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Relationship of Somatosensory Evoked Potentials and Cerebral Oxygen Consumption During Hypoxic Hypoxia in Dogs

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SUMMARY The effects of hypoxic hypoxia on cerebral hemodynamics and somatosensory evoked potential (SEP) were studied in 10 pentobarbital anesthetized dogs. Cerebral blood flow (CBF) was measured using the venous outflow technique and cerebral oxygen consumption (CMRO$_2$) was calculated from the arterio-cerebro-venous oxygen difference times CBF. SEP was evaluated by percutaneous stimulation of an upper extremity nerve and was recorded over the contralateral somatosensory cortex. The latencies of the initial negative wave (N1), second positive wave (P2) and the amplitude of the primary complex (P1N1) were measured. Animals were breathed sequentially with oxygen concentrations of 21, 10, 6, 5, and 4.5% for five minutes each. Animals were returned to room air breathing when the amplitude of the SEP decreased to < 20% of control and were observed for 30 minutes following reoxygenation. Severe hypoxia (4.5% O$_2$) increased CBF to 200% of control, decreased CMRO$_2$ to 45% of control, decreased amplitude and increased latency of SEP. Following reoxygenation, as CMRO$_2$ increased toward control, latency of SEP decreased and amplitude increased and CBF returned to baseline within 30 min. During hypoxia and reoxygenation, the latencies of N1 and P2 and the amplitude of P1N1 were correlated with CMRO$_2$ in individual animals. We conclude that changes in SEP amplitude and latency reflect changes in CMRO$_2$ despite high CBF during rapidly progressive hypoxic hypoxia and following reoxygenation.

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Previous studies have not explored the relationship between oxygen delivery and changes in brain electrical activity. Verification of the relationship of cerebral oxygen delivery and brain electrical activity allows assessment of adequacy of oxygen availability and utilization when cerebral blood flow (CBF) and other important factors such as blood oxygen carrying capacity cannot be measured. Studies of non-oligemic insults on SEP have failed to include information on CBF or cerebral oxygen consumption (CMRO$_2$). It is therefore unclear if a consistent relationship of changes in CMRO$_2$ and SEP exists.

We studied the relationship of CMRO$_2$ and SEP in a model in which CBF increases in response to cerebral oxygen deprivation to test the hypothesis that somatosensory evoked potentials reflect oxygen availability to the brain regardless of CBF.

Methods

Ten adult mongrel dogs (20–25 kg) of either sex were utilized in this study. Anesthesia consisted of sodium pentobarbital (30 mg/kg, i.v.) supplemented with increments of pentobarbital (60 mg, i.v.) as nec-

SOMATOSENSORY EVOKED POTENTIALS assess the nervous system from peripheral nerve to cortex. Clinical use emphasizes discrete lesions of the nervous system, brachial plexus,¹ brainstem,² spinal cord³ and hemisphere.⁴,⁵ Somatosensory evoked potential (SEP) is altered by systemic insults such as oligemia⁶,⁷ anemia,⁸ intracranial hypertension,⁹ and hypoxia.¹⁰ The changes in superficially recorded waves during systemic oxygen deprivation⁶,⁸,¹⁰ are similar to those recorded when the cortical region (somatosensory cortex) is directly involved¹¹ and therefore may be as useful in quantitating the degree of insult in generalized insult as in localized insult.
essary during the surgical preparation in response to
pedal and ocular reflexes. Pancuronium bromide (3–4
mg, i.v.) was administered to minimize muscle con-
tractions related to the electrocautery. Heparin (500
µg/kg, i.v.) was used as the anticoagulant.

After induction of anesthesia, animals were intubat-
ed and ventilated utilizing a positive pressure respira-
tor (Harvard respiration pump 607). Tidal volume and
respiratory rate were adjusted to give an alveolar (end-
expiratory) carbon dioxide of 4% as monitored by a
CO₂ analyzer (Beckman LB2). The CO₂ analyzer was
calibrated regularly with mixtures of CO₂ in air ana-
lyzed to a precision of 0.1%. Electrocautery was used to
expose one femoral artery and both femoral veins.
The femoral artery was cannulated for continuous
monitoring of mean arterial blood pressure (MABP).
One femoral vein was cannulated and was utilized to
return cerebral venous outflow while the other femoral
vein was cannulated and used for infusion of fluids and
drugs. Rectal temperature was maintained at 38° ± 1
centigrade using heating lamps. All pressures were
measured with Statham P-23 transducers, and all data
were recorded on a Gould-Brush recorder.

