



# Carbon monoxide poisoning: Recommendations for neuropsychological assessment

Simon B N Thompson<sup>1,2\*</sup>

<sup>1</sup>Professor of Clinical & Forensic Neuropsychology, Département de Psychologie, Université Paris Ouest, Nanterre La Défense, 200 Avenue de la République, 92000, France

<sup>2</sup>Formerly of Faculty of Law, Institute of Criminology, University of Cambridge, Sidgwick Ave, Cambridge CB3 9DA, UK

## \*Correspondence

Simon B N Thompson

Professor of Clinical & Forensic Neuropsychology, Département de Psychologie, Université Paris Ouest, Nanterre La Défense, 200 Avenue de la République, 92000, France

E-mail: nac.brain@hotmail.co.uk

- Received Date: 01 Jul 2022
- Accepted Date: 05 Jul 2022
- Publication Date: 08 Jul 2022

## Keywords

Assessment, Carbon Monoxide Poisoning, Diagnosis, Expert Witness Testimony, Neurological Damage, Neuropsychological Assessment, Neuropsychological Sequelae, Treatment

## Copyright

© 2022 Science Excel. This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International license.

## Abstract

Carbon monoxide poisoning is potentially significant in terms of mortality rates, and survivors can have serious deleterious neurological consequences. Diagnosis may be hampered because of symptoms that are similar to other benign conditions. Neuropsychological assessment is complicated due to the need to assess potentially global brain dysfunction. Assessment recommendations are presented and discussed together with an overview of treatment options.

## Introduction

### Carbon monoxide poisoning

The act of carbon monoxide (CO) entering a person's body is usually taken as being suspicious. Direct inhalation of CO as the result of a suicide attempt is familiar and may be catastrophic in terms of cognitive disability thereafter if the victim is found still alive. However, CO can enter via the lungs if inhaled unwittingly from the exhausts of a coal-burning or wood-burning stove fire.

Over two decades ago, CO poisoning was the most common cause of morbidity due to poisoning in the United States [1] and continues to affect about 50,000 people each year in the United States [2]. Typical clinical presentation is headache, dizziness, and coma, and with a mortality rate in the United States between 1 to 3 per cent although there is a significant number of victims who survive but suffer long-term neurological symptoms and conditions [3].

CO is a colourless, tasteless and odourless gas that is usually formed by the incomplete combustion of carbon compounds. A mechanism of toxicity is oxidative stress and impaired mitochondrial activity which sometimes makes it difficult to confirm diagnosis if the source of poisoning is unknown as symptoms can be similar to other benign disease processes [4]. Of course, the converse is true, i.e., if CO poisoning is unidentified and symptoms are attributed incorrectly to other neurological conditions then the consequences may be catastrophic and fatal [5-10]. Usually, the diagnosis can be confirmed with blood gases as most clinicians would agree that pulse

oximeters are not helpful because they tend to lack sensitivity and specificity.

CO binds to haemoglobin (Hb) in the blood forming COHb [2]. Exposure to 10 ppm of CO can lead to detectable COHb of approximately 2 per cent [11]. The World Health Organization suggests that levels that exceed 6 ppm are potentially toxic over a longer period of time [12]. In non-smokers, COHb levels of 2 per cent or greater, and 10 per cent or greater in smokers, is considered to produce symptoms [13].

For some time, CO poisoning has been known to be uniquely detrimental to myocardial tissue primarily because of its sensitivity to oxygen deprivation and particularly in patients who are pre-morbidly compromised such as cardiomyopathy, myocardial infarction and coronary artery disease [14]. Symptoms of CO cardiovascular toxicity include heart failure, myocardial infarction, pulmonary oedema, arrhythmias and myocardial dysfunction, which often subsides within 24 hours of exposure to CO [15].

### Cognitive decline, dementia and carbon monoxide poisoning

Deterioration in cognitive functioning is one of the signs of a dementia-process. However, there are more than 14 different types of dementia and each is characterised by origin or a specific set of symptoms [16-23]. CO can result in neurological conditions that may mimic other conditions [24-26].

