STUDIES OF ANAESTHESIA IN RELATION TO HYPERTENSION

IV: THE EFFECTS OF ARTIFICIAL VENTILATION ON THE CIRCULATION AND PULMONARY GAS EXCHANGES

C. PRYS-ROBERTS, P. FOËX, L. T. GREENE AND T. D. WATERHOUSE

SUMMARY

The cardiovascular responses, ventilation and pulmonary gas exchanges of 8 treated and 6 untreated hypertensive patients were studied during artificial ventilation under nitrous oxide/oxygen/relaxant anaesthesia, with and without halothane (1%). Mean arterial pressure (m.a.p.) fell to 69% and 62% of the awake values in the treated and untreated patients respectively during hypocapnia (mean $P_{aCO_2}$ 23 mm Hg) induced by IPPV under nitrous oxide anaesthesia. When halothane (1%) was added under the same conditions of IPPV, m.a.p. fell to 60% and 53% of the awake values respectively, as a result of reduced cardiac output (50% and 53%, respectively of awake values), whereas systemic vascular resistance was raised above the awake values in all patients. Electrocardiographic evidence of myocardial ischaemia was observed during these periods of arterial hypotension in 50% of the treated patients and in all the untreated patients. Increased alveolar-arterial $P_{aO_2}$ differences during IPPV were due to desaturation of mixed venous blood associated with raised arteriovenous oxygen content differences. Pulmonary venous admixture did not change significantly during the course of anaesthesia with IPPV and recovery. $Vd/Vt$ increased during spontaneous ventilation following induction of anaesthesia, but decreased significantly below awake values during IPPV, returning to control values in the postoperative period.

Previous studies in this series have shown that during nitrous oxide-halothane anaesthesia with spontaneous ventilation, patients with untreated or inadequately treated hypertension were prone to develop arterial hypotension associated with myocardial ischaemia (Prys-Roberts, Meloche and Foëx, 1971; Prys-Roberts et al., 1971). Premedicated, normotensive patients in a slightly younger age group (mean age 46 years) were less prone to arterial hypotension during spontaneous ventilation, but hypocapnia induced by artificial ventilation caused arterial hypotension due to low cardiac output (Theye, Milde and Michenfelder, 1966; Prys-Roberts et al., 1967, 1968). By contrast, Cullen, Eger and Gregory (1969) found that hypocapnia during artificial ventilation caused lesser falls of cardiac output in younger unpremedicated volunteers anaesthetized with cyclopropane.

It is thus clear that the patterns of cardiovascular response to anaesthesia and artificial ventilation cannot be regarded as typical for all patients, but only for specific groups of patients, characterized by at least two factors: their ages and any pre-existing cardiovascular disease. We describe in this paper the cardiovascular responses of treated and untreated hypertensive patients, and their pulmonary gas exchanges during anaesthesia and artificial ventilation. We also describe the cardiovascular responses and pulmonary gas exchanges of a normotensive subject who, by virtue of a permanent tracheostomy, was artificially ventilated at a constant tidal volume and frequency before, during and after anaesthesia.

METHODS

Clinical material.

Nineteen patients were studied during artificial ventilation, but since a complete set of measurements conforming to the protocol described below was only achieved in 14 patients, they alone form the material for this paper. Their medical and anthropometric details are shown in table I. Classification of these patients into treated and untreated hypertensive
Table I. Anthropometric and medical data of patients included in the study. Patient 2153 was studied again, 3 years after the first occasion, and as a treated hypertensive patient was renumbered 2156. Patients marked with an asterisk received practolol before intubation.

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>age</th>
<th>Weight (kg)</th>
<th>Body surface area (m²)</th>
<th>Arterial pressure (mm Hg)</th>
<th>Group</th>
<th>Therapy</th>
<th>Surgical procedure (medical complications)</th>
<th>Premedication</th>
<th>Induction agent</th>
</tr>
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<tbody>
<tr>
<td>2101</td>
<td>F</td>
<td>63</td>
<td>82.7</td>
<td>1.99</td>
<td>162/72</td>
<td>T</td>
<td>AMD</td>
<td>Parotidectomy</td>
<td>---</td>
<td>Thio.</td>
</tr>
<tr>
<td>2114</td>
<td>F</td>
<td>27</td>
<td>66.0</td>
<td>1.78</td>
<td>208/112</td>
<td>T</td>
<td>AMD, B, D-K</td>
<td>Tubal ligation</td>
<td>Pa 20</td>
<td>H 0.4</td>
</tr>
<tr>
<td>2121</td>
<td>F</td>
<td>69</td>
<td>46.5</td>
<td>1.39</td>
<td>204/112</td>
<td>U</td>
<td></td>
<td>Hysterectomy</td>
<td>---</td>
<td>NLA.</td>
</tr>
<tr>
<td>2126</td>
<td>F</td>
<td>44</td>
<td>79.0</td>
<td>1.80</td>
<td>170/92</td>
<td>T</td>
<td>Deb, D-K</td>
<td>Hysterectomy</td>
<td>---</td>
<td>NLA.</td>
</tr>
<tr>
<td>2135</td>
<td>F</td>
<td>54</td>
<td>49.5</td>
<td>1.43</td>
<td>281/143</td>
<td>U</td>
<td></td>
<td>Hysterectomy</td>
<td>Pa 20</td>
<td>H 0.4</td>
</tr>
<tr>
<td>2138</td>
<td>F</td>
<td>69</td>
<td>69.5</td>
<td>1.72</td>
<td>184/100</td>
<td>U</td>
<td></td>
<td>Hysterectomy</td>
<td>---</td>
<td>Prop.</td>
</tr>
<tr>
<td>2140</td>
<td>F</td>
<td>65</td>
<td>77.8</td>
<td>1.77</td>
<td>195/100</td>
<td>U</td>
<td></td>
<td>Hysterectomy</td>
<td>---</td>
<td>Metho.</td>
</tr>
<tr>
<td>2146</td>
<td>F</td>
<td>65</td>
<td>58.2</td>
<td>1.60</td>
<td>190/100</td>
<td>T</td>
<td>Guan</td>
<td>Vaginal hysterectomy</td>
<td>Pa 10</td>
<td>H 0.2</td>
</tr>
<tr>
<td>2150</td>
<td>M</td>
<td>66</td>
<td>49.0</td>
<td>1.46</td>
<td>235/112</td>
<td>U</td>
<td></td>
<td>Mandibulotomy, Partial Glossectomy, (chronic bronchitis)</td>
<td>---</td>
<td>Thio.</td>
</tr>
<tr>
<td>2152</td>
<td>F</td>
<td>66</td>
<td>68.5</td>
<td>1.67</td>
<td>150/90</td>
<td>T</td>
<td>Res, D-K</td>
<td>Parotidectomy, block dissection of neck (CVA after previous anaesthesia)</td>
<td>---</td>
<td>* Thio.</td>
</tr>
<tr>
<td>2153</td>
<td>F</td>
<td>28</td>
<td>91.2</td>
<td>1.89</td>
<td>240/140</td>
<td>U</td>
<td></td>
<td>Vaginal hysterectomy</td>
<td>P 100</td>
<td>A 0.6</td>
</tr>
<tr>
<td>2156</td>
<td>F</td>
<td>31</td>
<td>76.3</td>
<td>1.77</td>
<td>150/90</td>
<td>T</td>
<td>B, D-K</td>
<td>Dental clearance</td>
<td>Pa 20</td>
<td>H 0.4</td>
</tr>
<tr>
<td>2157</td>
<td>F</td>
<td>66</td>
<td>75.0</td>
<td>1.74</td>
<td>200/90</td>
<td>T</td>
<td>Res, D-K</td>
<td>Hysterectomy</td>
<td>---</td>
<td>* Thio.</td>
</tr>
<tr>
<td>2158</td>
<td>F</td>
<td>46</td>
<td>65.5</td>
<td>1.70</td>
<td>150/90</td>
<td>T</td>
<td>Guan, D-K</td>
<td>Vaginal hysterectomy (left ventricular hypertrophy)</td>
<td>---</td>
<td>* CT 1341</td>
</tr>
</tbody>
</table>