Measurement of Cerebral Blood Flow

The technique used to measure cerebral venous out-
flow has been described by Rapela and Green. The
confluence of the cerebral sinuses was cannulated and
the lateral sinuses and occipital emissary veins were
occluded with bone wax to minimize communication
between intracranial and extracranial venous circula-
tions. From the confluence of the sinuses blood then
passed through a previously calibrated electromagnetic
flow probe, before returning to the dog via the femoral
vein. With this technique approximately 50–70% of
the mass of the brain is drained at the confluence of
the sagittal and straight sinuses. Cerebral venous outflow
pressure was measured upstream from the flow probe.
This pressure measures the resistance to the flow of
blood induced by the flow transducer because the out-
flow cannula was set at the level of the right atrium and
all pressures were referred to this common zero refer-
ence plane. Brain perfusion pressure was estimated as
systemic arterial pressure minus cerebral venous out-
flow pressure. Intracranial vascular resistance was cal-
culated by dividing brain perfusion pressure by cere-
bral venous outflow.

The verification of the measurement of CBF utiliz-
ing this venous outflow technique has been described in
detail elsewhere. In addition, the viability and
responsivity of the cerebral vasculature to hypercap-
nia, hypoxia, and ability to autoregulate using this
technique has been previously demonstrated.

Measurement of Somatosensory Evoked Potential (SEP)

Stimulating needle electrodes were placed percuta-
neously in the volar surface of a foreleg in a location
which caused a distinct digital twitch and the stimulus
intensity just sufficient for a motor response (motor
threshold) was determined. The needles were secured
and a large surface ground pad was attached to the
extremity proximal to the stimulating electrodes. Sil-
ver ball electrodes with shielded cables were placed in
depressions drilled in the skull over the contralateral
somatosensory cortex. A needle electrode with shield-
ed cable was placed in the snout and acted as reference.
The junction of the lambdoidal and sagittal suture is an
easily indentifiable landmark in the dog and was chosen
as an anatomical reference point. Two parallel rows
of electrode locations were examined in each animal.
One row consisted of electrode locations 2 cm from the
midline, with electrode locations 4, 6, and 7.5 cm ante-
rior to the lambdoidal suture. A second row was
located 4 cm from the midline posteriorly and 3.5
cm from the midline anterior. Electrodes were placed
to 4, 6, and 7.5 cm anterior to the lambdoidal suture. In
each animal, the electrode location with maximum
amplitude was considered to be nearest the somatosen-
sory cortex and was analyzed for this study.

The SEP was developed using a 4 channel signal
averager (Nicolet Med 80, Nicolet Biomedical, Madi-
son, Wisc). A stimulus intensity twice motor threshold
and a stimulus duration of 150 usec was used. One
hundred twenty eight stimuli were delivered at a rate of
5.9/sec and averaged. Upper and lower band pass fil-
ters were 5 and 1500 Hz respectively. Waveforms
were stored on magnetic disk for later analysis. In the
control period, replicate waves were generated to en-
sure stability of the waveform. High amplitude electrical
artifact was automatically rejected by the comput-
er. The peripheral nerve was stimulated only during
study periods (approximately 45 sec each).

This active electrode and reference system yields a
consistent wave-form in the dog. The waveform con-
ists of a small positive wave (P1) and 15 ms after
stimulus, a large negative wave (N1) and about 20–25
ms after stimulus and a large positive wave (P2) occur-
ing 35–45 ms after stimulation. The amplitude and
latency of the waveform were evaluated using the cur-
sor mode of the computer. The latencies of the first
e positive (N1) and second positive (P2) waves were
determined. The latency was measured at the midpoint
of the wave. The amplitude of the initial complex
(P1N1) was measured from the maximum positive de-
flection of the initial positive wave (P1) to the max-
imum negative deflection at N1. A representative wave
is provided (fig. 1).

