Cerebral Blood Flow Responses to Hypocapnia During Hypotension

ALAN A. ARTRU, M.D., AND PETER S. COLLEY, M.D.

SUMMARY  Cerebral vascular responses to hypocapnia during hypotension to a mean arterial blood pressure (MAP) = 50 mm Hg induced with sodium nitroprusside (SNP, n = 12) or trimethaphan (TMP, n = 12) were examined in dogs. Cerebral vascular resistance (CVR) and cerebral blood flow (CBF) at $P_{\text{CO}_2}$ = 40 mm Hg, and $P_{\text{CO}_2}$ = 20 mm Hg were examined first at normal MAP then at hypotension in six dogs in the SNP group and six dogs in the TMP group. In both the SNP group and the TMP group, CO$_2$ responsiveness, as indicated by increased CVR and decreased CBF, was intact at normal MAP, but absent during hypotension. In the remaining 6 of 12 dogs in the SNP group and 6 of 12 dogs in the TMP group, CO$_2$ responsiveness at MAP = 50 mm Hg was examined without prior determination of CO$_2$ responsiveness at normal MAP. These additional studies were performed to rule out the possibility that absent CO$_2$ responsiveness during hypotension in the initial groups resulted from (1) physiologic deterioration of the preparation with time, or (2) adaptation of brain extracellular fluid pH to a preceding period of hypocapnia. Again, during both SNP- or TMP-induced hypotension CO$_2$ responsiveness was absent.

**Method**

Twenty-four unmedicated mongrel dogs (weight 12.6–23.6 kg) were anesthetized with halothane (>1%) and nitrous oxide (N$_2$O, 66%) in oxygen (O$_2$). The trachea was intubated and ventilation controlled with a Harvard pump. Ventilation was adjusted along with the inspired oxygen concentration to maintain initial blood-gas tensions (Radiometer BMS3 MK2 electrodes) at $P_{\text{O}_2}$ > 120 mm Hg and $P_{\text{CO}_2}$ = 39 ± 1 mm Hg (mean ± SEM). With the animal in the lateral position, a urinary catheter was placed and both femoral veins cannulated for fluid and drug administration. Intravenous infusion of succinylcholine 50–120 mg/h maintained muscle relaxation. The right femoral artery was cannulated for arterial blood sampling for blood-gas analysis and continuous monitoring of systemic arterial pressure and heart rate. MAP was determined by electronic integration.Expired CO$_2$ was continuously monitored via a Beckman LB-2 medical gas analyzer. Temperature was monitored by a nasopharyngeal thermometer probe and maintained at 37.0 ± 0.5°C by heat lamps. Depletion of vascular volume was minimized by continuous infusion of saline 4–6 ml·kg$^{-1}$·h$^{-1}$.

The animal was then turned to the prone position and the head slightly elevated and fixed in a stereotaxic frame. The zero reference for the strain gauge to measure systemic arterial pressure was set at the level of the

PREVIOUS STUDIES have reported that hypocapnia causes increased cerebral vascular resistance (CVR) and decreased cerebral blood flow (CBF) at mean arterial pressures (MAP) ≥ 60 mm Hg but not at MAP ≤ 50 when hypotension is induced with hemorrhage or halothane.$^{1-4}$ In clinical practice, elective, controlled hypotension is more commonly achieved with sodium nitroprusside (SNP) or trimethaphan (TMP) rather than hemorrhage or halothane. Based on currently available data, it is not certain whether cerebral vascular responses to hypocapnia also are absent at MAP ≤ 50 mm Hg when SNP or TMP are used to produce hypotension. Gregory et al examined the CO$_2$ responsiveness of CBF during SNP-induced hypotension (MAP = 37 mm Hg) in cats and reported that arterial $P_{\text{CO}_2}$ was altered between 17 and 51 mm Hg, some CO$_2$ responsiveness persisted though at less than half control levels.$^5$ However, in that study hypotension was not produced by SNP alone but by combining SNP with practolol, hemorrhage, and 0.5–0.7% halothane. Sullivan et al observed changes in the electroencephalogram (EEG) with hypocapnia (Pa$_{\text{CO}_2}$ = 18 mm Hg) during SNP-induced hypotension (MAP = 50 mm Hg) in man and speculated that hypocapnia caused cerebral vonasconstriction.$^6$ However, CVR or CBF were not measured and the EEG changes may have been the result of combining hypocapnia with enflurane anesthesia.$^7$ $^8$ Regarding TMP, Hamer et al reported that 5% CO$_2$ increased CBF in dogs during TMP-induced hypotension to MAP = 40 mm Hg.$^9$ Speculation about cerebral vascular reactivity to hypocapnia during TMP-induced hypotension could only be by extrapolation because CBF responses to hypocapnia were not examined. In contrast, Gregory et al. reported absent CBF responses when $P_{\text{CO}_2}$ was altered between 20 and 50 mm Hg during TMP-induced hypotension to MAP = 36 mm Hg in cats.$^5$ However, in that study hypotension was not produced by TMP alone but by combining TMP with practolol, hemorrhage, and 0.5–0.7% halothane.

