

2016 Joint Conference of Poison Control Centres Evaluation and Management of the Seriously Ill Poisoned Patients



Carbon Monoxide Poisoning

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26 SEPTEMBER 2016

Introduction



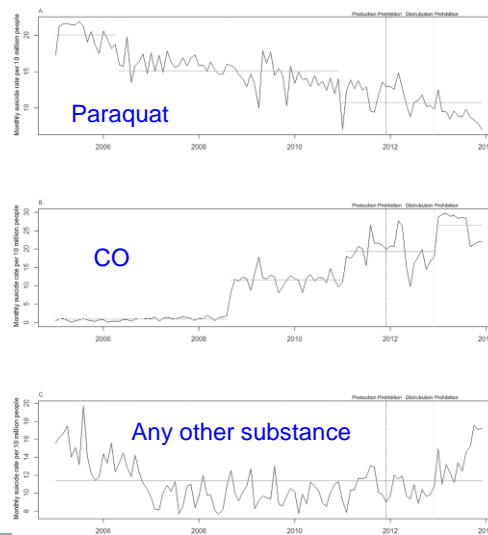
- Carbon monoxide is a colorless, odorless, tasteless, toxic gas
- Generated during incomplete combustion of carbon based compound
- When inhaled, can cause serious physical problems and even death

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^{1,2,3*}, Geung-Hee Lee^{2*}, Hong-Hee Won³, Maurizio Fava⁴, David Mischoulon⁴, Maren Nyer⁴, Doh Kwan Kim¹, Jung-Yoon Heo¹, Hong Jin Jeon^{1,4,5*}



PLOS ONE | DOI:10.1371/journal.pone.0128980 June 2, 2015

CASE REPORT

Open

Carbon monoxide poisoning-induced cardiomyopathy from charcoal at a barbecue restaurant: a case report

Hyun-Jun Kim¹, Yun Kyung Chung¹, Kyeong Min Kwak¹, Se-Jin Ahn¹, Yong-Hyun Kim¹, Young-Su Ju¹, Young-Jun Kwon¹ and Eun-A Kim²



Annals of Occupational and Environmental Med 2015;27:13

Unintentional deaths from carbon-monoxide poisoning due to a traditional practice observed during the post-partum period

[Med Sci Law](#). 2016 Sep 14. pii: 0025802416668459

Narghile Smoking

[Am J Case Rep](#). 2016 Sep 13;17:660-2



Suicidal asphyxiation by carbon monoxide within a polythene bag

[Med Leg J](#). 2016 Sep 12. pii: 0025817216669286

Pathophysiology (1)

- Incompletely understood
- $\text{CO} + \text{Hb} \rightarrow \text{CO-Hb}$
 - Competitive with O_2 with heme sites on Hb
 - >200 fold higher affinity than O_2
 - Increase affinity of remaining sites for O_2 , shift O_2 dissociation curve towards left
 - Decrease both the O_2 -carrying and 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 → 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 2. Incidence and Adjusted Hazard Ratios for Dementia During the 9-Year Follow-up Period

	Carbon Monoxide Poisoning	Reference Group
Dementia present	189	227
No. of person-years	81,017	353,206
Incidence/10,000 person-years	23.33	6.43
Crude hazard ratio	3.60* (2.97–4.37)	1.00 (reference)
Adjusted hazard ratio [†]	2.75* (2.26–3.35)	1.00 (reference)

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.

