SUMMARY Twenty rhesus monkeys were used to evaluate the effects of dexamethasone and dimethyl sulfoxide (DMSO) following experimental occlusion of the middle cerebral artery (MCA) for 17 hours.

Results show that the gross, microscopic and angiographical picture of dexamethasone and no-treatment controls was practically identical. In contrast, DMSO-treated monkeys showed significant protection from the severe neurological deficits seen in the other groups. It is concluded that DMSO has a positive effect in reducing the neurological deficits seen in this model and may be potentially useful in clinical embolic stroke.

Methods

Twenty male and female rhesus monkeys (Macaca mulatta) weighing 5 to 7 kg were used. The animals were divided into four groups as follows: (1) sham (N = 2); surgery, exposure and freeing of the MCA from arachnoid but no clipping; (2) controls (N = 8): clipping, 5 ml per kilogram physiological saline; (3) dimethyl sulfoxide (N = 5): 2.5 gm per kilogram in a 50% solution with saline; (4) dexamethasone (N = 5): 3 mg per kilogram in saline, first two doses intravenously and intramuscularly thereafter.

Treatments were given intravenously (except supporting doses of dexamethasone) in adjusted final fluid volumes according to body weight. All animals unable to drink water were kept hydrated by daily intravenous saline fluid replacement.

Respiration and blood pressure were monitored by pressure transducers connected to a Beckman Dynograph.
type R. Local cerebral tissue flow was monitored by the hydrogen electrode technique previously described. Light microscopy, blood gases, blood chemistry, arteriograms, CBC and dry/wet brain weights were taken in all animals. The animals were anesthetized with Sernylan, 0.04 mg per kilogram, and anesthesia was maintained with sodium pentobarbital i.v. as required during surgery. All animals were intubated and allowed to breathe spontaneously. Drugs were given by fast intravenous drip, 3 drops per second, and sur- tobarbital i.v. as required during surgery. All animals were given postoperatively during the recovery period. A catheterization of the femoral artery and vein, preoperative arteriograms were taken at one-half hour and sixteen and one-half hours following application of the clip, and 30 minutes after removing the clip. Arterial blood samples were taken before, during and after surgery for blood gases. Complete blood counts, electrolytes, hemoglobin and hematocrits were monitored during the surgical procedure. The monkeys were placed in a headholder in the prone position and draped. The left eyelids were sutured together and a horseshoe incision was made from the lateral canthus of the eye above and below the eyelids with an electrosurgical bovie. The skin flap was retracted medially and any bleeding was controlled with bipolar coagulation using a Malis forceps. A curved hemostat was clamped around the optic nerve and bipolar coagulating current was passed through the hemostat. The eyeball was collapsed by removing the vitreous humor and exenterated with enucleation scissors and bipolar coagulation. After removing the eyeball, the muscle cone near the optic foramen was trimmed with the curved scissors and hemostasis was achieved using moist cotton balls and pressure. Following hemostasis, the optic foramen was located and, using a Zeiss surgical microscope (50X), a 5-mm area above, lateral and below the optic foramen was removed using a high-powered dental drill. The transorbital craniectomy was extended with a 5-mm Schlesinger rongeur for another 3 mm (fig. 1). Very little or no bone bleeding results if craniectomy is not extended laterally for more than 10 mm from the optic foramen. The trajectory or the MCA was identified (large blush pulsating vessel under the dura), and the dura was cut above and perpendicular to the MCA and retracted near its branch to the internal carotid artery. After freeing the MCA from its arachnoid membrane with a curved jaw microcup forceps, a 2-mm wide 40°-angle Mayfield clip was applied around the entire circumference of the MCA about 5 mm from its bifurcation with the internal carotid artery. Local cerebral blood flow was taken by inserting a small platinum electrode in the brain tissue just above the MCA prior to and following the application of the clip and again following removal of the clip 17 hours later. The clip was left in place for 17 hours but treatment was begun four hours following occlusion with the clip still in place. The animal was removed from the headholder and the second postclip arteriogram was taken. Several cotton balls containing a few drops of 1% Neosporin were placed in the eye socket and the skin flap was sutured. Five milliliters of Bacitracin (10,000 units) were infiltrated subcutaneously around the suture incision and the area was covered with Gentamycin sulfate ointment. About 200 ml of physiological saline were administered during the surgical procedure to all animals and an additional 50 ml of saline were given postoperatively during the recovery period. A rectal thermometer was used to monitor body temperature and all animals were maintained at 39°C using a heat pad during and following surgery. All animals received 200,000 units of Procaine Penicillin G and 500 mg of streptomycin i.m. for four days following surgery. The animals were neurologically evaluated twice daily for the seven-day observation period. At the end of seven days the animals were killed with an overdose of sodium pentobarbital and samples for light and electron microscopy were taken 3 mm caudal to the clip and from both sides of the motor cortex and the basal ganglia. The results of the electron microscope study will be reported elsewhere. Any animal not surviving the 17-hour postocclusion insult was excluded from the series. Three such deaths occurred.

