Carbon Monoxide Poisoning

Introduction

- Carbon monoxide is a colorless, odorless, tasteless, toxic gas
- Generated during incomplete combustion of carbon based compound
- When inhaled, can cause serious physical problems and even death
Carbon Monoxide Poisoning

- Carbon monoxide is a common cause of poisoning worldwide including Hong Kong.

- Unintentional
  - Domestic accident
    - In-house heating (burning wood/charcoal) - during power failure in winter
    - Setting fire in enclosed space
  - Intentional
    - Car exhaust
    - Burning charcoal – as a comfortable way of suicide
      - Hong Kong & Taiwan

Paraquat Prohibition and Change in the Suicide Rate and Methods in South Korea

Woojae Myung, Geung-Ho Lee, Hong Hee Won, Maurizio Fava, David Mischoulon, Maren Nyer, Doh Woon Kim, Jung-Yoon Heo, Hong Jin Jeon

- Paraquat
- CO
- Any other substance
Pathophysiology (1)

- Incompletely understood
- \( \text{CO} + \text{Hb} \rightarrow \text{CO-Hb} \)
  - Competitive with \( \text{O}_2 \) with heme sites on \( \text{Hb} \)
  - >200 fold higher affinity than \( \text{O}_2 \)
  - Increase affinity of remaining sites for \( \text{O}_2 \), shift \( \text{O}_2 \) dissociation curve towards left
  - Decrease both the \( \text{O}_2 \)-carrying and \( \text{O}_2 \)-delivery capacity of blood
Pathophysiology (2)

- Disrupt the cellular oxidative processes by binding to intracellular proteins
  - Myoglobin, cytochromes a, a3
- During recovery, causing marked oxidative stress and inflammatory responses
  - NO generation $\rightarrow$ peroxynitrite production
  - Lipid peroxidation
  - Apoptosis (programmed cell death)
  - Immune-mediated injury
- Varying degrees of end-organ damage, especially the brain

Two syndromes

- Persistent neurologic sequelae
  - Symptoms immediately evident following poisoning
- Delayed neurologic sequelae,
  - Days or weeks later
  - Varies between reports from few % to two thirds
- Symptoms includes
  - Personality changes
  - Depressed mood,
  - Impaired short-term memory, poor attention & concentration
  - Parkinsonism or
  - Rarely focal neurological injuries or coma
Long-term risk of Dementia with CO poisoning
Wong CS et al., Medicine 95(3):e2549

Taiwan NHIRD 2004 – 2013
14,590 CO patients vs.
58,360 controls from comparison cohort

<table>
<thead>
<tr>
<th>TABLE 2. Incidence and Adjusted Hazard Ratios for Dementia During the 9-Year Follow-up Period.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbon Monoxide Poisoning</td>
</tr>
<tr>
<td>Dementia present</td>
</tr>
<tr>
<td>No. of person-years</td>
</tr>
<tr>
<td>Incidence/10,000 person-years</td>
</tr>
<tr>
<td>Crude hazard ratio</td>
</tr>
<tr>
<td>Adjusted hazard ratio</td>
</tr>
</tbody>
</table>

Values in parentheses are 95% confidence intervals.

*P value < 0.001.

Adjustments were made for diabetes mellitus, coronary artery disease, stroke, cancer, hypertension, hyperlipidemia, and Charlson comorbidity index.

FIGURE 1. Plot of dementia hazard curves based on the Cox model analysis for patients with carbon monoxide intoxication and comparison cohort.

History of potential exposure to a source of CO

Elevation of arterial or venous blood carboxyhemoglobin level (> 3-4% in nonsmokers or > 10% in smokers)

Symptoms consistent with CO poisoning (headache, dizziness, nausea, vomiting, confusion, fatigue, chest pain, shortness of breath, loss of consciousness)
## Signs and Symptoms

<table>
<thead>
<tr>
<th>CO-Hb</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4%</td>
<td>None - Normal</td>
</tr>
<tr>
<td>5-9%</td>
<td>Minor Headache</td>
</tr>
<tr>
<td>10-19%</td>
<td>Headache, Shortness of Breath</td>
</tr>
<tr>
<td>20-39%</td>
<td>Cherry Red discoloration is rare!</td>
</tr>
<tr>
<td>30-39%</td>
<td>Severe Headache, Vomiting, Vertigo, ALOC</td>
</tr>
<tr>
<td>40-49%</td>
<td>Confusion, Syncope, Tachycardia</td>
</tr>
<tr>
<td>50-59%</td>
<td>Seizures, Shock, Apnea, Coma</td>
</tr>
<tr>
<td>60% -</td>
<td>Coma, Death</td>
</tr>
</tbody>
</table>

