

CASE REPORT

The dangers of argon, an inert industrial gas: beware of asphyxiation

R.V. Peelen, B.P. Ramakers, A. Koopmans

Department of Intensive Care, Bernhoven Hospital, Uden, the Netherlands

Correspondence

R.V. Peelen - roel.peelen@radboudumc.nl

Keywords - inert gases, industrial accidents, argon, asphyxiation, resuscitation, hypoxia, hypoxemia.

Abstract

We describe the case of an industrial welder who died due to hypoxaemic hypoxia as a result of argon inhalation. Extensive resuscitation attempts could not prevent fatal organ damage, probably due to prolonged hypoxaemic hypoxia. Work-related accidents with industrial gases are rare. Nonetheless we feel that it is important to create awareness for this potentially life-threatening danger. Especially the combination of characteristics of the gases (colourless, tasteless and odourless), and the absence of alarming symptoms make it an easy to miss cause of a cardiac arrest. We describe the case in detail and discuss the danger of inert gases, especially argon, and their properties.

Introduction

In the Netherlands, work-related accidents result in approximately 50 deaths every year, mostly caused by trauma.^[1,2] A less common cause is the inappropriate use of industrial gases (e.g. argon, helium, carbon dioxide and nitrogen). These gases have numerous applications, from shielding gas, and fire extinguishing gas to insulation and chemical protection gas.^[3] In recent years, several of these gases have also been used in suicide attempts.^[4,5] So far only four case reports describe human asphyxiation due to argon inhalation.^[6,7] In 1987, unintentional refilling of a hospital oxygen supply with argon was the cause of death in three patients, who developed hypoxaemic cyanosis during routine surgical procedures.^[8] Second, in a nuclear power station reactor two people suffocated due to argon inhalation in 1992.^[9] Third, a welder's helper asphyxiated in 1994 while working with a argon welding installation in pipe during construction of a crude oil pipeline.^[11] And fourth, an engineer died in a small reaction vessel of a bulb factory in 2003 due to exposure to argon.^[12]

In this case report we describe a 48-year-old industrial welder who died of asphyxiation due to argon-induced hypoxaemia while welding.

Case report

A 48-year-old industrial welder, with mild obesity (32.8 kg/m²) but no other medical history, presented to the emergency department after a cardiac arrest. The patient was found unconscious, sitting on the floor just outside a small room near a working station where he had been welding just before. There were no signs of epilepsy (no tongue bite, no urinary incontinence, no signs of vomiting or airway obstruction) let alone a subsequent asphyxia as a cause. The chest X-ray did not show any significant abnormalities, especially no signs of aspiration. We cannot exclude cardiac arrhythmia as the primary reason for the out-of-hospital cardiac arrest; however, the patient had no cardiac history, and the position in which he was found did not suggest collapse without prodromal signs. The patient was last seen at least 10 minutes before he was found and the ambulance was called. In retrospect, co-workers found him acting strangely at that moment. After an initial 10 minutes of basic life support by colleagues, advanced life support was initiated by paramedics. The initial evaluation showed asystole with return of spontaneous circulation (ROSC) after 20 minutes, followed a few minutes later by a period of pulseless electrical activity. Upon arrival in the emergency room the patient (E1 V tube M 1) was resuscitated for another 30 minutes; with periods of pulseless electrical activity, asystole, and a single block of ventricular fibrillation. The total time of resuscitation was approximately 50 minutes. After ROSC, the patient had severe bradycardia at 30 beats/min. Since the aetiology of this cardiac arrest was unknown, we decided to insert an external pacemaker. During CPR, we evaluated all possible causes using the '4Hs and 4Ts'.^[12] Laboratory results ruled out glycaemic and electrolyte disorders using point of care measurements. We found a pH of <6.75 and a lactate of 24 mmol/l. Together with an initial PaCO₂ of 13.8 kPa we concluded there was a combined respiratory and metabolic acidosis during resuscitation. The patient had a normal temperature and was treated for possible hypovolaemia. There were no signs of trauma. The ECG

showed no signs of ischaemia. An echocardiography performed by the cardiologist during a period of bradycardia and after treatment with adrenaline showed normal contractility of the heart without signs of cardiac tamponade or right ventricular failure. There were no signs of hypoxaemia as seen in *table 1*. A cerebral CT scan ruled out intracranial haemorrhage as seen in *figure 1*. However, this scan showed severe diffuse cerebral oedema and did not rule out cerebral herniation. Next, the patient was admitted to the ICU where he was treated with increasingly high doses of norepinephrine and dobutamine (0.8 gamma and 8 gamma respectively) to maintain tissue perfusion. As the patient's condition further deteriorated we decided to withdraw treatment.

