greatly prolonged over that found in normal cats (unpublished observations). Furthermore, the administration of dexamethasone at the same time as pentobarbital in cats subjected to acute cerebral ischemia results in a plasma half-life of pentobarbital which is not significantly different from normal (unpublished observations). Dexamethasone, however, does not reduce the volume of brain infarction. Therefore, it appears that dexamethasone may counteract adrenal insufficiency in one of the following two ways (or a combination of them): (1) It may supply additional circulating glucocorticoids and; (2) by reversion of the half-life of pentobarbital to its normal duration in LMCA occluded cats, it may permit the adrenal cortex to recover more quickly from the barbiturate suppression.

In view of the deleterious side effects of the administration of high doses of pentobarbital, we feel a clinical trial of barbiturate therapy in acute cerebral ischemia must await the development of a protocol which avoids the severe drug-induced complications which accompany high plasma barbiturate concentrations. Furthermore, a thorough understanding of the altered pharmacokinetics of pentobarbital in cerebral ischemia must be secured before clinical trials in this therapeutic area can begin.

REFERENCES


Rat Brain Osmolality During Barbiturate Anesthesia and Global Brain Ischemia

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SUMMARY Ischemic brain damage can be partially ameliorated by barbiturate therapy applied postinsult. Catabolism-induced brain hyperosmolality during ischemia may contribute to the development of brain edema after restoration of circulation. To determine changes in brain osmolality during ischemia and the effect of barbiturate anesthetics in altering its course, we measured whole and regional (cerebral cortex, diencephalon-midbrain, and cerebellum) brain osmolality for up to 2 hours after decapitation ischemia in unanesthetized and pentobarbital anesthetized rats. Normal (nonischemic) brain osmolality in pentobarbital anesthetized rats was 319 ± 2 mOsm/l (mean ± sem) and higher than in unanesthetized rats (307 ± 6 mOsm/l). The rate of increase in whole brain osmolality was 60% slower in pentobarbital anesthetized rats in the first 60 minutes of ischemia and regional brain osmolality increased by a maximum of 32 mOsm/l compared to 45 mOsm/l in unanesthetized rats. The potential for edema based on percent change in brain osmolality as well as the rapidity of the change was greater in unanesthetized rats. The significance of the increase in brain osmolality with barbiturate anesthesia and its attenuation of the rate and magnitude of increase during ischemia is discussed.

INCREASED INTRACRANIAL pressure (ICP) after cerebral ischemic anoxia adds to brain damage sustained after the initial insult often leading to brain death with complete cessation of cerebral blood flow (CBF). The pathophysiological and biochemical mechanisms leading to water imbibition by the brain are poorly understood. During ischemic anoxia, rapid biochemical changes occur with failure in maintenance of the energy state of the brain. A 7-fold increase in glycolytic rate, lipolysis occurs while brain high energy phosphates fall. These metabolic alterations eventually lead to lysosomal enzyme activation and cytolysis and could result in a tremendous increase in solute concentration and osmolality leading to brain edema after restoration of circulation.

One approach toward an understanding of the pathogenesis of postischemic encephalopathy is to study pathophysiological and biochemical processes with and without therapies of proven benefit. Earlier studies suggested increased tolerance of the brain to hypoxia and ischemic anoxia during anesthesia. More recent studies in animal models of stroke demonstrated the efficacy of barbiturate administered postischemia. Bleyaert, et al. demonstrated remarkable neurologic recovery with thiopeptinal therapy in a monkey model of 16 minutes global brain ischemia with long-term survival. Smith, et al. showed that while barbiturate anesthetics prevented the development of brain edema after cortical freeze lesions in dogs, inhalation anesthetics (isoflurane, enflurane, and halothane) were in-
effective. These studies clearly demonstrate the efficacy of barbiturate anesthetics in ameliorating ischemic brain damage. Our aim in this study was to determine the magnitude and time course of brain osmolality changes during ischemia and the effect of barbiturates.

We studied whole and regional (cerebral cortex, diencephalon-midbrain and cerebellum) rat brain osmolality changes after various durations of global brain ischemia (decapitation) in unanesthetized and sodium pentobarbital anesthetized rats. Whole brain osmolality in sodium pentobarbital anesthetized rats was higher than in unanesthetized rats before ischemia. The rate and magnitude of the increase in whole and regional brain osmolality during global ischemia was attenuated during pentobarbital anesthesia.