Neurological and cognitive deterioration can lead to vascular dementia when small vessel disease and stroke is also involved [27-31] or as the result of mini strokes (e.g.,

**Citation:** Carbon monoxide poisoning: Recommendations for neuropsychological assessment. *Neurol Neurosci.* 2022; 3(2):1-4

transient ischaemic attacks or TIAs). Vascular dementia tends to have step-wise cognitive decline as opposed to the insidious and gradual decline characterised by Alzheimer's disease [32]. Psychological and physical trauma can also contribute to memory disorders, dementia and dementia-like presentations [33].

### Neuropsychological assessment recommendations

It is generally agreed that CO poisoning can result in global dysfunction as many regions of the brain can be affected that in turn cause neurological deficits and neuropsychological difficulties. Therefore, it is helpful to assess specific functions and of course, those functional problems that are salient in the affected patient.

**(i) Orientation** Asking specific questions that taps into the patient's knowledge and

awareness of their current location, home address, personal details (date of birth), name, and perhaps names of people they should know including famous figures (e.g., premier, president) and members of their own family.

**(ii) Logical Memory** (Auditory Verbal Memory) Using the Wechsler Memory Scale [34], the patient's memory is tested by reading out loud two fictitious stories and recording the responses of the patient. This assesses the patient's ability to remember information and again with a delay of about 20 minutes. Responses can be compared to comparative age-related norms drawn from the general population.

**(iii) Current Intelligence Quotient** Using the Wechsler Adult Intelligence Scale [35], the patient is assessed and compared with their age-related norms across a number of domains. Their Intelligence Quotient (IQ) can also be compared with an estimate of their premorbid IQ to see if there has been a change and attributable to CO poisoning (or other medical conditions or injuries according to their medical history). Subtests of the WAIS include:

**(a) Information** – taps the individual's fund of general knowledge, alertness to the environment and long-term memory;

**(b) Digit Span** – measures an individual's short-term auditory memory for number

sequences and reflects the individual's attention span and ability to concentrate;

**(c) Arithmetic** - requires the individual to solve numerical problems without the aid of pencil and paper where low scores may be associated with anxiety and poor concentration;

**(d) Comprehension** - taps common sense reasoning and the ability to exercise social

judgement in practical situations as well as the individual's degree of exposure to the mainstream culture;

**(e) Similarities** - calls for the ability to see relationships between things and ideas and to categorise them into logical groups; it also measures the capacity to form conceptual units from verbal material and to express these concepts in words;

**(f) Picture Completion** - measures the individual's alertness to visual detail and the ability to grasp the meaning of details within a complete picture using visual memory;

**(g) Picture Arrangement** - requires the individual to evaluate the social relevance of pictured situations, to anticipate the consequences of actions, to distinguish essential from irrelevant details and to possibly demonstrate planning ability;

**(h) Block Design** - measure of the ability to analyse abstract figures visually and construct them from their component parts using the ability to handle spatial relations;

**(i) Digit Symbol** - measures visuo-motor speed which is affected by visual memory, co-ordination, and the ability to learn non-verbal material.

**(iv) Estimate of Pre-Morbid Intelligence Quotient** Using the National Adult Reading Test [36], an estimate of the patient's premorbid IQ is obtained across verbal, performance and full-scale parameters which can then be compared with current values to discern any significant changes as compared with normative values.

**(v) Verbal Fluency** Controlled Oral Word Association Test [37] is an assessment of verbal fluency and assesses the patient's ability to produce words for each of three letters provided and compared to normative age-related values.

**(vi) Anxiety and Depression** Using the well-known Hospital Anxiety and Depression Scale [38], the patient is assessed on a number of statements that attempts to describe mood over categories that are considered to demonstrate levels of anxiety and depression.

**(vii) Test of Memory Malingering** This is a memory, recall and retention test that is considered to identify both malingering performances and degree of effort in tests [39]. It is particularly useful in identifying those patients who may be in denial, or who are trying to exaggerate their responses for financial gain (compensation) or for the achievement of other psychological gains.

**(viii) Trail Making Test** Trail Making Test [40] is a psychomotor task that tests information processing ability and visual-motor ability. It has part A, a relatively simple task, followed by part B which is a more complex task. Comparative age-related norms are available.

**(ix) New Learning** attempts to assess ability to learn new abstract concepts and the ability to learn new material in a trial-by-trial paradigm using word-pairs to be remembered and recalled [41].

