ASSESSMENT OF THE SIZE OF LEFT VENTRICLE IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

THESIS

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BY
SAHAR AHMED AHMED ABOU EL-EZZ
(M.B., B.Ch. & M.Sc.)

Under The Supervision of

Prof. Dr. GAMALAT MEGAHED
Professor of Internal Medicine
Faculty of Medicine, Cairo University

Prof. Dr. ERNEST BEBAWY
Professor of Internal Medicine
Faculty of Medicine, Cairo University

Prof. Dr. AHMED HAMZA
Professor of Internal Medicine
Faculty of Medicine, Cairo University

FACULTY OF MEDICINE
CAIRO UNIVERSITY

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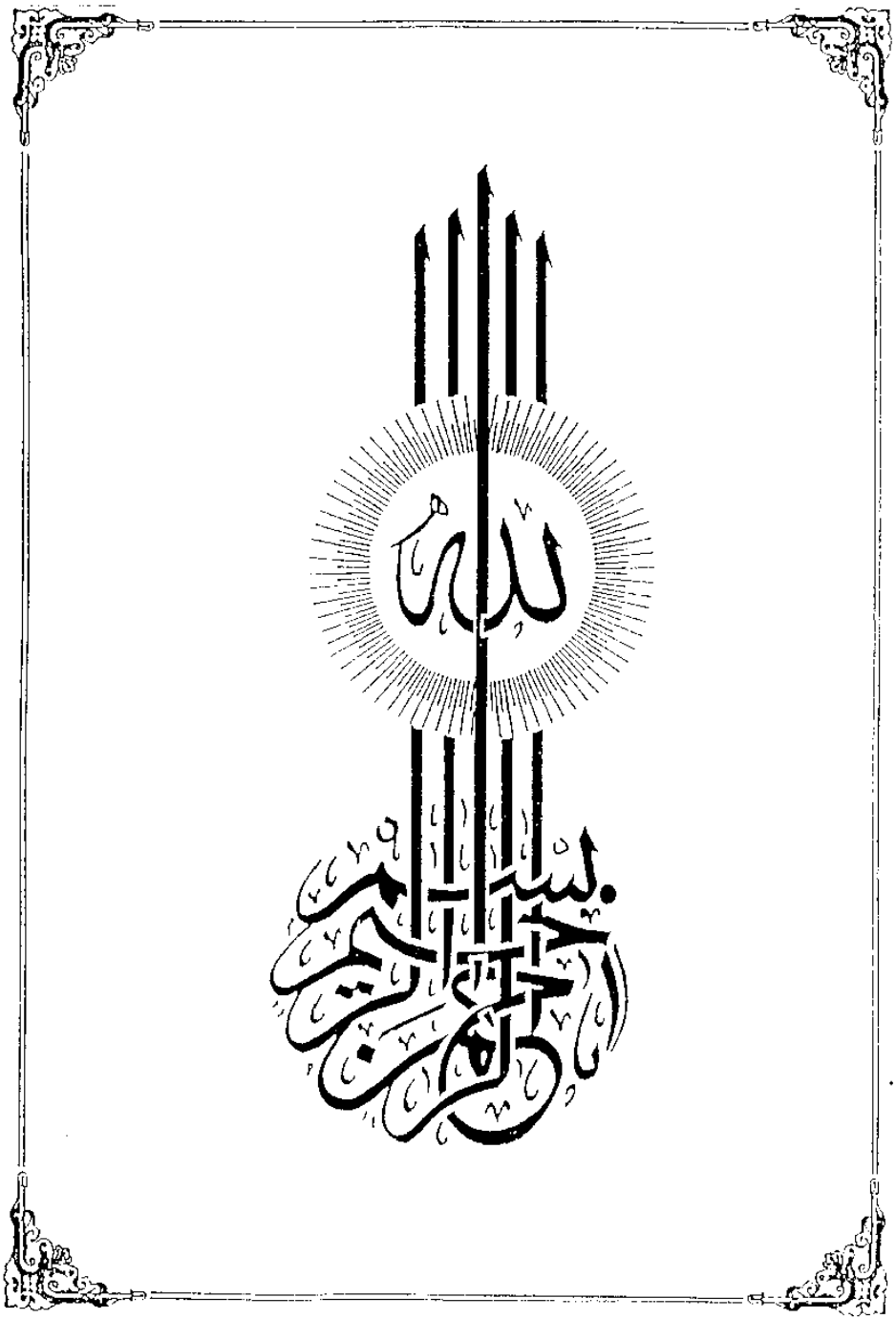

