

A Relationship between a Level of Carboxyhemoglobin and Hemoglobin in Bus Mechanics

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Abstract. Carbon monoxide (CO) is a highly toxic gas; the result of incomplete combustion emitted from motor vehicle fumes. Bus mechanics are workers who are at risk of CO exposure from motor vehicle fumes. This gas will enter the bloodstream and bind Hemoglobin (Hb) to Carboxyhemoglobin (COHb). This can reduce the capacity of the blood to send oxygen to body tissues. As compensation, the body will increase the process of erythropoiesis, which can increase Hb production. The purpose of this study was to determine the average picture of COHb, Hb levels, and the relationship of increased levels COHb with Hb levels in bus mechanics in Tasikmalaya. Design research uses a cross-sectional approach against 30 subjects. Examination of the level of COHb and Hb in laboratory STIKes Muhammadiyah Ciamis. Subjects exposed to CO with an average COHb level of 42.3 mg / L and an average level of Hb of 14.0 g/dL. Statistical test results show no relationship between COHb levels with an increase in Hb levels in bus mechanics in Tasikmalaya $p > 0.01$ ($p = 0.573$; $r = 0.107$). Conclusion, the average COHb level in bus mechanics in Tasikmalaya exceeds the allowable limit, while the Hb level is still in the reference value and an increase in COHb level not obtained with an increase in Hb.

1. Introduction

Carbon monoxide (CO) is a colourless and odourless toxic gas that is produced mainly as a result of burning incomplete hydrocarbon fuels such as oil, natural gas, wood, and coal [1]. Symptoms of CO poisoning are not specific. Mild exposure causes dizziness, headaches, myalgia, or neuropsychological disorders. Severe exposures to CO result in, loss of consciousness, and even death [2]. CO enters the body through the lungs and reversibly binds to oxygen, which carries the component of haemoglobin to form carboxyhemoglobin (COHb). Decreases the carrying capacity of oxygen in haemoglobin in binding to iron (II) atoms, where CO binds Hb 200-250 times more readily [3]. CO increases cytosolic heme levels, causes oxidative stress, and binds to platelet heme proteins and cytochrome c oxidase interferes with cellular respiration, and causes the production of reactive oxygen species, which in turn causes neuronal necrosis and apoptosis. CO exposure also causes inflammation through several pathways that are not dependent on the hypoxic pathway, which results in neurological and cardiac injury [2]. As compensation, the body will increase the process of erythropoiesis, which can increase Hb production. Thus, Hb levels in the blood will increase [4].

CO poisoning is quite common among the population, but certain occupational groups carry a higher risk of being exposed to this gas because of their work. Working groups include working in the auto services mechanical division, auto service maintenance personnel, exhaust plumbers, gas pumpers,



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and traffic police officers [1]. Research conducted by Sepriatno and Sainab in 2009 in Makassar, there were six mechanics (27.3%) whose COHb levels in their blood exceeded the threshold value determined by the ACGIH (American Council of Government Industrial Hygienists) of 3.5%, or threshold value determined by occupational safety and health (OSHA) of 35 mg/L [5][6]. The research objectives are to know the average overview of the COHb, Hb level, and COHb levels with an increase in Hb levels in bus mechanics in Tasikmalaya.

2. Methods

The population of this study is all mechanical mechanics in the City of Tasikmalaya. There are five PO buses in the city of Tasikmalaya. The sample in this study was thirty bus mechanics in the City of Tasikmalaya, who agreed to be asked to participate in the study. This research is an analytic survey research using cross-sectional, that is, the data is observed at the same time, meaning that each research subject is observed only once, together with between COHb levels and Hb bus mechanics. The technique of collecting data in this study is to use the type of primary data that is data obtained by collecting samples obtained directly to determine COHb and Hb levels in bus mechanics in Tasikmalaya. Data analysis of the results of the study was carried out using statistical tests if the data were normally distributed using Pearson Correlation. If the data are not normally distributed then use the Spearman Correlation.

The equipment used in the study is Hematology Analyzer (Sysmex XP-300). UV-Vis spectrophotometer (Camspec M550), Conway plate, volumetric flask, and micropipette. The material used in the study are: K₃EDTA vacuum tubes, 3 mL syringes, alcohol swabs, distilled water, blood control (EIGHTCHECK-3WP), 0.00442 mol/L palladium(II) chloride (Merck), 0.01N hydrochloric acid, 5N sulfuric acid, and 5% potassium iodide.

2.1. Hb Procedure

Hb discusses using a hematology analyzer, which has carried out quality control with normal level control material (Sysmex lot 8286 0822) with results still in the control range of 12.6-13.4 g/dL and high levels (Sysmex lot 8285 0823) with results still in the control range of 15.9 -16.9 g/dL.

2.2. COHb Procedure

Standard Curve : Prepared six volumetric flasks of 10 mL, each pumpkin added 5 mL of distilled water and 1 mL of 5% potassium iodide solution. Then make a solution of 0, 0.1, 0.2, 0.3, 0.4, and 0.5 mL of palladium (II) chloride in a 10 mL volumetric flask with distilled water. Then each is read on a spectrophotometer with a wavelength of 420 nm.[4] With the regression equation $y = 249.32x + 0.03$ and $r^2 = 0.9901$. Shows high linearity, where the linearity of the standard curve can see from the value of the conversation coefficient, which is 0.9901, meaning $\pm 99.01\%$ change in absorbance changed by changes in COHb concentration, while $\pm 0.99\%$ depends on other factors.

