Stereotactic laser ablation of symptomatic cavernous malformations: imaging and clinical outcomes at one year

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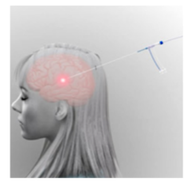
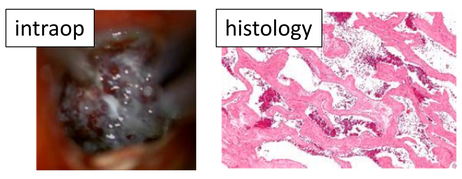
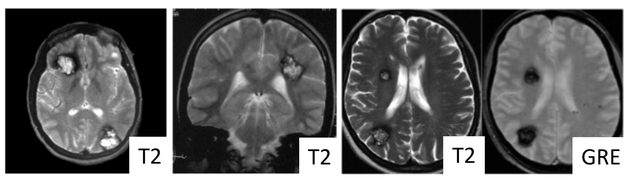


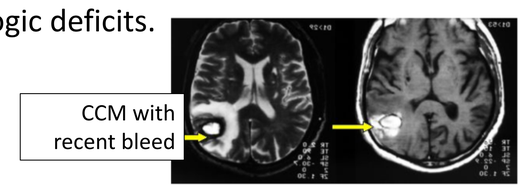
BACKGROUND: Cerebral cavernous malformations (CCMs) are abnormal vascular lesions, lobular in shape. Histologically they look like thin-walled vascular sinusoids, lined with endothelial cells, with no intervening brain parenchyma.



Because there is minimal blood flow through the sinusoids, they do not show up on catheter angiography. MRI is gold standard for imaging.



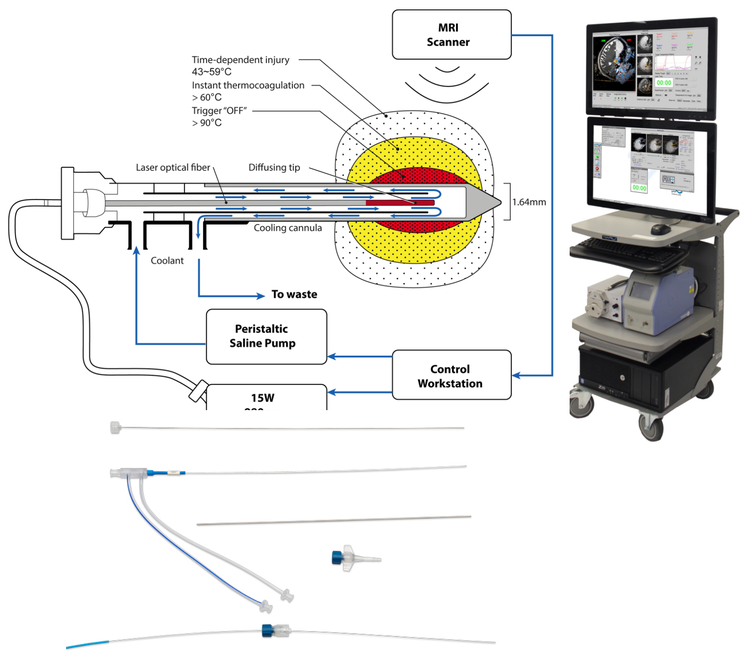
The flimsy sinusoids tend to hemorrhage at a rate of 2.4% over 5 years.[1] This presents as headaches, seizures, or other neurologic deficits.



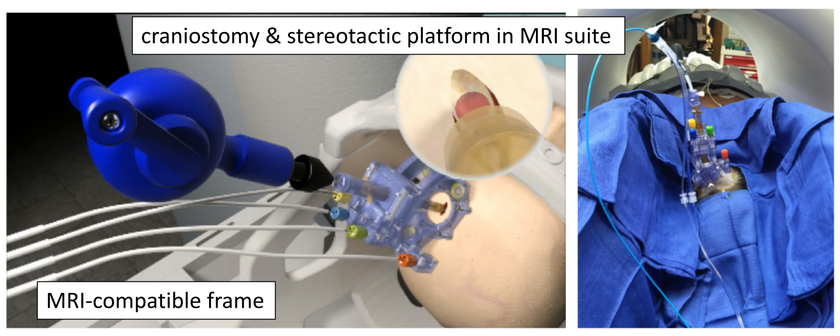
Surgical resection is the gold standard treatment; however, it carries the risks of rupture, stroke, or damaging surrounding healthy tissue. The risk of death of non-fatal stroke after surgery is 6% over 2-3 years.[1]

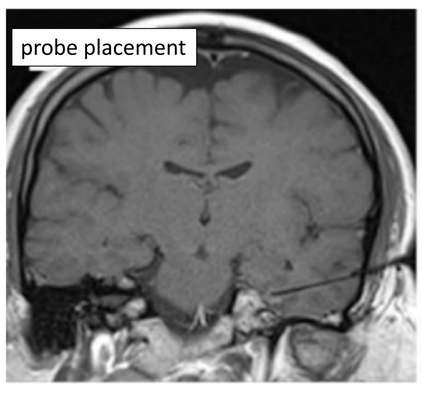
STEREOTACTIC LASER ABLATION (SLA)
SLA is an emerging technique to create lesions via magnetic resonance thermography-guided laser interstitial thermal therapy.

