Woes to the O’s?
Do We Really Need to Use Supplemental O$_2$?

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Questions to Ponder

- Do we really need to administer supplemental oxygen?
- Are there medical conditions where supplemental oxygen is harmful?
- Are there traumatic conditions where supplemental oxygen is harmful?
Oxygen-Hemoglobin Dissociation Curve
Myocardial Ischemia and Infarction
Oxygen relieved pain during episodes of angina pectoris
  - Steele C. Severe angina pectoris relieved by oxygen inhalations. BMJ 1900, 2:1568.

Hypoxia of the myocardium described as the cause of angina
100% oxygen either pronounced or prolonged electrocardiographic changes
Failed to prevent the onset of anginal pain
Failed to influence the duration of pain

Breathing high concentrations of oxygen for at least 30 minutes in the first 24 hours after MI
- Decreased heart rate
- Reduced cardiac output
- Increased systemic vascular resistance

High concentration – 85% to 90%

Administration of 40% oxygen for 20 minutes

- 17% decrease in cardiac output
- 5% increase in arterial blood pressure

90% oxygen for one hour
- Reduced stroke volume
- Reduce cardiac output
- Increased arterial pressure
- Increased systemic vascular resistance

Normal subjects
- Hypoxia does not affect the availability of oxygen for myocardial metabolism until arterial oxygen saturation falls to as low as 50%

Patients with coronary artery disease
- Myocardial ischemia is not observed until oxygen saturation fell below 85%
- Hyperoxia did not improve myocardial oxygen availability
- Subset of patients with severe triple-vessel disease
  - 6 minutes of high-flow oxygen reduced coronary blood flow sufficiently to induce myocardial ischemia


Breathing 100% oxygen for 10 minutes

- Increases vascular resistance in the left anterior descending artery by 23%
  - Diameter of the large conduit coronary arteries was not appreciably affected
  - Suggests vasoconstriction at the level of the myocardial microcirculation

<table>
<thead>
<tr>
<th>First Author</th>
<th>Changes in Coronary Blood Flow (%)</th>
<th>Changes in Coronary Vascular Resistance (%)</th>
<th>Changes in Myocardial Oxygen Consumption (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ganz</td>
<td>-17.1</td>
<td>25</td>
<td>-15.3</td>
</tr>
<tr>
<td>Ganz</td>
<td>-8.6</td>
<td>21.5</td>
<td>-16.1</td>
</tr>
<tr>
<td>Mak</td>
<td>-7.9</td>
<td>Not reported</td>
<td>Not reported</td>
</tr>
<tr>
<td>McNulty</td>
<td>-28.9</td>
<td>40.9</td>
<td>-26.9</td>
</tr>
<tr>
<td>McNulty</td>
<td>-19.8</td>
<td>22.2</td>
<td>Not reported</td>
</tr>
</tbody>
</table>

Analysis 1.1. Comparison 1 Oxygen versus air, Outcome 1 Death in hospital for participants with acute MI.

Review: Oxygen therapy for acute myocardial infarction

Comparison: 1 Oxygen versus air

Outcome: 1 Death in hospital for participants with acute MI

<table>
<thead>
<tr>
<th>Study or subgroup</th>
<th>Experimental</th>
<th>Control</th>
<th>Risk Ratio</th>
<th>Weight</th>
<th>Risk Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n/N</td>
<td>n/N</td>
<td>M-H,Fixed,95% CI</td>
<td></td>
<td>M-H,Fixed,95% CI</td>
</tr>
<tr>
<td>Rawles 1976</td>
<td>9/80</td>
<td>3/77</td>
<td></td>
<td>55.8 %</td>
<td>2.89 [ 0.81, 10.27 ]</td>
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<tr>
<td>Ukholkina 2005</td>
<td>1/58</td>
<td>0/79</td>
<td></td>
<td>7.7 %</td>
<td>4.07 [ 0.17, 98.10 ]</td>
</tr>
<tr>
<td>Ranchord 2012</td>
<td>1/68</td>
<td>2/68</td>
<td></td>
<td>36.5 %</td>
<td>0.50 [ 0.05, 5.39 ]</td>
</tr>
<tr>
<td><strong>Total (95% CI)</strong></td>
<td><strong>206</strong></td>
<td><strong>224</strong></td>
<td></td>
<td><strong>100.0 %</strong></td>
<td><strong>2.11 [ 0.78, 5.68 ]</strong></td>
</tr>
</tbody>
</table>

