Editorials

Who is for supplemental oxygen?

Cardiopulmonary complications account for over 50% of the serious adverse events associated with upper gastrointestinal endoscopy.[1,2] Many of these adverse events may relate to use of intravenous drugs[3,4] and one of the aims of both the American[5] and British[2] Working Party Reports on safety and monitoring during gastrointestinal endoscopy was to make recommendations that might improve patient safety by minimizing the risk of adverse cardiopulmonary events.

Two Australian papers[6,7] on the use of supplemental oxygen at the time of endoscopy appear in this issue. It is my purpose to discuss (1) the different oxygen delivery systems available, (2) the worries many endoscopists have regarding hypercapnia as a consequence of oxygen supplementation, and (3) the present two studies[6,7] in the light of other relevant studies on the same topic. I do not intend to review the now extensive literature relating to the occurrence of oxygen desaturation at the time of both upper and lower gastrointestinal endoscopy nor the merits and drawbacks to pulse oximetry, since well-referenced articles on these subjects are available elsewhere.[1,8]

Oxygen delivery systems may be roughly divided into those of variable and fixed performance. Variable performance devices include nasal prongs, nasal cannulas, some face masks, and, particularly relevant to the articles in this issue, low-flow oxygen delivered via the mouth or an oxygenating mouthguard. In all such devices, the actual oxygen concentration breathed in (FiO_2) depends on the rate and depth of respiration: the faster the respiratory rate and the greater the tidal volume, the lower the actual inspiratory oxygen concentration achieved[9]; furthermore, with variable performance systems undesirably high FiO_2 concentrations may be produced in patients with acute or chronic respiratory failure.[10] Few studies have measured the actual FiO_2 obtained with low-flow variable performance systems. In one study[11] the mean oropharyngeal oxygen concentrations when mouth breathing and using nasal catheters were 32.2% and 44.1%, respectively, at flow rates of 2 and 4 liters/min. Similar results were obtained in a second study[12] with both reports nicely demonstrating the marked intersubject variability in the measured FiO_2.[11,12]

Fixed performance devices[13–15] are designed to give a specific oxygen and air mixture to the patient and to be unaffected by the varying respiratory rates and volumes encountered in clinical practice. Such masks incorporate the venturi entrainment principle originally described as high air flow oxygen enrichment.[14]

One of the best known are the cone-shaped Ventimasks which have been established fixed performance devices since 1966. These masks are designed to produce an inhaled oxygen concentration of 24, 28, 35%, or higher. As discussed below, it is now accepted practice that tight control over inspired oxygen concentration is necessary in ventilatory failure, particularly with regard to keeping inspired oxygen concentration (FiO_2) below about 35%.[13] It is equally accepted practice that such tight control is unneccessary in nearly all other clinical situations.[13]

The dangers of hypercapnia occurring as a consequence of oxygen administration to patients with acute exacerbations of chronic obstructive airways disease (COAD) are well recognized. In fact, so ingrained is this fear of inducing hypercapnia that in my experience many endoscopists appear reluctant to give oxygen to any patient with a hint of a chest problem of any kind. This is illogical since even patients with quite advanced chronic stable respiratory failure secondary to COAD can safely be given low-flow supplemental oxygen via nasal cannulas for 16 or more hours per day totally unsupervised in their own homes.[16,17] As originally suggested many years ago, the acute rise in arterial carbon dioxide tension ($PaCO_2$) during oxygen therapy is still thought[18] to be mainly due to a fall in hypoxic drive (due to the induced rise in PaO_2), an acquired insensitivity to carbon dioxide and consequent hypoventilation. It was this theory which led Campbell in 1960[19] to develop fixed performance devices to ensure that controlled oxygen therapy could be given to hypoxic patients with COAD at a rate which would produce a modest rise in arterial oxygen tension (PaO_2), sufficient to significantly increase arterial oxygen saturation (SaO_2), but not to seriously depress the patient's all important ventilatory drive.

So what of our two articles? The first,[6] entitled Supplemental Low Flow Oxygen Prevents Hypoxia During Endoscopic Cholangiopancreatography concerns 50 consecutive patients subjected to ERCP. All patients had continuous monitoring of their oxygen saturation using a pulse oximeter attached to an index finger. The patients were sedated with a benzodiazepine/opioid combination of midazolam and fentanyl. The patients were randomly allocated to no oxygen (group 1), oxygen via nasal prongs (group 2), or oxygen via a nasopharyngeal cannula (group 3). The oxygen flow rate of 2 liters/min chosen by the authors[6] was the same as our own group had previously used in 1987[20] but much less than the 4.5 liters/min employed in a subsequent study from Hong Kong.[21] This low oxygen flow rate raised the baseline oxygen saturation by a mean of 2.8 and 2.9%, respectively ($p < 0.01$) in groups 2 and 3. Following intubation, where no oxygen was given, the oxygen saturation in group 1 reduced by a mean of 6.8% ($p < 0.001$). Hypoxia (defined as an oxygen saturation of <90%) occurred in 47% of patients not receiving oxygen, and no less than 5 of 20 in this group were given oxygen because their saturation fell to below 84%. In contrast, in groups 2 and 3 patients who received supplemental oxygen during the ERCP, significant increases in saturation from baseline of 2.3% and 1.3%, respectively, were observed and no patient became hypoxic.

