Effects of experimental fluid-percussion injury of the brain on cerebrovascular reactivity to hypoxia and to hypercapnia

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To test the hypothesis that concussive brain injury interferes with the normal vasodilator response of the cerebral circulation to hypoxemia, 30 cats were subjected to mild (PaO$_2$ 50 mm Hg) and severe (PaO$_2$ 30 mm Hg) hypoxemia while measurements were made of arterial and intracranial pressure, regional cerebral blood flow (CBF), and arterial blood gases. Ten cats served as controls, 10 were subjected to mild fluid-percussion injury of the brain (0.8 to 1.7 atmospheres (atm)), and 10 to severe injury (2.4 to 4.1 atm). The CBF response to hypercapnia (PaCO$_2$ 50 mm Hg) was also tested in most animals, and the response of CBF autoregulation to hemorrhagic hypotension was tested in four animals of each group. Trauma was found to severely attenuate the capacity of CBF to increase during hypoxemia. Responsiveness to hypoxemia appeared to be better preserved in traumatized animals than was autoregulation, but was less robust than the response to hypercapnia.

KEY WORDS • brain injury • concussion • cerebral blood flow • hypoxia • autoregulation

Of the secondary physiological insults that may stress the patient with a severe head injury at any time after impact, the most common is hypoxemia. In a recent survey of 225 comatose patients, the authors found more than 30% to have an arterial pO$_2$ of less than 60 mm Hg on admission. The presence of this insult was significantly associated with a poor outcome from injury.13

Under normal circumstances, severe hypoxemia provokes cerebral vasodilatation, which by increasing cerebral blood flow (CBF) compensates, up to a point, for the reduction in the oxygen content of arterial blood.12 This response may be attenuated or lost under certain circumstances: in areas of severe local brain damage or following a severe ischemic insult.6 Cerebrovascular responsiveness to hypercarbia, hypoxemia, and arterial hypotension is frequently grouped together in the literature, with the implication that when one response is impaired, the others are also. In fact, this is not always the case.2,16

In previous studies, we have investigated the effect of varying the severity of concussive brain injury in the cat on the subsequent cerebrovascular response to changes in arterial pCO$_2$ and to arterial hypotension.11,15 Injuries of mild degree, sufficient to produce temporary stunning, were associated with attenuation of the cerebral vasodilator response to CO$_2$. More severe injuries, of a severity that produces prolonged coma, were associated with abolition of the response to CO$_2$. While the effects of graded trauma on the CO$_2$ response were very clear, the effects of trauma on autoregulation to reduced arterial pressure were more variable. Overall, it could be said that concussive brain trauma was associated with impaired pressure autoregulation, but this impairment usually took the form of an elevation of the minimum arterial pressure at which CBF could still be maintained in the normal range.

In the present series of experiments, the effect of graded concussive brain injury in the cat upon the subsequent cerebrovascular response to mild and severe hypoxemia was studied. The cerebrovascular responses to CO$_2$ and arterial hypotension were also measured, to permit conclusions to be drawn concern-
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...ing the relative frequency of coexisting impairment of these responses.

Materials and Methods

Preparation

Thirty mongrel cats, weighing from 2.4 to 3.5 kg, were anesthetized with methohexital sodium (30 mg/kg), then tracheotomized and artificially ventilated. Suxamethonium chloride in a 5% glucose solution buffered with sodium bicarbonate was administered intravenously via a cannula in a femoral vein using a Harvard infusion pump.* and artificial ventilation with a mixture of 70% N₂O and 30% O₂ was instituted. All operative sites were infiltrated with 1% lignocaine to provide local anesthesia during the remainder of the surgical preparation.

A cannula was introduced into the brachial artery for measurement of arterial blood pressure and for withdrawal of blood samples for gas analysis. An arterial cannula was inserted into the abdominal aorta via a femoral artery, and was connected to a heparinized reservoir to be used later for reduction of blood pressure. The animal was fitted into a Trent-Wells stereotaxic frame and a drill hole made in the temporoparietal region to accommodate an epidural catheter for measurement of intracranial pressure (ICP). Biparietal and frontal screws were inserted for recording tracings from the electroencephalogram (EEG) and multimodality evoked potentials (MEP). A large burr hole, 11 mm in diameter, was made over the parieto-occipital region, and a hollow metal screw was tightly inserted into this hole without opening the underlying dura mater. The fluid-percussion device described previously by Sullivan, et al., was then attached and fixed with dental acrylic to this hollow injury screw.

