Occurrence and Mechanisms of Sudden Oxygen Desaturation in Infants Who Sleep Face Down

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ABSTRACT. Objective. Infants who sleep prone and face down on soft bedding are particularly vulnerable for sudden infant death syndrome. It has been suggested that 1 mechanism of death in this situation involves rebreathing of expired air. Many infants tolerate rebreathing while lying prone face down for long periods with stable saturations. Others occasionally have rapid desaturations and may require intervention to terminate rebreathing. The present study had 3 objectives: 1) to determine the frequency of rapid desaturations in a large group of healthy infants, 2) to elucidate the mechanism of these desaturations, and 3) to determine the timing of these events during periods of rebreathing.

Methods. We studied respiratory tracings and videotapes of 56 healthy 1- to 6-month-old infants who were sleeping face down and rebreathing on soft bedding in our laboratory. We compared the frequency of desaturations during rebreathing and nonrebreathing periods. We measured respiratory frequency and apnea occurrence before desaturation and nonrebreathing control episodes. We also measured minute ventilation during steady state before desaturation and just before desaturation.

Results. There were 25 desaturation episodes in infants while rebreathing, occurring in 11 (19.6%) of the 56 infants. Episodes were significantly more frequent during rebreathing infants reach steady state with regard to ventilation and oxygenation, even with documented airway obstruction, but there has been no documentation of this. Desaturation in a rebreathing environment is of special concern because central nervous system (CNS) depression occurs at saturations <50%, and this could prevent the infant’s escape from the asphyxiating environment.

Anecdotal accounts of infants dying while face down indicate that death may occur relatively rapidly. In contrast, animal models of rebreathing indicate that death occurs after a longer period of time. Therefore, we sought to establish the onset of rapid desaturation after assuming the face-down position, as this could be the immediate precursor to death.

METHODS

Participants

We studied 56 healthy infants from the St Louis community (mean age: 103 days; range: 40–180), all of whom were healthy at the time of the study. Four of these were premature and were studied at a mean post conceptional age of 339 days (range: 292–377); 2 additional infant’s sibling had died of SIDS. The remaining infants had no history of significant medical problems. Of the 56, 11 (6 boys, 5 girls) experienced significant episodes of desaturations during the study (mean age at study: 73 days; range: 19–120). These infants formed the primary group for study of desaturations. The study was approved by the Washington Uni-
desaturations in which movement artifact was detected by instability in the pulse tracing and/or the ECG tracing to eliminate artifactual desaturations. We evaluated 25 desaturations with a stable pulse signal or a single spike in the pulse signal. To confirm the validity of this approach, we evaluated the change in saturation associated with this pattern during nonrebreathing periods. It was not associated with any change in saturation during nonrebreathing periods. In 6 of the 25 desaturation episodes that were studied, the pulse signal was not recorded, in which case we relied on presence or absence of movement artifacts in the ECG because movement artifacts in the ECG were invariably present when the pulse trace reflected infant movements.

Desaturation episodes were classified as occurring during rebreathing periods (inspired CO₂ ≥1%) or during nonrebreathing periods (inspired CO₂ undetectable). We calculated the duration (in minutes) of rebreathing and nonrebreathing periods for each infant. We then calculated for each infant the frequency of desaturation episodes per rebreathing and nonrebreathing periods. The timing of onset of desaturations after the infants assumed a face down position was noted.

Respiratory Parameters: Rebreathing Desaturation Episodes Versus Nonrebreathing Control

The number of desaturations was determined for each infant during control and rebreathing periods. We then randomly selected nonrebreathing control periods with no desaturations for each infant by a random sampling procedure based on that described by Huntsberger and Leaverton.13 Each page of the tracings represented 30-second epochs of the study. We used a random-number generator to select a page (www.randomizer.org). If the selected page met control criteria, then we selected that page. If not, then another number was drawn until a nonrebreathing page without desaturation was selected. The beginning of the page was used as the control period. This entire method was repeated for each desaturation for each infant using this method. We established 25 controls, 1 for each desaturation. We also evaluated the respiratory pattern 10 seconds before the onset of the desaturation and control periods, comparing respiratory frequency and occurrence of brief apneas (Fig 1A).

