Role of Hyperbaric Oxygen Therapy in Carbon Monoxide Poisoning and Other Environmental Pollution Induced Poisoning

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The Asia-Pacific Society Undersea and Hyperbaric Medical Society

Compassion · Accountability · Effectiveness 關懷 · 專業 · 效率

Content

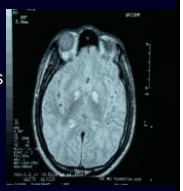
- A general introduction to acute carbon monoxide poisoning
- Delayed neurological syndrome of carbon monoxide poisoning
- The therapeutic effects of HBO on acute carbon monoxide poisoning and possible mechanisms
- Smoking inhalation
- Cases presentation

CO poisoning, Smoking Inhalation, and Flame Burns

A case saved by Hyperbaric Oxygen Therapy successfully

Chronic CO Toxicity

Delayed Neurological Syndrome



- A 32 y/o male
- C/O: Deep coma, suicide attempt
- GCS: E2V2M4→ E1V1M2 ; pin-point pupils
- COHb: 37.5% (our ER) ; ICU care
- Endotracheal tube with ventilator support
- EEG: diffuse cortical dysfunction

  Course: 1st~3rd day: E2VEM2
  4th~5th day: E2VEM4
  6th day: E4VEM6 → E4V5M6
  12th day: discharged
- 1 month later: Came back to OPD with metal retardation, and Parkinsonism syndrome

Cerebral Ischemic of a Firefighter post Smoke Inhalation – (1)

Pre HBO

Post HBO

- A 43 y/o fireman : after a fire fighting.
- 1ATA O_2, and seemed fine.
- 2 weeks later suddenly lapsed into coma.
- Awake with personality changes, non-verbal, and fed with NG-tube.
- SPECT: marked diffuse cerebral deficits, mostly frontal and basal ganglia.
- HBOT: 1hr/1.5 ATA, 65 HBOTs treatments, combined with PT and OT.

(Richard A Neubauer, 2001)

Cerebral Ischemic of a Firefighter Post Smoke Inhalation – (2)

- Result: More alert, cognitive improvement, returned to society.
- SPECT: Overall improvement in CBF and perfusion.

Conclusion:

- Repeated insults and unrecognized.
- Multiple poisoning not shown by HbCO%.
- Long term firefighters receive SPECT after any intensive exposure and routinely each 3 years.

Richard A Neubauer, 2001

Introduction

- Frequent
  - 5000~8000 cases a year in France
  - 12000 fire-associated death a year in USA
- Severe
  - Delayed neurological squeal and death
- Under-diagnosed
  - 30% overlooked or misdiagnosed

Sources of Carbon Monoxide (1)

- **Endogenous**
  - End product of metabolism
    - Byproduct of Heme to biliverdin
  - Air pollution
    - < 0.001% in the atmosphere
  - Nonsmokers > : 1~3% HbCO
  - Smokers: 10~15% HbCO
- **Exogenous**
  - Taiwan:
    - Incompletely combusted gas leak from bath heater
    - Poorly function heating system

Beauty parlor workers poisoned by leaking gases during a long term service

Sources of Carbon Monoxide (2)

- **Exogenous :**
  - High risk workers:
    - Exposed to motor vehicle exhaust fumes
    - Fork lift trucks
    - Casting worker
    - Miner
    - Car repair shop worker
    - Mechanic
    - Fire fighter
    - Exposed to methylene chloride (paint remover), propane and methane (undergo more complete combustion, skin and lungs absorb and metabolized to CO in liver).

Number of Suicide Attempt Patients Causing a Fire (2006~2012)

	2006	2007	2008	2009	2010	2011	2012
Suicide	61	42	32	39	25	22	38

Number of CO Poisoning Sent to ER in Taiwan (2006~2012)

