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# THE FORCED EXPIRATORY VOLUME AFTER EXERCISE, FORCED INSPIRATION, AND THE VALSALVA AND MÜLLER MANŒUVRES

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During exercise a patient suffering obstructive airway disease may ventilate his lungs at a rate greater than his maximum voluntary ventilation volume per minute (M.V.V.) measured at rest. Though this paradox has been known for a number of years (Filley, 1953), it has received but brief comment (Hugh-Jones and Lambert, 1952; Comroe, Forster, Dubois, Briscoe, and Carlsen, 1955; Gandevia and Hugh-Jones, 1957). Lewis and Morton (1954) reported an increase in the M.V.V. after exercise in healthy people.

If exercise increases the M.V.V. then it should increase the one-second forced expiratory volume ($F.E.V._1$) since these are closely related (Gaensler, 1951). We have been able to show that this increase occurs in patients with obstructive airway disease but not in healthy people (Experiment 1). An explanation was sought.

Beyond a certain point increasing expiratory effort is unlikely to increase the $F.E.V._1$ of patients with obstructive airway disease (Fry, Ebert, Stead, and Brown, 1954). The influence of increasing inspiratory effort was therefore investigated (Experiment 2).

During exercise the volume of blood in the lung capillaries of healthy people probably increases (Filley, MacIntosh, and Wright, 1954). Exercise may also increase the volume of blood in the lung capillaries of patients with obstructive airway disease. This must alter the mechanical properties of the lung. The effect of changes in lung blood volume on the $F.E.V._1$ was therefore investigated (Experiment 3).

We used a spirometer similar to that described by Bernstein, D'Silva, and Mendel (1952) recording on a fast-moving drum. When two similar spirograms were obtained from the patient (usually his first two attempts), the better was recorded. Patients were chosen at convenience, and all those tested are included in the report. All were more or less breathless on effort. The presence of obstructive airway disease (chronic bronchitis and emphysema or asthma) was confirmed from the forced expiratory ratio (F.E.R.: ratio of $F.E.V._1$ to forced vital capacity) when this was less than 60% (Capel and Smart, 1958).

## EXPERIMENT 1

The effect of exercise on the $F.E.V._1$ was studied in 48 patients, 30 with obstructive airway disease (mean F.E.R. 37%, range 23% to 54%) and 15 with heart disease (not in failure), or lung disease without airway obstruction (mean F.E.R. 67%), and in six healthy subjects (mean F.E.R. 78%).

The $F.E.V._1$ was measured at rest, immediately following exercise, and after five minutes' recovery. The test was then repeated after inhalation of adrenaline spray. The subjects mounted a 10-in. step 15 times a minute for five minutes or for as long as they were able to do so.

In all patients with obstructive airway disease the $F.E.V._1$ was increased immediately after exercise. The mean increase was 24% (range +3% to +52%) of the resting value. After five minutes' recovery the $F.E.V._1$ returned to a mean 3% above the resting value (Fig. 1).

After adrenaline inhalation the $F.E.V._1$ of the patients with obstructive airway disease rose to a mean of 21% (range −15% to +76%) above the $F.E.V._1$ at rest. The patients then exercised again, and once more the $F.E.V._1$ was increased immediately after exercise. The mean rise was 18% (range −8% to +54%) above the resting value after adrenaline inhalation.

Neither the patients suffering heart disease or pulmonary fibrosis without obstructive airway disease nor the healthy subjects showed much change in $F.E.V._1$ after exercise. After adrenaline spray inhalation the patients without obstructive airway disease showed little change in $F.E.V._1$.

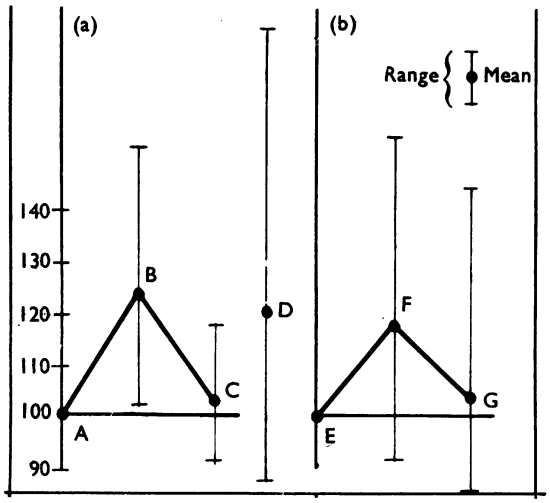


FIG. 1.—(*a*) Means of values of F.E.V.$_1$ in 30 patients with obstructive airway disease, expressed as percentages of the values at rest (A). B—after exercise. C—after five minutes' recovery from exercise. D—after adrenaline spray inhalation. (*b*) Means of values of F.E.V.$_1$ in the same patients after inhalation of adrenaline, similarly expressed. E—at rest. F—after exercise. G—after five minutes' recovery from exercise.

