Current Stroke Management

Roughly 3.1% of adult Americans have experienced a stroke (a figure I expect to rise from the COVID-19 vaccines). Each year, this translates to about 800,000 people in the United States having a stroke, and in 2022, 165,393 died (making it the fifth most frequent cause of death in the United States), with between 20-40% of survivors experiencing long term disability from the stroke.

Because of the harm strokes pose to society, and the rate at which brain tissue deteriorates once its blood supply is lost, the medical system emphasizes doing everything that can be done to identify and treat strokes as soon as possible.

Unfortunately, different types of strokes exist. In most cases, the blood supply is cut off due to something (e.g., a clot) blocking the artery (an ischemic stroke). However in 13% of cases it's instead due to a blood vessel rupturing and leaking out. This is problematic because the primary treatment for strokes is to inject a powerful clot busting medication (tPA) but in cases where the stroke is coming from a bleed, this can be disastrous. As a result, nothing can be done until the patient is accurately diagnosed (which requires a brain CT scan at the hospital), which in turn results in an even longer delay before tPA can be used to save a patient's brain tissue.

Note: there are a few diagnostic signs that are more suggestive of a hemorrhagic stroke (e.g., a severe headache or unusual neurologic symptoms), but to our knowledge, no reliable method besides a CT scan exists to differentiate the two.

Worse still, the statistics on tPA (approved in 1996 and still the only FDA approved treatment for ischemic strokes) aren't actually that good. Presently, tPA is only approved to be given within 3 hours of a stroke starting (as its likelihood of benefitting

a patient decreases with time)and in practice, it is often given up to 4.5 hours after symptoms start (since some degree of benefit still exists).

When that window is met (which only happens about 25% of the time and ultimately results in roughly 1.8%-8.5% of ischemic stroke patients receiving tPA), the existing data shows that only 13% percent of patients who receive tPA significantly benefit from it (39% return to normal, compared to 26% who would return to normal without treatment), with an additional 19% of tPA users experiencing some degree of improvement (but not a full recovery) from it.

Worse still, tPA can cause significant bleeding, which is sometimes minor (e.g., gum bleeding), but also carries a 6.4% risk of a symptomatic brain bleed, and a 1.6% risk of a serious systemic hemorrhage (along with other issues such as a 1.3% to 5.1% risk of angioedema and tPA frequently causing reperfusion injuries). In turn, many risk factors exist for the increased bleeding (e.g., a few common risk factors can lead to a 33% chance of tPA causing a fatal bleed), and there have been many lawsuits for either giving or not giving tPA to a stroke patient. Additionally, tPA is a poor choice for larger obstructions (e.g., one within the internal carotid artery), which instead must be physically removed. In short—many ICU doctors I know are quite hesitant to use tPA as they have seen cases where it dramatically improved patients, many where it did not do anything, and quite a few disasters (especially in the early days of the therapy where it was used for heart attacks and then often caused the patient to have a fatal or debilitating brain bleed).

Note: the best data exists for tPa being injected directly into the obstructed artery with interventional radiology. Unfortunately, while many premier institutions offer this, it is a specialized procedure that is not available at most hospitals.

Finally, there is essentially no therapy for recovery from stroke—which in short explains why stroke is the second leading cause of death and the third leading cause of disability worldwide.

In turn, it would be paradigm shifting if an effective stroke therapy existed which:

- Effectively treated ischemic strokes.
- Had no risk of worsening a hemorrhagic stroke.
- Could easily be taken at home, and more importantly, be quickly given on ambulances.
- Protected brain tissue from dying.
- Prevented reperfusion injuries.
- Healed damaged brain tissue after a stroke.

> I have been in health chats where twice now, folks were in the chat and were having a stroke, they both had DMSO on hand & took it, both strokes were stopped within 10-15 min and any damage was.

The fact that it's been known DMSO does all of that for over 50 years (it's even therapeutic for hemorrhagic strokes and can cross the blood-brain barrier to heal damaged neurons), in a nutshell, summarized why quite a few people I know harbor great animosity towards the FDA.

For example, a 2002 clinical trial (which can be viewed here) was conducted where DMSO and FDP (fructose diphosphate, a metabolite which cells turn into energy through glycolysis) mixed in 5% dextrose was administered intravenously twice a day (averaging 12 days) to 11 patients (average age 65) who presented with an acute or subacute ischemic stroke. After being subject to an extensive series of tests, it was concluded that DMSO was well-tolerated, that it benefited patients if given with 12 hours of symptom onset, and that 63% of the patients achieved 'improved' or 'markedly improved' neurological status (whereas for the patients receiving standard treatment, only 20% achieved an "improved" status three months later.

