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Alarms, oxygen saturations, and SpO₂ averaging time in the NICU

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Abstract

BACKGROUND—Alarm overload is a significant concern in the Neonatal Intensive Care Unit (NICU). Selecting a longer oxygen saturation (SpO₂) averaging time will reduce the number of alarms but may mask fluctuations in oxygenation.

OBJECTIVE—Characterize bedside monitor alarms in the NICU and estimate the impact of longer SpO₂ averaging time and alarm delay.

METHODS—All bedside monitor alarms were analyzed over a 12-month period in the University of Virginia NICU, using the default averaging time (8 seconds) and SpO₂ alarm limits set at 88–95% for infants on supplemental oxygen. In 10 VLBW infants, SpO₂ averaging time was lowered to 2 seconds for 24 hours and events of SpO₂ out of the target range were estimated with 2-, 8-, or 16-second averaging time, with and without a 15-second alarm delay.

RESULTS—There were 3,263,590 alarms in the unit over 12 months. Low or high SpO₂ alarms constituted 79% of the total, and 55% of these events lasted <15 seconds. In 10 infants we estimated that increasing SpO₂ averaging time from 2 to 16 seconds would have led to 53% fewer SpO₂ alarms but the mean duration of alarms would have been 2.15-fold longer. Adding a 15-second alarm delay to 2-second SpO₂ averaging in this analysis decreased SpO₂ alarms by 67%.

CONCLUSION—Longer SpO₂ averaging times mask the number and severity of events of aberrant oxygenation in preterm infants without decreasing total alarm time. Incorporating an alarm delay with shorter SpO₂ averaging times can reduce alarm number and duration, and allow more accurate assessment of oxygenation, which may be important for research into consequences of aberrant oxygenation in this vulnerable population.

1. Background

Bedside monitor alarms may signal a decline in a patient's condition but an excessive number, duration, or intensity of alarms can lead to "fatigue" on the part of bedside caregivers [1]. Desensitization to alarms may lead to failure to respond to important or even critical alarms [2–5], and improving alarms management has been identified as a priority for patient safety for over a decade [6].

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Conflicts of interest
none.

In the Neonatal Intensive Care Unit (NICU), the majority of alarms are due to low or high oxygen saturation. Hypoxemia and iatrogenic hyperoxemia [7] are particularly common among very low birth weight (VLBW) preterm infants with lung disease and central and obstructive apnea [8], and aberrant oxygenation can contribute to adverse outcomes, including retinopathy of prematurity and neurodevelopmental impairment. Recent randomized trials showing lower mortality for preterm infants with SpO₂ targeted to 91–95% [9, 10] have heightened the focus on careful titration of supplemental oxygen [11, 12] which may lead to increased alarm burden. Since responding to monitor alarms diverts nurses' attention from other important patient care responsibilities, a more rational approach to alarm management is needed.

One strategy for reducing the number of alarms is selecting a longer SpO₂ averaging time. Most oximeters allow users to select averaging time from 2 to 16 seconds, and several studies have shown that longer averaging times lead to fewer SpO₂ alarms but also misrepresent the number and severity of low SpO₂ events [13, 14]. Many of these events are brief and self-limited, and it is not known to what extent they impact long-term outcomes of preterm infants.

In the current study, we aimed to characterize the number and types of all NICU bedside monitor alarms over a one-year period during which we used the oximeter default SpO₂ averaging time (8 seconds). We then focused on the very low birth weight (VLBW, <1500 grams) infant population during that time period to determine the amount of time spent at a low SpO₂ (<88%). Finally, we sought to estimate the impact of selecting shorter or longer SpO₂ averaging times and incorporating an alarm delay on the number and duration of alarms and the ability to accurately assess oxygenation in VLBW infants.

2. Methods

This study was approved by the University of Virginia Institutional Review Board and was conducted in the UVA NICU, a quaternary care unit with 45 beds in a “pod” structure (average 6 beds per pod). General Electric bedside monitors were in use during the study period, with internal pulse oximeters that employ Masimo technology with a default SpO₂ averaging time of 8 seconds and no alarm delay. Standard alarm settings included low SpO₂ <88%, high SpO₂ >95% (if on supplemental oxygen and without cyanotic heart disease), apnea >15 seconds with no detected chest impedance respiration signal, tachypnea >100 breaths/minute, bradycardia <90 beats/minute, and tachycardia >200 beats/minute. Blood pressure alarm parameters were infant-specific, generally setting the low threshold for mean arterial blood pressure as the infant's gestational age in weeks.