	2006	2007	2008	2009	2010	2011	2012
CO toxicity	919	921	1031	774	829	623	654

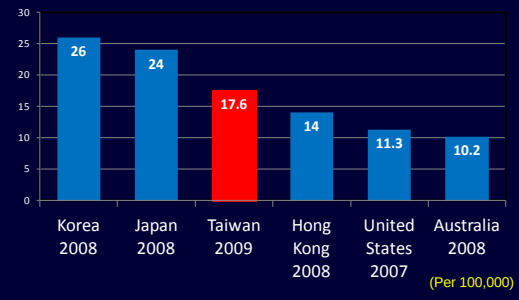
資料來源：內政部消防署

Chi Mei Medical Center: Smoke inhalation and gas toxicity

Year	CO Poisoning M	CO Poisoning F	Sum.	Smoke inhalation M	Smoke inhalation F	Total
2000、2001	12	5	17	1	0	18
2002	8	8	16	3	1	20
2003	17(82.4%)	15(66.7%)	32(75.0%)	74	14	120
2004	19(89.5%)	26(69.2%)	45(77.8%)	60	2	107
2005	65(75.4%)	71(74.6%)	136(75.0%)	30	2	168
2006	65(96.9%)	49(87.8%)	114(93.0%)	11	1	126
2007	62(87.1%)	30(96.7%)	92(90.2%)	24	0	116
2008	95(88.4%)	34(61.8%)	129(81.4%)	150	0	279
2009	34(76.5%)	44(86.4%)	78(82.1%)	5	0	83
2010	35(74.3%)	25(60.0%)	60(68.3%)*	4	0	64
2011	33(90.9%)	25(68.0%)	58(81.0%)**	1	1	60
2012	36(94.4%)	37(59.5%)	73(76.7%)***	5	2	80
總計	481(86.1%)	369(74.7%)	850(81.2%)			

* 2010年有97.6%自殺者使用燒碳方式，24.4%自殺者採燒碳及吞服不明藥物，使用呼吸器佔一氧化碳中毒之11.7%。
** 2011年有100%自殺者使用燒碳方式，36.2%自殺者採燒碳及吞服不明藥物，使用呼吸器佔一氧化碳中毒之3.4%。
*** 2012年有96.4%自殺者使用燒碳方式，28.6%自殺者採燒碳及吞服不明藥物，使用呼吸器佔一氧化碳中毒之11.0%。

A comparison of Crude Mortality Rate of Suicide Between Countries



Biochemical Effects of HBOT on CO Poisoning

- Colorless, odorless, and nonirritant toxic gas
- Affinity to Hb (COHb) : CO is 200~250x of O_2

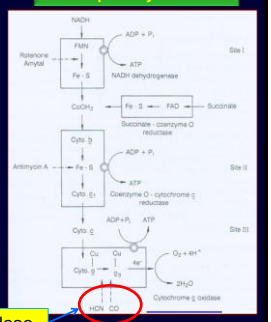
  Affinity to myoglobin (COMb) : 40x of O_2
- Toxicity:

  O_2-Hb dissociation curve shift to left ; resulting in impaired release of oxygen at the tissue level and cellular hypoxia

CO on Cytochrome a_3 Oxidase

- Toxicity: intracellular suffocation
  - ➢ Directly binding to mitochondria cytochrome a_3 oxidase to block ATP production
  - ➢ Direct CO–mediated damage at the cellular level
  - ➢ Most seriously damage to CNS

Schematic Mitochondrial Respirtaory Chain



Cytochrome a_3 Oxidase

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CO Poisoning

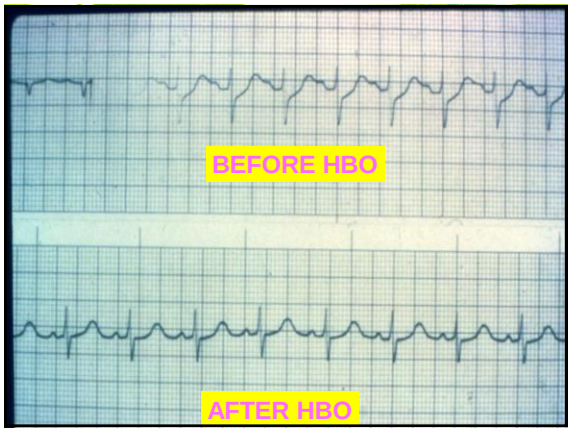
Clinical presentation (1)

- **Cardiovascular** system
  - ➢ Coronary vessel disease, hypotension, arrhythmia
- Respiratory
  - ➢ Pulonary edema, bronchospasm
- Nervous system
- Renal function
- Muscles: Rhabdomyolysis
- Skin: Erythema and blisters
- Endocrines
- Reproductive system
- Blood and hemorrhaeology

CO Poisoning

Clinical presentation (2)

- Tachycardia and tachypnea (compensation for cellular hypoxia)
- Headache, nausea, vomiting
- Presyncope, syncope and seizure (cellular hypoxia, cerebral vasodilatation→ cerebral edema)
- **Angina,** pulmonary edema and arrhythmias
- Classic finding: rare
  - ➢Cherry-red lips
  - ➢ Cyanosis
  - ➢ Retinal hemorrhage



CO Poisoning

Clinical presentation (3)

- **Headache (頭痛): 90%**
- **Nausea and vomiting (惡心嘔吐) : 50%**
- **Lethargy : 50%**
- **Altered conscious level (嗜睡) : 30%**
- **Subjective weakness (無力) : 20%**
- Rhabdomyolysis
- Parkinsonism and other movement disorders
- Implicated in a case of post-partum haemorrhage
- Arrhythmias
- Pulmonary oedema
- Hypotension
- Coma
- Exacerbation of existing disease
- Confusion
- Depression
- Ataxia
- Hearing problems

