

Acute Carbon Monoxide-Induced Cardiotoxicity: Clinical Study

*Mohamed AM Khalaf, **Nevin A El-Desouky, ***Ghada MA El-Galad and
*Ahmed H. Abbas,

Dept. of Forensic Medicine and Clinical Toxicology - Faculty of Medicine - *Al-Minya,
Cairo, and *El-Fayoum Universities.

ABSTRACT

Background: Carbon monoxide poisoning is a serious health problem that represents the 6th most frequent toxic exposure in Egypt. Cardiotoxicity may be one of the commonest causes of morbidity and mortality of such cases. **Objectives:** This study aimed at studying the commonest presentation of CO poisoning regarding the electrical and biochemical changes in relation to the carboxyhemoglobin level (COHb). **Patients and Methods:** Eighty six patients of both sexes were admitted at Al-Minya PCC with acute CO exposure during the period from 1st of October 2007 to 30th of April 2010. Diagnosis was confirmed by COHb blood level on admission. Two persons died on arrival and the study was carried out on the remaining 84 persons. They were classified into 4 different groups according to the COHb level: group I (control group): consists of 20 apparently healthy persons; group II (Mild toxicity); COHb% is 10-20 % (No. =34); group III (Moderate toxicity); COHb% is 20-40 % (No. = 36), and group IV (severe toxicity); COHb% is > 40% (No. = 14). All subjects were investigated for electrical changes by continuous electrocardiogram monitoring and measuring the cardiac Biomarkers (ALT, AST, LDH, CK-MB, and cTnT). **Results:** Regarding to pulse, there was no significant difference between the 3 intoxicated groups. Estimation of blood pressure revealed that most patients were normotensive or hypotensive, while hypertension was infrequent. In group II; 16 persons presented with tachycardia, and 6 with bradycardia. In group III; 22 persons presented with tachycardia, 5 with bradycardia. In group IV; 11 persons presented with tachycardia, 3 with bradycardia. There was a significant difference between groups II, IV and III, IV according to palpitation and dysrhythmias but not between groups II, III. One person of group III and 3 in group IV developed myocardial ischemia with a significant difference between groups II, IV and groups III, IV, but there was no significant difference at all between groups II, III. Interpretation of ECGs showed that: in group II; only one showed a depressed ST segment, 2 showed elevated ST segment, and the resting (31/34) had normal ECG, in group III; (27/36) had normal ECG and (9/36) showed abnormalities; depressed ST segment (2), elevated ST segment (2), inverted T-wave (1), paroxysmal ventricular contractions (PVCs) (2) and prolonged Q-T interval (2), in group IV; (6/14) had normal ECG and (8/14) showed abnormality; depressed ST segment (2), elevated ST segment (2), inverted T-wave (1), PVCs (2) and prolonged Q-T interval (1). There was a significant increase between group II, IV and III, IV when compared to each other regarding the ECG changes. There was no significant difference in ALT and AST levels in patient groups when compared together or to control. There was a significant increase of LDH in group IV when compared to group II, and a significant increase in LDH in all intoxicated groups when compared to control. There was a significant increase of CPK-MB and cTnT levels in groups III and IV when compared to group II or to control. There was a positive correlation of the severity of the cardiovascular manifestations and the electrical and biochemical changes to the blood level of COHb. **Conclusion:** CO poisoning can induce serious cardiotoxic effects which are directly proportional to the blood level of COHb.

KEYWORDS: Carbon monoxide, Cardiotoxicity, Electrocardiogram, Cardiac enzymes, Cardiac biomarkers.

INTRODUCTION

Carbon monoxide poisoning is a serious health problem resulting in approximately 40,000 visits to the emergency department annually in the United States (Weaver et al., 2002).

An Egyptian study by the Poison Control Center (PCC), Ain Shams University Hospitals in Cairo, showed that CO poisoning represented the 6th most frequent toxic exposure (2.28%) out of 25,555 cases admitted to the PCC in the year 2004 (Gamalludin et al., 2005).

As Cardiotoxicity may be one of the commonest causes of morbidity and mortality of such cases (Satran et al., 2005), the current study aimed at studying the commonest cardiotoxic presentation of CO poisoning regarding the electrical and biochemical changes in relation to the carboxyhemoglobin level (COHb).

SUBJECTS AND METHODS

This study was carried out at Al-Minya Poisoning Control Center (PCC) during the period from the 1st of October 2007 to the 3th of April 2010.