Cerebral trauma was produced by the release of a weighted pendulum, which impounded a saline-filled Plexiglas column attached to the hollow screw. An injury force of varying pressure but of constant duration (20 msec) was delivered to the intact dura under the injury screw and was recorded extracranially by a strain gauge and oscilloscope. The cats were divided randomly into three groups: a control group that received no trauma, a mild injury group that sustained injuries ranging from 0.8 to 1.7 atmospheres (atm) (600 to 1300 mm Hg), and a severe injury group that sustained injuries ranging from 2.4 to 4.1 atm (1800 to 3100 mm Hg).

Immediately following the imposition of trauma and at an equivalent time in the control animals, two further frontal drill holes were made in the skull, and platinum electrodes, 0.3 mm in diameter, were inserted and passed stereotaxically to lodge in each caudate nucleus. These electrodes were connected to a source of polarizing voltage and to an amplifier system, to be used for measurement of regional CBF by the hydrogen ion clearance technique. For each measurement of CBF, 2% hydrogen gas was administered into the inspired gas mixture for 2 to 3 minutes, sufficient to permit a full-scale deflection on the dynograph. Hydrogen administration was then stopped and blood flow determined from the half-time of the resulting washout curve, after 30 seconds' delay to allow arterial H₂ concentration to fall to baseline. To permit full polarization and stabilization of the electrodes, the first measurements of CBF were not made until a period of 1 hour had elapsed from initial electrode placement.

Arterial pressure, ICP, EEG, and end-expiratory CO₂ pressure were recorded continuously on a Beckman eight-channel dynograph recorder.† During each measurement of CBF, arterial blood gases were estimated with pH and MEP. Body temperature was kept at 37.5 ± 0.5°C by a heating blanket.

In calculating results, the mean value of the flow recorded from both cerebral hemispheres was used. Differences between the two hemispheres were small, and the responses did not differ qualitatively.

Experimental Protocol

After surgical preparation and stabilization of the platinum electrode system for 1 hour, two normoxic, normocapnic, and normotensive measurements of CBF were performed in all animals to determine resting control and posttraumatic value of CBF.

A mild hypoxic stimulus (PaO₂ = 50 torr) of 8 minutes' duration was then imposed by decreasing the oxygen percentage in the anesthetic gas mixture. This was followed by a severe hypoxic insult (PaO₂ = 30 torr) of the same duration. Hypercapnia was then imposed (PaCO₂ = 50 torr) to test the responsiveness of the cerebral circulation to CO₂. Each insult was separated by a period of normoxia and normocapnia of 30 minutes' duration.

In four animals of each group, the first insult of hypoxia was additionally preceded by hypercapnia. These two tests of the effects of hypercapnia were to measure the influence of hypoxia on cerebrovascular responsiveness of traumatized brain to CO₂. In these animals, following the second (post-hypoxic) hypercapnic insult, arterial pressure was also progressively lowered by bleeding the animal into the reservoir to a pressure of 80 mm Hg, and then by 20-mm Hg increments to test pressure autoregulation.

At each step of the experiment, CBF, MEP (visual, somatosensory (cortical and brain stem), and auditory (cortical and brain stem)) and blood gases were measured.

* Harvard infusion pump manufactured by Harvard Apparatus Co., Inc., 150 Dover Road, Millis, Massachusetts.

† Beckman eight-channel dynograph recorder manufactured by Beckman Instruments, Inc., 2500 Harbor Boulevard, Fullerton, California.
**TABLE 1**