The BedMaster system (Excel Medical Electronics, Jupiter FL, USA) was used to retrieve bedside monitor alarms and every-2-second SpO₂ from all NICU patients in 2013. We excluded the following categories of alarms: 1) a small number of alarms from patients in clinical studies with non-standard equipment that had alarms stored in the Bedmaster system, 2) rare cases in which the ventilator alarms were collected in the BedMaster system, 3) alarms whose duration was given as “0 seconds,” which represent a single SpO₂ value (<4 seconds) out of target range, and 4) alarms >600 seconds or with no duration recorded.

Some of the latter were identified as “lead fail,” “SpO₂ probe,” or “asystole” and may have been associated with infants being disconnected from the monitor rather than activating the alarm. Others were identified as low or high SpO₂ and may have been associated with inappropriate alarm parameters (for example infants on room air with the high saturation alarm set at 95% or infants with congenital heart disease with the low SpO₂ alarm set too high). We therefore excluded SpO₂ alarms >600 seconds to avoid overestimating alarm burden with properly set SpO₂ limits. In total, the excluded alarms represented 9.5% of all alarms recorded in the BedMaster system.

Since the majority of alarms were due to low oxygen saturations in VLBW preterm infants, we also assessed the amount of time VLBW infants spent with SpO₂ <88%. All available SpO₂ values (collected every 2 seconds with 8-second averaging) were analyzed for all VLBW infants in the unit in 2013.

Finally, we sought to determine the impact of shorter and longer SpO₂ averaging times on the number and duration of events of SpO₂ out of the set alarm limits of <88 and >95% SpO₂. We randomly selected 10 VLBW infants on respiratory support (ventilator or continuous positive airway pressure) with supplemental oxygen and changed the oximeter averaging time from the default setting which was standard in our unit (8 seconds) to the shortest averaging time (2–4 seconds) for 24 hours. The BedMaster system collects SpO₂ every 2 seconds, and for purposes of this report we used the lower end of 2–4 second averaging range to estimate what the SpO₂ would have been with longer averaging times. We thus averaged 4 values recorded every 2 seconds to estimate 8-second averaging, and 8 values to estimate 16-second averaging. This method (averaging an average) would be expected to underestimate the number and duration of desaturation events that would have been missed with longer averaging times.

We calculated the number and duration of alarms if 8- or 16-second SpO₂ averaging had been set on the oximeters, focusing on both the standard alarm limits in our unit at the time (<88% and >95%) and on more severe events (<70% and >98%). Consistent with the analysis of actual alarms, in which we excluded those with duration of “0 seconds” representing a single SpO₂ value out of range, for the averaging time analysis we required two SpO₂ values out of range to qualify as an alarm event. Since SpO₂ is recorded every 2 seconds, we thus excluded alarms representing <4 seconds of out-of-range oxygen saturations.

Many hypoxemia and hyperoxemia events in preterm infants are brief, and we calculated the impact of a 15-second alarm delay which may be selected on some NICU bedside monitors and pulse oximeters. This was done by excluding any event in which there were fewer than 8 sequential every-2-second SpO₂ values outside of the SpO₂ alarm parameters.

For statistical analysis, we compared the measured 2-second averaging SpO₂ values with the estimated 8- and 16-second averaging values, as well as the number and duration of low or high SpO₂ events for the 10 infants with and without a 15-second delay time, using Wilcoxon matched pairs signed rank tests with adjustment for multiple comparisons. Statistical testing was performed in GraphPad Prism 7 (LaJolla, CA).

3. Results

There were 3,263,590 bedside monitor alarms recorded in the 12-month period, during which time there were 710 infants admitted (mean $\pm$ SD gestational age 35.4 ± 4.1 weeks, and birthweight 2609 ± 960 grams). The average daily census was 36 and the nurse: infant ratio 1 : 3, thus there were, on average, 250 alarms per infant per day and 31 alarms per nurse per hour. The mean duration of alarms was 54 seconds, which translates to 134 cumulative hours of alarm noise per day throughout the unit.

Number of alarms by category is shown in Table 1. Equipment failure or artifact accounted for 7% of alarms, 12% were due to cardiovascular events, and 80.5% represented breathing or oxygenation-related events. High oxygen saturation alarms tended to have the longest duration (average 58 seconds) and bradycardia or high or low respiratory rate the shortest (19 seconds). Apnea alarms were relatively infrequent, as were tachypnea and tachycardia. Cardiac rhythm alarms constituted 3% of the total and specific rhythms identified (asystole, ventricular tachycardia) were generally considered to be artifact due to lead malposition or movement.

