

# The Risks of Acute Oxygen Therapy

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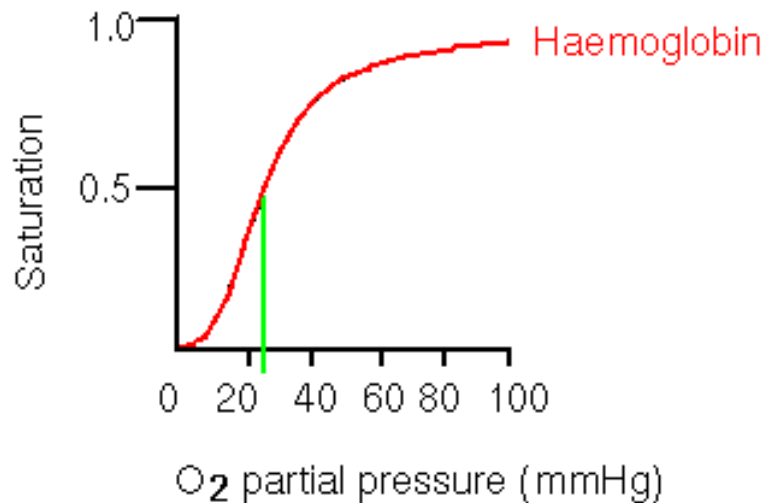
# Introduction

- Type 2 respiratory failure (low pO<sub>2</sub> and raised pCO<sub>2</sub>) was first recognised in 1949, and occurs in 10-15% of COPD patients.
- A large number of these patients will develop acidosis with oxygen administration

# Introduction ctd.

- Oxygen therapy is very beneficial in acute respiratory failure, and helps to stabilise their condition
- Excessive administration of oxygen is the most common error of oxygen prescription in hospitals
- The need for controlled oxygen therapy is often not recognised in hospital guidelines
- We need to try and limit hyperoxia, hypercarbia and acidosis, while avoiding inadequately correcting hypoxia

# Oxyhaemoglobin Dissociation Curve



- Health professionals are often concerned about allowing patients to get close to the “slippery slope” of the curve

# Risks of High Flow Oxygen

- Hyperoxia can lead to:
  - Respiratory acidosis
  - Increased systemic vascular resistance
  - Increased blood pressure
  - Decreased cardiac output
  - Reduced coronary, cerebral and renal blood flow
  - Atelectasis and increased airways inflammation
  - Increased intrapulmonary shunting
  - Production of toxic reactive oxygen species

# Risks of High Flow Oxygen ctd

- Hyperoxia can lead to a paradoxical decreased in oxygen consumption. This is thought to be due to maldistribution of blood flow and functional shunting, to protect vital organs from effects of toxic reactive oxygen species
- Hyperoxia in COPD is associated with increased mortality and morbidity, and is a good indicator of ICU, and increased complications during admission