Results

Gross and Histological Examination of the Brain

Seven days after occlusion of the middle cerebral artery, all animals were killed and the brains were removed for gross and histological examination. Coronal sections of the control brain often revealed hemorrhagic infarcts in the corticoparietal area extending into the suprasylvian region of the temporal lobe, and numerous minute infarcts in the caudate and lentiform nucleus. Ventricular compression and tissue softening extending from the prefrontal to the preocipital region in the occluded hemisphere was also noted. In the contralateral hemisphere, areas of punctate hemorrhage were often seen subcortically in the parietal area as well as small necrotic lesions in the white matter. There was sporadic leakage following injection of Evans-blue in both
the ipsilateral and contralateral hemisphere in these control animals.

Gross brain inspection of the dexamethasone-treated animals revealed few differences in the state of tissue pathology from the control animals.

Coronal sections of the DMSO-treated animals showed a small necrotic area posterior to the clipped MCA which extended in several animals several millimeters into the inferior thalamic peduncle and putamen. No large areas of hemorrhage were seen but petechial hemorrhages were seen in the caudate-putamen area. The traumatized hemisphere was found firm with no evidence of ventricular compression, and no leakage of Evans-blue tracer was observed in the tissue parenchyma. A summary of the histological examinations of brains from each group is shown in table 1. The caudate-putamen was chosen for detailed histological examination because its neuroanatomical and morphological identification in brain is easily recognized. Moreover, the caudate-putamen is fed by the lenticulostriate branches of the MCA and, because these arteries are not known to anastomose with one another, their occlusion almost always results in infarction of the tissue they supply, namely, the head of the caudate nucleus and the lentiform area (basal ganglia). Table 1 shows that, of the animals examined, a more severe histological picture is seen for the control and dexamethasone groups, particularly in the caudate lentiform area and the pre-occipital cortex. One sham animal was examined (surgery — no occlusion) and showed no significant pathological changes in the brain regions examined.

Angiography

Arteriograms were taken in all animals before occlusion, 30 minutes and sixteen and one-half hours after occlusion, and 30 minutes following unclipping of the MCA. Figure 2 shows the typical arteriograms seen for each group of animals in controls (a, b, c), DMSO (d, e, f), and dexamethasone (g, h, i). Figures 2a, d and g show the animals before occlusion of the MCA. Figures 2b, e and h show the animals sixteen and one-half hours following clip application. Figures 2c, f and i show the revascularization of the brain after removal of the clip at seventeen and one-half hours. All animals show the arrest of blood flow in the MCA and its branches (fig. 2b, e and h) following clipping. In addition, the control and dexamethasone groups show a narrowing (vasospasm?) of the internal carotid artery (ICA) just below the siphon.

This narrowing is seen in all animals 30 minutes after occlusion of the MCA. The narrowed ICA is still evident in the control and dexamethasone groups after sixteen and one-half hours following occlusion (fig. 2b and h) but not in the DMSO animals (fig. 2e, i).

Following removal of the clip, blood flow to the MCA returned in all animals (fig. 2c, f and i). The ICA remained stenosed in the control and dexamethasone animals (fig. 2c and i) but not in the DMSO animals (fig. 2f and h). Upon removal of the clip, it was noted that the control and dexamethasone groups showed lack of filling by the contrast media in the posterior parietal area (fig. 2c and i, \(\times 3\)). No lack of filling was observed in the DMSO group (fig. 2f, \(\times 2\)) and the filling of posterior parietal area resembled the preoperative arteriogram (compare fig. 2d and f).

