Oxygen (O2) Supplementation Use During Acute Coronary Syndrome (ACS)

PICOTS:
- **P** = Adults with suspected acute coronary syndrome (ACS) with a normal pulse ox ≥ 90%
- **I** = Supplemental oxygen by nasal cannula or facemask
- **C** = No supplemental oxygen
- **O** = reduction in mortality, progression of myocardial infarction, angina, and/or CV related complications
- **T** = Acute setting (within 12 to 24 hours and up to 30 days)
- **S** = Pre-hospital care or emergency department

**Note:** PICOTS stands for (P) for patient, (I) for intervention, (C) for comparison, (O) for outcome(s) of interest, (T) for timing, and (S) for setting.

**Summary:**
Patients with acute coronary syndrome (ACS; unstable angina, NSTEMI, STEMI) should initially receive 2-4 L/min supplemental oxygen (O2) per nasal cannula if they have an oxygen saturation by a pulse oximetry < 90%, are experiencing dyspnea, or have heart failure, as there is conflicting evidence about possible harm in normoxic patients.

- A Cochrane review of 4 trials in patients with acute MI is concerning for a possible greater risk of death in patients getting supplemental O2. However, there is conflicting evidence, and thus the use of supplemental oxygen should be studied in a clinical trial to verify its effect on morbidity and mortality. The current AHA guidelines both recommend its use in the above situations.
- There is some evidence in patients with stable CAD undergoing elective cardiac cath that supplemental O2 (breathing 100% FiO2 at 15 L/min via face mask) may increase coronary vascular resistance, reduce coronary blood flow, and increase mortality risk.
- Could also reduce respiratory drive in patients with known COPD or chronic hypercapnia, thereby worsening carbon dioxide retention and risk for respiratory acidosis.

### Supporting Guideline Statements:

**2015 ACLS Guidelines for ACS:** “The provision of supplementary oxygen to patients with suspected ACS who are normoxic has not been shown to reduce mortality or hasten the resolution of chest pain. Withholding supplementary oxygen in these patients has been shown to minimally reduce infarct size. The usefulness of supplementary oxygen therapy has not been established in normoxic patients. In the prehospital, ED, and hospital settings, the withholding of supplementary oxygen therapy in normoxic patients with suspected or confirmed acute coronary syndrome may be considered (Class IIb, LOE C-LD).”


**2014 NSTEMI Guidelines:** “Supplemental oxygen should be administered to patients with NSTE-ACS with arterial oxygen saturation less than 90%, respiratory distress, or other high-risk features of hypoxemia. (Class I, Level of Evidence: C)”


**2013 STEMI Guidelines:** “Indications: clinically significant hypoxemia (oxygen saturation <90%), HF, Dyspnea. 2 to 4 L/min via nasal cannula. Increase rate or change to face mask as needed. Caution with chronic obstructive pulmonary disease and CO2 retention.” (Note: no ranking or level of evidence provided.)

### Cochrane Review:


**Study Design:** Cochrane Review

**Sample Size:**
- n = 4 randomized controlled trials
- n = 430 patients with NSTEMI or STEMI presenting within 24 hours of onset

**Groups & Interventions:** Supplemental oxygen (under normal pressure) vs room air

**Primary Endpoint (Purpose):** The primary outcomes were death, pain and complications.

**Secondary Endpoint(s):**
- The pooled RR of death was 2.05 (95% CI 0.75 - 5.58) in an intention-to-treat analysis and 2.11 (95% CI 0.78 - 5.68) in patients with confirmed AMI.
- Analgesic use was used as an indicator of pain control and found to have a pooled RR of 0.97 (95% CI 0.78 to 1.20).

**Results:**
- There were 17 deaths in total and this could be due to a chance of occurrence.

**Conclusions:** There is no conclusive evidence from randomized controlled trials to support the routine use of inhaled oxygen in people with AMI. A definitive randomized controlled trial is urgently required, given the mismatch between trial evidence suggestive of possible harm from routine oxygen use and recommendations for its use in clinical practice guidelines.

**Comments:** This is an update from the Cochrane review done in 2010 that was suggestive of a possible 3-fold increase in the risk of death in patients treated with supplemental O2.

### Clinical Trials:


**Study Design:** Prospective, Single-Center Trial

**Sample Size:** n = 18

**Groups & Interventions:** Adults with stable coronary artery disease undergoing cardiac catheterization receiving 100% FiO2 at 15 L/min via a facemask

**Results:**
- Compared to breathing room air, breathing of 100% O2 increased coronary resistance by approximately 40%, decreased CBF by approximately 30%, increased the appearance of nitrotyrosine in coronary venous plasma, and significantly blunted the CBF response to ACh.
- Oxygen breathing elicited these changes without affecting the diameter of large-conduit coronary arteries, coronary venous concentrations of NO(2)(-) and NO(3)(-), or the coronary vasodilator response to adenosine.

**Conclusions:** The use of high FiO2 supplemental oxygen in patients undergoing cardiac catheterization can increase coronary vascular resistance, possibly due to oxidative quenching of NO within the coronary microcirculation.

**Comments:** It is important to note that these were not patients presenting acutely with symptoms of ACS or active myocardial infarction, but rather were stable patients undergoing an elective cardiac cath, who received high concentrations of FiO2 despite a lack of hypoxia.

**Location(s):** Pennsylvania State College of Medicine, Milton S. Hershey Medical Center, Hershey, Pennsylvania


**Study Design:** Prospective, Animal Study

**Sample Size:** n = 15 anesthetized dogs

**Groups & Interventions:** Induced intermittent occlusions of the LAD coronary artery in dogs who had occlusions assessed while getting supplemental O2 at FiO of 0.2 vs FiO2 of 0.4

**Results:**
- In dogs getting FiO2 of 0.20, the average ST-segment elevation (ST) was 4.0 +/- 0.6 mV (SEM) and the number of sites exhibiting ST-segment elevations exceeding 2 mV (NST) 15 minutes following occlusion was 6.2 +/- 0.7 sites vs. following occlusion with an FiO2 of 0.40 were 1.8 +/- 0.4 mV (P < 0.01) and 2.7 +/- 0.7 sites (P < 0;01), thereby...
suggested a reduction in acute myocardial ischemic injury

- Increasing the FiO2 to 1.0 did not decrease myocardial injury further.
- In 9 dogs where FiO2 was increased from 0.20 to 0.40 30 minutes after occlusion, CPK levels were less depressed in sites having comparable levels of ST-segment elevation at 15 minutes than in dogs that respired an FiO2 of 0.20 during the entire 24 hours.
- All (54) sites with ST-segment elevations greater than 3 mV in the 0.20 FiO2 group showed early signs of myocardial infarction, while only 49% of such specimens showed infarction in the 0.40 FiO2 group.

**Conclusions:**
In an experimental coronary artery occlusion in dogs, the use of FiO2 of 0.4 appeared to reduce acute ischemic injury and the eventual development of necrosis based on enzymatic and histological assessment.

**Comments:**
Comments or observations you feel should be known, considered about this study.

**Location(s):**
Limit to 5 study locations (if > than 5 then state "> 5 locations"): list country or countries

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