Stereotactic laser ablation as treatment for brain metastases that recur after stereotactic radiosurgery: a multiinstitutional experience

*Mir Amaan Ali, BS,¹ Kate T. Carroll, BA,¹ Robert C. Rennert, MD,¹ Thomas Hamelin, DNP,² Leon Chang, MD,³ Brian P. Lemkuil, MD,³ Mayur Sharma, MD,⁴ Jill S. Barnholtz-Sloan, PhD,⁵ Charlotte Myers, CNRN, MS, ANP-C,⁶ Gene H. Barnett, MD,⁴ Kris Smith, MD,⁶ Alireza M. Mohammadi, MD,⁴ Andrew E. Sloan, MD,⁵ and Clark C. Chen, MD, PhD¹

Departments of ¹Neurosurgery, ²Radiology, and ³Anesthesia, University of California, San Diego, California; ⁴Department of Neurosurgery, The Rose Ella Burkhardt Brain Tumor and Neuro-Oncology Center, and ⁵Department of Neurosurgery, University Hospitals, Cleveland, Ohio; and ⁶Department of Neurosurgery, Barrow Neurological Institute, Phoenix, Arizona

OBJECTIVE Therapeutic options for brain metastases (BMs) that recur after stereotactic radiosurgery (SRS) remain limited.

METHODS The authors provide the collective experience of 4 institutions where treatment of BMs that recurred after SRS was performed with stereotactic laser ablation (SLA).

RESULTS Twenty-six BMs (in 23 patients) that recurred after SRS were treated with SLA (2 patients each underwent 2 SLAs for separate lesions, and a third underwent 2 serial SLAs for discrete BMs). Histological findings in the BMs treated included the following: breast (n = 6); lung (n = 6); melanoma (n = 5); colon (n = 2); ovarian (n = 1); bladder (n = 1); esophageal (n = 1); and sarcoma (n = 1). With a median follow-up duration of 141 days (range 64–794 days), 9 of the SLA-treated BMs progressed despite treatment (35%). All cases of progression occurred in BMs in which < 80% ablation was achieved, whereas no disease progression was observed in BMs in which ≥ 80% ablation was achieved. Five BMs were treated with SLA, followed 1 month later by adjuvant SRS (5 Gy daily × 5 days). No disease progression was observed in these patients despite ablation efficiency of < 80%, suggesting that adjuvant hypofractionated SRS enhances the efficacy of SLA. Of the 23 SLA-treated patients, 3 suffered transient hemiparesis (13%), 1 developed hydrocephalus requiring temporary ventricular drainage (4%), and 1 patient who underwent SLA of a 28.9-cm³ lesion suffered a neurological deficit requiring an emergency hemicraniectomy (4%). Although there is significant heterogeneity in corticosteroid treatment post-SLA, most patients underwent a 2-week taper.

CONCLUSIONS Stereotactic laser ablation is an effective treatment option for BMs in which SRS fails. Ablation of ≥ 80% of BMs is associated with decreased risk of disease progression. The efficacy of SLA in this setting may be augmented by adjuvant hypofractionated SRS.

<http://thejns.org/doi/abs/10.3171/2016.7.FOCUS16227>

KEY WORDS brain metastases; stereotactic laser ablation; neurooncology; real-time MRI

ALTHOUGH the development of stereotactic radiosurgery (SRS) has fundamentally reshaped the treatment paradigm for brain metastases (BMs),^{1,16} there is an evolving recognition of its limitations. For tumor histological types that possess increased intrinsic

resistance to radiation, such as melanoma, treatment failure can be observed in > 25% of patients with BMs.⁶ For larger BMs or those adjacent to critical anatomical structures, delivery of radiation doses required for tumor control may incur neurological injury.⁸ Finally, as overall

ABBREVIATIONS BM = brain metastasis; BNI = Barrow Neurological Institute; CC = Cleveland Clinic; CW = Case Western; QID = 4 times daily; SLA = stereotactic laser ablation; SRS = stereotactic radiosurgery; TID = 3 times daily; UCSD = University of California San Diego.

SUBMITTED May 31, 2016. **ACCEPTED** July 27, 2016.

INCLUDE WHEN CITING DOI: 10.3171/2016.7.FOCUS16227.