Much more strenuous exercise (impossible for the patients with heart disease and pulmonary fibrosis) might have increased the F.E.V.$_1$ in the healthy subjects.

The forced vital capacity of the majority of patients and the healthy subjects was slightly reduced after exercise.

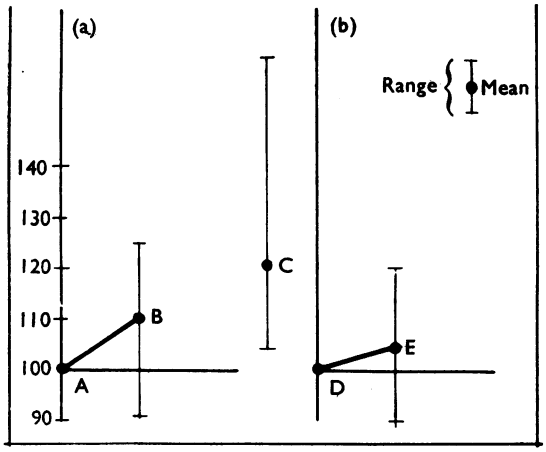


FIG. 2.—(*a*) Means of values of F.E.V.$_1$ in 31 patients with obstructive airway disease, expressed as percentages of the values at rest (A). B—after forced inspiration. C—after adrenaline spray inhalation. (*b*) Means of values of F.E.V.$_1$ in the same patients after inhalation of adrenaline, similarly expressed. D—at rest. E—after forced inspiration.

## EXPERIMENT 2

To permit study of the effect of variations in the speed of inspiration on the F.E.V.$_1$ 31 patients with obstructive airway disease (mean F.E.R. 43%, range 27% to 57%) and six healthy subjects recorded their F.E.V.$_1$ after a slow inspiration (as usual) and after a forced inspiration from the full expiratory position. The order was varied. The patients repeated both tests after adrenaline inhalation.

After forced inspiration the F.E.V.$_1$ of patients with obstructive airway disease rose to a mean value of 10.2% (range −9% to +25%) above the F.E.V.$_1$ measured after a slow inspiration. After adrenaline inhalation the mean rise was 3.8% (range −10% to +19%) above the slow inspiration F.E.V.$_1$ after adrenaline (Fig. 2). The normal subjects and the patients with heart disease showed little change.

## EXPERIMENT 3

To study the effect of a rise in lung blood volume the F.E.V.$_1$ was measured in 14 patients with obstructive airway disease (mean F.E.R. 40%, range 30% to 57%) before and after forced inspiratory efforts against the closed glottis with the chest in the mid-inspiratory position (Müller manœuvre) for four seconds. Full inspiration was then immediately completed and the forced expiratory volume recorded. The F.E.V.$_1$ rose a mean 7.1% (range −8% to +29%). There was little change in the forced vital capacity. After adrenaline inhalation and the Müller manœuvre the F.E.V.$_1$ showed a mean rise of 5.3% (range −2.7% to +15%) (Fig. 3). There was little change in the forced vital capacity. The Müller manœuvre caused little change in the F.E.V.$_1$ of healthy people.

Holding the breath alone at different levels of inspiration resulted in little change in the F.E.V.$_1$.