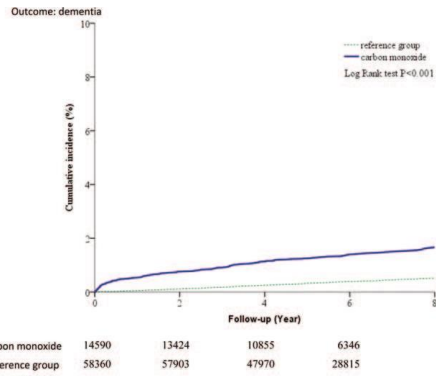
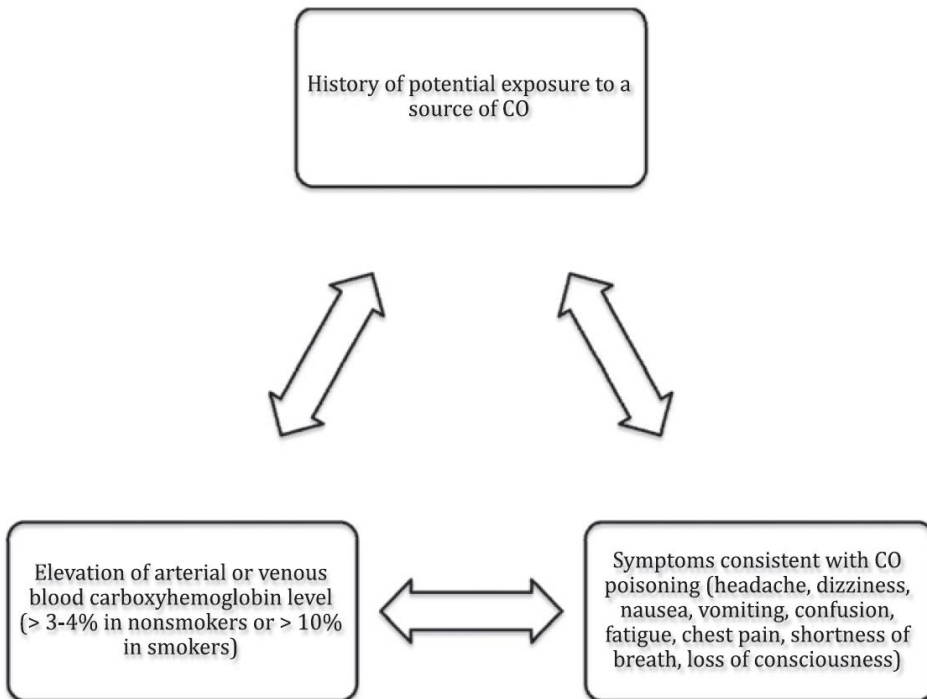


FIGURE 1. Plot of dementia hazard curves based on the Cox model analysis for patients with carbon monoxide intoxication and comparison cohort.



Signs and Symptoms

CO-Hb	Clinical Manifestations
0-4%	None - Normal
5-9%	Minor Headache
10-19%	Headache, Shortness of Breath
20-29%	Cherry Red discoloration is rare!
30-39%	Severe Headache, Vomiting, Vertigo, ALOC
40-49%	Confusion, Syncope, Tachycardia
50-59%	Seizures, Shock, Apnea, Coma
60% - >	Coma, Death

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

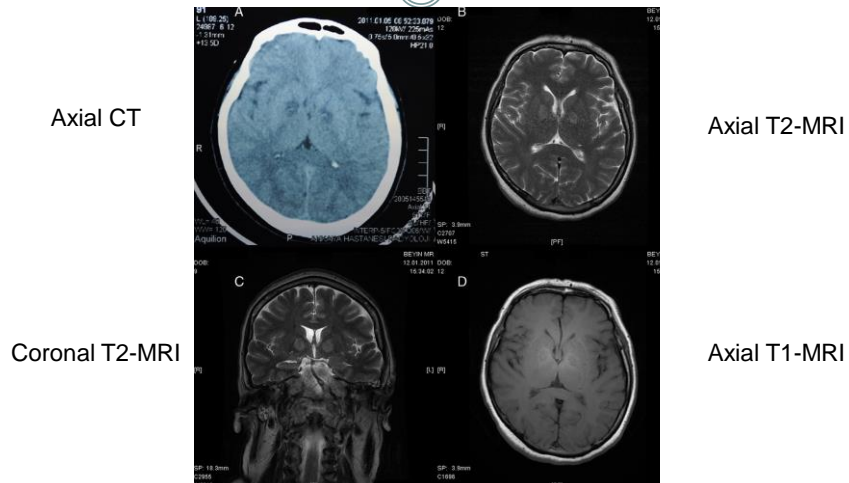
Measurement of carboxyhemoglobin (CO-Hb) level

