Pulmonary Function in Cardiac Disease

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Introduction

It has been well established that pulmonary function can be altered by cardiac disease. Conversely, pulmonary pathology may result in abnormal cardiac function and anatomy. Pulmonary function testing offers a simple method of quantitating and following these changes. The purpose of this paper is to illustrate the value of such measurements in cardiovascular disease. This is particularly useful in evaluating the effects of various modalities of therapy.

FIGURE 1: Ventilographic response on the Vitalor of a patient with cor pulmonale and "obstructive" emphysema to digitalization before and after nebulized bronchodilator therapy. (The vertical lines represent volume only—explanation has been previously described.†)

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Procedure

The parameters selected for study on patients in this series were: a total and timed vital expiratory capacity of the lung and the maximal expiratory flow rate, as obtained by the bellows vital capacity apparatus (the Vitalor†). Arterial oxygen and carbon dioxide studies also were carried out.

The patients studied were adult men in frank congestive failure. They were studied on the basis of either right or left heart failure. All testing was done in the standing position, when possible.

These patients were tested before and after various forms of treatment. The drugs used were: digitalis, aminophyllin, hydrochlorothiazide, isoproterenol, and prednisone.

In addition, circulation times, venous pressures, electrocardiograms and posteroanterior, right anterior oblique and left anterior oblique chest x-ray films were used in evaluating the clinical status of the patient. All of this information was correlated with the preliminary function tests.

Results and Discussion: Right Ventricular Failure

Eleven patients in right heart failure were studied by means of the Vitalor. Failure in these patients was caused mainly by cor pulmonale resulting from obstructive emphysema, inactive tuberculosis, pulmonary fibrosis, chronic empyema, silicosis, and Boeck’s sarcoidosis. Ventilatory studies in cor pulmonale were not as dramatic as in failure. This finding is to be expected, since the edema of right ventricular failure usually is found in the abdominal viscera and lower extremities, rather than in the

†The apparatus used in this paper was graciously furnished by the McKesson Appliance Company, Toledo, Ohio.
lungs. This may explain the symptomatic improvement seen when patients with chronic left ventricular failure develop right ventricular decompensation. It should be pointed out that the interpretation of abnormal results obtained in pulmonary function testing is made more difficult by the presence of coexistent lung disease. Therefore, this testing is of limited diagnostic value here. However, the patient with "obstructive" or "restrictive" lung disease who develops right ventricular failure usually shows a further decrease in total vital capacity. This may be due to progressive bronchopulmonary disease or the development of superimposed left ventricular decompensation. Figure 1 illustrates the response before and following the use of digitalis and isoproterenol where the total vital capacity increased as usually occurs after the cardiac therapy in this type of case with the maximum expiratory flow rate and the timed vital capacity remaining low.

Figure 2 illustrates pulmonary function studies done on a patient with severe cor pulmonale after irretreactable failure. Following prednisone therapy, there was a marked diuresis resulting in a dramatic improvement in the clinical cardiac status and in the pulmonary status as determined by the three ventilatory parameters measure by the Vitalor. This is probably because the steroid therapy reduced some of the bronchial spasm, bronchial edema, and bronchial inflammation (in addition to possible direct cardiovascular and renal action) thus relieving part of the right ventricular strain and hypoxia, increasing more adequate air exchange, and thereby also decreasing the work of breathing.

**FIGURE 3:** Serial ventilographic studies of a patient with left ventricular failure under therapy.
We also have found that blood arterial gas studies offer a better correlation with right ventricular failure than do ventilatory studies, and also are more useful in evaluating the response to therapy.

**Left Ventricular Failure**

The lungs, by virtue of their position in the circulation, have long been recognized as a sensitive indicator of inadequate performance of the left ventricle.

The 18 patients in left ventricular failure tested in this study demonstrated a marked reduction in the total vital capacity and to a lesser (often minimal) extent of the timed (forced) vital capacity and the maximum expiratory flow rate (Fig. 3). Why this occurs is readily understood.