**(x) Clinical Interview** This is a skilful tool that can sometimes reveal much more than simple testing paradigms. It is important not to use leading questions and to explore the use of both "open" and "closed" questioning dependent upon the type of information to be obtained.

### Treatment options

Biomarkers are increasingly being considered for diagnosis and prognosis of neurological disorders [23,42-45]. There is a growing trend towards presentation of biomarkers as evidence by court experts during legal trials [46,47].

Serious neurological sequelae are strongly predicted by prolonged loss of consciousness and elevated S-100 $\beta$  [48,49]. S100 $\beta$  is a cytoplasmic calcium-binding protein mainly expressed by glia. In serum or cerebrospinal fluid, it may predict lesion outcome and prognosis for brain or spinal cord injury [50].

The risk of neuropsychiatric sequelae may be reduced with hyperbaric oxygen but it has also been suggested that this might increase oxidative stress during recovery [48], or is complicated by barotrauma [51], seizures [52], pulmonary oedema [53], and claustrophobia [54].

Hyperbaric oxygen chambers for treatment regimens saw popularity following their use with deep sea divers who had

nitrogen narcosis after failing to re-compress appropriately or rose to the surface too quickly from deep dives [55].

It is now widely accepted that the administration of normobaric 100 percent oxygen is the therapy of choice for most cases of CO poisoning whereas hyperbaric oxygen therapy is reserved for the severe incidences of CO poisonings [56].

## Funding

This work was not funded and is the opinion of the author..

## Acknowledgement

I am grateful to my colleagues and friends for discussion and helpful comments. I am mindful of the valuable work at Fort Bovisand of the Late Dr Maurice Cross, Director of the Diving Diseases Research Centre, Plymouth, UK who was often the duty doctor and expert for those divers in need of urgent recompression using the hyperbaric chamber. He was gracious to my discussions and suggestions about research into the perception of cold in deep sea divers..

