Ischemic Strokes

After I learned how unconscionable the FDA's prohibition against DMSO was, I made a point to begin telling people (e.g., friends, relatives, patients) I felt were at risk of a stroke to stock DMSO at home, and since then, I've had instances where someone (or their caretaker) called me up, described a stroke, I gave them instructions on what to do (since they already had DMSO at home), and by the time they got to the ER, the stroke was "resolved" and in some cases, the ER was confused by the CT scan because it both looked like a stroke had happened and simultaneously that one had not.

Note: in my opinion, IV DMSO would have been ideal (and more effective) in those situations, but in each case, it was not feasible to implement.

Likewise, many compelling cases have been recorded of individuals who treated their strokes with DMSO:

> A Los Angeles school teacher had a major stroke shortly after the start of the Christmas break. She was unconscious on her living room floor. DMSO treatment was started immediately after the stroke. The DMSO was first applied topically to her head within minutes of the stroke. Less than one hour after the stroke she was given DMSO by intramuscular injection. This patient was never taken to the hospital for this stroke. A prominent surgeon who was a family friend told the husband of this patient that it was important to keep her out of the hospital. The surgeon said that even though the treatment was completely legal, it would be difficult to get approval to give the DMSO especially by injection at his hospital.
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> This patient made a dramatic recovery. She regained consciousness later in the day in which she had her stroke. Treatment continued for the next week. Each day she

> received two topical applications of DMSO, one intramuscular injection of DMSO, and two doses of one teaspoonful of DMSO in juice. Her condition improved each day. When school resumed after the first of January, this teacher was back in the school teaching the students as if nothing had happened during the Christmas vacation. She never even mentioned it to the other people at the school. She continued teaching until she retired. She retired healthy with no disability.

Note: small strokes can still cause significant long-term issues (which DMSO often completely prevents), so as a general rule, I advise using DMSO anytime someone has a suspected stroke. Additionally, if you drive someone to the ER (and call in ahead to let the ER know you are coming), you have numerous opportunities to administer DMSO prior to placing the patient in the ER without delaying their care there (e.g., emergency brain surgery for a hemorrhagic stroke).

> A lady was in a coma in a convalescent hospital and had been in the coma since her stroke three months ago. She was given little chance of recovery and was expected to remain in a vegetative state until her death.
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> When I first observed this lady, there was no response to any type of stimulus. She was alive, but appeared lifeless. It was decided that her treatment should be topical DMSO applied to her head daily either by her husband or by one of the nurses at the facility.
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> One month after the start of treatment, there were positive signs in the lady. Her brain was starting to respond to the DMSO. The treatment continued, and four months after treatment started this lady was able to return to her home. After her return to her home, this patient started drinking one teaspoonful of DMSO in a small glass of water each day in addition to the daily topical treatment. This treatment continued for a period of years.
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> Three years after the start of DMSO treatment this writer returned to visit this patient. At this time the lady was living a normal life, not the life of a stroke victim. She was able to look after the house and walked normally.

> The only lingering effect of the stroke was a slight speech defect. At this time she said that her memory was better than that of her husband who had not had a stroke and who was considered to be completely normal.

Note: there are also many reported cases of individuals who took DMSO for musculoskeletal or pain disorders (by far the most common use of DMSO) who then experienced a permanent improvement of stroke symptoms.

As shown earlier in this article, DMSO has numerous properties that make it uniquely suited to protect from the damage of ischemic strokes. These benefits have in turn been shown to occur for brain tissue. For example:

•In anesthetized cats, DMSO significantly enhanced brain oxygenation (particularly in the caudate nucleus).

•DMSO was shown to preserve the neurological function of hippocampal brain tissue samples once their oxygen or glucose were withdrawn (with similar results seen in this study).

