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## High-Flow Versus Titrated Oxygen



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**Review Of:** Austin MA, Wills KE, Blizzard L, et al. Effect of high-flow oxygen on mortality in chronic obstructive pulmonary disease patients in prehospital setting: Randomised controlled trial. *BMJ*. 2010;34:c5462

### The Science

This is a randomized controlled trial that sought to determine whether there's an association between administering high-flow versus titrated oxygen to patients with an acute onset of exacerbated chronic obstructive pulmonary disease (COPD). A total of 405 patients were initially enrolled with a total of 214 patients being diagnosed with COPD by lung function test within the past five years. The authors of this study report the following findings:

- Overall mortality in high-flow vs. titrated groups: 9% and 4%, respectively
- Titrated oxygen use reduced mortality by 58% when compared to all patients (relative risk 0.42, 95% confidence interval 0.20 to 0.89; 0.02) and by 78% for confirmed COPD patients (0.22, 0.05 to 0.91; P=0.04)
- Patients in the titrated group were less likely to have respiratory acidosis and hypercapnia.

The authors conclude titrated oxygen administration is associated with a reduction in mortality in the treatment of COPD exacerbation.

**Doc Wesley:** Well, if you wait long enough, what you learned 40 years ago comes back around as “new and improved” knowledge. I remember when we cautioned against the use of high-flow oxygen that would suppress the “hypoxic” drive of the COPD patient. I remember the Venturi Masks with their little colored adaptors for selecting oxygen concentration. But then oximeters came along, and it was soon evident that the COPD patient only needed a “wiff” of oxygen to get their saturations up to 100%. Yet, we continued to use high-flow oxygen with the comfort of knowing if they stopped breathing, we could ventilate them with a BVM or intubate them.

Research clearly showed that high-flow oxygen caused respiratory acidosis, which resulted in systemic acidosis. Additional research has shown that perhaps a little hypoxia is a good thing because it improves oxygen release in the capillary bed. Further studies have shown that high concentrations of oxygen in the body (hyperoxemia) results in poor neurologic outcome for cardiac arrest survivors and worsening ischemia for stroke and heart attack patients.

The authors clearly believe in the recommendations of the British Thoracic Society, which set prehospital protocol for most of the United Kingdom. The 2010 American Heart Association (AHA) guidelines echo these recommendation, stating the oxygen should only be given in the presence of documented hypoxia or server respiratory distress and then only to attain an O<sub>2</sub> saturation of 92–94% in patients with chest pain and status post cardiac arrest. They don't address the COPD patient, I assume, because these patients will be in distress or hypoxic.

So what do we do with this study? Does it support the recommendations? I'm not sure. There are several problems with the design and analysis of the author's research. First, what's the eligibility criteria for entrance into the study? Suspected COPD exacerbation. But how severe? We're not provided any information on initial work of breathing or aggressiveness of care (number of nebulizers, steroid administration, etc). Although there was no difference in initial oxygen saturation.

Secondly, there are problems with the number and math performed on their results. They reported a significant difference in mortality with 9% of high flow and 4% of titrated oxygen patients dying. But let's look more closely at that figure. They needed 100 patients in each arm to have an 83% probability of detecting a 70% difference in mortality. They choose 70% based on other studies that showed this difference. They had 97 in the titrated group but only 47 left after excluding those without confirmed COPD. They report a difference in mortality of 9% and 2% for the COPD group. Neither of these numbers can be considered statistically significant because you can only be about 70% sure the overall mortality and 50% sure that the COPD differences are real. That's no better than a coin toss.

Another issue with mortality is that 70% occurred up to five days after admission. Do the authors seriously intend to imply that 45 minutes of prehospital high-flow oxygen is the cause of these deaths? It's hard to fathom. What happened to these patients after they were admitted? Were they maintained on high-flow oxygen or titrated? I suspect that they were all titrated, which should cause them to acquire the survival benefit of this modality (if it exists) soon after admission.

Many studies have shown that the mortality rate from COPD exacerbation treated by EMS is as high as 25%. Why was their mortality rate so much lower? I suspect it's due to the fact that

the patients weren't as sick. This leads to a significant reduction in the treatment effect.

Finally, they state in their conclusion that the high-flow oxygen group had more respiratory acidosis and hypercarbia than the titrated group; however, looking at their data, less than half the patients had blood gases performed, and for those, there was no statistical difference in pH or partial pressure of carbon dioxide.

Now that I've totally confused you with statistical mumbo jumbo, you might be wondering, "what is the take-home message?" I'm in favor of treating the respiratory distress patient with titrated oxygen only if capnography is used. If the carbon dioxide is high and/or rising, then it makes sense to titrate the oxygen based on oxymetry. In the absence of capnography, I don't see any reason to have my first responders and basic EMTs titrate oxygen.

**Medic Marshall:** So the Doc has pretty much hit this one on the head—this is really questionable research and doesn't meet the general in clinical research, which is a p value of .05 or more. Simply put, there is only a 5 % probability the results occurred by chance alone.

The one thing that I asked myself when I read this study was, "Is the amount of time we spend giving these patients high-flow oxygen really the contributing factor to these patients' deaths?" Let's just say—for sake of argument—that in rural Tasmania, it's an average transport time of 45 minutes to the hospital. Well, factoring in on-scene time and transport time, your patient could be on high-flow oxygen for an hour to an hour and fifteen minutes. So for up to an hour and fifteen minutes, these patients were on high-flow oxygen ... and that made the difference in whether they were likely to live?

Like the Doc pointed out, the majority of the patients ended up dying five days later. I'm not sure I can accept the authors' conclusion because it would be difficult to conclude any type of association or mortality with pre-hospital administration of oxygen.

Another thing to mention is there were a total of 75 patients (out the 214) that weren't included in the analysis (54 or 56% in the titrated arm and 25 or 21% in the high-flow arm). These patients were excluded because their treatments followed the treatment protocol. So what were the reasons why the protocol wasn't followed or deviated? I have no idea; the authors don't report it.

At the end of the day, is this going to change the way we do things? I don't think so. Should it make us, as prehospital providers, start to think about what we do and how treat patients and how it may affect their lives? Absolutely.

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