Key: AMD, methyldopa; B, bethanidine; Guan, guanethidine; Deb, debrisoquine; Res, reserpine; D-K, diuretic with slow release potassium. Pa, papaveretum; P, pethidine; H, hyoscine; A, atropine. Doses in mg.

The normotensive patient (2401), aged 49, had suffered from severe myasthenia gravis for 8 years, but was otherwise in good health. As a result of persistent and severe dysphagia, a permanent tracheostomy and gastrostomy had been performed 7 years before the study. During the day, he was accustomed to treat his myasthenia with neostigmine (2.5 mg x 4 i.m.), but each night he connected himself to an East-Radcliffe ventilator, and slept under the influence of hypocapnia maintained by IPPV.

Procedure.

The general protocol and the specific methods used for cardiovascular measurements and Riley-Cournand analysis follow those described in detail in the previous papers (Prys-Roberts, Meloche and Foëx, 1971; Foëx, Meloche and Prys-Roberts, 1971), but modifications of technique appropriate to measurements during artificial ventilation are described below.

A complete set of cardiovascular measurements and Riley-Cournand analysis was initially obtained in the conscious, unpremedicated patient before induction of anaesthesia (stage A). Anaesthesia was induced with a variety of agents (table I) most commonly with 2.5% thiopentone 100–200 mg, or by neuroleptanalgesia (phenoperidine 1.0–1.6 mg and droperidol 4–6 mg) and steady-state maintenance anaesthesia with 1% halothane vaporized in 70% nitrous oxide and 30% oxygen was achieved before endotracheal intubation under muscle relaxation by suxamethonium 50–100 mg. Five patients (marked with an asterisk in table I) were treated with practolol 15–20 mg prior to intubation. Their cardiovascular responses to this agent and the subsequent modification of their response to intubation will be described in a subsequent communication (Prys-Roberts and Foëx, in preparation). Suffice it to say that in those
patients who received practolol prior to intubation, the artificial ventilation studies of the sequence (stages C and D) were carried out after the completion of surgery, an interval of between 60 and 90 min having elapsed since the administration of practolol. Following an intravenous dose of practolol 20 mg, it is unlikely that there would be any significant beta-adrenergic blockade at this time, since the blood level of practolol would be less than 0.3 μg/ml, a level which would be associated with less than 10% inhibition of tachycardia due to isoprenaline infusion (Fitzgerald, J. D., 1971, personal communication). After intubation, a further complete set of measurements was made during spontaneous ventilation of the same gas mixture (stage B). During surgery, all the patients were paralysed with pancuronium 4-8 mg and artificial ventilation was maintained with an East-Radcliffe ventilator, the pressure applied to the bellows being adjusted to provide a tidal volume of approximately 10 ml/kg at a frequency of 13/min. The concentration of halothane used during surgery was varied to suit the operative requirements and the condition of the patient, but a fixed concentration of 1% was used during the period of measurements. Two sets of measurements during artificial ventilation were made, before surgery in 4 patients, during and after surgery in the remainder. The first set of measurements in every patient was made during artificial ventilation with 1% halothane and nitrous oxide and oxygen (stage C), followed by a second set of measurements (stage D) not less than 20 min after withdrawal of halothane. The neuromuscular blockade was then reversed with neostigmine 2.5 mg given together with atropine 1.2 mg, and in some patients a further set of cardiovascular measurements was made during spontaneous ventilation. The patients were extubated before the final withdrawal of anaesthesia, and the final measurements (stage E) were made not less than 1 hour after the patients had recovered consciousness, and were able to converse rationally.

The ventilator settings used for patient 2401 during the study were those to which he was accustomed every night. He was studied before, during and after anaesthesia for surgery to shorten his tongue and introduce fascial slings in his face to prevent continuous dribbling of saliva. On the morning of the study, neostigmine was withheld and the same pattern and volume of ventilation was continued up to the beginning of the study. No premedication was given. Arterial and right ventricular catheters were introduced under local analgesia. Measurements were first made during IPPV with air, followed by a second set of measurements during ventilation with 100% oxygen. Anaesthesia was induced by adding halothane to the inspired oxygen, to achieve a steady-state of anaesthesia at an inspired halothane concentration of 1%. The measurements made before surgery were repeated at the same anaesthetic concentration after surgery had been completed. Finally, 1 hour after withdrawal of halothane, the measurements were repeated firstly during IPPV with 100% oxygen, and then with air.

Methods.
During the period of artificial ventilation, expired gas was collected for a 2-min period, from the expiratory port of the East-Radcliffe ventilator the circuitry of which had been checked to be effectively free from internal leaks and valve slip. Inspired gas was collected from a tap in the inspiratory port of the ventilator. The measured volume of gas collected during IPPV was corrected for the effects of gas compression in the ventilator bellows and tubing (Cooper, 1967a; Mushin, et al., 1969). In all other respects, the analytical methods, the calculations, and the statistical treatment followed the methods described in detail by Foex, Meloche and Prys-Roberts (1971).

RESULTS
Cardiovascular measurements.
Values of haemodynamic variables and their statistical relationships (stages A–E) for 8 treated and 6 untreated patients are shown in table II and figure 1. During stage A, the values for mean arterial pressure, cardiac output and systemic vascular resistance were all similar to those during the same stage in the study described by Prys-Roberts, Meloche and Foex (1971), although the higher mean arterial pressures in the untreated group in the present study reflect high values measured in two patients (2135, 2153). There was a significant difference (P<0.05) between the treated and untreated groups for the values of systolic and mean arterial pressures, and of systemic vascular resistance. During spontaneous ventilation (stage B) a significant fall of mean arterial pressure occurred in both groups as a result of reductions in cardiac output and systemic vascular resistance, the magnitude of the changes being comparable with those found in the previous study. During artificial ventilation with halothane (stage C), the values of arterial pressures and cardiac output were the lowest recorded in each patient, the mean
TABLE II. Summary of haemodynamic findings in 8 treated hypertensive patients (Group A) and 6 untreated hypertensive patients (Group B), during the five stages of the study.

