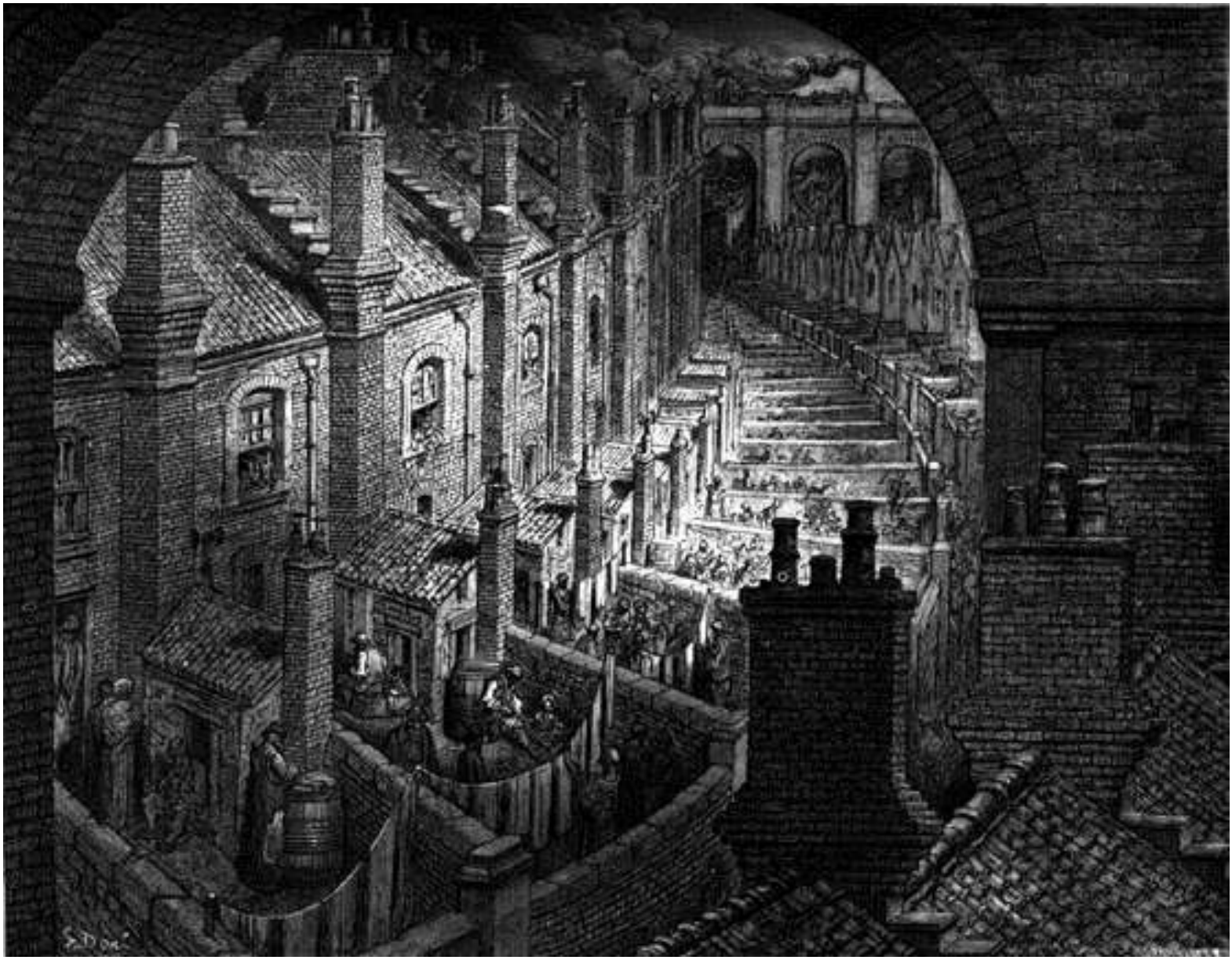
THE NORTHERN HOSPITAL

CARBON MONOXIDE POISONING



"Over the city by railway from London", woodcut print, Gustave Dore, 1872.

