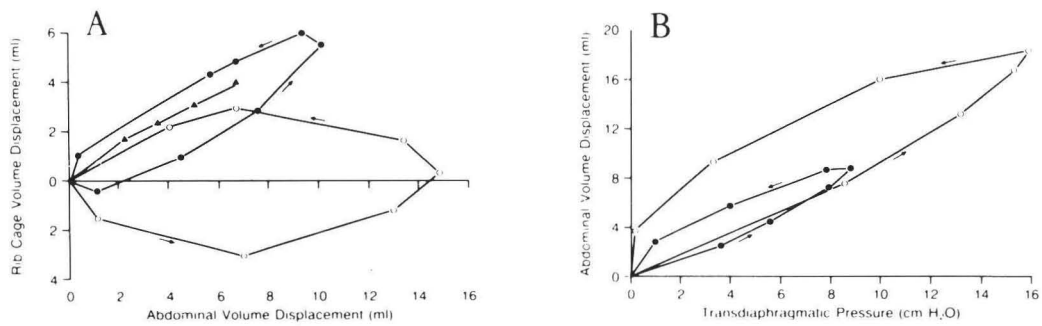

Fig. 7 A. Plot of rib cage and abdominal volume displacements during periods of minimal (closed circles) and maximal (open circles) distortion for a typical infant. Arrows show direction of inspiration and expiration. In the absence of distortion, chest wall expansion occurs along the relaxation line (filled triangles). Note that an increase in abdominal displacement is necessary to compensate for rib cage distortion. O represents the end-expiratory position.

B. Plot of abdominal volume displacement vs. transdiaphragmatic pressure during breaths of equal tidal volume. Symbols same as in A. Note the marked increase in transdiaphragmatic pressure required to maintain tidal volume in the presence of rib cage distortion. (Reprinted with permission from Guslits et al. : J Appl Physiol 62 : 1410-1415, 1987)

the neonatal EMG demonstrated the classic fall in power of high frequency components and increase in power of the low frequency spectrum characteristics of fatiguing muscle. The method was then simplified to produce an on-line index of EMG power spectrum by passing the EKG-gated signal through two band pass filters (one from 150-350 Hz and the other 20-40 Hz) and displaying the ratio of the power in the two bands : the Hi/Lo ratio. A fall in Hi/Lo ratio of greater than 20 percent for one minute was considered to be indicative of fatigue of the monitored muscle.[18] This device was then used to assess the response of infants to attempted weaning from ventilator assistance, and to explore the infant's strategies for adapting to muscle fatigue. A typical example of a weaning trial is shown in **Figure 9.** When this infant was weaned from intermittent mandatory ventilation (IMV) to CPAP alone the Hi/Lo ratio decreased substantially after ten minutes, pointing to diaphragmatic fatigue. Weaning failure was not apparent until 20 minutes later, however, when the infant became cyanotic and apneic. There was prompt recovery of the Hi/Lo ratio on reinstitution of IMV. In this study of ten infants, fatigue was evident from the fall in Hi/Lo ratio in all six infants who failed their weaning trial, and in none of the four infants who weaned successfully for 12 hours or more.

Lopes et al. went on to analyze infant patterns of response to the occurrence of fatigue.[19] He discovered that some infants exhibited an adaptive response to diaphragmatic fatigue, such that they recruited additional intercostal/accessory muscle activity, maintained tidal volume, and allowed the diaphragm to recover. Other infants failed to initiate this adaptive response.

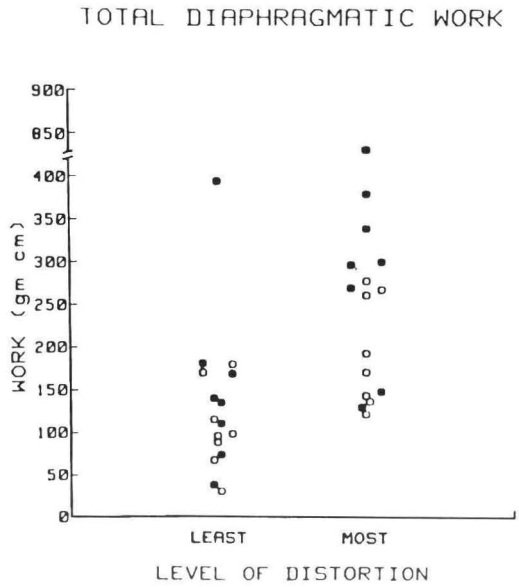

Fig. 8 Plot of total diaphragmatic work (methods same as Figure 7) during breaths matched for tidal volume with either minimal or maximal rib cage distortion. Observations were obtained in normal infants (open circles) and infants with BPD (closed square). Note that distortion increases diaphragmatic work in all infants, but the increase is substantially greater in infants with BPD. (Reprinted with permission from an M.Sc. Thesis by S.E. Howard, York University, 1987)