Koster LA, Rupp T. The Silent Killer, Recognizing and Treating Carbon Monoxide Poisoning. JEMS, October 2005

## Measurement of carboxyhemoglobin (CO-Hb) level

- **Pulse CO oximeter**
  - Masimo Rad-57 signal extraction
  - Rapid, continuous, field measurement
  - But, inaccurate (-11.6% to 14.4%)
  - As part of the routine A&E triage, especially during winter time

- **Lab Co-oximetry**
  - Gold standard
  - Confirm clinical diagnosis

- Correlate poorly with outcome

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Deniz T et al. CO poisoning cases presenting with non-specific symptoms. Toxicol Ind Health 2016 Aug 4;pii:0748233716660641
Radiological changes

Axial CT
Axial T2-MRI
Coronal T2-MRI
Axial T1-MRI

Management

- Removing patients from source of CO
- General Supportive Care
- Normobaric oxygen therapy
  - Speed up elimination of CO from body
- Hyperbaric oxygen therapy (HBOT)
Hyperbaric Oxygen Therapy (HBOT)

- Breathing of 100% oxygen by patients within hyperbaric chambers with more than one atmospheric pressure (> 1ATA)
- > 1.4 ATA

Chiew AL, Buckley NA, CO poisoning in the 21st century, Critical Care 2014;18:221

Goal of HBO

- Not for CO-Hb clearance nor short term hospital survival but
- Prevent or alleviate
  - Persistent neurologic sequelae &
  - Delayed neurologic sequelae
Elimination of CO-Hb over time

Indications for HBO – Undersea and Hyperbaric Medical Society (UHMS)

- Air or Gas Embolism
- Carbon Monoxide Poisoning
- Clostridial Myositis and Myonecrosis (Gas Gangrene)
- Crush injury, Compartment Syndrome and Other Traumatic Ischemias
- Decompression Sickness
- Arterial Insuffciencies
- Severe Anemia
- Intracranial Abscess
- Necrotizing Soft Tissue Infections
- Osteomyelitis (Refractory)
- Delayed Radiation Injury (Soft Tissue and Bony Necrosis)
- Compromised Graft and Flaps
- Acute Thermal Burn Injury
- Idiopathic Sudden Sensorineural Hearing Loss (8 October 2011)
HBOT for delayed neurologic sequelae

- Very Controversial

Acute carbon monoxide poisoning in a regional hospital in Hong Kong: historical cohort study

MY Chan *, Thomas TS Au, KS Leung, WW Yan

ABSTRACT

Objectives: This study aimed to describe the clinical profiles of all patients with carbon monoxide poisoning admitted to a regional hospital in order to enhance the vigilance of healthcare professionals for delayed neurological sequelae associated with carbon monoxide poisoning and to identify the prognostic factors associated with their development. This study also aimed to assess the impact of hyperbaric oxygen therapy on the development of delayed neurological sequelae in these patients.

Methods: This was a historical cohort study in which all patients with a diagnosis of carbon monoxide poisoning managed in a regional hospital in Hong Kong from 12 February 2003 to 8 November 2013 were recruited. Main outcome measures included delayed neurological sequelae.

Results: Of the clinical profiles of 93 patients analysed, 24 patients received hyperbaric oxygen therapy and did not develop delayed neurological sequelae. Seven patients who did not receive hyperbaric oxygen therapy developed delayed neurological sequelae. Comparison of groups...
CO Poisoning and Subsequent Dementia

Taiwan NHIRD 2000 – 2011
9,041 CO patients vs. 36,160 controls from comparison cohort

**TABLE 4. Incidence and Hazard Ratio for Dementia Stratified by the Severity of Carbon Monoxide Poisoning**

<table>
<thead>
<tr>
<th>Carbon Monoxide Poisoning Severity</th>
<th>Event</th>
<th>PY</th>
<th>Rate$^2$</th>
<th>Adjusted HR$^1$ (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-CO poisoning</td>
<td>174</td>
<td>178,311</td>
<td>9.76</td>
<td>1(Reference)</td>
</tr>
<tr>
<td>CO poisoning</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low severity</td>
<td>36</td>
<td>32,424</td>
<td>11.1</td>
<td>1.23(0.85, 1.79)</td>
</tr>
<tr>
<td>High severity</td>
<td>26</td>
<td>8513</td>
<td>30.5</td>
<td>2.18(1.42, 3.36)$^{***}$</td>
</tr>
<tr>
<td>$^P$ for trend</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