## References

- Ernst A, Zibrak JD. Carbon monoxide poisoning. *N Engl J Med*. 1998;339(22):1603-1608.
- Rose JJ, Wang L, Xu Q, et al. Carbon Monoxide Poisoning: Pathogenesis, Management, and Future Directions of Therapy. *Am J Respir Crit Care Med*. 2017;195(5):596-606..
- Smollin C, Olson K. Carbon monoxide poisoning (acute). *BMJ Clin Evid*. 2010;2010:2103.
- Nañagas KA, Penfound SJ, Kao LW. Carbon Monoxide Toxicity. *Emerg Med Clin North Am*. 2022;40(2):283-312.
- Kao LW, Nañagas KA. Toxicity associated with carbon monoxide. *Clin Lab Med*. 2006;26(1):99-125.
- Thompson SBN. Implications for cognitive rehabilitation and brain injury from exposure to Methyl Ethyl Ketone (MEK): a review. *Journal of Cognitive Rehabilitation*. 2010;28(Winter):4-14.
- Thompson SBN. Communicating with the Motor Cortex? Cortisol and Yawning as Possible Biomarkers for the Detection of Neurological Disease. *J Neurol Neurosci*. 2016, 7:S3.
- Thompson SBN. Hypothesis to explain yawning, cortisol rise, brain cooling and motor cortex involvement of involuntary arm movement in neurologically impaired patients. *J Neurol Neurosci*. 2017;8(1):1-5.
- Thompson SBN. Yawning as a Potential Diagnostic Indicator for Underlying Neurological Disorders. *Arch Neurol Neurol Disord*. 2022;5(1):132.
- Thompson SBN, Coleman A, Williams N. Yawning and cortisol levels in multiple sclerosis: Potential new diagnostic tool. *Mult Scler Relat Disord*. 2018;23:51-55.
- Raub JA, Mathieu-Nolf M, Hampson NB, Thom SR. Carbon monoxide poisoning--a public health perspective. *Toxicology*. 2000;145(1):1-14.
- Penney D, Benignus V, Kephelopoulou K, et al., 2010. WHO guidelines for indoor air quality: selected pollutants. Geneva: WHO.
- Hampson NB, Piantadosi CA, Thom SR, Weaver LK. Practice recommendations in the diagnosis, management, and prevention of carbon monoxide poisoning. *Am J Respir Crit Care Med*. 2012;186(11):1095-1101.
- Kalay N, Ozdogru I, Cetinkaya Y, et al. Cardiovascular effects of carbon monoxide poisoning. *Am J Cardiol*. 2007;99(3):322-324.
- Jung YS, Lee JS, Min YG, et al. Carbon monoxide-induced cardiomyopathy. *Circ J*. 2014;78(6):1437-1444.
- Kapur N, Scholey K, Moore E, et al. Long-term retention deficits in two cases of disproportionate retrograde amnesia. *J Cogn Neurosci*. 1996;8(5):416-434.
- Kapur N, Thompson S, Cook P, Lang D, Brice J. Anterograde but not retrograde memory loss following combined mammillary body and medial thalamic lesions. *Neuropsychologia*. 1996;34(1):1-8.
- North N, Thompson SBN. Neurological memory impairments. In P. Radcliffe G, Gudjonsson A, Heaton-Armstrong GG, Wolchover D (Eds.), *Witness testimony in sexual cases: evidential, investigative, and scientific perspectives* (2016). Oxford: Oxford University Press. pp.325-342
- Thompson SBN. *Dementia and memory: a handbook for students and professionals*. 2006. Aldershot: Ashgate.
- Thompson SBN. *Trauma psychology: clinical case histories, reviews, research*. 2016. Portsmouth: Blackwell-Yale-Academic.
- Thompson SBN. Hip Fracture As A Potential Contributor To Cognitive Decline And Dementia? *Neurol Neurosci*. 2022; 3(1):1-2.
- Thompson SBN, MacDonald J, Coates T. Improving visual memory with Aricept (Donepezil Hydrochloride, E2020) in mild-to-moderate Alzheimer's disease. *Clinical Gerontologist*. 2001;24(1/2):55-73.
- Thompson SBN, Daly S, Le Blanche A, et al., fMRI randomized study of mental and motor task performance and cortisol levels to potentiate cortisol as a new diagnostic biomarker. *Journal of Neurology & Neuroscience*. 2016;7(2):92: 1-8.
- Quinn DK, McGahee SM, Politte LC, et al. Complications of carbon monoxide poisoning: a case discussion and review of the literature. *Prim Care Companion J Clin Psychiatry*. 2009;11(2):74-79.
- Thompson SBN. Health psychology intervention - identifying early symptoms in neurological disorders. *International Journal of Medical, Health, Biomedical, Bioengineering & Pharmaceutical Engineering*. 2015;9(4):351-355.
- Kumarihamy P, Kularatne SAM, Pathirage M, Gunaratne WMSN, Waduge R. A case of delayed neurological manifestation following carbon monoxide poisoning in Sri Lanka: epidemiology of exposure and literature review. *BMC Pharmacol Toxicol*. 2019;20(1):17.
- Thompson SBN. Rehabilitation of cognitive and emotional problems. In R. Fawcus (Ed.), *Stroke rehabilitation: a collaborative approach*. (1999). Oxford: Blackwell. pp.147-159
- Thompson SBN. Art meets Science: empowering stroke patients to regain muscular control through creative graphics technology, psycho-physiology and neuroplasticity. *International Journal of Arts & Sciences*. 2012;5(4):79-85.
- Thompson SBN, Coleman MJ. A quantitative assessment of neuromuscular function for use with unilateral cerebrovascular accident patients. *International Journal of Rehabilitation Research*. 1987;10(3):312-316.
- Thompson SBN, Coleman MJ. An interactive microcomputer-based system for the assessment and prognosis of stroke patients. *Journal of Microcomputer Applications*. 1989;12(1):33-40.
- Thompson SBN, Morgan M. *Occupational therapy for stroke rehabilitation*. 1996. London: Chapman & Hall.
- Thompson SBN. Memory decline, Alzheimer's disease and vascular dementia: the clinical picture. *Journal of Cognitive Rehabilitation*. 2002;20(2):12-18.
- Thompson SBN. Saliva cortisol and yawning as a predictor of neurological disease. *International Journal of Medical, Health, Biomedical, Bioengineering & Pharmaceutical Engineering*, 2016;10(4):184-187.
- Wechsler D. *Wechsler Memory Scale – Fourth UK Edition (WMS-IV)*. 2010. Psychological Corporation: San Antonio, TX.
- Wechsler D. *Wechsler Adult Intelligence Scale – Fourth Edition*

