The Effect of Fluosol-DA on Oxygen Availability in
Focal Cerebral Ischemia

G. R. Sutherland, M.D., J. K. Farrar, Ph.D.,* and S. J. Peerless, M.D.

SUMMARY The effects of Fluosol-DA 35% (15 ml/Kg, IV) on cortical oxygen availability (O_2a), a relative measurement of cortical oxygen tension, were examined in 16 cats subjected to temporary middle cerebral artery (MCA) occlusion. The cats were divided equally into control and treatment groups and half of each group underwent MCA occlusion at room air ventilation and the other half at 100% O_2 ventilation. Prior to occlusion, Fluosol had no effect on arterial P_O_2 or cortical O_2a at normoxia, however, there was a significant increase in both arterial P_O_2 and O_2a following 100% O_2 ventilation (pre vs post Fluosol). In an additional 5 cats, Fluosol resulted in a transient decrease in blood oxygen content due to hemodilution. Concurrent to this decrease, cerebral blood flow increased substantially resulting in a net increase in oxygen delivery. Within ipsilateral hemispheres, MCA occlusion resulted in decreased O_2a to levels below the pre-occlusion normoxic values in all animals except those treated with Fluosol and ventilated with 100% O_2. During reperfusion, O_2a immediately recovered to hyperoxic levels in the Fluosol 100% O_2 animals, whereas in the other three groups, O_2a returned more gradually towards the pre-occlusion value. Since neither 100% O_2 alone nor Fluosol plus room air ventilation significantly improved O_2a in the ischemic cortex, we conclude that increased delivery of plasma plus Fluosol bound oxygen was responsible for the observed improvements in O_2a following MCA occlusion.

Although it has been suggested that Fluosol increases oxygen delivery to ischemic cortex, this has not been tested experimentally. Therefore, in this experiment, O_2a, a relative measurement of cortical oxygen tension using implanted noncalibrated electrodes, was measured from the cerebral cortex of cats prior to, during and following temporary MCA occlusion. The effect of Fluosol on O_2a in focal cerebral ischemia was evaluated in animals pretreated with Fluosol prior to occlusion.

Methods

Twenty-one adult cats (mean weight 3.1 ± 0.5 Kg) form the basis of this report. All were anaesthetized with ketamine HCl (30 mg/Kg intraperitoneally) and atropine sulfate (0.2 mg intraperitoneally). Each cat was intubated and artificially ventilated at normocapnia (PaCO_2 32 torr) following paralysis with gallamine triethiodide (0.3 mg/Kg). End-expired CO_2 was monitored continuously using a capnograph (Beckman, Model LB-2). Anaesthesia was maintained by hourly intravenous doses of ketamine HCl (6 mg/Kg) and gallamine triethiodide (0.2 mm/Kg). Twenty gauge polyethylene catheters were inserted into the left femoral artery and vein for continuous blood pressure recording, repeated assessments of arterial blood gases and the administration of maintenance fluid and electrolytes (Ringers' lactate 3 cc/Kg/hr).

Electrodes for the measurement of hydrogen clearance or O_2a were constructed from platinum wire 320 um, in diameter. The electrodes were sharpened electrochemically to a tip diameter of 15–20 um and were inserted into the cerebral cortex through small burr-holes using a microdrive. Three left hemisphere electrodes were inserted, one each into the anterior sylvian, the posterior suprasylvian, and the anterior marginal regions. One of the two contralateral electrodes was inserted into the posterior suprasylvian region and the other into the anterior marginal region (fig. 1). All electrodes were then sealed in place using cold-curing dental acrylic. A polarizing voltage of...
−600 MV (oxygen) or +350 MV (hydrogen) was applied between the platinum electrodes and a silver chloride reference placed in the shoulder and the output current was allowed to stabilize for a period of 60 minutes. The electrode outputs were amplified as described by Pasztor et al. and recorded on strip chart recorders (Brinkman, Model 2571).

For each CBF measurement, hydrogen was added to the inspired gas mixture for approximately 5 minutes and then abruptly discontinued. CBF was calculated from the initial portion of the clearance curve using the initial slope method. The O₂ electrodes provided a continuous monitor of cortical O₂a throughout the experiment.