Methods

Wistar albino rats 250 to 500 g body weight maintained *ad libitum* on Purina Chow and water were used either unanesthetized or anesthetized with sodium pentobarbital (60 mg/kg IP). Rectal temperature was monitored in anesthetized rats and kept between 37-39°C by an electric heating blanket. Global brain ischemia was induced by decapitation in unanesthetized or pentobarbital anesthetized rats. The head was immediately wrapped in a saline-soaked sponge prewarmed to 37°C for 4, 8, 16, 20, 30, 60, 90 and 120 minutes. One minute before the specified duration of ischemia the head was removed from the oven, the brain rapidly removed and immersed in liquid N2 to terminate the ischemic episode. The frozen brain was placed into a preweighed test tube at 0°C and immediately covered with Parafilm and weighed. Two ml's of 0.9% NaCl was added and the brain mechanically homogenized (Tri-R homogenizer) for 25 seconds, centrifuged at 1,200 X g (0°C) for 10 minutes and supernatant osmolality measured.

In a separate series of studies, we measured osmolality changes in cerebral cortex, cerebellum and diencephalon-midbrain. The procedures were as previously described for whole brain except that the different regions of the brain were separated prior to freezing in liquid N2 to terminate the ischemic episode. The frozen brain was placed into a preweighed test tube at 0°C and immediately covered with Parafilm and weighed. Two ml's of 0.9% NaCl was added and the brain mechanically homogenized (Tri-R homogenizer) for 25 seconds, centrifuged at 1,200 X g (0°C) for 10 minutes and supernatant osmolality measured.

In a separate series of studies, we measured osmolality changes in cerebral cortex, cerebellum and diencephalon-midbrain. The procedures were as previously described for whole brain except that the different regions of the brain were separated prior to freezing in liquid N2. The volume of 0.9% NaCl added to the test tubes containing the regional samples was adjusted to maintain a constant ratio of tissue to 0.9% NaCl volume as used for whole brain.

For control (no ischemia) brain tissue osmolality determinations in unanesthetized and pentobarbital anesthetized rats, a rapid brain sampling technique recently developed in our laboratory was used. The rats were placed in a supine position with the skull resting on 2 serrated blades welded in the form of a cross. A stainless steel basin containing liquid N2 was secured under the blades and the rats' head smashed on to the blades by a rapid blow with a rubber mallet tossing the brain (usually in quadrants) into the liquid N2. Brain osmolalities were measured using the techniques previously described.

Osmolality measurements were made by an Osmette A, automatic osmometer (Precision System, Inc.). The osmometer was calibrated with 100 and 500 milliosmole (mOsm/l) standards, prior to and after sample measurements. The osmolality of 0.9% NaCl solution added to the brain tissue was also measured. Brain tissue water osmolality was calculated from the measured supernatant osmolality according to the following formula:

\[
X = \frac{(A + (B \times C)) \times (Y - (D \times A))}{(B \times C)}
\]

Where:

- \(X\) = Brain H2O osmolality (mOsm/l)
- \(A\) = Volume 0.9% NaCl added to brain tissue (mls)
- \(B\) = Brain weight (grams)
- \(C\) = Brain tissue H2O content (0.78)
- \(Y\) = Measured osmolality of sample (mOsm/l)
- \(D\) = Osmolality of 0.9% NaCl solution (mOsm/l)

Brain tissue H2O content is assumed to be constant because of decapitation which precludes the imbibition of H2O by the brain (i.e., development of brain edema) although extracellular H2O shifts may occur.

Results

Normal (nonischemic) unanesthetized rat whole brain osmolality was 307 ± 6 mOsm/l (mean ± SEM) (fig. 1A). Following the first significant \((P < .01)\) rise at 30 minutes, whole brain osmolality increased to 339 mOsm/l or 32 mOsm/l higher than normal after 90 minutes of ischemia. The rate of increase in whole brain osmolality was about 0.5 mOsm/l/min in the first 60 minutes of ischemia.