Specimen Analysis : The middle part of the Conway cup is closed clean, after the middle part of the cup is smeared with vaseline then tilted. Then a blood sample of 1 mL and 0.5 mL of 5N sulphuric acid was put into the inner well. 2.0 mL palladium (II) chloride on the outside of the well. The cup was closed and rotated 360 ° so that it mixed with sulphuric acid, then incubated for 1.5 hours. Then the Conway cup lid is opened. The palladium (II) chloride solution in the outer well was piped at 0.5 mL using a micropipette, then transferred to a 10 mL volumetric flask containing 5 mL of distilled water and 1 mL of 5% potassium iodide. Add distilled water to the boundary mark. Read on a spectrophotometer with a wavelength of 420 nm with distilled water as a blank [4].

3. Result and Discussion

COHb and Hb examinations were carried out on thirty mechanical adult men in the city of Tasikmalaya. The type of specimen used in COHb and Hb examination is venous blood with K₃EDTA anticoagulant. Venous COHb levels predict arterial levels with a high degree of accuracy, with a difference of 2.1-2.4% COHb. Patients suspected of CO poisoning can be screened using venous blood, without the need for an arterial puncture [7].

Table 1. Results of COHb and Hb

No	Examinations	N	Mean	Minimum	Maximum
1.	COHb (mg/mL)	30	42,3	3,2	76,1
2.	Hb (g/dL)	30	14	11	18

Table 1 shows the mechanical bus in the city of Tasikmalaya has an average COHb level of 42.3 mg / mL, exceeding the threshold value determined by Occupational Safety and Health (OSHA), which is 35 mg/mL. It shows that workers like a mechanic bus at risk for clicking natural CO intoxication. Also, people such as firefighters, construction and garage workers, propane indoor forklift operators, welders, underground parking attendants, and outdoor workers in urban areas with heavy traffic can be expos to carbon monoxide at work [8].

CO gas in this research case originated from bus exhaust. Most of the accidental poisoning of poison gas has been reported to be associated with motor vehicle exhaust fumes.[9] Besides, CO is a natural molecule in the human body, to some degree; which is a product of haemoglobin degradation and produces 1-3% COHb saturation in non-smokers and 5-10% smokers [10].

Table 2. Correlations between COHb and Hb

	N	Pearson Correlation	Sig. (2-tailed)
Correlations between COHb and Hb	30	.107	.573

Correlation is significant at the 0.01 level (2-tailed)

Table 2 shows the mean Hb values in mechanics still within the range of adult male reference values of 14-18 g/dL [11]. Based on statistical tests using the Pearson correlation test in table 2 shows there is no relationship between the increase in COHb levels with Hb in the technique with a significance value of $p = 0.573$ ($p > 0.01$) with $r = 0.107$. Contrasts with previous studies conducted by Ischornia (2016), who conducted a study of the relationship of COHb levels with Hb and Hematocrit in smokers. However, the same with Priyanto's research (2016), which states there is no relationship between COHb and Hb [4]. This is consistent with Dubey's (2017) statement that in cases of CO intoxication, Hb concentration may be normal, but oxygen levels in the blood significantly reduced [12].

Where CO diffuses across the alveolar-capillary membrane and quickly binds to haemoglobin to form COHb, it can interfere with the ability of oxygen to bind to and detach from Hb [13]. CO affects organisms in various ways 1.CO binds to Hb and forms COHb molecules. Prevents oxygen transport and releases it to the tissues. Thus, it produces relative anemia, tissue asphyxia, and hypoxia. 2. COHb increases the adhesion of white blood cells to the endothelial surface, especially in brain tissue. CO causes inflammatory changes that depend on leukocytes and lipid peroxidation in the brain and cause oedema of white matter demyelination and focal necrosis, also causing reperfusion damage. 3. CO binds to myoglobin, also with high affinity, up to 20-50 times. It causes hypotension and myocardial depression by causing tissue hypoxia. 4. CO binds to muscle myoglobin, reduces O₂ pressure, and leads to rhabdomyolysis. Thus interferes with oxidative phosphorylation, reduces cellular respiration, and causes cellular hypoxia. 5. CO causes structural changes in the Hb molecule and makes it difficult to

provide oxygen to tissues [10]. Thus, CO toxicity manifests itself as a result of tissue and cellular hypoxia and causes clinically detected signs and symptoms when COHb is higher than 10-20%. In addition to tissue hypoxia, several other open CO toxicity mechanisms have recently described. These include CO-induced oxidative stress, peroxynitrite formation, inflammation, apoptosis, and immune-mediated injury. Thus, clinically relevant CO toxicity results from tissue hypoxia and direct cellular effects. The organs with the most significant aerobic activity, such as the brain and heart, are most susceptible to marked CO toxicity. Cardiovascular effects of acute CO poisoning include hypotension due to vasodilation, arrhythmias, ischemia, infarction, and heart attacks, while neurological manifestations include headaches, dizziness, impaired judgment, confusion, changes in mental status, seizures, syncope, stroke, and coma [13]. This shows that CO intoxication is not related to erythropoiesis.

4. Conclusion

The average COHb level in bus mechanics in Tasikmalaya exceeds the allowable limit, while the Hb level is still in the reference value and an increase in COHb level not obtained with an increase in Hb.

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