Carbon monoxide poisoning: correlation of neurological findings between accident and emergency departments and a hyperbaric unit

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Abstract

Objectives—To investigate and quantify the differences in neurological examination findings in patients acutely poisoned with carbon monoxide, between initial assessment at accident and emergency (A&E) departments and subsequently at a hyperbaric unit.

Methods—Retrospective case note review of all patients referred to the Hull Hyperbaric Unit for treatment of acute carbon monoxide poisoning between August 1998 and August 1999. Patients who were ventilated or less than 16 years old were excluded because of difficulty in assessing their neurological status.

Results—Thirty patients were included for analysis. The mean duration from exposure to assessment in A&E was four hours while patients were reviewed on average three hours later at the hyperbaric unit. Referrals came from 14 different hospitals. A history of loss of consciousness accounted for 70% of referrals. A mean of 3.2 neurological signs per patient was documented in A&E compared with 9.2 at the hyperbaric unit. Seventy nine per cent of abnormal neurological signs were not detected at A&E departments compared with 3% at the hyperbaric unit. The major source of discrepancy was in sharpened Romberg's test and heel-toe gait, in 13% of patients examined in A&E departments these signs were recorded as abnormal compared with 90% at the hyperbaric unit.

Conclusion—There is a large discrepancy in neurological findings between assessment in A&E departments and the Hull Hyperbaric Unit. A number of factors may account for this including interobserver variation, patient deterioration during transfer, poor documentation, lack of understanding of the sequelae of carbon monoxide poisoning and inadequate examinations. Further research is required to quantify the impact of the various factors that may contribute to the differences in neurological findings.

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Carbon monoxide poisoning accounts for a significant number of deaths in England and Wales each year,¹ and is estimated to be the leading cause of deaths from poisoning in both the United States² and the United Kingdom.³

Patients with sub-lethal exposure may exhibit abnormalities in cerebellar signs including sharpened Romberg's test⁴ and heel-toe gait.⁵

Hyperbaric oxygen therapy has been shown to significantly reduce the incidence of neuropsychiatric sequelae after carbon monoxide poisoning compared with treatment using normobaric oxygen therapy.⁶ However, its role remains controversial.⁷

Indications for hyperbaric oxygen therapy in acute carbon monoxide poisoning⁸ include history of loss of consciousness, unconsciousness, neurological abnormality, cardiac ischaemia⁹ and pregnancy.¹⁰

The carboxyhaemoglobin levels do not correlate well with the severity of poisoning.¹¹ Furthermore, as the diagnosis of carbon monoxide poisoning (sub-lethal exposure to carbon monoxide) is not always immediately apparent¹² and the examination findings are often subtle,¹³ careful neurological examination is essential.

In our clinical practice we have noticed a number of patients referred for hyperbaric oxygen therapy for treatment of carbon monoxide poisoning who exhibit remarkably different neurological signs than those that were present before transfer. As we are not aware of any previous studies that compared neurological findings elicited on examination at the time of presentation to an accident and emergency (A&E) department with that detected at a hyperbaric unit, we undertook this retrospective review following our first full year in operation.

Methods

A retrospective case note review was conducted on patients acutely poisoned with carbon monoxide and referred to the Hull hyperbaric unit for treatment between August 1998 and August 1999.

Data were collected from both the A&E and hyperbaric unit notes to identify any differences or omissions between the neurological examinations at either centre. Standard assessment forms were not used at the hyperbaric unit; thus all examinations were recorded on blank paper. Two patients, whose neurological status was documented as normal or "no focal deficit" on referral, were recorded as normal for all parameters during data collection.

All A&E departments referring patients for hyperbaric treatment for acute carbon monoxide poisoning were included in the study. It was not possible to identify the grade and clinical experience of the doctors who performed the initial assessment in the A&E departments. At

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Table 1 Sources of carbon monoxide poisoning

Gas boiler	15 (50)
Car exhaust	8 (26.7)
House fire	4 (13.3)
Gas operated machine	3 (10)

Percentages shown in parentheses.

Table 2 Reason for referral

Loss of consciousness	21 (70)
Neurological abnormality	5 (16.7)
Pregnant	3 (10)
Not stated	1 (3.3)

Percentages shown in parentheses.

the hyperbaric unit, 28 patients were examined by a consultant and two by a specialist registrar.

In particular the notes were scrutinised for documentation of sharpened Romberg's test, heel-toe gait and presence of any cerebellar signs, duration from exposure to examination at both the A&E departments and the hyperbaric unit. Sharpened Romberg's test was deemed to be abnormal if a patient was unable to stand in a heel-toe position with eyes closed for at least 30 seconds.¹⁴

Patients who were ventilated or less than 16 years old were excluded because of the difficulty in assessing their neurological status.