### Table 1  Histopathological Changes in 17-Hour MCA Occlusion

<table>
<thead>
<tr>
<th>Brain region</th>
<th>Control (N = 2)</th>
<th>Dexamethasone (N = 2)</th>
<th>DMSO (N = 2)</th>
<th>Sham (N = 1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caudate-lentiform</td>
<td>+2;+2</td>
<td>+1;+2</td>
<td>0;+1</td>
<td>0</td>
</tr>
<tr>
<td>Area posterior to clip</td>
<td>+3;+3</td>
<td>+3;+3</td>
<td>+2;+2</td>
<td>0;+1</td>
</tr>
<tr>
<td>Pre-occipital cortex</td>
<td>+2;+3</td>
<td>+2;+2</td>
<td>0;+1</td>
<td>0</td>
</tr>
</tbody>
</table>

0: no significant changes; +1: some neuronal swelling, few hemorrhagic infarcts, some vacuolization; +2: astrocytic, neuronal swelling, moderate hemorrhagic infarcts, moderate necrosis; +3: astrocytic neuronal swelling, numerous chromatolytic neurons, perivascular spaces, extensive vacuolization, extensive necrosis, "cellular degeneration" appearance.

### Blood Gases

Arterial blood gases taken before, during and after the surgical procedure showed a slight rise in the P\(_{O_2}\) and an 8% to 12% drop in the P\(_{CO_2}\) following surgery in most animals. The animals remained essentially normocapnic during surgery and any acid/base imbalance seen was promptly corrected with appropriate fluid administration.

### Brain Dry/Wet Weights

At the end of the seven-day observation period, animals underwent total craniectomy of the skull while under anesthesia. A fatal bolus of sodium pentobarbital was delivered and the brain was quickly removed, cross-sectioned and divided at the midline. Portions of each hemisphere were weighed and the tissue was baked in an oven for 24 hours at 100°C. The dry weight was obtained by re-weighing the residual tissue, and the percentage of swelling or edema was calculated using the formula of Elliott and Jasper.

\[
\text{dry weight} \times \frac{100}{\text{wet weight}} = \% \text{ dry weight (dw)}
\]

\[
\frac{\text{dwC} - \text{dwE}}{\text{dwE}} \times 100 = \% \text{ edema (edema)}
\]

where \(\text{dwC} = \% \text{ dry weight of control (uninjured hemisphere, dwe} = \% \text{ dry weight of experimental (injured) hemisphere, edema} = \% \text{ edema present in experimental hemisphere. The mean brain edema} \% \text{ found for each group was as follows: (a) controls (N = 8): 17%, (b) DMSO (N = 5): 0%, (c) dexamethasone (N = 5): 1.8%, (d) shams (N = 2): 0%.

### Blood Tests

Total protein, blood urea nitrogen, glucose and serum electrolytes were not appreciably changed in any of the groups except for a transient mean 15% and 20% lowering of serum sodium and potassium respectively.

Both hemoglobin and hematocrit were reduced in all animals by 10% to 15% after MCA occlusion. Slight leukocytosis was seen postsurgically in some animals but this was corrected with iron supplements and intravenous fluids.

### Diuresis

A Foley catheter was used to collect urine excretion for three hours following initial treatment. The total mean diuretic output in controls was 40 ml ± 15 ml. Dexamethasone-treated monkeys similarly showed a mean volume of 43
ml ± 13 ml while DMSO-treated animals excreted 110 ml ± 20 ml.

Neurological Evaluation

Table 2 summarizes the neurological status of each monkey at 24 hours and seven days following MCA occlusion. The animals were rated daily using the following criteria: (A) normal, no neurological deficits; (B) hemiparesis, alert, aggressive; (C) hemiparesis, slow, lethargic; (D) stuporous, comatose, dead.

It will be seen that the eight untreated controls and the five dexamethasone-treated monkeys were either stuporous or comatose (status D) 24 hours following stroke. After seven days, they remained stuporous or in coma except for two control animals which died on Days 5 and 6. Only one animal in the dexamethasone group recovered from status D to status C; the other four animals in this group remained in status D.

