PHYSIOLOGICAL STUDIES OF THE EFFECTS OF INTERMITTENT POSITIVE PRESSURE BREATHING ON CARDIAC OUTPUT IN MAN\textsuperscript{1,2}

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Three types of positive pressure breathing have been differentiated: a) continuous positive pressure breathing where a pressure above atmospheric is maintained in the lungs throughout the respiratory cycle, b) expiratory positive pressure breathing in which a pressure above atmospheric is present during the expiratory phase of breathing, and c) intermittent positive pressure breathing (IPPB), provided by automatic respirators, which consists in an active inflation of the lungs under an increasing positive (above atmospheric) pressure; while after cycling of the respirator, deflation of the lungs occurs almost to or to atmospheric pressure as a passive process engendered by the elasticity of the lungs and chest-wall structures. Only the latter type of pressure breathing (IPPB) is suitable for maintaining artificial respiration in the apneic subject. Moreover, IPPB as produced by automatic respirators is more comfortable and less exhausting for the conscious subjects when compared to continuous or expiratory positive pressure.

In a number of recent studies measurements of cardiac output were made on the same subjects during normal breathing at ambient pressure and during positive pressure breathing. Several observers (1, 2, 3,) have reported a reduction in cardiac output during positive pressure breathing in man, more or less proportional to the mean mask pressure used. Previous studies from this laboratory (4, 5) have shown that the changes in cardiac output are quite variable depending upon the types of intermittent positive pressure-breathing respirators used. In the same individual the cardiac output was decreased when on one type of respirator and increased when on another type. It has been suggested that these differences are related to the shape of the mask pressure curve produced by the intermittent positive pressure-breathing respirators (5).

In the present study, instead of grouping the data according to the type of automatic respirator used, the changes in cardiac output observed have been

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correlated to the type of mask pressure curve obtained. An attempt is then made to analyze the physiological factors involved in such correlation.

METHODS

The following automatic respirators were used to produce intermittent positive pressure breathing (IPPB): a) the pneumatic balance respirator, Burns model, PBR (6, 3), b) the Bennett aviation model respirator, Ben. X-1 (4, 5), c) the Bennett clinical research model respirator, Ben. X-2 (4, 5) and d) the Emerson intermittent positive pressure model (4, 5). All of the above respirators except the Emerson followed the slightest breathing efforts of conscious patients, and subjectively the action was smooth and the harshness of the ‘suck and blow’ type, with a fixed cycling pattern, was absent.

All of the mask pressure curves provided with IPPB have been classified in one of three types as shown in figure 1: Type I-IPPB consists of a symmetrical mask pressure curve with a gradual increasing and decreasing slope, with inspiratory and expiratory time approximately the same, and with a minimal expiratory pressure slightly above atmospheric. Type II-IPPB consists of an asymmetrical mask pressure curve with a rapidly increasing pressure during inspiration and a rapidly decreasing pressure during expiration, with a long inspiratory and a short expiratory time period, and the minimal expiratory pressure above atmospheric. Type III-IPPB consists of an asymmetrical mask pressure curve with a gradually increasing and decreasing slope, with inspiratory and expiratory time approximately the same, and with a minimal expiratory pressure slightly above atmospheric.

![Figure 1: Three types of mask pressure curves observed during IPPB studies. The dotted line is the mean mask pressure for the entire respiratory cycle.](http://ajplegacy.physiology.org/Downloaded_from http://ajplegacy.physiology.org)
curve with a gradually increasing pressure during inspiration and a pressure rapidly dropping to atmospheric early in expiration, and the expiratory time equal to or exceeding inspiratory time. The PBR and the Emerson intermittent positive pressure model produce only type I-IPPB, and the Ben. X-1 produces only type II-IPPB. The Ben. X-2 can be adjusted to produce the three types of mask pressure curves.