The following is an extract of evidence taken before the Parliamentary Committee on "Climbing Boys" in 1817 concerning the death of a boy aged eight, who most probably succumbed to the combined effects of burns and carbon monoxide poisoning.

On Monday morning, 29 March 1813, a chimney sweeper of the name of Griggs attended to sweep a small chimney in the brewhouse of Messrs Calvert and Co. in Upper Thames Street; he was accompanied by one of his boys, a lad of about eight years of age, of the

name of Thomas Pitt. The fire had been lighted as early as 2 o'clock the same morning and was burning on the arrival of Griggs and his little boy at eight. The fireplace was small and an iron pipe projected from the grate some little way into the flue. This the master was acquainted with (having swept the chimneys in the brewhouse for some years) and therefore had a tile or two broken from the roof, in order that the boy might descend the chimney. He had no sooner extinguished the fire than he suffered the lad to go down; and the consequence, as might be expected, was his almost immediate death, in a state, no doubt, of inexpressible agony.

The flue was of the narrowest description and must have retained heat sufficient to have prevented the child's return to the top, even supposing he had not approached the pipe belonging to the grate, which must have been nearly red hot; this however was not clearly ascertained on the inquest, though the appearance of the body would induce an opinion that he had been unavoidably pressed against the pipe. Soon after his descent, the master, who remained on the top, was apprehensive that something had happened and therefore desired him to come up; the answer of the boy was "I cannot come up master, I must die here". An alarm was given in the brewhouse immediately that he had stuck in the chimney, and a bricklayer who was at work near the spot attended, and after knocking down part of the brickwork of the chimney, just above the fireplace, made a hole sufficiently large to draw him through.

A surgeon attended, but all attempts to restore life were ineffectual. On inspecting the body, various burns appeared; the fleshy part of the legs and a great part of the feet more particularly were injured; those parts too by which climbing boys most effectually ascend or descend chimneys, viz. the elbows and knees seemed burnt to the bone; from which it must be evident that the unhappy sufferer made some attempts to return as soon as the horrors of his situation became apparent.

The committee recommended that the use of climbing boys be prohibited but it was not until 1864, after many years of campaigning, that an Act of Parliament was finally approved by the House of Lords, to outlaw the use of children for "climbing chimneys".

From "The Faber Book of Reportage",
Ed John Carey

Left: Child factory laborer, late Nineteenth Century Britain, identity unknown.

CARBON MONOXIDE POISONING

Introduction

Carbon monoxide (CO) poisoning is one of the commonest causes of death from poisoning.

Acute toxic effects are those of tissue hypoxia.

CO poisoning also has the potential for long term neurological sequelae.

Sources of exposure:

The usual clinical settings in which CO poisoning occurs include:

1. Fires, especially within a confined space.
    - The possibility of cyanide poisoning should also be kept in mind with fires.
2. Incomplete combustion of hydrocarbon fuels.
    - Stoves and portable heaters within a confined space.
    - Intentional self-harm, (most commonly by automobile exhaust).

Less commonly:

3. CO intoxication by inhalation of **methylene chloride** vapors, a volatile liquid found in degreasers, solvents, and paint removers.

Toxicology

1 Toxicity primarily results from **cellular hypoxia**.

- Lactic acidosis develops.

2. Binding to hemoglobin:
    - CO reversibly binds hemoglobin, resulting in relative anemia.
    - Because it binds hemoglobin **210** times more avidly than oxygen, even small concentrations can result in significant levels of carboxyhemoglobin.
3. Binding to myoglobin:
    - CO also binds to muscle myoglobin
4. The tissues that are especially vulnerable to the effects of CO include:

- CNS and peripheral nervous system.
- Myocardium
- The fetus is also at particular risk

The exact cause of delayed neurological sequelae are uncertain, however these are thought to be the result of endovascular oxidative injury and inflammation. Carbon monoxide initiates endothelial oxidative injury, lipid peroxidation and an inflammatory cascade.

Pharmacokinetics

Absorption:

- Carbon monoxide is a colorless, odorless, tasteless and generally non-irritating gas produced by incomplete combustion of organic compounds.

  It is readily absorbed from the lungs.

