SITOX 1972

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Società Italiana di Tossicologia

Intossicazione da monossido di carbonio in età pediatrica

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Da inizio novembre quasi un intossicato al giorno da monossido di carbonio: è emergenza per il caro energia

Sempre più persone sono costrette ad usare sistemi alternativi di riscaldamento, che possono però esser pericolosi

di **Redazione** - 07 Dicembre 2022 - 11:32

1 Commento Commenta Stampa
Invia notizia 3 min

Più informazioni su ravenna

https://www.ravennanotizie.it/cronaca/2022/12/07/da-inizio-novembre-quasi-un-intossicato-al-giorno-da-monossido-di-carbonio-e-emergenza-per-il-caro-energia/

Vivevano nel pavese, trovati morti dopo 4 giorni

Famiglia egiziana uccisa dal monossido di carbonio

I corpi senza vita della donna incinta all'ottavo mese, del marito e dei due figli della coppia, di 3 e 4 anni, trovati nell'appartamento dove vivevano a Landriano, vicino a Pavia. La morte della famiglia egiziana risalirebbe a 4 giorni fa.

La casa della famiglia a Landriano

Pavia, 05 Gennaio 2011

Verra' aperta un'inchiesta per omicidio colposo plurimo sulla morte della famiglia composta da madre, incinta, padre e due bambini di 4 e 3 anni, sterminati dal monossido di carbonio a Landriano, in provincia di Pavia.

Secondo quanto si apprende, la procura vorra' fare luce sulla regolarita' degli impianti e sui controlli e sulle manutenzioni condotte sul sistema di riscaldamento del bilocale in via Cirano.

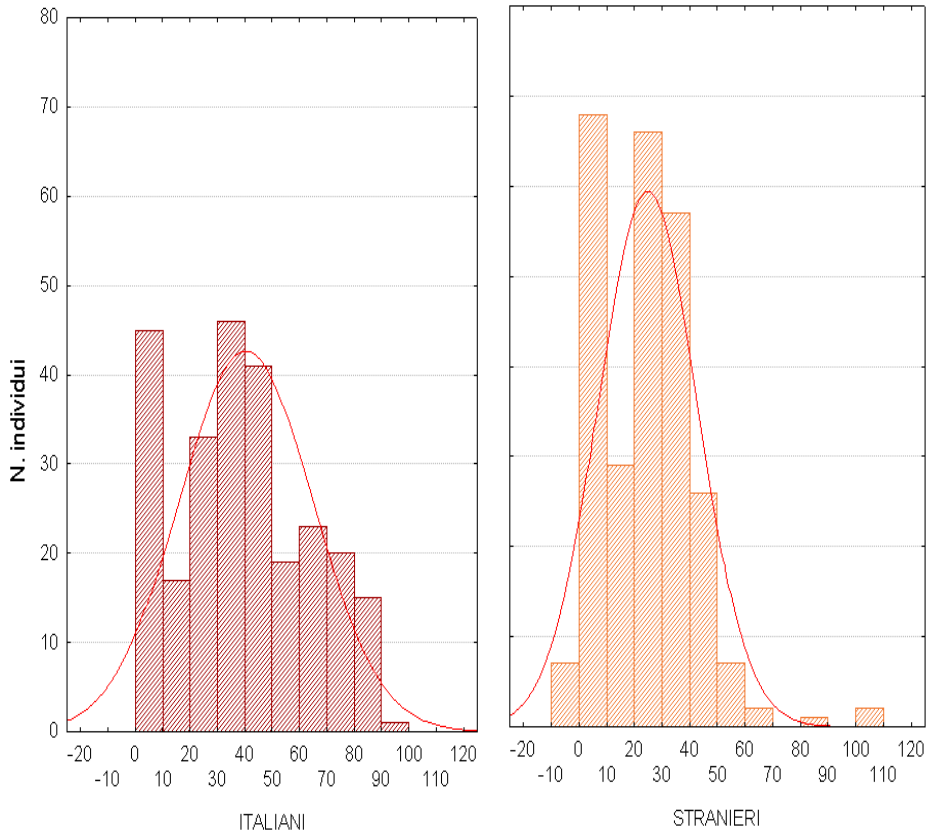
La famiglia, che viveva a Landriano da alcuni anni, era in regola con il permesso di soggiorno grazie all'impiego dell'uomo che lavorava in un magazzino. All'esterno dell'abitazione si sono radunati gli egiziani residenti nella zona.

Intossicazioni da CO

popolazione di origine italiana e quella immigrata - *confronto*

n=618 (2 anni di casisistica CAV Pavia)

Tab.1	stranieri	Italiani
impianti difettosi	26.4%	73.6%
stufe	60.5%	39.5%
bracieri	68.67%	31.32%



barriere linguistiche: adattamento degli strumenti valutativi usualmente utilizzati per l'inquadramento clinico anamnestico sia nella fase acuta che nella valutazione delle sequele a lungo termine, *nei bambini* come negli adulti

Regione Toscana

Centro Antiveleni Firenze
Centro Regionale di riferimento

IL MONOSSIDO DI CARBONIO PUÒ UCCIDERE!
CARBON MONOXIDE CAN KILL!
LE MONOXYDE DE CARBONE PEUT TUER!
أول أكسيد الكربون يمكن أن يقتل
一氧化碳可致人死亡!

Il **monossido di carbonio** è un gas tossico, incolore ed inodore prodotto dalla combustione di carbone, legna, benzina e altri combustibili.

ATTENZIONE!
- Non accendere bracieri a carbone o legna all'interno dell'abitazione!
- Utilizza stufe solo in locali ben areati, no in camere da letto, bagni e garage!
- Fai controllare stufe a carbone, a gas, a legna, caldaie, boiler, cucine, camini!
- Controlla che nei locali riscaldati ci siano prese d'aria!

Carbon monoxide is a colourless and odourless toxic gas that is produced by burning coal, wood, petrol and other fuels.

WARNING!
- do not light charcoal or wood burners inside the home!
- only use stoves in well-ventilated rooms and never in bedrooms, bathrooms or garages!
- have your charcoal, gas and wood-burning stoves, boilers, kitchens and fireplaces inspected!
- make sure that air vents are present in heated rooms!

Le **monoxyde de carbone** est un gaz toxique, incolore et inodore qui résulte de la combustion du charbon, du bois de chauffage, de l'essence et d'autres combustibles.

ATTENTION!
- n'allumez pas de brasero au charbon ou au bois à l'intérieur de la maison!
- utilisez des poêles uniquement dans des pièces bien aérées, pas dans les chambres à coucher, salles de bains et garages!
- faites vérifier les poêles à charbon, à gaz, à bois, les chaudières, les chauffe-eau, les cuisines et les cheminées!
- vérifiez la présence de bouches d'aération dans les pièces chauffées!

أول أكسيد الكربون غاز سام بدون لون أو رائحة وينتج عن احتراق الكربون والخشب والبنزين و غيرها من المواد القابلة للاحتراق

تحذير

لا تشعل مواقد الفحم أو الخشب داخل المساكن .

استخدم الدفايات فقط في الأماكن جيدة التهوية وليس في غرف النوم أو الحمامات أو الجراج .

احرص على فحص الدفايات التي تعمل بالفحم أو الغاز أو الخشب والسخانات و الغلايات و الأفران و المواقد .

تأكد من وجود مخارج للهواء في الأماكن التي بها تدفئة .

一氧化碳是一种煤炭、木材、汽油或其他可燃材料燃烧时释放出的无色、无味且有毒的气体。

注意!
- 切勿在屋内燃烧炭火盆!
- 仅在通风良好的室内点燃火炉,严禁在卧室、卫生间和车库内使用火炉!
- 对炭炉、煤气炉、木柴炉、燃烧炉、蒸煮器具、厨房和烟囱进行严格的检查!
- 检查加热区内是否存在通气口!

I primi sintomi di un'intossicazione sono
The first symptoms of poisoning are
Les premiers symptômes d'une intoxication sont les suivants
الأعراض الأولى لوقوع تسمم هي:
一氧化碳中毒后的前期反应包括

mal di testa/vertigini
headache/dizziness
maux de tête/étourdissements
الام في الرأس/ دوخة
头痛/头晕

palpitazioni
palpitations
palpitations
نبضان
心悸

nausea/vomito
nausea/vomiting
nausées/vomissements
غثيان/قيء
恶心/呕吐

confusione mentale
mental confusion
confusion mentale
تشوش ذهني
神志不清

disturbi della vista
impaired vision
troubles visuels
اضطرابات في الرؤية
视觉障碍

affanno
breathlessness
essoufflement
ضيق تنفس
呼吸急促

Se sei incinta ricordati che il monossido di carbonio può essere un serio pericolo anche per il tuo bambino!
If you are pregnant, remember that carbon monoxide can also seriously harm your baby!
Si vous êtes enceinte, rappelez-vous que le monoxyde de carbone peut constituer un grave danger également pour votre bébé!
في حالة الحمل، يرجى تذكر أن أول أكسيد الكربون يمكن أن يشكل خطرًا حقيقيًا على طفلك
若出现上述反应,请务必记得一氧化碳可能是让您和您的孩子面临一系列危险的原因!

Cosa fare in caso di pericolo:
- aprire porte e finestre
- spegnere gli apparecchi a combustione
- chiamare i servizi d'urgenza

What to do in the event of danger:
- open doors and windows
- turn off the combustion appliances
- call the emergency services

Que faire en cas de danger :
- ouvrir les portes et fenêtres
- éteindre les appareils à combustion
- appeler les services d'urgence

إجراءات يلزم عمله عند وقوع خطر
- فتح الأبواب والنوافذ .
- إطفاء الأجهزة التي تعمل بالاشتعال .
- طلب خدمات الطوارئ .

应急操作:
- 打开门窗
- 立即熄灭燃烧装置
- 拨打急救电话

Emergenza Sanitaria 112

Centro Antiveleni 055 7947819 — H24
Tossicologia Perinatale 055 7946731 — tutti i giorni 9.00 - 19.30

Ottobre 2022 – Newsletter SIMEUP
Gruppo Tossicologia Clinica

Società Italiana di Medicina di Emergenza e Urgenza Pediatrica

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I pericoli del Monossido di Carbonio

Dal Gruppo di studio Tossicologia

L'inizio della stagione fredda porta con sé i pericoli derivanti da un nemico subdolo che si insidia nelle nostre abitazioni: il monossido di carbonio.

Purtroppo ogni anno si registrano casi letali di intossicazione da monossido di carbonio che possono coinvolgere interi nuclei familiari.

L'attuale criticità di fornitura del gas è ulteriore motivo di allarme sui pericoli di intossicazioni derivanti dall'uso improvvisato di sistemi di riscaldamento casalinghi.

Per questo SIMEUP ritiene indispensabile ricordare a tutti i cittadini i pericoli del monossido di carbonio e gli accorgimenti da adottare per prevenire possibili intossicazioni.

COS'E' Il MONOSSIDO DI CARBONIO?

Il monossido di carbonio (CO) è un gas inodore, incolore e non irritante che si forma per combustione incompleta di sostanze organiche (carbone, legna, gas combustibile, benzina, ecc.). Negli ambienti di vita viene prodotto in quantità rilevanti dal malfunzionamento di caldaie, scaldabagni, caminetti, bracieri e stufe di ogni tipo: il rischio di intossicazione è ancora più elevato in ambiente confinato o poco areato.

COME CI ACCORGIAMO DEL PERICOLO ?

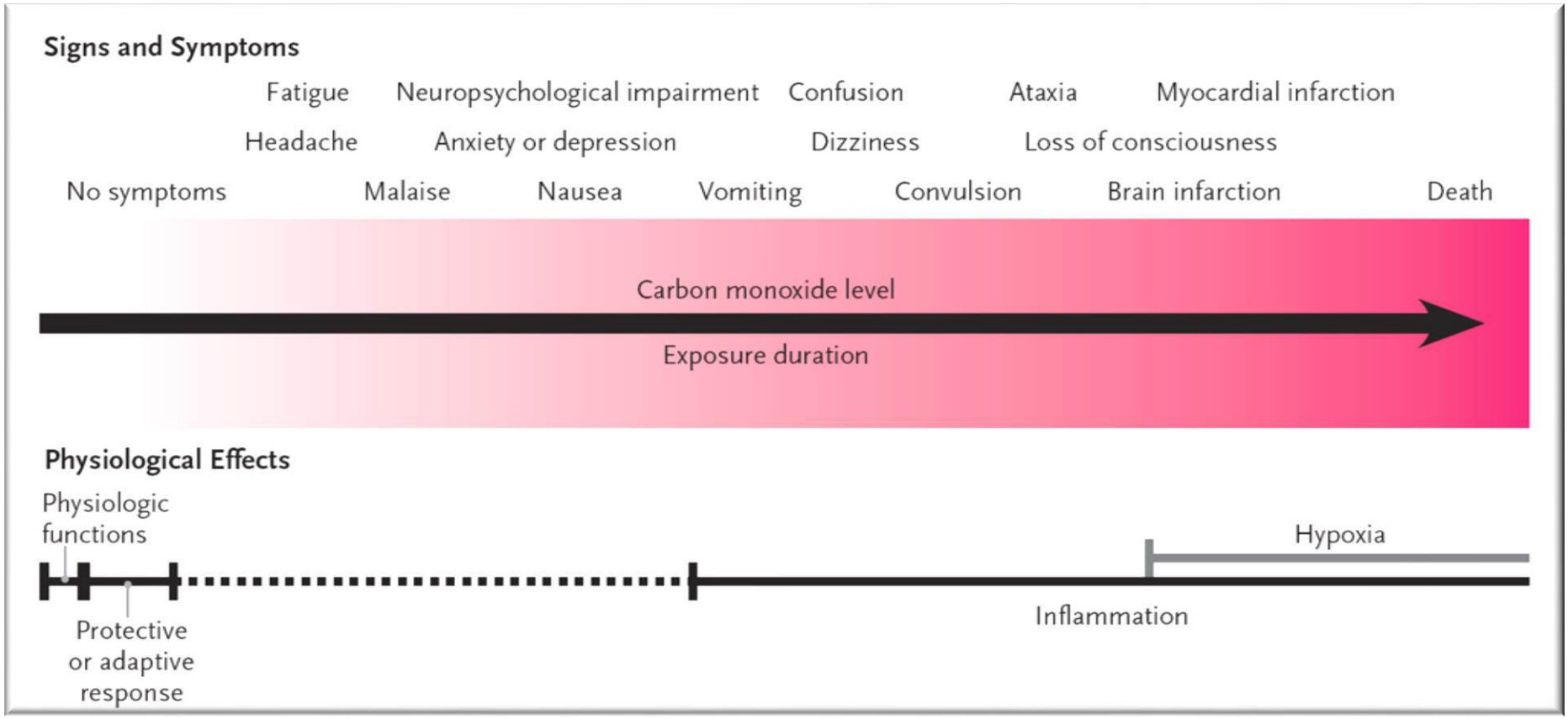
I primi sintomi dell'intossicazione da CO sono aspecifici e usualmente cefalea, vertigini e/o manifestazioni gastroenteriche quali nausea e vomito. Il quadro clinico può complicarsi fino alla manifestazione di convulsioni. I bambini e le gravide sono a maggior rischio per gli effetti tossici dell'avvelenamento.

