TRANSMURAL CENTRAL VENOUS PRESSURE DURING INTERMITTENT POSITIVE PRESSURE RESPIRATION

BY

W. E. WATSON, A. C. SMITH AND J. M. K. SPALDING

From the Respiration Unit, Department of Neurology, United Oxford Hospitals, and the Nuffield Department of Anaesthetics, Oxford University, England

During intermittent positive pressure respiration (IPPR) the mean intrathoracic pressure is elevated compared with the mean intrathoracic pressure in a comparable subject breathing spontaneously (Opie, Spalding and Smith, 1961). Venous return to the right side of the heart is impeded as the intrathoracic pressure rises (Visscher, Rupp and Scott, 1924; Shuler et al., 1942; Dupee and Johnson, 1943; Lauson, Bloomfield and Cournand, 1946; Brecher and Mixter, 1953; Donders, 1856), peripheral venous pressure rises (Humphreys, Moore and Barkley, 1939; Otis, Rahn and Fenn, 1946) and the blood flow along the superior vena cava to the right atrium may be reduced (Brecher, 1956). The difference between central venous pressure and intrathoracic pressure is described as “transmural central venous pressure” (Lee, Matthews and Sharpey-Schafer, 1954; Lee and Gimlette, 1957). This communication reports measurements of transmural central venous pressure made on patients receiving IPPR. Changes in transmural central venous pressure are believed to reflect changes in the degree of impairment of venous return.

METHOD

Nine patients receiving IPPR were investigated, and their clinical features are summarized in table I. All patients had clinically and radiologically normal lungs at the time of investigation and received intermittent positive pressure respiration through cuffed tracheotomy tubes. The patients normally received IPPR from Radcliffe respiration pumps (Russell et al., 1956), but during the course of these investigations they were ventilated with a respiration pump capable of providing IPPR with tracheal pressure of various waveforms (Watson, Spalding and Smith, 1962). Tidal volumes were between 550 and 650 ml and ventilatory frequency was 13 per minute. All patients lay supine during investigations.

Measurements.

All measurements of pressure were made with capacitance manometers and recorded on a direct-writing oscillograph. Unless otherwise stated pressures are relative to atmospheric pressure. Oesophageal pressure was measured with a liquid-filled catheter as in previous observations (Opie, Spalding and Stott, 1959).

Central venous pressure was measured with a liquid-filled catheter of 0.75 mm internal diameter passed percutaneously and directed centrally until respiratory fluctuations were observed in the recorded trace. The catheter was periodically flushed with 0.5 ml normal saline containing heparin 0.1 mg/ml.

Arterial blood pressure was measured through a Cournand needle inserted into the brachial or femoral artery: this system was periodically flushed with a solution of heparin in saline.

Tracheal pressure was measured at the external end of the tracheotomy tube by an air-filled system.

End-tidal Pco₂ was derived from the record of the Pco₂ of gas sampled continuously from the external end of the tracheotomy tube and analyzed with an infra-red CO₂ analyzer calibrated as described elsewhere (Smith, Spalding and Watson, 1962).

Tidal volume was obtained by measuring the expired respiratory minute volume with a Wright respirometer and dividing the result by the observed respiration rate.

Mean oesophageal pressure and mean central venous pressure were derived by measuring the
Summary of clinical features of the nine patients studied.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Duration of IPPR</th>
</tr>
</thead>
<tbody>
<tr>
<td>A:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T.K.</td>
<td>M</td>
<td>20</td>
<td>Poliomyelitis</td>
<td>9 months</td>
</tr>
<tr>
<td>G.W.</td>
<td>M</td>
<td>33</td>
<td>Poliomyelitis</td>
<td>1 year</td>
</tr>
<tr>
<td>A.W.</td>
<td>M</td>
<td>27</td>
<td>Poliomyelitis</td>
<td>3 weeks</td>
</tr>
<tr>
<td>R.J.</td>
<td>M</td>
<td>66</td>
<td>Basilar insufficiency</td>
<td>3 days</td>
</tr>
<tr>
<td>B:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.D.</td>
<td>M</td>
<td>26</td>
<td>Cervical cord injury (C4-5)</td>
<td>9 months</td>
</tr>
<tr>
<td>V.W.</td>
<td>M</td>
<td>36</td>
<td>Cervical cord injury (C2-3)</td>
<td>4 months</td>
</tr>
<tr>
<td>S.W.</td>
<td>M</td>
<td>10</td>
<td>Polyneuritis</td>
<td>4 weeks</td>
</tr>
<tr>
<td>E.P.</td>
<td>F</td>
<td>24</td>
<td>Polyneuritis</td>
<td>2 weeks</td>
</tr>
<tr>
<td>B.H.</td>
<td>M</td>
<td>10</td>
<td>Irreversible cerebral damage from anoxia</td>
<td>3 weeks</td>
</tr>
<tr>
<td>C:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R.J.</td>
<td>M</td>
<td>66</td>
<td>Basilar insufficiency</td>
<td>3 days</td>
</tr>
<tr>
<td>T.K.</td>
<td>M</td>
<td>20</td>
<td>Poliomyelitis</td>
<td>9 months</td>
</tr>
</tbody>
</table>