Results

A total of 52 patients, deemed to meet national criteria, were treated at the Hull Hyperbaric Chamber for carbon monoxide poisoning during the study period. Eight patients were excluded because they were less than 16 years

old and nine because they were ventilated thus making neurological assessment either unreliable or impossible. Of the remaining 35 we were unable to find any notes for five patients. The following results are based on the remaining 30 patients.

Eighteen male patients mean age 38.7 years (range 16–77) and 12 female patients mean age 32.3 years (range 18–53) were treated during the study period, 50% during the period November to February inclusive.

The most common source of carbon monoxide poisoning was faulty gas boilers (table 1) and the principal reason for referral for hyperbaric treatment was a history of loss of consciousness (table 2).

The mean carboxyhaemoglobin level was 28% (5–52.9%). The mean duration from exposure to examination in an A&E department was four hours while the mean interval between examination in A&E and the hyperbaric unit was three hours (fig 1). A total of 14 different A&E departments provided referrals.

Ninety seven neurological signs in total were documented in the A&E departments (mean per patient = 3.2) and of these 76 were recorded as normal and 21 as abnormal (table 3). In comparison 275 signs (mean per patient = 9.2) were documented at the hyperbaric unit with 177 of these recorded as normal and 98 as abnormal.

Only one patient examined in A&E had more neurological signs documented compared with examination at the hyperbaric unit, however the four abnormal neurological signs identified at the hyperbaric unit were not

Figure 1 Times from exposure to assessment.

Table 3 Discrepancies between neurological findings

Neurological sign	Hyperbaric unit			Accident and emergency		
	normal	abnormal	not stated	normal	abnormal	not stated
Sharpened Rhomberts	3	27	0	3	4	23
Heel-toe gait	3	27	0	3	4	23
Ataxia	4	8	18	2	1	27
Dysdiadochokinesis	16	8	6	3	1	26
Finger nose testing	9	6	15	4	1	25
Serial sevens	3	6	21	2	2	26
Fine movements	19	3	8	3	0	27
Nystagmus	14	3	13	4	0	26
Reflexes	22	2	6	11	1	18
GCS	21	3	6	17	4	9
CNS	22	1	7	7	0	23
PNS	23	1	6	10	1	19
Intention tremor	10	1	19	3	0	27
Speech	8	2	20	4	2	24

Total number of patients = 30. GCS = Glasgow coma score, CNS = central nervous system, PNS = peripheral nervous system.

detected at the referring A&E department. Altogether only four patients (13.3%) had both identical neurological examinations documented and identical abnormal signs recorded based on their examination at both centres.

Eighteen patients examined in A&E departments had no abnormal neurological signs identified compared with only two at the hyperbaric unit. The indications for accepting these patients for hyperbaric oxygen therapy are detailed in table 2. Of these 18 patients, 13 had no neurological examination documented in their A&E department case notes compared with only one patient examined at the hyperbaric unit.

The major sources of discrepancy between the neurological examination findings are listed in table 3. It is readily apparent that both the sharpened Rhombert's test and heel-toe gait were poorly recorded at the referring A&E departments. In 23 patients (76.6%) these signs were not recorded at the source of referral and were subsequently elicited as abnormal in 20 of these 23 patients at the hyperbaric facility. In three patients these signs were recorded as normal when on subsequent assessment at the hyperbaric unit they were found to be abnormal. Of the four patients whose sharpened Rhombert's test and heel-toe gait were assessed as abnormal at the referring A&E department these findings were identical when reviewed at the hyperbaric unit. Thus, 27 patients (90%) had abnormal sharpened Rhombert's test and heel-toe gait when examined at the hyperbaric unit compared with 13% (four patients) in A&E.

Other significant differences occurred with the assessment and documentation of dysdiadochokinesis, finger nose testing, serial sevens, fine movements, nystagmus and ataxia.

Differences also occurred when the A&E departments detected abnormalities in speech, Glasgow Coma Score and power in three separate patients that were not recorded at the hyperbaric unit. Ninety seven per cent of abnormal neurological signs were detected at the hyperbaric unit but only 21% of these were documented in A&E departments.

There was no appreciable difference in the number of abnormal neurological signs identified between those patients who presented

Table 4 Neurological signs identified in patients presenting with self inflicted and accidental carbon monoxide poisoning

Exposure	Number of patients	Neurological signs	
		Accident and emergency	Hyperbaric unit
Self inflicted	8	16 (2)	70 (8.7)
Accidental	24	81 (3.4)	205 (8.5)
Total	30	97 (3.2)	275 (9.2)

Mean values in parentheses.

after self inflicted carbon monoxide poisoning and those after accidental exposure (table 4).

Discussion

Recent publications have highlighted the importance of carbon monoxide poisoning¹⁵ and this indeed has been emphasised by the Chief Medical Officer writing to all medical practitioners in the United Kingdom in 1998⁵ highlighting the risk of carbon monoxide poisoning and its propensity to go undetected. Within the group studied only those deemed to meet the recommended criteria⁸ for hyperbaric oxygen therapy have been seen.