Si deve sospettare l'esposizione a CO quando i sintomi sopra indicati sono presenti in più persone che vivono nello stesso ambiente.

COSA DOBBIAMO FARE IN CASO DI INTOSSICAZIONE ?

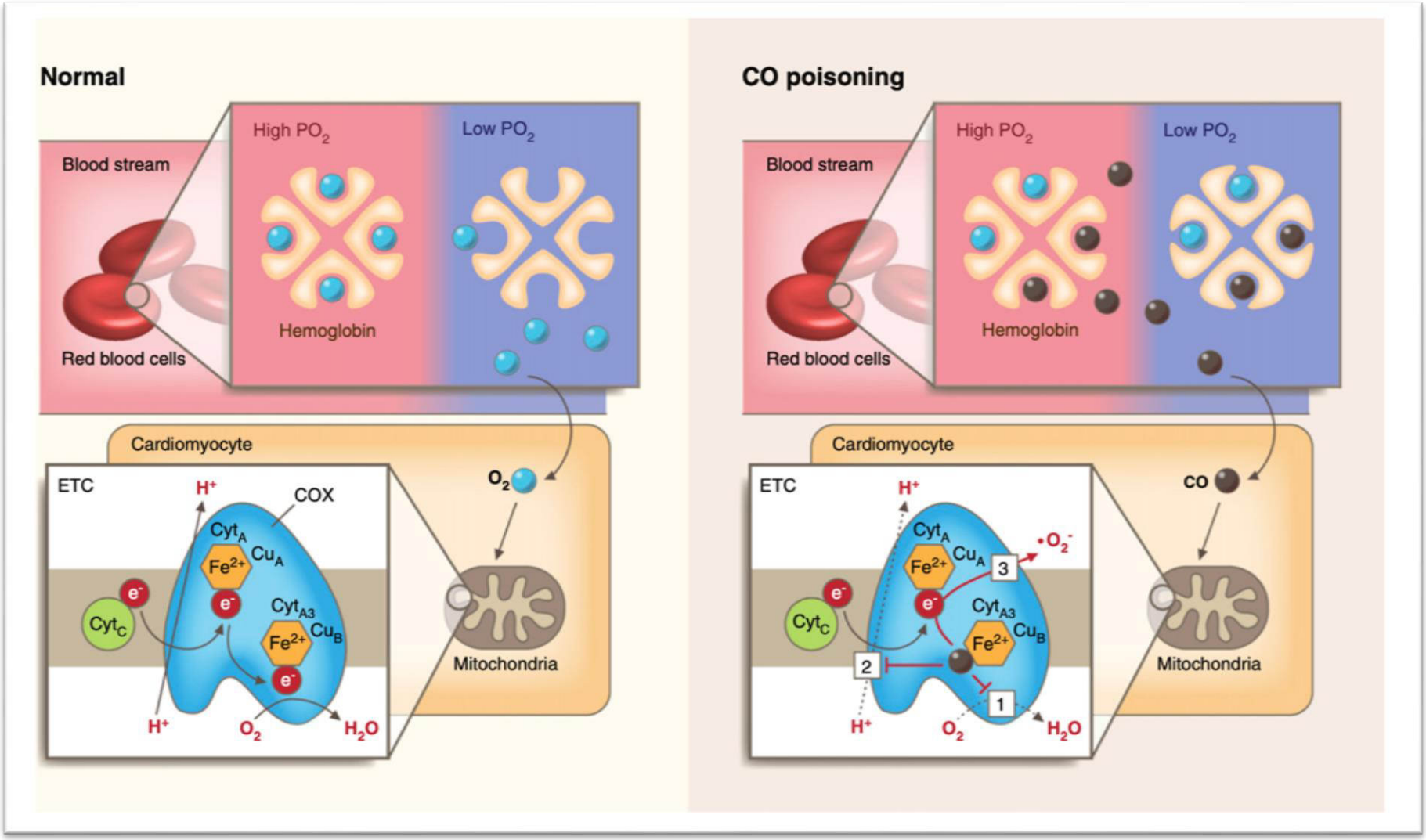
https://simeup.it/i-pericoli-del-monossido-di-carbonio/

Clinical and physiological effects of CO



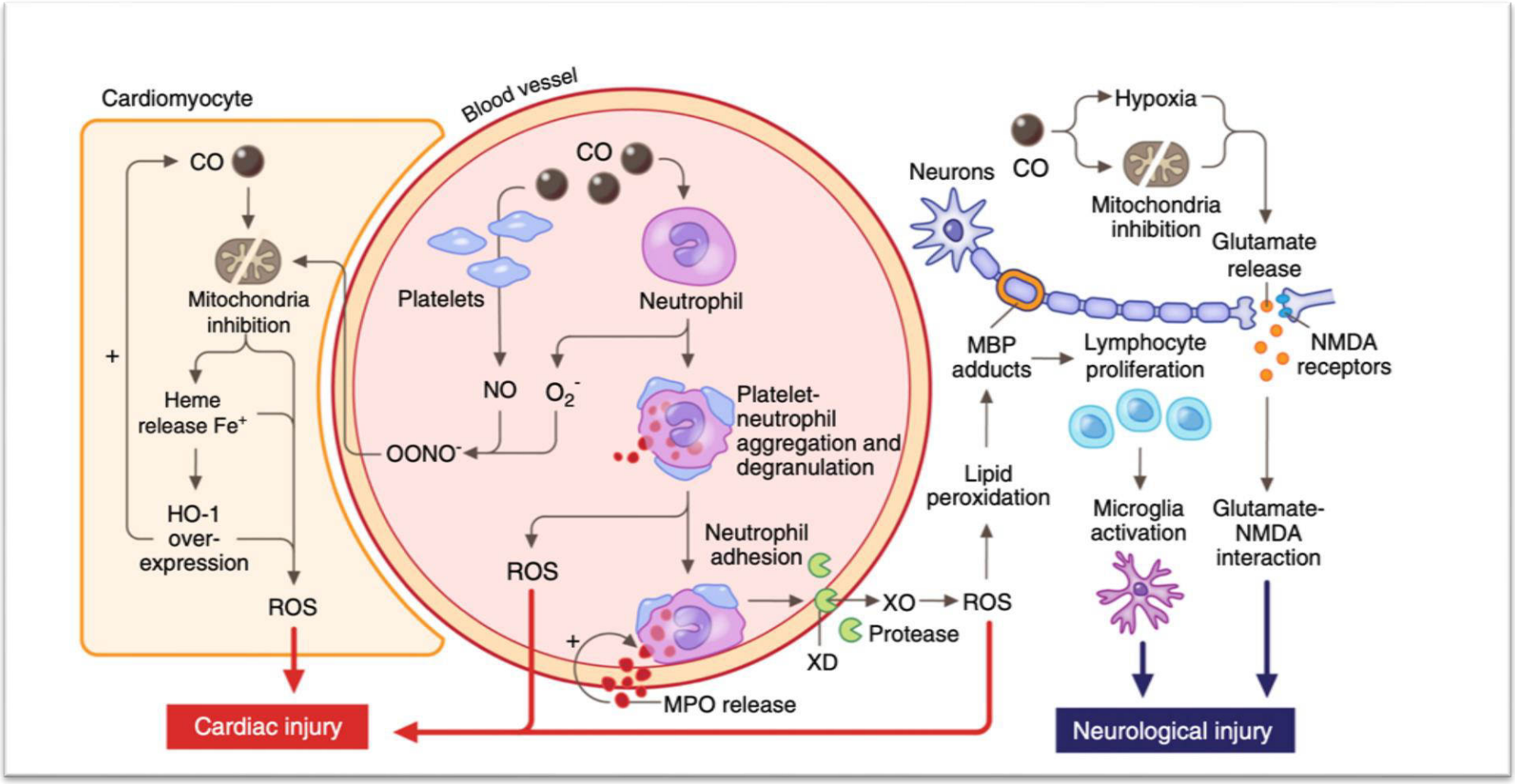
Weaver, 2009

Hemoglobin (Hb) and mitochondrial effects of CO



Rose JJ et al., Am J Respir Crit Care Med,

Inflammatory mechanisms of CO toxicity



Rose JJ et al., Am J Respir Crit Care Med,

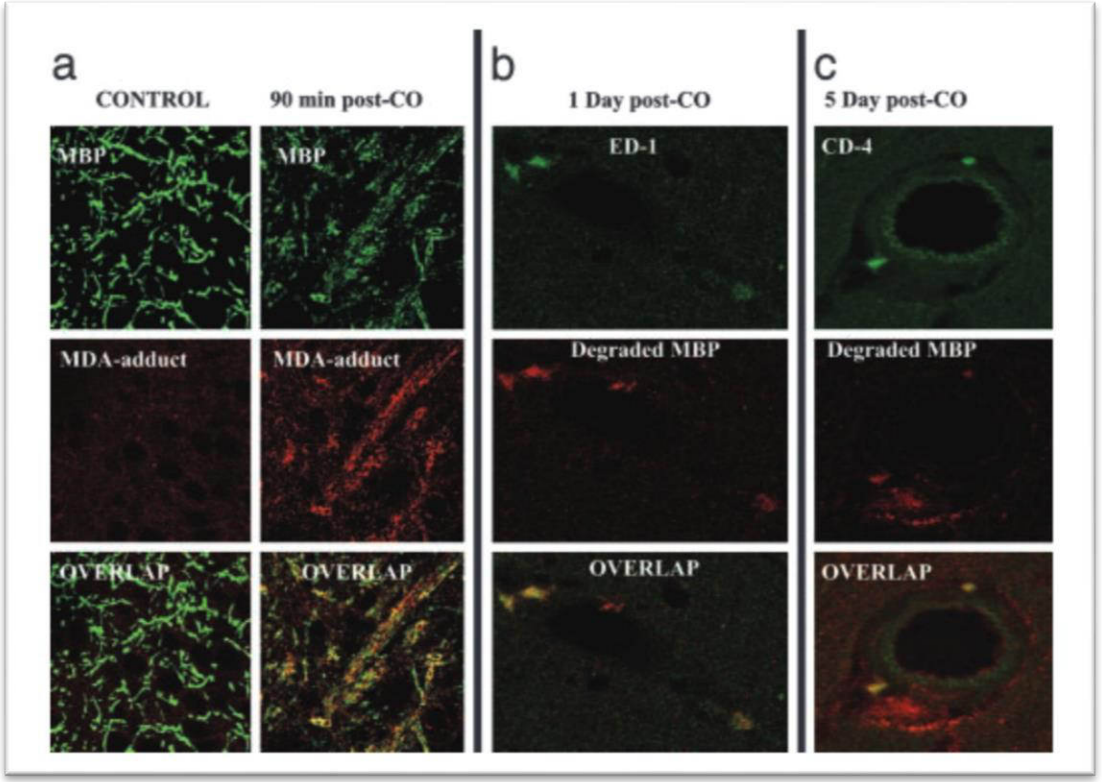
Delayed neuropathology after carbon monoxide poisoning is immune-mediated

Stephen R. Thom*†‡, Veena M. Bhopale*, Donald Fisher*, Jie Zhang*, and Phyllis Gimotty§

*Institute for Environmental Medicine, †Department of Emergency Medicine, and §Department of Biostatistics and Epidemiology, University of Pennsylvania Medical Center, Philadelphia, PA 19104-6068

Communicated by Robert E. Forster, University of Pennsylvania School of Medicine, Philadelphia, PA, August 3, 2004 (received for review February 4, 2004)