The present study was designed to determine whether hypotension induced solely with SNP or TMP alters the response of CBF to hypocapnia in dogs. Further, the possibility of differences between SNP- and TMP-induced hypotension was considered. Certain studies suggest that TMP, unlike SNP, does not directly vasodilate cerebral vessels and preserves autoregulation of CBF.$^{10-11}$ Thus, if direct vascular effects of hypertensive drugs are important to CO$_2$ responsiveness during hypotension, cerebral vascular responses to hypocapnia during SNP-induced hypotension should differ from those during TMP-induced hypotension.

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These studies were undertaken to rule out the possibility that in groups I and II the CBF response to normocapnia and hypocapnia during hypotension was influenced either by time (2 hr elapsed before hypotension was induced) or by the 30 min exposure to hypocapnia at normal MAP. In both groups three dogs were examined first at normocapnia and then at hypocapnia, while the other three dogs were examined first at hypocapnia and then at normocapnia. In these two groups the duration of each condition was 30 min. Hypotension was induced with SNP in group III and with TMP in group IV.

Cerebral and systemic variables were compared within groups using repeated-measures two-factors analysis of variance with PaCO2 and MAP comprising the two treatments. One way analysis of variance was employed to make comparisons between groups I and II, and between groups III and IV, using observed values at normocapnia, and percent change from normocapnia values for comparisons at hypocapnia. A p value of less than 0.05 was considered significant.

Results

At normal MAP, hypocapnia significantly increased CBF and decreased CBF in both groups I and II (table I). In contrast, during hypotension hypocapnia caused no statistically significant change in CVR or CBF. CO2 responsiveness was absent during SNP- and TMP-induced hypotension both in the dogs previously tested for CO2 responsiveness at normal MAP (groups I and II, tables 2 and 3), and in the dogs not previously tested for CO2 responsiveness at normal MAP (groups III and IV, table 4). The effects of hypocapnia on cerebral variables in the SNP groups (I and III) generally were similar to those in the TMP groups (II and IV). Exceptions were that the decrease of sagittal sinus blood O2 tension with hypocapnia during TMP-induced hypotension in group II did not achieve statistical significance, and CVR and CBF did not return to initial values 60 min after hypopcapnia during normal MAP in group II (TMP). The SNP infusion rates to achieve MAP = 50 mm Hg were 3.9 ± 0.5 μg·kg⁻¹·min⁻¹ for group I and 2.8 ± 0.6 μg·kg⁻¹·min⁻¹ for group III. The TMP infusion rates to achieve MAP = 50 mm Hg were 10.3 ± 1.7 μg·kg⁻¹·min⁻¹ for group II and 12.8 ± 2.0 μg·kg⁻¹·min⁻¹ for group IV.

As regards systemic variables, hypocapnia increased arterial blood pH and decreased bicarbonate in all dogs. During hypocapnia arterial blood pH and bicarbonate were not significantly different in the SNP groups compared to the TMP groups. During normocapnia bicarbonate was increased in SNP group I compared to TMP group II. Generally, other systemic variables were not significantly altered by hypotension or hypocapnia. An exception was heart rate which was greater during SNP-induced hypotension than during TMP-induced hypotension.