Effect of hypoxia and hypercapnia in the control group*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Rest</th>
<th>Mild Hypoxia</th>
<th>Rest</th>
<th>Severe Hypoxia</th>
<th>Rest</th>
<th>Hypercapnia</th>
</tr>
</thead>
<tbody>
<tr>
<td>MABP (mm Hg)</td>
<td>145.0 ± 3.4</td>
<td>152.0 ± 4.3</td>
<td>146.0 ± 4.7</td>
<td>151.5 ± 5.0</td>
<td>138.5 ± 6.7</td>
<td>149.5 ± 6.9</td>
</tr>
<tr>
<td>ICP (mm Hg)</td>
<td>10.2 ± 1.9</td>
<td>10.1 ± 1.7</td>
<td>8.6 ± 1.7</td>
<td>11.3 ± 2.1</td>
<td>8.7 ± 1.5</td>
<td>9.2 ± 1.7</td>
</tr>
<tr>
<td>CPP (mm Hg)</td>
<td>134.8 ± 4.3</td>
<td>141.9 ± 4.3</td>
<td>137.4 ± 4.3</td>
<td>140.2 ± 5.7</td>
<td>129.8 ± 6.8</td>
<td>140.0 ± 7.9</td>
</tr>
<tr>
<td>CBF (ml/100 gm/min)</td>
<td>34.3 ± 3.2</td>
<td>37.6 ± 3.2</td>
<td>30.7 ± 2.8</td>
<td>47.2 ± 4.9</td>
<td>29.5 ± 2.9</td>
<td>51.6 ± 4.8</td>
</tr>
<tr>
<td>CVR (mm Hg/ml/100 gm/min)</td>
<td>4.2 ± 0.4</td>
<td>4.0 ± 0.3</td>
<td>4.8 ± 0.3</td>
<td>3.1 ± 0.2</td>
<td>4.7 ± 0.2</td>
<td>2.8 ± 0.2</td>
</tr>
<tr>
<td>PaO2 (mm Hg)</td>
<td>128.5 ± 3.6</td>
<td>52.3 ± 0.7</td>
<td>121.0 ± 4.6</td>
<td>29.8 ± 1.4</td>
<td>125.5 ± 4.5</td>
<td>133.0 ± 4.2</td>
</tr>
<tr>
<td>PaCO2 (mm Hg)</td>
<td>32.2 ± 0.8</td>
<td>33.0 ± 1.0</td>
<td>32.1 ± 0.7</td>
<td>33.0 ± 0.4</td>
<td>32.3 ± 0.7</td>
<td>51.6 ± 1.0</td>
</tr>
<tr>
<td>pH</td>
<td>7.42 ± 0.13</td>
<td>7.41 ± 0.07</td>
<td>7.41 ± 0.09</td>
<td>7.41 ± 0.13</td>
<td>7.4 ± 0.08</td>
<td>7.41 ± 0.14</td>
</tr>
</tbody>
</table>

* MABP = mean arterial blood pressure; ICP = intracranial pressure; CPP = cerebral perfusion pressure; CBF = cerebral blood flow; CVR = cerebral vascular resistance.

**Neuropathological Examination**

At the end of each experiment, all brains were removed and placed in buffered neutral formalin. Gross neuropathological examination of each brain was performed in order to assess and confirm the graded injury level to which animals were subjected and grouped. Inspection, sectioning, and classification of brains into control, and mild and severe trauma groups were also performed on pathological grounds, without prior knowledge of the intensity of the injury and according to a protocol described previously.11

**Results**

**Resting Values and Physiological Response to Injury**

Arterial Blood Pressure. Initial mean arterial blood pressure (MABP) of the control group before electrode placement and of the mild and severe trauma groups before trauma was 146.5 ± 4.4, 137.5 ± 6.2, and 140.5 ± 6.2 mm Hg, respectively. The MABP for control animals 1 hour after electrode placement was 145.0 ± 3.4 mm Hg. Thus, the experimental procedure did not affect MABP.

Immediately after injury, there was an average increase in MABP of 81.0 ± 15.2 mm Hg in the mild trauma group. One hour after trauma, MABP was again at the normal level (135.0 ± 8.3 mm Hg). In the severe trauma group, the MABP rose by 96.5 ± 8.2 mm Hg, but 1 hour later was 11.4% below the initial value.

Intracranial Pressure. Resting ICP values for the control, and mild and severe trauma groups were 10.4 ± 2.0, 8.3 ± 0.8, and 12.3 ± 1.7 mm Hg, respectively. The ICP level 1 hour following placement of electrodes did not differ from the initial value.

Immediately after fluid-percussion injury below 2.0 atm, the ICP rose by 8.8 ± 3.5 mm Hg. Injury above 2.0 atm caused a transient maximum increase of ICP by 32.4 ± 6.7 mm Hg. In both groups, ICP remained slightly elevated at 1 hour after trauma.

Cerebral Perfusion Pressure. The cerebral perfusion pressure (CPP) was calculated as the difference between the MABP and ICP. It did not change significantly within the 1st hour in the control group. In traumatized animals, there was a transient increase in CPP immediately following trauma because arterial pressure rose more than ICP. Within 1 hour, CPP returned to the initial value in the mild trauma group and was 13% below baseline in the severe trauma group.