Cardiac output was determined by the direct Fick method employing the right heart catheterization technique (7-10) with the subject resting supine in a basal state. Control cardiac output measurements were determined at ambient pressure (normal spontaneous breathing) before and 30 minutes after each period of IPPB. The mixed venous blood was obtained from the right ventricle or pulmonary artery. Measurements were usually obtained after 10 minutes of IPPB; however, in four subjects serial cardiac output measurements were taken over a 70-minute period of continuous IPPB. The arterial blood was obtained through an intra-arterial needle placed in most cases in the brachial and in a few cases in the femoral artery. Sampling of arterial blood and of mixed venous blood and the measurement of oxygen consumption were made simultaneously for each cardiac output determination (10). Oxygen consumption was measured by analysis on the Haldane apparatus of expired air collected in a Tissot spirometer. In order to make all the calculations comparable, the inspired air was supplied in all instances from a compressed air cylinder, the gas content of which was checked by repeated analyses.

Simultaneous pressure tracings were recorded from the right heart, peripheral artery and face mask using Hamilton manometers (11) or an electric recording system (12). In addition, intrapleural pressures were also recorded in five cases with a therapeutic pneumothorax. The mean pressures were determined by planimetric integration of the area under the curve for complete respiratory cycles for each of the various tracings recorded.

The Van Slyke-Neill apparatus was used to analyze the blood samples for oxygen content and capacity and carbon dioxide content. The blood pH, was determined directly using a glass electrode and the pCO₂ was calculated.

RESULTS

The data obtained in 33 IPPB experiments on 29 human subjects with essentially normal circulation are recorded in table 1.

1. Mask pressure. The mean mask pressure with type II was almost twice that with type III and 50% greater than with type I, although the average peak mask pressure was only slightly higher. However, for a given peak mask pressure (the significant one in inflating the lung in artificial respiration) the mean mask pressure will be higher for type II than for type III as can be seen from the shape of the respective curves (fig. 1), with type I mask pressure curve being intermediate between the other two types.

2. Ventilation. During IPPB both abdominal and thoracic respiratory excursions were increased with a preponderance of diaphragmatic movement as viewed with the fluoroscope. The respiration rate was decreased the most with type I
PRESSURE BREATHING AND CARDIAC OUTPUT

(33%) and the least with type III (18%, table 1). The minute volume of ventilation was only slightly increased with type I (by 23%), but markedly increased with type II (by 67%), and type III (by 132%). The ventilation rise was due to an increase in tidal volume, since the respiration rate was decreased with all three types. The pH increased from 0.05 units with type I to 0.12 units with type II, and accordingly the calculated pCO₂ decreased 4 and 13 mm. Hg, respectively. The correlation was good between the degree of hyperventilation

TABLE 1. EFFECT OF THREE TYPES OF INTERMITTENT POSITIVE PRESSURE BREATHING, AS DIFFERENTIATED BY THE SHAPE OF THE MASK PRESSURE CURVE, ON VENTILATION, BLOOD PRESSURE AND CARDIAC OUTPUT