The local authorities informed us later that there was a problem with the welding equipment at the place of the incident. For no known reason, the argon flow was set a factor 10 higher than usual while welding in a small and insufficiently ventilated space.

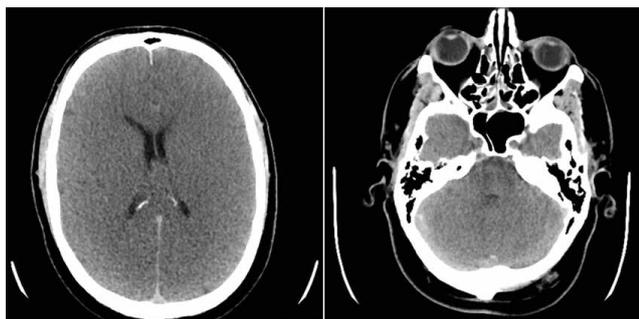


Figure 1. CT scan

A Cross-section at the level of the eyes. No visible sulci. There is no visible differentiation between the white and grey matter

B Cross-section at the level of the lateral ventricles. The ventricles are very small, no focal lesions or visible demarcation of white and grey matter is seen, and there is a complete loss of gyri and sulci pattern. These findings suggest diffuse signs of acute deterioration due to global brain ischaemia or swelling

Table 1. Initial laboratory findings in the emergency room

Unit (normal values)	Initial ER laboratory results	After resuscitation in ER
pH (7.35 - 7.45)	<6.75	6.95
PaCO ₂ , kPa (4.7 - 6.4)	13.8	9.7
PaO ₂ , kPa (10.0 - 13.3)	30.3	24.6
Bicarbonate, mmol/l (22 - 29)	-	16
Base excess, mEq/l (-3.0 - 3.0)	-	-15.3
Sodium, mmol/l (135 - 145)	143	143
Potassium, mmol/l (3.5 - 5)	4.8	5.3
Lactate, mmol/l (<2.2)	24	18
Glucose, mmol/l (3.5 - 7.8)	19.8	15.7
Haemoglobin, mmol/l (8.5-11.0)	8.3	7.2

Post-mortem analysis

The autopsy suggested hypoxaemic hypoxia as the cause of death. Overall there were signs of severe hypoxia in the heart (lactate dehydrogenase reaction, without signs of coronary artery disease), which occurred just a few hours before death, severe pulmonary oedema (probably due to acute heart failure), and severe swelling of the brain (acute hypoxic encephalopathy). By exclusion the pathologist concluded hypoxaemic hypoxia as the cause of death. Although argon possibly could have been detected in the lung, no additional research was performed by the forensic pathologist.^[11]

Discussion

Argon is an inert gas, colourless, tasteless and odourless. In the earth atmosphere, it accounts for 0.934% of the total volume, which makes it the most common inert gas.^[13] Argon is used for numerous industrial applications, such as shielding gas in welding but also as fire extinguishing gas. Argon gas can displace oxygen and other gas molecules due to its higher volumetric mass density and its relative inertia.^[3] Despite its inert properties, argon shows biological reactions under special circumstances. Argon is known for its anaesthetic properties under hyperbaric conditions (>4 ATM).^[14] The exact mechanism of action is unclear and it is assumed that under hyperbaric conditions argon acts as a GABA_A agonist.^[15,16] Under normobaric conditions the medical use of argon is currently the subject of research for its possible neuroprotective properties.^[17]

Bulk-used industrial gas, such argon, could easily lead to situations of hypoxaemic hypoxia.^[11,18] These inert gases do not take an active role in our alveolar gas exchange, but displace the regular air. Due to the abundance of available gas, in this case argon, the partial air pressure rises at the expense of other partial pressures, most importantly the PaO₂. The process of displacement is increased when the density of the gas is higher than regular air, as this decreases the degree of evaporation. Gases with these characteristics are used in numerous industrial applications. For example, nitrogen oxide is often used in industrial refrigerators to obtain inert atmospheres or as propellant, whereas carbon dioxide is used for extinguishing fires, for anaesthesia in slaughterhouses, and as dry ice in commercial, industrial and scientific applications. For commonly used industrial gases and their properties and applications see *table 2*.^[3,19,20]