Houck PM. J Emergency Med, 1997

Degree of Severity of CO Poisoning, COHb Levels, and Clinical Features

Severity	COHb level	Clinical features
Occult	> 5%	No apparent symptoms, Psychological deficits on testing
	5-10%	Decreased exercise tolerance in P't with COPD Decreased threshold for angina and Increased threshold for visual stimuli
Mild	10-20%	Dyspnea on vigorous exertion Headache, dizziness, Impairment of higher cerebral function, Decreased visual acuity
Moderate	20-30%	Severe headache, irritability, impaired judgement Visual disturbance, nausea, dizziness, increased RR
Severe	40-50%	Fainting on exertion, Mental confusion
Very severe	60-70% > 70%	Coma, frequently fatal within a few minutes Immediately fatal

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CO Poisoning

Clinical presentation (4)

- **COHb levels don't correlate with the severity of symptoms in most cases**
- Duration of exposure: important factor mediating toxicity; CO environment exposure > 1 hour may increase morbidity
- Animal study show the symptoms can be minimal even with high level of COHb if no dissolved CO presents in the plasma
- **Administering HBO therapy shouldn't always undergo according to COHb level**

Diagnosis

- Clinical history
- Highly suspected by clinicians
- Check COHb alone maybe insufficient to rule out the diagnosis
- Detailed neurological examination
- CO neuropsychological screen battery
- CT is not helpful to diagnose CO poisoning, can use to rule out other brain conditions

Laboratory Diagnosis of CO Poisoning (1)

1. Determination of CO in the blood
2. Arterial blood gases and lactic acid levels
3. Screening tests for drug intoxication and alcohol intoxication
4. Biochemistry

   Enzyme: Creatine kinase, Lactate dehydrogenase, SGOT, SGPT, Serum glucose

Laboratory Diagnosis of CO Poisoning (2)

5. Myoglobin
6. Alkine phosphotase, CK total
7. Complete blood count
8. EKG
9. EEG
10. CT scan and MRI if needed
11. Neuropsychological testing

Work Plan of HBO Therapy

Hyperbaric Oxygen (HBO) versus Normobaric Oxygen		
Hyperbaric facilities available	COHb>25%	HBO
	COHb<25%	HBO if symptoms, NBO if none
No hyperbaric facilities	COHb>40%	Immediate referral to HBO center
	COHb<40% no symptoms	NBO
	COHb<40% /c symptoms	Referral to HBO center

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Normobaric vs Hyperbaric Oxygen

- CO elimination: minute ventilation
  duration of exposure
  FiO_2 if inspired oxygen
- Half-life of COHb:
  - room air 1ATA → 5 h 20 min
  - 100% O_2 1ATA →1 h 20 min
  - 100% O_2 3 ATA → 23 min

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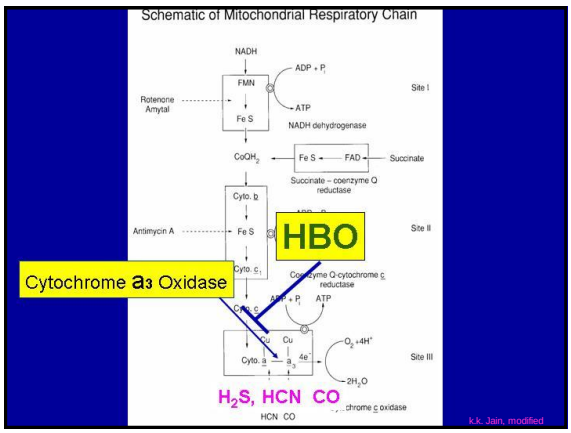


CO on Re-oxygenation

- Similar brain pathological lesions between post-ischemic reperfusion injury and CO poisoning
  - Lipid oxidation in rat brain
  - Conjugated diene and malonyldialdehyde ↑

    Thoms, 1990
  - Catalase and glutathione↓
  - Hydrogen perioxide and hydroxyl salicylate ↑

    Brown, 1992
- HBO ↓ Leukocyte adhesion by beta-2 intergrin expression

  Thom, 1993

Oxygen-Dependent Antagonism of Lipid Peroxidation

Thom SR, Elbuken ME
Free Radical Biol Med
10: 413-426 1991

Brain lipid peroxidation caused by CO is prevented by 100% oxygen at 3 ATA

A Brief Introduction to Hyperbaric Oxygen Therapy

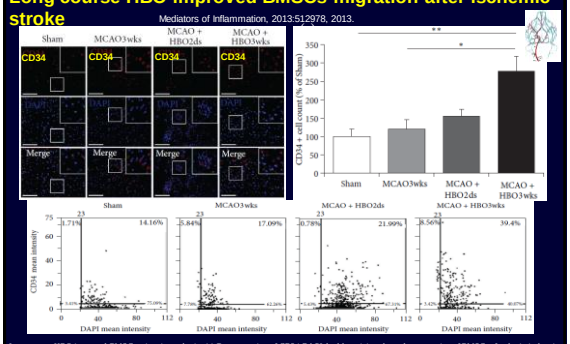
Mechanisms of Hyperbaric Oxygen Therapy (1)