A single bipolar EEG channel was monitored ipsi-
lar to the stimulated extremity. One electrode was
placed posteriorly and the anterior electrode was
placed in the same coronal plane as the active SEP
electrode.

Hypoxia Administration and Blood Gas Analysis

Animals were subjected to hypoxic hypoxia by ad-
ministration of a mixture of air and nitrogen. The in-
spired oxygen concentration was measured utilizing an
oxygen analyzer (Beckman LB-2). The inspired oxy-
gen concentration was decreased sequentially from 21
to 10, 6, 5, and 4.5% and each level of hypoxia was
maintained for approximately 5 minutes or until the
SEP was suppressed to < 20% of control. Endtidal
This degree of SEP suppression usually occurred when CO₂ was maintained at 4% by altering the ventilator stimuli delivered at 5.91 sec.

Degree of hypoxia (between 6 and 4.5% inspired oxygen) caused a decrease in MABP. The animals were then returned to room air breathing. Arterial and cerebral venous blood samples were obtained from the femoral artery and cerebral venous cannulae respectively at the mid-point of the SEP determination. SEP acquisition required 30–45 seconds.

A final set of data (for hypoxia) was obtained when SEP amplitude was decreased to < 20% of control. This degree of SEP suppression usually occurred when the EEG was flat or showed burst suppression. This degree of hypoxia (between 6 and 4.5% inspired oxygen) caused a decrease in MABP. The animals were then returned to room air breathing. Arterial and cerebral venous blood samples and SEP waveforms were obtained 2, 4, 6, 8, 10, 15, 20, and 30 minutes following reoxygenation. Again, blood samples were obtained at the midpoints of each SEP determination.

Arterial O₂ tension (PaO₂), CO₂ tension (PaCO₂), and pH were measured at 37°C immediately after the samples were obtained by use of Radiometer BMS-3 electrodes and analyzer. The electrodes were calibrated with air (20.8% O₂) and mixtures of O₂ in N₂ (8%) and CO₂ in air (5 and 10% CO₂) to a precision of 0.1%. The pH electrode was calibrated with standard phosphate buffers (6.840; 7.381). O₂ saturation and hemoglobin were also measured immediately after samples were taken with an Instrumentation Laboratories Co-Oximeter (Model 282). Electrodes were calibrated before and after each set of samples were analyzed. This instrument computes oxygen content from the saturation and hemoglobin. Cerebral oxygen consumption (CMRO₂) was calculated by multiplying the arterial (CaO₂) to cerebrovenous (CvO₂) O₂ content difference by CBF. Fractional O₂ extraction was calculated using the formula:

\[
\frac{\text{CaO}_2 - \text{CvO}_2}{\text{CaO}_2}
\]

Data in the text and figures are presented as mean ± standard error. Regression analysis was performed using a microcomputer to compare changes in waveform parameters (latency of N₁, P₂ and amplitude of N₁P₂) with changes in CMRO₂. Because the absolute rate of change in CMRO₂ was similar during hypoxia and recovery, a single slope was estimated for each animal. Variance components analysis was used to calculate confidence intervals for each parameter. This statistical technique acknowledges two sources of uncertainty in the slope estimate for a given animal: the first being the measurement errors in that animal’s data; the second being the naturally occurring differences in the rates among animals.

Results

Hypoxia

Decreasing the inspired O₂ concentration from control (21%) to 10, 6, 5 and 4.5%, decreased PaO₂ from 92 ± 5 to 31 ± 1, 19 ± 1, 17 ± 1 and 14 ± 1 mmHg, respectively (table 1). Fractional O₂ extraction increased markedly from control (0.47 ± 0.02) at a PaO₂ of 92 ± mmHg, to 0.74 ± 0.02 at a PaO₂ of 14 ± 1 mmHg. pH and PaCO₂ were unchanged throughout the different O₂ levels. The cerebral hemodynamic changes with hypoxia are shown in figure 2. MABP increased from 127 ± 4 to 150 ± 6 mmHg as PaO₂ was lowered from control to 31 ± 3 mmHg but decreased markedly when PaO₂ was lowered to 14 ± 1 mmHg. CBF increased markedly to 240% of control at