- 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



[#]Deniz T et al, CO poisoning cases presenting with non-specific symptoms, *Toxicol Ind Health* 2016 Aug 4;pii:0748233716660641

Radiological changes



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

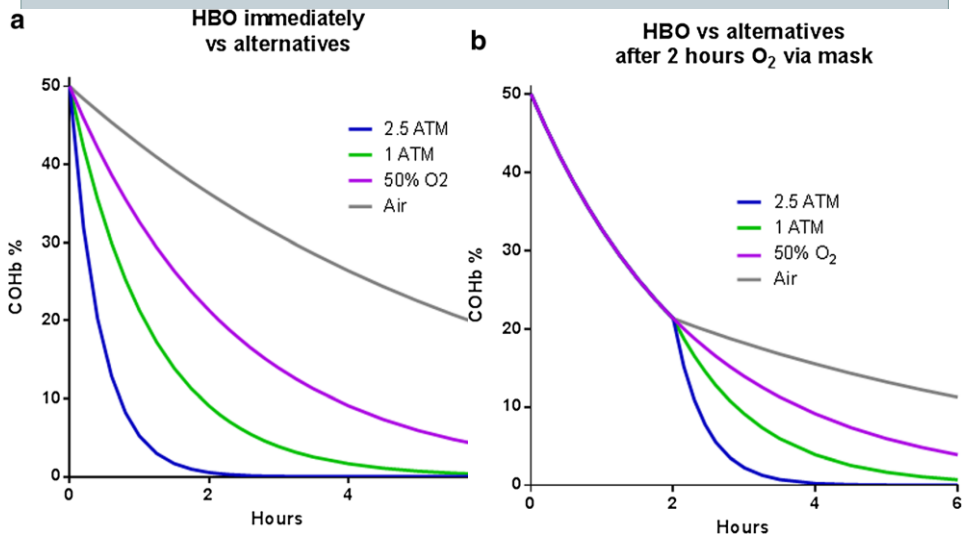
Chiew AL, Buckley NA, CO poisoning in the 21st century, Critical Care 2014;18:221

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 Insufficiencies
- 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 health care 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

急性一氧化碳中毒在香港一所分區醫院的歷史隊列研究

陳銘賢、歐德信、梁啟城、殷榮華

目的：描述所有入住一所分區醫院的一氧化碳中毒患者的臨床情況，從而加強醫療界人士對一氧化碳中毒與避發性神經系統後遺症（DNS）的警惕性和識別DNS的預後因素。本研究也旨在評估高壓氧治療對DNS發展的影響。

方法：這項歷史隊列研究納入由2003年2月12日至2013年11月8日期間，於香港一所分區醫院確診一氧化碳中毒的患者。主要結果測量包括DNS。

結果：分析了93名符合研究納入標準的患者，其中24人接受高壓氧治療而沒有出現DNS。沒有接受高壓氧治療的患者中，7人出現DNS。比較DNS和非DNS組別（不包括高壓氧治療的患者）後發現，對發展DNS的可能預後因素分別為：昏倒（ $P=0.038$ ）、格拉斯哥昏迷指數（GCS）為3（ $P=0.012$ ）、心肌鈣蛋白水平上升（ $P<0.001$ ）、肌酸激酶水平上升（ $P=0.008$ ）以及氣管插管（ $P=0.007$ ）。

