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Chronic Exposure to Cooking Gas – it's effect on Pulmonary Function Indices and Arterial Oxygen Content.

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SUMMARY

This study was conducted to assess the effect of chronic exposure to cooking gas on pulmonary function and arterial oxygen content. Eighty individuals: 30 (cooking gas vendors) and 50 (controls) entered the study. Lung function tests; forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and peak expiratory flow rate (PEFR) were performed. Peripheral oxygen saturation (S_pO_2) and haemoglobin levels were obtained from both the study and the control groups. Approximate values of the arterial oxygen partial pressure were calculated from the measured S_pO_2 . Approximate values of arterial oxygen content (CaO_2) were thus calculated. The duration of exposure (x) to cooking gas was also calculated. The values of the CaO_2 and lung function indices were related to the duration of cooking gas. The anthropometric parameters of both groups were similar with p-value being greater than 0.05. However, the difference between the ventilatory function parameters of the test and control groups were statistically significant $p < 0.05$, 0.05, 0.001 for FVC, FEV_1 and PEFR respectively. The relationship was significant for duration of exposure in relation to PEFR, FVC and FEV_1 ($p < 0.001$). The mean arterial oxygen content (CaO_2) of the vendors were found to be generally low $14ml\ dl^{-1}$ (normal = $20ml.\ dl^{-1}$) when compared to the control, $17.8ml.\ dl^{-1}$ ($p < 0.05$). There was also a gradual decline in the arterial oxygen content with increased duration of exposure to the cooking gas. Chronic inhalation of cooking gas should be avoided because it is injurious to health.

KEY WORDS: *Cooking gas; chronic inhalation, pulmonary function, arterial oxygen content.*

INTRODUCTION

Spillage of gases is common in all cooking gas shops in Nigeria. This spillage occurs as a result of leakage from faulty gas cylinder nipples and leakage during refilling of cylinders. Cooking gas contains butane and nitrogen dioxide. Both gases produce pungent and unagreeable odour. However, cooking gas vendors who chronically inhale these gases become adapted to the odour. Thus they now become completely unaware of the odour and any potential adverse effects.

Many researchers have established various deleterious effects of these gases such as; myocardial toxicity, ventricular fibrillation and respiratory function impairment^{1,2,3,4}.

This study was prompted by the persistent odour in the cooking gas shops and the fact that some cooking gas vendors sleep overnight in the unventilated shops. In addition, the recent report of two deaths among the cooking gas vendors who died in their sleep in two different shops at night underscored the need to conduct

this study.

This study, therefore, seeks to investigate the major physiological changes occurring in cooking gas vendors, who chronically inhale cooking gas.

Materials and Methods

This prospective study was carried out at cooking gas shops (study group) and the University of Calabar Teaching Hospital (Control group). The study group was made up of 30 cooking gas vendors while the control group was made of 50 medical students who were all within the same age range. The information obtained from each subject/control included the following; age, sex, weight, height, educational status and duration of exposure to cooking gas which was evaluated thus:

Exposure (x) = Years of contact (X_1) x Duration of contact per day (X_2)

($x = x_1 \times x_2$)

History of respiratory symptoms were also obtained. The following vital signs were recorded for each subject and control: pulse rate by digital plethysmography, blood pressure using sphygmomanometer, and peripheral oxygen saturation percent (SpO_2) using the pulse oximeter. Lung

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function tests performed included: forced vital capacity (FVC) forced expiratory volume in one second (FEV₁) using a vitalograph and peak expiratory flow rate (PEFR) using the peak flow meter. Blood samples were obtained for haemoglobin (Hb) level from both the subjects and controls. In the absence of a blood gas machine, PO₂ was calculated from the measured S_pO₂.

Oxygen content (CaO₂) was therefore calculated as follows⁵:

$$CaO_2 = Hb \times 1.34 \times S_pO_2 (\%) \times (0.003 \times PO_2)$$

The values of the CaO₂ and the lung function indices were related to the duration of exposure to cooking gas to see if there was any departure from normal physiological parameters.

Results

The age distribution of the gas vendors is shown in table 1. The bulk of the subjects were males 25 (83.3%) while only 5 (16.7%) were females. Majority of the subjects were relatively young (15 – 20years) in age. Only 10% were older than 40 years.