Subjects:

Eighty six patients of both sexes (Males = 53, Females = 31) were admitted at the PCC with history or presentation of acute CO poisoning which was variable. Two persons presented died and the study carried out on the remaining 84 persons (Males = 51, Females = 31). The diagnosis was confirmed by serum level of COHb on admission. The investigated subjects were classified into 4 different groups according to the COHb level

Group I (control group): consists of twenty healthy persons.

Group II (Mild toxicity): the level of COHb% is 10-20% (34 patients).

Group III (Moderate toxicity): the level of COHb% is 20-40% (36 patients).

Group IV (severe toxicity): the level of COHb% is > 40% (14 patients).

Exclusion criteria

The patient with a delay time between CO exposure and arrival to PCC more than two hours were excluded. The chosen subjects were with no past history of neurological, cardiovascular, respiratory, renal, gastrointestinal, or metabolic disorders. Those with risk factors such as pregnancy, smoking, obesity, and extremities of age, were also excluded from this study.

Investigations

Biochemical analyses

Biochemical Parameters

The laboratory work of the study was conducted at the laboratory of Al-Minya university hospitals. Five milliliters of venous blood were collected from every patient on admission by using sterile plastic syringes. The samples were used for estimation of the following parameters:

- Carboxyhemoglobin level using blood gas analyzer with co-oximeter (Bayer 855).
- Serum alanine aminotransferase (ALT) was determined by a colorimetric method using automated chemistry analyzer (Thermo Electron, model Konelab 20i, Finland). Normal range of ALT up to 40 U/L for men and up to 32U/L for women in 37 °C.
- Serum Aspartate aminotransferase (AST) was determined by a colorimetric method using automated chemistry analyzer (Thermo Electron, model Konelab 20i, Finland). Normal range of AST up to 40 U/L for men and up to and up to 32U/L for women in 37 °C.

- Lactate Dehydrogenase (LDH) concentration is determined by a colorimetric method using automated chemistry analyzer (Thermo Electron, model Konelab 20i, Finland). Normal range of LDH 200-400 U/L in 37 °C.
- Creatine kinase-MB (CPK-MB) concentration is determined by a colorimetric method using automated chemistry analyzer (Thermo Electron, model Konelab 20i, Finland). Normal range (up to 24 U/L).
- Troponin T (cTnT) was studied with immunoassay method using monoclonal antibodies (Enzymum Test System ES 300; Boehringer Mannheim, Mannheim, Germany). Levels >0.1 µg/ml were accepted as indicating myocardial damage.

Electrocardiogram (ECG)

Electrocardiographic recording was done for every patient on admission then repeated for those admitted to ICU using FuKuda densti Cardimex (model Fx-2111, Japan).

Statistical analyses

Data were checked, coded, and analyzed by using SPSS (version 11.0 software package). Numerical data were expressed as mean ± SD. Comparison between 2 independent groups of categorical data (e.g., incidence of symptoms) was done using Pearson's chi-square test. Comparison between 2 independent groups of mean of numerical data (e.g., mean B.P., pulse, ALT, AST, ...etc.) was done using student-*t* test. The correlation of the level of COHb to the electrical and biochemical changes was assessed by regression analysis. P-value was considered significant if < 0.05, high significant if < 0.01 and non significant if > 0.05 for all tests.

RESULTS

There was no significance between three different intoxicated groups

according to pulse. Estimation of blood pressure revealed that in group II, there were (15 /34) normotensive, (15/34) were hypotensive and (4/34) were hypertensive, in group III; (17/36) were normotensive, (15/36) were hypotensive and (4/36) were hypertensive, while in group IV; (3/14) were normotensive, (9/14) were hypotensive and (2/14). In group II; 16 persons presented with tachycardia, and 6 with bradycardia. In group III; 22 persons presented with tachycardia, 5 with bradycardia. In group IV; 11 persons presented with tachycardia, 3 with bradycardia. There was a significant difference between groups II, IV and III, IV according to palpitation and dysrhythmias but not between groups II, III. One person of group III and 3 in group IV developed myocardial ischemia with a significant difference between groups II, IV and groups III, IV, but there was no significant difference at all between groups II, III (Table 1).

Electrocardiographic changes (ECGs)

Interpretation of ECGs showed that: in group II; only one showed a depressed ST segment, 2 showed elevated ST segment, and the resting (31/34) had normal ECG, in group III; (27/36) had normal ECG and (9/36) showed abnormalities; depressed ST segment (2), elevated ST segment (2), inverted T-wave (1), paroxysmal ventricular contractions (PVCs) (2) and prolonged Q-T interval (2), in group IV; (6/14) had normal ECG and (8/14) showed abnormality; depressed ST segment (2), elevated ST segment (2), inverted T-wave (1), PVCs (2) and prolonged Q-T interval (1). There was a significant increase between group II, IV and III, IV when compared to each other regarding the ECG changes. (Table 2, Fig 1-2).