The results[1] are very similar to the Hong Kong study[21] in which 51.3% of control patients sedated with a diazepam and pethidine (meperidine) combination prior to ERCP had decreases in oxygen saturation to below 90% for more than 60 sec compared with only a 9.8% incidence of a similar degree of oxygen desaturation in those given 4.5 liters of intranasal oxygen per minute. Both groups of authors[6,21] conclude that their studies support providing low-flow supplementary oxygen during ERCP as an "effective, cheap and simple way of minimizing desaturation." Oxygen given via nasal prongs at a rate of much above 4 liters/min is uncomfortable, and if 2 liters are effective in most cases (and that is certainly our own experience), then we would recommend the lower flow rate as being (1) more comfortable to the patient, (2) more economical, and (3) safer in those with chronic obstructive airways disease. The most profound drop in oxygen saturation usually occurs at or shortly after intubation of the esophagus, especially when the examination is carried out by inexperienced endoscopists.[22] We recommend that patients be pre-oxygenated for a couple of minutes prior to administration of the intravenous sedative, since predictably if the supplemental oxygen is not turned on until the endoscopist starts to pass the instrument, the patient has often already begun to desaturate.[23]

In the United Kingdom it costs less than 5 pence to administer 2 liters of oxygen for 15 min, but the disposable nasal cannulas used to deliver it to the patient cost approximately 95 pence each. Thus, 95% of the cost of supplemental oxygen is the delivery system and only 5% the oxygen itself.[23] We used a dual nasal and oral thermistor technique to demonstrate that the vast majority of patients start to breath through the mouth rather than the nose as soon as an endoscope is passed into the upper esophagus, and, furthermore, this breathing pattern persists until the instrument is removed.[24] The study thus indicated that supplemental oxygen given during upper gastrointestinal endoscopy might be delivered via a modified mouthpiece rather than nasal cannulas which would make it more efficient and considerably cheaper.[24] This has now been followed up by a formal prospective randomized study comparing the efficacy of continuous supplemental oxygen given at 2 liters/min via the nasal or oral route.[23]

I read the second article, entitled Oxygenating Mouthguard Alleviates Hypoxia during Gastroscopy

with special interest because this particular mouthguard is shortly to become commercially available in the United Kingdom (Borody, personal communication). As illustrated in their article,[7] the oxygenating mouthguard looks like a standard plastic endoscopy mouthpiece in terms of its size and shape except it has a side arm to which oxygen tubing can be attached. The oxygen enters the mouthpiece from the side arm via two tunnels within the mouthpiece from whence it passes simultaneously either posteriorly into the oral cavity or upwards toward the patient's nostrils.

In the oxygenating mouthguard study,[7] all patients were given an intravenous injection of a benzodiazepine/opioid combination and had their oxygen saturation monitored using a pulse oximeter. The patients were randomized to either continue simply breathing room air or receive supplemental oxygen (3 liters/min) via the mouthguard. In 25% of patients breathing room air, significant oxygen desaturation occurred compared with only 3% of those given supplemental oxygen via the mouthguard. Although administering oxygen via the mouthguard greatly alleviated hypoxemia during gastroscopy, it did not totally prevent it, and consequently the authors recommended pulse oximetry as well as supplemental oxygen. I totally agree with this conclusion, but suggest, as discussed above, that the authors would achieve significantly better results by pre-oxygenating their patients for 1 or 2 min prior to administering the intravenous sedative.[23]

I would predict that oxygenating mouthguards will become standard for upper gastrointestinal endoscopy since, as the authors state,[7] they reduce the complexity of the equipment attached to the patient, are cheaper than disposible nasal cannulas, and, more important, mean that inserting an oxygenating device becomes an automatic part of the procedure. In all but those with advanced COAD who are in the throws of an acute exacerbation,[10] 2 liters of supplemental oxygen per minute can be safely given via variable performance devices such as nasal cannulas or oxygenating mouthguards. It is my personal view that all patients undergoing endoscopic procedures under intravenous sedation should not only be closely monitored clinically but also be fitted with a pulse oximeter. With very few exceptions a case can also be made for giving these patients supplemental oxygen. Oxygen is one of most abundant elements on earth. I cannot believe that the "Great Endoscopist in the sky" did not wish us all to make a little more use of it!

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