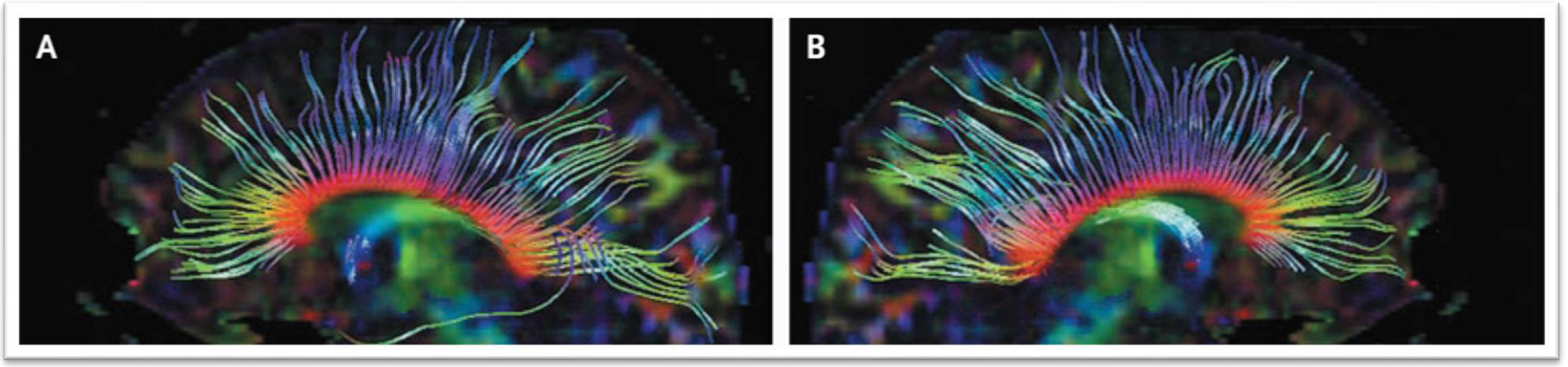
The neuropathological sequelae of carbon monoxide (CO) poisoning cannot be explained by hypoxic stress alone. CO poisoning also causes adduct formation between myelin basic protein (MBP) and malonylaldehyde, a reactive product of lipid peroxidation, resulting in an immunological cascade. MBP loses its normal cationic characteristics, and antibody recognition of MBP is altered. Immunohistochemical evidence of degraded MBP occurs in brain over days, along with influx of macrophages and CD-4 lymphocytes. Lymphocytes from CO-poisoned rats subsequently exhibit an autoreactive proliferative response to MBP, and there is a significant increase in the number of activated microglia in brain. Rats rendered immunologically tolerant to MBP before CO poisoning exhibit acute biochemical changes in MBP but no lymphocyte proliferative response or brain microglial activation. CO poisoning causes a decrement in learning that is not observed in immunologically tolerant rats. These results demonstrate that delayed CO-mediated neuropathology is linked to an adaptive immunological response to chemically modified MBP.



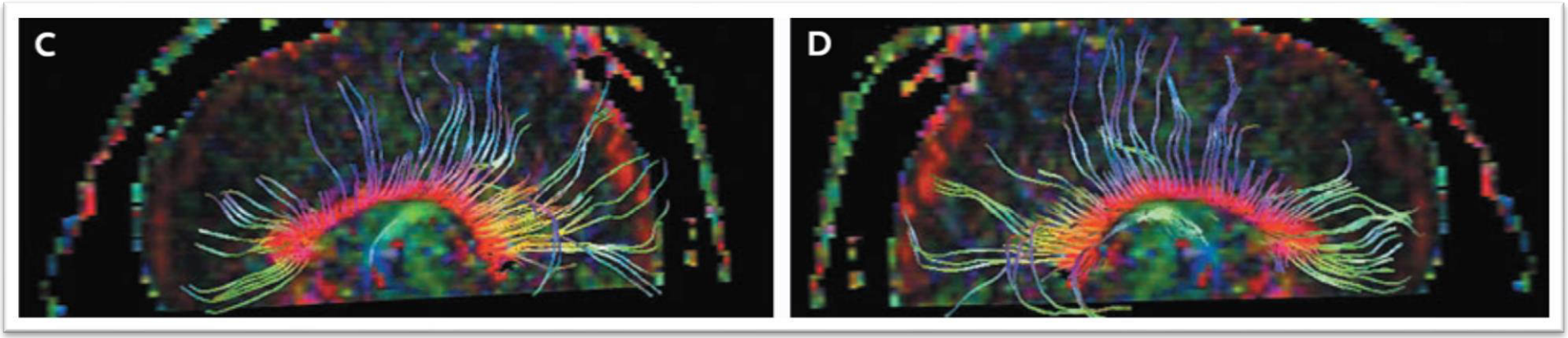
Thom SR et al, 2004

Highly sensitive 3-T MRI

21-years-old normal volunteer



age-matched patient with hystory of CO poisoning



Weaver, 2009

Lesson of the Week

Carbon monoxide poisoning in childhood

F GEMELLI, R CATTANI

Every winter the prolonged use of poorly maintained gas stoves in badly ventilated houses gives rise to several cases of carbon monoxide poisoning. The formation of this gas is a severe hazard because of its affinity for haemoglobin, which is about 300 times more than that for oxygen.[1] Moreover, carbon monoxide shifts to the left the dissociation curve of any oxyhaemoglobin that remains—and hence the liberation of oxygen is further reduced. The resultant cellular anoxia affects those organs that are particularly susceptible to ischaemic damage, such as the central nervous system and the heart. In adults symptoms and signs include a red complexion, frontal headache, dizziness, semiconsciousness, and chest pains.[2] The diagnosis is facilitated by the presence of a similar syndrome in people living in the same house and by the results of investigating its heating apparatus. Nevertheless, a particular problem in childhood is that the features may principally affect the bowel, at a time when, because of their higher tolerance, the adults living in the same house are showing no features of poisoning.

Here we describe three children erroneously treated for gastroenteritis who were eventually found to be suffering from carbon monoxide poisoning.

Case histories

Case 1—A 7 year old boy who had previously been admitted to the paediatric department with attacks of asthma and convulsions presented with a two day history of sickness, abdominal pain, and headache and eight hours' history of vomiting and fever. The next day his symptoms got worse and the other inhabitants of the house began to complain of headache and malaise. On admission the child's general condition was satisfactory and gastroenteritis was diagnosed; however, on the same day his father was admitted to hospital, where his blood carbon monoxide concentration was found to be 23%. Thereafter analysis of the child's blood showed a carboxyhaemoglobin concentration of 16%. Investigation showed that the kitchen of their house was heated by a gas stove that, because of the extremely cold weather, was working throughout the 24 hours. Though the stove was not defective, its draught was badly regulated and the incomplete combustion of household gas in a confined room gave rise to the formation of carbon monoxide.

Case 2—A 9 year old girl who had complained of headache for two days began to vomit and developed fever. On admission to hospital with a diagnosis of gastroenteritis the findings on physical examination were normal. Again, the results of routine investigations, including full blood count, electrolyte concentrations, and transaminase activities, were normal. Her father was admitted to hospital and found to have a carboxyhaemoglobin concentration of 25%. For this reason gas analysis of the child's blood was performed and showed a carboxyhaemoglobin concentration of 18%. The house was heated by a gas stove, and more detailed investigations showed that carbon monoxide was present when the stove was alight.

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F GEMELLI, MD, assistant
R CATTANI, MD, assistant
Correspondence to: Dr Gemelli.

In children apparently suffering from a gastrointestinal illness of obscure cause consider monoxide poisoning

Case 3—A 7 year old boy who had had diarrhoea and vomiting for several days did not improve with routine treatment. Examination showed patchy erythema on his chest and back and for this reason gas analysis was performed on a blood sample; this showed a carboxyhaemoglobin concentration of 32%. A boiler that was found to be defective was situated in a room communicating with the house. It had worked intermittently for 20 days, and further inquiries by the local health authorities showed that the combustion of household gas was incomplete. Three other people living in the same house were subsequently admitted with a carboxyhaemoglobin concentration even higher than that of the boy (40%).

Discussion

Little has been written about carbon monoxide poisoning in children, yet its manifestations are often so different from those in adults that it is important to emphasise the differences. In our cases the features resembled those of gastroenteritis and deceived the practitioner about the true nature of the illness. We found that the carboxyhaemoglobin concentrations of three children were considerably lower than those of 12 adults (22% *v* 35·6%), so that children must be particularly susceptible to carbon monoxide poisoning. These two features—the different symptoms and the earlier presentation—often make it difficult to diagnose the condition in childhood.

References

1 Haltuber MJ, Kürchmair H, de Luterotti A. *Vademecum di diagnostica e terapia d'urgenza*. Padua: Piccin Editore, 1974.
2 Barrès A, Grosbois S, Goulon M. Les intoxications aiguës par l'oxyde de carbone et les gaz de chauffage. *Rev Prat* 1979;29:1211-31.

A baby born with myelomeningocoele has hyperextension of the knees, hyperflexion of the hips, and club feet. What treatment is advised?

A child with myelomeningocoele and with these deformities will almost certainly have a neurological lesion at the L3/4 level. This results in unopposed activity of the hip flexors, adductors, and knee extensors. There may or may not be activity in the tibialis anterior. The hips will probably be dislocated, or will dislocate during the first few years of life as a result of the muscle imbalance. Management will require extensive orthopaedic care including multiple operations, but with this muscle pattern it should eventually be possible to achieve worthwhile walking with below knee calipers. A prolonged course of orthopaedic management is probably justified provided that the general condition of the child warrants such treatment. Details of the specific surgery and physiotherapy are well known to orthopaedic surgeons caring for these children.—GEOFFREY WALKER, consultant orthopaedic surgeon, Carshalton.

Elementi circostanziali che orientano alla diagnosi.

Criteri	Elementi da ricercare
Presenza di una fonte d'esposizione	• Presenza di fonti di produzione di CO in locali dell'abitazione (per es. stufe, scaldabagni, camini, caldaie, bracieri), non necessariamente malfunzionanti; motori (per es. veicoli, taglia-erba, generatori e pompe) tenuti accesi in ambiente confinato • Provenienza dei pazienti da ambienti confinati (per es. pista di pattinaggio, abitacolo dell'automobile) o da situazioni ambientali a rischio (motoscafo fuoribordo, viaggio su cassone di camion o di *pick-up*) • Occupazioni a rischio (per es. vigili del fuoco, vigili urbani, garagisti, autisti, lavoro in galleria)
Condizioni favorenti	• Condizioni atmosferiche (per es. forte vento) ostacolanti la fuoriuscita dei fumi dai camini • Stagionalità (si tratta di intossicazione prevalentemente invernale)
Criterio epidemiologico	• Interessamento contemporaneo di più soggetti di uno stesso nucleo familiare, anche se i quadri di presentazione possono essere multiformi nello stesso gruppo di pazienti • Insorgenza precoce della sintomatologia nei bambini, in coloro che trascorrono più tempo nei locali maggiormente a rischio o nei piccoli animali domestici • Reiterazione di una sintomatologia simil-influenzale, di scompenso cardiaco, di sincope • Miglioramento della sintomatologia al di fuori di un determinato ambiente

Emergency Care Journal, 2017

EB MEDICINE

PEDIATRIC EMERGENCY MEDICINE PRACTICE

AN EVIDENCE-BASED APPROACH TO PEDIATRIC EMERGENCY MEDICINE ▲ EBMEDICINE.NET

An Evidence-Based Approach To Pediatric Carbon Monoxide Poisoning

September 2011
Volume 8, Number 9

Author

Abby M. Williams, MD
Clinical Fellow, Division of Pediatric Emergency Medicine, Department of Pediatrics, Monroe Carell Jr. Children's Hospital at Vanderbilt, Nashville, TN

Società Italiana di Tossicologia

Position Paper sulla gestione delle intossicazioni acute da CO nel bambino: consenso multidisciplinare di esperti.

Nonostante l'intossicazione da monossido di carbonio (CO) sia molto studiata, molti aspetti riguardanti la diagnosi e il trattamento risultano ancora dibattuti. La gestione clinica dei pazienti intossicati è spesso differente in varie realtà del nostro paese. Aspetti cruciali quali criteri diagnostici, classificazione della gravità, indicazioni e durata del trattamento con ossigenoterapia iperbarica, tempi e modalità del follow-up clinici, non vengono applicati in maniera uniforme.

Tali aspetti sono ancora più controversi proprio in caso di intossicazione pediatrica.

Ad oggi, non sono disponibili raccomandazioni condivise e validate dalle società scientifiche di settore che possano essere di riferimento per le figure professionali coinvolte nella gestione del bambino con intossicazione acuta da CO.

L'obiettivo di questo progetto è quello di stilare un documento di consenso multidisciplinare sugli aspetti più controversi e dibattuti che riguardano la gestione delle dell'intossicazione acuta da CO.

La fascia di età considerata è quella compresa tra 0 e 14 anni includendo anche raccomandazioni per la donna gravida. Il presente documento non prende in considerazione la gestione del bambino con intossicazione da cloruro di metilene. Saranno incluse alcune considerazioni sull'intossicazione da monossido in conseguenza a esposizione a fumi di incendio.

Emergency Care Journal, in press

Metodo utilizzato

Metodo

E' stato utilizzato il metodo *Delphi*. Il questionario è lo strumento utilizzato per raccogliere le opinioni degli esperti coinvolti. L'esperto ha espresso il proprio parere in termini di: completamente d'accordo, parzialmente d'accordo, parzialmente in disaccordo, completamente in disaccordo. Durante le varie fasi della discussione dell'iter o del processo, sono state apportate le modifiche proposte dagli esperti ad ogni singolo statement fino al raggiungimento del 100% di accordo. Gli esperti sono stati individuati in considerazione delle rispettive aree di interesse professionale, per le diverse competenze maturate, e le pubblicazioni scientifiche prodotte. I componenti del *panel*, in base alle reciproche competenze, hanno valutato in modo esaustivo ad ogni singola domanda (*statement*), completando una cartella a quesito. Al termine di una prima fase (o primo *round*), sono stati analizzati tutti i contributi. Successivamente è stato stilato un primo documento di sintesi e un secondo questionario che è stato nuovamente inviato al panel per raccogliere il consenso. Terminata anche la seconda fase (secondo *round*), il processo è stato ripetuto per la terza fase conclusiva.