Change in Minute Ventilation During Rebreathing Before and Immediately Before Desaturation Episodes

We calculated the relative change in minute ventilation by measuring respiratory frequency and tidal volume \( (V_e) \) by the height of the integrated flow signal or the height of the Respitrace sum signal during 2 time periods: 1) the 8 seconds before the onset of the desaturation through to the desaturation nadir (to account for the 5- to 7-second averaging time of the pulse oximeter) and 2) the previous 8 seconds to the aforementioned time period if the infant's saturations were stable at that point (Fig 1B). If the satu-
rations were not stable, then we evaluated 8-second intervals sequentially preceding the interval until a stable time period was found. Both measures of $V_T$ were used in the infants, but the same measure was used for each desaturation and preceding interval to allow for a measure of relative change in minute ventilation. Average minute ventilation was calculated by averaging $V_T$ over the number of breaths counted multiplied by the frequency. This procedure was done in 17 of 25 desaturation episodes in which $V_T$ could be calculated for both time periods by the same method. In addition, we measured average end-inspiration CO$_2$ during both time periods.

Cause of Desaturation

We reviewed the polygraph tracings and videotapes of the infants to evaluate for a correlation in ventilatory patterns or behavioral features 10 seconds before the onset of desaturation. Arousal was defined as evidence of vocalization and/or body movements.

Data Analysis

Statistical analyses were performed using the Wilcoxon rank test and the paired Student $t$ test. Results are reported as mean ± standard error of the mean. Results were considered significant at $P < .05$.

RESULTS

Desaturation Occurrence and Timing During Rebreathing

Eleven (19.6%) of the 56 infants we studied experienced 25 episodes of desaturations with a mean of 2.3 spells per infant (range: 1–7; Fig 2). Spells did not occur in the SIDS siblings or any of the preterm infants. There was no apparent correlation of gender or age of the infant and occurrence of spells. Desaturations occurred more frequently during rebreathing than during nonrebreathing periods ($0.07 ± 0.02$ vs $0$ episodes/min, rebreathing and nonrebreathing, respectively; $P < .01$). Eight desaturation episodes reached ≥90%. Three additional desaturations occurring in 3 infants reached ≤85%. Onset of desaturation was variable but often occurred within as short a time as 1 to 3 minutes after infants assumed the face-down position.

Respiratory Parameters: Rebreathing Desaturation Episodes Versus Nonrebreathing Control

Respiratory frequency was not significantly different during the 10 seconds before desaturation than nonrebreathing control periods ($40.3 ± 2.3$ vs $45.4 ± 3.7$, desaturation and control, respectively; $P > .05$). Brief apneas occurred more frequently before desaturations than nonrebreathing control periods ($72 ± 20\%$ vs $4 ± 4\%$, desaturation and control, respectively; $P < .01$). The mean apnea duration was 2.98 seconds (range: 1.0–12.2 seconds).

Change in Minute Ventilation During Rebreathing Before and Immediately Before Desaturation Episodes

The average change in minute ventilation across the 17 desaturation episodes that could be accurately evaluated was a decrease from $466.5 ± 79.3$ to $429.7 ± 48.4$ (arbitrary units mm/min; $P = .56$). In 9 of theses 17 episodes, minute ventilation decreased by an average of 33.5%. In 8 of these episodes, the decrease in ventilation was associated with brief apnea.

Cause of Desaturation

Eleven of the 25 desaturation episodes were preceded by 1 or more short apneas. These were all central apneas with the exception of 1 in which there were clear indications of completely obstructed respiratory efforts. Three of these apneic episodes were associated with grunting or crying. One episode was associated with a transient decrease in minute ventilation without accompanying apnea. This was attributable to a decrease in $V_T$. An additional 3 episodes were associated with a failure to increase minute ventilation despite rising inspired CO$_2$. Five desaturation events were preceded by a spontaneous shift in head position, which increased airway contact with the bedding during the 10 seconds before desaturation. Snorting or snoring sounds, suggesting
upper airway obstruction, preceded 2 episodes in 2 infants in 1 of whom this was associated with evidence of arousal of sleep (Fig 3).

Most episodes of desaturation resolved when the infant turned and/or lifted his or her head. However, 2 infants had 1 episode each of desaturations <85% while rebreathing that required intervention by the examiner to prevent additional desaturation. One of these infants awoke before intervention, but the behavioral response was ineffective at escaping the rebreathing environment. He grunted, cried, lifted, and turned his head slightly after saturations reached 87% but then dropped his head into the rebreathing pocket once again. He was turned when the saturations reached 84% and he had not successfully repositioned his head. The other infant had a desaturation to 80% in which she demonstrated no signs of arousal and was turned supine by the examiner. None of these episodes was associated with bradycardia.