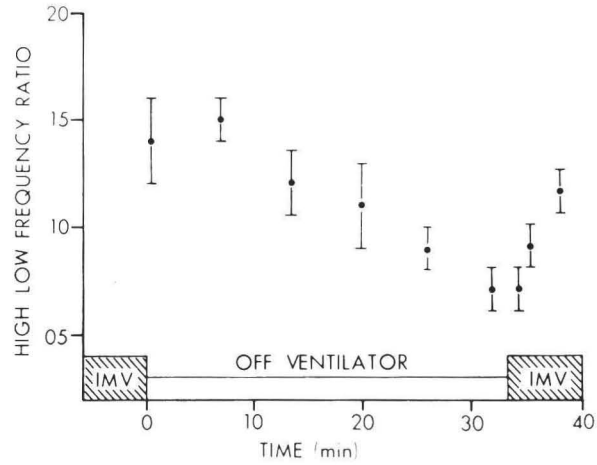

Fig. 9 Time course of high low frequency ratio in an infant being weaned from IMV to CPAP alone. See text for discussion. (Reprinted with permission from Muller et al. : Intensive Care in the Newborn III, pp. 263-269)

Intercostal muscle activity continued unchanged, and the diaphragmatic fatigue led to apnea. They concluded that the intercostal/accessory muscles and the diaphragm can work synergistically in the neonate to increase the overall endurance and minimize fatigue, but that this adaptation is not universally utilized in all infants.

Therapeutic Approaches

So far we have established that the neonate is prone to chest wall distortion, and that this distortion impairs breathing efficiency, potentiates muscle fatigue, and reflexly inhibits inspiration. It is therefore important to explore methods to minimize these problems without actually taking over from the respiratory muscles completely, since the latter approach carries the risk of disuse atrophy and long-term weaning difficulties. In this context it is interesting to note that many of the therapies we already pursue for various reasons may produce at least some of their benefits through chest wall stabilization. For example, neonates are often given theophylline to treat apnea, presumably via its influence on the respiratory control centre. However, theophylline may well operate through other mechanisms as well. It is well known that theophylline enhances muscle contractility. Infants given theophylline generate more pressure against an occluded airway for any given level of diaphragmatic EMG than they do without theophylline (A.C. Bryan : personal communica-

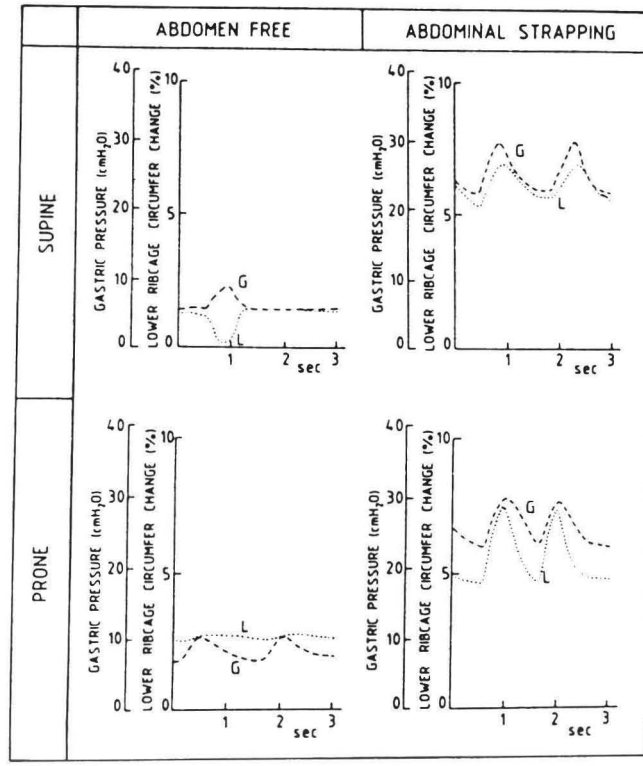

Fig. 10 Tracings of gastric pressure (G) and lower rib cage circumference (L) in an infant before and after abdominal strapping, strapping, in both supine and prone positions. Supine, diaphragmatic contraction (as manifested by an increase in gastric pressure) and lower rib cage motion are totally out of phase with the abdomen free, but become synchronous after abdominal strapping. Prone, lower rib cage motion is less discordant during free breathing but is again enhanced by abdominal strapping. Note that both the end-expiratory value and the tidal swing in abdominal pressure are increased during abdominal strapping. (Reprinted with permission from a thesis by H. Devlieger, Katholieke Universiteit Leuven, 1987)

tion). This force enhancement could reflect action on the diaphragm alone, plus possible added benefits from rib cage stabilization via increased intercostal muscle activity.