Of the five DMSO-treated animals, four had status B and one status C at the end of 24 hours postsurgery. After seven days, two animals showed no neurological deficits (status A) and three had status B. The results are significant at the p < 0.001 using the t-test.

All animals which did not feed themselves (status D) received daily iron supplements and intravenous dextrose 5%.

Sham controls (surgery, no occlusion) all had status A 24 hours following surgery and remained free of neurological deficits until killed. In addition, the same iron supplements and intravenous fluids were used in these animals as a
counter-check for the control and dexamethasone groups which were similarly treated.

An interesting detail observed in several dexamethasone and control monkeys following occlusion was a pronounced and palpitating superficial temporal artery on the left (occluded hemisphere) side of the head. This phenomenon was not seen in DMSO or sham animals.

Local Cerebral Blood Flow

The mean average cerebral blood flow value taken before occlusion in the area of the MCA was 89 ml/100 gm per minute with a range of 76 to 115 ml/100 gm per minute for all animals. Immediately following clipping of the MCA, this mean average value dropped by 43% to 54% (of 100). Following unclipping of the MCA, there was little group difference in the local cerebral blood flow as flow returned to essentially pre-occlusion values regardless of treatment. Some individual variation of blood pressure and CO₂ was observed in a few monkeys, thus making cerebral blood flow correlation that much more difficult to interpret.

Discussion

Early in this study, it was found that occlusion of the MCA for four hours was not sufficient to produce the desired severe neurological deficits in control animals. For this reason, and also because survival with sensorimotor loss was preferred to death in evaluating the possible drug reversal of the deficits, 17-hour occlusion was found optimum for this study. Treatment after four hours with the MCA still occluded was chosen because we wanted to test the efficacy of drug therapy while the problem of embolic occlusion was not yet resolved. In addition, treatment after four hours of occlusion is not unreasonable in terms of therapeutic delay such as may be seen clinically.

The ability of steroids, particularly dexamethasone, to improve the status of subjects with cerebral infarction seen either clinically or experimentally induced, remains doubtful. Experimentally, it has been reported in a number of studies which included cats, 16, 11 squirrel monkeys, 12 rats 14 and gerbils 15, 18 that steroids are ineffective in induced cerebral infarction.

However, an experimental study using dexamethasone reported a reduction in the mortality and morbidity in gerbils subjected to carotid ligation, 16 but no difference was found when the steroid was given eight hours after the infarction. Another study, by Wexler, 17 has shown that carotid ligation in the gerbil is accompanied by a significant drop in the plasma cortisol levels at 24 and 48 hours following the infarct. It is therefore possible that dexamethasone may be acting initially to protect these gerbils from the ensuing adrenal insufficiency brought on by the stroke.

Clinically, three studies have reported a beneficial use for steroids following embolic stroke. 10-12 Two of these reports lacked statistical analysis of the data and scoring of patients’ disability throughout the period of treatment. 10, 11 In addition, one of the studies did not include control patients. 10 The third report, by Patten et al., 12 was carried out double-blind and contained statistical values and ratings for the initial and final neurological status for each patient. The conclusion in that study, that steroids are beneficial in embolic stroke, is not, in our opinion, supported by the data presented. For example, there was no statistical difference between the 17 placebo patients and the 14 dexamethasone-treated patients when the author’s own rank-order Wilcoxon test was applied.

When the 15 most severely affected patients were analyzed, a significant difference was found if the initial and final scores were compared for the placebo versus dexamethasone. However, when the total numbers of improved patients for the severely disabled placebo versus dexamethasone were compared, no statistically significant differences could be found (Fisher’s exact test). In addition, of the eight severely disabled placebo patients, four were in their 80s and one was diagnosed as having cerebral hemorrhage (none of these five patients improved). On the other hand, of the seven patients in the steroid group, none was an octogenarian or had cerebral hemorrhage.

Since another study showed that the risk of death from embolic stroke increases by 75% for patients in their 80s (regardless of treatment) compared to 45% for those under 80, 19 and since steroids have been shown in numerous studies to be ineffective in cerebral hemorrhage, 11, 12, 19, 21 and since the total series of patients comparing placebo versus steroids showed no statistical difference between the two groups, we believe that the conclusion made by Patten and his group 10 on the usefulness of dexamethasone in cerebral infarction is not supported by good evidence.

