HAEMODYNAMIC EFFECTS OF A NEGATIVE (SUBATMOSPHERIC) PRESSURE EXPIRATORY PHASE DURING ARTIFICIAL VENTILATION

D. B. SCOTT, G. W. STEPHEN AND I. T. DAVIE

SUMMARY

The effects of a negative expiratory phase on cardiac output, arterial blood pressure, central venous pressure and heart rate were measured in six seriously ill patients undergoing artificial ventilation in an intensive care unit. Cardiac output was increased in one patient and decreased in two others. The circulatory benefit of a negative phase was considered to be minimal.

It is widely held that the introduction of a negative (subatmospheric) pressure expiratory phase during intermittent positive pressure respiration assists cardiac output. As a result, most ventilators are capable of producing a negative expiratory phase and this should, therefore, assist the venous return and be of value in the management of patients with cardiovascular impairment who require artificial ventilation.

In order to test this concept and, if possible, to quantitate the effect of a negative pressure phase, cardiac output was measured in six seriously ill patients receiving positive pressure ventilation.

METHODS

Six patients receiving treatment in the Assisted Ventilation Unit of the Royal Infirmary, Edinburgh, were studied. Details of the patients are shown in table I.

An intra-arterial catheter was inserted percutaneously either into the brachial or the femoral artery. A central venous catheter was introduced via a suitable vein in the cubital fossa. Continuous recordings of arterial pressure, central venous pressure and e.c.g. were made. Cardiac output was measured by the dye dilution method using indocyanine green and a Waters 302 cuvette and densitometer.

All patients were being ventilated at the time of the study and the ventilator settings were not altered from those chosen by the clinician in charge of the case. Ventilatory control had been achieved in each case without the use of muscle relaxants. Some cases had received opiates for this purpose but none had been given within 1 hour of the investigation. Cape ventilators were used in all cases. The positive pressures used varied from 20 to 40 cm H₂O. Four control measurements of cardiac output were obtained at 2-minute intervals in each patient following which a negative pressure expiratory phase was added without changing the tidal volume setting. Cardiac output estimations continued to be taken at 5-min intervals until four further observations had been made. The negative pressure used varied from -5 to -7 cm H₂O. Introduction of the negative phase did not appear to alter the positive pressure obtained by the fixed tidal volume.

Blood-gas values were measured during the control period and at the completion of the study (after 20 min of positive/negative pressure ventilation).

RESULTS

The mean results are given in table II.

Cardiac output. The mean cardiac output did not change during the period of positive/negative pressure ventilation. In individual patients there was a small sustained increase in one, but in two other patients there was a small but definite fall in output. Individual results are shown in figure 1.

Mean arterial pressure. The mean arterial pressure during positive pressure ventilation was 83 mm Hg (SE 4.6) and 86 mm Hg (SE 4.9) after the introduction of a negative pressure phase. Two patients had a slight increase and one had a fall in arterial pressure (fig. 2).

Heart rate. This did not change appreciably (fig. 3).


Present addresses:
* Queen Charlotte's Hospital, Goldhawk Road, London, W.6.
† Western General Hospital, Edinburgh.
TABLE I. Clinical details of the six patients investigated.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39</td>
<td>Crushed chest</td>
<td>Discharged home</td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>Crushed chest</td>
<td>Discharged home</td>
</tr>
<tr>
<td>3</td>
<td>62</td>
<td>Duodenal leakage—post-gastrectomy; respiratory failure</td>
<td>Died</td>
</tr>
<tr>
<td>4</td>
<td>52</td>
<td>Crushed chest</td>
<td>Discharged home</td>
</tr>
<tr>
<td>5</td>
<td>53</td>
<td>Pancreatitis, diabetes; renal failure; respiratory failure</td>
<td>Died</td>
</tr>
<tr>
<td>6</td>
<td>16</td>
<td>Crushed chest</td>
<td>Died</td>
</tr>
</tbody>
</table>

TABLE II. Cardiac output, mean arterial pressure, heart rate and central venous pressure during IPPR and IP/NPR in six patients. Mean values are shown with standard error in parentheses.

<table>
<thead>
<tr>
<th>Positive pressure period (min)</th>
<th>Positive/negative pressure period (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output (L/min)</td>
<td>Mean (SE)</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>7.7 (0.8)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>82.4 (4.6)</td>
</tr>
<tr>
<td>Central venous pressure (mm Hg)</td>
<td>6.0 (0.9)</td>
</tr>
</tbody>
</table>

![Fig. 1. Cardiac output measurements in six patients during IPPR and IP/NPR.](https://academic.oup.com/bja/article-abstract/44/2/171/393019)

![Fig. 2. Mean arterial pressure in six patients during IPPR and IP/NPR.](https://academic.oup.com/bja/article-abstract/44/2/171/393019)

![Fig. 3. Heart rate in six patients during IPPR and IP/NPR.](https://academic.oup.com/bja/article-abstract/44/2/171/393019)

![Fig. 4. Central venous pressure in six patients during IPPR and IP/NPR.](https://academic.oup.com/bja/article-abstract/44/2/171/393019)
EFFECTS OF A NEGATIVE PRESSURE EXPIRATORY PHASE

173

Table III. Airway pressures and blood-gases in individual patients.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Positive pressure period</th>
<th>Positive/negative period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>cm H2O</td>
<td>pH</td>
</tr>
<tr>
<td>1</td>
<td>0/25</td>
<td>7.47</td>
</tr>
<tr>
<td>2</td>
<td>0/30</td>
<td>7.49</td>
</tr>
<tr>
<td>3</td>
<td>0/33</td>
<td>7.25</td>
</tr>
<tr>
<td>4</td>
<td>0/30</td>
<td>7.45</td>
</tr>
<tr>
<td>5</td>
<td>0/20</td>
<td>7.37</td>
</tr>
<tr>
<td>6</td>
<td>0/40</td>
<td>7.38</td>
</tr>
</tbody>
</table>

Central venous pressure. Taken as a mean of the six patients, there was a slight fall in the central venous pressure but this was not statistically significant (Student $t = 0.707$; $P > 0.24$). The central venous pressure fell by 2 mm Hg in two patients and by less than 1 mm Hg in a third. The other three showed no change (fig. 4).