Cerebral Blood Flow. Initial values for CBF, 1 hour after electrode placement, were 34.3 ± 3.2, 30.1 ± 1.8, and 25.7 ± 4.4 ml/100 gm/min in control, and mild and severe trauma groups, respectively.

Cerebral Vascular Resistance. The cerebral vascular resistance (CVR) was calculated as CPP divided by CBF. The CVR values in the control and mild trauma groups were almost equal. Higher mean CVR in the severe trauma group resulted from very low CBF in one animal that received a 3.0-atm injury.

Effect of Graded Hypoxia and of Hypercapnia

Results in Control Animals. The results of hypoxia and hypercapnia in control animals are presented in Table 1. The insult of mild hypoxia (PaO2 = 52.3 ± 0.7 mm Hg) caused an increase of CBF by 9.6% as compared with the initial normoxic value. After 30 minutes of subsequent normoxia, CBF was again reduced to control levels. The second hypoxic insult (PaO2 = 29.8 ± 1.4 mm Hg) was reflected in a sharp increase of CBF in all animals, by 53.7% on average. Restoration of normoxic conditions produced a return of CBF to the previous control value. Hypercapnia (PaCO2 = 51.6 ± 1.0 mm Hg) imposed after the hypoxic insults caused an increase of CBF in all animals by 74.9% on average.

Results in Traumatized Animals. In the group of animals subjected to trauma of between 0.8 to 1.7 atm, mild hypoxia caused an increase of CBF by 18.9% as compared with the initial value; CBF returned to baseline as normoxia was restored (Table 2). Severe hypoxia produced a greater increase of CBF in all mildly traumatized animals, by 41.1% on average. As in the control group, all injured cats subsequently reacted to hypercapnia, with a mean increase of CBF by 65.9%.

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The animals subjected to severe trauma (between 2.4 and 4.1 atm) did not respond in the same manner to mild hypoxia. In four of them, slight reduction in CBF was seen. Return to normoxic conditions was associated with a decrease of CBF in all but one animal (a 13.2% decrease as compared with the level in the mild hypoxia group). Severe hypoxia caused a mean increase of CBF of 27.5%, but two animals (trauma of 2.5 and 2.7 atm) reacted paradoxically with a decrease of CBF and then with an increase of 22% and 67% when normoxia was restored. Three of these severely injured cats did not respond to a subsequent hypercapnic insult by cerebral vasodilatation, and the reaction in others was significantly depressed (mean increase in CBF of 14.9%). The results are presented in Table 3.

**Changes in CVR.** The changes in CBF during hypoxia could not be explained solely by the changes in CPP that occurred. There was a fall in CVR in control animals, with a decrease of 4.8% and 35.4% as mild and severe hypoxia were applied. This implies cerebrovascular dilatation has occurred to supply an adequate amount of oxygen to the brain. Hypercapnia also caused a sharp reduction in CVR, by 40.4%.

The animals of the mild trauma group reacted to mild hypoxia, severe hypoxemia, and hypercapnia in the same manner; CVR was reduced by 13.9%, 24.3%, and 27.9%, respectively. In severe trauma animals, the response to hypoxic insults was much more variable. On average, mild and severe hypoxia reduced CVR by 12% and 24%, respectively, but two animals reacted with an increase of CVR. Hypercapnia decreased CVR by 9.6% on average, but in three cats there was an opposite effect.

The data suggest that trauma diminishes the responsiveness of cerebral vessels both to hypoxia and to hypercapnia. Severe trauma has more pronounced effects.

**Autoregulation Following Hypoxia and Hypercapnia.** At the end of the experiment in four animals of each group the animals subjected to severe trauma (2.4 and 4.1 atm) did not respond in the same manner to mild hypoxia. In four of them, slight reduction in CBF was seen. Return to normoxic conditions was associated with a decrease of CBF in all but one animal (a 13.2% decrease as compared with the level in the mild hypoxia group). Severe hypoxia caused a mean increase of CBF of 27.5%, but two animals (trauma of 2.5 and 2.7 atm) reacted paradoxically with a decrease of CBF and then with an increase of 22% and 67% when normoxia was restored. Three of these severely injured cats did not respond to a subsequent hypercapnic insult by cerebral vasodilatation, and the reaction in others was significantly depressed (mean increase in CBF of 14.9%). The results are presented in Table 3.

### Changes in MABP and ICP

The animals of the control and mild trauma groups reacted to hypoxic and hypercapnic insults with a slight increase in CBF. There was not such a tendency in severely traumatized animals. A similar dependence was seen in ICP. In two severely injured cats, with preserved arterial pressure and CBF reaction to hypoxia, ICP rose significantly during severe hypoxic insult.