Gli statement sono stati discussi in sessione plenaria durante i seguenti congressi:

- Antidotes in Depth a Pavia, Settembre 2017
- Congresso Nazionale SITOX a Bologna, Aprile 2018
- Antidotes in Depth a Pavia, Settembre 2018

Durante la discussione in sessione plenaria, i membri del panel si sono confrontati con il pubblico presente.

Position Paper sulla gestione delle intossicazioni acute da CO nel bambino: consenso multidisciplinare di esperti.

Panel di esperti identificati

Esperto	Disciplina	Affiliazione
Claudia Bondone	Pediatria d'urgenza	Regina Margherita, Torino
Patrizia Botarelli	Pediatria d'urgenza	Meyer, Firenze
Virna Carmellino	Pediatria d'urgenza	Regina Margherita, Torino
Carla Debbia	Pediatria d'urgenza	Ospedale Gaslini, Genova
Marcello Lanari	Pediatria d'urgenza	Ospedale S. Orsola-Malpighi, Bologna
Mara Pisani	Pediatria d'urgenza	OPBG, Roma
Eduardo Ponticiello	Pediatria d'urgenza	Ospedale Santobono Pausillipon, Napoli
Salvatore Renna	Pediatria d'urgenza	Ospedale Gaslini, Genova
Vincenzo Tipo	Pediatria d'urgenza	Ospedale Santobono Pausillipon, Napoli
Renata Passi	Pediatra di Libera Scelta	Pavia
Marco Marano	Rianimazione Pediatrica	OPBG, Roma
Matteo Pessina	Rianimazione Pediatrica	ICP Buzzi, Milano
Salvatore Talia	Rianimazione Pediatrica	Ospedale Santobono Pausillipon, Napoli
Roberto Zoppellari	Rianimazione	Arcispedale Sant'Anna, Ferrara
Rachele Adorisio	Cardiologia Pediatrica	OPBG, Roma
Andrea Drei	Medicina d'Urgenza	Ospedale di Faenza
Lidio Maffi	Medicina Iperbarica	OTIP, Torino
Gianmariano Marchesi	Medicina Iperbarica	OTIP, Zingonia
Giuliano Vezzani	Medicina Iperbarica	OTIP, Fidenza
Vincenzo Zanon	Medicina Iperbarica	OTIP, Brescia
Marta Crevani	Tossicologia clinica	CAV, Pavia
Mariapina Gallo	Tossicologia clinica	CAV, Bergamo
Francesco Gambassi	Tossicologia clinica	CAV, Firenze
Carlo A. Locatelli	Tossicologia clinica	CAV, Pavia
Davide Lonati	Tossicologia clinica	CAV, Pavia
Valeria M. Petrolini	Tossicologia clinica	CAV, Pavia
Azzurra Schicchi	Tossicologia clinica	CAV, Pavia
Salvatore Savasta	Neurologia Pediatrica	OSM, Pavia
Umberto Balottin	Neuropsichiatria Infantile	Ospedale Mondino, Pavia
Paola De Rose	Neuropsichiatria Infantile	OPBG, Roma
Simona Orcesi	Neuropsichiatria Infantile	Ospedale Mondino, Pavia
Ivo Casagranda	Revisore esterno	Pavia
Massimo Pesenti Campagnoni	Revisore esterno	Aosta
Carolina Prevaldi	Supervisore	San Donà di Piave

Definizione di intossicazione da CO

Statement 1

Definizione di intossicazione da monossido di carbonio (CO): Condizione patologica conseguente all'esposizione più o meno prolungata a CO. In soggetti esposti essa è identificata dalla rilevazione di valori positivi di carbossiemoglobina (COHb) e/o dalla presenza di manifestazioni cliniche eterogenee. I valori di COHb sono da considerarsi indicativi per esposizione se superiori al 5% (non fumatori). L'intossicazione da CO non può essere esclusa in caso di indicatore di esposizione (COHb) negativo, anche in conseguenza del tempo intercorso dal termine dell'esposizione. Tale definizione si applica sia all'esposizione acuta che a quella cronica. Quest'ultima è definita come esposizione ripetuta o prolungata a CO. Si ricorda che nei primi sei mesi, per catabolismo della HbF, i livelli fisiologici di COHb possono arrivare al 7%.

Quando pensare al CO?

Statement 2

Il sospetto di intossicazione da CO deve essere formulato (escludendo la rilevazione ambientale da parte del personale del soccorso) in presenza di almeno una delle seguenti situazioni:

- fonte di produzione di CO in ambienti confinati (es. braciere, stufa, camino) non necessariamente malfunzionanti
- miglioramento della sintomatologia dopo l'allontanamento dall'ambiente ritenuto a rischio
- sintomatologia comparsa in più componenti della stessa famiglia (anche se i quadri possono essere eterogenei) - (criterio epidemiologico)
- malessere/decesso in animali domestici (es. gatto, cane)
- provenienza dei pazienti da ambienti in cui si è verificato un incendio
- provenienza dei pazienti da ambienti in cui siano stati impropriamente utilizzati apparecchiature a motore (es. generatori)
- stagionalità o condizioni atmosferiche ostacolanti la fuoriuscita di fumi di scarico (es. vento forte)
- ricomparsa della sintomatologia simil-influenzale, sintomi gastroenterici, convulsioni non febbrili al rientro nell'ambiente a rischio

Indicazioni per equipaggi del soccorso e triage

Statement 3

E' raccomandato che tutti gli equipaggi del soccorso siano dotati di rilevatore ambientale di CO.

Statement 4

E' consigliabile che l'equipaggio del soccorso sia dotato di saturimetro per la rilevazione non invasiva della COHb e dotato di adattatori/presidi per l'utilizzo nel paziente pediatrico.

Statement 5

In considerazione (i) delle possibili difficoltà della procedura nel paziente pediatrico, (ii) della potenziale inattendibilità del dato, (iii) del ritardo nel trasferimento in ospedale non è consigliabile eseguire il prelievo venoso sulla scena dell'evento. Il prelievo venoso sulla scena dell'evento è da prendere in considerazione solo in casi in cui il tempo di trasporto superi le 2 ore.

Statement 6

La somministrazione di ossigeno è indicata in tutti i pazienti per i quali si sospetti l'intossicazione acuta da CO.

Triage

Statement 7

Nel paziente che non necessita intubazione, la somministrazione di ossigeno durante il trasporto del paziente deve essere effettuata almeno con maschera *reservoir* a tenuta per ottenere FiO_2 massimale e con taglia idonea all'età.

Statement 8

Il paziente con sospetta esposizione a CO è valutato con codice colore giallo in tutti i casi indipendentemente dalla sintomatologia. Il codice rosso è definito come: compromissione di almeno una funzionale vitale (cardiaca/respiratoria/neurologica), parametri vitali critici per età, convulsione in atto o nell'ultima ora).

La percentuale di COHb non ha valore prognostico ma (eventualmente) può confermare l'esposizione.

Bibliografia:

- SIMEUP - Linee guida su intossicazione da CO in età pediatrica

Valutazione clinica della gravità

Statement 9

In tabella si riporta la suddivisione per l'assegnazione della gravità clinica

Grading	**Segni e Sintomi per fasce di età**		
	0 - 2 anni	**3-7 anni**	**8-14 anni**
grado 1	Asintomatico		
grado 2	Nausea (conati) Vomito diarrea		
	Irritabile Pianto non consolabile Rifiuta l'alimentazione	Cefalea Dolore addominale con o senza diarrea	
			Vertigini
grado 3	I sintomi precedenti + tachipnea (senza movimento delle pinne nasali oretrazione sternale) Anomalie comportamentali Riferite alterazioni dello stato di coscienza (perdita transitoria di coscienza, sopore, stupore, coma) Riferite convulsioni Atassia		
	Refill cutaneo rallentato Iporeattività Pianto lamentoso	Disorientamento Tachicardia Alterazione della vista Dolore toracico	
grado 4: segni e sintomi presenti alla valutazione in Pronto Soccorso	Tachipnea con segni di fatica respiratoria Sincope Convulsioni Alterazione dello stato di coscienza (sopore, letargia, coma) Arresto cardiaco		
	In caso di esposizione a **fumi di incendio**, oltre alla sintomatologia sopra riportata, considerare anche: presenza di depositi di sulla cute , aditus vie aeree (cavo orale e narici) grave instabilità emodinamica **arresto cardiaco → ripristino attività cardio-respiratoria (PALS)-> priorità di trattamento antidotico**		

Valutazione clinica della gravità

Statement 9

In tabella si riporta la suddivisione per l'assegnazione della gravità clinica

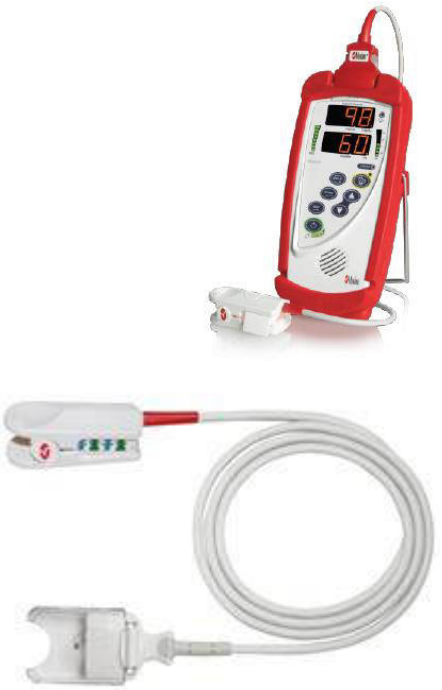
<table>
<tr><th>Grading</th><th colspan="3">Segni e Sintomi per fasce di età</th></tr>
<tr><td></td><th>0 - 2 anni</th><th>3-7 anni</th><th>8-14 anni</th></tr>
<tr><td>grado 1</td><td colspan="3">Asintomatico</td></tr>
<tr><td rowspan="3">grado 2</td><td colspan="3">Nausea (conati)
Vomito
diarrea</td></tr>
<tr><td>Irritabile
Pianto non consolabile
Rifiuta l'alimentazione</td><td colspan="2">Cefalea
Dolore addominale con o senza diarrea</td></tr>
<tr><td></td><td></td><td>Vertigini</td></tr>
<tr><td rowspan="2">grado 3</td><td colspan="3">I sintomi precedenti +
tachipnea (senza movimento delle pinne nasali oretrazione sternale)
Anomalie comportamentali
Riferite alterazioni dello stato di coscienza (perdita transitoria di coscienza, sopore, stupore, coma)
Riferite convulsioni
Atassia</td></tr>
<tr><td>Refill cutaneo rallentato
Iporeattività
Pianto lamentoso</td><td colspan="2">Disorientamento
Tachicardia
Alterazione della vista
Dolore toracico</td></tr>
<tr><td>grado 4: segni e sintomi presenti alla valutazione in Pronto Soccorso</td><td colspan="3">Tachipnea con segni di fatica respiratoria
Sincope
Convulsioni
Alterazione dello stato di coscienza (sopore, letargia, coma)
Arresto cardiaco</td></tr>
<tr><td></td><td colspan="3">In caso di esposizione a fumi di incendio, oltre alla sintomatologia sopra riportata, considerare anche:
presenza di depositi di sulla cute , aditus vie aeree (cavo orale e narici)
grave instabilità emodinamica
arresto cardiaco → ripristino attività cardio-respiratoria (PALS)-> priorità di trattamento antidotico</td></tr>
</table>

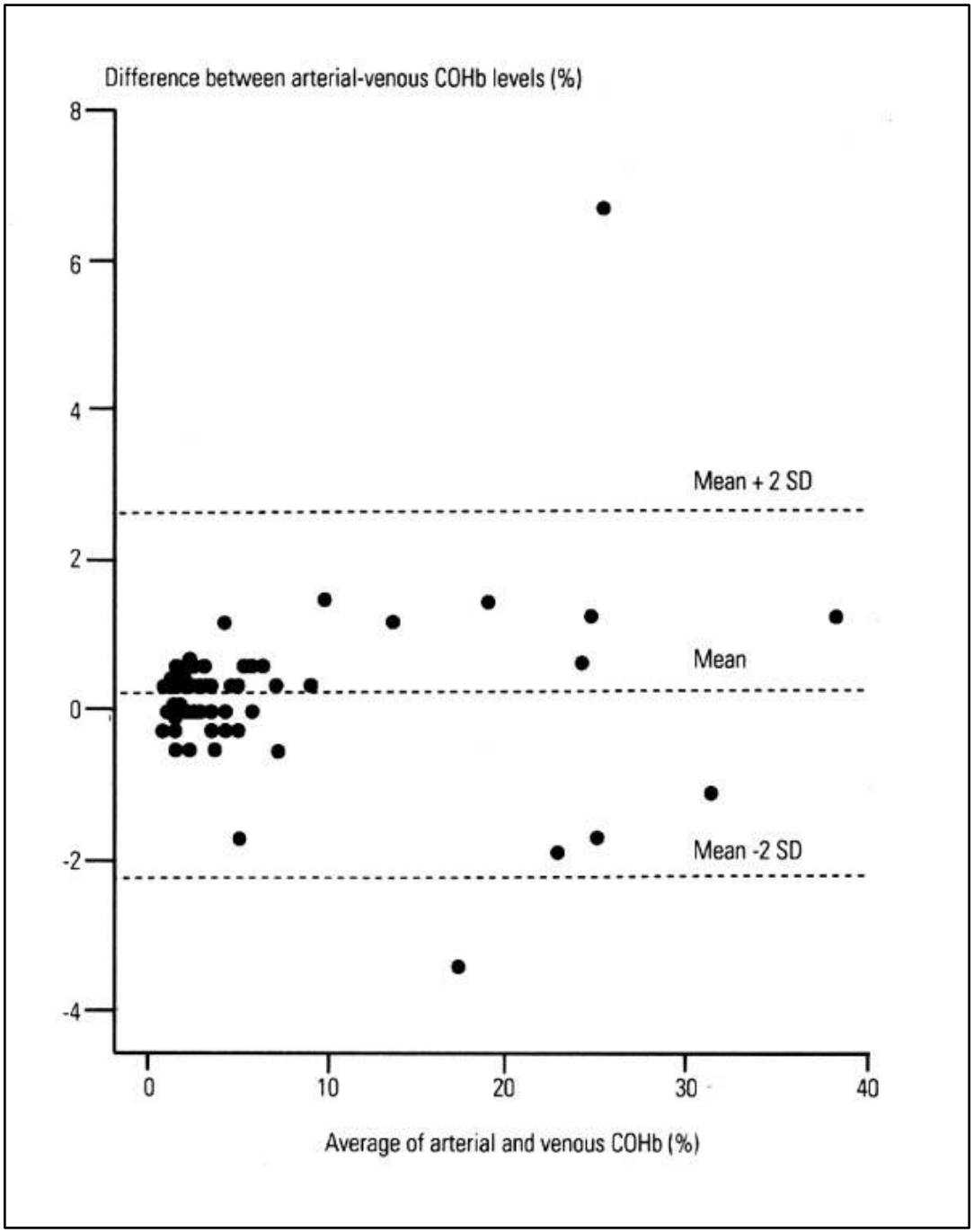
Statement 10

La determinazione della COHb utilizzando il CO-ossimetro per la rilevazione non invasiva (dotato di adattatori/presidi per l'utilizzo nel paziente pediatrico) è raccomandato a supporto della valutazione clinica e del triage.

