بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها

علي هذه الأفلام قد أعدت دون أية تغيرات

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To be Kept away from Dust in Dry Cool place of 15-25- c and relative humidity 20-40%

بعض الوثائـــــــق الاصليـــــة تالفـــة

بالرسالة صفحات لم ترد بالاصل

STUDY OF THE EARLY EFFECTS OF HYPERBARIC OXYGEN BREATHING ON THE CELLULAR AND BIOCHEMICAL PROFILE OF BRONCHOALVEOLAR LAVAGE IN DIVERS

Thesis Submitted to
Benha Faculty of Medicine
In partial fulfilment of the requirement of
Degree of

Doctor of Chest Diseases

By

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1998

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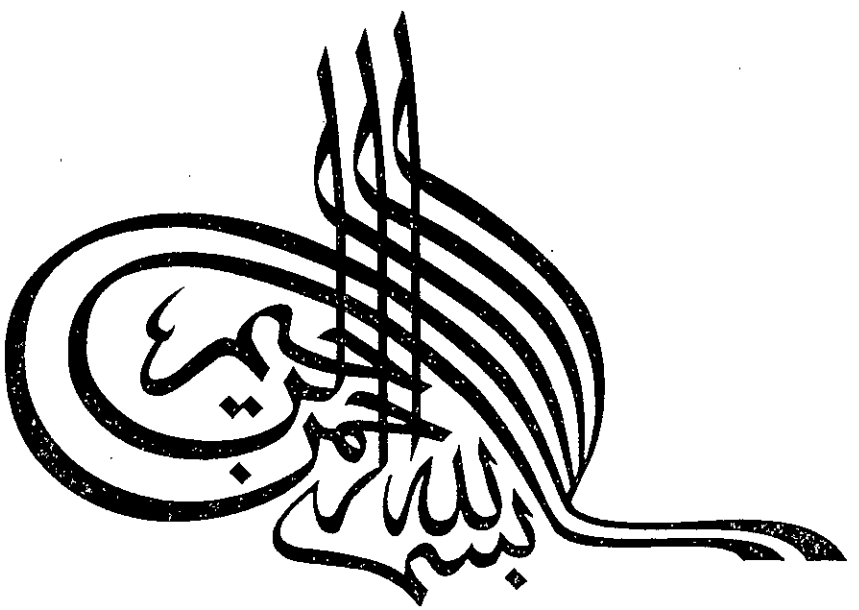
بسم الله الرحمن الرحيم

ACKNOWLEDGEMENT

Firstly, thanks to God who gave me the power to carry out the present work honestly and faithfully.

My deepest thanks to ***Professor Dr. Medhat Negm***, Professor of Chest diseases, Benha Faculty of medicine, for his thoughtfull ideas, meaningfull guidance, constant encouragement, valuable advices and willingness to denote a lot of his precious time and vast experience during the supervision of this work.

My deepest thanks to ***Professor Dr. Maysa Sharaf El Din***, Professor of Chest diseases, Cairo University, for her continous guidance, review and careful patient reading.

My special thanks to ***Professor Dr. Said Morsy*** and ***Professor Dr. Amr Badr El Din*** for their careful patient reading, for comments, and constructive creticism.

I am particularly indebted to our great couple ***Professor Dr. Mohamed Ahab Atta - Safaa Hussein*** for their continuous unlimited help and their excellent supervision and technical assistance.

My unlimited appreciation to ***Dr. Mohamed Abdel Hamid***, our consultant in Chest diseases in the Navy Military Hospital, for his constant moral and technical support and encouragement.

I am also grateful to ***Dr. Tarek El Halawany, Dr. Moustafa Esmat, Dr. Sameh Moris*** and their laboratory team which undoubtedly facilitated the completion of the present work, as well as to all of the people who have helped with this work.

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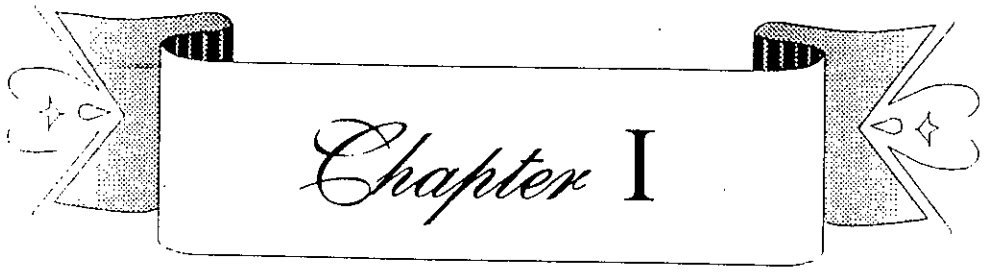
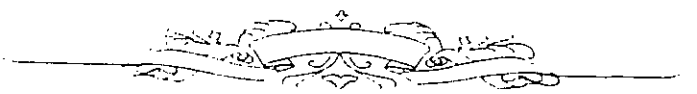
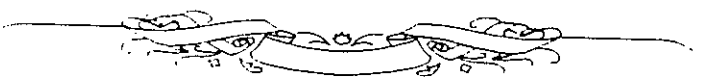




INTRODUCTION AND AIM OF THE WORK



INTRODUCTION AND AIM OF THE WORK

Nowadays, diving is an expanding field, not only for military operations, but also in commercial and scientific tasks, oceanographic studies, marine biological research activities and a wide spread of sports.