Total events: 11 (Experimental), 5 (Control)

Heterogeneity: Chi² = 1.81, df = 2 (P = 0.40); I² =0.0%

Test for overall effect: Z = 1.48 (P = 0.14)

Test for subgroup differences: Not applicable
Respiratory Processes and Oxygen
Patients with stable congestive heart failure

- Administration of 100% oxygen for 20 minutes
  - Cardiac output decreased by 16%
  - Stroke volume decreased by 16%
  - Systemic vascular resistance increased

• Titrated oxygen treatment in a pre-hospital setting
• Target SpO₂ of 88% - 92%
• Used compressed air to nebulize bronchodilators

### Mortality

<table>
<thead>
<tr>
<th>Presumed Acute COPD Exacerbation</th>
<th>RR 0.42 (95% CI 0.20-0.89)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confirmed COPD</td>
<td>RR 0.22 (95% CI 0.05-0.91)</td>
</tr>
</tbody>
</table>

### Adverse Outcomes

• Hyperoxia OR 9.1 (95 CI 4.08 – 20.6) – SpO₂ > 96%
• Hypoxia OR 2.16 (95CI 1.11 – 4.20) – SpO₂ < 88%


SpO2 < 92% associated with greater mortality/morbidity at 30 days.

High inspired oxygen leads to hypercapnia
Should be provided only in the face of hypoxia

Stroke
Healthy volunteers

Administration of 100% oxygen for 10 to 15 minutes
  - 20% to 33% decrease in cerebral blood flow
  - Independent of arterial partial pressure of carbon dioxide


No benefit in clinical performance scores or outcome

Lower survival at 1 year in non-hypoxic patients with mild to moderate strokes
  - OR 0.45 (95% CI 0.23-0.90) if supplemental oxygen administered
  - Nasal cannula for first 24 hours versus room air

Severe strokes and consequent hypoxemia trend towards increased mortality.


Hyperoxia independently associated with higher mortality than normoxia
  - OR 1.8 (95% CI 1.5 – 2.2)

PaO$_2$ increase if 25 mm is associated with a 6% increase in the RR if death

PaO$_2$ increase of 100 mm is associated with a 24% increase in RR of death


Medical Illnesses and Oxygen
Sepsis

SIRS
Temp. >38°C or <36°C, HR >90, RR >20 or PaCO₂ <32, WBCs >12,000 or <4,000 or >10% bands

Sepsis
SIRS + Infection

Severe Sepsis
Sepsis + End Organ Damage

Septic Shock
Severe Sepsis + Hypotension
Hyperoxia may impair oxygen deliver in sepsis

Hyperoxia decreases whole-body oxygen consumption in critically ill patients

Surviving Sepsis Campaign Guidelines
- Peripheral oxygen saturation should be maintained between 88% and 95% in patients with ARDS (Does not advocate hyperoxia)


Oxygen therapy

- Has not been shown to affect the duration of pain crisis
- Has not been shown to be useful in patients with acute chest syndrome with normoxemia

Oxygen should be administered only if hypoxemia is present


Hemorrhagic Shock
**Affects of Increasing FiO₂**

- Does not affect survival
- Compromises hemodynamics
  - Increased MAP
  - Decrease aortic blood flow

The Oxygen Cascade

![Graph showing the oxygen cascade with two lines: one for breathing 100% oxygen and another for breathing air.](image)

- **pO₂, kPa**
  - Gas type inhaled
  - Lung
  - Artery
  - Capillary
  - Extracellularly
  - Mitochondria
  - Venous

**Breathing 100% oxygen**

**Breathing air**
MI – Avoid hyperoxemia
CHF – Avoid hyperoxemia
COPD – Avoid hyperoxemia (SpO₂ 88% - 92%)
Pneumonia – Avoid hypoxemia
Asthma – Avoid hyperoxemia
Stroke – Avoid hyperoxemia
Severe stroke – Avoid hypoxemia
CPR – Avoid hyperoxemia
Septic Shock – Avoid hyperoxemia
Sickle Cell Crisis – No help unless hypoxemia present
Hemorrhagic Shock – Avoid hypoxemia and hyperoxemia