Mean transmural central venous pressure was derived by subtracting the mean oesophageal pressure from the mean central venous pressure.

Mean arterial blood pressure was calculated as the sum of mean diastolic blood pressure and one-third of the mean pulse pressure (Wood, 1956).

Procedure.

At the beginning of each investigation the "Valsalva" manoeuvre was performed on each patient. As a patient with respiratory paralysis cannot strain against his closed glottis, the tracheal pressure was elevated to about 40 mm Hg by stopping the respiration pump in the inspiratory phase for about 20 seconds. This procedure was curtailed if the patient complained of faintness or of pain in the chest. The arterial blood pressure was continuously recorded throughout this manoeuvre, and the nature of the response of the arterial blood pressure was the criterion by which patients were divided into groups (table I).

Each patient was subjected to brief periods of apnoea by disconnecting him from the respiration pump, and arterial blood pressure was observed. The effect on the arterial blood pressure of alterations in mean intrathoracic pressure before the period of apnoea and of the administration of oxygen before apnoea were also observed.

The inspiratory period was then reduced to 0.5 second, tidal volume remaining at 550 to 650 ml and frequency at 13 per minute. In these circumstances the end-tidal $P_{CO_2}$ was high at a given respiratory minute volume because physiological deadspace increases when inspiration lasts only 0.5 second (Watson, 1962).

During the rest of each investigation the end-tidal $P_{CO_2}$ was kept at this level ($\pm 2$ mm Hg) while the circulatory effect of changes in duration of inspiration and tracheal positive pressure waveform were observed. The tidal volume remained 550 to 650 ml and the frequency 13 per minute, and to keep the end-tidal $P_{CO_2}$ constant external deadspace was added between the respiration pump and the patient.

The transmural central venous pressure was measured with inspiration lasting 0.5, 1, 1.6 and 2.7 seconds using a "square" tracheal positive pressure waveform, in which the peak tracheal pressure was reached within 0.1 second and maintained for the rest of the inspiratory cycle. The transmural central venous pressure was also measured when the mean intrathoracic pressure was either raised by immersing the outlet of the respiration pump in water, or reduced by applying a subatmospheric pressure (0 to $-10$ cm H$_2$O) to the trachea during expiration.
The tracheal positive pressure waveform was varied while the duration of inspiration was 1, 1.6 or 2.7 seconds. Two waveforms were investigated: one was “square” and is described above. In the second waveform the tracheal pressure rose slowly to a peak at the end of inspiration, and the inspiratory flow rate was almost constant. In each case mean oesophageal and transmural central venous pressures were determined.

RESULTS

Responses to Valsalva’s manoeuvre.

The normal response to Valsalva’s manoeuvre is shown in figure 1. There is a characteristic “overshoot” of arterial blood pressure when intrathoracic pressure is lowered, and patients who had this normal response are shown in table IA. Figure 2 shows abnormal responses to Valsalva’s manoeuvre, and in particular there is no overshoot of arterial blood pressure when the intrathoracic pressure is lowered. This is a blocked response (Greene and Bunnel, 1950). Patients with a blocked response who could nevertheless maintain a normal blood pressure during artificial respiration are shown in table IB. Two patients had a blocked response to Valsalva’s manoeuvre and were also unable to maintain their arterial blood pressure at normal levels during artificial respiration. They are shown in table IC, and are the same as two patients in table IA but at a later stage of the illness.