結論：儘管統計上不顯著，本研究顯示高壓氧治療對嚴重一氧化碳中毒患者提供了100%防止DNS的保護作用。須進一步使用更好的研究設計來探討這問題。昏倒、低GCS、氣管插管以及提升心肌鈣蛋白和肌酸激酶水平是可能的DNS預後因素。在此建議要治療嚴重一氧化碳中毒的病人，須先定立一個明確的治療方案再加上適當的精神情緒測試和覆診時間表及一所位於醫院內的高壓氧氣室。

Hong Kong Med J 2016;22:46-55

DOI: 10.12809/hkmj144529

CO Poisoning and Subsequent Dementia

Lai CY et al, *Medicine* 95(1):e2418

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

Carbon Monoxide Poisoning Severity [†]	Event	PY	Rate [‡]	Adjusted HR [§] (95% CI)
Non-CO poisoning	174	178,311	9.76	1(Reference)
CO poisoning				
Low severity	36	32,424	11.1	1.23(0.85, 1.79)
High severity	26	8513	30.5	2.18(1.42, 3.36) ^{***}
<i>P</i> for trend				<0.001

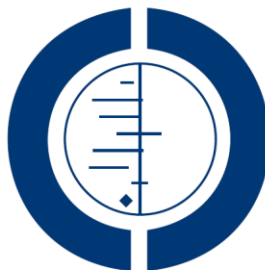
TABLE 3. Cox Proportional Hazards Regression Analysis for Hazard Ratio of Dementia-Associated Carbon Monoxide Poisoning With Interaction of Gender, Age, and Comorbidity

Variables		Adjusted HR [†] (95% CI)	<i>P</i> -Value [‡]
Carbon monoxide poisoning	Hyperbaric oxygen therapy		
No	No	1(Reference)	
Yes	No	1.45(1.05, 2.01) [*]	
Yes	Yes	1.80(0.96, 3.37)	

2011

Hyperbaric oxygen for carbon monoxide poisoning (Review)

Buckley NA, Juurlink DN, Isbister G, Bennett MH, Lavonas EJ



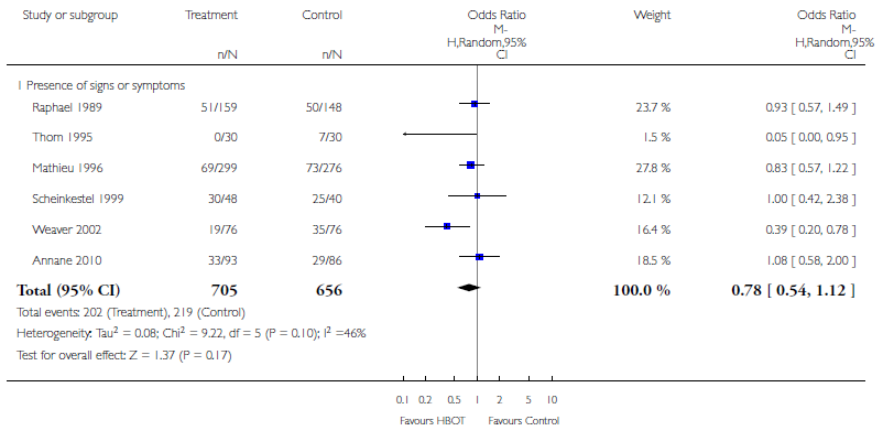
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Analysis I.1. Comparison I Hyperbaric Oxygen (HBO) vs. Normobaric Oxygen (NBO), Outcome I Presence of symptoms or signs at time of primary analysis (4-6 weeks).

