

Clinical Records

Carbon monoxide poisoning and sensorineural hearing loss

M. SHAHBAZ HASSAN, M.R.C.S., J. RAY, F.R.C.S.*, F. WILSON, F.R.C.S.†

Abstract

Exposure to carbon monoxide is a well-recognized cause of morbidity and mortality. Both acute accidental poisoning and chronic exposure are associated with a range of adverse health effects. We report two cases of carbon monoxide poisoning with the associated phenomenon of sensorineural hearing loss. Although hearing loss as a result of acute carbon monoxide exposure has previously been described, here we emphasize the need to consider chronic exposure to carbon monoxide as a potentially reversible cause of sensorineural hearing loss if diagnosed and treated early.

Key words: Carbon Monoxide Poisoning; Hearing Loss; Sensorineural

Introduction

Carbon monoxide (CO) is a common indoor pollutant and an important cause of adverse health effects. In the UK domestic exposure at levels exceeding current air quality guidelines is predominantly the result of malfunctioning or poorly ventilated fuel burning appliances¹ and is associated with an estimated 50 fatalities each year.² The peak

incidence of deaths occurs during the autumn and winter months in the northern hemisphere.² Exposure to high levels of CO most commonly causes minor symptoms of headache, nausea and dizziness. In survivors of acute poisoning and in those subject to chronic exposure however, CO intoxication may also be associated with more disabling neurological symptoms. Sensorineural hearing loss in association with acute CO poisoning is

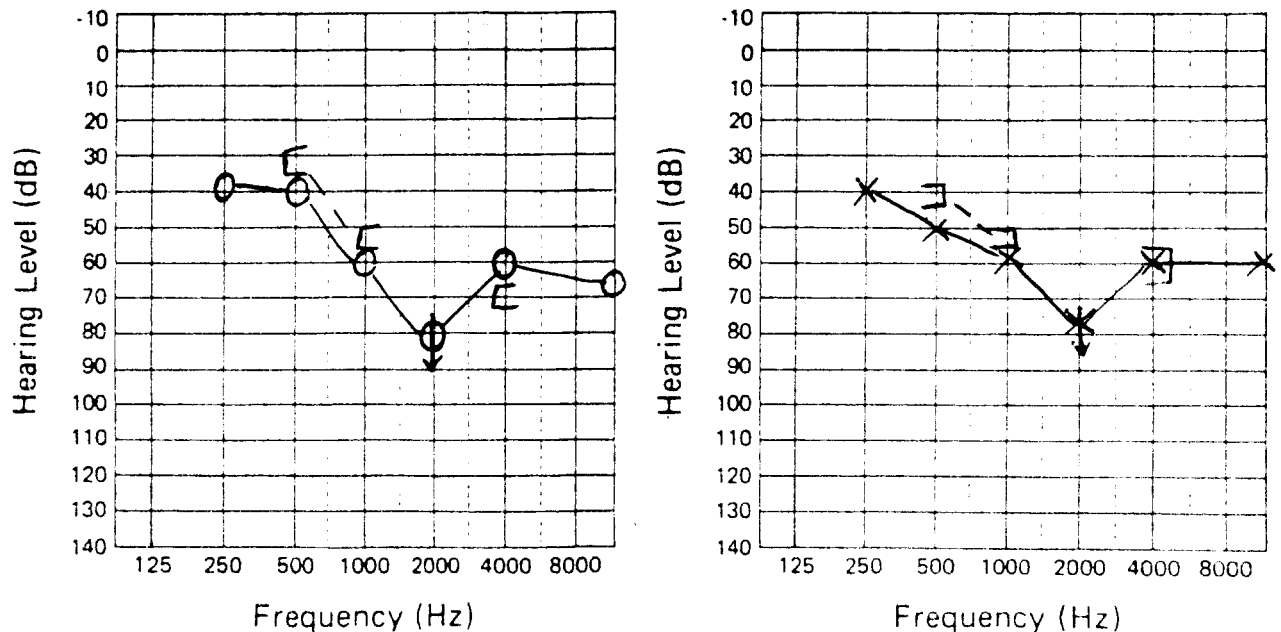


FIG. 1

Case 1: audiogram on day 6 following admission for acute CO poisoning showing bilateral sensorineural hearing loss.

From the Departments of ENT, Queen Elizabeth Hospital, Birmingham, West Midlands Rotation* and the Royal Wolverhampton Hospitals NHS Trust†, UK.

