

When an Alarm is not an Alarm, the Monitor that Cried Wolf

Rob Graham, R.R.T./N.R.C.P.

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.

“On the cockpit voice recorder, the sound of the takeoff warning system (TOWS) alarm could be heard sounding throughout the takeoff roll. The TOWS alerts pilots that the aircraft is not configured correctly for takeoff.”

On Aug 1, 1999, a Boeing 737-204C passenger jet operated by Líneas Aéreas Privadas Argentinas (LAPA) as flight LAPA 3142 pushed back from the gate at Aeroparque Jorge Newbery in central Buenos Aires. The plane taxied to its assigned runway, and then, after failing to get airborne, the plane crossed a road at the end of the runway. Before coming to rest at a small embankment, the plane crashed through the airport perimeter fence and crossed a road at the end of the runway. In its path was a Dodge Neon with two occupants; both were killed when the plane collided with it. The plane continued to strike a gas regulation plant before stopping at a small embankment. In addition to the two ground fatalities, of the 100 passengers and crew in the plane, 65 were killed, and 17 suffered severe injuries. At the time, it was the second deadliest aviation crash in Argentinian history.

On the cockpit voice recorder, the sound of the takeoff warning system (TOWS) alarm could be heard sounding throughout the takeoff roll. The TOWS alerts pilots that the aircraft is not configured correctly for takeoff. In the case of LAPA flight 3142, the plane could not generate enough lift to lift off because the flaps were fully retracted. Why did the pilots proceed through V1 (the point after which an aircraft cannot abort takeoff) to rotation speed (when the pilot pulls back on the yoke to lift the nose gear off the ground)?

As is the case in most incidents, many factors (of which there were many) culminated in the crash. A “sterile cockpit” (no discussion that is not relevant to the task at hand) was not maintained during pre-flight procedures, causing the pilots to miss setting flaps on the checklist. The airline was known for shoddy maintenance, and lax procedural adherence and nuisance alarms were so commonplace that pilots were accustomed to ignoring them. The pilots of LAPA 3142 ignored the TOWS alarm, thinking it was just another glitch. This situation is a direct consequence of what is known as alarm fatigue.

Alarms in the NICU are plentiful and frequent to the point that, just as in LAPA 3142, they are frequently ignored by those at the bedside. This is ubiquitous and undermines patient safety. For instance, arterial line alarms sound during blood withdrawal; too often, they are silenced before the procedure and not turned back on again. This missing step is a dangerous practice that could easily be avoided were there an option for silencing the alarm long enough to complete the task. As it is, alarm silence is not long enough, and the alarm sounds during a blood draw.

Of all the alarms in the NICU, oxygen saturation (SpO₂) alarms are by far the most frequent and spurious. Motion artifact and/or poor perfusion are often the trigger, but other factors increase the frequency of the alarms and contribute to alarm fatigue, chiefly the high and low limit settings. How do we protect our babies from hypo/hyperoxia without the frequency of alarms sabotaging our efforts?

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2018 saw the publication of a large multi-centre study on high versus low SpO₂ target ranges found that lower targets are associated with increased mortality compared to higher ones. The trade-off is increased incidents of retinopathy of prematurity (ROP) requiring treatment in the high SpO₂ cohort (1). In clinical

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practice this has led to raising low SpO₂ limits, narrowing the target range. Not only is there little evidence to support this change, it may increase the conditions it is meant to reduce (2). (This is an excellent and thought-provoking reference. I highly recommend reading it.)

Tightening the alarm limit range should reduce hyper/hypoxia, but this may not be true in clinical practice. False low SpO₂ alarms may lead to higher FiO₂ and, thus, higher SpO₂ as those at the bedside attempt to reduce the number of alarms (2). While high alarms may be increased simultaneously, these are usually less irritating because they are generally softer and lower in volume. The choice of monitor may also influence the number of false alarms (3) and the incidence of severe ROP (4).

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What, then, are appropriate high and low SpO₂ limits? The determination must consider both the risk to the baby and the risks associated with alarm fatigue. Surprisingly, the risk of hyperoxia in the <33 weeks PMA to ≤36 weeks PMA infant does not occur until a SpO₂ of 97-98%. Even more surprising is that in the ≥36 weeks PMA infant, this occurs at 96%, a level lower than the high alarm settings commonly used in the near term. On the other hand, the risk of hypoxia does not occur until a SpO₂ of 85-86% **independent of PMA** (2).

Low and high SpO₂ for near-term babies is often set at 90-92% and 98-99%, respectively, often for fear of the baby “flipping” into persistent pulmonary hypertension of the newborn (PPHN). This is likely to result in more hyperoxia and likely has little effect on the risk of PPHN. It is worth noting that maintaining higher than necessary SpO₂ in the near term may blunt the effect of inhaled nitric oxide (iNO) should the baby be in or develop PPHN. This is due to increased free radical production, which deactivates vasodilator production and thus promotes vasoconstriction (5).

Animal models (lamb) have shown a similar decrease in pulmonary vascular resistance (PVR) with a FiO₂ of 0.21, 0.50, or 1.0. However, prior exposure to a FiO₂ of 1.0 decreased the effectiveness of iNO. Additionally, resuscitation with FiO₂ increased pulmonary vasoconstriction with norepinephrine (6). This is a significant

finding since vasopressors are often used to manage PPHN.

Reducing nuisance alarms can be achieved in a myriad of ways, the simplest of which is to reduce low alarm limits and/or increase averaging time. Reducing alarm limits reduces the safety buffer before hypoxia occurs, and increasing average time increases the likelihood of missing events. One could argue that a desaturation lasting less than 10 seconds is of little or no physiological consequence, but decreasing alarm limits in conjunction with increased averaging time may miss consequential events.

For example, decreasing alarm limits in an adult ICU from 90% to 88% or 85% reduces alarms by 45% and 75%, respectively; increasing averaging time to 15 seconds at a limit of 90% reduces alarms by 70% (7). In the same setting, combining a limit decrease to 88% and a delay of 15 seconds decreased alarm frequency by a whopping 85% (7).

Combining a slight decrease in lower limit and a delay time of 15 seconds is the most effective action because it reduces alarms drastically while preserving a safety margin. Massimo® does not recommend delays of more than 16 seconds. Adaptive alarm technology currently in development holds great promise. Because it factors in individual patient baseline SpO₂, responding to changes in that baseline may be significant, but that would not be recognised by a regular saturation monitor (7).

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Reducing nuisance alarms and alarm fatigue should be a top priority for clinicians and device manufacturers. Do not let your patients be on flight LAPA 3142.

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