To study the effect of a fall in lung blood volume the F.E.V.$_1$ was measured in 10 patients with obstructive airway disease (mean F.E.R. 47%, range 29% to 57%) before and immediately after forced expiratory efforts against the closed glottis with the chest in the full inspiratory position (Valsalva manœuvre) for four seconds. The F.E.V.$_1$ fell a mean 8.4% (range −20% to +5%). The forced vital capacity changed little. After adrenaline inhalation the Valsalva manœuvre caused a mean fall in the F.E.V.$_1$ of 8.8% (range −20% to 0%) (Fig. 3). The mean forced vital capacity fell 9.4% following the Valsalva manœuvre after adrenaline. This was

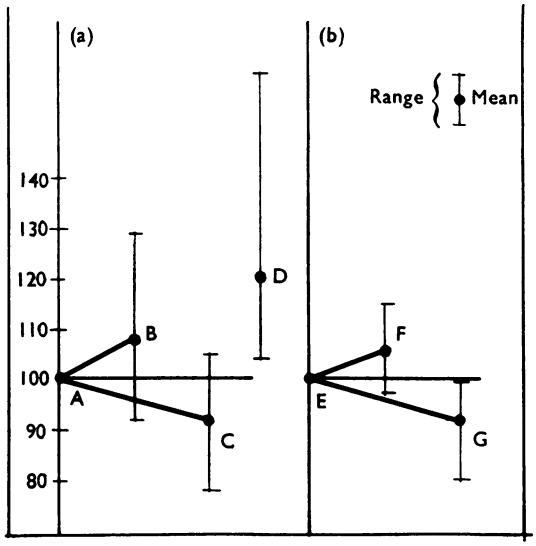


FIG. 3.—(*a*) Means of values of F.E.V.$_1$ in patients with obstructive airway disease, expressed as percentages of the values at rest (A). B—after the Müller manœuvre (14 patients). C—after the Valsalva manœuvre (10 patients). D—after adrenaline spray inhalation. (*b*) Means of values of F.E.V.$_1$ in the same patients after inhalation of adrenaline, similarly expressed. E—at rest. F—after the Müller manœuvre. G—after the Valsalva manœuvre.

due to unusually low volumes registered by three subjects. In six healthy subjects the Valsalva manœuvre resulted in a fall of 5% (range 0% to −7%).

## DISCUSSION

An increase in the F.E.V.$_1$ followed exercise only in patients with obstructive airway disease. After adrenaline inhalation the increase was a little less. Healthy people showed no increase, neither did patients with heart disease or lung disease without airway obstruction. The increase must therefore be related to airway obstruction. The immediate cause of the increase in the F.E.V.$_1$ must be a change in the force applied to the lungs or a decrease in airway resistance.

At a given initial lung volume an increasing force applied to the lungs of patients with obstructive airway disease does not produce an increasing air flow: instead of driving the air it soon traps it (Fry and others, 1954).

A stronger " respiratory drive " after exercise is unlikely to increase the F.E.V.$_1$ since holding the breath alone does not do so. Lewis and Morton (1954) using this method found the same to be true of the maximum voluntary ventilation of healthy subjects.

An increase in the depth of the inspiration which precedes it causes an increase in the F.E.V.$_1$. This would be expected because the first part of the forced expiration is always the fastest delivered. That an increase in the maximum inspiratory position of the ribs may play no part in causing the exercise increase was shown clinically. In spite of his fixed chest a patient with ankylosing spondylitis and obstructive airway disease increased his F.E.V.$_1$ by 15% after exercise. Another patient with ankylosing spondylitis and healthy lungs showed no increase. Whether exercise causes a rise in the maximum inspiratory position of the lungs of patients with obstructive airway disease is not known. It does not do so in healthy people (Prime, 1958).

It does not seem likely, therefore, that the rise in the F.E.V.$_1$ which follows exercise is due either to an increase in the force applied to the lungs or to an increase in the lung volume at which the force is applied.

Release of adrenaline within the body during exercise (Lewis and Morton, 1954) is unlikely to be important, since the increase after exercise was observed both before and after adrenaline inhalation.

We therefore suggest that the rise in the F.E.V.$_1$ which followed exercise was due to a fall in airway resistance. An apparent fall in airway resistance followed forced inspiration whether voluntary or due to exercise (Experiment 2). An apparent fall in airway resistance also followed an increase in lung blood volume (Experiment 3). Immediately after our subjects had exercised it is possible that there was an increase in their rate of inspiration and in the volume of blood in the capillaries and larger vessels of their lungs.

