Open access Journal of
Neurology & Neurosurgery
ISSN: 2476-0501

Juniper Publishers
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Commentary
Volume 4 Issue 4 - June 2017
DOI: 10.19080/OAJNN.2017.04.555642

Open Access J Neurol Neurosurg


# A Commentary on the Neurological Findings Associated with Overexposure to Liquefied Petroleum Gas

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**Submission:** June 14, 2017; **Published:** June 30, 2017

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## Introduction

In countries in the BRICS (Brazil, Russia, India, China and South Africa) Association, the use of alternatives to electricity as means for heating and cooking in the home has increased. The most commonly used alternative is liquefied petroleum gas (LPG) [1].

With this has come the development of a cohort of patients, presenting with non-specific symptoms associated with overexposure to LPG. This is commonly referred to as 'Gas Geyser Syndrome', but this has also beenused to describe cases of carbon monoxide (CO) exposure [2]. What further confounds the diagnosis, is the fact that the spectrum of symptoms closely mimics that of the exposure to other gases. New literature now shows us that findings on neurological imaging, can better elucidate this diagnostic conundrum.

### Understanding LPG

LPG is generally composed of the high-density gases propane (1.8988 kg/m3 at 15 °C) and butane (2.5436 kg/m3 at 15 °C), with a propensity to sink to lower levels in the environment which they occupy [3]. An odorant, usually Ethyl Mercaptan, is used to give LPG its distinctive smell. Naturally propane and butane are both odourless, thus a collection of the gases could go undetected if not odourised. In a small enclosed environment, LPG is able consume atmospheric oxygen to form deadly CO, and especially so if ignited, such as with gas geysers and cooking appliances. This, paired with the ability to have an anaesthetic effect, as well as being an asphyxantcan prove to be fatal. LPG is mainly used in small enclosures, such as bathrooms for gas geysers.Bearing this in mind, it is understandable that LPG has great potential to harm the uninformed user [4].

## Diagnosis

### History

It is paramount to the diagnosis and further management of the patient to determine the following:

Was a LPG canister seen?

Was the enclosure small?

Was there good ventilation?

The approximate duration of exposure

Have they received any oxygen pre-hospital?

The clinician should also enquire whether the patient was positioned at ground level or not. Non-specific symptoms that conscious patients may complain of include: myalgia, malaise, headaches and dizziness, as well as nausea and vomiting [4].

### Clinical findings of importance to neurological presentation

The above determines the clinical findings, in that they affect the level of consciousness of the patient, the duration of exposure to the agent and the likelihood that the patient was exposed to CO. Thus, it must not be forgotten that these patients are likely to be hypoxic.

On inspection – the level of consciousness may range from mild confusion to being comatose. Cherry red lips and/or oral mucosa may be noted if CO was present. This is due to the conformational change that haemoglobin undergoes when bound to CO. It also interacts more favourably with CO than oxygen. Pulse oximetry will thus, indicate a hypoxic state, which may appear to be a misnomer in the presence of the reddened mucosa. If a urinary catheter is inserted, tea coloured urine may be noted if rhabdomyolysis has developed. This is a late complication and is usually found in prolonged exposure. Complications of rhabdomyolysis should also be prevented. Palpation-there will likely be a tachycardia and an associated tachypnoea. This is only likely to be present once the anaesthetic effect has worn off or there was insufficient exposure to the gas to induce this. Patients with reduced levels of consciousness also tend to present with dilated pupils, intact corneal reflexes and normal to reduced limb reflexes globally.

### Special investigations specific to neurological diagnosis

An arterial blood gas is important as a respiratory acidosis will develop early on due to the asphyxant and anaesthetic properties. However, metabolic acidosis is usually in the late phase of overexposure. To definitively exclude CO inhalation, a carboxyhaemoglobin level should be done. However, it should be noted that this may be slightly elevated in smokers and those living in heavily air polluted areas [4].

### Development of diagnostic techniques

Commonly forgotten in the armamentarium of clinicians, is the correlation of neurological pathology, with clinical findings and evidence. All of the systemic manifestations of overexposure to LPG, have either a direct, or indirect effect on the neurology of the patient. The most obvious of these being the hypoxic state as well electrolyte and acid-base imbalances. Thus, it makes sense that resolution of these factors will result in resolution of neurological pathology. However, this cannot be thought of as a directly proportional correlation. There is more literature which speaks of CO overexposure than there is of LPG itself, and in many instances there is a mixed exposure. However, the presentation of overexposure to both gaseous mixtures is very similar. This is even shown in imaging studies.

Computed tomography (CT) and mangnetic resonance imaging (MRI), have both been used successfully to identify areas of injury and the development of lesions in the brain [5,6]. The most common sites where lesions are found are the globuspalladus (50%), putamen (30%) and the caudate nucleus (20%). However, this data is based on only a handful of reports and studies [5-7].

## Comments

Although there is little research and data available, a good foundation for further research has been laid. With further investigation, and the refinement of imaging techniques and interpretation, criteria can be developed to predict the outcome and further management of these patients. It also opens new avenues for radiology and neurology in the emergency room and thus will likely reduce morbidity and mortality in the long run.

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DOI: 10.19080/OAJNN.2017.04.555642





**How to cite this article:** Luke Sampson. A Commentary on the Neurological Findings Associated with Overexposure to Liquefied Petroleum Gas. Open Access J Neurol Neurosurg. 2017; 4(4): 555642. DOI: 10.19080/OAJNN.2017.04.555642.