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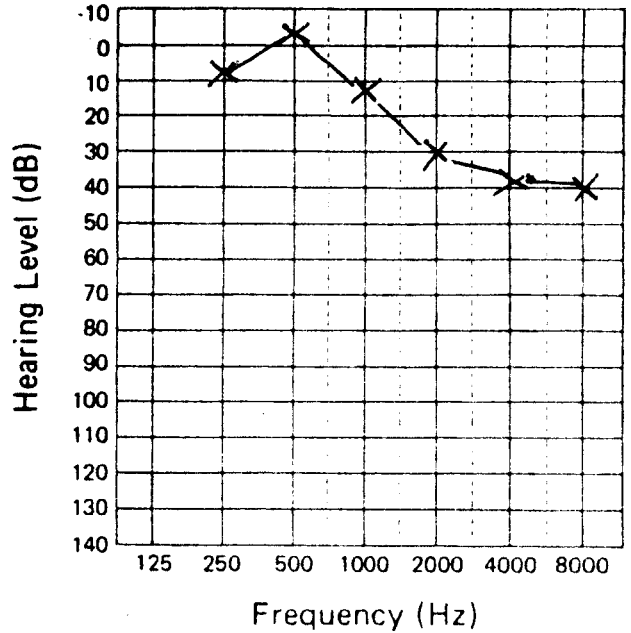
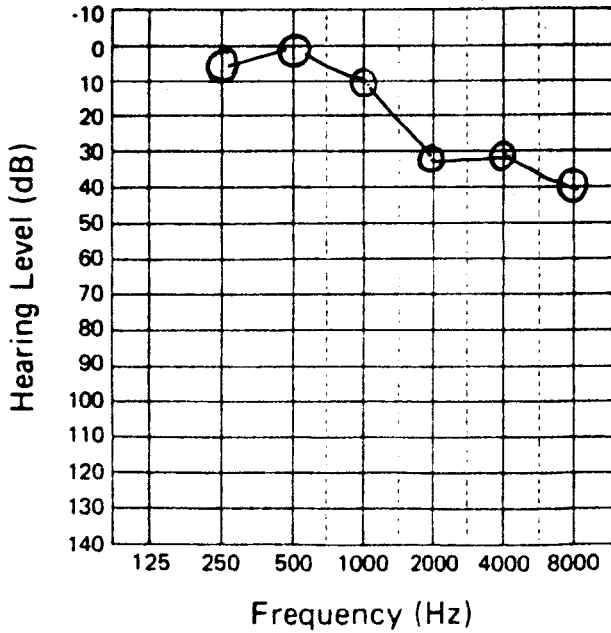


FIG. 2

Case 1: audiogram 2 months following acute CO poisoning showing some recovery in hearing.

one such symptom that has previously been described in the literature. It is less well recognized that the same effect may occur as a result of chronic lower dose exposure. We report two different cases of carbon monoxide poisoning, one acute and one chronic but both associated with sensorineural hearing loss.

Case reports

Case 1

A 30-year-old man was found in unconscious after repairing his car in a closed garage with the car engine running. He had no previous history of hearing loss or

otological complaint. There was no suggestion of suicidal intent. On presentation at hospital, he was deeply comatose with no clinical signs of cyanosis. Arterial blood gas analysis showed 29.9 per cent carboxyhaemoglobin, 69.3 per cent oxyhaemoglobin and 0.4 per cent methaemoglobin. He was treated with continuous 100 per cent oxygen. His arterial carboxyhaemoglobin content reduced to 6.6 per cent and four hours, and 1.8 per cent after seven hours, while his oxyhaemoglobin content increased to 96.9 per cent. Eighteen hours following admission, he regained consciousness but was suffering with amnesia, blurred vision, deafness and tinnitus. His vision rapidly returned to normal. Pure tone audiometry