Negative results in the use of steroids for cerebral infarction were reported in four studies. 15, 19, 26-28 One of the above reports was double-blind and controlled but included only 12 patients. 28 The second report was not double-blind but comprised 36 patients. 27 The third report by Bauer and Tellez 25 was a double-blind, controlled study in 54 patients which appears to be the most conclusive in its evaluation of dexamethasone for cerebral infarction. These authors reported no significant difference in the outcome of 26 placebo patients and 28 patients treated with dexamethasone for ten days beginning with 24 mg on the first day and tapering gradually to 8 mg on the final day, with an average of 12 mg per day per patient. This finding is supported by Norris 26 who recently reported on the ineffectiveness of steroid therapy in patients with cerebral infarction.

Our results in monkeys using high doses of dexamethasone following experimental cerebral infarction similarly show no difference between this therapy and no-treatment controls. It is of interest to point out that in our study and that of Rubenstein 29 in patients with hemorrhagic stroke, the reduction of edema in brain tissue secondary to cerebral infarct (see table 1) was not associated with an

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### Table 2: Neurological Status of Each Monkey at One Day and Seven Days Following MCA Occlusion

<table>
<thead>
<tr>
<th></th>
<th>1 Day postocclusion A*</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>7 Days postocclusion A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>DMSO</td>
<td>—</td>
<td>4</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>2</td>
<td>3</td>
<td>—</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>—</td>
<td>—</td>
<td>5</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>4</td>
<td>—</td>
</tr>
<tr>
<td>Sham (no occlusion)</td>
<td>2</td>
<td>—</td>
<td>—</td>
<td>2</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

*Neurological status: A = no neurological deficit; B = contralateral paresis, alert, aggressive; C = contralateral paresis, slow, indifferent; and D = severe lethargy, coma or death.*

*Two deaths on fifth and sixth day.*

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1. Our results in monkeys using high doses of dexamethasone following experimental cerebral infarction similarly show no difference between this therapy and no-treatment controls. It is of interest to point out that in our study and that of Rubenstein in patients with hemorrhagic stroke, the reduction of edema in brain tissue secondary to cerebral infarct (see table 1) was not associated with an...
amelioration of the neurological status when dexamethasone was used. This implies that while cerebral edema may be a critical factor in the ensuing pathological sequelae following stroke, the reduction of such swelling does not necessarily guarantee significant recovery of symptoms. This might also explain why hyperosmotic agents may be useful in acute brain trauma with swelling but not in swelling secondary to cerebrovascular occlusion or hemorrhage.

In a previous study, we reported that DMSO given to squirrel monkeys following a four-hour occlusion of the MCA reduced the mortality rate from 60% in controls and hemodilution-treated animals to 20%. That study also indicated the possible usefulness of hyperbaric oxygenation in occlusive stroke since mortality in these animals was also reduced to 20%. The present study appears to support the findings seen earlier using DMSO in a more severe cerebral infarction model using squirrel monkeys.7 Our study reveals several consistent differences among no-treatment, dexamethasone-treated animals, and the DMSO series. These differences can be summarized as follows.

1. Internal carotid artery narrowing is seen below the siphon in all animals 30 minutes following MCA occlusion. After sixteen and one-half hours and with MCA still occluded, the narrowed carotid is still seen in the former two groups but not in the DMSO animals (fig. 2b, e and h, ^). Filling of the posterior parietal and temporal branches of the MCA is more pronounced 30 minutes following unclipping of the MCA in DMSO than in controls or dexamethasone-treated animals (fig. 2c, f and i, ^). Gross and histological examination reveals less damage in brain tissue of DMSO-treated animals than in controls or dexamethasone-treated animals.

2. Brain swelling of the traumatized hemisphere is moderately high in controls, greatly reduced in dexamethasone, and not present in DMSO.

3. Diuretic output is twice as high for DMSO animals following treatment than the two other groups.

4. Neurological status of DMSO-treated monkeys is significantly better after 24 hours or at the end of seven days than in controls or dexamethasone-treated monkeys.