Another relatively simple "therapy" is adjustment of body position. We noted earlier that the anatomy of the neonatal chest well is such that there is a high potential for the lower rib cage to be sucked in during diaphragmatic contraction, thereby wasting a significant portion of the respiratory

effort. In a series of careful studies, Devlieger analyzed the phase relationships of motion of different parts of the chest wall supine, prone, and with abdominal strapping (**Figure 10**)[11]. In the upper left quadrant it is clear that diaphragm contraction (as reftected by an increase in gastric pressure) and lower rib cage motion are totally out of phase in the supine position. However, the simple measure of placing the infant prone stabilizes the system somewhat ; the lower rib cage does not really expand but at least it is not sucked inwards. Of interest is the impact of gentle abdominal strapping on the pattern of chest wall motion. In both supine and prone positions, abdominal strapping induced expansion of the lower rib cage that became completely in phase with diaphragmatic contraction.

Abdominal strapping is a therapeutic manoeuvre that has been suggested periodically over the last 12 years but is rarely practised. Henderson-Smart and Read inflated a blood pressure cuff around the abdomen of neonates and found that during REM sleep collapse of the rib cage was prevented[20]. Fleming et al. demonstrated that abdominal strapping could abolish paradoxical breathing during REM sleep without increasing diaphragmatic work of breathing and without impairing minute volume[21]. Chest radiographs during abdominal strapping show a number of changes compatible with improved chest wall efficiency, namely a cranial shift of the diaphragm with a more acute costodiaphragmatic sinus (i.e. an area of apposition is created), and an increase in anteroposterior chest wall diameter without a decrease in lateral diameter (i.e. rib cage circumference increases)[11]. Following abdominal strapping both the end-expiratory gastric pressure and the inspiratory swing in gastric pressure increase, evidence of an increase in the force available to drive rib cage expansion.

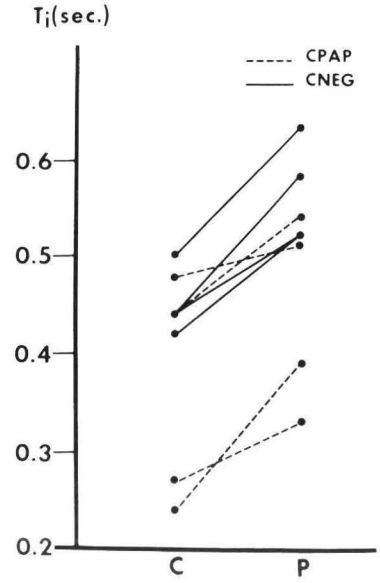

Fig. 11 Changes in inspiratory time (Ti) from control REM periods (C) to REM periods when either CPAP or CNeg (P) was applied in seven infants. In all cases CPAP or CNeg significantly reduced the rate of chest wall distortion, as well as inducing the increases in Ti plotted here. Each line represents one infant. (Reprinted with permission from Hagan et al. : J Appl Physiol 42 : 362-367, 1977)

Abdominal strapping will not help all infants, however. It is possible that some anecdotal reports of people trying abdominal strapping and abandoning it because the neonate deteriorated have arisen because the therapy was tried inappropriately. Devlieger emphasizes that strapping should not be attempted in infants who have low functional residual capacity and low lung compliance, such as infants with RDS[11]. In

these infants strapping will only further reduce FRC without benefit. It is when infants are in the recovery phase and weaning is inhibited by discoordinated movements of the chest wall that gentle abdominal strapping with an elastic bandage may be beneficial.

Rib cage stabilization can also be achieved by the use of CPAP via the airway or continuous negative pressure (CNeg) applied to the body surface. Unquestionably both of these therapies will increase lung volume and improve oxygenation on that basis, but they also have an additional beneficial effect on respiratory pattern via their impact on rib cage distortion. We noted earlier that rib cage distortion can actually inhibit inspiration via stimulation of chest wall afferents. Hagan et al. found that low level CPAP or CNeg acted both to decrease rib cage distortion and increase inspiratory time[13]. These data suggest that at least part of the reduction in apneic spells often achieved by low level CPAP administration can be attributed to rib cage stabilization causing a decrease in inhibitory neural traffic.

Many of the studies quoted in this review are small：neonates are not an easy population to study. Nonetheless a convincing pattern of evidence has accumulated that encourages us to pay close attention to chest wall physiology in our neonatal nurseries. Undoubtedly as we train ourselves to look for these mechanical aspects of chest wall function we will also learn new ways to optimize respiratory efforts in this vulnerable population.

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