In 5 control animals, baseline CBF measurements were obtained and Fluosol-DA 35% (15 ml/Kg) (Green Cross Corporation, Osaka, Japan) was injected intravenously. Additional CBF measurements were obtained at 5, 30, 60, 120 and 360 minutes. Blood samples were withdrawn at the appropriate intervals for blood gas analysis. Additional blood was withdrawn for measurements of hemoglobin concentration (Hb; gm/dl), O₂ saturation (%), hematocrit (Hct; vol %) for blood gas analysis. Additional blood was obtained at 5, 30, 60, 120 and 360 minutes. Blood drawn for measurements of hemoglobin concentration were obtained and Fluosol-DA 35% (15 ml/Kg) intravenous infusion with room air ventilation resulted in no change in the mean arterial blood pressure (MABP) (130 ± 10 vs 134 ± 12 mmHg: pre vs post), PaCO₂ (32 ± 1 vs 32 ± 2 mmHg) or PaO₂ (94 ± 8 vs 96 ± 7 mmHg). Ventilating with 100% O₂, however, resulted in an increase in the PaO₂ to 124 ± 8% of the pre-infusion value obtained at the same inspired oxygen tension (432 ± 16 vs 349 ± 21 mmHg). Secondary to a transient hemodilution, blood oxygen content decreased irrespective of the inspired oxygen tension. With room air, the O₂ content followed the resolution of hemodilution returning to pre-infusion values at 60 minutes. With 100% O₂ ventilation, O₂ content equaled the pre-infusion value at 30 minutes and plateaued at a value of 116 ± 2% of the pre-Fluosol measurement at 120 minutes. There was a

**Figure 1. Electrode placement.**

O₂ content = HbO₂ + plasma O₂ + Fluosol O₂

where:

\[
\text{HbO}_2 \text{ content} = 1.36 \times \text{Hb concentration (gm/dl)} \times \%
\]

\[
\text{Plasma O}_2 \text{ content} = 0.003 \times \text{PaO}_2 \text{ (mmHg)} \times (100-\text{Fct})/100
\]

Fluosol-DA 35% has a linear O₂ dissociation curve with a slope of 0.014 vol%/mmHg PO₂. Assuming that the aqueous component of Fluosol has an oxygen solubility equivalent to that of plasma (0.003 vol%/mmHg PO₂), the volume of oxygen carried by the fluorocarbon component can be expressed as:

\[
\text{Fluosol O}_2 \text{ content} = 0.034 \times \text{PaO}_2 \text{ (mmHg)} \times \text{Fct}/100.
\]

Oxygen delivery was calculated as the product of the O₂ content and CBF.

In 16 cats, the left MCA was exposed by transorbital approach and prepared for the application of a temporary vascular clip: 5–10 gm closing pressure. (Kees Surgical: Temporary clip) The O₂a electrodes were then inserted into the cerebral cortex, as described previously, and allowed to stabilize. Several measurements of the change in O₂a from room air ventilation to 100% O₂ ventilation were then obtained. The change in O₂a following this change in inspired oxygen tension was defined as the O₂a response. Subsequent changes in O₂a were measured as deviations from the baseline (normoxic) value expressed as a percentage of the control O₂a response.

The 16 cats were divided into two groups of eight. One group received Fluosol-DA 35% (15 ml/Kg intravenously) following which a series of O₂a responses was recorded. All animals then underwent temporary MCA occlusion during which half of each group were ventilated with room air and the other half with 100% O₂. The MCA clip was removed 60 minutes after its application and changes in O₂a recorded for 15 to 20 minutes.

Throughout the experiment, oxygen cycles were observed at all electrode sites and changes in their amplitude and frequency recorded. Blood gas analysis was performed following each change in inspired oxygen tension. In all cats, temperature was maintained at 38°C with a homeothermic blanket control.

The data is presented as mean values ±SEM and statistical comparisons were performed using an analysis of variance. For cats undergoing MCA occlusion, the data from electrodes 1 and 3 (ischemic region) and the data from electrodes 4 and 5 (contralateral hemisphere) were combined. The data from electrode 2 (border zone) was considered separately.