- (WAIS-IV). 2008. Psychological Corporation: San Antonio, TX.
36. Nelson HE, Willison J. The National Adult Reading Test (NART). Windsor: NFER-Nelson. 1991.
  37. Patterson J. Controlled Oral Word Association Test. In Kreutzer JS, DeLuca J, Caplan B, (eds) Encyclopedia of Clinical Neuropsychology. 2011. Springer: New York, NY.
  38. Zigmond AS, Snaith RP. The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica*. 1983;67(6):361-370.
  39. Tombaugh TN. TOMM. Test of Memory Malingering. 1996. Multi-Health Systems Inc.: North Tonawanda, NY.
  40. Tombaugh TN. Trail Making Test A and B: Normative data stratified by age and education. *Archives of Clinical Neuropsychology*. 2004;19(2):203-214.
  41. Barnett JH, Blackwell AD, Sahakian BJ, Robbins TW. The Paired Associates Learning (PAL) Test: 30 Years of CANTAB Translational Neuroscience from Laboratory to Bedside in Dementia Research. *Curr Top Behav Neurosci*. 2016;28:449-474.
  42. Thompson SBN. Pathways to yawning: making sense of the Thompson Cortisol Hypothesis. *Medical Research Archives*. 2015;3:1-7.
  43. Thompson SBN. Diagnostic biomarkers – exploring the potential of cortisol and yawning in the detection of neurological disease processes. *Journal of Neurology & Neuroscience*. 2016;7(3):112:1-2.
  44. Thompson SBN. Richer S. How yawning and cortisol regulates the attentional network. *Journal of Neuroscience & Rehabilitation*. 2015;2(1):1-9.
  45. Thompson SBN, Simonsen M. Yawning As a New Potential Diagnostic Marker for Neurological Diseases. *J Neurol Neurosci*. 2016, 6:3.
  46. Thompson SBN. Testamentary capacity and cognitive rehabilitation: implications for head-injured and neurologically impaired individuals. *Journal of Cognitive Rehabilitation*. 2009;27(Fall):11-13.
  47. Thompson SBN. Misrepresentation of expert witness testimony in neurology for neurological sequelae and disorders explained by situational action theory. *Archives of Neurology & Neurological Disorders*. 2022;5(1):1-20.
  48. Chiew AL, Buckley NA. Carbon monoxide poisoning in the 21st century. *Crit Care*. 2014;18(2):221.
  49. Hafez AS, El-Sarnagawy GN. S-100 $\beta$  in predicting the need of hyperbaric oxygen in CO-induced delayed neurological sequelae. *Hum Exp Toxicol*. 2020;39(5):614-623.
  50. Kanner AA, Marchi N, Fazio V, et al. Serum S100beta: a noninvasive marker of blood-brain barrier function and brain lesions. *Cancer*. 2003;97(11):2806-2813.
  51. Weaver LK. Clinical practice. Carbon monoxide poisoning. *N Engl J Med*. 2009;360(12):1217-1225.
  52. Sanders RW, Katz KD, Suyama J, et al. Seizure during hyperbaric oxygen therapy for carbon monoxide toxicity: a case series and five-year experience. *J Emerg Med*. 2012;42(4):e69-e72.
  53. Wu CT, Huang JL, Hsia SH. Acute carbon monoxide poisoning with severe cardiopulmonary compromise: a case report. *Cases J*. 2009;2(1):52.
  54. Hillard JR. Severe claustrophobia in a patient requiring hyperbaric oxygen treatment. *Psychosomatics*. 1990;31(1):107-108.
  55. Vrijdag XC, van Waart H, Sleigh JW, Mitchell SJ. Pupillometry is not sensitive to gas narcosis in divers breathing hyperbaric air or normobaric nitrous oxide. *Diving Hyperb Med*. 2020;50(2):115-120.
  56. Ilano AL, Raffin TA. Management of carbon monoxide poisoning. *Chest*. 1990;97(1):165-169.