DISCUSSION

Although the majority of rebreathing infants reach steady state with regard to ventilation and oxygenation, even with documented partial airway obstruction, a few infants have been noted to desaturate rapidly, occasionally requiring rescue. Such occurrences are relatively infrequent. Furthermore, it should be noted that although the definition used in the present study was based on established norms for desaturation, most spells that we observed would not be regarded as medically significant. However, our findings do indicate that sudden small desaturations while rebreathing are not uncommon. Furthermore, they indicate that potentially more serious larger decreases can occur in healthy infants, some of whom fail to escape or arouse. It should be noted, however, that the bedding we used was selected for its high potential for causing rebreathing.

When minute volume reaches maximum levels during rebreathing, there is a tenuous balance between O2 consumption and diffusion of O2 into the bedding beneath the infant’s face. Recovery of O2 saturation to the previous level after a large decrease would be unlikely to occur in the absence of significant change in head position allowing access to fresh air. This is because O2 concentration in inspired air during rebreathing correlates closely with the stores of O2 in the infant’s body; hence, a continued downward trend in saturation would be expected to occur after a significant decrease in O2 stores. Such a situation therefore could constitute a critical event in the pathway to death because hypoxemia is a poor stimulus for arousal and CNS hypoxia produces rapid onset of hypoxic coma, at which point spontaneous escape from the hypoxic environment becomes impossible.

SIDS deaths are called “sudden”; however, the actual time from onset of respiratory or circulatory compromise to actual death is usually uncertain. Deaths in infants who have assumed the face-down position have been anecdotally reported to occur in as short a time as 10 to 20 minutes. This contrasts with the anesthetized animal model of rebreathing in which death occurs after 1 or 2 hours when acidosis and hypoxemia result in hypotension and bradycardia. The present findings suggest that death might occur much more rapidly in sleeping infants in situations in which respiratory pauses associated with otherwise normal infant behaviors produce rapid and potentially irreversible decreases in saturation.

In infants who sleep in the prone position, we found that desaturations occurred more often while they were rebreathing than not rebreathing. This is likely attributable to the hypoxic environment that coexists with hypercarbia in a rebreathing setting. Partial pharyngeal obstruction as suggested by snoring or snorting was heard in 2 infants, and complete obstruction was present in an additional infant; this may have played a role in some desaturation episodes. However, the primary mechanism that we found for desaturations was brief central apneas. In 9 episodes, a decrease in minute ventilation was noted, the majority of which were associated with apnea. Decreased minute ventilation was also associated with behavioral features of arousal such as crying or grunting, which have been previously been shown by others to be associated with desaturation.

Crying for help is an appropriate protective response for the infant at risk, although it may prevent an appropriate ventilatory response to increasing environmental CO2. Previously, we have shown that minute ventilation increases 3-fold in infants in similar rebreathing environments. Others have shown in experimental settings that minute ventilation increases by a factor of 1.25 to 2.75 with rising CO2 in a similar range to that observed in our participants when rebreathing. Increasing minute ventilation is a primary mechanism for achieving a steady state during rebreathing. Because ventilation reaches maximum attainable levels during rebreathing, it would be difficult for the infant to recover from a transient decrease in respiratory rate or VT, producing desaturation without getting access to fresher air.

Most infants demonstrated an appropriate motor response after desaturation was present by either moving the head to the side or lifting it up. Two (8%)
of the infants studied had arousal responses that were ineffective in avoiding a potentially dangerous environment. One of these infants reassumed the face-down position during his arousal response, which led to an increase in inspired CO₂ and additional desaturation. Previous work by Lijowska et al⁸ has shown that the arousal response to an asphyxial environment may sometimes aggravate an already dangerous situation if the infant is ineffective at clearing the airway and/or turns the head further into the bedding. Thus, although arousal is believed to play an integral role in protecting infants from sudden death while sleeping, such as by head repositioning, the associated cardiopulmonary changes, such as altered respiratory patterns or apnea, may also precipitate hypoxemia in some infants.

CONCLUSION

Normally occurring respiratory patterns associated with normal infant behavior usually have little consequences for blood gas homeostasis. However, in a low-O₂ environment associated with the infant’s face being covered by bedding, these same respiratory patterns may produce a rapid desaturation. When associated with arousal activity that is ineffective in gaining access to fresh air or failure to arouse, such desaturations can produce an immediate danger, potentially leading to death.

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