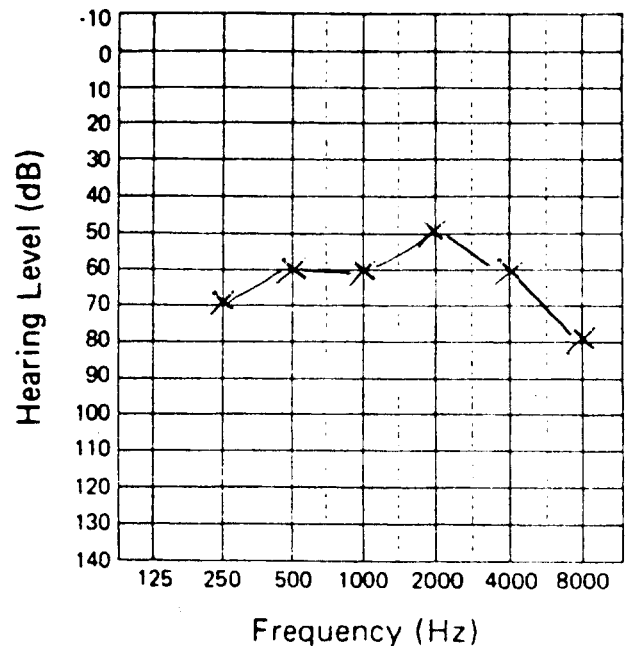
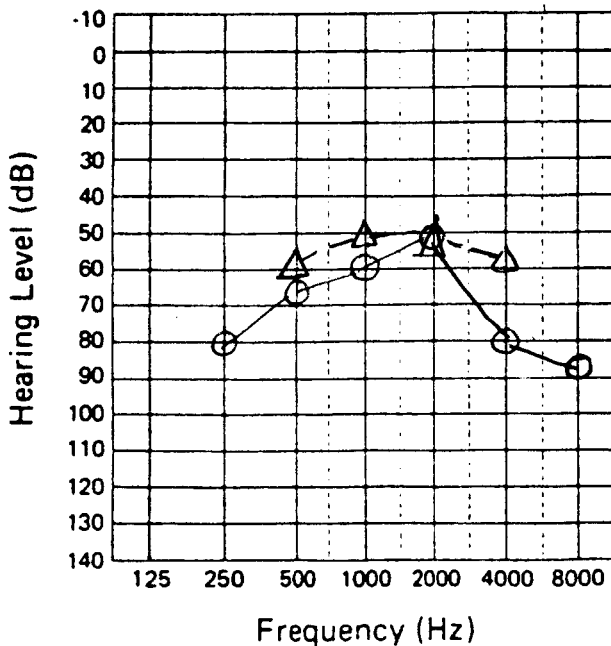


FIG. 3

Case 2: audiogram following chronic CO poisoning showing bilateral sensorineural hearing loss.

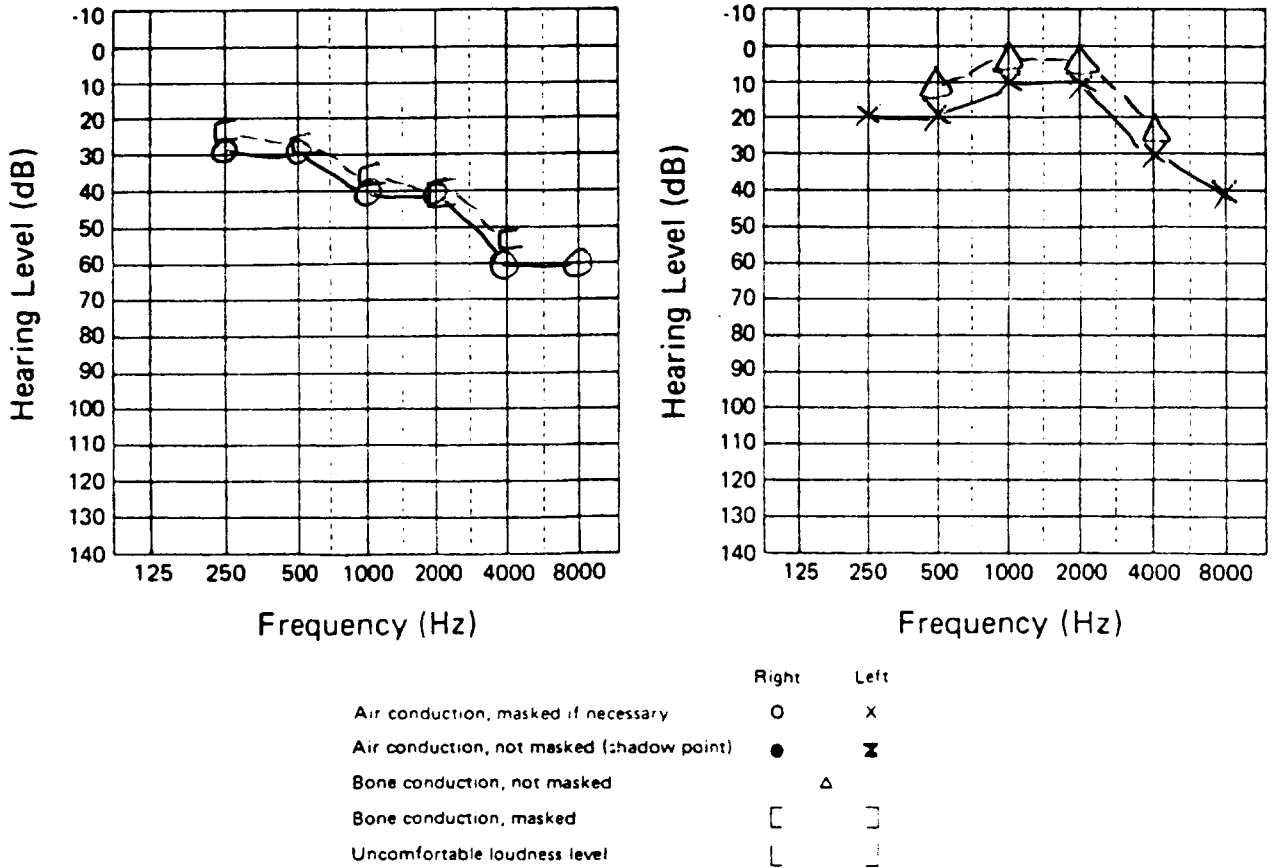


FIG. 4
Case 2: audiogram six weeks following chronic CO poisoning showing improvement in hearing.