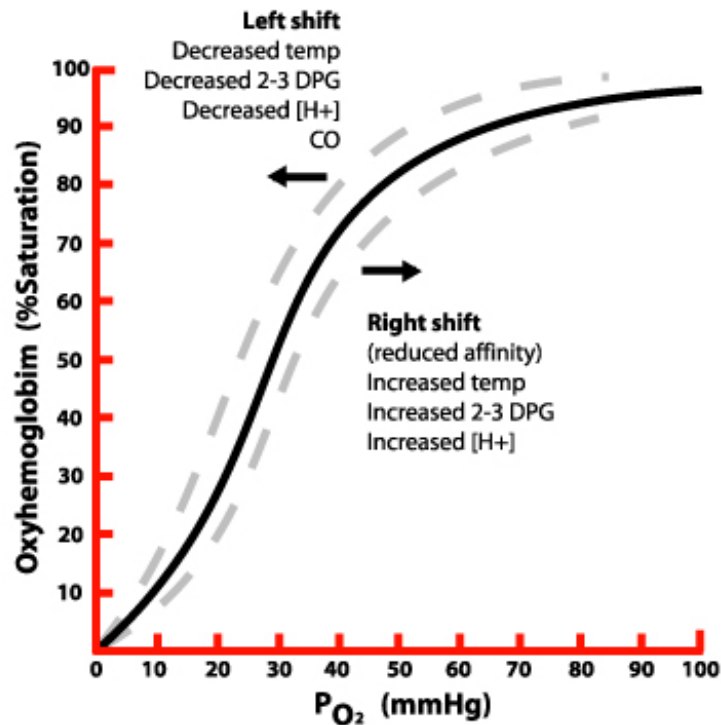
# Hypercapnia

- There are several mechanisms thought to be responsible for increased O<sub>2</sub> leading to hypercapnia in susceptible individuals:
  - Decreased hypoxic drive
  - Decreased minute ventilation
  - Increased ventilation perfusion mismatch

# Hypercapnia ctd

- Hypercapnia can lead to depression of the neurological and cardiorespiratory function
- These effects occur slower than effects of hypoxia, but can last for hours or days, and can be fatal
- A resultant falling pH can eventually be fatal
- Chronic hypercapnia can occur, and is related to severity of respiratory failure, however the highest PaCO<sub>2</sub> levels are seen after oxygen therapy

# Hypercapnia ctd



- Reduction in pH leads to rightward shift in the dissociation curve, and therefore a reduced oxygen saturation at a given PaO<sub>2</sub>

# Evidence (retrospective and prospective audits)

- Durrington et al (2005)
  - high concentration oxygen caused significant and inappropriately high PaO<sub>2</sub> and PaCO<sub>2</sub> compared to initial low concentration oxygen
  - 25% of patients receiving low inspired O<sub>2</sub> throughout admission had complications
  - 41% of those exposed to high O<sub>2</sub> in the ambulance and converted to low concentration in ED had complications (particularly when the ambulance journey exceeded 30mins)
  - 65% of those receiving high inspired O<sub>2</sub> throughout had complications

# Evidence ctd.

- Plant, Owen & Elliott (2000)
  - a significant negative correlation between pH and PaO<sub>2</sub>. The more oxygenated patients became, the greater the magnitude of respiratory acidosis
  - 47% were hypercapnic, 20% of these were acidotic if PaO<sub>2</sub> was >75mmHg
  - It is not the absolute level of PaCO<sub>2</sub> that is important, but the magnitude and speed of any change, which is reflected by pH
  - Acidosis is associated with increased ICU use, but not with mortality (however, this may be confounded by the use of NIV)

# Evidence ctd

- Joosten et al (2007)
  - The use of oxygen flow rates of greater than 4L/min in patients with high CO<sub>2</sub> levels was associated with a significant increase in length of stay, an increased need for NIV, and a higher admission rate to the HDU
- Soo Hoo, Hakimian & Santiago (2000)
  - Increasing severity of respiratory acidosis was associated with an increased likelihood of intubation and mortality

# Evidence ctd

- Denniston, O'Brien & Stableforth (2000)
  - In-hospital mortality was 14% for patients administered an O<sub>2</sub> concentration > 28% and 2% for those administered a lesser concentration. These groups were comparable in relation to triage category, age, and smoking history
  - In-hospital mortality was greatest in the group with severe acidosis (29%), followed by the group with mild acidosis (11%), those with unknown acid-base status (6%) and the non-acidotic group (4%)

# Recognition of Deterioration

- One of the least recognised risks of routine high flow oxygen is a delay in recognition of deterioration
- Contrary to common assumption that high flow oxygen will have a protective effect in the event that pulmonary function deteriorates

# Is Low Flow O<sub>2</sub> Safe?

- There is varying opinions on what constitutes a 'safe' level of hypoxaemia. There is a reasonable consensus around a pO<sub>2</sub> of 50mmHg, leading to recommendations of sats between 85-92%
- Some patients will acclimatise to significantly lower pO<sub>2</sub>, due to an increase in minute ventilation, and secondary polycythaemia