A, awake before anaesthesia; B, spontaneous ventilation (N\textsubscript{2}O, O\textsubscript{2}, halothane 1%); C, IPPV (N\textsubscript{2}O, O\textsubscript{2}, halothane 1%); D, IPPV (N\textsubscript{2}O, O\textsubscript{2}); E, awake after anaesthesia. Mean values shown for both groups, with SD in parentheses. Significances in [ ] are the levels determined by unpaired t-tests between mean values for the same stage in the two groups. Significance levels indicated in the r-matrix are for paired two-tailed t-tests between mean values for each stage of the study.

<table>
<thead>
<tr>
<th>Stage</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>173 (19)</td>
<td>108 (21)</td>
<td>95 (33)</td>
<td>110 (26)</td>
<td>160 (16)</td>
</tr>
<tr>
<td>Diastolic arterial pressure (mm Hg)</td>
<td>89 (14)</td>
<td>61 (16)</td>
<td>59 (24)</td>
<td>67 (19)</td>
<td>81 (12)</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>119 (16)</td>
<td>77 (16)</td>
<td>72 (28)</td>
<td>82 (22)</td>
<td>109 (13)</td>
</tr>
<tr>
<td>Cardiac output (l./min/70 kg)</td>
<td>5.14 (0.64)</td>
<td>4.15 (0.62)</td>
<td>2.76 (0.83)</td>
<td>3.27 (0.79)</td>
<td>4.65 (0.69)</td>
</tr>
<tr>
<td>Systemic vascular resistance (dyne.sec.cm\textsuperscript{-5})</td>
<td>1800 (284)</td>
<td>1412 (247)</td>
<td>2012 (705)</td>
<td>1931 (593)</td>
<td>1845 (424)</td>
</tr>
<tr>
<td>Heart rate (beat/min)</td>
<td>67 (18)</td>
<td>67 (17)</td>
<td>62 (12)</td>
<td>62 (11)</td>
<td>68 (15)</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>82 (21)</td>
<td>65 (14)</td>
<td>45 (11)</td>
<td>55 (13)</td>
<td>72 (19)</td>
</tr>
<tr>
<td>Right ventricular end-diastolic pressure (mm Hg)</td>
<td>2.6 (1.2)</td>
<td>5.0 (1.4)</td>
<td>4.6 (2.2)</td>
<td>3.0 (1.4)</td>
<td>2.5 (1.0)</td>
</tr>
</tbody>
</table>

GROUP B

<table>
<thead>
<tr>
<th>Stage</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>211 (39)</td>
<td>125 (20)</td>
<td>96 (21)</td>
<td>120 (21)</td>
<td>182 (17)</td>
</tr>
<tr>
<td>Diastolic arterial pressure (mm Hg)</td>
<td>105 (23)</td>
<td>70 (19)</td>
<td>57 (11)</td>
<td>68 (9)</td>
<td>85 (16)</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>144 (36)</td>
<td>92 (22)</td>
<td>77 (11)</td>
<td>90 (13)</td>
<td>125 (19)</td>
</tr>
<tr>
<td>Cardiac output (l./min 70 kg)</td>
<td>5.12 (0.60)</td>
<td>4.04 (0.56)</td>
<td>2.57 (0.88)</td>
<td>3.22 (0.84)</td>
<td>4.89 (0.55)</td>
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<tr>
<td>Systemic vascular resistance (dyne.sec.cm\textsuperscript{-5})</td>
<td>2493 (731)</td>
<td>1914 (611)</td>
<td>2596 (1031)</td>
<td>2422 (921)</td>
<td>2213 (566)</td>
</tr>
<tr>
<td>Heart rate (beat/min)</td>
<td>86 (14)</td>
<td>76 (25)</td>
<td>65 (11)</td>
<td>73 (18)</td>
<td>77 (13)</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>58 (20)</td>
<td>53 (19)</td>
<td>34 (14)</td>
<td>43 (15)</td>
<td>60 (17)</td>
</tr>
<tr>
<td>Right ventricular end-diastolic pressure (mm Hg)</td>
<td>1.8 (1.7)</td>
<td>7.8 (2.7)</td>
<td>5.3 (2.1)</td>
<td>4.3 (1.7)</td>
<td>2.5 (1.9)</td>
</tr>
</tbody>
</table>

Mean pulmonary arterial pressure (mm Hg) | 13 | 20 | 13 | 15 | 12 |

Significance levels: * P<0.05; ** P<0.01; P<0.001.
cardiac output in treated and untreated patients being 53% and 50% respectively of the control values, while the systemic vascular resistance was higher than at any other stage during the study. The low cardiac outputs reflected severe reductions in stroke volume despite elevation of right ventricular end-diastolic or right atrial pressures. Although the actual values of cardiac output were lower in the untreated patients, there was no significant difference between the two groups either during this or the subsequent stage D. Following withdrawal of halothane, cardiac output increased in every patient,
although with respect to stage C, the differences between the mean values were statistically significant only for the treated hypertensive group and for the two groups combined.

Following recovery from anaesthesia, arterial pressures and cardiac output were marginally lower than the preanaesthetic values, although the difference was only significant in respect of cardiac output in the treated hypertensive group. No patients in this series exhibited overt shivering during recovery, and the commonest pattern of events was that arterial pressures and cardiac output increased to the levels measured at stage E within 5 min of the withdrawal of anaesthesia. In 5 treated hypertensive patients, the values of cardiac output measured within 5–10 min of withdrawal of anaesthesia were higher (3–14%) than those measured during stages A or E, and were associated with higher arteriovenous oxygen content differences. Such a pattern indicated that the derived values of oxygen uptake were higher than the predicted basal values, which would be consistent with increased muscular activity of a lower degree than would be evident as overt shivering.

**Electrocardiographic changes.**

During the period of artificial ventilation with halothane in nitrous oxide (stage C), significant T-wave depression or inversion (0.2 mV or more) was evident in more than one lead in 4 of 8 treated patients, 2 of whom also showed S-T segment depression. By contrast, all six untreated hypertensive patients showed T-wave depression or inversion, 3 of whom also showed marked changes in the S-T segment, and one of whom developed nodal rhythm at a rate of 42 beats/min (fig. 3).

Following withdrawal of halothane, T-wave depression persisted during IPPV with nitrous oxide in 4 untreated patients only, 1 of whom the changes were partially reversed by elevating Pco₂ to normal levels while maintaining the volume of ventilation (fig. 2). T-wave inversion persisted following recovery from anaesthesia in 1 patient only (2153), in whom the most severe T-wave inversion had occurred during IPPV (fig. 3).