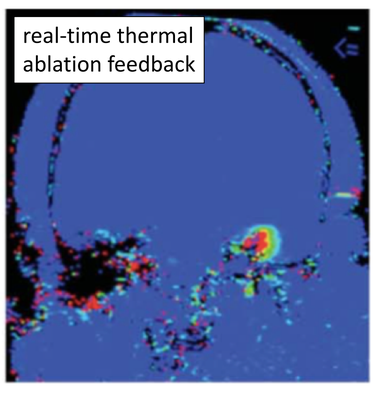
Via twist-drill craniostomy, we pass a flexible saline-cooled outer catheter (1.65mm diameter) with an internal fiber optic probe to deliver light energy (up to 15W using 980nm wavelength). This heats tissue from 50-90C causing irreversible parenchymal coagulation and microvascular thrombosis in 1-3 minutes.

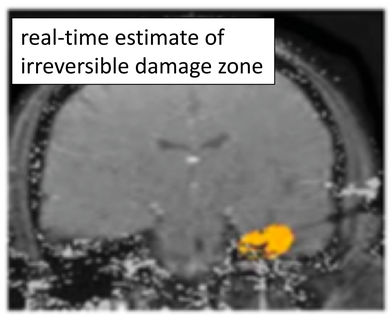


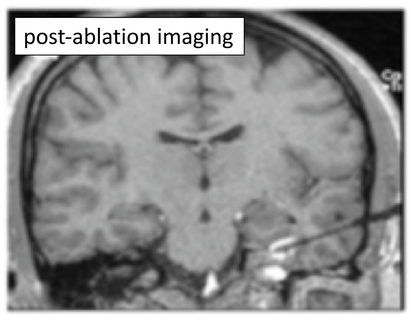
The procedure takes place within the MRI providing accurate probe placement and near real-time thermal feedback back to the computer console.











OBJECTIVE: To describe the use of SLA for treating symptomatic cavernous malformations with respect to feasibility, safety, imaging, and clinical outcomes in a series of patients.

Why use a laser?
- CCM has low flow so unlikely to hemorrhage with probe insertion
- Minimally invasive
- Larger ablation volumes than radiofrequency lesions
- MRI compatibility allows real-time verification of therapy goals
- Target CCM and surrounding gliotic/epileptic tissue
- Our initial experience treating five CCMs and over 50 epileptic lesions combined with other studies have shown its effectiveness and safety.[2-5]

METHODS: We retrospectively analyzed 20 consecutive patients who had CCMs associated with medically-refractory epilepsy (16), intractable headaches (3), or aggressive natural history (1). To confirm concordance between symptoms and location of pathology, each patient underwent a combination of anatomic and functional MRI, EEG, PET, and neuropsychometric testing. Intraop MRI provided confirmation of cannula placement and near-real-time feedback on extent of thermocoagulation. Patients were followed for symptom recurrence and imaging to estimate CCM involution.

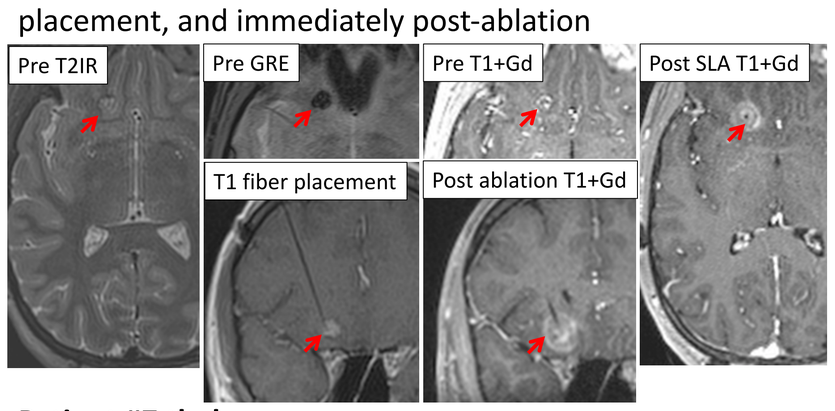
PATIENTS: 20 patients were included (17 seizures, 3 headache/deficit). Male/female 9/11. Age 37±7 y. Locations: temporal lobe (11), followed by frontal (4) and parietal (2), thalamus (2), pallidum (1). CCM volume averaged 0.9 cm^3 (±0.9, range 0.1-8.0).