Bibliografia:

- McKenzie LB, Roberts KJ, Kaercher RM, Collins CL, Comstock RD, Fernandez S, Abdel-Rasoul M, Casavant MJ, Mihalov L. Paediatric emergency department-based carbon monoxide detector intervention: a randomised trial. Inj Prev. 2017 Oct;23(5):314-320.
- Hampson NB. Noninvasive pulse CO-oximetry expedites evaluation and management of patients with carbon monoxide poisoning. Am J Emerg Med. 2012 Nov;30(9):2021-4.





Differenza tra i livelli di COHb arteriosa e venosa

Touger et al, Ann Emerg Med, 1995

… clinically significant inaccurate carboxyhemoglobin measureme

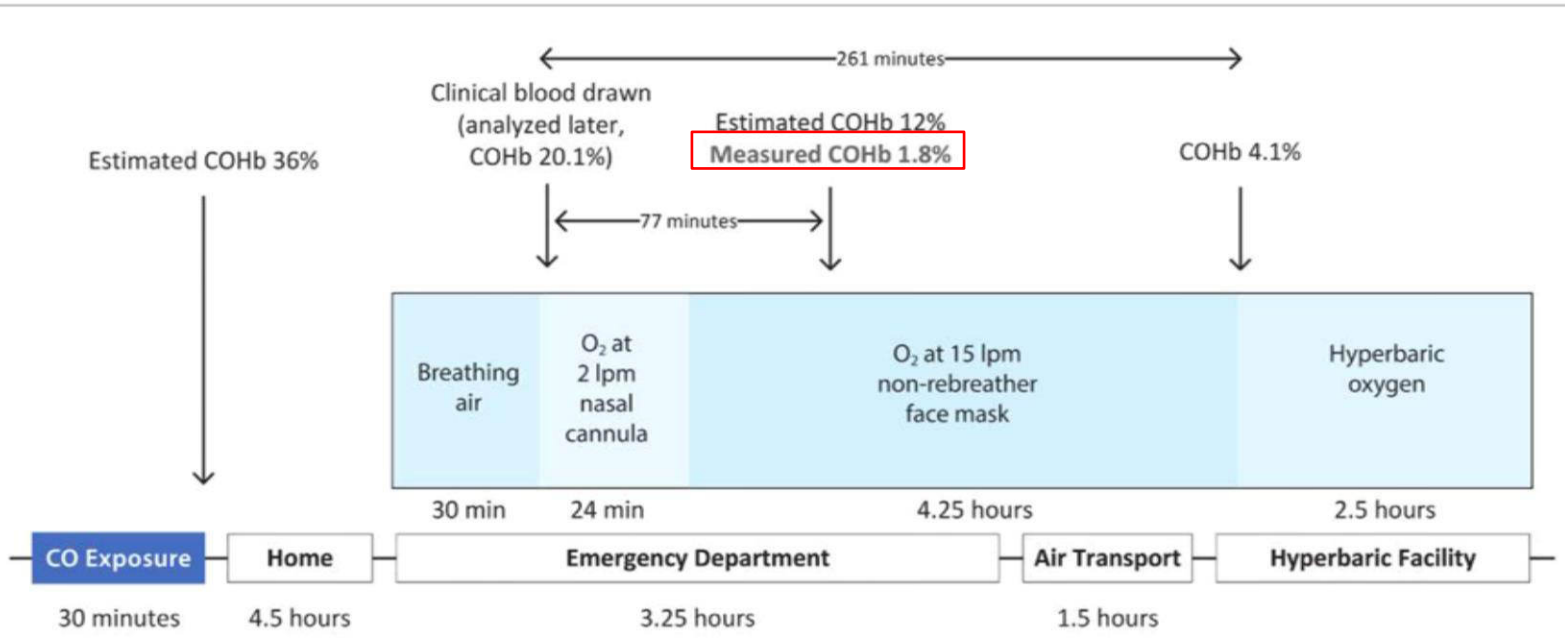


Figure 1. Case 1 patient management timeline.

The patient's first ordered COHb measurement returned a false low value (1.8%), but the patient's symptoms and history strongly suggested CO poisoning. A repeated COHb and later co-oximetry analysis of blood drawn for initial clinical laboratory confirm that the 1.8% COHb was erroneous.

Weaver LK al., UHM, 2022

Condizioni fisiologiche / patologiche / ambientali

Statement 11

Le scelte terapeutiche sono condizionate dalle seguenti condizioni fisiopatologiche/ambientali

Condizioni fisiologiche	Condizioni patologiche/ambientali
presenza di HbF (età inferiore a 6 mesi-1 anno)	disfunzione ventricolare / storia di scompenso / cardiopatie congenite
immaturità del SNC	anemia
metabolismo accelerato	anemia falciforme o talassemia
condizioni che richiedono aumentato consumo di ossigeno (es. esercizio fisico, febbre)	anemia emolitica
	leucemia
	ittero neonatale
	ipovolemia
	emoglobinopatie (es. metHb)
	ipossia
	inalazione di fumi di incendio, ustione coane nasali e/o vibrisse, ustioni cutanee

Statement 12

Recommended **laboratory tests and imaging** studies for initial evaluation of patients:

- blood gases determination (at least venous blood) with lactate level
- complete blood count
- fetal hemoglobin in patients younger than 1 year
- hemoglobinopathies (e.g. metHb)
- muscular damage bio*markers* (creatine kinase)
- myocardial damage bio*markers* (troponins)
- B-type natriuretic peptide or N-terminal-pro hormone BNP (BNP or NT-proBNP)
- glycemia
- creatinine
- EKG
- thorax XR
- pregnancy test in women of child-bearing age

- cardiotocography control in pregnant woman

NBO and HBO Treatment

Statement 13

Normobaric oxygen therapy (15 liters/minute with at least a non-rebreathing mask) should be administered to **every patient**, no matter the severity grade of symptoms

Statement 14

For those patients treated with hyperbaric oxygen therapy, it's not indicated further adjuvant treatment with normobaric oxygen therapy after the hyperbaric oxygen treatments.

NBO and HBO Treatment

Statement 15

Inclusion criteria for hyperbaric oxygen therapy :

- asymptomatic pregnant patient
- patient severity grade 3 and 4, independently form COHb levels
- in case of fire smokes exposure, hyperbaric oxygen therapy is indicated also in those patients with severity grading 2 and with COHb levels below 10% and but this does not substitute the specific antidote therapy for cyanide poisoning if necessary (lactic acidosis)

Inclusion criteria requiring a hyperbaric physician's case-by-case evaluation:

- patient severity grade 1 (asymptomatic) with COHb level presentation greater than 10%,
- patient severity grade 2

And with a specific risk-benefit oriented choice in case of:

- a patient younger than 6 months of age because of the presence of fetal hemoglobin (independently from the value of COHb)

HBO treatment protocol

Statement 16

The more appropriate treatment regimen for hyperbaric oxygen therapy and the number of sessions will be evaluated in every single case by the hyperbaric medical specialist.

One single treatment session can be sufficient for patients asymptomatic at the end of the session.

In case of clinical indication to additional hyperbaric sessions, it is not considered beneficial to exceed an overall of 3 sessions in number. The number of sessions can increase in case of indication to treatment of delayed neurocognitive sequelae .

Statement 17

Hyperbaric oxygen therapy (HBO) should be initiated, when possible, within 6 hours and, in any case, no later than 12 hours from the diagnosis.

Contra-indications to HBO

Statement 18

- impossibility to perform compensatory maneuvers (for age); in severe selected cases, consider the opportunity of a bilateral myringotomy, and the eventual positioning of trans-tympanic drainages (grommets),
- not drained pneumothorax,
- ongoing acute asthmatic attack,
- status epilepticus,
- patients suffering from an ethmoid bone fracture and rhinorrhea (risk of a dysbaric pneumocephalus occurrence while performing the required compensatory maneuvers),
- therapy with doxorubicin or cisplatin (rare in pediatric age)

Main relative contra-indications:

- epilepsy under pharmacological treatment,
- history positive for spontaneous pneumothorax,
- pacemaker or ICD (on the basis of the technical sheet of these devices),
- claustrophobic traits.

Statement 19

Normobaric oxygen therapy (15 liters/minute with at least a non-rebreathing mask with reservoir) should be administered until symptoms resolution, and in any case for at least 12 h, to those patients exposed to CO acute poisoning and not presenting indication to hyperbaric oxygen therapy or having indication to HBO but with concomitant contraindication to this line of treatment.

Statement 20

To those patients for which, despite indication, it is not possible to perform hyperbaric oxygen therapy, normobaric oxygen therapy (15 liters/minute with at least a non-rebreathing mask with reservoir) should be administered for at least 24 h. The continuation of treatment will be decided considering a neurological clinical re-evaluation.

Statement 21

The patient in-chamber assistance follows the directives and rules of every hospital

Statement 22

Echocardiography may not be performed in asymptomatic patients with biomarkers and EKG within normal ranges. In case of alteration of one or more biomarkers, it is strongly recommended to have the patient undergo echocardiography within 24 hours.

Statement 23

The young patient can be discharged after a minimum of 12-24 hours of observation from the end of the normobaric/hyperbaric therapy and with normal laboratory analysis and imaging studies, according to clinical evaluation.

Statement 24

Follow-up and long-term evaluation.

45 days after hospital discharge it is advisable patient revaluation by the pediatrician for cardiac evaluation and/or for possible appearance of symptoms attributable to delayed neurological sequelae (NMR CNS) (in formative letter to parents).

2022

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SHORT COMMUNICATION

Hyperbaric oxygen should be used for carbon monoxide poisoning

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Funding information
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This short review addresses the mechanisms of injury mediated by carbon monoxide (CO) and current information on efficacy of hyperbaric oxygen therapy (HBOT). Recent clinical series involving large, country-wide databases and prospective randomized trials are summarized. We conclude that there is an abundance of basic science and preclinical and clinical research supporting the use of HBOT for acute CO poisoning. With appropriate consideration for pathology and therapeutic mechanisms, HBOT at a dose of 2.5–3.0 atm absolute is a necessary treatment for this toxidrome.

KEYWORDS
carboxyhaemoglobin, neurological sequelae, randomized trials, β_2 integrins

TABLE 1 Randomized control trials for hyperbaric *vs.* ambient pressure oxygen treatment listed by increasing dose of hyperbaric oxygen (HBO)

Study	Maximum O_2 dose	Time tx initiated[a]	Acute treatment protocol[b]	Source	Sample size (*n*)	Results
Raphael et al.[25]	2.0 ATA	5.3 ± 2.3 to 6.4 ± 2.8 h	6 h NBO *vs.* 2 h HBO + 4 h NBO or 4 h NBO with 1 *vs.* 2 HBO treatments	Non-fire, accidental domestic exposure	343	No benefit
Annane et al., 2011[22]	2.0 ATA	2 to 6 h	Same as Raphael et al.	Non-fire, accidental domestic exposure	385	Negative
Ducasse et al.[23]	2.5 ATA	Mean: 53 min	12 h NBO *vs.* 2 h HBO + 10 h NBO	Unknown	26	Positive
Mathieu et al.[24]	2.5 ATA	Not given	12 h NBO *vs.* 90 min HBO	Unknown	575	Positive
Thom et al.[27]	2.8 ATA	2.0 ± 1.1 h	6 h NBO *vs.* 2.8 ATA for 30 min followed by 2.0 ATA for 90 min	Auto, furnace, fires	65	Positive
Scheinkestel et al.[26]	2.8 ATA	5.7 to 8.6 h	Three days NBO *vs.* three daily tx 2.8 ATA for 60 min + 40 min taper plus 3 days NBO	Auto, furnace, no fires	191	Negative
Weaver et al.[10]	3.0 ATA	5.8 ± 2.9 h	125 min NBO *vs.* 3.0 ATA for 50 min followed by 2.0 ATA for 60 min	Auto, furnace, no fires	152	Positive

Abbreviations: ATA, absolute atmosphere; HBO, hyperbaric oxygen; NBO, normobaric oxygen.
[a]Treatment initiation times are the interval from removal from the CO source to start of treatment. They are shown as mean ± SD or range when only that information was included.
[b]Note that some protocols involved multiple treatments after initial intervention. These details are not shown.

