EFFECT OF SODIUM NITROPRUSSIDE-INDUCED HYPOTENSION ON PULMONARY DEADSPACE

H. J. KHAMBATTA, J. G. STONE AND R. S. MATTEO

SUMMARY
Deliberate hypotension with nitroprusside produced a 40% reduction in mean arterial pressure, but it did not cause any change in the pulmonary deadspace to tidal volume ratio (Vd/Vt). This was achieved by adequate hydration both before and during hypotension, as indicated by constant filling pressures. Thus we have shown that deliberate hypotension per se does not increase Vd/Vt, and that vasodilatation may bring about a relatively hypovolaemic state and be responsible for the increase in Vd/Vt observed by previous workers.

Deliberate hypotension induced by ganglionic blockers was reported to produce an increase in pulmonary deadspace (Eckenhoff et al., 1963), and this led the authors to recommend the use of a high inspired oxygen concentration during anaesthesia with induced hypotension. In the most recent edition of the textbook General Anaesthesia (Gray, Nunn and Utting, 1980), this recommendation still holds, but no one has studied pulmonary deadspace ventilation during hypotensive anaesthesia using current vasodilating drugs. The question whether induced hypotension itself is responsible for the increase in pulmonary deadspace has not been answered. This is the subject of the present study.

METHOD
Ten patients (six male), average age 30 yr (range 23–44 yr), were studied with their informed consent and approval by the Institutional Review Board for human subjects. All patients underwent lumbar laminectomy to remove degenerated herniated disc material and spinal fusion with autogenous iliac bone graft. Preoperative history, physical and laboratory examination revealed no medical problems except those related to their forthcoming surgery.

Premedication consisted of oral diazepam 10 mg the night before surgery and i.m. atropine 0.5 mg and quinalbarbitone 100 mg 1 h before induction of anaesthesia. Anaesthesia was induced with thiopentone 250–300 mg i.v. and the trachea was intubated after muscle relaxation with suxamethonium 100 mg. Anaesthesia was maintained with 1.2% halothane in oxygen. Muscle relaxation was re-established with tubocurarine 0.6 mg kg⁻¹. The lungs were mechanically ventilated with an Air Shields Ventimeter ventilator at a constant minute ventilation and a constant inspired gas mixture throughout the entire procedure. The ratio of inspiration to expiration was 1:3. Tidal volume was set at 10 ml kg⁻¹ and the rate was adjusted to keep PaCO₂ in the range 4–4.5 kPa. All measurements were made with the patients in the prone position. The head and torso were at the same level. The arms were outstretched and in front. The patients were supported with well-padded bolsters with the abdomen free.

A 20-gauge Teflon catheter was placed in the radial artery and a 5F balloon-tipped catheter was floated into a pulmonary artery through an arm vein for the recording of pressures and the collection of blood samples. Pressure recordings were made using a Hewlett-Packard monitoring system. Transducers were zeroed at the heart level.

Measurements, which included mean arterial pressure, mean pulmonary artery pressure, pulmonary capillary wedge pressure, cardiac output, arterial carbon dioxide tension (PaCO₂) and mixed expired carbon dioxide tension (PICO₂), were made after steady-state periods of at least 60 min. Measurements were made: during anaesthesia and surgery (control); during deliberately induced hypotension with nitroprusside (hypotension); after nitroprusside had been discontinued and arterial pressure had returned to the control value (recovery). Blood samples for blood-gas analysis were collected in heparinized glass syringes and analysed immediately in duplicate using an Instrumentation © The Macmillan Press Ltd 1982
Laboratories 313 blood-gas analyser. Cardiac output was measured by the dye-dilution technique using indocyanine green and a Beckman Cardiodensitometer. Mixed expired gas samples were collected in a 30-litre Douglas bag using a non-rebreathing one-way valve system. Mixed expired carbon dioxide tension was measured in triplicate by an Instrumentation Laboratories 313 gas analyser which was calibrated with three known carbon dioxide standards to obtain a three-point line.

Hypotension was induced with an i.v. infusion of 0.04% sodium nitroprusside. The infusion rate was regulated with a Holter infusion pump to achieve a 40% reduction in mean arterial pressure from the control value. During the operation these patients received an average of 500 ml of 5% dextrose in water and 2500 ml of Ringer's lactate solution. Of this total of 3 litres of fluid, 2 litres was given before induction of hypotension. Blood loss during surgery was estimated to be less than 500 ml. The average duration of the whole procedure was 3.5 h and the period of hypotension was 80 min.

Pulmonary deadspace to tidal volume ratio (VD/VT) was calculated using the standard formula:

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VD/VT = \frac{P_{ACO_2} - P_{ECO_2}}{P_{ACO_2}}
\]

Statistical analysis was done by paired t test. Values represent mean ± SEM. P < 0.05 was considered significant.

RESULTS

The results are summarized in table I. Mean arterial pressure was reduced by 40.8% during induced hypotension (P < 0.01) and returned to the control value during the recovery period. Mean pulmonary artery pressure and pulmonary capillary wedge pressure were essentially unchanged during the entire procedure. Cardiac index increased by 20% during induced hypotension (P < 0.05) and returned to control value during recovery. Neither the arterial carbon dioxide tension nor the pulmonary deadspace to tidal volume ratio changed in any of the three periods of measurement.

DISCUSSION

We have demonstrated that during anaesthesia and nitroprusside-induced hypotension the pulmonary deadspace to tidal volume ratio is not increased. Thus the contention that deliberately induced hypotension always causes an increase in VD/VT is refuted by this study.