1. To release hypoxia-edema induced vicious cycle by increasing tissue oxygen tension and blood flow.
2. To improve the wound healing and tissue repair.
3. To eliminate the secondary damage of ischemia-hypoxia injury in different organs.
5. To increase the bacteria killing capacity of neutrophil.
6. To provide bacteriostatic or bacteriacidal effect on anaerobic bacteria.

Mechanisms of Hyperbaric Oxygen Therapy (2)

5. To eliminate CO from blood.
6. To attenuate oxidative stress, inflammatory cytokines, but uprising anti- inflammatory cytokines in severe injury, catastrophic inflammatory response, and critical condition.
7. To improve mobilization of stem cells to an ischemic area for tissue regeneration.
8. To accelerate medication delivery to tissue by improving blood flow.

Long course HBO improved BMSCs migration after ischemic stroke



Long course HBO improved BMSCs migration to brain. (a) Demonstration of CD34-DAPI double staining showed presentation of BMSCs after brain ischemia. (b) Representative image of tissue cytometry using Tissue Quest software. CD-34 positive cells and DAPI positive cells were counted and the signal intensity was quantified. (c)The amount of double positive cells with CD34 and DAPI in the ischemic boundary was recorded as the percentage of CD34 positive cells in all cells. The difference was significant, as compared with MCAO3wks group and HBO3wks group (+p < 0.05). The difference was more significant, as compared with Sham group and HBO3wks group (++p < 0.01).

Guidelines for the Management of CO Poisoning

1. Remove patient from the site of exposure
2. Immediately administer oxygen, if possible after taking a blood sample for COHb.
3. Endotracheal intubation in comatose patients to facilitate ventilation
4. Removal of patient to HBO facility when indicated.
5. General supportive treatment: for cerebral edema, acid-base imbalance, etc.
6. Keep patient calm and avoid physical exertion by the patient.

k.k. Jain

Classification of Tissue Poisoning Where HBO Has Been Used Successfully

- **Action by combination with cytochrome a_3 oxidase and P-450**
  - Carbon monoxide
  - Hydrogen sulfide
  - Cyanide
- **Hepatotoxic ROS mediated by P-450**
  - Carbon tetrachloride
- **Drug-induced methemoglobinemias**
  - Nitrites
  - Nitrobenzene
- **Others**
  - **Quinine (vision)**
  - **Organophosphorus Compounds**
    - Paroxon (rabbits)
  - **Amentia Phalloides** (Mushroom)
  - **Ethacrynic Acid** (Otooxicity)

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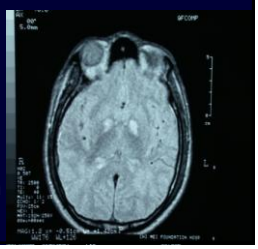
Delayed Neurological Syndrome

神經後遺症

- 3 days ~ 8 months after insults
- Prevalence: 10~40% Ginsberg, 1973
- Cause
  - Misdiagnosis Barret,1985
  - Inadequate therapy Smith, 1973
  - Delayed therapy Barois, 1979
- CT showed low density in globus pallidus



Delayed Neurological Syndrome (1)

- Delayed onset of neuropsychiatric symptoms about 3~240 days after recovery from the acute intoxication
- Occur in 10~30% of victims (even 67%) k.k. Jain
- In large clinical series
  - 50~75% DNS complete recovery (mild to moderate)
  - 25~50% DNS incomplete recovery (Severe)
    - Cognitive and
    - Personality changes
    - Parkinsonism
    - Incontinence
    - Dementia
    - Psychosis
    - Unsteady gait
    - Paralysis
    - Cortical blindness,
    - Vegetative state

Myers et al

Chronic CO Toxicity DNS

Pathophysiology and Mechanisms

I-R injury, inflammation and Oxidative Stress

- I-R injury brain 腦部缺氧-再灌流傷害
- Lipid peroxidation 脂肪過氧化傷害
- Platelets induced free radicals and NO^- 血小板異常引發氧游離基和一氧化氮增加

$$NO \rightarrow ONOO^- \xrightarrow[e^- \text{ transport}]{\text{Hemoprotein}} \text{mitochondria}$$