**TABLE 3. Cox Proportional Hazards Regression Analysis for Hazard Ratio of Dementia-Associated Carbon Monoxide Poisoning With Interaction of Gender, Age, and Comorbidity**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Adjusted HR$^1$ (95% CI)</th>
<th>$P$-Value$^1$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbon monoxide poisoning</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1(Reference)</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1.45(1.05, 2.01)$^*$</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1.80(0.96, 3.37)</td>
<td></td>
</tr>
</tbody>
</table>

**2011**

Hyperbaric oxygen for carbon monoxide poisoning (Review)

Buckley NA, Juurlink DN, Isbister G, Bennett MH, Lavonas EJ
Analysis 1.1. Comparison of Hyperbaric Oxygen (HBO) vs. Normobaric Oxygen (NBO), Outcome 1: Presence of symptoms or signs at time of primary analysis (4-6 weeks).

**Review:** Hyperbaric oxygen for carbon monoxide poisoning

**Comparison:** Hyperbaric Oxygen (HBO) vs. Normobaric Oxygen (NBO)

**Outcomes:** Presence of symptoms or signs at time of primary analysis (4-6 weeks)

<table>
<thead>
<tr>
<th>Study or subgroup</th>
<th>Treatment</th>
<th>Control</th>
<th>Odds Ratio</th>
<th>Weight</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n/N</td>
<td>n/N</td>
<td>H/random</td>
<td>C</td>
<td>H/random</td>
</tr>
<tr>
<td>Presence of signs or symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raphael 1989</td>
<td>51/139</td>
<td>53/148</td>
<td>2.22</td>
<td>0.97</td>
<td>0.59</td>
</tr>
<tr>
<td>Thiem 1995</td>
<td>7/10</td>
<td>7/20</td>
<td>3.31</td>
<td>0.02</td>
<td>0.03</td>
</tr>
<tr>
<td>Mahieu 1996</td>
<td>66/299</td>
<td>70/276</td>
<td>1.08</td>
<td>0.72</td>
<td>0.72</td>
</tr>
<tr>
<td>Schenkel-Kap 1999</td>
<td>30/48</td>
<td>25/40</td>
<td>1.06</td>
<td>0.03</td>
<td>0.03</td>
</tr>
<tr>
<td>Weafer 2002</td>
<td>19/76</td>
<td>36/76</td>
<td>1.04</td>
<td>0.03</td>
<td>0.03</td>
</tr>
<tr>
<td>Annane 2010</td>
<td>33/93</td>
<td>29/96</td>
<td>1.04</td>
<td>0.03</td>
<td>0.03</td>
</tr>
<tr>
<td>Total (95% CI)</td>
<td>78/5</td>
<td>65/6</td>
<td>1.00</td>
<td>0.78</td>
<td>0.54, 1.12</td>
</tr>
</tbody>
</table>

**Total observed patients:** 222 (Treatment) 220 (Control)

**Heterogeneity test:** $I^2 = 0.00$, $Q = 9.22, df = 9 (p = 0.10), P = 0.56$

**Test for overall effects:** $Z = 1.39 (p = 0.17)$

Hyperbaric Oxygen (HBO) compared to Normobaric Oxygen (NBO) for carbon monoxide poisoning

**Patient or population:** Patients with carbon monoxide poisoning

**Setting:** Hospital

**Intervention:** Hyperbaric Oxygen (HBO)

**Comparison:** Normobaric Oxygen (NBO)

**Outcomes**

<table>
<thead>
<tr>
<th>Illustrative comparative risks * (95% CI)</th>
<th>Relative effect (95% CI)</th>
<th>No of Participants (studies)</th>
<th>Quality of the evidence (GRADE)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Assumed risk</strong></td>
<td>Corresponding risk</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normobaric Oxygen (NBO)</td>
<td>Hyperbaric Oxygen (HBO)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Presence of symptoms or signs at time of primary analysis (4-6 weeks)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Study population | OR 0.78 (0.54 to 1.12) | 1351 (6 studies) | |}

**Quality of the evidence:** Very low 1, 2, 3, 4, 5.
### Raphael 1989

**Methods**
Prospective, randomized, unblinded trial. Randomization stratified according to history of loss of consciousness. Allocation by sealed opaque envelopes, not sequentially numbered. Only those with no history of LOC randomized to HBO vs. NBO; more severe patients randomized to different regimens of HBO. Jadad score 3/5.