However, with appropriate consideration for pathology and therapeutic mechanisms, we conclude that HBOT at a dose of 2.5–3.0 ATA is a necessary treatment for this toxidrome.

2022

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REVIEW ARTICLE

BJCP BRITISH PHARMACOLOGICAL SOCIETY

Hyperbaric oxygen should not be used routinely for carbon monoxide poisoning

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[2]Ontario Poison Centre, Toronto, Ontario, Canada

[3]Sunnybrook Research Institute, Sunnybrook Health Sciences Centre, Toronto, Ontario, Canada

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KEYWORDS: carbon monoxide poisoning, hyperbaric oxygen

«Every day, thousands of patients around the world are poisoned by carbon monoxide. Some die in the prehospital setting, but many more present in need of treatment. Clinicians who opt to treat them with HBO do so in the hope that it might help, not because they know it will. Even as a card-carrying HBO sceptic, I myself recommend the therapy from time to time, but I do so with the recognition that I am operating …»

Pediatric Carbon Monoxide Poisoning

Effects of Hyperbaric Oxygen Therapy on Thiol/Disulfide Balance

Zafer Bağcı, MD, Abdullah Arslan, MD,† and Salim Neşelioğlu, MD‡*

Objectives: Carbon monoxide (CO) poisoning remains the foremost cause of poisoning worldwide. This study aimed to investigate the effects of hyperbaric oxygen therapy (HBOT) and normobaric oxygen therapy (NBOT) on thiol/disulfide homeostasis in children with CO intoxication.

Methods: Eighty-one children aged 0 to 18 years with CO intoxication were included in this cross-sectional study. No changes were made in the routine clinical evaluation and treatment practices of the patients. Thirty-two children who received HBOT and 49 children who received NBOT were compared for serum native thiol, disulfide, and total thiol levels, as well as for the changes in disulfide/native thiol, disulfide/total thiol, and native thiol/total thiol ratios before and after treatment.

Results: Antioxidant levels, such as native thiol and total thiol, were significantly decreased in patients who received HBOT and increased in those who received NBOT ($P = 0.02$ and $P = 0.01$, respectively). There was no statistically significant difference between the 2 groups concerning the change of native thiol/total thiol ratios ($P = 0.07$). In addition, there was no significant difference regarding changes in disulfide, disulfide/native thiol, and disulfide/total thiol levels before and after treatment ($P = 0.39$, $P = 0.07$, and $P = 0.07$, respectively).

Conclusions: Although thiol-disulfide balance is maintained in patients treated with HBOT, antioxidant levels decrease significantly compared with NBOT. Despite efficiency of HBOT in CO intoxication, oxidative stress and reperfusion injury due to hyperoxygenation should be considered in the treatment of HBOT.

Key Words: carbon monoxide poisoning, thiol-disulfide, oxidative stress

(*Pediatr Emer Care* 2022;38: 104–107)

Delayed encephalopathy of acute carbon monoxide intoxication in rats: potential mechanism and intervention

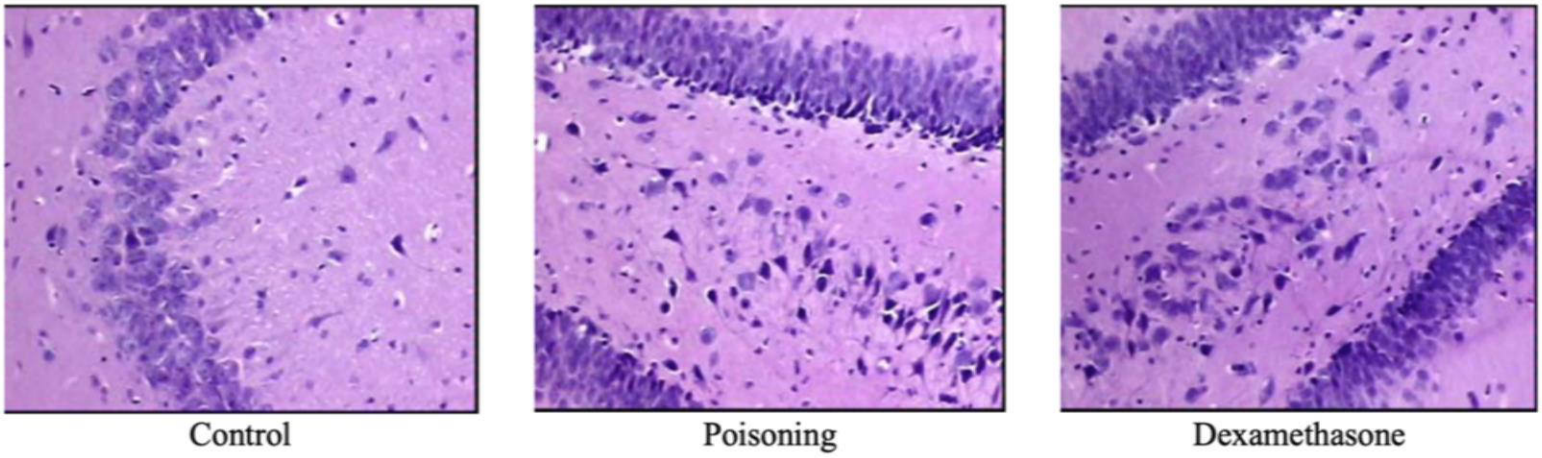


Fig. 1: Hematoxylin and eosin (H&E) staining (×100)

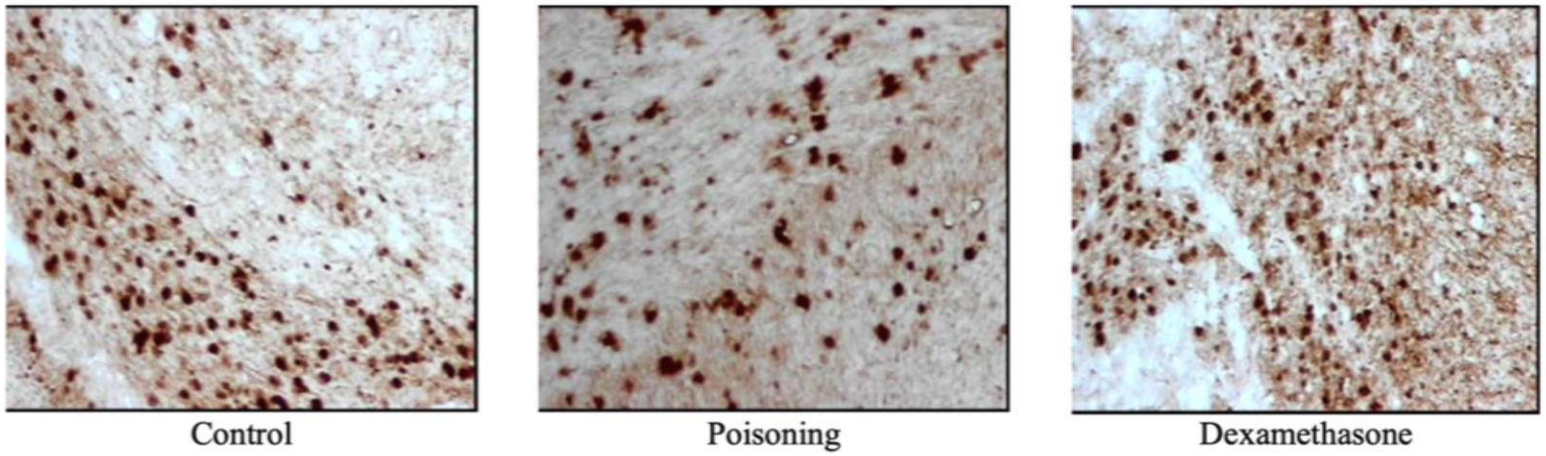


Fig. 2: MBP immunohisto chemistry staining (×100)

Xiang W-P. 2014

2023

CLINICAL RESEARCH

Dexamethasone therapy prevents delayed neuropsychiatric sequelae after carbon monoxide poisoning: a prospective registry-based study

Sechan Kim[a], Sungwoo Choi[a], Yujin Ko[b,c], Choung Ah Lee[d], Gi Woon Kim[a], Ji Eun Moon[e], Sangun Nah[a*] and Sangsoo Han[a*]

[a]Department of Emergency Medicine, Soonchunhyang University Bucheon Hospital, Bucheon, Republic of Korea; [b]Department of Psychiatry, Soonchunhyang University Bucheon Hospital, Bucheon, Republic of Korea; [c]Department of Psychiatry, Gangnam Severance Hospital, Yonsei University College of Medicine, Seoul, Republic of Korea; [d]Department of Emergency Medicine, Hallym University Dongtan Sacred Heart Hospital, Hwaseong, Republic of Korea; [e]Department of Biostatistics, Clinical Trial Center, Soonchunhyang University Bucheon Hospital, Bucheon, Republic of Korea

ABSTRACT

Background: Delayed neuropsychiatric sequelae are major complications of carbon monoxide poisoning; carbon monoxide triggers brain oxidation and inflammation. Corticosteroids such as dexamethasone modulate neurological damage after carbon monoxide poisoning through anti-inflammatory actions and immune response inhibition. However, it is not known whether corticosteroids prevent delayed neuropsychiatric sequelae. We thus studied whether dexamethasone reduced the incidence of delayed neuropsychiatric sequelae.

Methods: This registry-based study enrolled patients with carbon monoxide poisoning treated in a Korean tertiary care hospital from March 1st, 2020 to November 30th, 2021. Data of patients were prospectively collected during the study period, and retrospectively analyzed. One group received intravenous dexamethasone. We performed multivariable logistic regression analysis to identify factors associated with delayed neuropsychiatric sequelae.

Results: A total of 128 patients were enrolled, of which 99 patients received dexamethasone therapy and 29 patients did not. The incidences of delayed neuropsychiatric sequelae in the dexamethasone and non-dexamethasone groups were 16.2% and 37.9%, respectively. Multivariable logistic regression analysis revealed that dexamethasone use (odds ratio =0.122, 95% confidence interval 0.031–0.489) and a higher Glasgow Coma Scale (odds ratio = 0.818, 95% confidence interval 0.682–0.981) was associated with a lower incidence of delayed neuropsychiatric sequelae.

Conclusion: Early dexamethasone treatment was significantly associated with a decreased incidence of delayed neuropsychiatric sequelae. A higher *Glasgow Coma Scale* at presentation also was associated with a lower incidence of delayed neuropsychiatric sequelae.

ARTICLE HISTORY
Received 28 June 2022
Revised 5 January 2023
Accepted 12 January 2023

KEYWORDS
Carbon monoxide; dexamethasone; poisoning; neuropsychiatric sequelae

Table 2. Comparison of the non-dexamethasone and dexamethasone patient groups.

	Non-dexamethasone group ($n = 29$)	Dexamethasone group ($n = 99$)	*P* Value
Age, years	46.5 ± 17.8	42.6 ± 13.7	0.289
Sex, *n* (%)			>0.99[a]
Female	10 (34.5)	32 (32.3)	
Male	19 (65.5)	67 (67.7)	
Vital signs			
Systolic blood pressure, mmHg	130 [110–140]	130 [115–140]	0.890
Diastolic blood pressure, mmHg	80 [70–90]	80 [70–90]	0.594
Heart rate, beats/min	93.1 ± 19.4	91 ± 17.9	0.591
Respiratory rate, breaths/min	20 [20–20]	20 [18–20]	0.9
Comorbidities, *n* (%)			
Diabetes	5 (17.2)	3 (3.0)	0.015[b]
Hypertension	4 (13.8)	8 (8.1)	0.467[b]
Current smoker, *n* (%)	16 (55.2)	53 (53.5)	>0.99[a]
Glasgow Coma Scale at presentation	15 [12–15]	15 [12.5–15]	0.352[b]
Score < 15, *n* (%)	13 (44.8)	32 (32.3)	0.308[a]
Symptoms, *n* (%)			
Headache	3 (10.3)	9 (9.1)	>0.99[b]
Loss of consciousness	14 (48.3)	26 (26.2)	0.043[a]
Dizziness	2 (6.9)	13 (13.1)	0.518[b]
Dyspnea	2 (6.9)	6 (6.1)	>0.99[b]
Chest pain	1 (3.5)	1 (1)	0.403[b]
Laboratory findings			
White blood cell count, $10^3/\mu L$	10.9 [8.7–13.7]	11.8 [8.8–15.4]	0.734
Hemoglobin, g/dL	14.5 [13.1–15]	15.1 [13.5–16]	0.106
Protein, g/dL	7.2 ± 0.5	7.3 ± 0.6	0.581
Albumin, g/dL	4.3 ± 0.4	4.4 ± 0.4	0.327
Glucose, mg/dL	124 [95.8–171.5]	113 [101.5–131]	0.559
Blood urea nitrogen, mg/dL	12.5 [10.3–20.6]	13 [11.1–16.8]	0.988
Creatinine, mg/dL	1.05 [0.8–1.3]	1 [0.9–1.2]	0.476
Creatine kinase, U/L	134 [81.5–538]	121 [84–433]	0.569
Troponin I, ng/mL	0.1 [0.1–0.2]	0.09 [0.05–0.15]	0.008
C-reactive protein, mg/L	0.2 [0.1–1.3]	0.1 [0–0.4]	0.206
Lactate, mg/dL	1.4 [1.2–4.2]	2.1 [1.8–4.7]	0.553
Carboxyhemoglobin, %	9.0 [3.8–13.7]	10.2 [5.5–17.6]	0.330
Duration of carbon monoxide exposure, min	150 [30–240]	180 [85–420]	0.236
Type of exposure, *n* (%)			0.936[a]
Unintentional	7 (24.1)	21 (21.2)	
Intentional	22 (75.9)	78 (78.8)	
Hyperbaric oxygen therapy, *n* (%)	21 (72.4)	85 (85.9)	0.101[b]
Delayed neuropsyciatric sequelae, *n* (%)	11 (37.9)	16 (16.2)	0.023[a]

Values are presented as means ± standard deviations, medians [interquartile ranges], or numbers (proportions).
[a]Pearson's χ^2 test,[b]Fisher's exact test.