COMPLICATIONS: No incidence of hemorrhage upon cannula insertion into CCM. Complications only occurred in subcortical cases, and included transient scalp numbness (thalamus, n=1), transient hemiparesis associated with hemorrhage during ablation (pallidum, n=1, see below), and worsening hemiparesis persistent at early follow-up (thalamus, n=1).

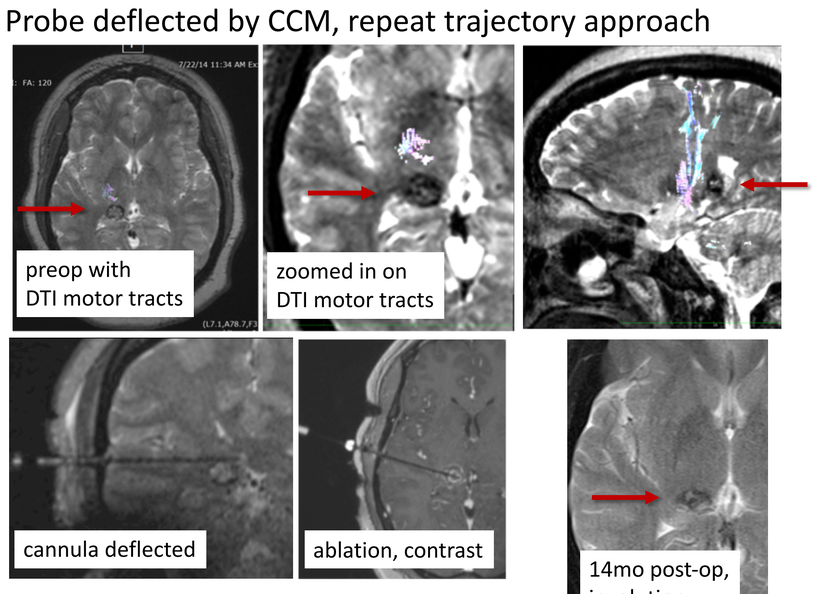
OUTCOMES: Eleven of 12 epileptic patients with >1-year follow-up were seizure-free (92% Engel class 1 outcome) from ablation alone. 5 of 6 remaining epilepsy patients with <1-year follow-up were seizure-free at last follow-up. Both headache patients with >1-year follow-up were improved.

CONCLUSION: Minimally invasive MR-guided ablation of symptomatic CCMs is a potentially safe and effective alternative to open resection. Additional experience and longer follow-up are needed.

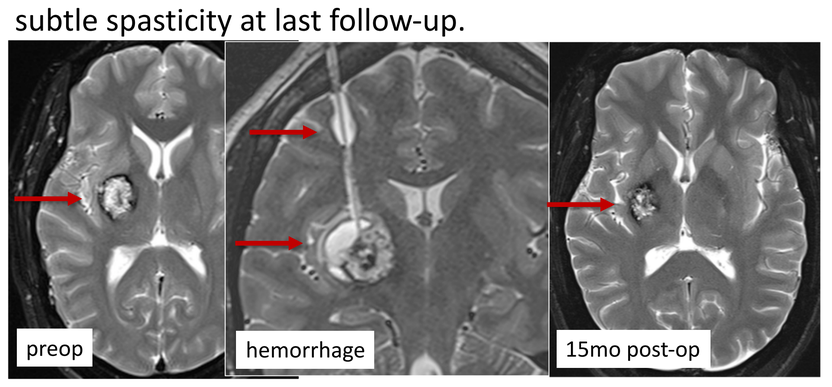
Patient #6
An example CCM with preop images, intraop cannula placement, and immediately post-ablation



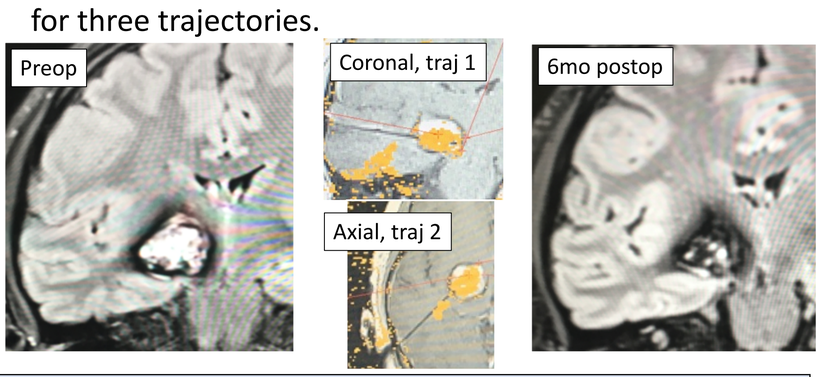
Patient #7 thalamus
Probe deflected by CCM, repeat trajectory approach



Patient #11 pallidum of basal ganglia, intra-op hemorrhage
CCM volume: 4.12 cm^3, second largest in series
Began bleeding during procedure, aborted. Moderate post-op hemiparesis, recovered most within 2 hours, subtle spasticity at last follow-up.