<table>
<thead>
<tr>
<th>TYPE OF MASK PRESSURE CURVE</th>
<th>I</th>
<th>II</th>
<th>III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of experiments</td>
<td>16</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>Peak mask pressure (mm. Hg)</td>
<td>14.2</td>
<td>16.7</td>
<td>12.3</td>
</tr>
<tr>
<td>Mean mask pressure (mm. Hg)</td>
<td>7.0</td>
<td>10.6</td>
<td>5.7</td>
</tr>
<tr>
<td>Respiration rate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>per min.</td>
<td>14</td>
<td>13</td>
<td>12</td>
</tr>
<tr>
<td>per cent change¹</td>
<td>-33</td>
<td>-28</td>
<td>-18</td>
</tr>
<tr>
<td>Ventilation rate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liters per min.</td>
<td>10</td>
<td>13</td>
<td>16</td>
</tr>
<tr>
<td>per cent change¹</td>
<td>+23</td>
<td>+67</td>
<td>+132</td>
</tr>
<tr>
<td>Arterial blood, pCO₂ and pHₙ</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1) pCO₂</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mm. Hg</td>
<td>34</td>
<td>28</td>
<td>30</td>
</tr>
<tr>
<td>change¹ mm. Hg</td>
<td>-4</td>
<td>-13</td>
<td>-8</td>
</tr>
<tr>
<td>(2) pHₙ</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pHₙ</td>
<td>7.47</td>
<td>7.55</td>
<td>7.50</td>
</tr>
<tr>
<td>change¹ in pHₙ units</td>
<td>+.05</td>
<td>+.12</td>
<td>+.08</td>
</tr>
<tr>
<td>Blood pressure, syst/diast. (mm. Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1) Arterial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal breathing</td>
<td>120/77</td>
<td>126/77</td>
<td>120/77</td>
</tr>
<tr>
<td>Pressure breathing</td>
<td>135/82</td>
<td>129/79</td>
<td>133/82</td>
</tr>
<tr>
<td>(2) Right ventricle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal breathing</td>
<td>28/2.5</td>
<td>25/1</td>
<td>28/2</td>
</tr>
<tr>
<td>Pressure breathing</td>
<td>20/5</td>
<td>30/8</td>
<td>33/5</td>
</tr>
<tr>
<td>Cardiac output</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liters per min.</td>
<td>5.9</td>
<td>5.8</td>
<td>6.3</td>
</tr>
<tr>
<td>per cent change¹</td>
<td>-14.5</td>
<td>-16.5</td>
<td>+6.0</td>
</tr>
</tbody>
</table>

¹ With respect to normal breathing at atmospheric pressure.
produced by types I and II-IPPB and the change in pCO₂. Although the hyper-
ventilation was most marked with type III-IPPB, there was a larger dead space
in the hose-line connection of the mask to the respirator, with a resulting greater
rebreathing; this probably accounted for the observed smaller change in pCO₂
and pHₐ. Changes in blood gases and pHₐ observed after the respirators were
in use for several hours were of the same magnitude as the changes observed after
10 to 15 minutes. The respiratory alkalosis observed was apparently of little
clinical significance. Even in the cases on type II-IPPB, where the respiratory
changes from hyperventilation were most marked (pCO₂ 28 mm. Hg and pHₐ
7.55), no manifestation of tetany was observed. Observations after 60 minutes
(3) on subjects at a 25,000-foot simulated altitude and with hyperventilation
showed still lower pCO₂ and a higher pHₐ without evidence of tetany.

3. Blood pressures. The blood-pressure changes produced by the three types
of IPPB were very small as shown in table 1 and figures 2, 3 and 4. There was a
slight rise in the right ventricle blood pressure with all three types. The right
ventricle end diastolic pressure rise was the largest with type II (7 mm. Hg as
compared to 2.5 and 3.0 mm. Hg with the others). The systolic pressure rise
was the least with type I. The arterial pressure, both systolic and diastolic,
was increased slightly with types I and III and decreased by 3 mm. Hg with
type II. The latter decrease may be related to the decrease in pulse pressure
present after about five heart beats after inspiration starts, which persists over
the balance of the long inspiratory period and is followed by an increase in pulse
pressure over only a short expiratory period.

4. Cardiac output. The cardiac output was decreased on an average by 14.5
per cent with type I-IPPB and 16.5 per cent with type II-IPPB when compared
to control cardiac output measurements made during ambient breathing at
atmospheric pressure. There was no decrease with type III-IPPB as compared
to the controls but a slight increase of 6.0 per cent. When the decrease in car-
diac output, which occurred with types I and II, was plotted against the mean
mask pressure for each individual case, there was a good correlation between
the height of the mean mask pressure and the decrease in cardiac output (figs.
5 and 6). There was no decrease in cardiac output with type III, hence no cor-
relation between the height of the mean mask pressure and the change in car-
diac output as with the other two types of IPPB (fig. 7).