| Table 1 Blood Gas Changes During Hypoxia and Recovery |
|----------------------------------------|----------------|----------------|----------------|
| Inspired oxygen concentration          | pH             | PaO₂ (mm Hg)  | PaCO₂ (mm Hg)  | Fractional extraction |
| 21%                                    | 7.40 ± 0.01    | 92 ± 5        | 30 ± 1         | 0.47 ± 0.02         |
| 10%                                    | 7.43 ± 0.01    | 31 ± 1*       | 31 ± 1         | 0.55 ± 0.02*        |
| 6%                                     | 7.46 ± 0.01    | 19 ± 1*       | 30 ± 1         | 0.56 ± 0.03*        |
| 5%                                     | 7.43 ± 0.01    | 17 ± 1*       | 31 ± 1         | 0.65 ± 0.04*        |
| 4.5%                                   | 7.37 ± 0.01    | 14 ± 1*       | 31 ± 1         | 0.74 ± 0.02*        |

| Recovery                               | pH             | PaO₂ (mm Hg)  | PaCO₂ (mm Hg)  | Fractional extraction |
| 2                                       | 7.28 ± 0.1*    | 71 ± 7*       | 36 ± 2*        | 0.13 ± 0.02*         |
| 4                                       | 7.29 ± 0.1*    | 86 ± 4        | 33 ± 2         | 0.15 ± 0.03*         |
| 6                                       | 7.30 ± 0.1*    | 85 ± 4        | 33 ± 2         | 0.20 ± 0.03*         |
| 8                                       | 7.31 ± 0.1*    | 87 ± 5        | 31 ± 1         | 0.23 ± 0.04*         |
| 10                                      | 7.31 ± 0.1*    | 88 ± 5        | 32 ± 1         | 0.25 ± 0.02*         |
| 15                                      | 7.32 ± 0.1*    | 86 ± 6        | 31 ± 1         | 0.35 ± 0.04*         |
| 20                                      | 7.33 ± 0.1*    | 86 ± 5        | 30 ± 1         | 0.44 ± 0.04         |
| 30                                      | 7.35 ± 0.1     | 87 ± 5        | 31 ± 1         | 0.47 ± 0.04         |

* = P < 0.05.
Hemodynamic Changes Following Return To Room Air

\( n = 10; \bar{x} \pm \text{SEM} \)

- Mean Arterial Blood Pressure (mmHg)
  - 200
  - 150
  - 100
  - 50
  - 0
- Cerebral Blood Flow (ml/min)
  - 60
  - 40
  - 20
  - 0
- Cerebral Oxygen Consumption (ml/min)
  - 3.0
  - 2.5
  - 2.0
  - 1.5
  - 1.0
  - 0.5
  - 0

TIME AFTER RETURN TO ROOM AIR (minutes)

Figure 2. Hemodynamic and cerebrovascular changes to a progressive decrease in inspired oxygen concentration (21, 10, 6, 5, and 4.5% \( \text{O}_2 \)) is shown \( n = 10, \text{mean} \pm \text{SEM} \). Animals were returned to room air when the amplitude of SEP decreased to less than 20% of control. Differences from control value \((* = p < .05)\) were evaluated using analysis of variance for repeated measures.

\( \text{PaO}_2 \) of 19 ± 1 mmHg but then decreased to 160% of control as \( \text{PaO}_2 \) was reduced to 14 ± 1 mmHg. CMRO₂ was reduced (75% of control) at \( \text{PaO}_2 \) of 19 ± 1 mmHg and was 54 and 36% of control at \( \text{PaO}_2 \) of 17 ± 1 and 14 ± 1 mmHg, respectively. Two minutes following the return of the animal to room air breathing, \( \text{PaO}_2 \) was increased to 71 ± 7 mmHg and by 4 minutes was at the control value (86 ± 4 mmHg) (table 1). Brain oxygen extraction did not return to normal until 20 minutes after reoxygenation (table 1). Following reoxygenation, CBF was elevated and required 20 min to return to its control value, whereas CMRO₂ had returned to control by 10 min (figure 3). MABP decreased immediately after reoxygenation and remained unchanged over the 30 minute period.