Discussion

The present study was designed so that dogs in groups I and II were tested for CO2 responsiveness at normal MAP before testing for CO2 responsiveness at
### Table 1 Effect of Hypocapnia on Cerebral and Systemic Variables at Normal MAP, Groups I and II (mean ± SEM)

<table>
<thead>
<tr>
<th></th>
<th>SNP, group I, n = 6</th>
<th></th>
<th>TMP, group II, n = 6</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normotension</td>
<td>Hypocapnia</td>
<td>Normotension</td>
<td>Hypocapnia</td>
</tr>
<tr>
<td>Cerebral Variables</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVR, mm Hg/ml·min⁻¹·100g⁻¹</td>
<td>0.98 ± 0.07</td>
<td>1.55 ± 0.17*</td>
<td>1.24 ± 0.15</td>
<td>2.34 ± 0.29*</td>
</tr>
<tr>
<td>CBF, ml·min⁻¹·100g⁻¹</td>
<td>100.6 ± 10.6</td>
<td>61.6 ± 8.0*</td>
<td>81.8 ± 16.7</td>
<td>38.3 ± 5.4*</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>58 ± 3</td>
<td>42 ± 2*</td>
<td>56 ± 2</td>
<td>33 ± 2*</td>
</tr>
<tr>
<td>Systemic Variables</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>97 ± 5</td>
<td>90 ± 6</td>
<td>92 ± 8</td>
<td>84 ± 5</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>111 ± 4</td>
<td>113 ± 6</td>
<td>106 ± 6</td>
<td>109 ± 5</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>39 ± 1</td>
<td>20 ± 1*</td>
<td>39 ± 1</td>
<td>19 ± 1*</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>170 ± 5</td>
<td>201 ± 4*</td>
<td>183 ± 30</td>
<td>187 ± 17</td>
</tr>
<tr>
<td>pH</td>
<td>7.35 ± 0.01</td>
<td>7.50 ± 0.03*</td>
<td>7.33 ± 0.01</td>
<td>7.52 ± 0.02*</td>
</tr>
<tr>
<td>Bicarbonate, mEq/l</td>
<td>20.1 ± 0.6†</td>
<td>14.8 ± 0.7*</td>
<td>18.0 ± 0.8†</td>
<td>15.2 ± 0.7*</td>
</tr>
<tr>
<td>Hemoglobin, g/dl</td>
<td>12.5 ± 0.5</td>
<td>12.5 ± 0.5</td>
<td>12.6 ± 0.6</td>
<td>12.5 ± 0.7</td>
</tr>
<tr>
<td>Temperature, nasopharyngeal, °C</td>
<td>37.1 ± 0.1</td>
<td>36.8 ± 0.1</td>
<td>37.1 ± 0.2</td>
<td>36.9 ± 0.2</td>
</tr>
</tbody>
</table>

* = significant difference compared to values at normotension and normocapnia, p < 0.05.
† = significant difference SNP value vs. TMP value, p < 0.05.

CVR = cerebral vascular resistance, CBF = cerebral blood flow, PaO₂ = sagittal sinus blood oxygen tension, MAP = mean arterial blood pressure.

### Table 2 Effect of Hypocapnia on Cerebral and Systemic Variables during SNP-induced Hypotension, Group I (mean ± SEM), n = 6

<table>
<thead>
<tr>
<th></th>
<th>Normotension</th>
<th>Hypotension</th>
<th>Hypotension</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normocapnia</td>
<td>Hypocapnia</td>
<td>Normocapnia</td>
</tr>
<tr>
<td>Cerebral Variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVR, mm Hg/ml·min⁻¹·100g⁻¹</td>
<td>1.36 ± 0.11</td>
<td>0.89 ± 0.08†</td>
<td>0.96 ± 0.07</td>
</tr>
<tr>
<td>CBF, ml·min⁻¹·100g⁻¹</td>
<td>76.5 ± 3.5</td>
<td>59.9 ± 4.6</td>
<td>53.1 ± 4.4</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>46 ± 3</td>
<td>48 ± 3</td>
<td>38 ±3*</td>
</tr>
<tr>
<td>Systemic Variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>103 ± 6</td>
<td>52 ± 2†</td>
<td>50 ± 1</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>114 ± 8</td>
<td>135 ± 6†</td>
<td>137 ± 6†</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>39 ± 1</td>
<td>39 ± 1</td>
<td>21 ± 1*</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>177 ± 8</td>
<td>161 ± 6</td>
<td>184 ± 6</td>
</tr>
<tr>
<td>pH</td>
<td>7.30 ± 0.01</td>
<td>7.30 ± 0.02</td>
<td>7.44 ± 0.02*</td>
</tr>
<tr>
<td>Bicarbonate, mEq/l</td>
<td>17.6 ± 0.8</td>
<td>18.5 ± 0.5†</td>
<td>14.5 ± 0.8*</td>
</tr>
<tr>
<td>Hemoglobin, g/dl</td>
<td>11.7 ± 0.6</td>
<td>11.8 ± 0.6</td>
<td>11.7 ± 0.6</td>
</tr>
<tr>
<td>Temperature, nasopharyngeal, °C</td>
<td>37.0 ± 0.2</td>
<td>37.2 ± 0.1</td>
<td>37.2 ± 0.1</td>
</tr>
</tbody>
</table>

* = significant difference compared to values at hypotension and normocapnia, p < 0.05.
† = significant difference SNP value vs. TMP value at same condition (table 3), p < 0.05.