**Pulmonary gas exchanges.**

The change in ventilation, pulmonary gas exchanges and blood-gas variables were studied in 12 patients (6 treated and 6 untreated) whose data are combined and summarized in table III. The mean values for minute, tidal and alveolar ventilation in the conscious patients (stage A) were lower than the mean values during the same stage in the previous study (Foëx et al., 1971); the difference can be accounted for by the values in 5 patients who were premedicated with opiates and 1 patient (2150) who had chronic obstructive airway disease. During spontaneous ventilation (stage B), the reductions in ventilation volumes were proportionally similar to those measured in the previous study. During artificial ventilation (stages C and D), the mean tidal volume achieved was 10.8 ml/kg (SD 1.5 ml/kg), and alveolar ventilation was nearly double the value in the conscious patient. Ventilation in the postoperative period was essentially the same as in the preanaesthesia stage.

As in the previous study the ratio Vd/Vt increased during stage B, though Vb was decreased as a result of endotracheal intubation. During both stages of IPPV Vd/Vt was significantly lower than at all other stages, and Vd was raised. The postoperative values of Vd and Vd/Vt were not significantly different from the values measured in the preanaesthesia stage.

Mean values of oxygen uptake (Vo₂) during both the preanaesthesia (119 ml STPD/min/m²) and postoperative periods (120 ml STPD/min/m²) were close to the basal levels (114 ml STPD/min/m²; SD 5) predicted from the data of Boothby, Berkson and Dunn (1936). During all three stages under anaesthesia, Vo₂ was decreased and remained constant at about 75% of the preanaesthesia level, whereas carbon dioxide elimination (Vco₂) was decreased by 23% during stage B, but by only 10% during the two stages of IPPV. Reflecting the disproportionate reduction of cardiac output relative to Vo₂, CaO₂-CVO₂ was significantly raised during both stages of IPPV, the highest values being recorded during the administration of halothane (fig. 4).

Comparisons of arterial Po₂ at all the different stages were not strictly meaningful since the inspired oxygen concentration (FiO₂) was higher during anaesthesia. During air breathing in the preanaesthesia period the mean PAO₂ was 76 mm Hg reflecting an alveolar–arterial Po₂ difference (PAO₂–PAO₂) of 26 mm Hg, equivalent to a pulmonary venous admixture (Qs/Qt) of 12%. Mean postoperative PAO₂ was 73 mm Hg reflecting a PAO₂–PAO₂ of 27 mm Hg, equivalent to a Qs/Qt of 13%: a fall of more than 10 mm Hg in PAO₂ between stages A and E occurred in only 2 patients, and was due to increased PAO₂–PAO₂ in only 1 of them. During anaesthesia FiO₂ was maintained at about 0.3, and the mean values of PAO₂ were thus maintained at a level slightly below 100 mg Hg, there being no significant dif-
Fig. 2. Electrocardiographic evidence of myocardial ischaemia induced by hypocapnia (Pa\textsubscript{\textit{CO}}\textsubscript{2} 20.5 mm Hg) during IPPV in patient 2126. The T-wave inversion was reversed when Pa\textsubscript{\textit{CO}}\textsubscript{2} was elevated to 40.8 mm Hg. This was achieved by maintaining the same volume and frequency of ventilation from an East-Radcliffe ventilator, utilizing a circle system without a soda-lime absorber, and a fresh gas flow of 4 l./min. Awake spontaneous Pco\textsubscript{2}=33 mm Hg.

Fig. 3. Development of myocardial ischaemia associated with nodal bradycardia as a result of adding halothane (1%) to the inspired gas mixture in patient 2153. The second panel shows depression of the S-T segment and the T-waves occurring during arterial hypotension under nitrous oxide/oxygen/relaxant anaesthesia. Halothane (1%) was added for a brief period, but was withdrawn following the development of nodal bradycardia leading to further arterial hypotension and deep T-wave inversion (third panel). Ten minutes after withdrawal of halothane, T-wave inversion was still present even though arterial pressure had risen with the return to sinus rhythm, albeit with a prolonged P-R interval (fourth panel).
<table>
<thead>
<tr>
<th>Stage</th>
<th>n</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>Significance matrix</th>
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<tr>
<td></td>
<td></td>
<td>A-B</td>
<td>A-C</td>
<td>A-D</td>
<td>B-C</td>
<td>B-D</td>
<td>B-E</td>
</tr>
<tr>
<td>Minute volume (Vt) (l/min) BTPS</td>
<td>12</td>
<td>6.16</td>
<td>4.20</td>
<td>9.11</td>
<td>9.23</td>
<td>6.59</td>
<td>A-B *** A-C *** A-D *** B-C *** B-D *** B-E ***</td>
</tr>
<tr>
<td>Tidal volume (Vt) (ml) BTPS</td>
<td>12</td>
<td>444</td>
<td>224</td>
<td>674</td>
<td>684</td>
<td>427</td>
<td>A-B *** A-C *** A-D *** B-C *** B-D *** B-E ***</td>
</tr>
<tr>
<td>Alveolar ventilation (Vt) (l/min) BTPS</td>
<td>12</td>
<td>3.68</td>
<td>2.23</td>
<td>6.11</td>
<td>6.07</td>
<td>3.69</td>
<td>A-B *** A-C *** A-D *** B-C *** B-D *** B-E ***</td>
</tr>
<tr>
<td>Total dead space (Vd) (ml) BTPS</td>
<td>12</td>
<td>174</td>
<td>102</td>
<td>221</td>
<td>235</td>
<td>169</td>
<td>A-B *** A-C *** A-D *** B-C *** B-D *** B-E ***</td>
</tr>
<tr>
<td>Vt/Vr (%)</td>
<td>12</td>
<td>40</td>
<td>47</td>
<td>33</td>
<td>34</td>
<td>42</td>
<td>A-B * A-C * B-C *** B-D *** B-E *</td>
</tr>
<tr>
<td>Va/Qc</td>
<td>12</td>
<td>0.75</td>
<td>0.58</td>
<td>2.39</td>
<td>1.96</td>
<td>0.81</td>
<td>A-B n.s. A-C *** A-D *** B-C *** B-D *** B-E ***</td>
</tr>
<tr>
<td>Oxygen uptake (Vo2) (ml/min/m2 STPD)</td>
<td>12</td>
<td>119</td>
<td>91</td>
<td>87</td>
<td>87</td>
<td>120</td>
<td>A-B *** A-C *** A-D *** B-C n.s. B-D n.s. B-E n.s.</td>
</tr>
<tr>
<td>CO2 elimination (Vco2) (ml/min/m2 STPD)</td>
<td>9</td>
<td>90</td>
<td>69</td>
<td>80</td>
<td>81</td>
<td>95</td>
<td>A-B * A-C * B-C * A-D n.s. B-D * B-E *</td>
</tr>
<tr>
<td>Respiratory exchange ratio</td>
<td>9</td>
<td>0.78</td>
<td>0.77</td>
<td>0.93</td>
<td>0.94</td>
<td>0.80</td>
<td>A-B n.s. A-C * B-C * A-D * B-D * B-E *</td>
</tr>
<tr>
<td>P02 (mm Hg) Pao2 (mm Hg)</td>
<td>12</td>
<td>149 (2)</td>
<td>223 (12)</td>
<td>222 (13)</td>
<td>226 (11)</td>
<td>149 (2)</td>
<td>A-B *** A-C *** A-D *** B-C *** B-D *** B-E ***</td>
</tr>
<tr>
<td>Venous admixture (Qs/Qt) (%)</td>
<td>12</td>
<td>26</td>
<td>68</td>
<td>99</td>
<td>99</td>
<td>27</td>
<td>A-B *** A-C *** A-D *** B-C *** B-D *** B-E ***</td>
</tr>
<tr>
<td>Cao2-Cvo2 (ml/100 ml)</td>
<td>12</td>
<td>4.06</td>
<td>3.78</td>
<td>6.09</td>
<td>5.02</td>
<td>4.55</td>
<td>A-B n.s. A-C * B-C * A-D * B-D * B-E *</td>
</tr>
<tr>
<td>Pao2 (mm Hg)</td>
<td>12</td>
<td>76</td>
<td>102</td>
<td>93</td>
<td>99</td>
<td>73</td>
<td>A-B *** A-C *** A-D *** B-C n.s. B-D n.s. B-E n.s.</td>
</tr>
<tr>
<td>Pco2 (mm Hg)</td>
<td>12</td>
<td>38.7</td>
<td>50.2</td>
<td>22.7</td>
<td>23.8</td>
<td>40.6</td>
<td>A-B *** A-C *** A-D *** B-C *** B-D *** B-E ***</td>
</tr>
<tr>
<td>Pvco2-Paco2 (mm Hg)</td>
<td>10</td>
<td>4.6</td>
<td>5.3</td>
<td>7.6</td>
<td>6.9</td>
<td>4.3</td>
<td>A-B n.s. A-C ** B-C ** A-D * B-D * B-E *</td>
</tr>
<tr>
<td>Pvco2 (mm Hg)</td>
<td>10</td>
<td>45.3</td>
<td>57.9</td>
<td>30.3</td>
<td>30.7</td>
<td>44.6</td>
<td>A-B *** A-C *** A-D *** B-C *** B-D *** B-E ***</td>
</tr>
<tr>
<td>Pvco2-Paco2 (mm Hg)</td>
<td>10</td>
<td>4.6</td>
<td>5.3</td>
<td>7.6</td>
<td>6.9</td>
<td>4.3</td>
<td>A-B n.s. A-C ** B-C ** A-D * B-D * B-E *</td>
</tr>
</tbody>
</table>
ANAESTHESIA IN RELATION TO HYPERTENSION