We have therefore tried to show how an increase in inspiratory rate and an increase in lung blood volume may each cause an apparent fall in airway resistance. An apparent fall in airway resistance following increase in the rate of inspiration might occur in the following way. In obstructive airway disease some parts of the lungs may be more severely affected than others: the resistance to airflow would then be higher in some parts of the lungs than in others. During a fast inspiration the narrower high resistance airways would offer a disproportionately greater opposition to airflow. The airways offering lower resistance would be ventilated instead. The alveoli served by these low resistance airways would be filled to greater capacity than if the same lung volume were achieved by a slow

inspiration. They would also be more tense: as the stretch is greater so is the strain. This sequence of events has been postulated to explain the stiffening of emphysematous or asthmatic lungs which has been found to follow an increase in the respiratory rate. From the simultaneous measurement of lung pressures and lung volumes, Christie (1934) and McIlroy and Christie (1952) observed that the distensibility of the lungs of emphysematous patients depends largely on the respiratory rate. This has been confirmed and discussed by Mead, Lindgren, and Gaensler (1955), Otis, McKerrow, Bartlett, Mead, McIlroy, Selverstone, and Radford (1956), Cherniack (1956), McIlroy and Marshall (1956), Attinger, Herschfus, and Segal (1956), and Prime (1958). The lungs of healthy people were not found to stiffen as their respiratory rate increased.

The relative tenseness of the "over-distended" alveoli would enable them to empty into their low resistance airways with less assistance from intrathoracic pressure. With lower expiratory intrathoracic pressures expiratory collapse of the airways and "trapping" would be less (Dayman, 1951). There would be an apparent improvement in the elastic recoil of the lungs and an apparent fall in airway resistance. As expected the rise was less after adrenaline inhalation since airway resistance was then lower and more evenly distributed. In health the rise was not observed.

In the patients with obstructive airway disease the increase in the mean of the $F.E.V._1$ measurements following forced inspiration (10%) was perhaps too small to account completely for the increase in the mean of the $F.E.V._1$ measurements after exercise (24%). Something else may have influenced the change, possibly an increase in lung blood volume during exercise. This is suggested because an increase in lung blood volume by the Müller manœuvre increased the $F.E.V._1$ at rest by a mean 7% (Experiment 3). An increase in the volume of blood in their capillaries and larger vessels would make the lungs more turgid. This might help to support them, increase their power of recoil, and keep their airways open during forced expiration.

The influence of positive and negative intrathoracic pressures on lung blood volume has been discussed by Fenn, Otis, Rahn, Chadwick, and Hegnauer (1947) and Fowler, Guillet, and Rahn (1951). Fenn and his colleagues showed that an intrathoracic pressure of 30 cm. of water (during the Valsalva manœuvre) might displace half the blood contained in the lungs.

Our experiments do not explain the increase in the maximum voluntary ventilation rate of healthy subjects after exercise (Lewis and Morton, 1954). Ogilvie, Stone, and Marshall (1955) reported three healthy subjects who increased their maximum voluntary ventilation at rest when the test was performed with inspiratory rather than expiratory emphasis. This shift of emphasis might have occurred when Lewis and Morton's subjects carried out the maximum ventilation test after exercise.

The increase in the $F.E.V._1$ which followed exercise was therefore probably due to an increase in the rate of inspiration and possibly to an increase in the volume of blood in the lungs. This might be evidence for an adaptation important in maintaining "the just equilibrium between blood and air which meet to undergo mutual and chemical change within the lungs" (Watson, 1871).

These changes may explain the paradoxical increase in the maximum ventilatory capacity which may occur during exercise in patients with obstructive airway disease.

## Summary

In patients with obstructive airway disease the one-second forced expiratory volume ($F.E.V._1$) usually increased immediately following exercise both before and after adrenaline spray inhalation. This did not occur in healthy people or in patients with heart disease or pulmonary fibrosis without airway obstruction.

In patients with obstructive airway disease the $F.E.V._1$ usually increased immediately after forced inspiration and immediately after increase in lung blood volume by the Müller manœuvre. This did not occur in healthy subjects or in the patients without obstructive airway disease. Therefore the increase in the $F.E.V._1$ after exercise in patients with obstructive airway disease was due probably to an increase in the rate of inspiration and possibly to an increase in lung blood volume. We discuss a way in which uneven distribution of air within the lung exaggerated by forced inspiration may cause an apparent fall in airway resistance.

In patients with obstructive airway disease, patients with heart disease, and healthy subjects, the $F.E.V._1$ was reduced immediately after reduction in lung blood volume by the Valsalva manœuvre.

The relationship of these findings to the paradoxical increase in maximum ventilatory

capacity during or immediately after exercise is discussed.

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