CVR = cerebral vascular resistance, CBF = cerebral blood flow, PaO₂ = sagittal sinus blood oxygen tension, MAP = mean arterial blood pressure.

MAP = 50 mm Hg. That hypocapnia increased CVR by an average of 73% and decreased CBF by an average of 47% at normotension in these groups is consistent with previous studies and suggests that the cerebral vasculature of these dogs was normally reactive to CO₂ and that the surgical preparation was suitable to measure those responses. Our subsequent observation in those same groups of no statistically significant cerebral vascular response to hypocapnia during SNP- or TMP-induced hypotension suggests that hypocapnia does not cause a clinically relevant decrease of CBF during hypotension to a MAP = 50 mm Hg produced by either direct or indirect acting vasodilator drugs. We wished to eliminate the possibility that absent CO₂ responsiveness in groups I and II during hypotension occurred because the surgical preparation deteriorated after 2 hr or because 30 min hypocapnia at normal MAP impaired subsequent CO₂ responsiveness due to adaptation of brain extracellular fluid pH. Thus, in groups III and IV, CO₂ responsiveness during hypotension was examined as soon as systemic and cerebral variables stabilized after surgical preparation and with-
out prior testing for CO₂ responsiveness at normal MAP. Our observation in these latter groups of no statistically significant cerebral vascular response to hypocapnia during SNP- or TMP-induced hypotension confirmed the results of groups I and II, and suggests that those results were not artifacts caused by the duration of the study or by the earlier exposure to hypocapnia and normotension.

In the present study CVR decreased when MAP was lowered to 50 mm Hg using either SNP or TMP. This observation suggests that the present experimental preparation preserved normal cerebral vasodilatory responses to hypotension as well as CO₂ responsiveness (seen in Groups I and II at normal MAP). CBF decreased at MAP = 50 mm Hg in all groups suggesting that this level of MAP was below the lower limit of

### Table 3: Effect of Hypocapnia on Cerebral and Systemic Variables during TMP-induced Hypotension, Group II (mean ± SEM), n = 6

<table>
<thead>
<tr>
<th>Cerebral Variables</th>
<th>Normocapnia</th>
<th>Hypotension</th>
<th>Hypotension</th>
<th>Hypotension</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normocapnia</td>
<td>Normocapnia</td>
<td>Normocapnia</td>
<td>Normocapnia</td>
</tr>
<tr>
<td>CVR, mm Hg/ml·min⁻¹·100g⁻¹</td>
<td>1.74 ± 0.20§</td>
<td>1.16 ± 0.15‡</td>
<td>1.28 ± 0.11</td>
<td></td>
</tr>
<tr>
<td>CBF, ml·min⁻¹·100g⁻¹</td>
<td>54.9 ± 10.7§</td>
<td>46.9 ± 8.3</td>
<td>40.0 ± 3.8</td>
<td></td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>48 ± 6</td>
<td>45 ± 7</td>
<td>34 ± 2</td>
<td></td>
</tr>
<tr>
<td>Systemic Variables</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>88 ± 6</td>
<td>50 ± 2‡</td>
<td>49 ± 2</td>
<td></td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>106 ± 7</td>
<td>111 ± 8†</td>
<td>115 ± 7‡</td>
<td></td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>38 ± 1</td>
<td>39 ± 1</td>
<td>22 ± 1*</td>
<td></td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>170 ± 7</td>
<td>162 ± 9</td>
<td>156 ± 13</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.30 ± 0.01</td>
<td>7.29 ± 0.01</td>
<td>7.44 ± 0.01*</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate, mEq/l</td>
<td>16.9 ± 0.2</td>
<td>16.8 ± 0.2†</td>
<td>14.1 ± 0.7*</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin, g/dl</td>
<td>11.9 ± 0.6</td>
<td>11.9 ± 0.6</td>
<td>11.8 ± 0.6</td>
<td></td>
</tr>
<tr>
<td>Temperature, nasopharyngeal, °C</td>
<td>36.8 ± 0.1</td>
<td>36.8 ± 0.1</td>
<td>36.8 ± 0.1</td>
<td></td>
</tr>
</tbody>
</table>