![Graph](image-url)

**Figure 1.** Brain tissue osmolality in unanesthetized rats during decapitation-induced global brain ischemia in: (A) whole brain (WB), and (B) cerebral cortex (CC), diencephalon-midbrain (D/MB) and cerebellum (CER). Each point represents observations made in 4 to 5 rats.
Nonischemic osmolalities for cerebral cortex, diencephalon-midbrain and cerebellum were 290 ± 3 mOsm/1, 303 ± 6 mOsm/1 and 297 ± 2 mOsm/1, respectively, (fig. 1B). Osmolality increased rapidly in all regions to about 325 mOsm/1 after 4 minutes of ischemia, plateaued for 16 minutes, then gradually increased between 16 and 90 minutes. Between 20 and 90 minutes of ischemia, cerebral cortex and cerebellar osmolalities were lower (P < .05) than diencephalon-midbrain osmolality. In all regions, the average maximal increase in osmolality was about 45 mOsm/1.

In pentobarbital anesthetized rat, normal whole brain osmolality was 319 ± 2 mOsm/1 (mean ± SEM) and higher (P < .02) than in unanesthetized rat (fig. 2A). Whole brain osmolality increased gradually during ischemia to about 340 mOsm/1 at 90 minutes or 25 mOsm/1 higher than control. The rate of increase in whole brain osmolality for the first 60 minutes of ischemia was about 0.3 mOsm/1 · min⁻¹ or 60% of the rate observed in unanesthetized rats. The increase (P < .05) in osmolality after 8 minutes of ischemia coincided with a higher (P < .01) rectal temperature (Tr) in the rats prior to the ischemic insult which may affect the rapidity of the increase in brain osmolality after decapitation although the brains were kept in a 37°C oven postdecapitation. At a Tr comparable to control, whole brain osmolality was significantly elevated only after ischemia of 20 minutes longer.

Osmolalities in normal, pentobarbital anesthetized rat cerebral cortex, diencephalon-midbrain and cerebellum were 320 ± 4 mOsm/1, 325 ± 2 mOsm/1, and 334 ± 3 mOsm/1, respectively. Cerebellar osmolality was significantly higher (P < .05) than cerebral cortex (fig. 2B). In the first 16 minutes of ischemia, osmolality changed little in all regions. Between 16 and 20 minutes, however, diencephalon-midbrain osmolality increased sharply to about 350 mOsm/1 while the increase in cerebellum and cerebral cortex was delayed until 30 to 60 minutes of ischemia. The change in osmolality was about 35, 32, and 29 mOsm/1 for cerebral cortex, diencephalon-midbrain and cerebellum, respectively, with a mean increase of 32 mOsm/1 compared to 45 mOsm/1 in unanesthetized rats. Cerebral cortex osmolality was consistently lower than diencephalon-midbrain and cerebellum after 30 minutes of ischemia.

The percent increase in ischemic whole and regional brain osmolality relative to control (nonischemic) osmolality for unanesthetized and pentobarbital anesthetized rats are shown in figures 3 and 4, respectively. In unanesthetized rats, there was an initial rapid 3% increase in whole brain osmolality after 8 minutes of ischemia followed by a plateau between 8 and 20 minutes and a second rapid increase between 20 and 60 minutes to 11% higher than control. No further change occurred between 60 and 120 minutes.

The percent increase in regional osmolality, which ranged from 6% greater than control for diencephalon-midbrain to 9% for cerebral cortex in the first 4 minutes of ischemia, was more rapid than for whole brain. There was a further linear increase in all regions between 4 and 90 minutes of ischemia to peak values of 15%, 14% and 13% above control for

![Figure 2](http://stroke.ahajournals.org/)

Figure 2. Brain tissue osmolality in pentobarbital anesthetized (60 mg/kg) rats during decapitation-induced global brain ischemia: (A) whole brain (WB); and (B) cerebral cortex (CC), diencephalon-midbrain (D/MB) and cerebellum (CER). Each point represents observations made in 4 to 5 rats.

![Figure 3](http://stroke.ahajournals.org/)

Figure 3. Percent increase in brain tissue osmolality compared to preischemic control values during decapitation-induced global brain ischemia in unanesthetized rats. WB = whole brain, CC = cerebral cortex, D/MB = diencephalon-midbrain, and CER = cerebellum.
cerebral cortex, diencephalon-midbrain and cerebellum, respectively. The magnitude of the increase observed in regional osmolality was greater than for whole brain throughout the ischemic period, but was especially marked in the first 20 minutes.

In pentobarbital anesthetized rats, the percent increase in whole brain osmolality for the first 30 minutes roughly paralleled the increase observed in unanesthetized rats with a 5% rise after 30 minutes of ischemia. Between 30 and 60 minutes, however, there was little, if any, further increase. A maximum increase of 7.7% above control was observed between 90 and 120 minutes of ischemia compared to the 11% increase observed in unanesthetized rats at 60 minutes.

