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Original Article

Physiology Section

Should the Functional Residual Capacity be Ignored?

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ABSTRACT

Aim and Objectives: The functional residual capacity was given the least importance than the other lung volume parameters. Studies have revealed the restrictive pattern of lung disease in patients with liver cirrhosis. We aimed to analyze the importance of the functional residual capacity and other lung volumes of cirrhotic patients.

Subjects and Methods: Forty (40) patients with cirrhosis (Child's-B) were enrolled in this study. The vital capacity was measured by an instrument called V02 Max 22. The other lung volumes which were measured were derived parameters. The functional residual capacity was measured by the nitrogen wash-out method.

Results: The measured value of the functional residual capacity was below normal as compared to the reference value. The total lung capacity and the vital capacity were positively correlated with the functional residual capacity. The residual volume was found to be increased in twelve out of forty cirrhotic patients.

Conclusion: The functional residual capacity can be determined by the compliance of the lung and the chest wall. The patients with a reduced functional residual capacity may be suffering from dyspnoea, probably due to the restrictive pattern of the lung disease. Hence, the reduced lung volumes of the subjects may be due to the abnormalities in the mechanics of ventilation.

Key Words: Functional residual capacity, Total lung capacity, Vital capacity, Compliance

INTRODUCTION

Normally, the lung volumes are maintained by the elevation and depression of the thoracic cavity. Usually, in practice, the clinicians concentrate on the lung volume parameters like the Total Lung Capacity (TLC), the Residual Volume (RV) and the RV/TLC ratio, to diagnose the restrictive pattern of lung diseases. Though the Functional Residual Capacity (FRC) is given the least importance, it maintains the lung volumes at the end of the expiration, which should be adequate for the subsequent respiration. FRC is physiologically important because it keeps the small airways open [1] and prevents the complete emptying of the lungs during each respiratory cycle. If there is no FRC, the alveolar PO_2 and PCO_2 will vary widely during breathing and will interfere with the diffusion of the respiratory gases. So, FRC acts as a buffer against such fluctuations [2].

At the Resting Respiratory Level (FRC), the opposing forces like the elastic recoil of the lungs and the chest wall are equal and they balance each other. The volume which is present in the lung at this point is the relaxation volume, which is equal to the FRC. To make clinical determinations of pulmonary compliance, one must be able to measure the changes in the pressure and the volume. The volume [3] changes can be measured with a spirometer, but measuring the pressure changes is more difficult because the changes in the transpulmonary pressure gradient must be taken into account. Hence, the FRC can be considered to determine the compliance [4].

The lung compliance is reduced in pulmonary fibrosis, atelectasis, oedema and ascites [5]. It is well known that cirrhosis alone, in the absence of other disease processes, affects the respiratory functions in humans. The most Persistent Pulmonary Function Test (PFT) abnormalities in the cirrhotic patients are reduced the Total Lung Capacity (TLC), diffusion and hypoxaemia, due to the alterations in the chest wall mechanics, which also lead to a decreased total respiratory compliance [6].

Among the lung volume parameters which are assessed to know the ability of the lung to distend, the FRC is measured. The total respiratory resistance and work are increased in cirrhotic patients. These PFT abnormalities are more pronounced in cirrhotic patients and in those with the Hepato Pulmonary Syndrome (HPS) [7]. So, we studied the lung volumes and the FRC of the cirrhotic patients who did not have any evidence of other lung diseases. Furthermore, we have described the compliance of the cirrhotic patients, based on their lung FRC and have discussed the potential liver function abnormalities which are responsible for the difference.

MATERIALS AND METHODS

Study population

During a 12- month period, forty (40) male cirrhotic patients who were aged 42-62 years, who were referred to the pulmonary function test laboratory at the Tuberculosis Research Centre, were screened for the presence of pulmonary function abnormalities. A written informed consent was obtained from all the cirrhotic patients. Depending upon the severity of the diseased subjects' clinical records, the liver function tests and other relevant data were reviewed to identify the Child's-B type of cirrhotic patients with ascites (Grade II). The patients with Child's-A and Child's-C type of cirrhosis were excluded. This study was approved by the institutional review board.