一氧化氮 粒線體傷害

- Oxidative stress and inflammatory responses 過氧化傷害及發炎反應

Delayed Neurological Syndrome

- Neurological and psychiatric presentation
  - Impaired cognition, poor memory, vertigo, ataxia, dementia, parkinsonism, muscle rigidity, gait disturbance, disorientation, mutism, urine and stool incontinence,
  - Cortical blindness, hearing loss, tinnitus, nystigmus
  - Depression, anxiety
- Pathology
  - **Demyelination**
    - Cerebral cortex
  - **Neuronal death**
    - Cortex, hippocampus, substantia nigra, globus pallidus

Chronic CO Toxicity DNS

Risk Factor

- The initial symptoms: unconsciousness
- Duration longer than one hr exposure
- Pre-existing cardiovascular disease
- Pre-existing CNS disease
- Older than 60 yrs
- Pregnancy

Delayed Neurological Syndrome

744 cases

• Group 0 Headache or nausea	100% O_2
• Group 1 NE abnormal	HBO
• Group 2 Consciousness loss	HBO
• Group 3 Comatose (GCS>6)	HBO
• Group 4 Deep comatose (GCS≤6)	HBO

Mathieu, 1985

Chronic CO Toxicity DNS

Clinical Symptoms

- Fatigue
- Headache
- Dizziness
- Flu-like illness
- Tearfulness
- Depression
- Agitation
- Anxiety
- Decreased memory, attention and concentration skill
- Poor reasoning
- Irritability
- Euphoria
- Overall personality changes

Chronic CO Toxicity *DNS*

Delayed Neuropsychological Sequelae

3 days to 8 months after the initial insult

- Impaired cognition
- Memory dysfunction
- Vertigo
- Ataxia
- Parkinsonism
- Muscle rigidity
- Gait disturbance
- disorientation
- Mutism
- Urinary incontinence
- Fecal incontinence
- Cortical blindness
- Hearing loss
- Tinnitus
- Nystagmus
- Seizure
- Coma
- EEG abnormality
- Cerebral edema
- Leukoencephalopathy
- Globus pallidus necrosis

Smoke Inhalation and Gases Toxicity

(52 cases)

A fire occurred in a pharmacy and science university

10 fire-fighting cars
1 chemicals supression car
88 fire fighters

- **Dizziness**
- **Headache**
- **Nausea**
- **Prechest fullness**
- **Cough**
- **Black sputum**
- **Burnt nose hair**

2003 • 3 • 6

Smoke Inhalation and Gases Toxicity

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- **Cough**
- **Black sputum**
- **Burnt nose hair**

A Gp:Remained CNS symptom noted 2~3 days later (29 cases)

B Gp:CNS symptom noted on the scene, but disappeared 2~3 days later with mild residual sign (5 cases)

CGp: Most CNS residual symptom disappeared 2~3 days later

Only mild respiratory discomfort (18 cases)

2003 • 3 • 6

Smoke Inhalation Injury and Gas Toxicity

Suspension of toxic gases, irritant gases, heat and combustion products

HEAT

- **Direct injury**
  - Dry cough and chest pain
  - Dyspnea
  - Pulmonary congestion
  - Alveoli layer slough

TOXIC

- **CO** **H_2S**
- **CO_2** **CN**
- **ORGANIC**
- **BENZENE**
  - Intracellular suffocation
  - Cerebral and whole body ischemia
  - Polychlorinated biphenyl, PCB
    - CNS
    - Endocrines
    - Sex hormones

IRRITANTS

- **HCl** **H_2**
- **Cl_2** **H_2SO_4**
- **AMONIA**
- **PHOSPHORIC**
  - Acute cough
  - Severe chest pain
  - Acute bronchospasm and dyspnea
  - Acute pul. congestion

PARTICLES

- **Burnt Carbon** adhere trachea and alveoli layers, suffocates and erode the epithelial cells
  - Burnt nose hair
  - Bloody secretion with black burnt particle
  - Dry cough with chest pain
  - Dyspnea

$C12HnCl(10-n)_2$

職業病通報系統

Smoke Inhalation Injury and Gas Toxicity

A fire occurred in a pharmacy and science university

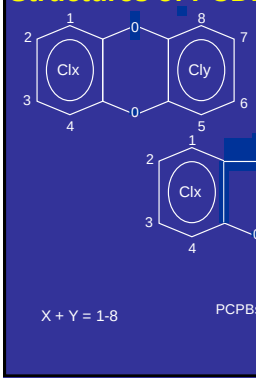
IRRITANTS
- HCl
- Cl_2
- H_2
- H_2SO_4
- AMONIA
- PHOSPHORIC.....

IRRITANTS and TOXIC
- Hexane,n- (正己烷)
- Methanol (甲醇)
- Ethanol (乙醇)
- Acetone (丙酮)
- Dichloromethane (二氯化甲烷)
- ..... .