Review: Hyperbaric oxygen for carbon monoxide poisoning

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

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



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

Patient or population: patients with carbon monoxide poisoning

Settings: hospital

Intervention: Hyperbaric Oxygen (HBO)

Comparison: Normobaric Oxygen (NBO)

Outcomes	Illustrative comparative risks* (95% CI)				Relative effect (95% CI)	No of Participants (studies)	Quality of the evidence (GRADE)
	Assumed risk		Corresponding risk				
	Normobaric (NBO)	Oxygen	Hyperbaric (HBO)	Oxygen			
Presence of symptoms or signs at time of primary analysis (4-6 weeks)	Study population				OR 0.78 (0.54 to 1.12)	1361 (6 studies)	⊕○○○ very low ^{1,2,3,4,5}
	334 per 1000		281 per 1000 (213 to 360)				
	Medium risk population						
	338 per 1000		285 per 1000 (216 to 364)				

Raphael 1989

Methods	Prospective, randomized, <u>unblinded</u> 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% (nonsmoker) Exclusion: other 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	<u>Only those without history of loss of consciousness randomized to HBO vs. NBO.</u> 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 <u>by neurologist (unblinded)</u> 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.)	
<i>Risk of bias</i>		
Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

Thom 1995

Methods	Prospective, randomized, <u>unblinded</u> trial of HBO vs. NBO. Treatment allocation by computer-generated random numbers within sealed opaque envelopes, not sequentially numbered. Jadad score 3/5.	
Participants	<u>65 patients</u> 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 begun 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 subtest upon retesting.	
Outcomes	<u>Outcome assessors not blind to treatment allocation.</u> 5 patients lost to follow up (2 control, 3 HBO). 7/30 patients in control arm had sequelae consistent with DNS vs. 0/30 patients in HBO arm.	
Notes	<u>No statistical adjustment for multiple comparisons</u> (previous analysis published as abstract in 1992) raising concerns of spurious false positive results, particularly in light of recruitment and outcome pattern of the final seven patients recruited to trial.	
<i>Risk of bias</i>		
Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

Mathieu 1996	
Methods	Prospective, randomised, <u>unblinded trial</u> .
Participants	575 <u>non-comatose nonpregnant patients</u> with no evidence of mixed poisoning, recruited over 3 years. COHb > 10%
Interventions	HBO at 2.5 ATA for 90 minutes (plus 15 minutes each for compression and decompression) vs 12 hours of NBO
Outcomes	Neuropsychologic 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
Notes	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

Scheinkestel 1999

Methods	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. Jadad score 5/5.	
Participants	230 patients sequentially referred to single center in Australia. Inclusion: all referred. Excluded (n=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%).	
Interventions	All patients given high-flow O2 prior to randomization. Daily treatment (x3) of HBO (100 minutes; 60 minutes at 2.8 ATA) OR <u>NBO (100 minutes of 100% O2 at 1 ATA) as a sham dive</u> . 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 "poor outcome".	
Outcomes	191 randomized (104 HBO NBO 87, discrepancy due to cluster) No mortality difference at discharge. <u>Poor follow-up attendance (46%) at one month</u> . 34/52 symptomatic in HBO arm vs. 20/34 symptomatic in NBO arm (NS).	
Notes	Several other conclusions in text, based upon repeated neuropsychologic testing. However, <u>no adjustment for multiple comparisons</u> ; high likelihood of spurious statistical significance.	
<i>Risk of bias</i>		
Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

Weaver 2002

Methods	Prospective, randomized, 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 6.	
Participants	152 patients with CO poisoning (symptomatic and COHb > 10% or symptoms and signs unequivocally due to CO exposure). Exclusions: Pregnancy, > 24h since exposure, < 16 years 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. <u>NBO patients received sham treatment at 1 ATM.</u> 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	<u>Endpoint in published trial different from that described in initial report of first interim analysis and earlier published descriptions of trial.</u>	
<i>Risk of bias</i>		
Item	Authors' judgement	Description
Allocation concealment?	Yes	A - Adequate

Annane 2010

Methods	Prospective, randomised <u>unblinded</u> 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	
Participants	179 patients ≥ 15 years 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 attempt, non-domestic poisoning, inhalation of smoke or other toxic	
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 diazepam 10 mg IM	
Outcomes	Outcome measures included <u>self-assessment questionnaire and examination by a blinded neurologist at 1 month.</u> 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 in 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