**Participants**
629 adults admitted within 12 hours of termination of CO exposure. Inclusion: age > 15 y; admitted within 12 h, COHb > 10% (smoker) or < 5% (non-smoker). Exclusion: alcohol intoxication, pregnancy, CV collapse, pulmonary edema, non-feasible HBO (technical problems etc.), difficulty in stratifying into groups A or B (by LOC), refusal by patient. Of enrolled patients, 343 were randomized to receive either HBO or NBO.

**Interventions**
Only those without history of loss of consciousness randomized to HBO vs. NBO. A0 - 100% oxygen x 6h - other patients randomized to HBO x 1 vs. HBO x 2; not included in analysis. A1 - HBO x 2h followed by 100% oxygen x 4h (where HBO regimen included 30 mins compression & decompression flanking 60 mins at 2.0 ATA.)

**Outcomes**
Intention to treat analysis. Outcome measures included self-assessment questionnaire and physical examination by neurologist (unblinded at one month, with no difference in outcome (symptoms present in 50 of 158 patients (32%) treated with NBO vs. 51 of 159 patients (32%) treated with HBO at one month.)

**Risk of bias**

<table>
<thead>
<tr>
<th>Item</th>
<th>Authors’ judgement</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allocation concealment?</td>
<td>Unclear</td>
<td>B - Unclear</td>
</tr>
</tbody>
</table>

### Tchon 1995

**Methods**
Prospective, randomized, unblinded trial of HBO vs. NBO. Treatment allocation by computer-generated random numbers within sealed opaque envelopes, not sequentially numbered. Jadad score 3/5.

**Participants**
65 patients referred from local emergency departments, within 6 hours of removal from exposure. Inclusion criteria: history of acute exposure, elevated COHb, symptoms consistent with CO poisoning. Exclusion criteria: history of LOC, active ischemia. Two groups largely similar (higher average COHb in HBO group 24.6% vs. 20.0%).

**Interventions**
All patients in HBO arm given 100% O2 until HBO initiated. HBO began within 6 h of end of exposure. HBO @ 2.8 ATA for 30 minutes, then 2.0 ATA x 90 minutes. NBO 100% O2 until all symptoms resolved (mean 4.2 +/- 0.3 h). After intervention, neuropsychologic baseline testing (6 tests) performed (some up to 12 hrs. post-Rx). Occurrence of DNS self-reported as (1) recurrent symptoms or (2) new symptom consistent with DNS, plus deterioration in 1 or more subnet upon retesting.

**Outcomes**
Outcome assessors not blinded to treatment allocation. 5 patients lost to follow up (2 control, 3 HBO). 7750 patients in control arm had sequelae consistent with DNS vs. 0/50 patients in HBO arm.

**Notes**
No statistical adjustment for multiple comparisons (previous analysis published as abstract in 1992) raising concern of spurious false positive results, particularly in light of recruitment and outcome pattern of the final seven patients included in trial.

**Risk of bias**

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<tbody>
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</tr>
</tbody>
</table>
### Mathieu 1996

<table>
<thead>
<tr>
<th>Methods</th>
<th>Prospective, randomised, unblinded trial.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participants</td>
<td>575 non-comatose nonpregnant patients with no evidence of mixed poisoning, recruited over 3 years. COHb &gt; 10%</td>
</tr>
<tr>
<td>Interventions</td>
<td>HBO at 2.5 ATA for 90 minutes (plus 15 minutes each for compression and decompression) vs. 12 hours of NBO</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Neuropsychological testing at 1, 3, 6, and 12 months. Persistent neurological manifestations were present in 23% of HBO arm and 26% of NBO arm at 1 month, but detailed data were not presented</td>
</tr>
<tr>
<td>Notes</td>
<td>Data from abstract of 1996 interim analysis only. This trial is not registered and no later data were available for analysis at the time of the 2005 or 2011 review. Author contacted in 2004 and 2010 but no further information provided</td>
</tr>
</tbody>
</table>