Table 2 shows the duration of exposure of the subjects to cooking gas. Majority of the subjects (43.2%) were exposed to it for a period of less than 24 months, while 30% were exposed for longer duration of above 48 months.

Table 3 shows the general characteristic of both the subjects and control population. A total of 30 subjects and 50 control population were used for the study. Table 3 also shows the comparison of anthropometric and

Table 1: Age and Sex Distribution of Gas Vendors and Controls.

Age	Total n	Subjects (n = 30)			Controls (n = 50)	
		Men	Women	Total	Men	Women
15 – 20	11 (36.6)	9	2	14	8	6
21 – 25	4 (13.5)	4	–	20	13	7
26 – 30	3 (10)	–	3	13	9	4
31 – 35	5 (16.6)	5	–	2	2	–
36 – 40	4 (13.5)	4	–	1	1	–
40+	3 (10)	3	–	–	–	–
Total	30	25	5	50	33	17

Table 2: Correlation between ventilatory function parameters and duration of exposure of the vendors to cooking gas.

Parameter	PEFR	FVC	FEV1 or	FEV%
Duration of exposure Males n = 25	–0.92 0.001	–0.89 0.001	–0.85 0.001	0.24>0.05
Duration of exposure Female n = 5	–0.95 0.001	–0.91 0.091	–0.83 .001	0.19>0.05

Table 3: General characteristics of subjects and controls.

Parameters	Subjects n = 30	Controls n = 50	p. Value
Mean Height (M)	1.62 ± 2.54	1.65 ± 2.80	>0.005 Ns
Mean Weight (kg)	56.0 ± 6.2	57.5 ± 6.5	>0.05 Ns
Mean Age	18.0 ± 3.5	19.2 ± 3.8	>0.05 Ns
Mean PEFR (L/mm)	346 ± 14.0	523 ± 16.11	<0.001
Mean FVC (L/mm)	28 ± 0.25	3.5 ± 0.28	<0.05
FEV ₁ (L/m)	2.38 ± 0.45	3.2 ± 0.56	<0.05
FEV ₁ %	86.1 ± 7.8	92 ± 8.1	<0.05
CaO ₂ (ml.dl ¹)	14.0	17.8	<0.05

Table 4: Duration of exposure of subjects to cooking gas.

Months	Total	Men	Women
0 – 24	13 (43.33)	10 (40)	3 (60)
25 – 48	8 (26.6)	6 (20)	2 (40)
49 – 72	6 (200)	6 (200)	–
73 – 96	2 (67)	2 (8)	–
96	1 (33)	1 (4)	–
	30	25	5

Table 5: Mean CaO₂ of subjects and duration of exposure.

Duration Months	Subjects	Mean CaO ₂ (ml.dl ⁻¹)
0 – 24	13	14.25
25 – 48	8	14.01
49 – 72	6	13.75
73 – 96	2	13.20
> 96	1	12.30

ventilatory function parameters between cooking gas vendors and their controls. The anthropometric parameters of both groups were similar with p-value being greater than 0.05. However, the differences between the ventilatory function parameters of the test and control groups were statistically significant p<0.05, 0.05, 0.001, 0.001 for FVC₁, FEV₁ % and PEFR respectively. The difference between the CaO₂ of the subjects and that of the control group was statistically significant with p-value less than 0.05.

Table 4 shows a correlation of ventilatory function parameter PEFR, FVC FEV₁ and FEV₁ with duration of exposure of men and women to cooking gas. The negative coefficient of correlation (r) indicates a general decline of ventilatory functions values with increase in the duration of exposure to cooking gas. The relationship was significant for duration of exposure versus PEFR, FVC and FEV1 (p<0.001).

Table 5 shows the mean CaO₂ of subjects and the duration of exposure to cooking gas. The majority of the subjects 13 (43.3%) were exposed for a period less than or equal to 24 months, yet the mean CaO₂ was 14ml.dl⁻¹ (normal value = 20ml.dl⁻¹). One (3.3%) subject was exposed for a period > 96 months and his CaO₂ was the least; 12.30 ml.dl⁻¹.