Seventy-nine percent of all alarms were due to low or high SpO₂, with alarm parameters for infants on supplemental oxygen generally set at <88% and >95%. We analyzed every-2-second SpO₂ values stored in the BedMaster system from all patients in the 12-month period and 2.8 out of 456 million values (0.6%) were <88%. Limiting the analysis to VLBW preterm infants, 13% of SpO₂ values were <88%. Figure 1 shows the percentage of time at SpO₂ <88% in the first 8 weeks after birth for extremely low birth-weight infants (<1000 grams, $n = 36$) and for infants with birth weight 1000–1499 grams ($n = 56$). The number of low SpO₂ events peaked at 4–6 weeks of age for these infants.

To estimate the impact of shorter and longer SpO₂ averaging times on alarms, we randomly selected 10 VLBW infants on respiratory support and supplemental oxygen (GA 22–28 weeks, postmenstrual age 23–32 weeks). We reset the averaging time from the default (8 seconds) to the lowest setting (2–4 seconds) for 24 hours. We then analyzed SpO₂ values collected every 2 seconds and averaged them to estimate what the SpO₂ would have been with 8- or 16-second averaging (see methods). SpO₂ values were significantly lower with 2-second averaging compared to 8- or 16-second averaging values for each of the 10 infants ($p < 0.001$). We also calculated the number and duration of desaturation events < 88% and < 70% and high saturation events >95% and >98% with 2-second averaging and estimated these for averaging times of 8 or 16 seconds (Table 2). With 16-second averaging, about half of the low or high SpO₂ events detected with 2-second averaging would be missed, and the duration of events would be longer. As a result, the total amount of time that alarms would be sounding for SpO₂ out of the target range would not be significantly different with longer averaging times.

Since many SpO₂ alarm events in preterm infants are brief (55% of those in our 12-month analysis lasted <15 seconds, Table 1), we also estimated the impact of choosing an alarm delay which is an option on some pulse oximeters and cardiorespiratory monitors. Table 2 shows the potential impact of setting a 15-second alarm delay with the shortest and longest

averaging times, based on our analysis of 10 VLBW infants on respiratory support with frequent events. The 15-second alarm delay in this analysis would reduce the number of alarms by 67% (for 2-second averaging) and 33% (for 16-second averaging). With this 15-second delay, there were significantly fewer events <95% with 16-second compared to 2-second averaging, but no significant difference in events <70%, <88%, or >98%.

4. Discussion

Alarms are a double-edged sword in ICUs. Critical alarms can be life-saving, while an overwhelming number of non-critical alarms creates excess noise and caregiver desensitization which may lead to failure to respond to critical alarms. We report over three million bedside monitor alarms over the course of one year in a Neonatal Intensive Care Unit, which translated to more than 30 alarms per nurse per hour. Through computations, we show that selecting longer pulse oximeter averaging times would be expected to lead to fewer but longer alarms and underrepresentation of the frequency and severity of brief episodes of hypoxemia and hyperoxemia.

The fact that the majority of alarms in the NICU are due to low oxygen saturation in VLBW preterm infants is not surprising, since these infants have apnea and lung disease which lead to frequent fluctuations in oxygenation. We have previously reported that bedside monitor apnea detection is inaccurate, with both false negative and false positive alarms [5, 15]. In the current analysis, apnea accounted for only 1% of the 3.2 million alarms, while approximately 80% were due to SpO₂ out of the set target range. Some of the SpO₂ alarms may represent motion or perfusion artifact, and efforts to improve accuracy of apnea and SpO₂ analysis would be expected to lead to some improvement in alarm burden [16].

In recent years there has been a heightened focus on avoiding hypoxemia in extremely preterm infants, after a large prospective meta-analysis of three randomized clinical trials reported lower mortality and less necrotizing enterocolitis when SpO₂ is targeted at 91–95% versus 85–89% [9]. Achieving this narrow range of SpO₂ is a very difficult task, and efforts to improve oxygen targeting include lowering nurse:patient ratios for high risk infants, regularly reviewing SpO₂ histograms with all caregivers, careful titration of supplemental oxygen, and optimizing respiratory support and caffeine therapy [17–19]. Ventilators which automatically adjust FiO₂ based on SpO₂ show promise. In a clinical trial of preterm infants on non-invasive respiratory support, time spent with SpO₂ in target range was 62% with automated FiO₂ adjustment compared to 54% with manual FIO₂ adjustment [20]. In the manual control group, over 100 adjustments in FiO₂ were made by bedside caregivers each hour. It is unrealistic to expect nurses to respond to all bedside monitor alarms and accurately titrate oxygen on multiple preterm infants in addition to their many other patient care responsibilities, and further development of automated oxygen adjustment systems is warranted.