Patients with normal response to Valsalva’s manoeuvre.

If the tidal volume and frequency of respiration are unchanged, the mean intrathoracic pressure rises as the duration of inspiration is increased (fig. 3), and the range of mean intrathoracic pressure obtained in this way is approximately equal to that which occurs during clinical treatment.
TRANSMURAL CENTRAL VENOUS PRESSURE

with IPPR. In patients whose response to Valsalva's manoeuvre is normal, changes in intrathoracic pressure within this range are accompanied by similar changes in central venous pressure, and the transmural central venous pressure therefore remains unchanged (fig. 4(i)).

The range of mean intrathoracic pressure was extended in one direction by applying subatmospheric pressure within the trachea between inspirations, and in the other direction by obstructing the outflow of air from the chest. In one patient (R.J.) mean transmural central venous pressure fell when the mean intrathoracic pressure exceeded about 3 cm H₂O, but in patient T.K. it was sustained until the mean intrathoracic pressure rose to 6 cm H₂O (fig. 4(i)). Figure 4(ii) shows mean arterial blood pressure in these circumstances. In every case a fall in mean transmural central venous pressure, usually a considerable fall, occurred before any change in mean arterial blood pressure.

Patients with blocked response to Valsalva's manoeuvre.

In patients with a blocked response to Valsalva's manoeuvre the mean transmural central venous pressure falls progressively as the mean intrathoracic pressure rises from -0.2 to +4 cm H₂O (fig. 5(i)). The mean arterial blood pressure in two patients was little affected by change in mean intrathoracic pressure (fig. 5(ii)). In the other two patients mean arterial blood pressure was unaltered with mean intrathoracic pressures between -2.0 and +1 cm H₂O, but fell progressively as mean intrathoracic pressure rose above +1 cm H₂O.

Patients with a blocked response to Valsalva's manoeuvre who were also unable to maintain a normal blood pressure during IPPR.

Two patients who had previously had normal responses to Valsalva's manoeuvre were later unable to sustain a normal arterial blood pressure during IPPR, and at that time had a blocked response to Valsalva's manoeuvre. Mean transmural central venous pressure and mean arterial blood pressure fell progressively as mean intrathoracic pressure rose (fig. 6).

Substitution of the "slowly rising" tracheal positive pressure waveform for the "square" tracheal positive pressure waveform never significantly changed either arterial blood pressure or transmural central venous pressure.

Arterial blood pressure during apnoea.

When a patient with complete respiratory paralysis and a normal response to Valsalva's manoeuvre was disconnected from his respiration pump, a biphasic elevation of arterial blood pressure commonly occurred. The second component of this biphasic rise did not usually occur if the patient breathed oxygen before the period of apnoea (fig. 7). The first component was greater when inspiration during the preceding period of IPPR was long than when it was short (fig. 8).

DISCUSSION

In patients whose circulatory control is normal, both clinically and in response to Valsalva's manoeuvre, the mean transmural central venous pressure remains unchanged until the mean intrathoracic pressure reaches 3 to 5 cm H₂O (fig. 4(i)). The mean arterial blood pressure remains unchanged until the mean intrathoracic pressure reaches 5 to 8 cm H₂O (fig. 4(ii)). These pressures are higher than those normally occurring during clinical IPPR, for when the tidal volume is
500 ml, the frequency 16 per minute and the duration of inspiration does not exceed one-third of the respiratory cycle, the mean intrathoracic pressure is about 0.5 to 2.5 cm H$_2$O (Opie, Spalding and Smith, 1961). One of the factors which may be concerned in the maintenance of transmural central venous pressure is active contraction of the walls of capacity vessels when the intrathoracic pressure is raised (Watson, 1961). It is probable that when the mean intrathoracic pressure is further increased and the mean transmural central venous pressure falls, the cardiac output also falls. The mean arterial blood pressure, however, is sustained until the intrathoracic pressure reaches 5 to 8 cm H$_2$O and this is probably achieved by constriction of the arterioles. The "overshoot" of
Response of arterial blood pressure to apnoea in a curarized patient with tetanus. The patient was receiving 100 per cent oxygen until A, when he began to receive air. Upper trace, seconds; second trace, arterial blood pressure; third trace, oesophageal pressure; fourth trace, tracheal pressure.