Blood gases. These are given in table III. As will be seen, the introduction of a negative phase did not alter the blood gases, the tidal volume having remained constant.

Discussion

A negative (subatmospheric) pressure expiratory phase during positive pressure respiration has been advocated for many years as a means of minimizing the adverse effects of the positive pressure upon the circulation especially in those cases with circulatory embarrassment (see review by Mushin et al., 1969). The negative pressure is said to reduce the mean intrathoracic pressure, thereby encouraging the venous return to the right heart.

As a result of this concept, virtually all ventilators have a built-in mechanism for applying a negative phase during expiration. In practice, however, this seldom appears to be used and it is common experience that little benefit seems to accrue from the use of negative pressure, as judged clinically even in seriously ill patients undergoing intensive care.

Surprisingly little data on the circulatory effects of positive/negative pressure respiration are available from studies in man. Cournand and associates (1948) showed that cardiac output was reduced if the airway pressure was positive throughout the respiratory cycle and if inspiration was long compared with expiration. However, of the three types of ventilatory pattern described by them, the worst (positive pressure throughout respiration and a prolonged inspiratory time) reduced cardiac output by only 14 per cent. The best pattern, which did not incorporate a negative phase, increased output by 6 per cent over control figures.

The same workers (Motley et al., 1948) compared in detail various ventilators, one of which utilized a negative pressure phase. They again showed the deleterious effects of a high mean airway pressure but found no evidence that negative pressure increased cardiac output. They conclude: "The negative pressure phase has not been found advantageous over simple intermittent positive pressure".

Maloney and associates (1953) strongly advocated a negative phase and produced evidence that, while this only caused a 14 per cent increase in cardiac output in normal subjects, in those with circulatory impairment a 42 per cent increase was noted compared with positive pressure ventilation. However, only four cases of "circulatory impairment" were studied and the methodology used then would be very suspect at the present time. It is extremely difficult to understand how assistance to the circulation of a mechanical kind works only if the venous return is low.

Evidence from animal experiments is also somewhat conflicting.

Hubay and associates (1954), studying dogs with implanted flowmeters, were able to demonstrate a marked improvement in blood flow when a negative phase was introduced, especially in dogs suffering from blood loss. Similar results were obtained by Maloney and Handford (1954). The reasons why hypovolaemic dogs react differently are not clear.

Adams and associates (1970), however, did not observe any important haemodynamic change consequent upon variations in the pattern of positive pressure ventilation in dogs whether normo-, hyper- or hypovolaemic.

The results of the present investigation show that no circulatory benefit accrued from the use of a negative pressure phase in the acutely ill patients receiving ventilator therapy who were studied (although admittedly none was considered hypovolaemic clinically). There are several possible explanations for this.
The classical theory that augmentation of venous return to the right heart by a negative pressure expiratory phase depends upon the production of a negative pressure within the thorax itself. That is, the negative pressure in the airway must be transmitted from the lung to the great veins. Even with normal lungs, it takes 2–3 seconds for the airway and intrathoracic pressures to equalize. With damaged lungs of poor compliance, this process will be prolonged and, unless the respiratory rate is very slow and considerable negative pressure applied, the intrathoracic pressure will be little affected. It can be assumed from the high inflation pressures used in our cases that compliance was poor in all of them.

It is also necessary to consider not only the blood flow to the right heart but also the flow across the pulmonary capillaries. Changes in pressure within the alveoli are transmitted with much greater ease to the capillaries, separated as they are by a single endothelial layer, than to the great veins. Lowering pressure within the alveoli is likely to decrease flow which is dependent upon the pressure gradient between the pulmonary artery and the left atrium. Thus, a retardation of flow with engorgement of the capillary bed could occur during the negative phase, the blood being “pumped” on by the succeeding positive phase. Whether any such increment or decrement to flow occurs is difficult to say and such rapid fluctuations in flow are extremely difficult to measure. Reasons have been given elsewhere why high positive pressures exert their main effect upon the pulmonary capillaries and not on the great veins (Scott, Slawson and Taylor, 1969).

Remembering the deleterious effects, particularly air-trapping, which may occur during positive/negative pressure respiration (Galloon and Rosen, 1965), there appears to be little place in clinical practice for this respiratory pattern.

REFERENCES


EFECTOS HEMODINAMICOS DE UNA FASE ESPIRATORIA CON PRESION NEGATIVA (SUBATMOSFERICA) DURANTE LA VENTILACION ARTIFICIAL

RESUMEN
Los efectos de una fase espiratoria negativa sobre el gasto cardiaco, presión sanguínea arterial, presión venosa central y frecuencia cardíaca fueron medidos en seis pacientes gravemente enfermos bajo ventilación artificial en una unidad de cuidados intensivos. El gasto cardiaco estaba aumentado en un paciente y disminuido en otros dos. Se consideró que el beneficio circulatorio de una fase negativa es mínimo.