Regional brain osmolality in pentobarbital anesthetized rat cerebral cortex and cerebellum followed the same time course with a linear increase to about 9% of control in the first 60 minutes of ischemia and remained at this level between 60 and 120 minutes. Diencephalon-midbrain osmolality was essentially unchanged in the first 16 minutes of ischemia, but increased to 7.5% of control between 16 and 30 minutes followed by a gradual linear rise to 12.5% of control between 30 and 120 minutes of ischemia. At 1 hour of ischemia, the percent increase in regional osmolality was about 9% in anesthetized rats compared to about 13% in unanesthetized rats.

Discussion

Tissue hyperosmolality relative to serum reported in earlier studies was largely due to autolytic changes post-mortem and during preparation for cryoscopic measurements. More recent studies using rapid sampling and quick freezing in liquid N₂ confirmed the generally accepted view that tissue is at least nearly iso-osmolar to serum (see table 1 below) except for secreting tissue such as the kidney. Our values of 307 and 319 mOsm/l in unanesthetized and anesthetized rat whole brain are in the range of values obtained by others in earlier studies. However, earlier data do not enable comparison of the effects of anesthesia on brain osmolality.

Approximately 30% of the increase in whole brain osmolality during anesthesia can be attributed to increased brain glucose and phosphocreatine. Barbiturate anesthesia also increases glucose-6-phosphate and fructose-6-phosphate, but these changes are probably offset by reductions in glycolytic and citric acid cycle intermediates beyond phosphofructokinase. Other factors which may contribute to the increase in brain osmolality during anesthesia are: (a) altered membrane permeability to water, Na⁺ and K⁺; (b) a shift in metabolic pattern from free water producing metabolism (carbohydrate and fat) to the production of free osmoles (protein metabolism). Until these factors can be quantitatively evaluated the increase in brain osmolality during anesthesia is unexplained.

Regional brain osmolality in unanesthetized rats was not significantly different (P < 0.05) between the regions analyzed but the ascending order of the mean values were cerebral cortex, diencephalon-midbrain and cerebellum which corresponds inversely to their water content and directly with their myelin content. During pentobarbital anesthesia cerebellar osmolality was significantly greater (P < 0.05) compared to cerebral cortex which may be attributable to the greater increase in cerebellar phosphocreatine, glucose and glycogen than in the cerebral cortex during anesthesia.

<table>
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<td>pentobarbital</td>
<td>35</td>
</tr>
<tr>
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<td>313</td>
<td>pentobarbital</td>
<td>24</td>
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<tr>
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<td>whole brain</td>
<td>319 ± 2</td>
<td>pentobarbital</td>
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</table>

TABLE 1 Comparison of Brain Osmolality Values
Unless the higher absolute values of whole and regional brain osmolality in pentobarbital anesthetized rats is partly the cause of decreased brain metabolism, the efficacy of barbiturates in ameliorating ischemic brain damage cannot be attributed to the suppression of brain tissue hyperosmolality during ischemia. Indeed, osmolality for whole and regional brain was higher in pentobarbital anesthetized rats before and after various duration of ischemia than in unanesthetized rats.

The major difference between osmolality changes in whole and regional brain with and without barbiturate anesthesia is the greater rate and magnitude of the change in osmolality with the onset of ischemia in unanesthetized rats. These results suggest that the osmoregulating reactions are greatly reduced during ischemia under pentobarbital anesthesia. The more rapid and greater increase in cerebral cortex, diencephalon-midbrain and cerebellar osmolality compared to whole brain (especially in unanesthetized rats) indicates that the osmoregulating reactions occur more rapidly in grey matter than in white matter. Gatfield, et al. showed reduced rates of glucose, glycogen and ATP breakdown and formation of lactate and inorganic phosphate during 30 second decapitation ischemia in phenobarbitone anesthetized (150 mg/kg) mice. However, Michenfelder, et al. reported no alteration in rates of ATP degradation and lactate production in dog brain with decapitation ischemia during thiopental anesthesia (46 mg/kg) plus 0.8% halothane. Using a technique of sequential sampling from different regions of the dog cerebral cortex, they obtained samples before decapitation and at 0.25, 1.25, 2.25, 4.25, 6.25 and 9.25 minutes postdecapitation. At subanesthetic concentrations of halothane (< 0.1%) the regression equations for ATP depletion and lactate production were; ATP (µmole/g) = 2.21 - 0.37X and lactate (µmole/g) = 3.08 + 1.76X, respectively, for the first 4 minutes postdecapitation. In dogs anesthetized with 46 mg/kg thiopental and 0.8% halothane, the equations were, ATP (µmole/g) = 2.19 - 0.40X and lactate (µmole/g) = 2.40 + 2.36X indicating no difference between the two anesthetic states. However, the technique of sequential brain sampling with "1 to 2 cm of nontraumatized cortex" between biopsy sites probably masked any effects of the anesthetics.