The Use of HBOT for CO poisoning in Europe

Undersea Hyperb Med 2016 Jan-Feb;43(1):49-56



- 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

The Use of HBOT for CO poisoning in Europe

Undersea Hyperb Med 2016 Jan-Feb;43(1):49-56



- 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

HBO Indications	No. of cases (%)	HBO given (%)
Present *	59 (19.5)	4/59 (6.8)
Absent	244 (80.5)	0/244 (0)
Total	303 (100)	4/303 (1.3)

* 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









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

47

- 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

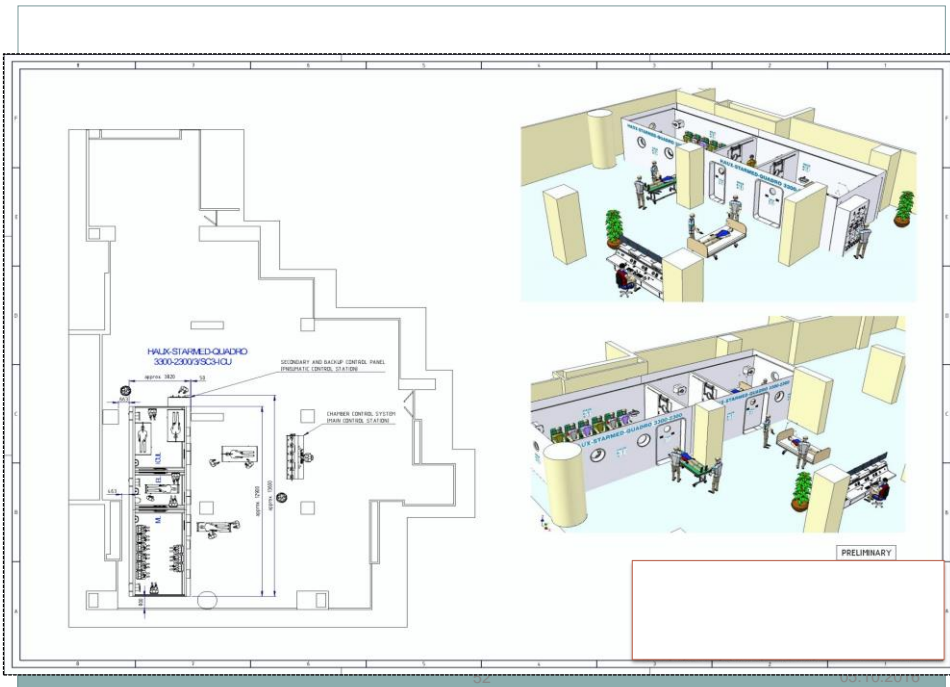
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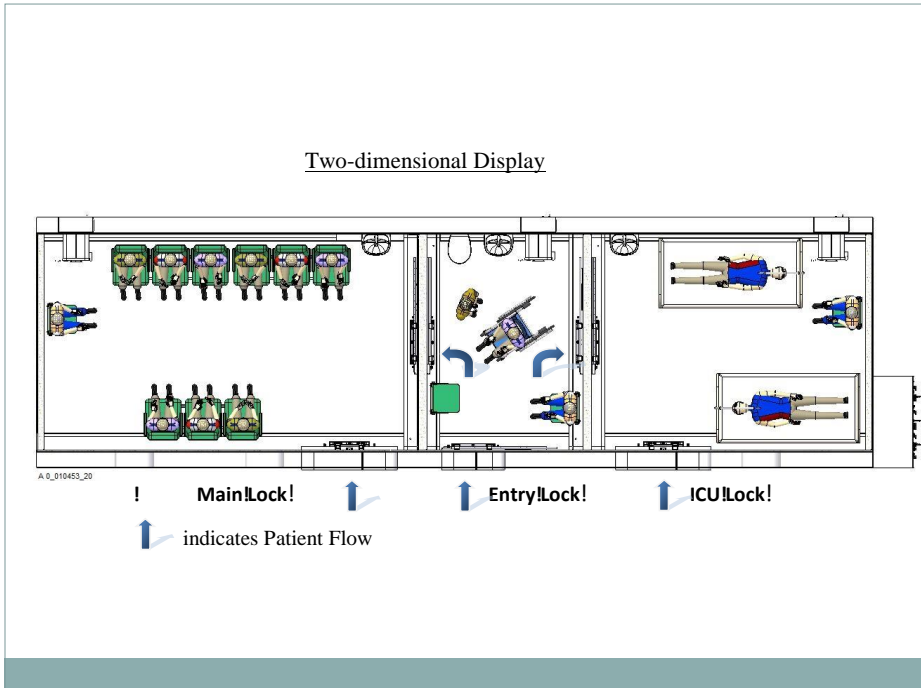
Hospital-based HBOT Centre in Hong Kong