**Results**

Control Measurements

The measurements obtained from five control animals are presented in figure 2. Fluosol-DA 35% (15 ml/Kg) intravenous infusion with room air ventilation resulted in no change in the mean arterial blood pressure (MABP) (130 ± 10 vs 134 ± 12 mmHg: pre vs post), PaCO₂ (32 ± 1 vs 32 ± 2 mmHg) or PaO₂ (94 ± 8 vs 96 ± 7 mmHg). Ventilating with 100% O₂, however, resulted in an increase in the PaO₂ to 124 ± 8% of the pre-infusion value obtained at the same inspired oxygen tension (432 ± 16 vs 349 ± 21 mmHg). Secondary to a transient hemodilution, blood oxygen content decreased irrespective of the inspired oxygen tension. With room air, the O₂ content followed the resolution of hemodilution returning to pre-infusion values at 60 minutes. With 100% O₂ ventilation, O₂ content equaled the pre-infusion value at 30 minutes and plateaued at a value of 116 ± 2% of the pre-Fluosol measurement at 120 minutes. There was a
transient increase in CBF following Fluosol. CBF increased to 164 ± 8% of the pre-infusion control value at 5 minutes. At 30 minutes, CBF was still elevated at 131 ± 8% returning to a normal value at 120 minutes.

With the large increase in CBF outweighing the initial decrease in \( O_2 \) content, \( O_2 \) delivery was initially increased to 131% of the pre-infusion value during room air ventilation and to 151% during ventilation with 100% \( O_2 \). By 30 minutes, \( O_2 \) delivery had fallen to 118% with room air ventilation and 135% with 100% \( O_2 \) ventilation. This trend continued with \( O_2 \) delivery returning to pre-infusion levels by 60 minutes when ventilated with room air and continuing at a plateau of 116% with 100% \( O_2 \) ventilation.

**O\(_2\) Responses and Oxygen Cycles**

In the 16 cats prepared for MCA occlusion, initial baseline changes in \( O_2_a \) from room air to 100% \( O_2 \) ventilation (\( O_2_a \) response) were obtained. As the electrodes were not calibrated, all results are presented as a percentage of these baseline \( O_2_a \) responses. Oxygen cycles were found to increase in amplitude following this change in inspired oxygen tension (fig. 3).

When Fluosol was infused during room air ventilation, no significant changes in \( O_2_a \), oxygen cycle amplitude or frequency were observed (fig 3). As in the control animals, there were no significant changes in MABP, \( PaO_2 \) or \( PaCO_2 \). Ventilation with 100% oxygen resulted in an increase in \( O_2_a \) to 148 ± 12% above baseline (ie. 48% greater than the control response: \( p < 0.05 \)). \( PaO_2 \) also increased from 366 ± 17 mmHg (100% \( O_2 \), pre-Fluosol) to 435 ± 21 mmHg (an increase of 21 ± 4%). The increased \( O_2_a \) response was maximal immediately following Fluosol infusion and slowly decreased with time. However, the \( O_2_a \) response was still significantly elevated (33 ± 13%) two hours after the infusion. In addition, we noted an increase in the oxygen cycle amplitude as shown by the recording in figure 3.

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**FIGURE 2.** Control measurements: Illustrating the effect of a Fluosol-DA infusion (35%, 15 ml/Kg) on Hb concentration and oxygen content, plasma + Fluosol oxygen content, total oxygen content, oxygen delivery, and CBF at both room air (dotted line) and 100% \( O_2 \) ventilation, (solid line). The asterisk indicates a significant change from baseline values (\( p < 0.05 \)).

**FIGURE 3.** Electrode 1 output: Fluosol treated animal showing 3-10/min. oxygen cycles that increased in amplitude with 100% \( O_2 \) ventilation. The change in \( O_2_a \) from room air to 100% \( O_2 \) ventilation was defined as the \( O_2_a \) response. The infusion at room air ventilation had no effect on \( O_2_a \), however, the \( O_2_a \) response was significantly increased. MCA occlusion resulted in a fall in \( O_2_a \), however, not to ischemic levels and with release of the clip \( O_2_a \) quickly returned to the pre-occlusion value.
The effects of MCA occlusion on cortical O$_2$a in animals not pretreated with Fluosol are presented in figure 4 and table 1. MCA occlusion and subsequent release had no effect on O$_2$a in the contralateral hemisphere. Application of the clip resulted in a rapid fall in O$_2$a in the ipsilateral hemisphere and this decline was considerably greater in the ischemic regions than in the border zone. (p < 0.05 at 60 minutes post-occlusion) Ventilation with 100% O$_2$ did not significantly improve O$_2$a in the ipsilateral hemisphere during the occlusion (p > 0.05). Release of the MCA clip resulted in a rapid increase in O$_2$a. In the ischemic region, O$_2$a returned to pre-occlusion values (-8 ± 5%) by 15 minutes in the room air ventilated subgroup. In the 100% O$_2$ ventilated subgroup, O$_2$a initially increased to 95 ± 20% at 5 minutes and then decreased to 75 ± 24% by 15 minutes. In both subgroups, O$_2$a in the border zone returned to within 10% of the pre-occlusion values by 15 minutes.