Patient #19
CCM volume 8.0 cm^3, largest in series. Used ROSA robot for three trajectories.



Case	Age Sex	Indication	Lesion Location	Frame	Preop (cm^3)	Ablation (cm^3)	Follow up* (cm^3)	Adverse Events	Symptoms at 12mo	Symptoms at last followup	Follow up (mo)
1	37M	Epilepsy	L fusiform gyrus	CRW	0.3	1.0	0.03	None	Engel 1A	Engel 1A	40
2	29M	Epilepsy	R Hippocampus	MRGF	0.4	4.8	0.08	None	1B	1B	17
3	67F	Epilepsy	R middle frontal gyrus	MRGF	0.2	1.9	n/a	None	4B	4B	9
4	66M	Epilepsy	L fusiform and inferior temporal gyrus	MRGF	2.2	4.0	0.2	None	1D	1D	25
5	76F	Epilepsy	L inferior temporal gyrus	CRW	0.6	4.6	0.3	None	1D	1A	25
6	34F	Epilepsy	R orbitofrontal	MRGF	0.6	2.5	0.2	None	1A	1A	13
7	27F	HA + deficit	R posterior thalamus	MRGF	1.0	3.8	0.4	C1-C2 numbness (resolved)	n/a	mild-moderate improvement in HA	14
8	40F	Epilepsy	R uncal apex (head)	MRGF	0.5	4.8	n/a	None	n/a	n/a	n/a
9	37F	Epilepsy	R anterior temporal pole	MRGF	1.0	2.6	n/a	None	1B	1B	16
10	21M	Epilepsy	R anterior temporal pole & stem	MRGF	0.8	9.3	0.4	None	1A	1A	13
11	41F	HA + deficit	R posterior putamen & pallidum	MRGF	4.1	1.1	1.5	Hemorrhage during procedure, aborted incomplete. Initial hemiparesis (resolved)	decreased frequency & severity	decreased frequency & severity, trace spasticity	15
12	29M	HA + deficit	R medial occipital (cuneus)	MRGF	0.1	3.6	n/a	None	n/a	n/a	n/a
13	21M	Epilepsy	L fusiform gyrus & perirhinal cortex	MRGF	0.1	4.4	0.0	None	1A (1 aura)	1A	14
14	16M	Epilepsy	L posterior hippocampus	MRGF	1.2	7.0	n/a	Minor superior quadrant anopsia that resolved	1A	1A	14
15	31F	Epilepsy	R inferior temporal gyrus (anterior)	MRGF	1.2	10.0	0.1	None	1A	1A	14
16	19M	Epilepsy	L frontal precentral gyrus	MRGF	0.7	2.0	0.1	None	1A	1A	13
17	56F	Epilepsy	L inferior temporal gyrus (posterior)	CRW	0.9	2.1	n/a	None	-	1A	1
18	33M	Epilepsy	R frontal white matter	MRGF	0.1	1.1	n/a	None	-	n/a	0
19	14F	HA + deficit	R midbrain/thalamus	Robot (ROSA)	8.0	8.2	4.0	Temporary worsening field cut	-	subjective improvement	6
20	50F	Epilepsy	L parietal perisylvian	MRGF	0.1	1.1	n/a	None	-	2	6

HA=headache, R=Right, L=Left, CRW=Cosman-Roberts-Wells, MRGF=MRI-Guided Mini-frame (ClearPoint SmartFrame, MRI Interventions Inc, USA); *Follow-up MRI between 6-12mo

[1]Poorthuis, *J Neurol Neurosurg Psychiatry* 2014 doi:10.1136/jnnp-2013-307349; [2]Willie, *Neurosurg* 2014 doi:10.1227/NEU.0000000000000343; [3]McCracken DJ, *Oper Neurosurg* 2016 doi: 10.1227/NEU.0000000000001033; [4]Curry, *Epilepsy Behav* 2012 doi:10.1016/j.yebeh.2012.04.135; [5]Carpentier, *Lasers Surg Med* 2011 doi:10.1002/lsm.21138