Von Basch in 1891 described the rigidity and stiffness of the lungs in heart failure ("Lungenstarre") with the subsequent observation revealing the dyspnea in heart disease, particularly in left ventricular failure and in decompensated mitral stenosis, is as closely related to lung compliance as any other single observation. Since compliance (i.e., ratio of change in lung volume to change in transpulmonary pressure: a low value signifying increased lung rigidity) is shown to vary directly with the total vital capacity, it is apparent that reduction in compliance, diminished vital capacity, and dyspnea in congestive failure are closely related. Figure 4 illustrates the direct linear correlation between lung compliance and the total vital capacity.

![Graph](image-url)
FIGURE 5: Serial ventilographic changes obtained prior to digitalization for left ventricular failure and then five days later.

FIGURE 6: Response of patient to hydrochlorothiazide therapy before and after nebulized bronchodilator administration.
Thus, the total vital capacity may be inversely proportional to the degree of pulmonary congestion. Because of this, pulmonary function testing offers the practitioner a simple and extremely useful tool for the evaluation of the response of left ventricular failure to the various modes of therapy. This is demonstrated in Figure 5 with an improvement in the three parameters measured. This was also true following the use of nebulized isoproterenol as demonstrated in Figures 1 and 6. The effect of an oral diuretic, hydrochlorothiazide, also is demonstrated in Figure 6 in an individual with left ventricular failure. This figure also illustrated the importance of bronchospasm reversible by nebulized bronchodilator therapy which may be present in certain cases of left ventricular failure associated with bronchial edema due to infection or with the fluid retention or bronchospasm associated with the pulmonary edema.

In addition, pulmonary function is a valuable tool differentiating the cardiac patient with a superimposed cardiac neurosis with hyperventilation syndrome from one in left ventricular failure (Fig. 7).

In the dyspneic patient with thyrotoxicosis, anemia, obesity, or neurosis, normal pulmonary function testing may help eliminate congestive failure as a cause. Normal function in a resting patient, however, does not necessarily mean that failure is not the cause of the symptoms which occur only on effort.

**SUMMARY**

1. Measurements of the (forced) total and timed expiratory vital capacities and maximum expiratory flow rates have been made in 11 patients with obvious right ventricular failure and in 18 with left ventricular failure.
2. Pulmonary ventilation studies were of limited value in right ventricular failure except in following the response to therapy. Arterial gas studies were more informative here.

3. However, ventilation tests were a sensitive indicator of incipient or frank left ventricular failure and its response to therapy.

4. Normal parameters obtained on pulmonary ventilation testing as described in this paper were helpful in differentiating acute left ventricular failure from other causes of dyspnea.

RESUMEN

1. La medida de la expiración forzada total y por segundos de la capacidad vital así como la máxima tasa del flujo expiatorio se llevaron a cabo en 11 enfermos que tenían evidente insuficiencia ventricular derecha y en 18 con insuficiencia ventricular izquierda.

2. Los estudios de la ventilación pulmonar fueron de valor limitado en la insuficiencia ventricular derecha exceptuando después de la respuesta al tratamiento. Los estudios del gas arterial fueron más informativos en ese caso.

3. Sin embargo las pruebas ventilatorias fueron de valor limitado en la insuficiencia incipiente o franca ventricular izquierda y se reespecia al tratamiento.

4. Los parámetros normales obtenidos en las pruebas ventilatorias según se describen en este trabajo fueron útiles para diferenciar la insuficiencia ventricular izquierda aguda de otras causas de disnea.

RESUME

1. Des mesures de la capacité totale et de la capacité vitale ainsi que de la ventilation maximale ont été faites chez 11 malades présentant une atteinte ventriculaire droite évidente, et chez 18 malades avec atteinte ventriculaire gauche.

2. Les études de la ventilation pulmonaire furent de valeur limitée dans l'atteinte ventriculaire droite, sauf pour suivre la réponse à la thérapeutique. L'étude des gaz artériels donna plus d'informations dans ce cas.

3. Cependant, les tests de ventilation donnant des indications sensibles de l'atteinte naissante ou franche du ventricule gauche et de sa réponse au traitement.

4. Des paramètres normaux obtenus par les tests de ventilation pulmonaire, comme il est décrit dans cet article, furent utilisés pour différencier l'atteinte aigu du ventricule gauche des autres causes de dyspnée.

ZUSAMMENFASSUNG


3. Jedoch waren Ventilationsprüfungen ein empfindlicher Indikator für eine beginnende oder offensichtliche Linksinsuffizienz und ihre Reaktion auf die Behandlung.


REFERENCES