The amplitude and frequency of oxygen cycles in the contralateral hemisphere were not affected by MCA occlusion. In the border zone electrodes, MCA occlusion resulted in a decrease in the amplitude of the oxygen cycles which then returned to control levels with reperfusion. In the ischemic region, the fall in O$_2$a during occlusion resulted in a decrease in amplitude of oxygen cycles in all electrodes and their disappearance in 65%. With release of the clip, oxygen cycles returned to their pre-occlusion amplitude and frequency in 30%. In the remaining electrodes, the oxygen cycles were markedly abnormal showing an increase in amplitude and low frequency.

**TABLE 1 Effects of MCA Occlusion on Oxygen Availability* in Control Animals**

<table>
<thead>
<tr>
<th>Electrode positioning</th>
<th>Ventilation</th>
<th>Control</th>
<th>Occlusion</th>
<th>Reperfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>5 min.</td>
<td>30 min.</td>
<td>60 min.</td>
</tr>
<tr>
<td>Contra lateral hemisphere</td>
<td>room air</td>
<td>0 ± 1</td>
<td>1 ± 1</td>
<td>-2 ± 2</td>
</tr>
<tr>
<td></td>
<td>100% O$_2$</td>
<td>98 ± 4</td>
<td>103 ± 3</td>
<td>100 ± 3</td>
</tr>
<tr>
<td>Border zone</td>
<td>room air</td>
<td>0 ± 3</td>
<td>1 ± 9</td>
<td>1 ± 9</td>
</tr>
<tr>
<td></td>
<td>100% O$_2$</td>
<td>10 ± 24</td>
<td>12 ± 30</td>
<td>9 ± 32</td>
</tr>
<tr>
<td>Ischemic region</td>
<td>room air</td>
<td>0 ± 1</td>
<td>-22 ± 32</td>
<td>-71 ± 24</td>
</tr>
<tr>
<td></td>
<td>100% O$_2$</td>
<td>100 ± 15</td>
<td>-51 ± 21</td>
<td>-81 ± 33</td>
</tr>
</tbody>
</table>

*Oxygen availability is given as the deviation from the baseline normoxic value expressed as a percentage of the control O$_2$ a response (see text for details). Data are presented as mean ± SEM.

**Figure 4.** The effect of MCA occlusion in animals not pretreated with Fluosol expressed as a percent of the pre-occlusion O$_2$a response showing significant variability between electrode 2 (border zone) and electrodes 1 and 3 sites (ischemic region) (p < 0.05 at 60 mins.) In both the room air (left) and 100% O$_2$ ventilated animals (right) O$_2$a fell to ischemic levels at electrode 1 and 3 sites with no significant difference between subgroups (p > 0.05). Release of the clip resulted in a return of O$_2$a to pre-occlusion values. Contralateral electrodes 4 and 5 remained unchanged during and following MCA occlusion.
The results obtained in animals pretreated with Fluosol are presented in figure 5 and table 2. As in the untreated animals, MCA occlusion had no effect on $O_2a$ in the contralateral electrodes. The fall in $O_2a$, which occurred in the ischemic region, was considerably greater than that in the border zone although this failed to achieve statistical significance due to the variability of responses. Oxygen availability in the ischemic region in cats ventilated with room air was not significantly different from non-Fluosol treated animals ($p > 0.05$). Ventilation with 100% $O_2$, however, resulted in a significantly lower reduction in $O_2a$ compared to the other three subgroups ($p < 0.05$ at 60 minutes). Release of the MCA clip resulted in a rapid increase in $O_2a$. In the ischemic region, $O_2a$ returned to pre-occlusion values ($7 \pm 18\%$) by 15 minutes in the room air ventilated subgroup. In the 100% $O_2$ ventilated subgroup, however, $O_2a$ increased to hyperoxic values of $174 \pm 32\%$ at 5 minutes and then decreased to $158 \pm 25\%$ by 15 minutes. In room air ventilated cats, $O_2a$ in the border zone returned to pre-occlusion values by 15 minutes ($-9 \pm 10\%$), however, in the 100% $O_2$ ventilated animals it increased to hyperoxic values of $158 \pm 28\%$ at 5 minutes and then decreased to $136 \pm 24\%$ by 15 minutes.