Biochemical changes

There was non significant difference in liver enzymes (ALT and AST) in patient groups when compared together or to control (Table 3-4).

There was a significant increase of LDH in patients with severe poisoning when compared to patients with mild poisoning, and there was a significant increase in LDH in all groups of intoxicated patients when compared to control (Table 3-4).

There was a significant increase of CPK-MB in patients with moderate

and severe poisoning when compared to patients with mild poisoning or to control (Table 3-4).

Troponin-T level was significantly increased in the patients of the groups III and IV when compared to the level of the patients of group II and the control group.

There was a positive correlation of the severity of the cardiovascular manifestations and the electrical and biochemical changes to the blood level of COHb (Table 5).

Table (1): Cardiovascular manifestations of the studied patients.

Cardiovascular manifestations	Group			P-value and Chi					
	II	III	IV	II vs. III		II vs. IV		III vs IV	
				chi	P	Chi	P	chi	p
Tachycardia	16	22	11	1-1	5.52	5.22	0.021*	5.56	0.019*
Bradycardia	6	5	3	0.58	0.35	0.31	1.09	0.64	0.27
Dysrhythmias				0.41	0.74	14.1	0.001**	14.3	0.001**
Normal Bl.P	16	18	3	0.92	0.06	0.13	2.39	0.09	2.95
Hypotension	14	14	9	0.75	0.09	0.16	1.93	0.08	2.99
Hypertension	4	4	2	0.82	0.07	0.93	0.03*	0.96	0.007**
M.I	0	1	3	0.39	0.72	5.4	0.02*	4.1	0.04*

Bl.P: blood pressure; M.I: Myocardial ischemia; *: significant; **: high significant.

Table (2): ECG changes of the studied patients.

ECG findings	Group			P-value and Chi					
	II	III	IV	II vs. III		II vs. IV		III vs. IV	
				Chi	p	chi	P	chi	p
Normal ECG	31	27	8	0.30	1.05	10.2	0.001*	6.82	0.009*
Abnormal ECG	3	9	6	0.30	1.05	10.2	0.001*	6.82	0.009*
Elevated ST	2	2	2	0.74	0.10	0.27	1.17	0.37	0.77
Depressed ST	1	2	2	0.80	0.05	0.27	1.17	0.15	2.04
Inverted T wave	0	1	1	0.39	0.72	0.06	3.5	0.15	2.04
PVCs	0	2	2	0.39	0.72	0.19	1.70	0.52	0.39
Q-T interval	0	2	1	0.39	0.72	0.19	1.70	0.52	0.39

N.B. In group IV: One patient recorded to have a combined depressed ST-segment with inverted T-wave, and another patient was recorded to have a combined elevated ST-segment with paroxysmal ventricular contractions (PVCs). *: significant; **: high significant.

Table (3): Values of the biochemical parameters (mean ± SD) of the different studied groups.

Parameter	Group			
	I	II	III	IV
ALT (U/L)	21±5	22±6	24±11	23±8
AST (U/L)	20±6	21±11	23±6	22±9

LDH (U/L)	170±30	230±57	330±280	380±260
CPK-MB (U/L)	17±5	23±17	42±22	54±31
cTnT (µg/ml)	0.04±0.01	0.05±0.02	0.36±0.01	1.1±0.02

Table (4): Comparison of the biochemical parameters (Mean ± SD) of different studied groups.

Parameter	Groups											
	II Vs III		II Vs IV		III Vs IV		II Vs I		III Vs I		IV Vs I	
	t-value	P-value	t-value	P-value	t-value	P-value	t-value	P-value	t-value	P-value	t-value	P-value
ALT	0.42	0.83	0.66	0.46	0.77	0.33	0.57	0.61	0.27	1.18	0.39	0.92
AST	0.37	0.91	0.77	0.31	0.66	0.48	0.72	0.39	0.09	1.79	0.45	0.79
LDH	0.09	1.77	2.81	0.009**	0.57	0.61	4.27	0.001**	2.57	0.01*	3.61	0.001**
CK-MB	3.64	0.001**	4.22	0.001**	0.13	1.57	0.14	1.54	5.11	0.001**	5.29	0.001**
cTnT	1.44	0.003**	1.27	0.001**	1.76	0.001**	0.78	0.06	1.64	0.001**	1.21	0.001**

*: significant. **: high significant.