### Autoregulation Following Hypoxia and Hypercapnia

At the end of the experiment in four animals of each group...
group, hemorrhagic hypotension was induced to a MABP of 80 mm Hg and then by 20-mm Hg increments. The data should be interpreted in the light of knowledge of prior episodes of hypoxia and hypercapnia.

Control cats had a stable CBF despite the reduction of MABP to 60 mm Hg. At this point, CBF fell in one animal that had been spontaneously hypertensive. In the remaining cats, CBF fell in a pressure-passive manner after the reduction of MABP to 40 mm Hg. All control animals had preserved cerebrovascular reactivity to hypoxia and to hypercapnia. The responsiveness of CBF to hypoxia and hypercapnia in the mild trauma group was only slightly depressed, and three of the four animals had preserved autoregulation until MABP was reduced to 60 mm Hg. In the severely injured group, in which hypoxic and hypercapnic cerebrovascular reactivity were impaired, autoregulation was partially preserved in two cats until MABP was reduced to 60 mm Hg.

Comparison of Cerebrovascular Response to Different Stimuli

Of the 18 cats in the control and mild injury groups in which the cerebral vasodilator response to hypoxemia was judged to have been preserved, 16 had an intact and one an impaired response to hypercapnia (Table 4). In eight of these animals, tests of hypotensive autoregulation were also carried out. This was judged to have been preserved until CPP fell to 40 mm Hg in four cats, to 60 mm Hg in two cats, and was frankly impaired in the other two cats.

In the 10 cats with an impaired cerebrovascular response to hypoxemia (less than 25% increase in CBF despite PaO₂ of 30 mm Hg), six had some hypercapnic response (more than 25% increase in CBF when PaCO₂ rose by 20 mm Hg) while four did not. Autoregulation was partially preserved (to 60 mm Hg CPP) in two of the four animals tested and impaired in the remaining two.

The effect of graded cerebral trauma on the cerebrovascular response to hypoxia and hypercapnia seems to be more predictable than on the response to arterial hypotension. Trauma tends to raise the autoregulatory pressure threshold. The relationship between CBF responsiveness to hypoxia and to hypercapnia and the severity of trauma is clearer, being significantly depressed only in more severely injured animals. The hypercapnic response is possibly more robust than the hypoxic response.

Effects of Hypoxia on Responsiveness to Hypercapnia

To test the effects of hypoxia on cerebrovascular reactivity to hypercapnia in normal and traumatized brains, hypercapnia (PaCO₂ = 50.0 ± 0.5 mm Hg) was applied prior to hypoxic insults in four animals of each group. Then, following severe hypoxia, a second hypercapnic stimulus (PaCO₂ = 52.0 ± 0.7 mm Hg) was applied. In control animals, CBF rose by 72.3% and by 92.8%, respectively, during pre- and post-hypoxic stimuli of hypercapnia.

Mild trauma animals reacted by smaller increases of CBF (25% and 41%, respectively) during the two episodes of hypercapnia. The difference in the percentage increase between pre- and post-hypoxic CO₂ stimulus can be partly explained by the slightly higher value of PaCO₂ during the second test of hypercapnia. Increases of CBF by 13.2% and by 6.8% were noted during two hypercapnic stimuli in the severe trauma group.

It appears that a hypoxic insult may further impair cerebrovascular responsiveness to hypercapnia in severely traumatized brain. Analysis of CVR supports this opinion.

Cerebrovascular Responsiveness in Relation to Severity of Trauma and Pathological Changes

Subarachnoid hemorrhage (SAH) and petechial hemorrhages in the brain stem were seen mostly in severely traumatized animals. No cats with trauma below 1.5 atm had visible neuropathological changes. Two cats with 1.7 atm of trauma had SAH and petechial hemorrhages in the brain stem, but reactivity to hypoxia and to hypercapnia was preserved. The remaining five cats with trauma below 2.0 atm had only mild pathological damage. Of 10 animals with trauma of 2.4 atm and above, six had severe hemorrhages. In two of them, CBF reacted somewhat to changes in PaO₂ and PaCO₂ while CBF responses were poor in four. On the other hand, two animals with only mild pathological changes did not respond normally to hypoxia and to hypercapnia. There was not a strict-ranking correlation between the degree of trauma and the neuropathological changes, nor between these two factors and reactivity of CBF to hypoxia and to hypercapnia.