- Skin
- Mucosa
- Respiratory
- CNS and endocrine

2003 · 3 · 6

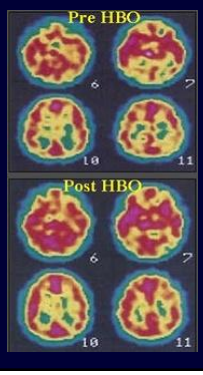
Structures of PCDDs, PCDFs, and PCPBs



Like Cholesterol
- Dioxin-like compounds
- Polychlorinated biphenyl, PCB

- CNS
- Endocrine
- Sex hormone

Exposure to Exhaust Fumes for 3 Months Prior to HBOT



- 20 y/o female
- Memory loss, confusion
- Poor coordination
- Inability to resolve problems
- Advised to seek psychiatric help
- Referred to HBOT Dr.

HBOT
- 69 sessions HBOTs
- SPECT: Improvement in CBF
- The patient was completely recovered

R.A. Neubauer, 2000

Factors Affecting the Prognosis of Patients with Delayed Encephalopathy after Acute Carbon Monoxide Poisoning

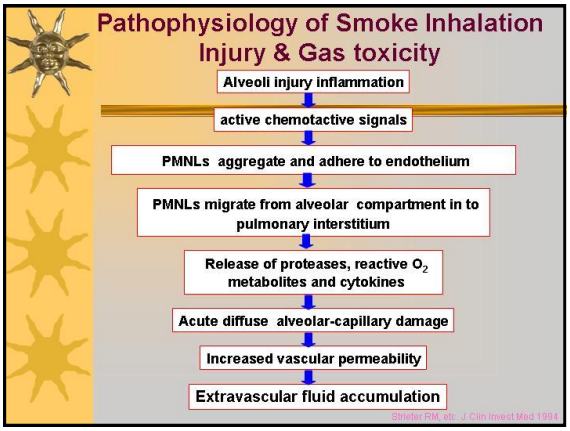
American Journal of Emergency Medicine (2011) 29, 261–264

Methods: In a retrospective study, 46 DEACMP patients

- A clear history of acute CO poisoning, a distinct "lucid interval," and neuropsychologic symptoms.
- First-time admittance to a hospital (within 1 week of onset of disease).
- Completion of standard treatment period. All patients received HBOT.

CONCLUSION:
- HBOT appears useful in treating DEACMP patients (50% : 23 / 46).
- Age, lucid interval, danger–activities of daily living scores, and complications were related ($P < .05$) to the prognosis.
- Whereas sex, fundamental diseases, HBOT in acute stage, intoxication time, unconsciousness duration, and GM1 ganglioside administration were not related to prognosis ($P > .05$).

Pathophysiology of Smoke Inhalation Injury & Gas toxicity

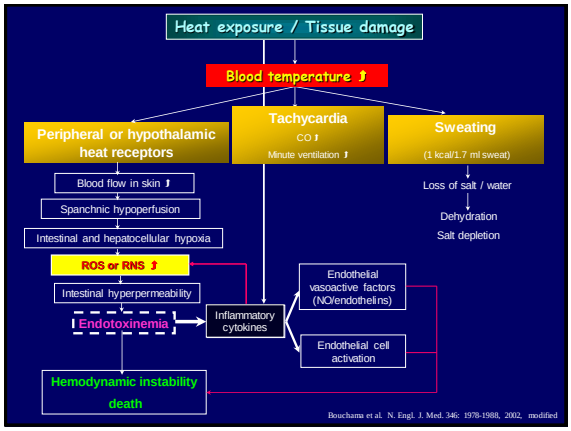


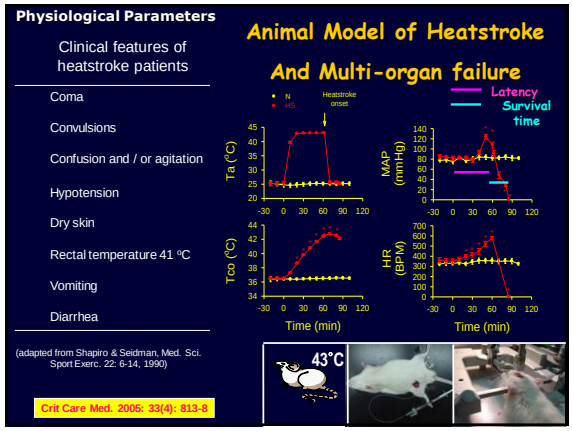
Strieter RM, etc. J Clin Invest Med 1994

