# Management of carbon monoxide poisoning using oxygen therapy

TWL Mak, CW Kam, JPS Lai, CMC Tang

**The management of carbon monoxide poisoning requires an accurate assessment of the extent of blood oxygenation. Measuring the fractional oxyhaemoglobin content by using co-oximetry gives a true picture of the oxygen-carrying capacity of blood in the presence of carboxyhaemoglobin. The use of readings from pulse oximetry or a standard blood gas analyser is insufficient and can be misleading. We report on a case of carbon monoxide poisoning to illustrate this potential pitfall.**

## Introduction

Carbon monoxide poisoning reduces the oxygen-carrying capacity of blood. The accurate measurement of fractional oxyhaemoglobin ($FO_2Hb$) by using co-oximetry is essential in the calculation of the oxygen-carrying capacity. Oxygen saturation is measured by using pulse oximetry or calculated by using the arterial oxygen partial pressure ($PO_2$), as measured with a standard blood gas analyser. Oxygen saturation, however, does not take into account the presence of carboxyhaemoglobin. We report on a case of carbon monoxide poisoning to illustrate the accurate approach to measuring the oxygen content of blood.

## Case report

A 30-year-old woman presented to the Accident and Emergency Department of the Tuen Mun Hospital in January 1999 after having attempted suicide by burning coal inside her home while all the windows were closed. The relative who had found the woman reported that she was drowsy, her limbs were rigid, and her eye gaze was abnormal. The ambulance staff reported that the woman was confused and had twitching limbs during transit to hospital. Oxygen supplementation (between 60% and 90%) was given through a non-rebreathing facial mask with a reservoir.

On arrival at the Accident and Emergency Department, the patient had regained consciousness and scored 15/15 on the Glasgow coma scale. There was no detectable neurological deficit. The blood pressure was 125/107 mm Hg and the pulse rate was 123 beats per minute. The skin colour was normal. The oxygen saturation as measured by using pulse oximetry ($SpO_2$) ranged between 95% and 99%; oxygen therapy was continued. Electrocardiography showed sinus tachycardia (pulse rate, 118 beats per minute) without ischaemic features or other arrhythmia. The chest X-ray did not reveal any abnormality.

An arterial blood gas profile was determined while the patient was given 60% to 90% of oxygen. The blood pH, $PO_2$, and arterial carbon dioxide partial pressure ($PCO_2$) were 7.36 (reference range, 7.35-7.45), 20.4 kPa (reference range, 10.0-13.3 kPa), and 4.4 kPa (reference range, 4.7-6.0 kPa), respectively. Oxygen saturation as measured from the blood gas profile ($SaO_2$) was 99%. The $FO_2Hb$ was measured directly by co-oximetry 15 minutes later and was found to be 64.8%; the carboxyhaemoglobin (COHb) content was 34.6%. The patient's vital signs remained stable during oxygen therapy of 60% to 90% through a non-rebreathing facial mask with a reservoir, at an oxygen flow rate of 12 L/min. She was transferred to a psychiatric hospital for further treatment 2 days later.

**Tuen Mun Hospital, Tsing Chung Koon Road, Tuen Mun, Hong Kong:**
**Department of Clinical Pathology**
TWL Mak, MRCPath, FHKAM (Pathology)
JPS Lai, MSc
CMC Tang, MRCPath, FHKAM (Pathology)
**Accident and Emergency Department**
CW Kam, MRCP, FHKAM (Emergency Medicine)

Correspondence to: Dr T W L Mak

## Discussion

Oxygen saturation is defined as the fraction of oxyhaemoglobin (oxyHb) in relation to the amount of haemoglobin that is capable of carrying oxygen.[1] The oxygen-carrying haemoglobins are oxyHb and deoxyhaemoglobin (deoxyHb). Oxygen saturation, defined as the concentration of oxyHb divided by the sum of the concentrations of oxyHb and deoxyHb, can be measured non-invasively by using pulse oximetry or calculated from the $PO_2$, as measured by using a standard blood gas analyser. In normal situations, the oxygen saturation that is obtained by either method is a good indicator of the extent of blood oxygenation. In the presence of other haemoglobin derivatives such as COHb, methaemoglobin, or sulphaemoglobin, however, the oxygen saturation does not reflect the underlying pathological changes.

Pulse oximetry measures the arterial oxygen saturation ($SpO_2$) by recording the amount of light that is transmitted through a body part, typically in the terminal phalanx of a finger. It uses dual-wavelength absorbance measurements, one in the visible and one in the near infrared range, to estimate the concentrations of oxyHb and deoxyHb.[2] The basis of $SpO_2$ measurement relies on the difference in the optical absorbance of oxyHb and deoxyHb. Because COHb has optical absorbance characteristics similar to oxyHb, the pulse oximeter cannot distinguish between the two. Hence, in a case of carbon monoxide poisoning, the $SpO_2$ becomes a misleading indicator of oxygenation. It is important to recognise this shortcoming of pulse oximetry to avoid mismanaging suspected cases of carbon monoxide poisoning.