Table 2. Commonly used industrial gases and their properties and applications

Gas	Density (normal air: 1.293)	Additional dangers	Example of Applications
Nitrogen (N ₂)	1.169	Anaesthetic properties in hyperbaric situations	Coolant Packaging Fire extinguishing Diving Fertiliser
Nitrous oxide	1.53	Anaesthetic	Anaesthetic Oxidiser Propellant
Oxygen	1.429	Corrosive Explosive	Welding Diving Medicine
Helium	0.179		Coolant Diving Shielding gas Aviation
Argon	1.782	Anaesthetic properties in hyperbaric situations	Shielding gas Fire extinguishing Thermal insulation Laboratory gas
Xenon	5.88	Anaesthetic properties	Gas-discharge lamps Anaesthetic
Hydrogen	0.089	Highly explosive	Fuel Coolant
Carbon dioxide	1.98		Dry-ice Fire extinguishing Side product
Carbon monoxide	1.14	Toxic; binding to haemoglobin	Feedstock Side product
Acetylene	1.1	Highly explosive	Feedstock Welding fuel

Symptoms

In the presence of an inert gas in high concentration, oxygen and carbon dioxide (CO₂) will be displaced, creating alveolar hypoxia. Since the pH and P_a[CO₂] are our main respiratory stimuli, the human respiratory system will give minimal if any alarm signals in conditions of low PaO₂.^[21] Even for trained professionals such as pilots, hypoxia is difficult to recognise. Nonetheless, patients do show warning symptoms such as passiveness, fatigue, confusion and finally fainting and bystanders could be alarmed by altered behaviour.^[7,11,18] Even when the oxygen content of the air decreases to just below 21 vol.% tiredness is experienced accompanied by an increased heart rate. Until 11 vol.% movement will be limited and intellectual performance will decrease rapidly.^[11] Lower oxygen content will lead to headaches, dizziness and fainting after some time. In environments where the oxygen concentration has fallen below 6 vol.%, the person would almost immediately faint, after which irreversible damage to vital organs, most significantly the brain, occurs.^[11,18,22] Much research has been performed on the time of useful consciousness in the aviation industry. At an oxygen content of 12 vol.%, it would take 3-5 minutes before a person is no longer able to implement proper corrective or protective measures, due to rapid decrease of the intellectual

level.^[23] The combination of rapid increasing damage and growing inability to react is potentially lethal.

Treatment

Treatment of hypoxia due to inhalation of an inert gas consists of discontinuing the causative agent and support with adequate oxygenation.^[18]

In contrast to carbon monoxide intoxication, where high levels of oxygen are necessary to counteract the haemoglobin binding, inert gases such as argon only reduce the alveolar oxygen fraction, causing hypoxaemic hypoxia. Nonetheless, in both cases a non-rebreathing mask could be adequate, if the patient has spontaneous adequate ventilation; otherwise non-invasive or when unconscious invasive ventilation may be necessary.^[18] In this case the primary hypoxaemia led to severe tissue-hypoxia resulting in cardiac arrest. The combination of hypoxia, the delay in adequate oxygen administration and the extended duration of CPR caused irreversible damage to the organs, in particular the brain.

Post mortem analysis

Proving argon-related asphyxiation may be difficult, due to the inert properties of argon and its natural presence in the atmosphere and the human body. Argon wash out from the tissue can be augmented by the length of oxygen administration and resuscitation. Nonetheless Auwärter described a technique which enables us to prove excessive argon inhalation. Gas chromatography can detect elevated tissue concentrations of argon in the respiratory and circulatory tract, stomach, liver and lung.^[11]

Conclusion

Work-related accidents or suicide attempts with industrial gases are rare. Nonetheless, the combination of the characteristics of inert gases (colourless, tasteless and odourless), and the absence of alarming symptoms, is potentially lethal. When using the 4 Hs and 4 Ts any physician working in the emergency room or ICU should think of hypoxia as a cause, also when the arterial blood gas analysis shows normal or high PaO₂ (in the presence of oxygen therapy). If asphyxia due to inert gases is suspected, high flow oxygen therapy is required. In case of an unconscious patient, the airway should be secured. Preventive measures are key to prevent subsequent incidents. Reliable and portable oxygen meters are available and would possibly have prevented this tragic accident.

Disclosures

All authors declare no conflict of interests. No funding or financial support was received.

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