The relationship between amplitude (P1N1) and latency (N1, P2) of the SEP and CMRO₂ during hypoxia and recovery is shown in figure 4. Hypoxia (\( \text{PaO}_2 = 14 \pm 1 \text{ mmHg} \)) decreased P1N1 amplitude to 17% of control, increased N1 latency to 111% of control, and increased P2 latency to 107% of control. As \( \text{PaO}_2 \) decreased to the lowest level (14 ± 1 mmHg), EEG progressively slowed and showed electrocortical silence or prolonged burst suppression (5–10 sec) occurred when the SEP amplitude was decreased to 17% of control. Two minutes after reoxygenation, P1N1 amplitude had increased to 53% of control while N1 latency was still elevated at 112% of control, and P2 latency was 109% of control (fig. 4). Thirty minutes after reoxygenation, P1N1 amplitude, and N1 and P2 latency had returned to control values.

Table 2 shows the slope of changes in CMRO₂ and changes in latency of N1 and P2 and amplitude P1N1 in individual animals during both hypoxia and recovery. It can be seen that the slopes of these responses are similar in the group of animals and that a correlation exists in each animal. The values during hypoxia and recovery were pooled in each animal. The average slope of latency N1 versus CMRO₂ is \(-0.46 \pm 0.19\) (95% confidence interval), P2 versus CMRO₂ is \(-0.19 \pm 0.13\), and amplitude vs CMRO₂ N1P2 is \(0.15 \pm 0.06\).

Discussion

The model of cerebral oxygen deprivation (hypoxic hypoxia) differs from other models used to assess changes in SEP, oligemia, and increased tissue pressure because increases in CBF may preserve oxygen delivery to the brain despite low oxygen content of blood. Previous studies have related SEP and CBF without assessment of CMRO₂. In those studies, the insult was delivered over a prolonged period of time and the experimental design excluded cerebral com-
Return with normalization of O$_2$ delivery and good correlation with neurological outcome has been shown. The present experiments evaluated the adequacy of O$_2$ delivery and its effect on SEP in a rapidly changing situation. The stress of O$_2$ deprivation was rapid and reoxygenation was accomplished quickly at a point of exhaustion of cerebral compensatory mechanisms (increased CBF). Lack of equilibration could appear to decrease the relationship of the changes in cerebral O$_2$ uptake and parts of the SEP waveform. However, despite the potential for a non steady state condition, changes in SEP were well correlated with CMRO$_2$. The parameters used to acquire SEP data allowed data acquisition in 30–45 seconds and should minimize the effect of changes of neural function occurring during the data acquisition period. It is unlikely that oxygen deprivation of peripheral structures (peripheral nerve or spinal cord) contributed to the SEP changes noted. The spinal cord components of SEP are less sensitive than cortical components during ischemia and during hypoxic hypoxia. The SEP was evaluated for changes of both amplitude and latency. Latency of waves from peripheral nerve to the cortex is a function of white matter and the amplitude of the cortical waves is primarily a function of gray matter. Amplitude of SEP is easier to evaluate visually than latency, particularly in a situation of rapid change. Early parts of the SEP wave, representing arrival of the afferent volley at the cortex, were chosen for evaluation because of their constancy in this preparation and the resistance of these early wave to anesthetic drugs which make them preferable to later waves for intraoperative or intensive care monitoring. Our studies demonstrate that changes in wave latency correlated well with CMRO$_2$. The delay from peripheral nerve to cortex increased as O$_2$ availability was decreased and then decreased toward normal as O$_2$ became available following reoxygenation. The change in latency N1 from room air to severe hypoxia was 11% and the latency of P2 was changed by 8%. The magnitude of amplitude change was much greater (84% decrease) and changes in amplitude (pN1) appears to be a more reliable indicator than changes in latency (as indicated by a more narrow confidence interval of the slope). For rapid assessment of CMRO$_2$ change, amplitude appeared preferable because of the much larger change.