Fig. 4. Interrelationships between cardiac output, oxygen uptake and carbon dioxide elimination, and \( C_\text{ao2} - C_\text{vo2} \) during the five stages of the study. Note that the stages are not depicted in the order described in the text.

Differences between the values at the three stages (B, C and D). At the constant levels of \( F_\text{tO2} \) maintained in each patient during anaesthesia, \( P_{\text{aO2}} - P_{\text{ao2}} \) was significantly lower during stage B, when the mean \( P_{\text{aco2}} \) was 50 mm Hg, then during hypopnoea induced by IPPV (stages C and D). The increased \( P_{\text{aO2}} - P_{\text{ao2}} \) during IPPV was related to the decreased mixed venous oxygen content, since the calculated \( Q_\text{s}/Q_\text{t} \) was not increased.

Patient 2401. The results of the study of this patient are shown in table IV, and are largely self-explanatory. Cardiac output decreased by 11% following the change of inspired gas from air to 100% oxygen. During steady-state anaesthesia with halothane in oxygen before surgery, cardiac output fell to 75% of the air-breathing control value and largely accounted for the 66% fall in the mean arterial pressure. Following surgery, mean arterial pressure had risen to 90% of the awake control, but cardiac output had fallen further to 66%, returning to within 5% of the awake control 1 hour after withdrawal of anaesthesia. The fall in cardiac output was associated with a decrease in myocardial contractility as reflected in the values of \( \text{Max right ventricular } \frac{dP}{dt} \) and the derived value of \( \text{Max } \frac{dP}{dt}/\text{IP} \) where IP is the difference between right ventricular end-diastolic pressure and the instantaneous pressure developed at the time of Max \( dP/dt \) (Gersh et al., 1970). During anaesthesia, oxygen uptake fell to 85% of the awake value, thus the proportionally greater fall in cardiac output was reflected in the increased \( C_\text{ao2} - C_\text{vo2} \), the latter being associated with the increased \( P_{\text{aO2}} - P_{\text{ao2}} \) at a constant \( P_{\text{tO2}} \). Pulmonary venous admixture and \( VD/VT \) remained effectively unaltered throughout the study.

DISCUSSION

In many respects the cardiovascular responses of hypertensive patients to the induction and maintenance of anaesthesia with artificial ventilation, were qualitatively similar to those which have been described for normotensive patients of a wide range of ages. Cardiac output in the conscious hypertensive patient is similar to that in normotensive patients of the same age (Frohlich, Tarazi and Dustan, 1969; Prys-Roberts, Meloche and Foex, 1971), and the essential difference between hypertensive and normotensive patients is that the high arterial pressures in the former group reflect their high systemic vascular resistance. Folkow (1971) has recently emphasized that the high vascular resistance in hypertensive patients is due to "thickening" or adaptive hypertrophy of the arterial media to such an extent that the lumen diameter of the vessel is reduced without alteration of the resting length of the vascular smooth muscle. Thus the high vascular resistance is not the result of abnormal vasoconstriction, but of structural changes in the vessel wall. This implies that when the arterioles of hypertensive patients are maximally vasodilated, the resistance they offer to blood flow is greater than that of their normotensive counterpart, yet maintaining a complete range of vasodilatation/vasoconstriction within the confines of a different resistance/smooth muscle shortening relationship (Sivertsson, 1970) (fig. 5). The importance of this relationship lies in the increased "reactivity" of the vessels of hypertensive patients to stimuli which cause shortening of the vascular
TABLE IV. Summary of measurements in Patient 2401 before, during and after anaesthesia with 1% halothane. Patient's weight 62 kg. Haemoglobin concentration 12.9 g/100 mL. Values shown are means of duplicate estimates of each variable.