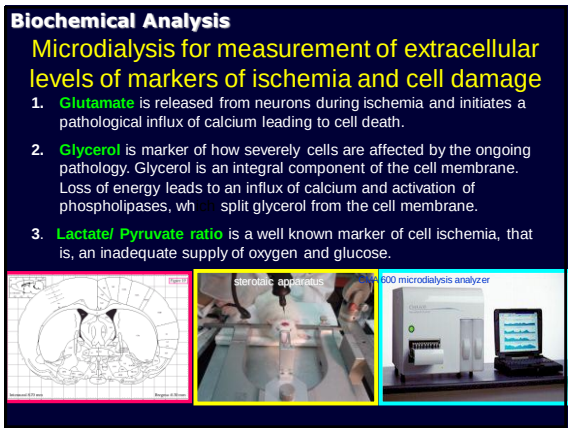
Unsuccessful local Inflammatory Response Induces Systemic Inflammatory Response

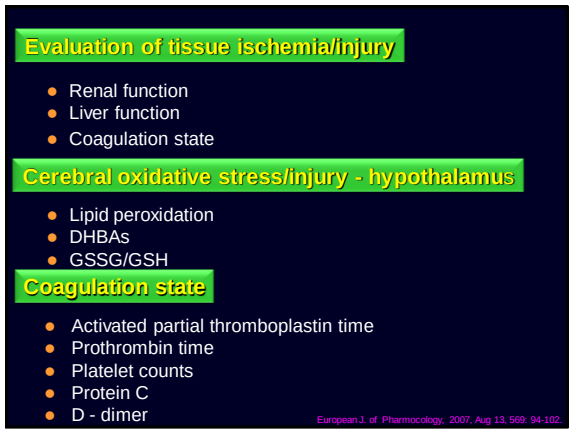
The Cytokine "Storm"

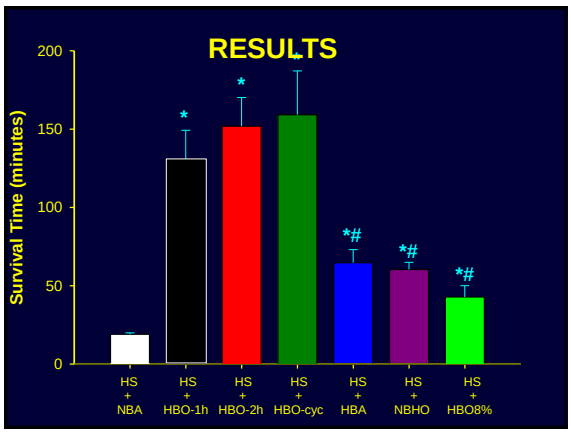
- The cytokine release leads to destruction rather than protection.
- The subsequent activation of numerous humoral cascades
- and subsequent loss of circulatory integrity.
- This leads to organ multi-organ dysfunction.

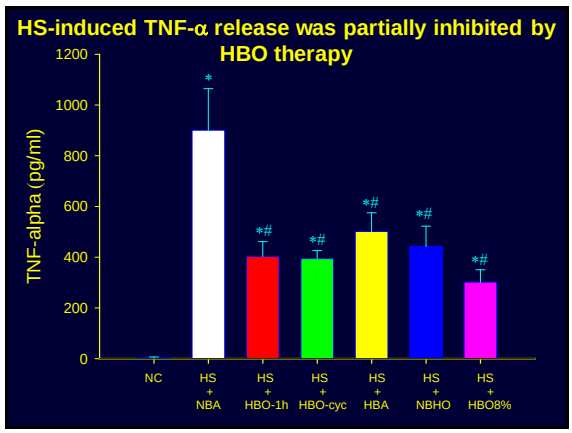

Heat exposure / Tissue damage
Blood temperature ↑
Peripheral or hypothalamic heat receptors
Tachycardia
CO ↑
Minute ventilation ↑
Sweating
(1 kcal/1.7 ml sweat)
Loss of salt / water
Dehydration
Salt depletion
Blood flow in skin ↓
Spanchnic hypoperfusion
Intestinal and hepatocellular hypoxia
ROS or RNS ↑
Intestinal hyperpermeability
Endotoxinemia
Inflammatory cytokines
Endothelial vasoactive factors (NO/endothelins)
Endothelial cell activation
Hemodynamic instability
death
Bouchama et al. N. Engl. J. Med. 346: 1978-1988, 2002. modified


Physiological Parameters
Clinical features of heatstroke patients
Coma
Convulsions
Confusion and / or agitation
Hypotension
Dry skin
Rectal temperature 41 ºC
Vomiting
Diarrhea
(adapted from Shapiro & Seidman, Med. Sci. Sport Exerc. 22: 6-14, 1990)
Crit Care Med. 2005; 33(4): 813-8
Animal Model of Heatstroke
And Multi-organ failure
Latency
Survival time
Heatstroke onset
Ta (ºC)
Tco (ºC)
MAP (mmHg)
HR (BPM)
Time (min)
43°C