The effect of a 70-minute period of continuous IPPB with type I curve was
studied on four essentially normal subjects by serial cardiac output determina-
tions. The cardiac output was taken 10 minutes after starting type I-IPPB,
after 40 minutes and after 70 minutes. Thirty minutes after the IPPB was
stopped, the final ambient cardiac output measurement was taken. The de-
crease in cardiac output was greater during the first 10-minute period (23%) as
compared to a 17 per cent decrease present after the 40-minute and 70-minute
periods of IPPB. Most of the studies of the effects of IPPB on cardiac output
have been made after 10 minutes of pressure breathing, which apparently is a
period of maximal change in cardiac output for any given type and pressure of
IPPB employed.
FIG. 2. SIMULTANEOUS PRESSURE RECORDINGS with Hamilton manometers and the electrocardiogram.

FIG. 3. SIMULTANEOUS PRESSURE RECORDINGS with Hamilton manometers and the electrocardiogram. Note the decrease in arterial pulse pressure during the latter half of inspiration with type II-IPPB.

FIG. 4. SIMULTANEOUS PRESSURE RECORDINGS with Hamilton manometers and the electrocardiogram. Note there are as many heart beats during expiration as during inspiration.
5. Net pressure filling in right ventricle. In an attempt to analyze the mechanism underlying the changes observed in cardiac output, net filling pressure measurements in the right ventricle were made during IPPB in three subjects with a therapeutic pneumothorax. The net filling pressure for the right ventricle for a given heart beat was calculated by subtracting the simultaneously recorded pleural pressure from the pressure in the right ventricle at the end of diastole. A beat-to-beat analysis was made of mask pressure, pleural pressure, net filling pressure of the right ventricle and the duration of the cardiac cycle for consecutive pulses and for complete respiratory cycles, and representative graphic analyses have been made for both ambient and IPPB as shown in figure 8 for type I,

![Type I Mask Pressure Curve](image1)

**FIG. 5.** RELATION BETWEEN MEAN MASK PRESSURE INCREASE and change in cardiac output from control ambient. Note the proportional decrease in cardiac output as the mean mask pressure is increased.

**FIG. 6.** NOTE A SIMILAR PROPORTIONAL DECREASE in cardiac output as the mean mask pressure is increased as shown in figure 5.

**FIG. 7.** NOTE THE ABSENCE OF A DECREASE in cardiac output correlated with the mean mask pressure as shown in figures 5 and 6.

in figure 9 for type II and in figure 10 for type III. It can be seen that the curves of net filling pressure, although in phase, varied inversely as the pleural pressure. The net filling pressure of the right ventricle decreased during the phase of increasing mask pressure and increased during the phase of decreasing mask pressure. It is well shown in figures 9 and 10 that the variations in net filling pressure were not due to lengthening or shortening of the duration of the cardiac cycle.

It was also found that when the mean right ventricular net filling pressure calculated for the several entire respiratory cycles was decreased during IPPB as compared to the control value, there was also a decrease in cardiac output, as observed with type I and II-IPPB (figs. 8 and 9). The greater the decrease in the mean right ventricular net filling pressure, the greater the decrease in cardiac output. On the other hand, when the mean right ventricular net filling
pressure for entire respiratory cycles was unchanged or slightly increased as compared to the control value, there was no decrease in cardiac output as observed with type III-IPPB (fig. 10). All these observations made with two types of IPPB on each of the three subjects with a therapeutic pneumothorax are summarized in figure 11. In this figure the differences between the mean

![Fig. 8: Beat-to-beat analysis giving the correlation between pleural pressure and hemodynamic changes with ambient and respirator breathing.](image)

**Fig. 8.** A beat-to-beat analysis giving the correlation between pleural pressure and hemodynamic changes with ambient and respirator breathing. The mean net-filling pressure was measured over several respiratory cycles both by planimetric integration of the curves and by averaging the summated values of each separate beat. The difference between the mean values calculated by both methods was insignificant. The cardiac output was measured immediately before and after the pressure records were taken.

**Fig. 9.** A beat-to-beat analysis giving the correlation between pleural pressure and hemodynamic changes with ambient and respirator breathing. For further explanation see figure 8 and text.