Distribution:

- CO binds avidly to hemoglobin and myoglobin

Metabolism and elimination:

- CO is eliminated unchanged via the lungs.
- The elimination half-life is directly related to the dissolved oxygen tension in the blood. The half-life of CO under various conditions are as follows:
  - ♥ **In room air (i.e 21% oxygen) it is 4 hours.**
  - ♥ **One hundred percent oxygen reduces the half-life to 90 minutes.**
  - ♥ **100% oxygen in hyperbaric pressures of 3.0 atmospheres will reduce it to 23 minutes.**

Risk Assessment

High risk features in **acute** exposures include:

1. History:
   - There is a history of **confusion** or **loss of consciousness** at the scene.
   - There is a history of exposure within a confined space.
2. Co-morbidity:

- The patient has significant CVS or respiratory co-morbidity.
- Age over 55 years.

3. Clinical features on presentation:

   - **Persistent** neurological dysfunction, in particular:
     - ♥ Altered conscious state/ confusion.
     - ♥ Cerebellar dysfunction.
   - Myocardial ischemia.

4. Pregnancy:

   - Fetal haemoglobin binds CO more avidly, thus rendering the fetus more susceptible to injury.

5. Biochemical results:

   - Lactic acidosis
   - Carbon monoxide levels, have only a rough correlation to outcome, (**see below**)

Risk of delayed neurological sequelae:

Acute deliberate self-poisoning by car exhaust fumes usually involves exposure to high concentrations of CO for a limited duration. This has a relatively *lower* risk of long-term sequelae.

Accidental occupational or domestic exposure often involves exposure to lower concentrations of CO for a **prolonged duration**. This has a relatively higher risk of long-term sequelae.

Clinical Features

The signs and symptoms of CO poisoning are essentially those of hypoxia.

Important points of history:

1. Symptoms at the scene:

   - The history of what the patient was like *at the scene* is important, especially a history of **confusion or loss of consciousness.** This is because CO levels done *late* do not correlate well with symptoms.

2. Nature of the exposure:

- A high index of suspicion must be maintained for any patient exposed to fires, (especially within a confined area).

  Did the event occur within a **confined space?**

- Misdiagnosis commonly occurs because of the vagueness of complaints. *Specifically inquiring* about possible exposures when considering the diagnosis is therefore important, especially in cases of **chronic** exposure.

3. Co-morbidities:

   - The severity of signs and symptoms will also depend on a patient's pre-existing medical problems especially with respect to **respiratory and cardiovascular disease.**

Important points of examination:

1. Immediate attention to any ABC issues.

2. Vital signs:

   - Note that **pulse oximiter** readings may be **normal.**

   - Tachycardia

   - Hypertension (or hypotension in severe cases)

   - Tachypnea

3. Check the patient's level of alertness and orientation:

   - More subtle signs of toxicity may be detected on the **mini mental status** examination.

4. Respiratory distress:

   - Check for signs of non-cardiogenic pulmonary edema or wheeze.

5. Neurological:

   - In particular the cerebellar examination.

Clinical toxic effects include:

1. Acute neurological:

   CNS:

- Confusion/ impaired mental state.
- Altered conscious state
- Coma/ seizures
- Hypoxic brain injury
- Death

Peripheral:

- Cerebellar dysfunction in particular.

Delayed neurological sequelae:

- Higher concentrations for short periods pose less risk than lower concentrations for prolonged durations.

  Delayed sequelae appear after 2 - 40 days - especially the form of impaired judgment and memory. [5]

2. Respiratory:

   - Bronchospasm
   - An acute non-cardiogenic pulmonary edema

3. Cardiovascular:

   - ACS may be precipitated, especially in the over 55 years age group.
   - Hypotension and arrhythmias in severe cases.

4. Metabolic:

   - Lactic acidosis.
   - Rhabdomyolysis.
   - Hyperglycemia.

5. Hematological:

   - Disseminated intravascular coagulation

6. Pregnancy:

   - CO is toxic to the fetus.

Investigations

Blood tests:

1. FBE

2. U&Es/ **glucose**

   - **Urgent K** if rhabdomyolysis is suspected

3. **Beta HCG**:

   - This should be considered in any woman of child bearing age.