## ctd

- There is some evidence that pO<sub>2</sub> of >75mmHg is associated with acidosis in 33-50% of hypercapnic COPD patients
- “O<sub>2</sub> sats of 85% is of concern because it indicates severe underlying disorder, not because of inadequate oxygenation. The crucial therapeutic response is to recognise the severity of the underlying disorder and institute the required aggressive therapy, in addition to appropriate O<sub>2</sub> therapy” (Beasley, McNaughton & Robinson, 2006)

# Evidence

- Murphy, Driscoll & O'Driscoll (2001)
  - 13 of 15 patients with COPD developed a pO<sub>2</sub> less than 50mmHg when they undertook light exercise in simulated aircraft cabin conditions
  - All of these patients had pO<sub>2</sub> >70mmHg when breathing room air at sea level
  - All the patients were asymptomatic

# Evidence ctd

- NICE guidelines 2004, citing Agusti et al
  - Oxygen was given to 18 patients with COPD with acute respiratory failure, within 48 hours of admission
  - Oxygen was given 2-4L/min via NP, or 24-28% via Venturi mask
  - This raised the O<sub>2</sub> sats to >90% immediately in all cases

# Evidence ctd

- Plant, Owen & Elliot (2000)
  - “The data suggest that acidosis is minimised in the pO<sub>2</sub> range 55-75mmHg, or sats 85-92%”

# Other Thoughts

- When confronted with a dyspnoeic, cyanotic patient, it is difficult to refrain from administering high flow oxygen
- There is a persistent view that COPD patients with an adequate resp rate cannot be harmed with high flow oxygen (ie, only concerned about hypoxic drive, not acidosis, or hyperoxia)
- One opinion is that inadequate O<sub>2</sub> accounts for more deaths and disability than can be justified by relatively small risks associated with high flow oxygen

## ctd

- One author suggests that several studies conclude that COPD patients are difficult to oxygenate, and require higher flows of oxygenate to achieve adequate pO<sub>2</sub>. This is not supported by other data (and the opposing view is often stated)

# ctd

- McCrory et al (2001)
  - “based on 4 observational studies, we concluded that O2 administration may lead to hypercarbia, but that at risk patients can be identified”
    - (should be noted that all 4 studies used fixed concentration O2, or started with low flow O2, and titrated up)

# ctd

- Gomersall et al (2002)
  - “From our study, not only is there no deleterious effect of outcome from correction of hypoxaemia, there was a slight trend toward improvement. The dangers of correction of hypoxaemia in these patients may be overstated”
    - NB: respiratory stimulants (eg doxapram) were used where indicated in this study. Study looked at loss of hypoxic drive, not at acidosis and hyperoxia. They didn't define what 'correction of hypoxaemia' was

## ctd

- Soo Hoo, Hakimian & Santiago (2000)
  - “our study found there was no difference in immediate mortality rates, but there was an increased 1-year mortality rate for the hypercapnic group. There was no analysis based on the initial level of acidosis”
    - NB: ?increased 1-year mortality rate due to disease severity

# Patient Assessment

- Detection of hypercapnia and acidosis is difficult without ABGs, as signs are similar to that of hypoxia
  - Headache, depression, drowsiness, combativeness, reduced level of consciousness
- Ideally, ABGs should be measured before, and one hour after starting O<sub>2</sub> therapy
- Pulse oximetry is non-invasive, and useful for trending and adjusting oxygen therapy settings

# Guidelines

- GOLD Guidelines (2006)
  - Supplemental oxygen should be titrated to improve the patients hypoxaemia
  - Adequate levels of oxygenation ( $pO_2 > 60\text{mmHg}$  or  $SpO_2 > 60\text{mmHg}$ ) are usually easy to achieve
  - $CO_2$  retention can occur insidiously with little change in symptoms
  - ABG should be checked 30-60min after  $O_2$  is commenced