Several possibilities may be advanced to explain these differences. If one accepts, in principle, the idea that narrowing of the internal carotid artery (fig. 2b and h, ^) results from the experimental occlusion of the MCA, and the arteriograms seem to support this, then it seems reasonable that the blood flow to the anterior cerebral artery (ACA) may be sufficiently compromised to affect the normal anastomosis between the ACA branches and those of the MCA even while the latter is occluded. If this is the case then the lack of revascularization to the posterior parietal area as seen in the control (fig. 2c, ^) and dexamethasone (fig. 2i, ^) animals can be explained by a reduction of collateral flow to this region. It would also follow that the reversal of the internal carotid artery's narrowing (fig. 2e, ^) would provide sufficiently adequate flow to the ACA and, subsequently, to the branches which anastomose with the MCA such as the pericallosal artery. Thus, adequate vascular anastomoses to the posterior parietal areas may prevent ischemia and necrosis of the cerebral tissue by maintaining these regions with a minimum supply of oxygen and cellular nutrients derived from the blood.

If collateral flow is a passive phenomenon determined by intravascular pressure differences in the circulation, then a reduction of such flow can be caused by platelet aggregation, vasospasm or compression of vessels by parenchymal edema. The action of DMSO might then be explained along these lines since the drug has been reported to disaggregate platelets in vessels16 and to reduce tissue edema,1, 16 often by protecting cellular disruption12 and preventing the release of lysosomal enzymes.16 Little et al.16 reported that there is extrusion of neuronal lysosome contents into the neuropil 12 hours after brain ischemia secondary to MCA occlusion.

It is generally accepted that a release of lysosomal enzymes following tissue injury can support or exacerbate the formation of edema. De Duve17, 18 has demonstrated that autolysis of cells results from the rupture of lysosomes and the release of acid hydrolases which further compound the existing tissue damage and cellular degradation. In fact, de Duve17 referred to the intracytoplasmic lysosomes as "suicide bags" in reference to their potential danger following their rupture.

The lack of hemispheric brain swelling, the diuretic output and the histological examinations suggest that DMSO is able to prevent edema formation in ischemic areas thus assuring minimal cell necrosis. Dexamethasone also substantially reduced swelling but the neurological recovery was not much different from that of the no-treatment controls. Gross examination of the brain following sacrifice showed extensive necrosis in the controls and dexamethasone series extending from the prefrontal to the preoccipital lobes. The DMSO series showed tissue necrosis just posterior to the clipped MCA compromising 3 to 5 mm of tissue.

The return of flow following unclipping of the MCA appears to be proportionately related to the amount of tissue oxygenation available to the ischemic tissue during embolism of a cerebral vessel. Two findings reinforce this suggestion. The first is that hyperbaric oxygenation in the MCA-occluded squirrel monkeys in another study increased survival and good recovery in these animals.7 Secondly, DMSO has been reported to increase tissue oxygenation in some organ tissues,19, 20 or to reduce oxygen demand or consumption by the tissue.20 The results of this study, then, seem to indicate that a reversal in the narrowed supraclinoid portion of the internal carotid artery by DMSO may be responsible for preventing the severe neurological deficits seen after the MCA is occluded for 17 hours in these models. It is possible that, by reversing the internal carotid artery stenosis, an adequate supply of collateral blood flow may be maintained in the affected hemisphere. This collateral flow increase could support adequate tissue oxygenation levels in the infarcted regions and thus result in a significant reduction of the neurological deficits.

Finally, it is of interest to note that an external carotid artery sign, characterized by a highly pronounced superficial temporal artery in the occluded hemisphere, was seen in some controls and dexamethasone animals the day following infarction but not in the DMSO series. This condition can arise as a result of internal carotid artery narrowing or occlusion, an event which can shuttle more blood to the external carotid artery thus causing the superficial carotid artery to become more visible in the scalp. This sign, according to Olivarius,20 is not necessarily accompanied by
neurological deficits in humans nor is it pathognomonic of internal carotid artery occlusion, but may be helpful in drawing attention to the possibility of occlusion or partial thrombosis of the internal carotid artery on the ipsilateral side.

In summary, our data show that in this injury model, DMSO is significantly effective in preventing the severe neurological deficits seen in these monkeys after occluding the MCA when compared to no-treatment animals. We further conclude that the administration of the corticosteroid dexamethasone does not provide any benefit to these animals following the vascular insult.

Acknowledgment

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