**Fig. 10.** A beat-to-beat analysis giving the correlation between pleural pressure and hemodynamic changes with ambient and respirator breathing. For further explanation see figure 8 and text.

right ventricular net filling pressure measured for several respiratory cycles during ambient pressure breathing and IPPB are plotted against the corresponding differences in cardiac output. It shows a very good correlation between changes in the right ventricular net filling pressure and cardiac output. It can be seen that a decrease of 0.5 and 1.8 mm. Hg in the mean right ventricular
net filling pressure resulted in a reduction in cardiac output of 25 per cent and 39 per cent, respectively, while an increase of 0.5 mm. Hg in the mean right ventricular net filling pressure resulted in an increase of 12 per cent in cardiac output.

Right intraventricular pulse pressure variations were also studied in relation to the shape of the mask pressure curve, but no consistent relationship was found. It should be pointed out that in contrast to net filling pressure, pulse pressure in the right ventricle is influenced both by stroke volume and pulmonary vascular resistance changes. The effects of a changing stroke volume upon the pulse pressure in the right ventricle may be masked by the effects in opposite directions of variation in pulmonary vascular resistance caused by IPPB. Therefore, no conclusion may be derived concerning stroke volume variations during IPPB from a beat-to-beat analysis of pulse pressures in the right ventricle.

**DISCUSSION**

It is well known that variations in intrathoracic pressure may have an influence upon the filling pressure and therefore the output of the right heart. Hamilton *et al.* (13) found that the pressure gradient forcing blood through the lungs is decreased by a prolonged rise in intrathoracic pressure and increased immedi-
ately afterwards in the unanesthetized dog. Lauson, Bloomfield, and Cournard (14) have given evidence indicating that the stroke volume of the two ventricles is unequal in man during deep breathing. The following mechanism was suggested: with an increase in pressure in the thorax during expiration, the right heart filling pressure from the large veins was reduced and the stroke volume decreased. At the same time the output from the left heart increased during expiration with the increased venous return from the pulmonary circuit. Studies of IPPB indicate that the same mechanism is operating in a reverse fashion and to a greater extent with the phase of increasing intrathoracic pressure during inspiration and the phase of decreasing pressure during expiration. The decrease in output from the right heart, which occurs during the phase of rising pressure in the mask and in the thorax, may be made up during the phase of falling mask pressure. The reverse occurs in the left heart but with limitations, for the increased output during inspiration with IPPB can be maintained for short periods only (3–5 pulses) by the displacement of pulmonary blood, for if the inspiratory mask pressure is prolonged, the arterial pulse pressure decreases after the initial rise (fig. 3). Thus the variation in stroke volume in the right ventricle is the important part to consider in relation to changes in cardiac output.

The right ventricular net diastolic filling pressures were lower with the IPPB during periods of increasing intrapleural pressure than during the periods of ambient breathing with all three types of mask pressure curves. The reverse occurred during the period of decreasing intrapleural pressure when the mask pressure decreased to or almost to atmospheric. If one may apply here what is commonly referred to as Starling's principle in the sense that increased diastolic filling pressure results in increased stroke volume, then these changes suggest that the deficit in cardiac output incurred during the phase of increased intrapleural pressure is compensated for during the phase of decreased intrapleural pressure. If after cycling of the respirator the pressure falls rapidly to atmospheric and if the duration of expiration is slightly longer than inspiration, then compensation may be complete and no decrease in cardiac output occurs, as observed with type III-IPPB. Compensation during expiration is not, however, complete with types I and II-IPPB, because the pressure does not drop rapidly to atmospheric with type I-IPPB and expiratory time is too short, and the end pressure is still above atmospheric with type II-IPPB.

An overall confirmation of Starling's principle is provided by the observations on mean net filling pressure in the right ventricle measured over entire respiratory cycles. Accordingly, in these observations a change in this mean net filling pressure is associated with a proportional change in cardiac output and it is well to emphasize that even small changes, demonstrated with accurate methods

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6 Actually this pressure-flow relationship was proposed by O. Franck in 1904, and discussed by Patterson, Piper, and Starling in 1914, who put forth the proposition that the fiber length or diastolic volume rather than the diastolic filling pressure determined the energy of mechanical ejection.
of recording pressure in the heart, may have a considerable effect upon cardiac output.