As in untreated controls, the amplitude and frequency of oxygen cycles in the contralateral hemisphere were not affected by MCA occlusion. In the border zone electrodes, MCA occlusion resulted in a decrease in the amplitude of oxygen cycles which then returned to control levels with reperfusion. In the ischemic region, the fall in $O_2a$ during occlusion resulted in a

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Effects of MCA Occlusion on Oxygen Availability* in Fluosol Treated Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrode positioning</td>
<td>Ventilation</td>
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<tr>
<td>Contralateral hemisphere</td>
<td>room air</td>
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<tr>
<td></td>
<td>100% $O_2$</td>
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<tr>
<td>Border zone</td>
<td>room air</td>
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<td>100% $O_2$</td>
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<td>Ischemic region</td>
<td>room air</td>
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<td></td>
<td>100% $O_2$</td>
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</table>

*See Table 1 for explanation of symbols.
decrease in the amplitude of oxygen cycles in all electrodes and, in the room air ventilated subgroup, their disappearance in 70%, whereas in the 100% O₂ ventilated subgroup, they disappeared in only 20%. With release of the clip, in room air ventilated cats, oxygen cycles returned to their pre-occlusion amplitude and frequency in 32%. In the 100% O₂ ventilated animals, the oxygen cycles returned to their pre-occlusion amplitude and frequency in 55%. In the remaining electrodes, the oxygen cycles were abnormal showing an increase in amplitude and low frequency.

Discussion

Control Measurements

A significant increase in CBF following the intravenous infusion of Fluosol-DA 35% (15 ml/Kg) was demonstrated. This observation was first made by Rosenblum in 1975 where he found a decreased Fluorescein transit time through the cerebral microcirculation in mice treated with a fluorocarbon emulsion (FC-80). More recently, Nagasawa et al found CBF to be increased by 12% following the infusion of Fluosol-DA 20% (8 ml/Kg) in the Macaca irus monkey. This increase in CBF is in all probability secondary to a decreased blood viscosity evoked through hemodilution by Fluosol. With an initial decrease in Hct, CBF increased, returning to baseline with resolution of the hemodilution by Fluosol. Although Fluosol has a viscosity significantly less than blood, the relatively small amount infused would not be expected to result in a significant decrease in the viscosity in the absence of hemodilution.

Since the amount of oxygen dissolved in blood greatly exceeds that in Fluosol, this hemodilution resulted in a decrease in blood oxygen content. With the resolution of hemodilution, blood oxygen content increased to pre-infusion levels. As the amount of O₂ dissolved in Fluosol at low PaO₂ is small, blood oxygen content returned to baseline during room air ventilation. However, Fluosol is capable of binding a relatively large volume of oxygen at high PaO₂ so that total oxygen content plateaued above baseline during 100% O₂ ventilation due to the addition of Fluosol bound oxygen. Since the increase in CBF outweighed the initial decrease in blood oxygen content, O₂ delivery initially increased at both inspired oxygen tensions. With resolution of hemodilution, O₂ delivery returned to baseline at the lower inspired oxygen tension, but showed a sustained increase with 100% O₂ ventilation due to the Fluosol bound oxygen (i.e. due to the sustained increase in total O₂ content).

O₂ Responses

In agreement with previous reports, we found that local PO₅ oscillates with a frequency of three to ten peaks per minute. Halsey and McFarland have suggested that these oscillations represent local variations in CBF (and hence, cortical pO₂) in response to a metabolic autoregulatory feedback mechanism constantly searching for an optimal local blood flow. The increase in cycle amplitude following an increase in arterial PO₂ both prior to and following Fluosol infusion should not be interpreted as a measure of increased metabolism or increased fluctuation in CBF. Under normal conditions, oxidative metabolism is closely coupled to tissue demands and would not be expected to change with an increase in arterial PO₂. Furthermore, changes in tissue metabolism would be expected to alter the cycle frequency whereas frequency was unchanged during variations in arterial PO₂ in the present studies. Mean CBF decreases with increasing arterial PO₂ but this effect is very small over the range of PaO₂ examined in the present study and is unlikely to affect the magnitude of CBF fluctuations about the mean. The most likely explanation for the increase in cycle amplitude would be that for a given change in blood oxygen content (constant metabolic demand, fluctuating flow), the associated change in PaO₂ increases with increasing values of mean PaO₂ due to the nonlinear nature of the O₂ dissociation curve. Since the cortical tissue will be in diffusion equilibrium with blood supplying that area, the nonlinear characteristics of blood (PO₂ vs oxygen content) would also be apparent in the tissue PO₂ recordings thus increasing the amplitude of the O₂ cycles.