In an ordinary blood gas analyser, the $SaO_2$ is derived from the $PO_2$ value.[3] Under normal situations, there exists a relationship between $SaO_2$ and $PO_2$, which is depicted in the standard oxygen dissociation curve. The $PO_2$ is a measurement of the oxygen tension in blood, which is a measure of the amount of oxygen dissolved in the plasma. The majority of oxygen in the blood is carried by haemoglobin, which forms a readily reversible complex with oxygen and enhances substantially the oxygen-carrying capacity of blood. In the presence of COHb, the $PO_2$ is not affected because the capacity of blood to dissolve oxygen physically is unchanged. Hence, the $SaO_2$ derived from the $PO_2$ is also an unreliable indicator of oxygenation when COHb is present. This is a second pitfall when dealing with carbon monoxide poisoning cases. Co-oximetry overcomes this problem by simultaneously measuring the absorbance of blood in multiple wavelengths to accurately quantify the proportions of oxyHb, deoxyHb, COHb, and other dyshaemoglobins (eg methaemoglobin and sulphaemoglobin).[4] The $FO_2Hb$, defined as the percentage of oxyHb with respect to the total concentration haemoglobin, is then derived from the measured values.

Because the amount of oxyHb and all haemoglobin moieties are measured directly, the $FO_2Hb$ gives a true picture of the oxygen-carrying capacity of blood in the presence of COHb. This is the only reliable way to assess the oxygenation status in cases of carbon monoxide poisoning in a clinical laboratory. The numerical values of $SpO_2$, $SaO_2$, and $FO_2Hb$ are usually very similar because most patients do not have a significant amount of dyshaemoglobins. This fact overshadows the potential pitfalls of using $SpO_2$ and $SaO_2$ to assess the extent of blood oxygenation. The affinity of carbon monoxide for haemoglobin is approximately 250 times that of oxygen.[5] The oxygen content of the blood is thus severely lowered in the presence of a significant level of COHb (Fig). The oxygen content (in mmol/L) is defined as[6]:

$$(tHb \times FO_2Hb \times 1.39) + (PO_2 \times \alpha)$$

where 'tHb' is the total haemoglobin concentration and '$\alpha$' is the solubility coefficient of oxygen in the plasma.
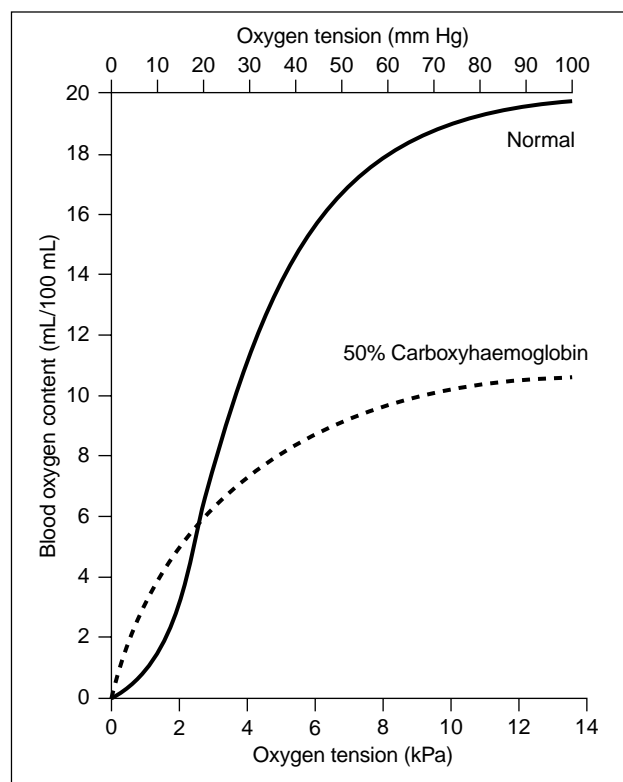


**Fig. Relationship between arterial oxygen partial pressure and blood oxygen content** [5]
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To maintain the oxygen supply, the cardiac output would have had to be increased by a factor of 1.5 in this case. Severe hypoxia would otherwise have resulted if the cardiac function of the patient were compromised. Depending on the level of carbon monoxide in the blood, pure oxygen or hyperbaric therapy is required to shorten the time required to reconvert COHb to oxyHb. The half-life of COHb is usually 270 minutes. This time is reduced to approximately 25 minutes when a patient is breathing 100% oxygen at a pressure of 3 atm (304 kPa).[7] Furthermore, hyperbaric oxygen therapy may convey benefits other than rapidly reducing the COHb concentration, such as reducing the free radical concentration.[8] The indications for hyperbaric oxygen therapy that are used at the Poisons Unit at Guy's Hospital, London, United Kingdom, are as follows: a loss of consciousness, neurological or cognitive abnormality, electrocardiographic evidence of cardiac ischaemia, a COHb level of more than 20%, and pregnancy.[7] Hence, it is important to accurately assess the oxygenation status to determine the appropriate approach to treating suspected cases of carbon monoxide poisoning. In Hong Kong, hyperbaric oxygen therapy is available at the Government Dockyard on Stonecutters Island. When indicated, one can contact the Fire Services Control Centre by using the emergency telephone number (999) or by calling 2723 2233 to mobilise the resources to transfer the patient and to start treatment.

In conclusion, when managing cases of carbon monoxide poisoning, there is a pitfall in using pulse oximetry or the estimated $SaO_2$ value from the $PO_2$, as determined by using a standard blood gas analyser. Co-oximetry is mandatory for measuring the $FO_2Hb$, whether or not COHb is present. This service should be available on a 24-hour basis at all hospitals dealing with these cases.

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T Lane
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*Hong Kong Medical Journal*