<table>
<thead>
<tr>
<th></th>
<th>Awake</th>
<th>Anaesthetized</th>
<th>Awake</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Air</td>
<td>Oxy</td>
<td></td>
</tr>
<tr>
<td>Minute volume (l/min)</td>
<td>13.55</td>
<td>13.67</td>
<td>13.72</td>
</tr>
<tr>
<td>Tidal volume (ml)</td>
<td>903</td>
<td>911</td>
<td>903</td>
</tr>
<tr>
<td>FIO2 (mm Hg)</td>
<td>151</td>
<td>724</td>
<td>717</td>
</tr>
<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>125</td>
<td>117</td>
<td>90</td>
</tr>
<tr>
<td>Diastolic arterial pressure (mm Hg)</td>
<td>83</td>
<td>74</td>
<td>51</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>97</td>
<td>88</td>
<td>64</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>5.17</td>
<td>4.62</td>
<td>3.87</td>
</tr>
<tr>
<td>Systemic vascular resistance (dyne.sec.cm⁻⁵)</td>
<td>1415</td>
<td>1488</td>
<td>1270</td>
</tr>
<tr>
<td>Heart rate (beat/min)</td>
<td>92</td>
<td>87</td>
<td>78</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>57</td>
<td>53</td>
<td>50</td>
</tr>
<tr>
<td>RV Max (dP/dt/IP)</td>
<td>40</td>
<td>37</td>
<td>22</td>
</tr>
<tr>
<td>RV Max (dP/dt/IP)</td>
<td>425</td>
<td>395</td>
<td>155</td>
</tr>
<tr>
<td>PAO₂ (mm Hg)</td>
<td>130</td>
<td>708</td>
<td>698</td>
</tr>
<tr>
<td>Paco₂ (mm Hg)</td>
<td>88</td>
<td>617</td>
<td>589</td>
</tr>
<tr>
<td>Paco₂ (mm Hg)</td>
<td>18.6</td>
<td>18.0</td>
<td>15.7</td>
</tr>
<tr>
<td>PH (arterial)</td>
<td>7.558</td>
<td>7.565</td>
<td>7.580</td>
</tr>
<tr>
<td>CaO₂-CO₂ (ml/100 ml)</td>
<td>4.45</td>
<td>4.25</td>
<td>5.01</td>
</tr>
<tr>
<td>Qs/Qt (%)</td>
<td>9</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Vb/Vt (%)</td>
<td>26</td>
<td>29</td>
<td>28</td>
</tr>
<tr>
<td>Vo₂ (mlsSPD/min)</td>
<td>230</td>
<td>197</td>
<td>194</td>
</tr>
</tbody>
</table>

Fig. 5. Diagrammatic representation of the relationship between flow resistance and smooth muscle shortening (tone) in the resistance vessels of normotensive (N) and hypertensive (H) animals. The position of curve H implies that an increase in wall thickness of the vessel has occurred with consequent decrease in lumen, even at maximal dilatation. Because of the increased wall thickness/lumen ratio, curve H is steeper than curve N throughout its course, and it follows that such a vessel would exhibit increased "vascular" reactivity, without implying a change in smooth muscle shortening. At normal smooth muscle tone, flow resistance would be increased in the hypertensive vessel (A) compared with the normotensive vessel (O), yet the hypertensive vessel is still capable of constriction or dilatation, with the appropriate change in flow resistance, along curve H. (Folkow, 1971; reproduced from Clin. Sci. (1971), 41, 1, by kind permission of the author and editor.)

smooth muscle, e.g. the effects of sympathetic nerve stimulation or catecholamine infusions, changes in transmural pressure, and the effects of changes in carbon dioxide levels or hypoxia. By "reactivity", we imply that a stimulus which causes a given degree of smooth muscle shortening elicits a greater change in resistance as reflected in the greater slope of the response in the hypertensive patient in figure 5. In the context of the present study, the vasoconstrictor effects of elevated pH and reduced PaCO₂ were much more marked in these hypertensive patients than the effects of the same stimulus in the normotensive patient (Prys-Roberts et al., 1967, 1968). The increased systemic vascular resistance as a result of hypocapnia alters the impedance to left ventricular ejection, and since no marked changes in left ventricular filling volume occur, and since myocardial contractility is not significantly altered during hypocapnia, the ejection fraction (stroke volume) decreases (Prys-Roberts and Foex, 1971). Thus the reduction of cardiac output during hypocapnia does not reflect direct depression of the ventricular myocardium by low PaCO₂ (Ng, Levy and Zieske, 1967), but a failure of the myocardium which is already depressed by nitrous oxide anaesthesia to eject against a raised vascular impedance (Gersh, 1970; Gersh et al., 1970). Further depression of myocardial contractility by the addition of halothane to the nitrous oxide reduces stroke volume further without a significant change of systemic vascular resistance (Gersh et al., 1970).
During both stages of artificial ventilation in the present study, the marked reduction of cardiac output associated with hypocapnia was of the same magnitude as that found in a group of middle-aged normotensive patients (Theye, Milde and Michenfelder, 1966; Prys-Roberts et al., 1967, 1968), but proportionally much greater than the changes described in young unpremedicated volunteers maintained in a state of eucapnia during IPPV (Price et al., 1969; Eger et al., 1970; Smith et al., 1970). Despite the reduction of cardiac output during artificial ventilation, the arterial pressures in our hypertensive patients did not fall to the same extent as those of normotensive patients (Prys-Roberts et al., 1968; Price et al., 1969), reflecting the high systemic vascular resistances during this stage, especially in the untreated group of patients.

For the hypertensive patient, particularly those with very high arterial pressures, mean arterial pressures of less than 80 mm Hg represent a serious risk of myocardial ischaemia, whereas the normotensive patient can tolerate much lower mean arterial pressures without electrocardiographic changes (Prys-Roberts et al., 1968). In this respect, halothane used as an additive to nitrous oxide-relaxant anaesthesia may be the critical factor reducing coronary blood flow to a degree where electrocardiographic evidence of ischaemia is present (fig. 3). It is difficult to assess the risk to the patient of such episodes of myocardial ischaemia, and while it is gratifying to report that with one exception the e.c.g. changes reported in this study were reversed on withdrawal of anaesthesia, the findings of Mauney, Ebert and Sabiston (1970) offer no room for complacency in dealing with patients with pre-existing e.c.g. abnormalities, whether symptomatic or not. These authors detected evidence of fresh or worsening myocardial ischaemia in 18% of their patients, of whom half had evidence of post operative myocardial infarction, the incidence of which was significantly higher in those patients whose arterial pressures fell by more than 30 mm Hg during anaesthesia and surgery. Renck (1969) surveying a group of elderly men undergoing prostaticctomy, stressed the importance of assessing the preoperative electrocardiogram during rest and exercise, since the postoperative deterioration in
many of his patients was more significant in those patients who had pre-existing coronary heart disease, many of these patients having a normal resting e.c.g. but exhibiting clinical and e.c.g. evidence of ischaemia during exercise. Because of the incidence of ischaemic heart disease associated with hypertension, Mauney, Ebert and Sabiston (1970), Breslin and Swinton (1970) and ourselves (Foëx et al., 1971) have concluded that preoperative stabilization of high arterial pressures should be achieved before elective surgery.