As reported by Hamilton-Farrell⁸ the peak incidence for acute carbon monoxide poisoning referrals to hyperbaric facilities is from November to February inclusive and our data are no different. However, he reported a mean interval of nine hours and fifteen minutes after removal from exposure to arrival in a hyperbaric facility. Our patients experienced a much shorter delay of seven hours. However, this is still outside the six hour interval of therapeutic benefit as evident in the work of Goulon *et al.*¹⁶

The observed differences in examination findings and documentation may be explained in a number of ways. Some examples of which are (1) Interobserver variation may account for the reported differences. (2) The disease process may be in evolution and thus from initial examination to subsequent evaluation at the receiving hyperbaric facility the patient's clinical condition may have altered or deteriorated. Indeed this would be consistent with the hypothesis of Thom¹⁷ who has suggested that there may be a component of hypoxia induced reperfusion injury within the pathophysiology of carbon monoxide poisoning. (3) As was apparent earlier in this paper, sharpened Rhombert's test and heel-toe gait accounted for the main source of discrepancy between both sets of examinations. The explanation for this may lie in the fact that patients often are only examined on the A&E department trolleys and therefore abnormalities in their gait and balance will easily be missed. (4) Patients may have been adequately examined but these findings may have been poorly recorded in their notes. (5) Patients may have been inadequately examined and this may be because of a lack of understanding of the neurological sequelae of carbon monoxide poisoning.

Additionally, it is possible that neurological examinations in A&E were curtailed once an indication for hyperbaric oxygen therapy was identified. Documentation of subtle neurological signs in these instances would not have

changed acute management. Conversely at the hyperbaric unit careful documentation of all neurological signs plays a crucial part in identifying the response to treatment and possible need for additional treatment sessions. Furthermore, neurological examinations were performed by consultants in all but two cases, at the hyperbaric unit, possibly reflecting the higher documentation rate of both normal and abnormal signs.

In the present era of clinical governance¹⁸ we need to demonstrate that we are treating patients appropriately in all aspects of their care including performing adequate examinations, making the correct diagnosis together with accurate documentation of our findings. A history of loss of consciousness prompted referral in 70% of patients. Thus the referring clinicians correctly identified loss of consciousness as an important indicator of serious poisoning requiring referral to a hyperbaric facility.

As this study is retrospective and involves only a small number of patients, these factors may be construed to be limitations. However, we believe that this paper reflects current clinical practice. Had it been performed prospectively it would have been difficult to quantify the impact that the Hawthorne effect¹⁹ would have had on the results.

Regardless of the fact that there is an element of equipoise in relation to the efficacy of hyperbaric oxygen treatment in carbon monoxide poisoned patients⁷⁻²⁰ it is incumbent on the medical profession who come into contact with these patients to make the correct diagnosis and record any neurological findings.

With the aforementioned in mind and in the light of the documented discrepancies in neurological findings, it is our intention to carry out a prospective multicentre study to identify which of the above possible explanations has the greatest impact on the recorded differences in neurological findings.

Conclusions

Significant differences in neurological examination findings between assessment at A&E departments and that at the Hull hyperbaric unit were noted. These differences may reflect interobserver variation, poor documentation of examination findings by doctors, a lack of understanding of the neurological abnormalities, which may be present after acute carbon monoxide poisoning, or be a reflection of inadequate examinations performed by doctors. Therefore in any patient presenting with carbon monoxide poisoning we suggest that an integral part of their examination should include getting the patient off the trolley and assessing their heel-toe gait and sharpened Rhomberg's test.³ These tests not only demonstrate abnormal neurology but also were closely correlated when examined both in A&E and the hyperbaric unit. Additionally, the sharpened Rhomberg's test is objective when measured against the clock.

An additional explanation is that some patients may deteriorate during the time inter-

val from initial assessment in an A&E department to that at a hyperbaric unit, as there is often a considerable distance between these centres. To help clarify this situation we plan to conduct a prospective multicentre trial in the near future.

Education should be directed at A&E doctors aimed at increasing their understanding of the neurological sequelae of carbon monoxide poisoning and improving their documentation of both relevant normal and abnormal examination findings.

As a result of this study we are issuing all A&E departments that have referred patients to Hull hyperbaric unit in the past year, with a summary of the relevant neurological signs that should be sought, and their presence or absence recorded clearly in the patients' notes.

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Contributors to paper

Richard Lynch discussed core ideas, collected the data, reviewed the literature, read the relevant journals, analysed the data and wrote the manuscript. Gerard Laden discussed core ideas, reviewed the literature and read relevant journals, assisted in writing and editing the manuscript. Paul Grout initiated the study, discussed core ideas and contributed to editing the manuscript. Richard Lynch will act as guarantor for this paper.

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