* = significant difference compared to values at hypotension and normocapnia, p < 0.05.
† = significant difference TMP value vs. SNP value at same condition (table 2), p < 0.05.
‡ = significant difference compared to values at normotension and normocapnia, p < 0.05.
§ = significant difference compared to values at the first condition of normotension and normocapnia (table 1), p < 0.05.

CVR = cerebral vascular resistance, CBF = cerebral blood flow, PaO₂ = sagittal sinus blood oxygen tension, MAP = mean arterial blood pressure.

### Table 4: Effect of Hypocapnia on Cerebral and Systemic Variables during Hypotension, Groups III and IV (mean ± SEM)

<table>
<thead>
<tr>
<th>Cerebral Variables</th>
<th>SNP, group III, n = 6</th>
<th>TMP, group IV, n = 6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hypotension</td>
<td>Hypocapnia</td>
</tr>
<tr>
<td>CVR, mm Hg/ml·min⁻¹·100g⁻¹</td>
<td>0.93 ± 0.07</td>
<td>0.96 ± 0.06</td>
</tr>
<tr>
<td>CBF, ml·min⁻¹·100g⁻¹</td>
<td>58.3 ± 4.2</td>
<td>53.8 ± 2.9</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>45 ± 3</td>
<td>39 ± 2*</td>
</tr>
<tr>
<td>Systemic Variables</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>50 ± 1</td>
<td>50 ± 1</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>137 ± 7†</td>
<td>140 ± 6†</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>39 ± 1</td>
<td>21 ± 1*</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>169 ± 6</td>
<td>182 ± 3</td>
</tr>
<tr>
<td>pH</td>
<td>7.31 ± 0.01</td>
<td>7.48 ± 0.03*</td>
</tr>
<tr>
<td>Bicarbonate, mEq/l</td>
<td>19.2 ± 0.8</td>
<td>15.5 ± 0.9*</td>
</tr>
<tr>
<td>Hemoglobin, g/dl</td>
<td>12.2 ± 0.4</td>
<td>12.0 ± 0.3</td>
</tr>
<tr>
<td>Temperature, nasopharyngeal, °C</td>
<td>36.9 ± 0.2</td>
<td>36.9 ± 0.2</td>
</tr>
</tbody>
</table>

* = significant difference compared to values at hypotension and normocapnia, p < 0.05.
† = significant difference SNP value vs. TMP value at same condition, p < 0.05.
CVR = cerebral vascular resistance, CBF = cerebral blood flow, PaO₂ = sagittal sinus blood oxygen tension, MAP = mean arterial pressure.
auto-regulation of CBF for dogs. This observation is in agreement with determinations of the lower limit of auto-regulation of CBF for dog at normocapnia.\(^\text{15, 16}\)

It has been proposed that the failure of cerebral vessels to constrict when \(P_{\text{a}}\) is lowered during severe hypotension indicates that maintenance of cerebral perfusion takes precedence over the maintenance of a normal tissue \(P_{\text{a}}\).\(^\text{1-15}\) The authors postulated that such an “over-ride” mechanism could be mediated through the tissue oxygen tension, which is presumably low due to inadequate blood flow, and could counteract the vasoconstrictive effect of hypocapnia. This explanation implies that loss of CBF response to hypocapnia should be observed using any technique that decreases MAP to \(\leq 50\) mm Hg and thus is consistent with our results and those of previous studies. However, tissue oxygen tension likely is not the sole mediator of this effect because in the present study both CBF and \(P_{\text{a}}\) appeared to be adequate at MAP = 50 mm Hg and normocapnia. A more complete understanding of the mechanisms regulating cerebral vessel diameter during hypoventilation and hypocapnia requires additional investigation.