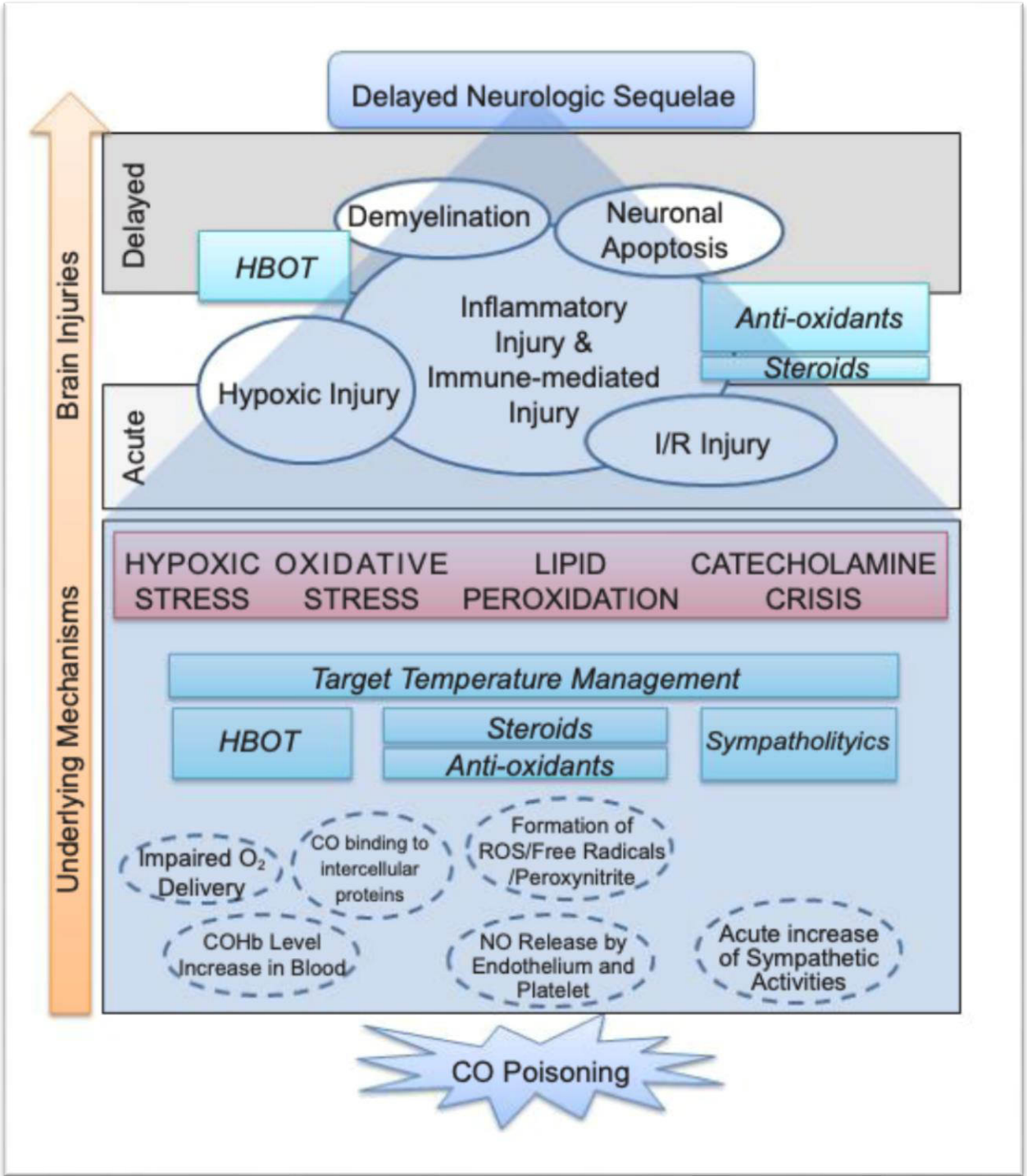
Kim S et al., 2023

Table 3. Multivariable logistic regression analysis of factors predictive of delayed neuropsychiatric sequelae.

	OR	95% CI
Dexamethasone use	0.122	0.031–0.489
Glasgow Coma Scale score	0.818	0.682–0.981
Carboxyhemoglobin, %	0.944	0.888–1.003
C-reactive protein, mg/L	0.988	0.836–1.168
Duration of exposure to carbon monoxide, min	1.001	0.999–1.003
Hyperbaric oxygen therapy	5.662	0.517–62.011

OR: odds ratio; CI: confidence interval.

Kim S et al., 2023



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PSYCHOLOGICAL SEQUELAE TO CARBON MONOXIDE INTOXICATION IN THE CHILD

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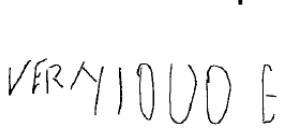
ABSTRACT

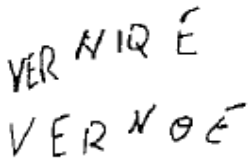
Twenty children from 25 months to 15 years old at the time of carbon monoxide intoxication have been studied in a short-term follow-up, and 14 children in a long-term follow-up, as far as their psychological and cognitive faculties are concerned. The quality of recovery is a function of the severeness of the intoxication, but the intellectual level of the children, and primarily their age at the time of the intoxication, also play a major role.

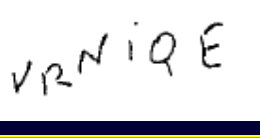
6.9 ys
coma
COHb 25%

after CO exp.



4 months after

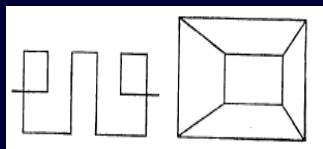




12 ys
coma

18 days after

1 year after



	age	n°	COHb	Psychological sequelae
Short term f-up 3 months (n=20) only 3 coma at ED	2-3 ys	6	16-37 %	irritable, anxiety
	4-9 ys	8	4-25%	mnestic/visuo-spatial disorder
	10-15 ys	6	26-36%	auditory/visual deficits
Long term f-up 2-11 years (n=14) 4 coma 3 somnolence at ED	2-3 ys	2	19-24%	1 serious spatial, spelling, lexical disorders
	4-9 ys	6	13-42%	2 mild (memory) and 3 serious disorders
	10-15 ys	6	23-32%	2 mild (memory) and 4 serious disorders

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BRIEF REPORT

Open issues in management of carbon monoxide poisoning in pregnancy: practical suggestions

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ABSTRACT

Carbon monoxide (CO) poisoning during pregnancy may cause deleterious effects to the fetus. Hyperbaric oxygen therapy (HBO) in pregnancy is proven to be safe and it is considered to be beneficial, reducing the severity of the fetal injuries. However, a number of issues are still to be discussed, among them the question of the carboxyhemoglobin (COHb) levels that trigger HBO therapy in pregnant CO poisoned patients. In this letter we report some practical suggestions for organizations wishing to develop their own protocols.

KEYWORDS

Carbon monoxide; pregnancy; poisoning; general gynaecology; general obstetrics

Chorea as the Neurological Symptom of Delayed Encephalopathy After Carbon Monoxide Intoxication in a Child

The Neurohospitalist
2016, Vol. 6(3) 130-131

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Muhammad Ubaidulhaq, MD[1], Young Ah Lee, MD[2], and Huiyuan Jiang, MD[1]

Keywords
chorea, delayed encephalopathy, carbon monoxide (CO) intoxication

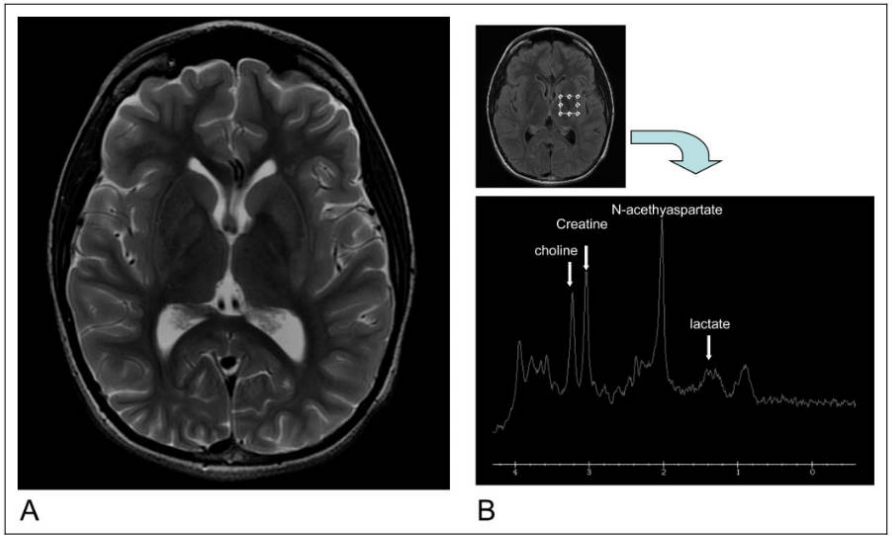

Figure 1. A, T2-weighted axial sequence of brain magnetic resonance imaging (MRI) shows hyperintensities in the bilateral basal ganglia. The signal changes are greater involving the left caudate and putamen and greater involving the right globus pallidus. B, Brain magnetic resonance spectroscopy (MRS) with voxel over the left basal ganglia shows small lactate peaks.

Cyanide !

A 10-year-old boy presented to the emergency department with abnormal movements 8 days after he was rescued from a house fire. At the scene, he was comatose and was found to have soot in his airway. He had an elevated carboxyhemoglobin level (24.5%). He received mechanical ventilation with 100% oxygen for 40 hours. He showed rapid recovery in mental status and was discharged home. Eight days after the injury, he developed tremors, dystonia, and chorea involving his face and all extremities (Video). Brain magnetic resonance imaging showed T2 hyperintensity in the caudate, putamen, and globus pallidus bilaterally, and magnetic resonance spectroscopy showed lactate peaks in the basal ganglia (Figures 1A and B).