Table (5): Correlation of the COHb level to the electrical and biochemical changes.

Group							
I		II		III		IV	
r-value	P-value	r-value	P-value	r-value	P-value	r-value	P-value
0.782	0.001**	0.884	0.001**	0.791	0.001**	0.962	0.001**

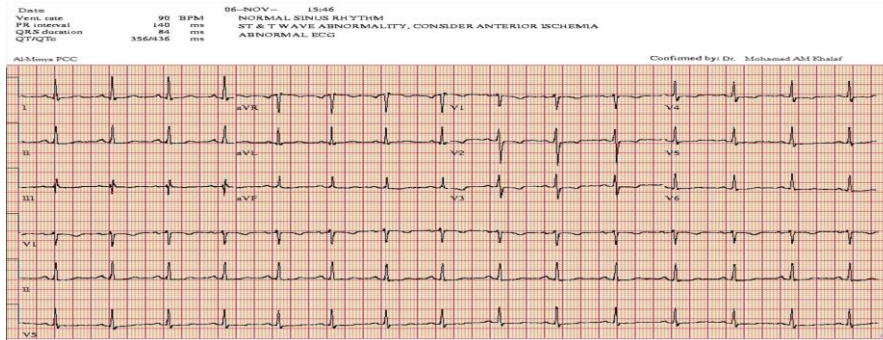


Fig. (1): A 12-lead electrocardiogram of a 47 male patient after prolonged exposure to CO (COHb = 32 %) showing normal sinus rhythm with abnormal ST & T- wave suggesting anterior ischemia.



Fig. (2): A 12-lead electrocardiogram of a 32 female patient 4 hours after CO exposure (COHb = 27 %) showing a prolonged QT interval. The longest QT interval was measured at 700 msec in lead III.

DISCUSSION

The current study concerned with studying the acute CO poisoning-induced cardiotoxicity and the possible correlation of the severity to the level of COHb. The results of this study revealed that tachycardia was the commonest manifestation detected in 58.33% (49/84) of the cases. Bradycardia was reported in 16.67% (14/84) of the patients while the resting 21 cases (25%) had normal heart rate.

These findings were in accordance with those of **Meert et al., (1998)**, who reported that tachycardia was one of the most common presenting sign in a study conducted to evaluate the clinical characteristics and neurological outcome of patients with CO poisoning treated with normobaric oxygen in the United States. Moreover, **Swank et al., (2004)**, stated that CO-induced sinus tachycardia was reported in a study in Ruby Memorial Hospital in the United States.

On the other hand, **Hampson and Zmaeff, (2001)**, stated that bradycardia was found among 10 cases out of 18 cases (55.5%) in a study to examine the outcome of group of patients with extreme CO poisoning in the United States.

Gandini et al., (2001a), reported that, although tachycardia is a common finding in CO poisoning and usually considered as a compensatory response to systemic hypoxia and cardiac dysfunction, yet bradycardia may be present, indicating rhythm disturbances.

In the present study, the incidence of hypotension, dysarrhythmia, and myocardial ischemia increased with increased severity of CO poisoning. These findings were in agreement with the findings reported by **Kao and Nanags (2004)**.

Hypotension was recorded in 46.43% (39/84) of the studied CO-poisoned patients. This agrees with

Raub et al., (2000), who stated that, severe CO poisoning may result in marked hypotension. Also, **Chamberland et al., (2004)**, observed hypotension in a study describing the cardiac dysfunction in acute CO poisoning in the United States.

The hypotensive effect of CO was explained by **Morita and Kourembanas, 1995**, who stated that CO may act as a regulator to the vascular tone either under physiological or pathophysiological conditions i.e. hypoxia. Also, **Kourembanas, (2002)** reported that, CO induced smooth muscle relaxation and vasodilatation of blood vessels as well as inhibition of platelet aggregation. This could be due to activation of guanyl cyclase enzyme and generation of cyclic GMP. In addition, **Gandini et al. (2001a)**, suggested that hypotension may be due to the vasodilatory effect of CO leading to ischemic hypotension and in turn may promote ischemic reperfusion injury. The authors also added that the action of CO as a physiological regulator of vascular tone may account, at least in part, for its vasorelaxant effect.

