Supplemental oxygen compromises the use of pulse oximetry for detection of apnea and hypoventilation during sedation in simulated pediatric patients.
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Abstract Excerpted:

The goal was to assess the time to recognition of apnea in a simulated pediatric sedation scenario, with and without supplemental oxygen. Methods: A pediatric human patient simulator mannequin was used to simulate apnea in a 6-year-old patient who received sedation for resetting of a fractured leg. Thirty pediatricians participating in a credentialing course for sedation were randomly assigned to 2 groups. Those in group 1 (N = 15) used supplemental oxygen, and those in group 2 (N = 15) did not use supplemental oxygen. A third group (N = 10), consisting of anesthesiology residents (postgraduate years 2 and 3 equivalent), performed the scenario with oxygen supplementation, to ensure validity and reliability of the simulation. The time interval from simulated apnea to bag-mask ventilation was recorded. Oxygen saturation and Paco(2) values were recorded. All recorded variables and measurements were compared between the groups. Results: The time interval for bag-mask ventilation to occur in group 1 (oxygen supplementation) was significantly longer than that in group 2 (without oxygen supplementation) (173 +/- 130 and 83 +/- 42 seconds, respectively). The time interval for bag-mask ventilation to occur was shorter in group 3 (anesthesiology residents) (24 +/- 6 seconds). Paco(2) reached a higher level in group 1 (75 +/- 26 mmHg), compared with groups 2 and 3 (48 +/- 10 and 42 +/- 3 mmHg, respectively). There was no significant difference between the groups in oxygen saturation values at the time of clinical detection of apnea (93 +/- 5%, 88 +/- 5%, and 94 +/- 7%, respectively). Conclusions: Hypoventilation and apnea are detected more quickly when patients undergoing sedation breathe only air. Supplemental oxygen not only does not prevent oxygen desaturation but also delays the recognition of apnea.

Commentary:

This paper is represents a nice use of simulation to prove a point that has been widely known and discussed in the last several years. In short – oxygen therapy causes a longer interval between apnea and oxygen desaturation. The logic follows that this longer interval could result in a longer interval to *recognize* apnea and thus a longer time to respond to apnea (since apnea is not noticed and probably not thought to be as urgent a problem) because of this. The authors do a nice job of describing some of the physiology behind this clinical observation in the Discussion section of this paper. The paper does bring up some obvious points for discussion that need to be appreciated:

1. To accept the conclusions of this study, it must be assumed that performance on a simulator in this setting is representative of performance on actual patients. Although some work in validating simulation has been completed, there is really very little information that would indicate that we can assume that findings on the simulator translate to real life performance in pediatric sedation. If we are depending on

observation of chest wall motion to appreciate apnea, it should be understood that chest wall motion is not perfectly replicated in a simulator – and at times can be somewhat subtle. In addition, we would question whether or not the physiology built into the METI simulator is absolutely representative of pediatric physiology – this has not been proven or validated to our knowledge. So, question number one: Are the results representative of actual practice? We are not sure.

2. There was no End Tidal CO2 (ETCO2) monitoring included in the study. This, in spite of the fact that according to the current AAP Guidelines, ETCO2 should be considered for deep sedation. It is a standard monitor for Anesthesiology providers in the operating room environment and for off-site anesthesia provision. We would hazard a guess that most sedation services in the United States make use of ETCO2 as this monitor is well accepted as a valuable adjunct to sedation monitoring. At this point many handheld ETCO2/SpO2 monitors are available at reasonable cost and continuous O2 sampling can be obtained while using nasal cannula or mask O2. Clearly if ETCO2 is being monitored it should be a considerable lead monitor for apnea over pulse oximetry. While the monitor usually does not provide accurate assessments of PaCO2, it can be a very reliable indicator of apnea. So we must ask, why not include ETCO2 in this study? Is this study relevant for most sedation providers since no ETCO2 monitoring was used? Perhaps a more interesting investigation (or follow up investigation) would ask the question,"is there any difference in time to react to apnea between patients receiving or not receiving O2 when ETCO2 is utilized?" When using ETCO2 a potent argument could be made to add O2 therapy since it would give more time to manage apnea prior to desaturation once it is noticed.
3. Reading the methods section, it appears this study was performed prior to sedation training for the providers involved in the comparison. Once again it would be important to understand whether or not the differences in recognition of apnea would hold up if all of the providers had more familiarity with the simulators and more training in ventilation monitoring.
4. In an era that emphasizes "outcomes", we must ask the question as to whether or not there are any clinical implications of a longer interval to recognize and treat apnea. Is there any harm to this longer interval? Is it a marker of potentially dangerous care? In general we would absolutely agree that (on face value) it is better to recognize apnea sooner rather than later. On the other hand, it is not absolutely the case that just because you can measure a difference in care, it indicates a safety issue or injury to patients. We must point out that the difference in time to notice apnea is highly dependent on the physiology built into the METI simulator, which (as mentioned above) is not validated. In order to answer the question as to whether or not the differences noted in this study have clinical significance we would require large demographic studies involving sedation care that are simply not available. The assumption of this study would be that the difference in time to recognize apnea is important – we feel it is necessary to call the question as to whether this is absolutely the case – or not?

So we would conclude that the authors have helped to prove a point that has been long accepted, but we would point out that there are some remaining questions. Not the least of which would be, "why use a measure of oxygenation to monitor ventilation?"