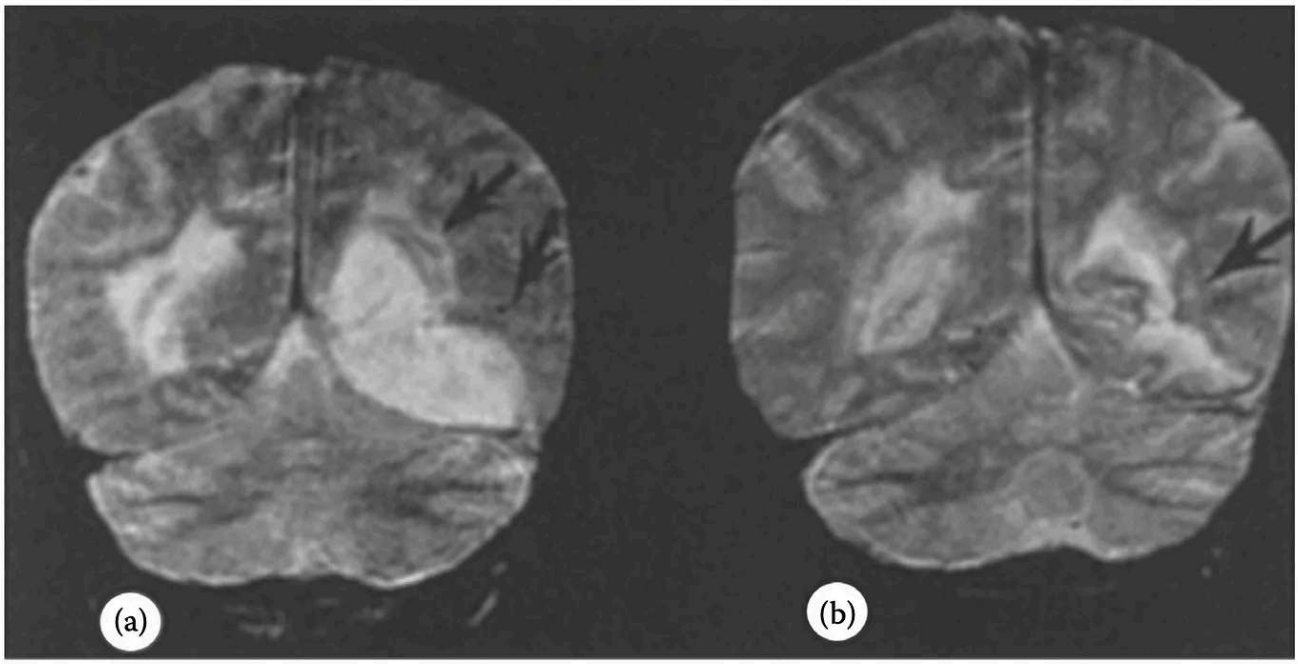
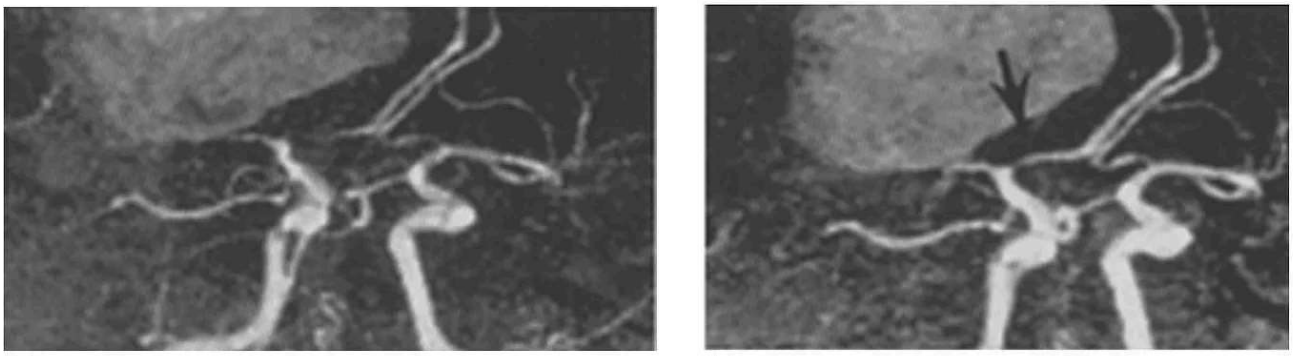


FIGURE 9.6 (a) T2-weighted coronal view MRI of posterior cerebral artery (PCA) territory infarct before treatment. White matter changes are seen with lateral mass effect on lateral ventricle, left thalamus involvement, and widespread edema involving the brainstem (arrows). (b) After 11 days of daily DMSO–FDP IV administration, dramatic reduction of edema and lower signal intensity are seen with an apparent improvement of gray matter and thalamic involvement (arrow). (From Karaça, M. et al., *Neurol. Res.*, 24(1), 73, 2002.)