on day six following admission, showed a bilateral moderate to severe sensorineural hearing loss (Figure 1). He was fitted with a hearing aid and no further treatment was necessary. His hearing showed partial recovery after two months with residual hearing loss between 1 to 8 kHz (Figure 2). No further hearing recovery was apparent after six months. He was able to cope well with his level of hearing loss and persistent tinnitus.

Case 2

A 61-year-old lady who lived alone in a bungalow presented with a two-week history of bilateral hearing loss, increasing dizziness and lethargy. There was no history of tinnitus or other otological symptoms. There was no previous history of hearing loss. Detailed otoneurological examination was unremarkable. Pure tone audiometry showed bilateral moderate sensorineural hearing loss (Figure 3), that was consistent with tuning fork and voice tests. The accuracy of the thresholds was confirmed by cortical evoked responses. Magnetic resonance imaging (MRI) scans excluded any intracranial cause.

During the course of her admission a fault with a gas fire at her home was found and high levels of carbon monoxide were detected. Elevated carboxyhaemoglobin levels then confirmed chronic exposure and treatment with continuous high flow oxygen was instigated. Following this, the patient made a good recovery with rapid resolution of her dizziness and lethargy and gradual improvement in her hearing (Figure 4).

Discussion

Our reported cases illustrated that both acute and chronic CO intoxication may be associated with sensorineural hearing loss.

As early as 1948, a report by Lumio documented a 78 per cent incidence of sensorineural hearing loss among 700 cases of chronic CO exposure.³ Since then however, reports have been limited to those patients presenting with hearing loss in association with acute CO poisoning. In 1967 Garland and Pearce reported four cases of accidental carbon monoxide intoxication with slight to moderate hearing loss, which apparently improved over 24 hours.⁴ Morris reported a case in 1969 of severe bilateral sensorineural hearing loss, that partly improved over a period of 11 months.⁵ In a study of 15 coal miners who survived a disastrous mine explosion in 1963, Makashima described mild sensorineural hearing loss.⁶ Young *et al.* reported that in an animal model carbon monoxide exposure potentiated high frequency auditory threshold shifts induced by noise.⁷

The auditory dysfunction produced by CO appears to be frequency specific and although the mechanism of this local cochlear effect remains unclear, one possibility is that the basal (high-frequency) region of the cochlea is selectively vulnerable to the effects of CO.⁷ Consistently, in our cases, CO poisoning appeared to affect high frequency hearing, i.e. 1 to 8 kHz.

Recognized treatment of CO poisoning consists of removal from the source of exposure and oxygen therapy. Although both 100 per cent normobaric oxygen and hyperbaric oxygen are accepted treatments, it remains unclear on present evidence whether hyperbaric oxygen

offers substantial advantage in clinical poisoning.⁸ Both of our patients, after removal from the CO source, were treated with high flow continuous oxygen and both showed gradual improvement in their hearing thresholds.

In the UK, 225 non-fatal injuries due to carbon monoxide poisoning were recorded during 1999–2000.⁹ Whilst the occurrence of CO poisoning in the home or workplace is far from uncommon⁸ the commonest symptoms are easily confused with those of other ailments. Patients frequently report vague symptoms of mild dizziness, headache and lethargy and as a result, missed diagnoses of CO intoxication may occur. Our second case effectively illustrates that chronic CO intoxication, if considered, may be found to be the underlying cause in cases of unexplained sensorineural hearing loss. For this reason, in the out-patient setting carboxyhaemoglobin measurement or breath testing, both inexpensive and non-invasive, could be added to routine investigations for unexplained hearing loss. This may be most appropriate when other associated symptoms are present.

There is a lack of evidence on which to base estimates of the incidence of CO-related sensorineural hearing loss. By recognition of this phenomenon however, the simple measures of removing the patient from the source of exposure and instigating oxygen therapy may reduce morbidity and improve recovery from any consequent sensorineural hearing loss.

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Address for correspondence:

Mr M. Shahbaz Hassan,
Department of ENT/Head and Neck Surgery,
University Hospital Birmingham, Queen Elizabeth,
Edgbaston,
Birmingham B15 2TH, UK.

Fax: +44 121 627 2299

E-mail: shahbazhassan@hotmail.com

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