TO MY PARENTS

MY Husband

AND Maï

MY Daughter

C O N T E N T S

LIST OF ABBREVIATIONS

ABGs	: Arterial blood gases.
AD	: Arterial deficiency.
$Alpha_1$ PI	: $Alpha_1$ protease inhibitor.
CAD	: Coronary artery disease.
CAL	: Chronic airflow limitation.
CAO	: Chronic airflow obstruction.
CO	: Cardiac output.
COLD	: Chronic obstructive lung diseases.
COPD	: Chronic obstructive pulmonary diseases.
dec.slope	: Deceleration slope.
DM	: Diabetes mellitus.
EF	: Ejection fraction.
ESS	: End systolic stress.
FEV_1	: Forced expiratory volume in the first second.
FRC	: Functional residual capacity.
FS	: Fractional shortening.
FVC	: Forced vital capacity.
HPV	: Hypoxic pulmonary vasoconstriction.
HF	: Heart failure.
IHD	: Ischemic heart disease.
IM	: Increased markings.
IVS	: Interventricular septum.
LA	: Left atrium.
LV	: Left ventricle.

$LVEDD_p$: Left ventricular end-diastolic dimension in parasternal view.

$LVEDD_s$: Left ventricular end-diastolic dimension in subcostal view.

LVEDP : Left ventricular end-diastolic pressure.

LVEDV : Left ventricular end-diastolic volume.

LVEF : Left ventricular ejection fraction.

$LVESD_p$: Left ventricular end-systolic dimension in parasternal view.

$LVESD_s$: Left ventricular end-systolic dimension in subcostal view.

LVESS : Left ventricular end-systolic stress.

LVESV : Left ventricular end-systolic volume.

LVH : Left ventricular hypertrophy.

MI : Mitral incompetence.

$P(A-a)O_2$: Alveo-arterial oxygen difference.

$PaCO_2$: Partial pressure of carbon-dioxide in the arterial blood.

PaO_2 : Partial pressure of oxygen in the arterial blood.

PAP : Pulmonary artery pressure.

PmCWP : Pulmonary capillary wedge pressure.

PEFR : Peak expiratory flow rates.

PFR : Peak flow rates.

PH : Pulmonary hypertension.

PND : Paroxysmal nocturnal dyspnea.

PVR : Pulmonary vascular resistance.

PWT : Posterior wall thickness.

RV : Right ventricle.

RV : Residual volume.

RVD : Right ventricular dimensions.

RVEDP : Right ventricular end-diastolic pressure.

RVEF : Right ventricular ejection fraction.

RVH : Right ventricular hypertrophy.

RVWT : Right ventricular wall thickness.

SPAP : Systolic pulmonary artery pressure.

STIs : Systolic time intervals.

SWT : Septal wall thickness.

TLC : Total lung capacity.

TPV : Time to peak velocity.

VA/Q : Ventilation perfusion ratio.

Vfc : Mean velocity of circumferential fibers shortening

VD-VT : Dead space/tidal volume ratio.

VE : Minute ventilation.

wTd : Wall thickness in diastole.

wTs : Wall thickness in systole.

2D echocardiography: Two dimensional echocardiography.

INTRODUCTION AND AIM OF THE WORK

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Cor pulmonale is an important cause of mortality in patients with chronic obstructive pulmonary disease (COPD).

Cor pulmonale is by definition pulmonary hypertension, right ventricular hypertrophy with or without failure, caused by primary chest disease. The clinical diagnosis of cor-pulmonale in patient with COPD can be difficult (Robin and Gaudio, 1970).

Physical findings of heart disease such as pulmonary rales or abnormal cardiac impulse may be obscured by lung hyperinflation, likewise, pulmonary hyperinflation, bullae and vertical position of the heart may obscure the radiographic evidence of right or left ventricular enlargement and pulmonary hypertension.