4. Troponin level:

   - If myocardial ischemia is suspected.

5. Myoglobin level if rhabdomyolysis is suspected

6. Consider coingestion:

   - **Blood alcohol**

   - **Paracetamol**

7. ABGs/ lactate level:

   - PaO_2 levels may be **normal.**

   - O_2 saturation levels may be normal (depends on how this is measured).

   - The essential investigation is the "co-oximetry" reading, where a direct CoHb level may be obtained.

7. COHb levels:

   - This can be done as **co-oximetry** on the arterial blood gas sample or - more conveniently - on a **venous gas** sample. [5]

   - Levels correlate best if taken **at the scene**, rather than at some later stage.

   - Elevated levels are significant; however, low levels do not entirely rule out significant exposure, especially if the patient already has received 100% oxygen or if significant time has elapsed since exposure.

   - Therefore, if COHb level is done late the patient may still have severe poisoning despite an apparently "non-toxic" level.

Correlation of COHb levels and clinical features:

COHb level	**Clinical features**
< 10%	Background levels in smokers.
10%	Asymptomatic or mild headache.
20%	Dizziness, headache, nausea/ vomiting dyspnea, mini mental state abnormalities/ impaired judgment.
30%	Ataxia, visual disturbances.
40%	Confusion, coma, seizures, syncope.
50%	Cardiovascular complications, respiratory failure, death.

CXR:

If there are significant respiratory symptoms.

ECG:

In particular for arrhythmias or ischemic changes.

Look for hyperkalemia changes if rhabdomyolysis is suspected.

CT scan brain:

Should be considered in all cases of altered conscious states or confusion.

It will be most useful in ruling out alternative causes for these symptoms.

MRI:

MRI is far better able to assess brain injury due to CO poisoning than is CT scan.

It is not necessary acutely but may be done later to assess prognosis, especially in those that show persisting symptoms.

It may show cerebral edema, basal ganglia injury or cortical demyelination in severe cases.

See Appendix 1 below

Management

1. Immediate attention to any ABC issues.

2. 100% oxygen therapy:

   Patients with minor exposures with symptoms should be treated with 100% normobaric oxygen. This should continue until all symptoms have resolved. The exact period of treatment required is not well defined.

   Current expert opinion suggests a period of *at least* **6 hours**. [5]

   - 100 % oxygen can be delivered initially via a Hudson mask fitted with a *reservoir*.

     A non-rebreathing system is best and this may be achieved with a Laerdal bag with reservoir, strapped to the patient's face or via a CPAP circuit.

   The threshold for treatment for *apparently* minor exposures, should be lower if:

   - **There is a history of confusion or loss of consciousness at the scene.**
   - **The patient has significant CVS or respiratory co-morbidity.**
   - **There is a history of exposure within a confined space.**

3. For more significant exposures or for patients who have not stabilized after 6 hours of treatment, or patients who are **pregnant** 100 % O_2 must be given.

   - For a *minimum* of **24 hours.**[1, 3]

   100% oxygen therapy should then be continued until the patient is: [5]

   - **Asymptomatic**
   - **Has no signs of CO poisoning.**
   - **< 5 % CO on co-oximetry**

4. Confused combative patients may require intubation for treatment.

5. **Hyperbaric oxygen**:

   The role of hyperbaric oxygen remains controversial

- Whilst it is not disputed that hyperbaric oxygen causes faster CO "off-gassing" than normobaric oxygen, what remains unknown is whether this results in a reduced incidence of persistent and delayed neurological sequelae.

- Current opinion in the state of Victoria is that hyperbaric oxygen treatment remains of unproven benefit, (in adults and non pregnant women)

- The indication in **children and pregnant women** is less clear and these cases should be discussed with a **Clinical Toxicologist.**

Disposition:

All symptomatic patients should be discussed with a Clinical Toxicologist.

Patients without symptoms or minimal symptoms may be suitable for a Short Stay Unit Period of Observation.

