# Respiratory Report: To Grunt or Not To Grunt: That is the Question Considerations in the management of transient tachypnea of the newborn (TTN)

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***I dedicate this column to the late Dr. Andrew (Andy) Shennan, the founder of the perinatal program at Women's College Hospital (now at Sunnybrook Health Sciences Centre). To my teacher, my mentor and the man I owe my career as it is to, thank you. You have earned your place where there are no hospitals and no NICUs, where all the babies do is laugh and giggle and sleep.***

Transient Tachypnea of the Newborn (TTN) is the most common respiratory condition seen in the newborn period with estimated occurrence in 0.5 to 4 percent of births. (1) It is a generally benign and self-limiting condition resulting from insufficient clearance of lung fluid during delivery. (2) Risk factors include birth by caesarian section (C/S) without labour, particularly if delivered before 39 weeks (doubles risk), gestational diabetes, small or large for gestational age infants, maternal asthma, perinatal asphyxia, male gender and prematurity. (3)

Clinically, TTN presents most commonly with exhalation against a partially closed glottis ("grunting," the infant's attempt to increase end-expiratory pressure), with or without retractions or nasal flaring. As severity increases, low oxygen saturation ($SpO_2$), respiratory acidosis, and finally, respiratory failure may occur without appropriate support and intervention. Treatment is supportive, the degree of which should be determined by how severely the infant presents.

In NICU's, it is sometimes said that "we make big babies sick." In my opinion, this stems from the tendency to over-treat these infants. Also, the fear of persistent pulmonary hypertension of the newborn (PPHN) may lead to interventions above and beyond what is required. Because of the severity of PPHN, it is hard to blame those at the bedside, however knowing risk factors ahead of time should lessen this fear. These include Cesarean section delivery, high maternal body mass index, maternal use of aspirin, nonsteroidal anti-inflammatory agents, and maternal diabetes mellitus are among the factors associated with an increased risk for PPHN.

Recent data suggest that maternal use of serotonin reuptake inhibitors might represent another important risk factor for PPHN. There appear to be no effective preventative strategies. (4) PPHN is, however, less likely to occur after 72 hours. (5) Further adding to the fear of PPHN is the fact that both birth by C/S and TTN are considered risk factors for PPHN, as well as RDS, post-term delivery, birth asphyxia, meconium aspiration, and sepsis. (6) That sepsis often first presents as mild respiratory distress compounds the dilemma of whether or not to escalate treatment and proper investigations to rule it out is prudent. Premature prolonged rupture of membranes (PPROM), particularly prior to 20 weeks gestation, also increases the risk of PPHN. (7)

Given the above risk factors and attending sequelae, it is not hard to see why so many clinicians immediately put these babies on some form of support, most notably CPAP; however, as benign as we believe CPAP is as a therapy, there are other forms of support available. Placing a baby on CPAP sets up a chain of events that may increase the length of stay unnecessarily, interfere with the establishment of oral feeding, and parental bonding. While, obviously, keeping a baby stable (and alive!) takes precedence over bonding, there are many babies who are well enough to simply monitor. Who might they be?

***"One quick way to assess respiratory distress in a newborn is to offer them something to suck on, preferably their mother's breast. As a general rule, infants in significant respiratory distress will not suck or latch."***

A baby who is grunting but otherwise well, in room air with $SpO_2$ 95-100%, is clearly managing their pathology well themselves and, personally, I am reluctant to intervene. I believe these babies can, I believe, be safely watched without support as long as $SpO_2$ remains in that range, especially bigger full-term babies who have an adequate reserve. They can carry on this way for quite some time without tiring provided they do not become either hypothermic or hypoglycemic, (both of which can lead to secondary surfactant deficiency and respiratory distress syndrome), something those used to dealing with premature infants tend to forget. This is often a case of "less is more," but, the most difficult procedure in the NICU seems to be sitting on one's hands, what one of my mentors refers to as "skillful neglect."

One quick way to assess respiratory distress in a newborn is to offer them something to suck on, preferably their mother's breast. As a general rule, infants in significant respiratory distress will not suck or latch. Offering skin to skin contact with their mothers quite often works like magic. Prone positioning may also help. Skin to skin can also be initiated post C/S in the operating room. (8)

Should $SpO_2$ fall, supplementary oxygen may be given by nasal prongs, and support may be escalated to high flow nasal prongs with supplemental oxygen. (9) Another advantage of these less cumbersome therapies is that bigger babies tend not to like CPAP interfaces and are more comfortable with low flow or high flow prongs, and with larger babies, a "hands-off", minimal handling approach is best whenever possible. In my experience, they are also more likely to be offered kangaroo care or other parental contact and be fed orally. As well, there are attending costs associated with escalating treatment in terms of equipment and length of stay.

Knowing existing risk factors for more serious pathologies both on the maternal and infant side is essential when managing TTN less invasively, and waiting and watching does require close and careful monitoring, especially of $SpO_2$. A caveat here: the fear of a baby "flipping" into PPHN leads many clinicians to maintain SpO2 high with supplemental oxygen artificially. While well-intentioned, hyperoxia leads to increased formation of free radicals, one of which is a potent vasoconstrictor that will blunt the effect of inhaled nitric oxide or prevent it completely. (10) It is my practice to

set low $SpO_2$ alarms at 90-92% to prevent "falling off the cliff" on the oxygen desaturation curve but to not keep $SpO_2$ above 98% artificially.

Pneumothoraxes (particularly pneumomediastinum) may also present as grunting, and these should be ruled out before initiating nasal CPAP as these babies are best managed with supplemental oxygen and not positive pressure which may exacerbate the problem. Very often these will resolve on their own without intervention, although pneumothoraxes increase the risk of PPHN. The largest risk factors for spontaneous pneumothoraxes are birth by C/S, being male, higher birth weight and being large for gestational age. (11)

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Addendum

July's column on MIST/LISA was inadvertently submitted missing two paragraphs, without which my view of the practice may be interpreted as overly negative. They are below, with apologies.

"There is also the endotracheal tube itself. Phthalates used in the production of PVC pose known health risks to humans, particularly to children, neonates being particularly at risk. These chemicals leach from the during the first 24 hours of placement.(12) PVC is used extensively in medication bags and tubing which are also used in the neonatal population thus isolating the effect of the ETT itself from a phthalate standpoint is impossible. Still, any reduction in phthalate exposure can only be a good thing. (13) Still, it is ostensibly what is done after the insertion of an ETT that creates the most harm, hence it is essential that clinicians have excellent knowledge of lung-protective ventilation strategies that are carried into practice as there are patients who will require intubation and mechanical ventilation.

Another factor to consider in very premature patients being managed with NIV is $FiO_2$. Until anti-oxidant production and supplementation are established, the premature infant has no protection from free radicals and is therefore very susceptible to oxidative stress. There seems to be great variance in clinical practice when it comes to just how high a safe $FiO_2$ is. I firmly believe less is best since $FiO_2$ is the barometer of pulmonary compliance. It is common practice to withhold surfactant replacement therapy until a threshold $FiO_2$ is reached, usually around 0.30. I do not embrace this practice, as there are consequences to delaying surfactant treatment. Increased length of exposure to higher $FiO_2$ in a patient lacking endogenous anti-oxidant protection and who's susceptibility to oxidative stress is increased is not, in my opinion, wise. Also, delayed treatment increases the risk of air leak, most notably pneumothorax. (14) (Please note I do not advocate waiting for FiO2 to be as high as this reference suggests. It is my practice to give intubated infants with any increased $FiO_2$ surfactant.) Any practice that reduces the reluctance of a clinician to give surfactant is a good thing in my mind."


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