The question may be raised as to whether the intrapleural pressure values are the same as the extracardiac pressures during both phases of respiration or vary in the same direction. Bookhart and Boyd (15) have reported higher pressures inside the pericardium than in a small closed pneumothorax in anesthetized dogs, but on enlarging the pneumothorax the pressure difference was decreased. It would appear that the pressure in a therapeutic pneumothorax in man involving one fourth to one third of the volume of one lung would approximate very closely the pressure outside the right auricle.

On the basis of the observations made with the three types of IPPB on changes in cardiac output in essentially normal unanesthetized subjects, a very desirable type of IPPB mask pressure curve produced by respirators may be described. The rise in pressure should gradually increase during inspiration to a peak not in excess of approximately 25 cm. water, and after cycling occurs, the pressure drop should be rapid early in expiration and with a mean mask pressure during the expiratory period as near atmospheric as possible, as with type III-IPPB (fig. 1). Inspiratory time should not exceed expiratory time, thereby allowing as many heart beats to occur during expiration, with the increased right ventricular net filling pressure, as during inspiration, with the decreased net filling pressure. Such a mask pressure curve as described above permits the decrease in output from the right heart during the inspiratory phase of rising intrathoracic pressure to be made up during the phase of falling mask pressure.

SUMMARY

1. The effects of three types of intermittent positive pressure breathing, as differentiated by the shape of the mask pressure curve, have been correlated with the changes in cardiac output observed in 33 experiments on 29 human subjects.

2. The three types of mask pressure curves were as follows: type I, symmetrical with gradual increasing and decreasing slope, expiratory time approximately the same as inspiratory and the end expiratory pressure above atmospheric; type II, asymmetrical with rapidly increasing pressure during inspiration and rapidly dropping during expiration, long inspiratory and short expiratory time intervals and the end expiratory pressure above atmospheric; and type III, asymmetrical with gradually increasing pressure during inspiration and suddenly dropping early in expiration to atmospheric and expiratory time equal to or exceeding inspiratory.

3. Cardiac output was decreased more or less in proportion to the increase in mean mask pressure with the first and second type curves. There was no decrease in cardiac output with the third type curve.

4. The net filling pressure of the right ventricle decreased during the phase of increasing mask pressure and increased during the phase of decreasing mask pressure with all types of curves. The curve of net filling pressure of the right
ventricle, although in phase with, varied inversely as the pleural pressure and mask pressure.

5. The mean net filling pressure of the right ventricle was calculated for complete respiratory cycles during intermittent pressure breathing and during ambient breathing. In cases where the cardiac output was reduced during intermittent pressure breathing as compared to ambient, the mean net filling pressure was reduced also. Conversely, when the cardiac output was increased the mean net filling pressure was increased.

6. Interpreted in terms of variation in stroke volume, these changes suggest that the deficit in cardiac output incurred during the inspiratory phase is compensated for during the expiratory phase. When the pressure drop is rapid in expiration with the resulting intrapleural pressure low and the right ventricular net filling pressure high, compensation is complete provided expiratory time is of sufficient duration. Expiratory time must equal or exceed inspiratory time in order that the number of heart beats during expiration may equal or exceed the number present during inspiration. The time and pressure relationships with the third type of curve permits compensation to be complete, as the mean right ventricular net filling pressure is not decreased. Compensation was incomplete with the first and second types, because the mask pressure does not drop rapidly after cycling with the first, expiratory time is too short and the end expiratory pressure is still above atmospheric with the second.

7. A very desirable type of IPPB should provide a mask pressure curve that shows: a) a gradual increase in pressure during inspiration, b) a rapid drop in pressure after cycling occurs, c) a mean mask pressure during the expiratory period as near atmospheric as possible and d) an expiratory time equal to or exceeding the inspiratory time. Adequate ventilation can be provided with the above type of pressure breathing in man with a minimal disturbance to the circulation, and this type would seem most desirable physiologically for administering artificial respiration.

REFERENCES