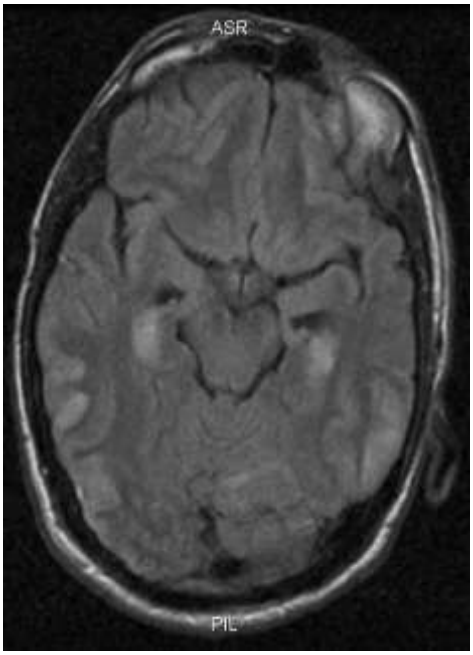
Patients with more significant symptoms will require monitoring in a HDU or ICU

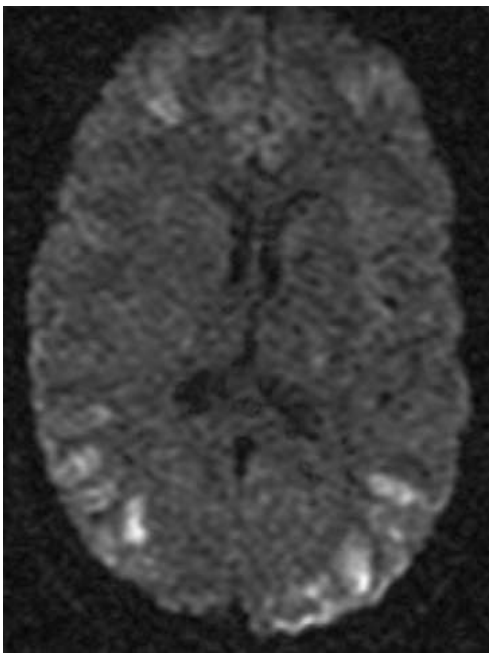
Organize appropriate psychiatric consultation in all cases of intentional self-harm.

Patients with more significant exposures will need neurological and neuropsychological follow-up assessments at 1 - 2 months after discharge from hospital for possible delayed sequelae.

It is important to note that patients who present following accidental occupational or domestic CO exposure represent index cases. There should be a thorough investigation of the source as well as an investigation into other potential victims.

Appendix 1





Left: Cerebral scan in a 39 year old male found unconscious in his car, (with hose attached from exhaust pipe to window), suffering from severe carbon monoxide poisoning. The CT without contrast shows no apparent abnormalities.

The MRI Flair (middle) and MRI DWI (right) scans show lesions within the basal ganglia and cortical regions, consistent with ischaemic/toxic changes characteristic of severe CO poisoning.

Appendix 2

Chemistry of Carbon

Carbon as diamond, charcoal and graphite and (Time-Life, "The Elements")

Elemental symbol	**C**
Discovery	Antoine Lavoisier
Atomic number	6
Atomic weight	12.011
Melting point	None - it sublimes directly into a gas.
Boling point	40827 ^{o} C
Classification	Non-metal
Physical Appearance	Black as graphite, transparent as diamond

References:

1. Emerson G, The Dilemma of Managing Carbon Monoxide Poisoning, Emergency Medicine Australasia 2004 16, 101-102.

2. Scheinkestel CD et al, "Where to now with carbon monoxide poisoning?" Emergency Medicine Australasia 2004 16, 151-154.

3. Carbon monoxide in L. Murray et al. Toxicology Handbook 3rd ed 2015.

4. Scheinkestel CD, Bailey M et al. Hyperbaric or normobaric oxygen for acute carbon monoxide poisoning: a randomised controlled clinical trial. Med J Aust 1999 Mar 1; 170(5): 203-10

5. Carbon Monoxide Poisoning in Austin Health Toxicology Clinical Practice Guidelines, February 2018.

Dr J. Hayes

Reviewed 2 February 2019.