# Guidelines ctd

- COPD-X Guidelines (2006)
  - Controlled oxygen therapy is indicated in patients with hypoxia, aiming for  $SpO_2 > 90\%$  ( $pO_2 > 50\text{mmHg}$ ). There is no point aiming for  $SpO_2 > 92\%$
  - Nasal prongs at 0.5-2L/min or a 24% or 28% Venturi mask is usually sufficient
  - Minimise excessive oxygenation, which can worsen hypercapnia
  - Careful monitoring with pulse oximetry or ABGs

# Guidelines ctd

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- BTS Guidelines (2004)
  - Initial oxygen management should be 24%-28%  
Venturi masks or 2L/min via nasal prongs
  - Measure ABG after 60min

# Guidelines ctd

- BTS Guidelines (2008) Emergency Oxygen Therapy
  - Aim for sats of 88-92% for patients at risk of hypercapnia (COPD patients, and some other conditions), using 24% or 28% Venturi masks
  - Pulse oximetry must be available in all locations where emergency oxygen is used

# Guidelines ctd

- NICE Guidelines (2004)
  - Oxygen therapy should be commenced at 40% during transfer, titrated upwards if sats fall below 90% and downwards if sats exceed 93-94% or if patient becomes drowsy
  - ABGs should be measured on arrival at ED

# Recommendations from Research

- There is a strong recognition that further research is required, with randomised, placebo-controlled trials
- One author suggests that patients could ethically be randomised to 28% or 40% initial inspired oxygen

# Recommendations ctd

- Oxygen alert cards can be used with patients, with or without a provided Venturi mask
- This may improve correct diagnosis by ambulance crew
- Some studies have shown very poor use of the cards.
- This may be improved with provision of Venturi masks

# The driver for change

- Examples from clinical practice
- Two separate events within one week
- Were the stimulus for us to find ways to improving practice and minimise the impact of oxygen therapy to an at risk group

# NZ Audit of oxygen therapy in AECOPD

- This audit was completed in 2006 – 2007
- Majority of patients received high flow oxygen
- About half had documented respiratory failure
- About one quarter had previous invasive or non-invasive ventilation
- 75% had oxygen saturations above the target range 88-92%
- Increased oxygen flow rate were associated with poor clinical outcomes

(Wijesinghe, M et al, 2009. Oxygen therapy in Acute Exacerbation of COPD. Unpublished)

# Additional factors to consider

- Only 35% of patients self identified themselves as having COPD/emphysema
- Ambulance crew correctly identified COPD in 32% of cases (Denniston et al, 2002)
- Ambulance are often the first medical contact for patients
- Ambulance crews currently don't have access to hospital medical records
- Patients perception

# Benefits of Oxygen Alert Cards

- Provides an alert to those giving emergency oxygen therapy
- Enables ambulance crew to identify at risk patients
- Educating patients about oxygen therapy
- Shown to improve outcomes
- Follows guidelines for emergency oxygen therapy in AECOPD

# Steps

- Discussed our ideas with ambulance service, physicians, emergency departments and other hospital in our region
- Designed the card
- Determined how to select patients
- Decided on how to distribute cards to at risk patients
- Decided on whether or not to issue nasal prongs/mask with the card
- Advised limiting nebulisation on oxygen driven nebulisers to 6l/6min
- Planned an audit

## COPD OXYGEN ALERT CARD

NAME:

NHI:

I have COPD & at risk of a raised CO<sub>2</sub> level.

**AIM FOR OXYGEN SATURATIONS 88-92%**

Limit initial O<sub>2</sub> driven nebuliser 6litres/6minutes

(NB: this is a guide and does not replace clinical judgement)



**Capital & Coast**  
**District Health Board**  
ŪPOKO KI TE URU HAUORA



## **COPD OXYGEN ALERT CARD**

Carry this card with you at all times.  
Show it to the ambulance or emergency  
department

# Conclusion

- There is a need for controlled oxygen therapy
- Use of high flow oxygen therapy in emergency oxygen therapy is associated with poor outcomes
- The BTS guidelines for emergency oxygen use recommend the use of alert cards for COPD patients
- Alert card will help identify those at risk
- Help to improve the way we administer emergency oxygen to this vulnerable group

# References

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