### Scheinkestel 1999

<table>
<thead>
<tr>
<th>Methods</th>
<th>Prospective double-blind RCT of HBO vs. NBO. Cluster randomization for patients presenting simultaneously. Allocation through sealed opaque envelopes, not sequentially numbered. Patients and outcome assessor blind to allocation, technicians and nurses not. Stratified by vent/non-vent and suicide vs. accidental exposure. Jailed score 5/5.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participants</td>
<td>230 patients sequentially referred to single center in Australia. Inclusion: all referred. Excluded: non-39; children, burn victims, pregnant. Two groups similar for all important variables. 89% male, coma in 50.6%, average COHb 21%. Large number of suicide attempts (69%), co-intoxication (44%), and severe poisonings (73%).</td>
</tr>
<tr>
<td>Interventions</td>
<td>All patients given high-flow O2 prior to randomization. Daily treatment (x3) of HBO (100 minutes: 60 minutes at 2.8 ATA) or NBO (100 minutes of 100% O2 at 1.6ATA) as a sham dose. After third treatment, patients with deficits were treated again, with high-flow oxygen in between. 3 additional courses of original therapy given to 28% HBO and 15% NBO because of &quot;poor outcome&quot;.</td>
</tr>
<tr>
<td>Outcomes</td>
<td>191 randomised (104 HBO NBO 87, discrepancy due to cluster) No mortality difference at discharge. Poor follow-up attendance (46%) at one month. 34/52 symptomatic in HBO arm vs. 20/54 symptomatic in NBO arm (NS).</td>
</tr>
<tr>
<td>Notes</td>
<td>Several other conclusions in text, based upon repeated neuropsychologic testing. However, no adjustment for multiple comparisons; high likelihood of spurious statistical significance.</td>
</tr>
</tbody>
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#### Risk of bias

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<tbody>
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</table>
### Weaver 2002

<table>
<thead>
<tr>
<th>Item</th>
<th>Authors' judgement</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allocation concealment?</td>
<td>Yes</td>
<td>Adequate</td>
</tr>
</tbody>
</table>

#### Methods
Prospective, randomised, double-blind RCT of HBO vs. NBO. Randomization method used sequentially numbers sealed envelopes. Jadad score 5/5. Allocation concealment possibly jeopardized by fixed block size of 5.

#### Participants
152 patients with CO poisoning (symptomatic and COHb > 10% or symptoms and signs unequivocally due to CO exposure). Exclusions: Pregnancy, > 24 wks since exposure, < 16 yrs of age, moribund, refused consent. Stratified by LOC, age < 40, and delay to treatment < 6h.

#### Interventions
HBO - 1 session 3ATA x 1h & 2ATA x 1h, followed by two sessions 2ATA x 2h at 6-12 hour intervals. NBO patients received sham treatment at 1 ATM. Oxygen not routinely used after first session.

#### Outcomes
Serial neuropsychological testing immediately after treatments 1 and 3, and then at 2, 6, 26 and 52 weeks follow-up.

#### Notes
Endpoint in published trial different from that described in initial report of first interim analysis and earlier published descriptions of trial.

### Amanze 2010

<table>
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</thead>
<tbody>
<tr>
<td>Allocation concealment?</td>
<td>Yes</td>
<td>Adequate</td>
</tr>
</tbody>
</table>

#### Methods
Prospective, randomised unblinded trial. Similar to the earlier trial by the same investigators, randomisation was stratified by history of "transient loss of consciousness" vs. "initial coma". Patients without impaired consciousness were excluded. Patients with "transient loss of consciousness" were randomised to HBO vs NBO ("Trial A") and are included in this review. A separate group of patients with "initial coma" was randomised to receive 1 vs 2 HBO treatment sessions ("Trial B"), and are not considered in this review.

#### Participants
179 patients ≥ 15 yrs of age presenting for therapy between Oct 1989 and Jan 2000 within 12 hours of exposure with a COHb of >5% if a non-smoker or >10% if a smoker and a history of transient (but not sustained) loss of consciousness. Key exclusion criteria included: suicide attempts, non-domestic poisoning, inhalation of smoke or other toxic material (other than CO).

#### Interventions
In "Trial A", patients with "transient loss of consciousness" were randomised to receive mask oxygen alone for 6 hours (NBO) or mask oxygen for 4 hours and HBO at 2.0 ATA for 120 minutes including 30 minutes compression/decompression. In addition, HBO patients received treatments 10-14 days.

#### Outcomes
Outcome measures included self-assessment questionnaire and examination by a blinded neurologist at 1 month. No difference in primary outcomes was evident, with symptoms present in 29 of 74 patients (39%) randomized to NBO vs 33 of 79 patients (42%) randomized to HBO.