Proposed timeline:

Year	2014/15	2015/16	2016/17	2017/18	2018/19	2019/20	2020/21	2021/22
Phase 1: PYNEH		Site preparation		First HBOT Centre				
Phase 2: Kai Tak Hospital				Development and Site Preparation				Second HBOT Centre







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

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急症解毒 臨牀毒理科 急症科的次專科

某夜，急症室當值護士長收到消息，救護車正趕送一位七十歲懷疑心臟有問題的女士來醫院。她有血壓高的病史，但總算健康，從未住院。她因腳痛在早上看了中醫，晚上約九時服過中藥，深夜一時，開始心悸及嘔吐。她在救護車上的血壓還算正常，脈搏卻是每分鐘185。

護士長聽到這消息後，二話不說使用擴音器呼叫：「R(搶救)房即將有case，醫生護士請理位。」負責搶救的醫生和護士立即放下手頭上的工作，趕到R房，有默契地組成搶救小組，趕緊在病人未抵達前檢查及準備急救儀器。

救護車火速把病人送到，救護員交代病情，護士第一時間量度病人的維生指數，接駁監察儀器，做心電圖、抽血和吊鹽水。因為病人還清醒，醫生也盡快問了病歷及檢查。心電圖初步顯示病人有室性心動過速(VT)，心跳達每分鐘186。正當醫生慶幸病人還支持得住，準備用藥時，病人卻突然全身抽搐。醫生憑着多年經驗，知道這並非普通的癱瘓抽筋，而是心跳驟停引致腦部缺氧而引發的短暫抽搐，抽搐之後便死亡。醫生立刻查看監察儀上的心電圖，果然已經轉化為心室顫動，病人已經沒有脈搏了。說時遲那時快，醫生未說完「除顫電擊，200焦耳(Joule)」，護士已經準備好電擊機給醫生。基於醫護的迅捷反應，病人在心跳停頓不到一分鐘，便接受了第一次除顫電擊，心電圖之後早一直線，很快便恢復實性心律，病人的脈搏也重新出現。兩分鐘內，病人已甦醒過來，卻不知道自己剛從鬼門關給救回來。

病人之後被送到深切治療部監察，她的心臟檢查基本健康。她的尿液、吃過的中藥殘渣和藥方都拿去化驗，證實是烏頭鹼過量中毒，引致室性心動過速和心室顫動，幸虧搶救及時，把她從奈何橋頭拉回來。烏頭鹼常見於川烏、草烏、附子等的中藥，主要功效是祛風除濕，溫經止痛。她的藥方內有製川烏及製草烏，但份量是衛生署建議的五倍，故中毒。

急症專科在香港已經發展二十年，今年又獲香港醫學專科學院批准，在香港急症科醫學院之下設立新的專科——「臨牀毒理科」。我們將在這個專欄內與讀者分享一些急症科與臨牀毒理科的小故事和知識。

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逢周五見報



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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Received 11 March 2015; Accepted 7 July 2015

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.