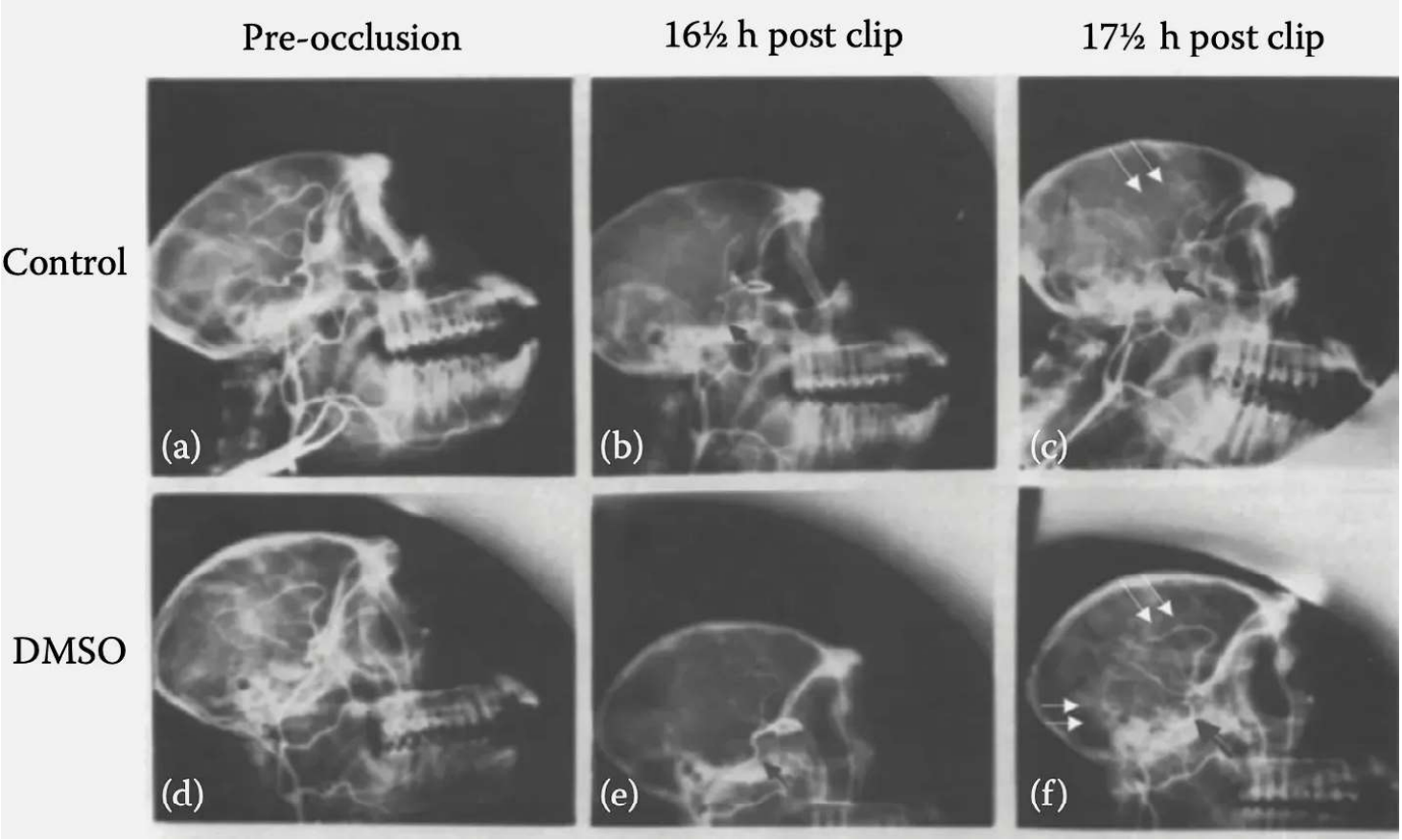
•Frequently in strokes, an area will form where blood has been impaired, but brain tissue has not yet died (known as the penumbra—and the key target of most stroke management). In a pivotal rat stroke study where DMSO was administered an hour after brain blood flow had been permanently cut off, MRI imaging showed that DMSO stopped the region of dying brain tissue from continuing to expand, hence allowing a penumbra (rather than additional dead tissue) to form around the stroke site (particularly within the cortex).
Note: beyond the classic penumbra, groups of cells can also enter a shocked state where their normal functions cease (and they eventually die). As discussed here, this "penumbra" also responds to DMSO (which is one reason tissue often comes back to life following DMSO treatment).

Many animal studies have found that if blood flow is cut off to the brain, typically by occluding (blocking off) either the MCA (a key artery in the brain frequently affected

in debilitating strokes) or the carotid artery, DMSO significantly reduced the resulting ischemic damage (along with the reperfusion damage resulting from the blood re-entering the ischemic brain tissue).

Note: these results argue that giving IV DMSO beforehand could reduce the complications of many challenging surgeries (e.g., a coronary bypass). Unfortunately, much in the same way ultraviolet blood irradiation dramatically reduces bad surgical outcomes, neither has been adopted for this purpose.

For example, a rhesus monkey study blocked the MCA for 4 hours, gave DMSO, dexamethasone, or nothing, and then opened the MCA after it had been blocked for 17 hours. DMSO gave significant protection from the severe neurological deficits and loss of arterial blood flow the other two groups developed.



A squirrel monkey study blocked the left MCA for 4 hours, and then given a variety of different treatments (e.g., saline, hemodilution, or hyperbaric oxygen at 2

atmospheres). Seven days after treatment, 8 of 10 DMSO treated monkeys were alive (with 2 having mild contralateral muscle weakness), while 75% of those receiving hyperbaric survived, and just 34% of those receiving hemodilution survived (with the last two groups **also having more significant neurological deficits**). Finally, combining either of these treatments with DMSO produced slightly worse results than just DMSO alone.