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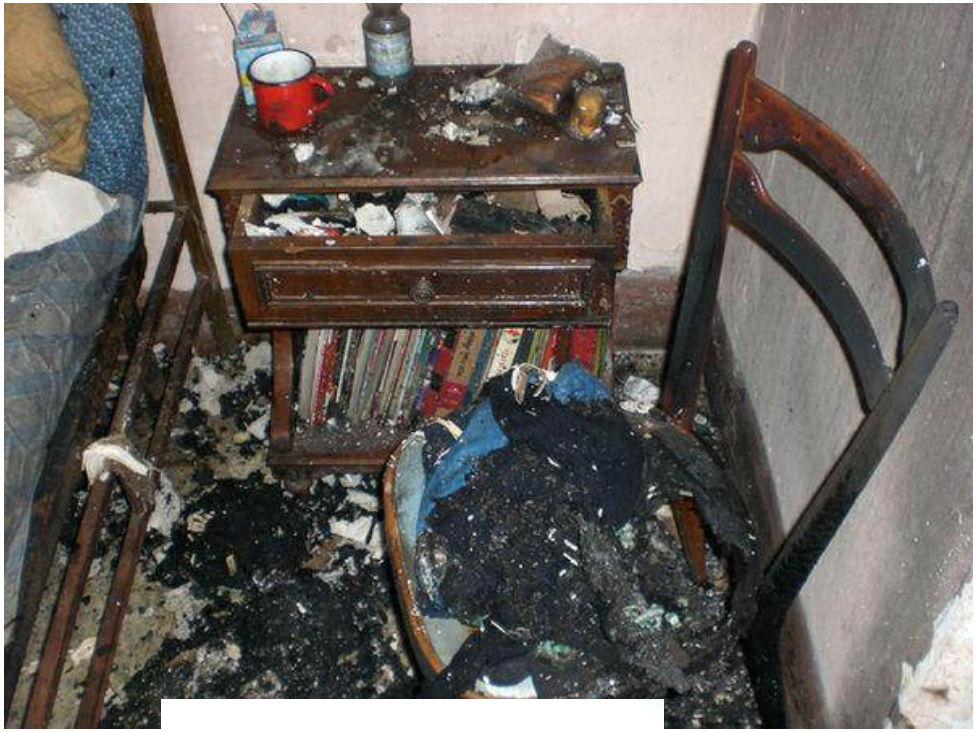
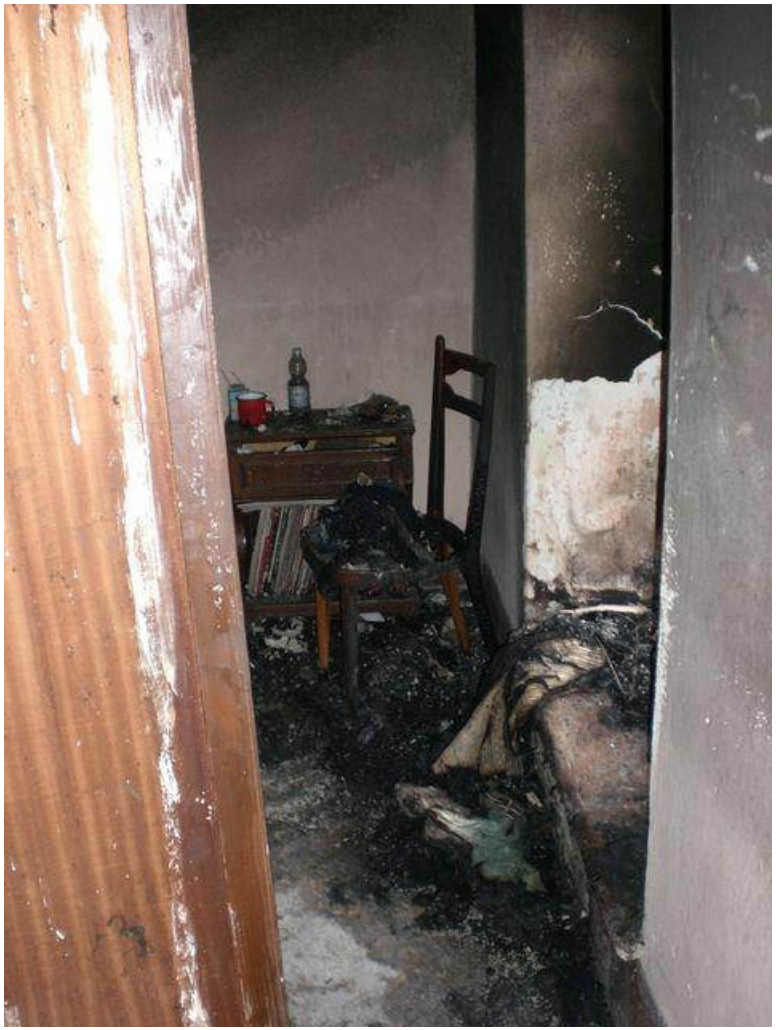
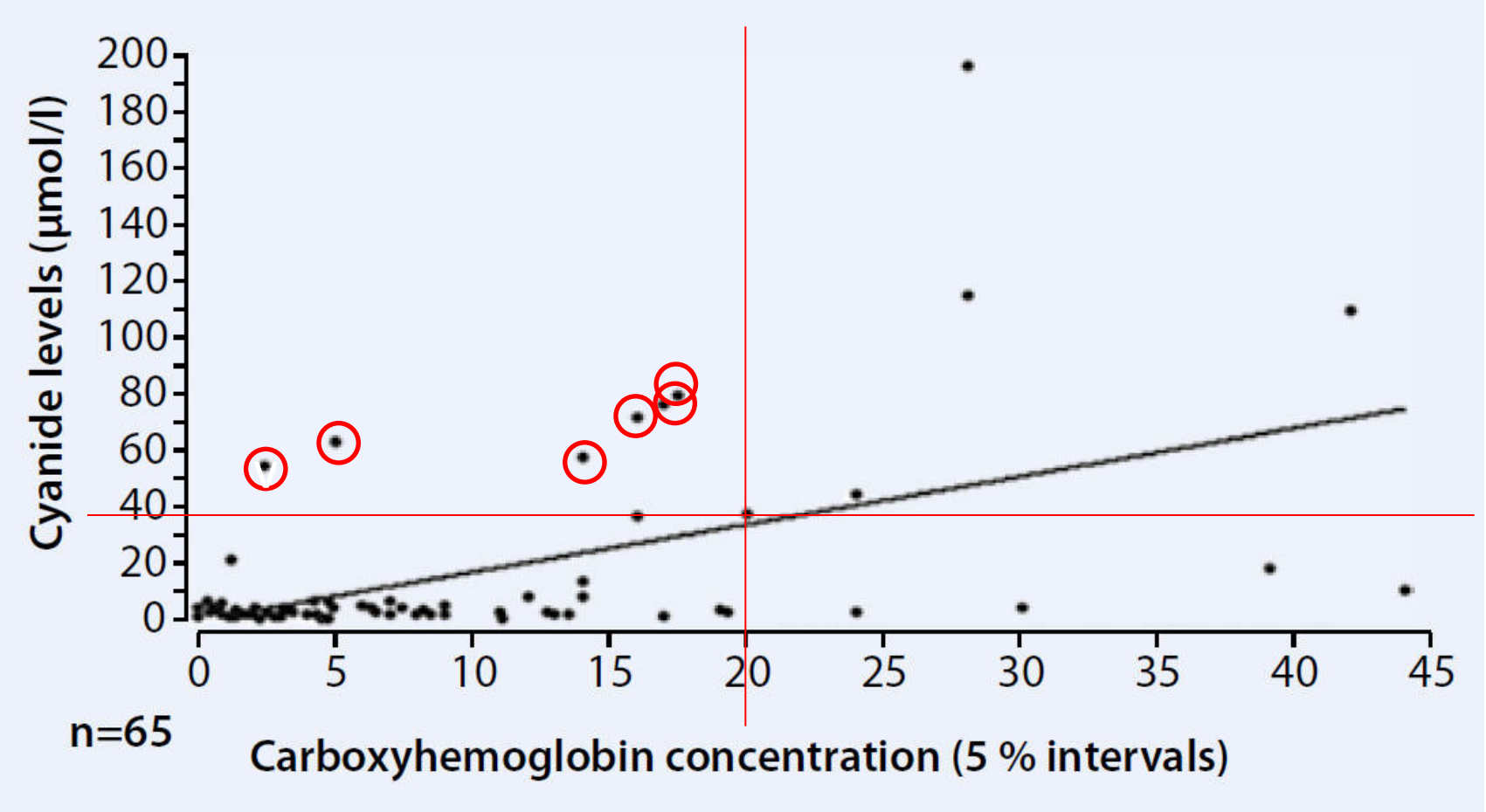




Correlation between cyanide levels (µmol/l) and COHb concentrations (%) in the blood of patients after smoke inhalation (correlation coefficient: 1.68; p<0.0001)



Intossicazione acuta da CO nel bambino
Conclusioni

- CO: veleno funzione e lesionale
- clinica di presentazione “subdola”
- insorgenza precoce nel bambino
- criterio epidemiologico

- pulsiossimetro (rilevazione non invasiva al triage)

- trattamento con ossigeno normobarico
  - Ossigeno iperbarico
  - Antiossidanti (es. NAC)
  - Steroidi

- follow-up
  - danno d’organo (miocardio “stordito”)
  - sequele neurologiche
- attenzione a esposizione a fumi di incendio domestico

21° Congresso Nazionale
Società Italiana di Tossicologia

Grazie per l'attenzione

Dr. Davide Lonati

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Linee guida SIMSI/SIAARTI/ANCIP per il trattamento con ossigeno iperbarico della intossicazione acuta da CO

M. Mordacci, G. Vezzani
AUSL Parma, P.O. Vaio-Fidenza
U.O. Anestesia-Rianimazione, Terapia Iperbarica e Antalgica

- PZ IN COMA
- PZ CON MOMENTANEA PERDITA DI COSCIENZA
- PZ CON SINTOMI NEUROPSICHIATRICI (cefalea, nausea, vomito, vertigini, modificazioni caratteriali, ecc.)
- PZ CON ACIDOSI METABOLICA SCOMPENSATA
- PZ CON DOLORE TORACICO E SEGNI ECG DI ISCHEMIA MIOCARDICA
- ARITMIE

- PZ IN GRAVIDANZA
- BAMBINI IN ETÀ < 6 MESI PER LA PRESENZA DI HbF

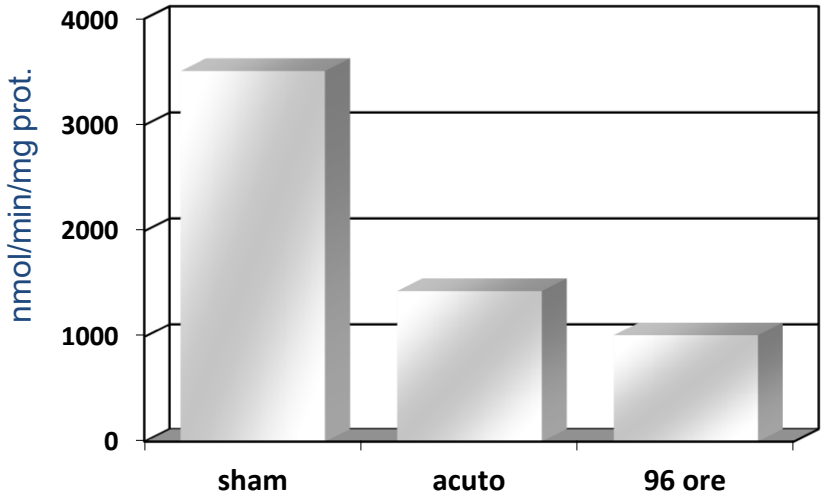
- PZ ASINTOMATICI CON COHB > 25%
- BAMBINI ASINTOMATICI CON ETÀ < 12 ANNI CON: COHB > 10%
- PZ ASINTOMATICI CON PREGRESSA ISCHEMIA MIOCARDICA CON COHB > 15%

Esperienza Centro Iperbarico di Fidenza – dr. Vezzani

- gennaio 1990 al dicembre 2010 (≅ 26 bambini/anno)
  - 520 bambini
    - età compresa tra zero (15 giorni) e 12 anni → 189; 36% (15 giorni e 4 anni)
- casco (nel quale venivano infilati fino al diaframma)
  - bambini età inferiore a 15-18 mesi
- maschere con valvola di non ritorno per rendere minimo lo spazio morto

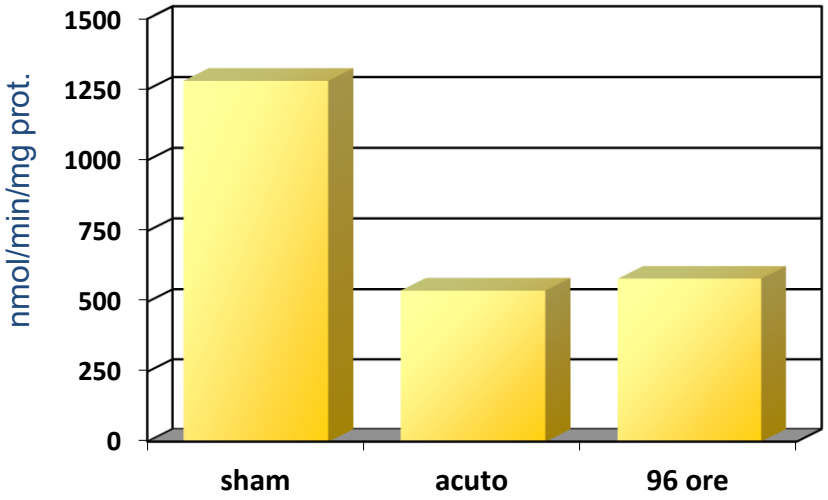
- non sono stati segnalati effetti avversi

CITOCROMO OSSIDASI

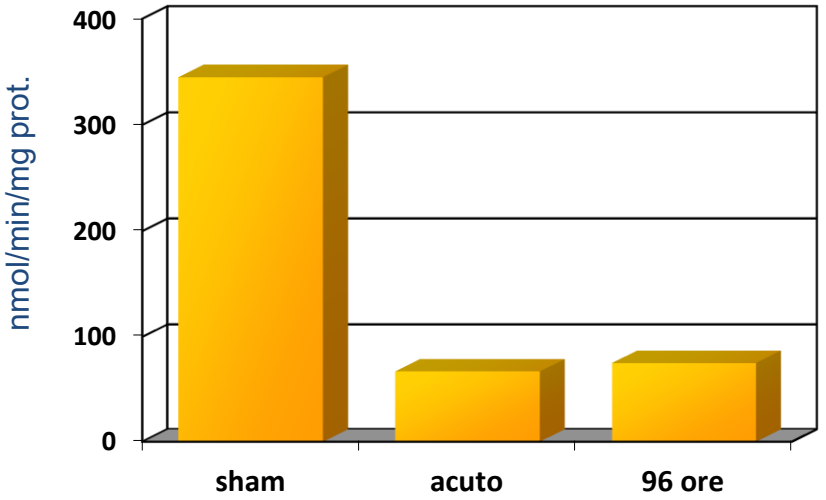


Persistenza delle alterazioni del metabolismo cellulare indotte da CO

PIRUVATO CHINASI



FOSFOFRUTTO CHINASI



Coccini T et al, 2004

Analisi retrospettive

N=28 Crocker PJ, 1985	%	N=74 Yarar et al, 2008 (1 – 18 anni)	%	N=30 Cho et al, 2008 (2-17 anni)	%
nausea	100	confusione mentale	52.7	alterazioni dello stato di coscienza	86.7
cefalea	93	vertigine	37.8	convulsioni	23.3
vomito	75	cefalea e nausea	32	cefalea e vertigini	10
letargia	63	vomito	31	vomito	3.3
sincope	56	sincope	23	-	-
convulsioni	3.5	astenia	16	-	-
-	-	convulsioni e coma	9.5	-	-
-	-	(6/74; 8.1%) asintomatici in PS; 4/6 CoHb 20-40 % (prelievo eseguito tra 30 minuti e 3 ore dal termine dell'esposizione)	-	intossicazione accidentali (16), volontarie (6) e secondarie a esposizione a fumi di incendio (8) (8/30; 26.7%) clinica più severa: acidosi metabolica severa (62.5%), ipotensione (25%) e convulsioni (25%)	-