The greater rate and magnitude of increase in cerebral cortex, diencephalon-midbrain and cerebellar osmolality compared to whole brain also indicates that these regions should have a greater predilection for the development of brain edema after restoration of circulation. This supports the concept that focal brain edema may occur which is not necessarily reflected by a generalized increase in ICP, causing increased local tissue pressure and decreasing cerebral perfusion pressure and blood flow. The selective vulnerability of these brain regions have been clearly demonstrated.

The ratio of ischemic brain osmolality relative to control gives an indication of the percentage increase in brain tissue water required to restore brain osmolality to preischemic or normal levels. This estimation is based on the assumption that only water is taken up by the brain without solute during edema development and therefore gives a minimum estimate of the potential increase in brain volume. Lofgren, et al. studied the cerebrospinal fluid (CSF) volume-ICP relationship in the dog and showed that an increase in CSF volume up to 4% can be tolerated without causing a generalized increase in ICP. However, if CSF volume increases by 5% or greater, there is a marked rise in ICP. A 10% increase in CSF volume increases ICP to about 100 mm Hg. In unanesthetized whole rat brain, osmolality increased by 5% relative to control after 30 minutes of ischemia and by 11% after 1 hour of ischemia. During pentobarbital anesthesia whole brain osmolality was also elevated by 5% after 30 minutes of ischemia, but there was little further increase thereafter. These results suggest that the propensity for the development of increased ICP after 30 minutes of ischemia based on whole brain osmolality is similar with or without barbiturate anesthesia, but greater after ischemia of 30 minutes or longer. Based on regional brain osmolality changes, however, the results show that regional brain edema tended to occur earlier in unanesthetized brain beginning at 4 minutes of ischemia.

In conclusion, the effect of barbiturate anesthesia in reducing the osmole-producing reactions (i.e., catabolism) during ischemia probably plays a role in attenuating the development of brain edema after restoration of circulation. Also, due to local differences in osmolality during ischemia, focal brain edema may develop which could produce local brain tissue pressure gradients without being reflected by a generalized increase in ICP.

Acknowledgments

The authors gratefully acknowledge the assistance of Henry Alexander, Rod Hartman, Ellis Gross, and Ms Diane Samber.

References

CONCLUSIONS  The results of this study suggest that the two symptom groups could be differentiated clinically and arteriographically. Clinical data assessed the degree of stenosis, the diameter of the narrowest part of the lesions, and ulceration. In establishing the diagnosis of either amaurosis fugax or hemispheric transient ischemic attacks, consideration was given to the age, presence of carotid bruit, diabetes, claudication, hypertension, and duration of symptoms before the attack. Because the number of patients studied was small, we undertook a review of a larger series to determine if any important clinical or angiographic differences exist between these groups.

Materials and Methods

Between January 1, 1972 and December 31, 1976, 121 consecutive patients were evaluated at one of our institutions and given the clinical diagnosis of either amaurosis fugax or hemispheric transient ischemic attacks. Amaurosis fugax was defined as transient monocular blindness; transient ischemic attacks were defined as focal neurologic deficits that lasted less than 24 hours and were attributable to ischemia in the distribution of the carotid arterial system. Amaurosis fugax and transient ischemic attacks were studied to determine whether the two symptom groups could be differentiated clinically and arteriographically. Clinical data assessed the degree of stenosis, the diameter of the narrowest part of the lesion, and ulceration. In establishing the diagnosis of either amaurosis fugax or hemispheric transient ischemic attacks, consideration was given to the age, presence of carotid bruit, diabetes, claudication, hypertension, and duration of symptoms before the attack. Because the number of patients studied was small, we undertook a review of a larger series to determine if any important clinical or angiographic differences exist between these groups.

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Stroke. 1978;9:249-254
doi: 10.1161/01.STR.9.3.249

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