Fig. 1 is a histogram showing the relationship between arterial oxygen content and duration of exposure of the vendors to cooking gas. There was a gradual decline in the arterial oxygen content with increase duration of exposure to the cooking gas.

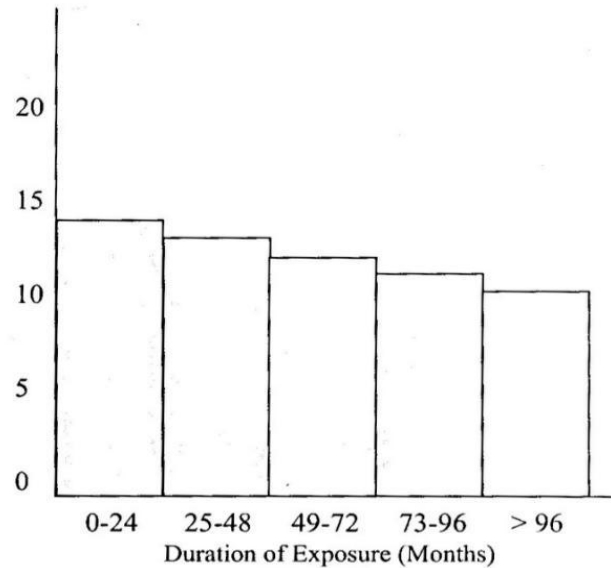


Figure 1: Mean CaO₂ and duration of exposure.

Discussion

This study has amply demonstrated that chronic exposure of gas vendors to cooking gas has deleterious effects on their oxygen carrying capacity and lung function.

The butane and nitrogen dioxide gases which form major components of cooking gas have been implicated in a number of studies for causing both acute, and chronic effects on health, particularly people with asthma^{1,2}.

From the result of the study, the gas vendors spend about 80% of their time indoors, taking over 10,000 breaths per day. Given the amount of time spent indoors in their shops, they are exposed to a variety of chemicals. Studies in the UK have found higher average concentrations of NO₂ in homes with gas cookers (25 – 70kg/m³) than in homes without gas cookers (13 – 40 kg/m³)^{6,7}. Similarly a maximum one hour average concentration of 1,115kg/m³ has been recorded in a gas cooker equipped kitchen. Thus considering the duration of exposure of gas vendors to cooking gas and the degree of gas spillage from cylinders, the vendors must have been exposed to higher concentrations of Nitrogen dioxide. High levels of NO₂ affect the airway and lungs causing respiratory illnesses and obstructive lung disease^{6,8}. Therefore, because NO₂ is only one of a number of potentially hazardous substances produced in cooking gas, it would be advisable to reduce the time of exposure to cooking gas.

The gradual decline in arterial oxygen content of the vendors with increased duration of exposure to cooking gas revealed in this study confirmed the impaired oxygen carriage capacity of vendors resulting from chronic exposure to cooking gas.

Similarly, there was a general reduction in the ventilatory function indices of vendors (FVC, FEV₁ &

FEV₁%, PEFR) as when compared with controls, (p<0.03, 0.05, 0.001, 0.001) respectively. This amply illustrates lung function impairment of the vendors resulting from chronic exposure to cooking gas. Also, in the correlation coefficient, between the duration of exposure to cooking gas and ventilatory function parameters, there was a negative correlation (r) with increase in duration of exposure of vendors to cooking gas. This shows that there is progressive lung function impairment with prolonged exposure to cooking gas. This is similar to the findings in other studies of the possible effects of cooking gas on ventilatory function^{4,6,8}.

In very high concentration such as have occurred in shops of gas vendors nitrogen dioxide can cause very severe lung damage⁹.

The mechanism by which nitrogen dioxide acts is most probably related to its properties as an oxidizing agent which can damage cell membranes and protein⁶. At relatively high concentration, nitrogen dioxide cause acute inflammation of the airways⁹. In addition, a short term exposure can affect the immune cells of the airway in a manner that might predispose people to an increased risk of respiratory infection. In addition, studies have further shown that inhalation of nitrogen dioxide at moderate concentrations may cause an inflammatory reaction in the lungs or may increase the susceptibility of individual to subsequent inhalation of allergens, such as those from house dust or grass pollen¹⁰. There is also some evidence that exposure to nitrogen dioxide can enhance the response of someone with asthma to inhalation of allergen¹⁰.

