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**STUDY OF SOME ENZYMES AS PREDICTORS
FOR THE SEVERITY OF ACUTE CARBON
MONOXIDE INTOXICATION**

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THESIS

Submitted to the Faculty of Medicine
University of Alexandria
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of
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By

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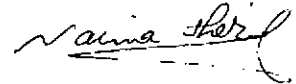
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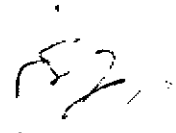
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LIST OF TABLES

No.	Name	Page
1	Neuro-psychiatric manifestations	11
2	The intensity of CO poisoning	15
3	Treatment guidelines of CO poisoning	23
	# Distribution of patients with acute intoxication by carbon monoxide admitted to APC at AUMH by :	
4	Age and sex	45
5	Duration of exposure	51
6	Latent period	52
7	Clinical presentation	53
8	Vital signs	54
9	Renal function tests	57
10	Complete blood picture	58
11	Arterial blood gases and acid-base status	59
12	Arterial blood gases, acid base status and level of consciousness	60
13	Carboxyhemoglobin %	61
14	Carboxyhemoglobin % and age	62
15	Carboxyhemoglobin % and sex	62
16	Carboxyhemoglobin % and duration of exposure	63
17	Carboxyhemoglobin % and latent period	64
18	Carboxyhemoglobin % and clinical presentation	65
19	Carboxyhemoglobin % and level of consciousness	66
20	Carboxyhemoglobin % and GCS	67
21	Carboxyhemoglobin %, arterial blood gases and acid base status	68

22	COHb % CPK, LDH and level of consciousness	69
23	GCS and blood gases, carboxyhemoglobin, CPK, LDH and acid-base status.	70
24	C.T findings and level of consciousness	71
25	Duration of stay in hospital and COHb %	75
26	Changes in CPK, LDH levels and kidney function tests in the case of rhabdomyolysis	76
27	Comparison between CPK and LDH levels on first and on discharge in patients stayed in the hospital > 1 day	77

LIST OF FIGURES

No.	Name	Page
1	Effects of anemia and carboxyhemoglobin on oxy-hemoglobin dissociation curve	2
2	Oxygen-Hemoglobin curve	7
3	Leucocyte-mediated carbon monoxide reperfusion injury pathway	9
4	Human maternal and fetal oxyhemoglobin saturation curves showing CO effect	10
5	CO elimination	26
6	Distribution of patients with acute intoxication by carbon monoxide admitted to APC at AUMH by sex	46
7	Distribution of patients with acute intoxication by carbon monoxide admitted to APC at AUMH by residence	47
8	Distribution of patients with acute intoxication by carbon monoxide admitted to APC at AUMH by level of education	48
9	Distribution of patients with acute intoxication by carbon monoxide admitted to APC at AUMH by mode of transportation	49
10	Distribution of patients with acute intoxication by carbon monoxide admitted to APC at AUMH by time of admission	50
11	Distribution of patients with acute intoxication by carbon monoxide admitted to APC at AUMH by levels of consciousness	55
12	Distribution of patients with acute intoxication by carbon monoxide admitted to APC at AUMH by severity of toxicity using Glasgow coma score (GCS)	56
13	Brain C.T showing brain oedema	72
14A.	Brain C.T showing hypodensity of the basal ganglia	73

14B. Brain C.T showing hypoxic insult in the basal ganglia	73
15 Brain MRI of the brain	74
16 Distribution of patients with acute intoxication by carbon monoxide admitted to APC at AUMH by Their outcome	78

CONTENTS

<u>CHAPTER</u>		<u>PAGE</u>
I	INTRODUCTION	1
II	AIM OF THE WORK	31
III	MATERIAL	32
IV	METHODS	33
V	RESULTS	34
VI	DISCUSSION	79
VII	SUMMARY	92
VIII	CONCLUSIONS	96
IX	RECOMMENDATION	98
X	REFERENCES	100
	PROTOCOL	
	ARABIC SUMMARY	

INTRODUCTION

INTRODUCTION

Historical Review

Carbon monoxide (CO) is a colourless, odourless, tasteless, and non irritant gas.⁽¹⁾ Because of these physical properties, CO gives no warning of its presence, hence its description as a silent killer.⁽²⁾

It normally appears in atmospheric concentration of 0.001% (10 ppm). However, The normal rate of endogenous CO production in man is 0.42 ml/hr as a result of normal hemoglobin (Hb) catabolic process.⁽³⁾

CO was first discovered with the introduction of hydrocarbon fuels which undergo incomplete combustion.⁽⁴⁾

Napoleon's surgeon; saw soldiers with CO-induced myonecrosis when billeted in huts heated by wood burning stoves.⁽⁵⁾

Incidence

True incidence of CO poisoning is not known, since non lethal exposures may go untreated or it may have been confused with other diseases as flu-like viral illness and gastroenteritis.^(6,7) It has been estimated that one-third of all cases of CO poisoning are undiagnosed.⁽⁸⁾

CO poisoning is the most common cause of toxin induced deaths in the U.S. and Great Britain and may account for as many as 5,000 deaths each year in the U.S., with an additional 10,000 patients seeking medical attention for toxic exposure.^(9,10) Mortality rates as high as 31% have been reported in United States (U.S).⁽¹¹⁾

CO poisoning may be deliberate or accidental, in the latter case, it may be : acute, subacute, or chronic.^(12,13,14)

Sources

I-Endogenous:

i. Hemoglobin catabolism:

Endogenous production of CO results from metabolism of the methane carbon atom in the protoporphyrin ring during hemoglobin catabolism and produces a COHb% of 0.4 – 0.7 %.⁽³⁾

Degradation of heme to biliverdine and CO appears to be the dominating source in most species.^(15,16) The enzyme heme oxygenase (HO), with 2 isoenzymes (HO-1 & HO-2), seems to be the rate limiting factor. HO-1 is reported to be irreducible, whereas HO-2 constitutively expressed⁽¹²⁾ as HO-2-like immuno reactivity is seen in local parasympathetic ganglia of human trachea and bronchi. These findings suggest that CO serves as a modulator of synaptic neurotransmission in the lung.⁽¹³⁾ The intensity of HO-1-like immuno-staining in the lung increases as the result of hypoxic challenge.⁽¹⁴⁾

Induction of HO-1 by reactive oxygen species followed by increased CO production has been put forward as a general cytoprotective mechanism against oxidative stress and therefore it is clinically useful in the detection and management of inflammatory lung disorders.⁽¹⁵⁾ This assumption is supported by increased levels of CO in which the exhaled air of asthmatic patients, during periods of non-steroidal treatment, during asthma exacerbation, and as a result of allergen-challenge.^(16,17)

Hemolytic anemia rises endogenous CO production and may increase COHb to 4-8 %.⁽¹⁷⁾ Effects of anemia and carboxyhemoglobin on oxyhemoglobin dissociation curve are illustrated in figure 1.⁽¹⁷⁾