In groups I and II control CBF values were higher than values observed during surgical levels of anesthetics (CBF = 60-70 ml-100 g\(^{-1}\)-min\(^{-1}\)),\(^\text{17-19}\) but less than or equal to values during light anesthesia with N\(_2\)O (60-70%) and halothane (< 0.1%) in \(O_2\) (CBF = 80-140 ml-100 g\(^{-1}\)-min\(^{-1}\))\(^\text{19-21}\) with this model. In previous studies high control CBF values during light anesthesia have not impaired subsequent determination of CBF, cerebral metabolic rate for oxygen, cerebral metabolites (ATP, lactate, pyruvate, phosphocreatine, etc.), EEG, or integrity of the “blood-brain” barrier during hypoxemia, hypercapnia, or administration of catecholamines, anesthetics, or sedative hypnotics.\(^\text{12, 21}\)

This experience, plus observance in the present study of normal CO\(_2\) responsiveness of CBF at normal MAP and a normal cerebral vasodilatory response to hypoventilation at normocapnia suggests that high control CBF values in the present study did not impair cerebral vascular reactivity later in the study.

In the present study CBF was lower at 120 min at normal MAP and normocapnia (condition 3) than after 30 min at normal MAP and normocapnia (condition 1). This decrease may be related in part to the surgical preparation. A time-related decrease of CBF previously was reported in dogs with craniotomy and cannulation of the sagittal sinus or torcular.\(^\text{22, 23}\) CBF was 25% lower than initial values, with the decrease occurring independent of the method used to determine CBF. The study of McDowall and Harper found that > 90% of the CBF decrease occurs within the first 60 min, with CBF remaining relatively stable thereafter.\(^\text{22}\)

These previous studies suggest that the time effect on CBF may explain, in part, the lower CBF values measured at 90 min after initial CBF determinations in this study. These earlier data further suggest that the time-related decrease of CBF after normal MAP and normocapnia at 120 min (condition 3) was small and did not obscure subsequent cerebral vascular reactivity to hypotension (condition 4) or hypocapnia (condition 5). A second contributing factor to the time-related decrease of CBF observed in the present study may be adaptation to the cerebral vasodilating effects of halothane. Albrecht et al reported that in goats anesthetized with halothane (1% inspired in \(O_2\)) CBF was doubled during the first 30 min then decreased and approached pre-halothane values over the next 2 hr.\(^\text{24}\)

In summary, our results do not support the proposal that hyperventilation during SNP- or TMP-induced hypotension further decreases CBF to cause ischemia. Further, our results suggest that the direct vascular effects of hypotensive drugs may not be important to CO\(_2\) responsiveness of CBF during hypotension to MAP = 50 mm Hg.

References


18. Nugent M, Artru AA, Michenfelder JD: Cerebral metabolic, vas-
PATIENTS WITH INFARCTIONS in the territory of the right middle cerebral artery (RMCA) may present with an agitated confusional state and a paucity of lateralized deficits. We have encountered two such patients in three years. A detailed description of this syndrome has been published only once, and we were unable to find any information concerning the frequency of this presentation among patients with RMCA infarctions. We therefore undertook this study to ascertain how common this presentation was, and to determine whether there were any clinical features distinguishing those patients presenting with agitated confusion from other patients with RMCA territory infarctions.

Methods

We reviewed the records of patients with RMCA strokes who were seen by the Neurology Services of San Francisco General and University of California, Moffitt Hospitals, between July 1, 1979 and June 30, 1982. Patients with coma, metabolic derangement, septicemia, preexisting dementia, or other conditions capable of causing an abnormal mental state were excluded from consideration, as were those with frank intracerebral hemorrhage on computed tomographic (CT) scan, and those whose CT scan and examination indicated lacunar infarction. During the period of the study, a CT scan was a standard part of the investigation of all patients with unexplained confusion. Forty-six patients fulfilled these criteria. Orientation and level of consciousness were recorded in all cases. An agitated confusional state was defined by the presence of disorientation, distractibility, agitation, impaired cognition and perceptual errors (illusions, delusions or hallucinations). CT scans confirmed the presence of cerebral cortical infarction in 35 patients. Of the remaining 11 patients, four had only deep cerebral infarctions on CT scan and seven showed no lesion on CT scan (in six of these seven, CT scans were done within 24 hours of onset of neurologic symptoms). All 11, however, had sensory deficits suggesting parietal cortical infarction, such as agaphesthesia, astereognosis, extinction to double simultaneous stimulations, impaired sensory localization, unilateral neglect, and anosagnosia. They were therefore included in the study. Two patients with infarctions in the right internal carotid artery (RICA) distribution were grouped with RMCA stroke patients.

Results

The two patients who presented with an agitated confusional state are described briefly. Both were seen...
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