To reduce the degree of hypotension during artificial ventilation, we would recommend the practice of maintaining Pco₂ at eucapnic levels during artificial ventilation, not by using low levels of ventilation, but by allowing partial rebreathing in a closed circuit without an absorber (Suwa and Yamamura, 1970). We believe that the use of pressor amines to maintain arterial pressure in the hypertensive patient is strongly contraindicated. The reduction of cardiac output during anaesthesia in these patients is the result of depressed myocardial contractility in the face of an unaltered or raised systemic vascular resistance (Gersh et al., 1970). To raise further the systemic vascular resistance by inducing arteriolar constriction prejudices ventricular performance, and may rapidly precipitate left ventricular failure. This may be illustrated by the results of an experiment in a dog under halothane anaesthesia (fig. 6), where infusion of phenylephrine, an almost pure arteriolar constrictor, raised arterial pressure, but simultaneously halved the stroke volume and cardiac output.

Pulmonary gas exchanges.

We concluded in our previous communication (Foëx et al., 1971) that the alterations in pulmonary gas exchange in elderly hypertensive patients, gave less cause for concern than their cardiovascular instability. The results of the present study do not modify that conclusion, even though the effects of artificial ventilation on oxygen transport and gas exchange were more marked than those observed during anaesthesia with spontaneous ventilation. With the exception of the changes in the deadspace/tidal volume ratio (Vd/VT), the other effects we have observed were more or less predictable from our previous knowledge of the behaviour of normotensive patients during anaesthesia (Prys-Roberts et al., 1968). The reduction of oxygen uptake during all three stages during anaesthesia was in accord with a number of recent studies in a wide range of patients, anaesthetized with a variety of volatile anaesthetic agents (Theye and Tuohy, 1964; Prys-Roberts et al., 1968; Cullen, Eger and Gregory, 1969; Marshall et al., 1969; Price et al., 1969; Eger et al., 1970). Less predictable, and of less moment were the changes in carbon dioxide elimination. Since this variable was measured by a gaseous technique which assumes the existence of a steady-state of the body carbon dioxide stores (Farhi and Rahn, 1960) its interpretation during IPPV is difficult. During spontaneous ventilation early in the course of anaesthesia, the reduction of alveolar ventilation caused retention of carbon dioxide in the body stores, and the reduced Vco₂ as measured at the lungs may not accurately reflect the true carbon dioxide production of the tissues. During IPPV, the apparent increase in Vco₂ relative to V̇o₂ probably reflects no more than the washout of the body stores of carbon dioxide, and that within the time course of the study equilibrium conditions had not been achieved. The increased values of Ca₀₂-Cv₀₂ during IPPV reflect the disproportionate reduction of cardiac output relative to V̇o₂, and the high values during administration of halothane are comparable with the values found in younger subjects during IPPV (Prys-Roberts et al., 1968) and during induced hypotension (Prys-Roberts, 1970).

The increased alveolar-arterial Po₂ difference during both stages of IPPV as compared with the stage of spontaneous ventilation (at a common FiO₂) reflects the effect of desaturation of mixed venous blood during hypocapnia (Prys-Roberts, Kelman and Greenbaum, 1967) and could not be attributed to increased pulmonary venous admixture. In both the present study, and the previous one (Foëx, Meloche and Prys-Roberts, 1971), we have been impressed by the constancy of Qs/Qt from before anaesthesia to the postoperative period, although similar values during anaesthesia and IPPV were comparable with the mean values derived, usually on the basis of an assumed Ca₀₂-Cv₀₂, by a number of authors (Sykes, Young and Robinson, 1965; Nunn, Bergman and Coleman, 1965; Marshall and Millar, 1965). Our preanaesthetic and postoperative values are also comparable with those of a group of normotensive subjects of the same age group studied by Colgan and Mahoney (1969) and a somewhat older group studied by Renck (1969). On the basis of this study, and the previous study during spontaneous ventilation, we conclude that the increased alveolar-arterial Po₂ difference which is widely observed during anaesthesia does not usually represent a change in the
condition of the lung occurring as a result of anaesthesia per se. The increased \( P_{A_0_2} - P_{A_0_2} \) during air breathing in the supine position, which is most marked in the elderly subject, may be interpreted as representing posture-dependent airway closure (Leblanc, Ruff and Milic-Emili, 1970), and as endemic that these conditions persist unchanged throughout anaesthesia and into the postoperative period.

The changes in the ratio of total deadspace to tidal volume (VD/VT) during artificial ventilation observed in this study reinforce our earlier contention (Foëx, Meloche and Prys-Roberts, 1971) that as an index of changes in pulmonary perfusion relative to ventilation, changes of VD/VT are only meaningful in situations where VT is tolerably constant. The mean values of VD and VD/VT during both stages of IPPV in the present study were markedly lower than those measured by Cooper (1967b), and even if we add 70 ml to the measured deadspace (Vd) as suggested by Cooper, the recalculated mean values for VD/VT (43% during stage C, 44% during stage D) are still well below Cooper's mean value. For the reasons outlined in our previous communication (Foëx, Meloche and Prys-Roberts, 1971), we regard the estimate of 70 ml as being too high to compensate for the reduction of anatomical deadspace as a result of tracheal intubation, and our values of VD/VT during IPPV are more in keeping with those of Nunn and colleagues (1965) and Askrog and colleagues (1964).

In any case the argument is somewhat academic, because if we base our interpretation of the observed difference between \( P_{A_0_2} - P_{A_0_2} \) as if this gradient represented the efficiency of carbon dioxide exchange, then we must conclude, that carbon dioxide exchange was inefficient during anaesthesia with spontaneous ventilation, and more efficient than normal during IPPV with only moderately high tidal volumes.

Patient 2401 was studied because of the unique opportunity afforded by the possibility of maintaining a constant volume of ventilation with different gas mixtures before, during and after anaesthesia. The striking finding was that despite marked changes in mean arterial pressure and cardiac output, to a degree comparable with the changes measured in our hypertensive patients, pulmonary venous admixture and total deadspace remained virtually unchanged. Since it is our experience that overnight ventilation of this patient does not cause any change in VD/VT or Qs/Qt, it would have been surprising if anaesthesia of 3 hours duration had produced a significant change, unless such a change was a pharmacological effect of the specific anaesthetic agent. On the same basis, the changes in VD/VT in our hypertensive patients reflect the changing character of the tidal ventilation rather than a change in the total dead space or its components.

**RECOMMENDATIONS**

On the basis of this and our previous studies of hypertensive patients, we are inclined to make the following recommendations concerning the management of these patients during anaesthesia and surgery involving the use of artificial ventilation.

1. The preoperative assessment of the patient should include a complete examination of the electrocardiogram with the patient at rest and during exercise. Electrocardiographic monitoring should be mandatory during anaesthesia and surgery in order to detect early warning signs of myocardial ischaemia.

2. Patients with high arterial pressures, whether due to lack of treatment, withdrawal of treatment or inadequate treatment, should have their blood pressures stabilized by anti-hypertensive therapy before embarking on elective anaesthesia and surgery.

3. During IPPV with the high tidal volumes necessary to maintain adequate lung expansion, the undesirable circulatory effects of hypocapnia may be avoided by the use of partial rebreathing techniques.

4. Halothane does not commend itself as an additive to nitrous oxide-relaxant anaesthesia with IPPV for the hypertensive patient, and should only be used when an electrocardiograph, and preferably direct arterial pressure monitoring are available, and then it should be used with caution.