The recovery phase of this experiment is particularly important. In this circumstance, changes in SEP correlated with decreased CMRO$_2$ due to metabolic impairment, when cerebral O$_2$ delivery was normal. Hence, alteration in CBF and cerebral O$_2$ availability do not alter SEP unless CMRO$_2$ is decreased whether due to limited O$_2$ availability or metabolic impairment. A potential area of usefulness of SEP monitoring is the brain injured patient. Since SEP is related to brain metabolic function, regardless of CBF or cerebral oxygen availability, it may be a useful tool to assess brain function during the period following brain injury in which hyperemia may be followed by oligemia. Hence a measure of function (SEP) might be more useful than compensatory mechanisms such as an increase in CBF in response to cerebral oxygen deprivation. Adequate cerebral O$_2$ delivery and utilization (CMRO$_2$) can be maintained at low PaO$_2$ if oxygen carrying capacity is adequate and CBF can increase sufficiently. In addition to an increase in CBF, an increase in fractional oxygen extraction also serves to maintain cerebral oxygen delivery constant in a variety of circumstances.

Since the venous outflow assesses both blood flow and metabolism from the cerebral hemispheres, values determined by this method should reflect the environment in the cortical area of generation of the cortical SEP waves.

In this study, parameters used to generate the SEP (band pass filter; number of stimuli) were chosen to emphasize the cortically generated waves. A recent report demonstrates that cortical components of the SEP are more sensitive to oligemia than are subcortical components. Additionally, failure of cortical SEP components and spontaneous cortical activity (EEG) at a similar level of oxygen deprivation is consistent with previous reports. Transient loss of SEP with rapid

**Figure 4.** Relationship of SEP amplitude and latency and cerebral oxygen consumption during hypoxic hypoxia and recovery ($n = 10$; mean ± SEM). The relationship of SEP amplitude and latency are plotted for each level of hypoxia (21, 10, 6, 5, and 4.5% O$_2$) and following reoxygenation (2, 4, 6, 8, 10, 15, 20 and 30 minutes).
CBF measurement in such circumstances. Validation of a clinically useful non-invasive method of assessing return of CNS function regardless of CBF will allow therapeutic manipulation to be based on such evaluation. The use of the SEP as a monitor gives more information than intracranial pressure, cerebral perfusion pressure or even CBF concerning brain function.

The cerebrovascular, cerebral metabolic and electrical (EEG) responses and recovery to hypoxic hypoxia are similar to those noted previously although we produced a more severe degree of hypoxia. The degree of hypoxic stress in our study was greater than in that study by effects on both CMRO₂ and EEG amplitude. The time course of return of brain high energy compounds to control reported previously are similar to the return of the brain’s ability to use O₂ (fractional oxygen extraction) in the present study. We have shown that the SEP reflects cerebral O₂ deprivation, even in a non-steady state situation.

It is important to note that both return of CBF and the ability of the brain to extract oxygen (fractional extraction) returned slowly toward control. Indeed, correlation of SEP components with CMRO₂ when the brain was metabolically impaired (increase CBF, decrease ability to extract oxygen) substantiates the importance of SEP as a monitor of O₂ adequacy. Studies using oligemic methods stress a pressure threshold of available oxygen.

SEP and correction of the abnormality will return the SEP toward normal. Indeed, uses of changes of SEP to evaluate therapy appears useful when other currently used methods are inadequate or not feasible. SEP evaluation is possible without movement of the patient to special facilities, and is useful at a stage of disease when other methods are too complicated (PET Scanner), or provide inadequate information concerning CBF, CMRO₂, or cerebral perfusion pressure.

In the brain injured patient, normal and acceptable values of MABP, ICP, CBF are determined based on the expected values for the patient rather than knowledge of normal, “control”, values for that patient. In the same context, “normal” values for brain electrical activity must frequently be presumed and therapy maneuvers undertaken to return the values toward that presumed normal value. Indeed, we and others, have used non-normal, that is SEP altered by anesthetist agents to prevent or diagnose neurological injury.

We have shown that evoked electrical activity of the brain is progressively altered by O₂ deprivation when CMRO₂ is decreased and returns to normal as the brain’s ability to utilize oxygen is restored. This suggests that SEP monitoring may be useful during oxygen deprivation and recovery to assess adequacy of cerebral O₂ delivery and the ability of the brain to use available oxygen.

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