VD/VT in the anaesthetized, intubated and mechanically ventilated prone patient was found in the present study to be 31%. It has been shown in the awake human subjects that changing from the upright to the supine position reduced VD/VT from 34% to 30% (Craig, Wahba and Don, 1971). We do not have data in the awake state, but our results are in agreement with published data obtained during anaesthesia (Nunn, 1977). Pulmonary disease may influence deadspace ventilation. Patients suffering from chronic lung disease, where there has been destruction of alveolar septa and capillaries, have an increased pulmonary deadspace because of emphysema and hypoperfusion (Donald et al., 1952). In such patients, a further increase in VD/VT occurs with the induction of anaesthesia (Pietak et al., 1975). The patients in our study had no pulmonary disease, and we expect no change in VD/VT with induction of anaesthesia.

In this study during deliberate hypotension induced with nitroprusside, mean arterial pressure was reduced from 76 ± 4 to 45 ± 22 mm Hg. VD/VT remained unchanged during hypotension. When hypotension is produced by haemorrhage, VD/VT can increase to 55–64% (Cournand et al., 1943; Gerst, Rattenberg and Holaday, 1959). This occurs

<table>
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<th>Table I. Haemodynamic and respiratory gas exchange values during nitroprusside-induced hypotension. Values represent mean ± SEM. Control v. hypotension, recovery v. hypotension:*P &lt; 0.01; †P &lt; 0.05</th>
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<tbody>
<tr>
<td>Control</td>
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<tr>
<td>Mean arterial pressure (mm Hg)</td>
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<td>Mean pulmonary artery pressure (mm Hg)</td>
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<td>Pulmonary capillary wedge pressure (mm Hg)</td>
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<td>Cardiac index (litre m⁻² min⁻¹)</td>
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<td>(P_{ACO_2}) (kPa)</td>
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<td>VD/VT (%)</td>
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because haemorrhage results in hypovolaemia and subsequent pulmonary hypoperfusion, thereby increasing $V_d/V_t$. Eckenhoff and his co-workers (1963) in their landmark paper showed that during nitrous oxide in halothane anaesthesia, deliberate hypotension with trimethaphan or pentolinium and a head-up tilt caused $V_d/V_t$ to increase to 75%. This observation was repeated by Askrog, Pender and Eckenhoff (1964). Anaesthesia, vasodilatation and pooling of blood in the periphery all combined to reduce venous return. Any decrease in filling pressure will result in diminished pulmonary blood flow and therefore will increase pulmonary deadspace (Nunn, 1977). Although in the above-mentioned studies pulmonary artery pressures were not measured, it is now well recognized that ganglionic blocking agents, like haemorrhage, bring about a reduction in filling pressures. Ganglionic blocking agents have been shown to decrease cardiac output probably by the same mechanisms (Scott et al., 1972). It has been shown that a decrease in cardiac output results in an increase in $V_d/V_t$ (Suwa, Hedley-Whyte and Bendixen, 1966).

In contrast to deliberate hypotension with ganglionic blocking agents, in our study with nitroprusside we observed no change in pulmonary artery pressures or pulmonary capillary wedge pressures. This was accomplished by giving adequate volume to fill the increased vascular capacity. The patients received a total of 3 litres of crystalloid fluid, 2 litres before hypotension and 1 litre during hypotension as has been our practice (Kambatta et al., 1978). This not only kept up filling pressures during hypotension, but it also permitted cardiac output to increase via afterload reduction and preload augmentation. In another study during nitrous oxide–halothane anaesthesia, intermittent positive pressure ventilation and deliberate hypotension with nitroprusside, an increase in $V_d/V_t$ from 49% to 54% was noted (Wildsmith, Drummond and MacRoe, 1975). However, neither pulmonary artery pressures nor cardiac output were measured and no attempt was made to hydrate the patients during nitroprusside-induced vasodilatation. It is thus quite possible that the observed increase in $V_d/V_t$ was the result of a decrease in filling pressures and cardiac output. Moreover, these patients had high $V_d/V_t$ values under anaesthesia, indicating possible pulmonary disease, which is often managed during anaesthesia with underhydration.

We conclude that deliberate hypotension, per se, does not cause an increase in $V_d/V_t$. Moreover, we believe that maintenance of adequate hydration, filling pressures and cardiac output during induced hypotension play a crucial role in maintaining the normal pulmonary deadspace.

REFERENCES


EFFET D'UNE HYPOTENSION PROVOQUEE PAR LE NITROPRUSSIATE DE SODIUM SUR L'ESPACE MORT PULMONAIRE

RESUME

Une hypotension provoquée au nitroprussiate entraîne une baisse de 40% de la pression artérielle moyenne, sans modification du rapport espace mort pulmonaire sur volume courant ($V_d/V_t$). Ceci est obtenu par une hydration correcte à la fois avant et pendant l'hypotension, comme le montrent les pressions de remplissage inchangées. Aussi nous avons montré qu'une hypotension délibérée n'augmente pas par elle-même $V_d/V_t$ et
que la vasodilataon peut démasquer une hypovolémie relative et être responsable de l’augmentation de Vd/Vt observée par de précédents chercheurs.

ZUSAMMENFASSUNG


SUMARIO

Una hipotension intencional con nitroprusiato tuvo por efecto una reducción del 40% de la presión arterial media, pero no provocó ningún cambio en la relación del periodo muerto pulmonar con el volumen respiratorio (Vd/Vt). Se consiguio este resultado mediante hidratación adecuada tanto antes como durante la hipotension, tal como indicado por las presiones de relleno constantes. Entonces, hemos demostrado que la hipotension intencional en sí no hace aumentar el Vd/Vt y que la vasodilatación puede causar un estado relativamente hipovolémico y dar origen al aumento del Vd/Vt observado por investigadores anteriores.