Measurements

The body height (cm) and weight (kg) were measured, with the subjects wearing indoor clothing. The lung volumes were determined by using an instrument which is known as VO_2 Max 22. The FRC was measured by the nitrogen washout technique and the Residual Volume (RV) was obtained as the FRC minus the Expiratory Reserve Volume (ERV). The TLC was calculated as RV plus VC. The recommendations for the standardized procedures for various lung function test measurements were followed [8,9] [Table/Fig-1].

STATISTICAL ANALYSIS

In our study, the quantitative variables are represented as means and standard deviations. The Student's-t test was used to describe the relationship between the predicted and the measured values of the lung volumes. P values which were < 0.05 were considered as significant values and those which were <0.001were considered as highly significant values.

RESULTS

In [Table/Fig-2], the measured value of VC (2.79±0.59 L) is significantly reduced as compared to the predicted values (3.19±0.36L). The TLC measured value (4.41±0.72L) is significantly lower than the predicted values (5.18±0.74l). In contrast to the previous two values, the RV measured value was found to be more (1.67±0.39L) than the predicted value (1.64±0.25L) but it was not statistically significant. The FRC measured value was significantly less (2.42±0.54L) than the predicted values (2.96±0.44L) and it was also normal. The ERV significantly showed a difference between the measured and the predicted values (0.71±0.24L, 1.37±0.14L) respectively. As compared to the normal RV/TLC ratio, our subjects showed >10% of the increased values, which were statistically significant.

[Table/Fig-3] indicates the size of the lung with the parameters like VC, TLC, and FRC. The predicted values are in %. The 5th percentile lung volumes were considered for analyzing the results. The small lung size was analyzed in >50% of the patients who had reduced lung volumes (<79%). The patients with enlarged lung sizes were identified by the patients with large lung volumes (>121%). The % predicted values were taken to compare the severity of the disease in cirrhotic patients.

Age (yrs)	42.13±10.39
Sex	Male (n-40)
Height (cm)	161.90±5.4
Weight (kg)	54.62±11.35
Smoking	NO
Classification	Child's-B

[Table/Fig-1]: Characteristics of cirrhotic patients

Parameters	Reference	Measured	P-value
VC (L)	3.19±0.36	2.79±0.59	<0.05
TLC(L)	5.18±0.74	4.41±0.72	<0.001
RV(L)	1.64±0.25	1.67±0.39	NS
FRC(L)	2.96±0.44	2.42±0.54	<0.001
ERV(L)	1.37±0.14	0.71±0.24	<0.001
RV/TLC%	31.51±1.84	38.43 ±8.68	<0.001

[Table/Fig-2]: Table-II Static lung volumes in cirrhotic patients

Results are expressed as mean ± SD. Reference and measured values of cirrhotic patients (n=40) are in liters VC-Vital capacity, TLC-Total lung capacity, RV-residual volume, FRC-Functional Residual capacity, ERV-Expiratory reserve volume.

Parameters	(< 79%)	(80- 120%)	(>121%)
VC (L)	14	26	0
TLC(L)	17	22	1
RV(L)	3	25	12
FRC (L)	24	14	2
ERV(L)	39	1	0

[Table/Fig-3]: Distributions of patients with specific lung volume abnormalities Total Number of Patients – 40

DISCUSSION

The reduced VC and TLC in our study proved the same restrictive pattern of the lung disease 7 which was shown in many studies. Previous studies had evidenced that this restriction may be due to the reduced compliance of the lung, which in turn might have caused resistance for the expansion of the chest wall. The reduced FRC of the cirrhotic patients may at least in part, be a consequence of the elasticity of the lung.

A further evidence which supports this hypothesis is that FRC is a point at which the inward elastic recoil of the lung normally opposes the outward elastic recoil of the chest wall and vice versa. This integrity of the lung and the chest wall system may be disturbed in cirrhosis due to the alteration of the vertical diameter of the chest wall which is caused by ascites. Hence, to know the elastic tissue abnormality among the lung volume parameters (VC,TLC), the FRC should be considered.