A prolonged Valsalva’s manoeuvre. If the type of IPPR is adjusted (or maladjusted) to produce a high mean intrathoracic pressure, an overshoot of arterial blood pressure occurs when the intrathoracic pressure is lowered again (fig. 8a). This probably indicates arteriolar constriction. If IPPR is employed in the normal manner the rise in intrathoracic pressure is smaller and there is no overshoot when it is discontinued (fig. 8b). This may indicate that in these circumstances the circulatory stress is insufficient to cause arteriolar constriction. Since subjects with normal circulatory responses to Valsalva’s manoeuvre compensate fully for the obstruction to venous return caused by clinical IPPR, there is no need to lower the mean intrathoracic pressure by applying subatmospheric pressure within the trachea during expiration.

In patients with impaired circulatory control, judged by blocked response to Valsalva’s manoeuvre, arterial blood pressure which occurs when the intrathoracic pressure falls after Valsalva’s manoeuvre (fig. 1) is attributed to arteriolar constriction occurring while the intrathoracic pressure was raised (Lee, Matthews and Sharpey-Schafer, 1954). IPPR causes a rise in mean intrathoracic pressure above the normal and may be regarded as
A progressive fall occurs in mean transmural central venous pressure as the mean intrathoracic pressure is raised throughout the range examined (figs. 5 and 6). This may be connected with the inability of patients with certain disorders, including severe polyneuritis and cervical cord transection, to contract the capacity vessels of their hands reflexly (Watson, 1961). In patients S.W., B.H., and E.P. the fall in mean transmural central venous pressure is accompanied by a fall in mean arterial blood pressure (fig. 5(ii)), probably due to inability to compensate for a fall in cardiac output by constriction of the arterioles. In patients V.W. and A.D., who had complete transection of the cervical part of the spinal cord, a fall in mean transmural central venous pressure occurred when the mean intrathoracic pressure was raised (fig. 5(i)), but arterial blood pressure was better maintained (fig. 5(ii)), suggesting that some arteriolar constriction was occurring. In all patients with a blocked response to Valsalva’s manoeuvre, subatmospheric tracheal pressure applied in expiration during IPPR increased the mean transmural central venous pressure and, in many of them, also the mean arterial blood pressure.

Maloney et al. (1953) showed that in patients with clinical circulatory failure (“shock”) from a variety of causes IPPR might cause a fall in blood pressure and cardiac output. The present observations indicate some of the mechanisms which may account for this. The practical lesson in the management of patients receiving IPPR is that circulatory dangers only arise in patients with impaired circulatory reflexes. If impairment of circulatory control can be demonstrated or is suspected, a subatmospheric tracheal pressure should be applied during expiration.

CONCLUSIONS

It is concluded that:

In these circumstances circulatory failure is reflected earlier in a fall of mean transmural central venous pressure than of mean arterial blood pressure.

In patients with normal circulatory reflexes a rise in mean intrathoracic pressure stimulates directly or indirectly a reflex which maintains mean transmural central venous pressure.

Patients with abnormal circulatory reflexes may be unable to maintain a normal mean transmural central venous pressure as mean intrathoracic pressure rises.

In intermittent positive pressure respiration subatmospheric pressure during expiration can be expected to help the circulation only in patients whose circulatory reflexes are impaired.

SUMMARY

Mean transmural central venous pressure, arterial blood pressure and intrathoracic pressure were measured in tracheotomized patients receiving intermittent positive pressure respiration.

When the mean intrathoracic pressure was raised in steps from −3 to +4 cm H₂O

(a) in patients with a normal response of the arterial blood pressure to Valsalva’s manoeuvre, no marked change of mean transmural central venous pressure was found;

(b) in patients with an abnormal response of the arterial blood pressure to Valsalva’s manoeuvre, mean transmural central venous pressure fell: this fall preceded a fall of arterial blood pressure.

The value of a subatmospheric airway pressure during the expiratory phase of the imposed respiratory cycle is discussed.

ACKNOWLEDGMENTS

We wish to thank Dr. W. Ritchie Russell for continued encouragement and Sister Mair for co-operation and assistance.

REFERENCES