Although cor pulmonale essentially involves the right heart, left ventricular function has been questioned in patients with COPD, because autopsy studies done in the past have exhibited the presence of left ventricular hypertrophy in cor pulmonale (Murphy et al., 1977).

Studies performed in individuals with congestive heart failure, caused by COPD have shown that as many as one third of those patients who develop signs and symptoms of cardiac failure, have normal systolic function. The cause of congestive heart failure in these individuals is abnormal diastolic function (Soufer et al., 1985).

Also the impairment of left ventricular function in COPD may be caused by compromised stroke output of right ventricle and hence may indicate a decrease in flow of left heart. Additionally changes in left ventricular geometry induced by the development of right ventricular dilatation and hypertrophy have been shown to have effect on left ventricular filling patterns.

More recently, the importance of ventricular interdependence in circulatory adjustment has been emphasized (Bemis et al., 1974).

Left ventricular catheterization and cine ventriculographic studies may provide a precise answer to the question of whether a particular patient has an element of left ventricular dysfunction contributing to his dyspnea; however, these procedures are not without risk and are difficult to justify in either clinical or ventricular contexts.

The advent of balloon-tipped flow-directed catheters has greatly facilitated the acquisition of right cardiac and pulmonary arterial pressures, and in most circumstances the pulmonary arterial wedge pressure accurately reflects the left ventricular end-diastolic pressure.

Right cardiac catheterization at bedside has been extremely useful for identifying dyspneic patients who have left ventricular failure in addition to pulmonary disease. Recently, echocardiography (De Maria et al., 1991) and radionuclide angiography (Bonow, 1991) have been established as valuable non-invasive techniques for evaluating left ventricular performance in patients without coexistent pulmonary disease; however, these have been applied rarely to patients with COPD, in whom technical problems might be anticipated to interfere with both performance and analysis.

Therefore the aim of the presented study is to:

1) Assess left ventricular size in COPD.
2) Assess left ventricular function (systolic and diastolic).
3) Detect pulmonary hypertension and its relation to left ventricular function in COPD.
4) To determine whether Eco Doppler techniques for left ventricular function could be successfully applied.

REVIEW OF LITERATURE

CHRONIC OBSTRUCTIVE PULMONARY DISEASES (COPD)

Definition:

It is a sweeping term that covers a spectrum of disorders. The common denominator of this spectrum is increased resistance to expiration (**Fishman, 1988**). It includes chronic obstructive bronchitis, emphysema, bronchiolitis, and asthma. These conditions are very different, even the way they produce airway obstruction is different, so the umbrella terms: chronic obstructive lung disease and chronic obstructive airway disease, have no place in medical use. It is commonly used to denote a combination of chronic bronchitis and emphysema. The clinical hallmark of this group are chronic cough, expectoration, chest wheezing and dyspnea during a mild exertion if not at rest. Characteristically, airflow is reduced and bronchodilators do nothing or little to relieve the obstruction.

Reduced airflow is detected on physical examination by diminution in the sounds of air movement, especially on expiration throughout both lungs. Reduced airflow may be measured by standard ventilation tests (maximum voluntary ventilation, forced expiratory volume, maximum mid. expiratory

flow) or by measuring increased airway resistance (in the body box) (Robin and Gaudio,1970).

Although the diagnosis of chronic bronchitis is made on the basis of clinical history and emphysema on the basis of morphology, COPD is characterized in functional terms. COPD consists of persistent, largely irreversible airway obstruction in which the underlying pathophysiology is not precisely known (Fletcher et al., 1984).

To retain any usefulness, the term should exclude conditions characterized by persistent obstruction in which the mechanism of obstruction is known, such as asthma, bronchiolitis, cystic fibrosis and alpha, protease inhibitor (alpha, PI) deficiency (Fraser et al., 1990). Synonyms for COPD include chronic obstructive lung disease (COLD), chronic airflow obstruction (CAO) and chronic airflow limitation (CAL). (COLD and CAO) are acceptable but (CAL) is misleading because it suggests that airflow limitation is unique to this entity. In fact every one has maximal expiratory airflow limitation, it is the severity of limitation that is abnormal in COPD (Fraser et al., 1990).

Thurlbeck et al. (1970) suggested that the term (CAO) is preferable to designate this group of diseases since obstruction may not be within the airways themselves, but may