Fluosol infusion did not affect cortical O₂a during room air infusion in spite of a transient increase in oxygen delivery (CBF x oxygen content). This observation suggests that cortical PO₂ is relatively insensitive to increases in oxygen delivery (i.e. increased CBF) through hemodilution at normoxia. This is in agreement with the results obtained by Crockard et al who found no change in cortical PO₂ during hypercapnia providing there were no concurrent changes in blood pressure. We found that Fluosol produced a 48% increase in the O₂a response to 100% O₂ ventilation. This enhanced O₂a response decreased with time but remained 33% greater than control two hours after infusion. CBF had returned to control levels at this time indicating that only a portion of the increase in O₂a may have been related to increased CBF. Fluosol also resulted in a sustained increase in arterial PO₂ (20-25%) during 100% O₂ ventilation and we would conclude that this increase in arterial PO₂ was responsible for the majority of the observed increase in cortical O₂a response.

MCA Occlusion

The maximal effect of occlusion on oxygen availability occurred in the anterior ectosylvian and posterior or suprasylvian regions and was significantly less within the anterior marginal region. As the anterior marginal region lies between the middle and anterior cerebral artery territories, it would be partially protected against MCA occlusion by leptomeningeal collaterals.

MCA occlusion resulted in an abrupt fall in cortical O₂a followed by a more gradual decline throughout the period of occlusion. When compared to the room air control animals, ventilation with 100% O₂ or treatment with Fluosol and room air ventilation did not alter O₂a significantly during occlusion and all three groups of
animals showed a severe fall in $O_{a}$ to hypoxic values. In contrast, $O_{a}$ remained at pre-occlusion normoxic levels in the Fluosol treated animals ventilated with 100% $O_{2}$ indicating a maintenance of oxygen delivery to the tissues. In the control animals, ventilation with 100% oxygen increased the oxygen content of blood by approximately 7% (1.2 ml/dl). However, the high viscosity of blood would restrict the amount of flow reaching the ischemic tissue through small collateral channels so that this change in oxygen content would not be expected to improve tissue oxygenation appreciably. As noted previously, the intravenous injection of Fluosol caused a marked increase in CBF of control animals, most likely as a result of decreased blood viscosity secondary to hemodilution. Nevertheless, increased flow — in itself — did not significantly improve oxygenation of ischemic tissue in the Fluosol, room air treated animals. The only significant difference between the Fluosol room air and 100% $O_{2}$ subgroups was in the amount of oxygen contained in the Fluosol + plasma component of blood. In the room air animals, this averaged 0.4 ml/dl (ie. 2% of total oxygen content in control animals) whereas in those ventilated with 100% $O_{2}$, the plasma + Fluosol component contained 2.2 ml/dl dissolved oxygen (13% of that contained in control animals). When combined with increased CBF, this relatively high volume of plasma + Fluosol bound oxygen appears to be able to prevent ischemic hypoxia in this model. This would suggest that red blood cells (which carry the majority of oxygen) cannot reach the ischemic region readily in the acute phase. During 100% $O_{2}$ ventilation, high $PO_{2}$ in normal tissue at the periphery of the ischemic zone may also have helped reduce the degree of hypoxia. Oxygen cycles were preserved at the majority of electrode sites in the Fluosol, 100% $O_{2}$ treated subgroup thus providing further evidence that metabolic activity (ie. attempts to regulate flow) was preserved in these animals.

In summary, these results indicate that Fluosol combined with 100% $O_{2}$ ventilation increased $O_{a}$ in both normal and ischemic tissue and appeared to enhance post-ischemic recovery. Since neither ventilating with 100% $O_{2}$ alone nor pretreating with Fluosol and ventilating with room air significantly improved $O_{a}$ in the ischemic cortex, we would conclude that increased delivery of plasma plus Fluosol-bound oxygen (ie. increased CBF and increased plasma plus Fluosol $O_{2}$ content) was responsible for the observed improvements in $O_{a}$ in the Fluosol 100% $O_{2}$ treated animals.

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