Similar results have also been seen in many other species. For example, in rats who experienced strokes:

•DMSO 30 minutes prior to MCA occlusion significantly reduced the amount of permanently damaged brain tissue.[1,2,3]

•DMSO 20 hours prior to MCA occlusion reduced infarct size by 65%, by 44% when given an hour after (or by 31% if a lower dose was given), and by 17% when given two hours afterwards. Additionally, all treated rats survived (whereas 50% of controls died), and when survivors were examined 3 days after the stroke, the infarct was significant reduced.

•DMSO immediately after occluded MCA blood flow was restored reduced infarct size (the region of lost brain tissue) and blood-brain barrier damage (as measured by MRI). When combined with DPI, this protection was enhanced and the activity of MMP-2 and MMP-9 (enzymes which break down brain tissue) was reduced. Similar results were found in this study.

•DMSO preserved neuronal loss and reduced astrocytic hyper-reactivity in the somatosensory cortex and hippocampal from MCA occlusion.[1,2]

•DMSO one hour before or after MCA and carotid occlusion significantly reduced brain edema and infarct volume.

•DMSO 30 minutes prior to 90 minutes of MCA occlusion significantly reduced cortical and striatal infarct volumes and significantly improved neurological motor

function (assessed 24 hours after occlusion). Additionally, no benefit was seen when a low dose of DMSO was used.

•Oral DMSO along with vitamins C and E, 12 hours after MCA occlusion, significantly reduced oxidative stress.

Note: in neonatal (7 day old) rats with hypoxia-ischemia (HI) brain damage, DMSO injected into the brain reduced infarct volume and brain injury (particularly within the cortex) along with inhibiting the breakdown of MAP2 and fodrin, suggesting neuroprotection via calpain inhibition.

Likewise in other animals:

•A gerbil study (this species is more susceptible to strokes) found blocking carotid blood flow to the brain and then restoring blood flow to the brain caused significantly less neuronal loss if DMSO was given 30 minutes before the carotid blood supply was cut off. Another gerbil study had similar results, another did as well, another did as well (which found the best results, such as reduced death, neuron damage, and retained motor function) were obtained with lower DMSO doses), as did a fourth (which specifically found DMSO protected against hippocampal pyramidal cell loss). *Note: DMSO was less protective in Gerbils than other species.*

•Another gerbil study found DMSO given 30 minutes prior to permanent occlusion of a carotid greatly reduced seven day mortality (60% in untreated animals vs. 33.4% with low dose DMSO and 14.3% with high dose DMSO), greatly reduced neurological symptoms (e.g., drooping eyelids, hemiparesis, walking circularly only in direction) and reducing damaged brain cells by 15.6 to 35%.

•A dog study cut off cerebral blood flow, then restored it and used a variety of biochemical measurements to monitor cellular metabolism (along with EEGs). Dogs who received DMSO (and an anti-platelet agent) had significantly higher mitochondrial function (which was almost identical to controls who had not suffered the occlusion).

Another dog study induced a stroke by introducing an embolus (clot) into the MCA and then giving DMSO. Compared to controls, those given DMSO were observed to have normal behavior and no neurological deficits afterward, whereas 3 of the 9 controls died (with significant tissue death in the brain), while the survivors had contralateral paralysis (a typical stroke consequence) and impaired consciousness.

Note: another dog study found IV DMSO shunted blood to the brain, increasing total cerebral blood flow by over 20%, with increased flow to the caudate nuclei and cerebral hemispheres along with increasing intravascular volume, lowering hematocrit, increasing cardiac index with enhanced ventricular blood flow and having the cerebral metabolic rate of oxygen remain stable.

A cat study found DMSO protected brain tissue from MCA occlusion and increased cerebral blood flow (CBF) by 27%. When DMSO was given in conjunction with PGI2, a greater improvement was seen (e.g., a 68% increase in CBF).

In another cat MCA occlusion study, IV DMSO reduced mortality by 75% (almost all cats survived), likely due to DMSO greatly reducing the severe postischemic vasogenic brain edema (which created life-threatening midline shifts in the brain). This study built upon a similar dissertation by one of the investigators.

In rabbits where the MCA brain blood flow was cut off for 4 hours, administering appropriate doses of 20% IV DMSO when blood flow was restored greatly reduced the resulting neuronal damage, reactive gliosis and multifocal spongiosis throughout the areas supplied by the MCA, along with reducing meningeal edema, erythrocyte extravasation, neurophil hemorrhage.

Additionally, a rat study found that when hemorrhagic shock was induced (causing a loss of blood flow to the brain), DMSO downregulated the inflammatory response (NF-kappaB) and upregulated a key protein cells use for survival (HSP70).

Note: in the studies, I reviewed, I came across two (a cat study and a gerbil study(where blood flow was cut off to the brain did not observe a benefit from DMSO.[1,2]