Usually, the volume of the gas which is left in the lung is determined by a) The increased inward elastic recoil of the lung and the decreased outward elastic recoil of the chest wall and b) the positivity of the recoil pressure of the chest wall at high lung volumes. At FRCs which are below 70% of the normal, the chest wall elastic recoil is outward; at FRCs which are above 70% of the normal, the recoil is inward. In our study, the increased FRC of our patients tended to hold the alveoli open and their reduced elasticity kept the alveoli expanded.

Therefore, at high lung volumes (FRC), (i.e.) above 70%, as has been discussed earlier, the patients may feel dyspnoeic because both the lung and the chest wall elastic recoil are inward.

The pleural pressure is always negative because it is essential to keep the lung partially inflated all the time. The further inflation of the partially inflated lungs by a normal FRC is easier than the fresh inflation of collapsed lungs. Hence, having partially inflated lungs saves considerable energy during breathing. In patients with lung volumes which are below FRC, the work which is done by them to expand the lung volume increases, because the work which is done during the outward recoil of the chest wall is greater than that which is done during the inward recoil of the lungs.

The distribution of the ventilation is determined almost entirely by the gravity-dependent pleural pressure gradient down the lungs, which acts in combination with the nonlinear volume elasticity of the lungs [10].

In patients with liver cirrhosis, the distribution of a breath which was taken from the Resting Lung Volume (FRC) was not preferential to the lower lung zones and so, there was also a marked

reduction in the ventilation of the lower zones. This was associated with a somewhat reduced lower zones perfusion in some patients. Compression of the lung tissue by ascetic fluid, as in patients with Child's-B, may be invoked to explain these abnormalities [11].

An increase in the closing volume can be caused by an increase in the magnitude of the pleural pressure gradient down the lungs, by the loss of the elastic recoil of the lungs, particularly in the dependent lung zones, and/or by a decreased resistance to the collapse of the dependent small airways [12]. This can be caused by several factors (a) loss of surfactants in the small airways, (b) changes in the bronchomotor tone, (c) mechanical compression of the small airways from the distended vessels, and (d) interstitial pulmonary oedema [12,13].

Hughes and Rosenzweig [13] showed that the interstitial pulmonary oedema resulted in an increase in the trapped gas volume, particularly in the dependent lung zones. The data from their study appears to be helpful for explaining the RV of our present results. They measured the static deflation volume-pressure relationship of the lung, both under control conditions and after the development of interstitial lung oedema in liver cirrhosis. The oedema was evidenced by the increased lung weight and the presence of peribronchial and perivascular cuffs on histological examination. In the oedematous lung, there was a marked increase in the trapped gas volume, i.e., the volume of the gas which was left in the lungs at a negative (- 6 cm H20) transpulmonary pressure, was increased. This marked increase in the trapped gas volume was due to the closure of the small airways in the oedematous lungs at higher lung volumes.

Whatever may be the mechanisms which cause the FRC, VC and the TLC abnormalities in hepatic cirrhosis, our results may indicate a marked ventilation perfusion impairment on a regional basis. Since the lower lung zones in cirrhotic patients presumably contain both open and trapped units during quiet breathing, the V/Q abnormality must be much greater. In other words, some airways in the lower lung zones may attain the opening pressure at the beginning of the inspiration, while others may only open towards the end of the inspiration, i.e., when the pleural pressures are the most negative [12]. Similarly, some airways may reach the closing pressure at the beginning of the expiration, while others will close only towards the end of the expiration, i.e., when the pleural pressures are the least negative. In addition, some units may remain closed throughout the breathing cycle.

We conclude that the reduced VC, TLC and FRC values proved the restrictive pattern of the lung disease in patients with hepatic cirrhosis, due to ascites. The restriction for the expansion of the lungs is due to the abnormalities in the respiratory muscles and the rib cage and also due to the elasticity of the lung tissues. VC and TLC provide the strength of the respiratory muscles but FRC (compliance) alone determines the amount of air which enters into and leaves the lungs. Hence, cirrhotic patients suffer from breathlessness which alters the ventilation- perfusion mismatch, thus leading to an impairment of the gas exchange within the lungs. In the present study, we had no direct evidence of the closing volume and hence , our tentative explanation of the results must necessarily remain speculative.

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