Discussion

Variations in arterial pO₂ and pCO₂ are considered to be a chemical component of the CBF regulatory mechanisms. Both are supposed to act by influencing hydrogen ion concentration in the perivascular space, in smooth-muscle cells of the brain vessels, and in the cerebrospinal fluid.4,10

Moderate arterial hypoxia does not cause any sig-

**TABLE 4**

Comparison of cerebrovascular response to different stimuli*

<table>
<thead>
<tr>
<th>Response to Hypoxemia</th>
<th>No. of Cats</th>
<th>Response to Hypercapnia</th>
<th>Autoregulation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Intact</td>
<td>Impaired</td>
</tr>
<tr>
<td>intact</td>
<td>18</td>
<td>16</td>
<td>1</td>
</tr>
<tr>
<td>impaired</td>
<td>10</td>
<td>6</td>
<td>4</td>
</tr>
</tbody>
</table>

* See text for full description. Not all animals were fully tested.
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significant change in CBF, but more marked arterial hypoxia, with the oxygen tension approaching 50 mm Hg, decreases CVR and increases CBF. Hypoxemia begins to be a vasodilator stimulus only when oxygen saturation and content are beginning to be affected. Before this stage is reached, however, change in brain energy metabolism may be well advanced. The CBF increase appears to be a threshold phenomenon triggered by brain tissue lactacidosis. The threshold oxygen tension at which cerebral vasodilatation occurs is also influenced by such factors as respiratory acidosis, hypoglycemia, and cerebral edema. The CO2 reactivity is the more sensitive mechanism, so even slight variations in PaCO2 are reflected in rapid changes of the CBF.

In previous work from this institution, we concluded that mild trauma impaired, and severe trauma abolished, the CBF response to increased CO2. According to the concept that chemical controls of CBF by O2 and by CO2 are basically the same, one would expect that brain injury causing impairment of CBF reactivity to hypercapnia has a similar effect on CBF response to hypoxia. This proved to be largely the case.

The relationship between traumatic SAH and cerebrovascular responsiveness appears to be more variable than the relationship in the model of "pure" SAH. Although trauma caused an immediate and significant increase in MABP, we did not see any relationship between the level of this increase and the impairment of chemical regulation of CBF. It has to be stated, however, that all the animals in which an increase of more than 80 mm Hg in MABP was noted as an immediate result of trauma had subsequent difficulty with autoregulation when MABP was lowered below 80 mm Hg.

Our results also confirm that severe head trauma causes a reduction in CBF, as seen 1 hour after trauma. This reduction is proportional to the severity of the impact and to the reduction in perfusion pressure resulting from a combination of a small reduction in arterial pressure and a small increase in ICP. Reduction of CBF below baseline was also seen 2 to 20 minutes after concussive trauma in rats, as reported by Nilsson and Nordström. Brain injury impairs the CBF response to hypoxia and to hypercapnia. This impairment appears to be related more to the severity of trauma than to the severity of neuropathological changes. It is difficult to state which CBF regulatory mechanism (chemical or autoregulatory) is more sensitive to brain injury. In our studies, mild trauma was more likely to disturb the autoregulation before any change in hypoxic and hypercapnic response appeared. In a few severely traumatized animals, however, we noted severe impairment of the hypoxic and hypercapnic response with some apparent preservation of autoregulation. This has also been observed in experimental focal cryogenic injuries.

The clinical implications of this study are clear. The commonest secondary insult suffered by patients with severe head injury is hypoxemia. It is present in a third of comatose patients seen in the first 6 hours after injury. Its presence is associated with a poorer outcome from injury. Within areas of brain contusion, normal cerebrovascular responses to changes in arterial pressure, PaCO2, and PaO2 are known to be in abeyance so that cerebral arteries cannot dilate adequately. What this study suggests is that even in areas of brain not directly damaged, this loss of vascular response is also present during the first 6 hours after injury.

Concurrence of hypoxemia and loss of the appropriate cerebrovascular dilator response may be an explanation for the frequent occurrence of ischemic brain damage in the basal ganglia and cortex of patients with fatal head injuries. Thus, the importance of ensuring a free airway and optimum oxygenation in patients with head injury is underscored.

References


J. Neurosurg. / Volume 56 / March, 1982


Manuscript received June 29, 1981.

This work was supported by NIH Grant NS 12587 and USPHS Fellowship FO5 TW02703.

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