Biochemical Analysis
Microdialysis for measurement of extracellular levels of markers of ischemia and cell damage
1. Glutamate is released from neurons during ischemia and initiates a pathological influx of calcium leading to cell death.
2. Glycerol is marker of how severely cells are affected by the ongoing pathology. Glycerol is an integral component of the cell membrane. Loss of energy leads to an influx of calcium and activation of phospholipases, wh split glycerol from the cell membrane.
3. Lactate/ Pyruvate ratio is a well known marker of cell ischemia, that is, an inadequate supply of oxygen and glucose.
stereotaic apparatus
600 microdialysis analyzer

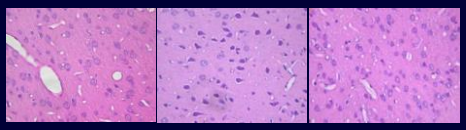

Evaluation of tissue ischemia/injury
Renal function
Liver function
Coagulation state
Cerebral oxidative stress/injury - hypothalamus
Lipid peroxidation
DHBAs
GSSG/GSH
Coagulation state
Activated partial thromboplastin time
Prothrombin time
Platelet counts
Protein C
D - dimer
European J. of Pharmocology, 2007, Aug 13, 569: 94-102.


RESULTS
Survival Time (minutes)
200
150
100
50
0
HS + NBA
HS + HBO-1h
HS + HBO-2h
HS + HBO-cyc
HS + HBA
HS + NBHO
HS + HBO8%


HS-induced TNF-α release was partially inhibited by HBO therapy
TNF-alpha (pg/ml)
1200
1000
800
600
400
200
0
NC
HS + NBA
HS + HBO-1h
HS + HBO-cyc
HS + HBA
HS + NBHO
HS + HBO8%

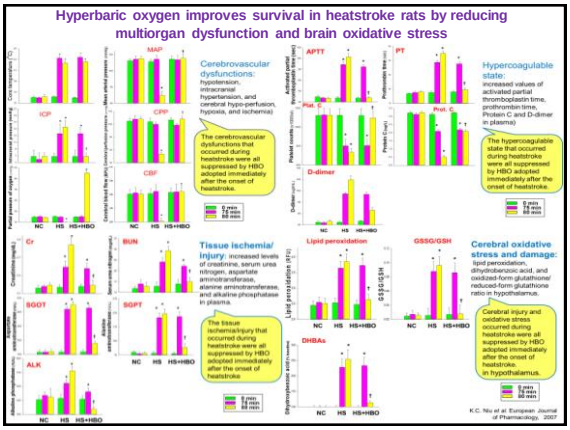
Histological examination of rat cortex by HE staining

NC | HS+NBA | HS+HBO

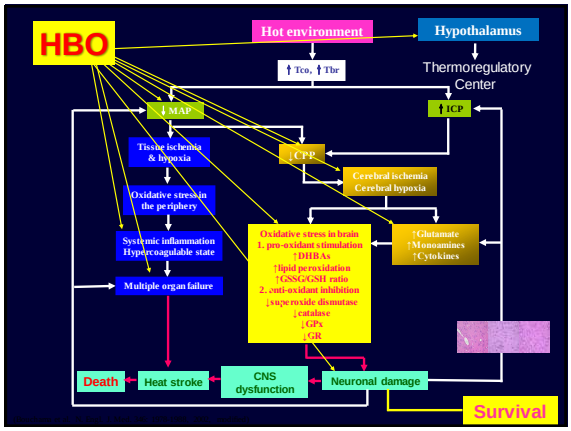


Treatment	Neuronal damage score (0 -3)
NC-treated rats at 26 °C	0 (0, 1)
NBA-treated rats at 43 °C	2 (2, 2)*
HBO-1h-treated rats at 43 °C	1 (0, 1)†
HBO-cyc-treated rats at 43 °C	1 (0, 1)†

Hyperbaric oxygen improves survival in heatstroke rats by reducing multiorgan dysfunction and brain oxidative stress



HBO



A Heatstroke Patient treated by HBOT

- Vital sign became stabilized rapidly.
- The patient regained consciousness at 48 hrs of HBOT.
- The ventilation was removed in three days.

Conclusion

- HBO can protect the CNS, cardiovascular system and multiple organs from heat-induced injury through the possible mechanisms of **attenuating levels of cytokines (TNF-α) and cerebral oxidative stress and subsequent multiorgan failure**.

- Cerebral ischemia
- Sepsis
- Burns
- Critical disorders induced multi-organ failure
- Crushing injury & other ischemia-reperfusion inj.
- Preventive medicine: inflammation related arteriosclerosis, arthritis, and post major operation

→

- Critical care medicine
- Emergency medicine
- Toxicology
- Neurology
- Neurosurgery
- Traumatology
- Burns
- Others