Additionally, hypotension may result from myocardial injury secondary to hypoxia, ischemia, a direct myocardial depressant activity from myoglobin binding, peripheral vasodilation, or a combination of the aforementioned and may persist even after neurologic and metabolic symptoms have resolved as reported by **Yanir et al (2002)**.

Hypertension was detected in 11.9% (10/84) of the studied cases, which agrees with a study done by **Meert et al. (1998)** who observed hypertension in a study evaluating the clinical characteristics and outcome of patients exposed to CO poisoning. Also, **Koskela et al. (2000)** observed

that the blood pressure was slightly higher in patients exposed to CO poisoning than those who were not exposed in a study on 931 male foundry workers in Finland to determine the predictors of ischemic heart disease among them.

The ECG recordings of the studied CO-poisoned patients on admission showed that 78.57% (66/84) of cases had normal ECG recordings, while 21.43% (18/84) of the patients had ECG changes. The commonest changes were in the form of ST-segment changes in 61.11 (11/18), PVCs in 22.22% (4/18), QT-interval prolongation in 16.67% (3/18) and T-wave changes in 11.11% (3/18).

These findings agree with **Satran et al., (2005)**, who reported 77 cases out of 230 CO-poisoned patients had diagnostic ischemic changes recorded in the ECG which were in the form of (26%) with ST-segment or T-wave changes "flat or inverted T-wave" and (4%) with ST-segment elevation, whereas (41%) had non specific ST-changes either elevation or depression of the segment and (27%) had no ischemic changes in a study to detect the cardiovascular manifestations of CO poisoning. Also, **Sorodoc et al., (2004)**, reported a non Q-wave anteroseptal acute myocardial infarction due to acute CO poisoning in a study to detect complications of CO poisoning in Italy. In addition,

Chamberland and his fellows, (2004), observed right bundle branch block in a study describing CO-induced cardiac dysfunction in the United States. The authors stated that the reversible nature of the conduction disease could have been consistent with acute CO-induced myocardial stunning.

The ECG changes in cases of acute CO poisoning can be attributed to the direct toxic effect of CO on the heart or due to CO-induced depression

of respiratory and central nervous systems causing cardiac affection (**Blumenthal, 2001**). On the other hand many authors suggested that CO-induced myocardial dysfunction might take place without ECG changes which could be attributed to myocardial stunning. **Tritapepe et al. (1998)** explained that restoration of the intracellular respiration caused by CO wash out might result in reperfusion injury due to production of reactive oxygen species leading to myocardial stunning and cardiac dysfunction. The probable diagnosis of reperfusion-induced myocardial stunning was supported by the good response to inotropic agents. The stunned myocytes, although unable to contribute to efficient ventricular contractility, still preserve metabolic viability suggesting that myocardial dysfunction can occur without ECG changes or elevation of enzyme levels typical of ischemia (**Yanir et al., 2002**).

Surprisingly, it has been found that there was no significant difference in ALT and AST levels in patient groups when compared together or to control. Regarding the recorded Ck-MB changes, there was a significant increase of CK-MB in patients with moderate and severe poisoning when compared to patients with mild poisoning or to control, a significant increase of LDH in patients with severe poisoning when compared to patients with mild poisoning, and there was a significant increase in LDH in all groups of patients with COP when compared to control. This is concomitant with the reports of **Brogan et al., (1997)**, who had found that measuring serum CK-MB was efficient to predict a wide range of short term adverse events in patients presented with chest pain. Moreover, **Collinson (1998)**, stated that the diagnostic criteria for myocardial

injury have been classically based on the triad of history, ECG and measurement of cardiac enzymes. The choice of the enzymes has been dictated by the evolution of laboratory techniques, starting from measuring serum AST and progressed to serum CK-MB levels. In addition, **Zhang and his colleagues, (2003)**, in a study to detect cardiac injury in 62 cases with acute CO poisoning stated that determination of serum myocardial enzymes was helpful to detect myocardial injury. These findings were in accordance with **Pach and his fellows, (1998)**, who stated that considering the results of ECG and enzymes (AST, CK-MB) activity alone without performing perfusion scintigraphy scans (which indicate decrease blood flow to the myocardium) were not sufficient to diagnose cardiac dysfunction in seven patients presented with acute CO poisoning. This may be in accordance with the results of the current study regarding the reports of AST and ALT, but not in agreement with our results regarding LDH and CK-MB activity.