5. Pressor amines should not be used to maintain arterial pressure during anaesthesia in hypertensive patients, since their use imposes a potentially overwhelming load on a depressed ventricular myocardium.

**ACKNOWLEDGEMENTS**

It is a pleasure to thank Mr E. Cope, Mr A. Williams, Mr T. J. S. Patterson and Mr P. R. Barton for allowing us to study hypertensive patients under their care, and Dr J. M. K. Spalding for his co-operation in the studies of the myasthenic patient in his care. We wish to thank Mr A. Ryder, Miss C. Ranson, S.R.N., Mr P. Childs and Mr J. Aspel for their skilled technical assistance. Dr P. Foëx was supported by grants from the Hôpital Cantonal et Universitaire, Geneva, and by a special grant from the Holderbank Stiftung, Aargau, Switzerland. The Wang 370 computer used for the Riley-Cournand analyses was bought by the Medical Research Council.

**REFERENCES**


**ETUDES DE L'ANESTHESIE EN RELATION AVEC L'HYPERTENSION**

SOMMAIRE

Dans le cadre d'une respiration assistée sous anesthésie par l'association protoxyde d'azote, oxygène, agent myorelaxant, avec ou sans halothane (1 %), les réponses cardiovasculaires, la ventilation et les échanges gazeux pulmonaires présentés par huit malades hypertendus traités et six autres malades hypertendus non traités, ont été étudiés. Au cours de l'hypocapnéie (tension moyenne de CO2 : PaCO2 = 23 mm Hg) induite par l'IPPV sous anesthésie au protoxyde d'azote, chez les malades traités et chez ceux non traités, la pression artérielle moyenne (PAM) a chuté respectivement à 69 % et 62 % par rapport aux valeurs enregistrées chez les sujets en état d'éveil. Lorsque l'halothane (1 %) a été ajouté dans les mêmes conditions d'IPPV, la pression artérielle moyenne a chuté respectivement de 6 % et de 53 % à partir des valeurs notées à l'état éveillé. Ceci doit être interprété comme étant le résultat d'une réduction du débit cardiaque (respectivement 50 % et 53 % des chiffres notés à l'état d'éveil), alors que la résistance...
ANAESTHESIA IN RELATION TO HYPERTENSION

vasculaire périphérique était augmentée chez tous les
malades au-dessus des valeurs notées à l’état d’éveil. Au
cours de ces périodes d’hypotension artérielle, les con-
trôles électrocardiographiques ont fourni la preuve de
l’existence d’une ischémie myocardique chez 50% des
malades traités et chez tous les malades non traités.
L’augmentation des différences existant au cours de
l’IPPV entre la tension en CO₂ au niveau des alvéoles et
celle mesurée au niveau du sang artériel était consécuti
tive à une désaturation du sang veineux mélangé, associée à
une augmentation des différences de concentration en O₂
du sang artériel et du sang veineux. Le mélange de sang
veineux pulmonaire n’a pas présenté de modifications
significatives au cours de l’anesthésie avec l’IPPV et de la
phase de récupération du malade. Le rapport Vd/Vt s’est
accru au cours de la ventilation spontanée consécutive à
l’induction de l’anesthésie, mais il a présenté une diminu-
tion significative en-dessous des taux notés à l’état d’éveil
au cours de l’IPPV, revenant aux chiffres témoins pendant
la période post-opératoire.

NARKOSE UND HYPERTENSION
ZUSAMMENFASSUNG
An acht behandelten und sechs unbehandelten Patienten
mit Hypertension wurden unter Intubations-Lachgas-
Sauerstoff-Narkose mit und ohne Halothan (1 Prozent) die
cardiovasculären Reaktionen, die Ventilation und der
pulmonale Gasaustausch gemessen. Der mittlere arterielle
Druck (map) fiel auch 69% und auf 62% der Werte im
Wachzustand bei den behandelten und unbehandelten
Patienten unter Hypokapnie (Paco₂: 23 mm Hg), hervor-
gerufen durch Intubations-Beatmung und Lachgasanaes-
thesie. Nach Zugabe von Halothan unter den gleichen
Anästhesiebedingungen fiel der map auf 60 bzw. 53
Prozent der Wachwerte, infolge eines verminderten
Schlagvolumens (50 bzw. 53 Prozent der Wachwerte),
bei einem systemischen Gefäßwiderstand bei allen
Patienten über die Wachwerte hinaus anstieg. Bei 50
Prozent der behandelten und bei allen unbehandelten Pat.
zeigen sich während der arteriellen Hypotension myokard-
diale Ischämiezeichen im EKG. Ein Anstieg der alveolär-
arteriellen PO₂-Differenz wurde verursacht durch Entäs-
tügung von venösem Mischblut, verbunden mit steigender
Differenz im arteriovenösen O₂-Gehalt. Die pulmonale
venöse Beimischung änderte sich unter der Narkose nur
unwesentlich, Vd/Vt nahm unter Spontanatmung nach
Narkoseeinleitung zunächst zu, unter der Intubation dann
deutlich ab, um in der postoperativen Periode wieder zur
Norm zurückzukehren.

ESTUDIOS SOBRE LA ANESTESIA EN
RELACION CON LA HIPERTENSION
RESUMEN
Fueron estudiadas las respuestas cardiovasculares, ventila-
ción e intercambio gaseoso pulmonar de ocho pacientes
hipertensos tratados y sin tratar durante ventilación
artificial bajo anestesia con óxido nitroso/oxígeno/relajante,
con y sin halotano (1 por ciento). La presión arterial media
(m.a.p.) descendió al 69 por ciento y 62 por ciento de
los valores en vigilia respectivamente en los pacientes
tratados y no tratados durante hipocapnia (Paco₂ media:
23 mm Hg) inducida por IPPV bajo anestesia con óxido
nitroso. Cuando se añadió halotano bajo las mismas con-
diciones de IPPV, la m.a.p. descendió a 60 por ciento y
53 por ciento de los valores en vigilia respectivamente,
como resultado de la reducción del gasto cardíaco (respec-
tivamente 50 por ciento y 53 por ciento de los valores
en vigilia), mientras que la resistencia vascular general
aumentó por encima de los valores en vigilia en todos los
pacientes. Fueron observados signos electrocardiográficos
de isquemia miocárdica durante estos períodos de hiper-
tensión arterial en el 50 por ciento de los pacientes tratados
y en todos los pacientes no tratados. El aumento de la
diferencia alveolar-arterial de PO₂ durante la IPPV era
debido a la desaturación de la sangre venosa mezclada
asociada con aumento de la diferencia arteriovenosa de
contenido en O₂. La mezcla venosa pulmonar no cambió
significativamente durante el curso de la anestesia con
IPPV y el restablecimiento. El Vd/Vt aumentó durante
la ventilación espontánea después de inducción de la
anestesia, pero disminuyó significativamente por debajo de
los valores de vigilia durante la IPPV, volviendo a los
valores de control en el período posoperatorio.