A study in East Anglia showed that young women who had gas cookers subsequently had more respiratory symptoms and poorer lung function than the average women from homes with electric cookers^{11,12}.

Apart from the effect of the gas on the respiratory system and blood oxygen carrying capacity, nitrogen dioxide may combine with other atmospheric chemicals, notably polycyclic aromatic hydrocarbon to produce nitroamines^{6,7,12}. Many nitroamines are in vitro mutagens and some are carcinogenic in animals exposed to high doses. It is also said to be a possible carcinogen in humans.

Various ways have been suggested to remove these pollutants generated indoors before they can build up to high concentrations¹⁴. The air exchange with the outside air (assuming, of course, that, the outside air remain relatively unpolluted) is essential in maintaining healthy indoor gravity.

In conclusion, cooking gas vendors should be educated and counselled to adopt various methods to prevent indoor pollution. These measures should include better careful attention to cylinders and then values to ventilation of the shops. From this study the damage to health in sleeping overnight in unvented gas shops is made patently clear.

The pulmonary function impairment and decreased

arterial oxygen content in cooking gas vendors put them at risk of impaired gaseous exchange and thus hypoxaemia in an event of anaesthesia and surgery. It is therefore, advisable to assess arterial oxygen content and to perform pulmonary function tests on all cooking gas vendors who present for anaesthesia and surgery. Regional anaesthetic technique should be the choice in this group of individuals if no contraindications exist.

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REFERENCES

1. Roberts M. J., McIvor R. A., Adgey A. A. Asytle following butane gas inhalation *Br. J. Med.* 1990; **44**: 294.
2. Williams D. R., Cole S. J. Ventricular Fibrillation following butane gas inhalation Resuscitation 1998; **37**: 43 – 45.
3. Esmail A., Anderson H. R., Ramsey J. D., Taylor J., Pottier A. Controlling deaths from volatile substance abuse. In under 18s: the effect of legislation. *Br. Med. Journal* 1992; **305**: 692.
4. Jarvis D., Chinn S., Leuzynska C. and Burney P. Association of respiratory symptoms and lung function in young adults with use of domestic gas appliances. *The Lancet* 1996; **347**: 426 – 431.
5. Tien Y. K., Garbel R. A. Prediction of PO₂ from SO₂ using the standard O₂-Hb equilibrium curve. *J. App Physio* 1977; **42**: 985.
6. The effect of Nitrogen dioxide in health, UK department of Environment food and rural affairs publication. 1998, 1 – 4.
7. Institute for Environment and health Assessment on indoor air quality in the home. *Bulletin Leicester* 1996; 1 – 2.
8. Reed C. E., Asthma; Chronic desquamating eosinophilic bronchitis; In; immunological mediated pulmonary disease. Lynch JP. Deremme RA eds. *JB Lippcott company peliadelphia* 1991; 322 – 39.
9. Devalia J. L., Campbell A. M., Sapsford R. J. Effect of nitrogen dioxide on synthesis of inflammatory cytokines expressed by human bronchial epithelial cells in vitro *Am J Respir Cell Mol. Bio* 1993; **9**: 271 – 278.
10. Reed C. E., Swanson M. C. Indoor allergens. Identification and quantification Environment Intermit. 1986; **12**: 15 – 20.
11. Mc Falden E. R. (Jr.) Asthma. In: Harrison's principle of internal medicine. Wilson JD. Ed. 12th ed. Vol. 2 McGraw Hill, New York 1991; 1048 –1052.
12. Gamamage and Kaye Indoor air quality and building energy efficient homes Boneville Power Administration office of Conservation publication. 1984; 1 – 6.
13. Nuess M., Prill R. Indoor Air quality. Washington State University cooperative extension publication. 1997; 1 – 6.
14. Berry R. W., Brown V. M. Coward S. K. et al. Index air quality in homes. The building research establishment indoor environment study report 1997; 1 – 4.