The impact of SpO₂ averaging time on number of alarms is intuitive and our findings are similar to those in two previous studies. Ahmed and colleagues recorded desaturation events in 22 neonates over a 24-hour period using two pulse oximeters simultaneously, one with averaging time set to 2 seconds and the other 16 seconds [13]. They reported detecting more

frequent and severe desaturation events with 2-second averaging and also found that the longer averaging time overestimated the duration of hypoxia by interpreting a cluster of desaturations as a single prolonged event. Similarly, Vagedes et al. studied recordings from 226 hours of data for 15 neonates to determine the effect of 7 different averaging times on the time spent in 5 different SpO₂ ranges [14]. They reported that shorter averaging times result in the detection of lower SpO₂ nadirs. In both of these studies, the total time spent within a targeted SpO₂ range was not significantly affected by different averaging times. Vagedes also developed a formula to estimate desaturation events of specific depth and duration with SpO₂ averaging times other than the one used to collect the original data [21], which may be useful for multicenter research among units that use different SpO₂ averaging times.

For research purposes, analyzing accurate, frequently sampled SpO₂ data is important to identify patterns and timing of aberrant oxygenation associated with preterm infant conditions such as necrotizing enterocolitis, retinopathy of prematurity, bronchopulmonary dysplasia, and neurodevelopmental impairment. Using the oximeter default averaging time (generally 8 seconds) or selecting an even longer averaging time will result in fewer but longer alarms and may lead to caregivers increasing FiO₂ for infants whose deep but brief desaturation events have resolved while the averaged SpO₂ remains low, potentially leading to rebound hyperoxemia. The optimal solution appears to be incorporating an alarm delay with shorter averaging times, which will substantially reduce the number and duration of alarms and facilitate collecting and responding to more accurate oxygenation data.

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Fig. 1.

Percent of time at SpO₂ <88% for VLBW infants. For each week after birth, the median percent of time with SpO₂ <88% was calculated for all infants <1000 grams birthweight ($n = 36$, black solid line) and 1000–1499 grams birthweight ($n = 56$, black dashed line). Interquartile range is shown in gray. During the 1-year period of study, the default SpO₂ averaging time of 8 seconds was used, and the low SpO₂ alarm limit was set at 88%.

Fig. 2.

Example of impact of SpO₂ averaging time on detection of desaturation events.

Representative example of a desaturation event in a preterm VLBW infant with SpO₂ averaging time at the shortest setting (2–4 seconds, thick black line), and calculated SpO₂ for the same 30 second time period if the oximeter had been set to 8 or 16 second SpO₂ averaging (thin black and dotted grey lines, respectively).

Table 1

Type, number, and duration of bedside monitor alarms over 12 months (with SpO₂ alarm limits 88% and 95% for infants on oxygen)

Alarm category	Alarm type	Number	% of total	Seconds/alarm (average)
Breathing	Apnea	36,869	1%	21
	High/low respiratory rate	1,529	0.5%	19
Oxygenation	Low SpO ₂ <15 seconds	991,973	30%	5
	Low SpO ₂ >15 seconds	694,486	21%	88
	High SpO ₂ <15 seconds	427,637	13%	6
	High SpO ₂ >15 seconds	488,384	15%	103
Cardiovascular	Bradycardia	90,786	3%	19
	Tachycardia	18,921	1%	30
	Blood pressure	179,246	5%	68
	Cardiac rhythm	91,813	3%	24
Equipment	Lead off, artifact	241,946	7%	22
Total		3,263,590	100%	54

Table 2Impact of SpO₂ averaging time on detection of low and high SpO₂ events

SpO ₂	Event number or duration	SpO ₂ averaging time				2 sec ave		16 sec ave	
		2 sec	8 sec	16 sec	+15 sec delay	2 sec	+15 sec delay	16 sec	+15 sec delay
<70%	# events/day/infant	50	33 ^a	21 ^a	13			12	
	Mean seconds/event	17	24 ^a	37 ^a					
	Seconds/day per infant	828	792 ^a	770 ^a					
<88%	# events/day/infant	511	362 ^a	243 ^a	170			162	
	Mean seconds/event	30	41 ^a	63 ^a					
	Seconds/day per infant	15,195	15,005	15,378					
>95%	# events/day/infant	453	323 ^a	207 ^a	156			132 ^a	
	Mean seconds/event	26	38 ^a	57 ^a					
	Seconds/day per infant	11,943	12,238	11,872					
>98%	# events/day/infant	63	47 ^a	29 ^a	16			14	
	Mean seconds/event	18	25 ^a	40 ^a					
	Seconds/day per infant	1,158	1,165	1,067 ^a					

^aSignificantly different than 2 seconds, $p < 0.01$. sec ave = second average.