Our results revealed that there was a significant increase in the level of Troponin-T in the moderately and severely intoxicated patients when compared to the level of the mildly intoxicated patients the control group. These findings were supported with those of **Aslan and his fellows, 2005**, who reported a strong link between the ECG changes, the biochemical changes and the level of COHb. Moreover, they suggested that significant myocardial damage and life-threatening cardiac hemodynamic changes do not develop in CO-poisoned patients with COHb level below 60 % and without any known underlying cardiac disease. Based on their findings, they stated that it is not necessary to routinely measure CK, CK-MB and troponin-T, and perform myocardial perfusion

SPECT in acute CO poisoning cases without any ECG abnormality, ischemic cardiac symptoms. In our opinion, this is not true as many previous studies reported that myocardial damage may exist with elevated cardiac biomarkers with no ECG abnormalities (**Teksam et al., 2010**).

The reported positive correlation of the severity of the cardiovascular manifestations and the electrical and biochemical changes to the blood level of COHb are supported by many previous literatures that assure that the diagnosis of Co poisoning is normally made by measuring the percentage of carboxyhaemoglobin in either arterial or venous blood samples and that there is an excellent correlation between the two, as would be expected for a stable compound such as COHb (**Harper and Croft-Baker, 2004**). However, these results were on the contrary of a recent case report that stated frankly, after studying a severe case of CO poisoning with severe cardiopulmonary compromise and a very high COHb level, that the COHb concentrations do not always determine the severity of toxic damage at the level of selected organs, or serve as a prognostic index (**Wu et al., 2009**).

In conclusion, the results of the current study demonstrated that CO poisoning-induced cardiovascular toxicity were not uncommon presentation in our country and may be one of the leading causes of death among such patients. In addition, the morbidity of the cardiotoxic effects of CO correlated excellently with the COHb concentrations.

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أقسام الطب الشرعى و السموم الإكائينيكية - كلية الطب - جامعة المنيا* - جامعة القاهرة** - جامعة
الفيوم***

يعتبر غاز أول أكسيد الكربون من أكثر الغازات التي تسبب وفيات وإصابات في مصر 0
وقد أجريت هذه الدراسة لتقييم وقياس التغيرات الكيميائية والالكترونية والقياسات الحيوية المصاحبة للتأثير
السمي لهذا الغاز 0
وقد شملت الدراسة على 86 مريض في مركز السموم جامعة المنيا في الفترة من أول أكتوبر 2007 إلى 30
إبريل 2010 وتم تقسيمهم على 4 مجموعات حسب نسبة الكربوكسي هيموجلوبين في الدم 0
المجموعة الأولى (الضابطة) وتتكون من 20 فرد طبيعي 0
المجموعة الثانية تتكون من 34 مريض نسب الكربوكسي هيموجلوبين تتراوح بين 10-20% 0
المجموعة الثالثة نسبه تتراوح من 20-40% 0
المجموعة الرابعة نسبة الكربوكسي هيموجلوبين \uparrow 40% 0
وتم عمل رسم قلب وقياس العوامل الحيوية (النبض - ضغط الدم - سرعة دقات القلب) وأخذ عينات من الدم
لقياس انزيمات الدالة على إصابة عضلة القلب (ALT, AST, LDH & C TnT) لكل منهم ومقارنته
بالمجموعة الضابطة 0
لوحظ ارتفاع ضغط الدم وسرعة دقات القلب كلما زاد نسبة الكربوكسي هيموجلوبين ولم نجد أي فرق في قياس
النبض ولوحظ عدم انتظام دقات القلب في المجموعة الرابعة وقد عانى مريض في المجموعة الثالثة و3 مرضى
في المجموعة الرابعة من أزمة قلبية وقد لوحظ اختلافات شديدة وعدم انتظام في جهاز رسم القلب مصاحبا
لإرتفاع نسبة الكربوكسي هيموجلوبين 0
وبالنسبة للإنزيمات وجد عدم اختلاف في نسبة ALT & AST في اي من المجموعات والمجموعة الضابطة
بالمقارنة بارتفاع ملحوظ في نسبة LDH في المجموعة الرابعة بالمقارنة للمجموعة الثانية وارتفاع كبير عن
المجموعة الضابطة 0
أيضا هناك ارتفاع لانزيم CPK-MB, C TnT في المجموعة الثالثة والرابعة بالمقارنة بالمجموعة الثانية
والضابطة 0
ومن نتائج هذه الدراسة والتي أظهرت علاقة طردية بين ارتفاع نسبة الكربوكسي هيموجلوبين في الدم والتغيرات
الكيميائية ورسم القلب يمكن استنتاج أن غاز أول أكسيد الكربون له تأثير ضار على قلب الإنسان 0