Note: since older patients are the most vulnerable to strokes and have had such a significant recovery (without adverse reactions), this indicates DMSO is an even more promising therapy for younger patients with strokes.

A magnetic resonance angiogram is shown in Figure 9.7 of an 80-year-old patient who was diagnosed with a right MCA infarct that resulted in a mild mass effect and large hematoma affecting the basal ganglia territory. This patient was treated with DMSO–FDP twice daily after 48 h poststroke and showed improved perfusion in the MCA territory and reduced basal ganglia involvement.



(a) (b)

FIGURE 9.7 Sagittal view of magnetic resonance angiograph of internal carotid artery territory. A right MCA infarction with mild mass effect, large hematoma, and midline shift involving basal ganglia before treatment is seen (a) in an 80-year-old patient 48 h before DMSO–FDP treatment. (b) Eight days of twice daily DMSO–FDP infusions revealed no change in hematoma size, but there appeared an increased perfusion of the MCA ischemic territory, increased blood flow of MCA (arrow), and lessened basal ganglia involvement. (From Karaça, M. et al., *Neurol. Res.*, 24(1), 73, 2002.)

One of the most important aspects of this trial was that while DMSO is the most helpful when given immediately after a stroke, the trial showed DMSO could save the neurons long after the stroke had happened.

Patient		Treatment	After 1 month	After 3 months	Tx time (h)
ES	(90/♀)	PHA-56	Markedly improved	Markedly improved	6–12
FG	(62/♀)	PHA-56	Slightly improved	Slightly improved	>48
IU	(80/♂)	PHA-56	Unchanged	Unchanged	>48
NT	(41/♂)	PHA-56 DMSO+FDP	Improved	Markedly improved	>48
NC	(61/♀)	PHA-56	Slightly improved	Improved	>48
HD	(59/♂)	PHA-56	Unchanged	Unchanged	>48
GK	(80/♀)	PHA-56	Markedly improved	Markedly improved	>48
FO	(75/♀)	PHA-56	Markedly improved	Markedly improved	13–48
EF	(60/♀)	PHA-56	Unchanged	Unchanged	>48
AE	(62/♂)	PHA-56	Improved	Markedly improved	6–12
IC	(63/♂)	PHA-56	Improved	Markedly improved	6–12
HH	(74/♂)	Standard tx	Unchanged	Slightly improved	6–12
RK	(59/♂)	Standard tx	Unchanged	Slightly improved	6–12
NA	(64/♀)	Standard tx	Slightly improved	Improved	6–12
MA	(61/♀)	Standard tx	Unchanged	Slightly improved	13–48
HK	(48/♂)	Standard tx	Unchanged	Unchanged	6–12

"Tx time" designates how long after the stroke symptoms treatment was initiated.

Given the existing options for strokes, a trial like this should have been immediately replicated by premier institutions around the world—but instead almost no one even knows it happened.

Additionally, there are also animal studies on the DMSO-FDP mixture:

•In a rabbit study, blood flow to their brains was cut off (via hypoxemia, hypotension, and a bilateral common carotid artery occlusion), which eventually caused them to develop isoelectric (flatlined) brainwaves. After 5 minutes of no brain activity, they received either DMSO and FDP or saline, and then after roughly 2 minutes had their blood supply restored (with the DMSO group having an extra 1.4 minutes of no blood flow). The DMSO group regained brain activity much faster (a result frequently seen in animal experiments), all survived and all had minimal brain tissue damage, whereas only 22% of the saline group survived (and were severely disabled with significant brain tissue damage).

•In a mouse study (which can be read here), mice were subjected to moderate or severe head impacts and then treated 5 minutes later with various compounds, then evaluated

for motor function (via a grip test), brain tissue damage, and survival. DMSO-FDP was the most protective, DMSO the second best, while the rest (e.g., FDP alone) did not provide a benefit.