Exposure to oxygen greater than one atmosphere (ATA) i.e. (Hyperbaric Oxygen) is mandatory in closed circuit military divers (Frogmen) [Davis 1977], in decompression elimination of inert gas and in treatment of ischemic organs [Davis 1988].

The expanded use of hyperbaric oxygen therapy to decrease the duration and increase the effectiveness of decompression procedures in military and commercial diving purposes, has exposed large number of normal men to increased oxygen pressures [Lambertsen 1967].

Paradoxically, the same gas that is required to sustain life in cases of severe hypoxemia, has toxic properties that affect all living cells at sufficiently high pressure and duration of exposure [Fridovich 1978].

Two possible mechanisms underlying oxygen-induced pulmonary disease. The first is that high inspired oxygen fraction induces formation and release of free toxic oxygen radicals. That result in protein degradation, lipid peroxidation, destruction of cell membranes and inactivate intracellular enzymes [Fisher et al.,1979].

Another theory is the cellular mechanism, based on the fact that stimulation of alveolomacrophages (AM) with hyperoxic exposure,

produces neutrophil (PMN) chemotactic and activating factor. This leads to PMN attraction and sequestration in the pulmonary microcirculation with release of toxic oxygen radicals and proteolytic enzymes [Cross 1978]. Also Davis et al., (1983) have demonstrated that alveolar macrophages can release fibronectin and growth factor both are chemoattractant to fibroblastic accumulation, proliferation and lung fibrosis [Bitterman et al., 1982; Rennard 1982].

Studies of bronchoalveolar lavage from a group of subjects exposed to toxic dose of oxygen [Lee et al.,1981; Mcguire et al., 1982], have shown the presence of free proteolytic activity as neutrophil elastase in alveolar fluid, while those without free proteolytic activity had excess active alpha-1-protease inhibitor in the lavage; which is considered as principal inhibitor of neutrophil elastase in plasma or tissue.

The free protease; induced by hyperoxia, could have a number of damaging effects on the lung, including injury to endothelial cells and type I epithelial cells resulting in alveolo-capillary leak, as evidenced by a significant increase in lavage fluid albumin [Katzenstein et al., 1976].

In addition, prolonged exposure to hyperoxia is associated with alveolar fibrosis, probably because hyperoxia; through stimulation of alveolar macrophage, increases the number of fibroblasts in the alveolar walls [Bitterman et al.,1982].

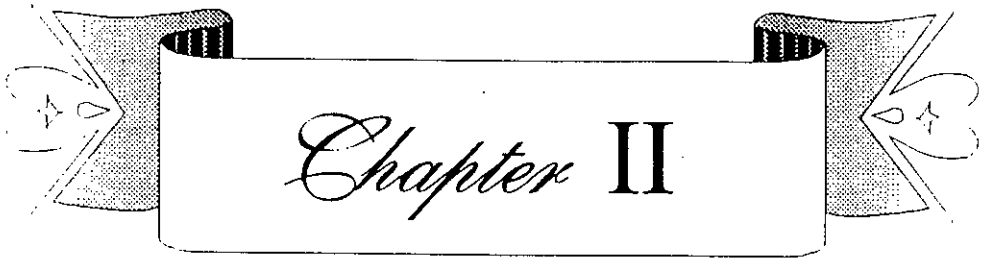
Clark et al [1987, 1988a, 1988b], have shown that pulmonary mechanical functions is impaired earlier and more severely than gas exchange function.

The present study was designed for the early detection (within 2 hours of exposure), of the biochemical composition and inflammatory cell changes in the lower respiratory tract and alveolar structure, that may precede the permanent derangement of lung parenchyma associated with hyperoxic injury.

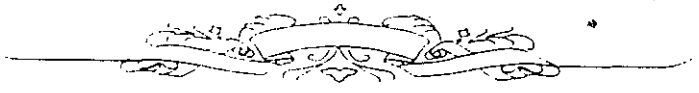
To accomplish this, bronchoalveolar lavage was used to recover different cells and the epithelial lining of the lower respiratory tract in divers exposed to hyperbaric oxygen (100% oxygen) at 3 ATA for two hours.

AIM OF THE WORK

Our aim of the work was to study the possible reversible changes in the pulmonary function as well as in the biochemical and cellular profile of bronchoalveolar lavage in divers breathing 100% oxygen at 3 ATA for 2 hours.



Chapter II



REVIEW OF LITERATURE