#### Notes
This trial was originally reported in Abstract 2004 (Raphael 2004) and included in our previous review. The trial protocol was retrospectively added to a clinical trials
Authors’ Conclusions

- Existing randomised trials do not establish whether the administration of HBO to patients with carbon monoxide poisoning reduces the incidence of adverse neurologic outcomes
  - HBO cannot be routinely recommended for the treatment of CO poisoning
  - It is possible that some patients, particularly those with more severe poisoning, may derive benefit from treatment, but this remains unproven

- Additional research is needed to better define the role, if any, of HBO in the treatment of patients with carbon monoxide poisoning.

Optimal HBO protocol

- Unknown
  - ? No. of session
  - ? Depth of dive
  - ? Duration of each session
To identify practice differences in CO poisoning treatment with HBOT among centres in Europe
Commercial online survey website
68 centres from 23 countries

- 39% (18/46) single session within 24 hours
- 19% (9/46) three sessions within 24 hours

### Indications of HBOT
- Transient or prolonged unconsciousness 100%
- Positive neurological findings, ischemic changes in ECG and pregnancy 95%
- Elevated carboxyhemoglobin 44%

A total of 21 different HBOT profiles used in European centres!
PyN ICU indications for HBO in CO poisoning

- Loss of consciousness at any time
- Neurological symptoms and signs
- Chest pain or evidence of myocardial ischemia
- Pregnancy
- CO-Hb > 25%

Use of HBO in CO poisoning in HK

<table>
<thead>
<tr>
<th>HBO Indications</th>
<th>No. of cases (%)</th>
<th>HBO given (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present *</td>
<td>59 (19.5)</td>
<td>4/59 (6.8)</td>
</tr>
<tr>
<td>Absent</td>
<td>244 (80.5)</td>
<td>0/244 (0)</td>
</tr>
<tr>
<td>Total</td>
<td>303 (100)</td>
<td>4/303 (1.3)</td>
</tr>
</tbody>
</table>

*Hx of syncope / coma, cardiac ischemia/arrhythmia or CO-Hb>25%

Hong Kong Poison Information Centre, data from 2006 - 2009
Reasons for low HBO referral in HK

- Evidence of efficacy of HBO therapy
- Risk of Transport and lack of support in RTC
- Occupational health risk
- Manpower shortage

Risk

Benefit
HBO facilities in Hong Kong for public hospitals

- The Recompression Treatment Centre at Stonecutter’s Island
  - Not attached to hospital

- Situated in a government dockyard

Year of 2010
RTC at Stonecutter’s island

- Not only for medical uses
  - Also for disciplinary forces training

- Not attached to hospital
- Only basic monitoring and resuscitative equipments available
- Crowded environment

- Occupational Health Division of Labour department
  - Not used to deal with clinical emergencies or resuscitation
  - No nursing nor clerical support
- In case of unexpected event, no immediate support
  - From own department or
  - From other clinical specialties
## Hospital-based HBOT Centre in Hong Kong

### Proposed timeline:

<table>
<thead>
<tr>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase 1: PYNEH</td>
<td></td>
<td>Site preparation</td>
<td>First HBOT Centre</td>
<td></td>
<td></td>
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<td></td>
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<td>Phase 2: Kai Tak Hospital</td>
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<td>Development and Site Preparation</td>
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**Image:** Hospital-based HBOT Centre in Hong Kong
Two-dimensional Display

indicates Patient Flow

Three-dimensional Display

indicates Patient Flow
Prevention

- Legislation for prevention of accidental exposure
  - E.g. Installation of heating machine by registered technicians
- Education
  - Hong Kong Poison Control Network
    - Hong Kong Poison Information Centre
    - Poison Treatment Centre
    - Toxicology Reference Lab
  - Hong Kong College of Emergency Medicine
RCT on Traditional Chinese Medicine

A Randomized Controlled Trial of Puncturing and Bloodletting at Twelve Hand Jing Points to Treat Acute Carbon Monoxide Poisoning as Adjunct to First Aid Treatment: A Study Protocol

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Conclusions

- CO poisoning is a popular form of suicide method
- Carries debilitating long term side effects, delayed neurological sequelae
- Management is mainly supportive, Oxygen therapy
- HBOT is still controversial, needs further studies
  - Indications
  - Timing
  - Treatment profile

Thank you for your attention.