* Mr. Ali and Ms. Carroll contributed equally to this work. Drs. Smith, Mohammadi, Sloan, and Chen share senior authorship of this work.

survival improves for select patient populations, reports of BM progression after SRS-induced growth arrest are emerging.^{11,13}

Therapeutic options for BMs that recur after SRS remain limited. Repeat SRS is an option;¹⁹ however, it is associated with increased risk of radiation necrosis.⁵ Effective chemotherapeutic options in this setting remain elusive.³ It is in this context that stereotactic laser ablation (SLA) has been used. For SLA treatment of SRS-resistant metastases, a fiberoptic probe with laser-firing capacity is inserted into the cancerous tissue, where activation of the laser induces tissue destruction through thermocoagulation.^{4,9,17} Because clinical experiences with this technique remain limited, we present the pooled experience of 4 institutions where 26 BMs that recurred after SRS were treated with SLA.

Methods

Patient Population and Data Collection

The study was conducted under protocols approved by the institutional review boards at the following institutions: University of California San Diego (UCSD); Barrow Neurological Institute (BNI); Cleveland Clinic (CC); and Case Western (CW). Patient characteristics (age, sex, primary tumor histology, tumor location), corticosteroid regimens, and surgical morbidity information were collected through chart review. For BMs treated using the NeuroBlate system (Monteris Medical), tumor volume and percent of ablation covered by the blue isotherm line (tissue heated to 43°C for ≥ 10 minutes) were calculated by the company software. Information pertaining to percent tumor ablation for lesions treated with the Visualase (Medtronic) system was not available. Tumor volumes for these lesions were calculated using 3D Slicer (www.slicer.org), ImageJ (<https://imagej.nih.gov/ij/>), or by estimation based on maximal diameters. Surveillance MRIs were performed every 1–2 months after SLA for all treated patients. Radiographic responses were assessed based on the Neurologic Assessment in Neuro-Oncology (NANO) criteria at the time of the last follow-up.¹²

The SLA Procedures

The SLA procedures were performed by the following authors: C.C.C. (UCSD); G.B. and A.M. (CC); K.S. (BNI); and A.S. (CW). Details of NeuroBlate and Visualase treatment have been reported elsewhere.^{2,15} All patients were treated with the NeuroBlate system, with the exceptions of the patients in Cases 12–14, who were treated using Visualase. All UCSD procedures were performed in a conventional MRI suite. Procedures at CC, BNI, and CW were done in intraoperative MRI suites.

Adjuvant Hypofractionated SRS

All patients treated at UCSD received postoperative hypofractionated SRS. Approximately 1 month after SLA, these patients underwent thin-cut MRI (1-mm cut) obtained with contrast (wherein images are obtained after systemic administration of contrast to better visualize blood vessel permeability) and CT scans obtained without contrast (35-cm field of view, 512 × 512 pixel size, 1.25-

TABLE 1. Demographic and clinical characteristics in 23 patients with BMs after SRS

Case No.	Age	Sex	Primary Tumor	Location of Metastasis	Institution
1	67	F	Colon	Lt thalamus	UCSD
2	58	F	Melanoma	Lt thalamus	UCSD
3	54	F	Breast	Rt thalamus	UCSD
4	60	F	Breast	Lt frontal	UCSD
4*				Lt parietal	UCSD
5	42	F	Breast	Lt frontal	CC
5*				Lt thalamus	CC
6	64	F	Bladder	Lt motor strip	CC
7	44	F	Breast	Rt insula	CC
8	48	F	Breast	Lt basal ganglia	CC
9	48	F	Breast	Lt basal ganglia	CC
10	42	F	Lung	Rt frontal	CC
11	74	F	Colon	Lt cerebellum	CC
12	73	M	Sarcoma	Rt parietal	BNI
13	58	M	Melanoma	Lt frontal	BNI
14	70	F	Ovarian	Lt parietal	BNI
14*				Lt occipital	BNI
15	77	F	Lung	Lt frontal	BNI
16	54	F	Lung	Lt frontal	CW
17	55	M	Lung	Lt thalamus	CW
18	58	F	Melanoma	Rt occipitoparietal	CW
19	45	F	Melanoma	Lt frontal	CW
20	74	M	Lung	Lt occipitoparietal	CW
21	64	M	Melanoma	Lt frontal	CW
22	54	M	Esophagus	Lt frontal	CW
23	77	M	Lung	Rt occipital	CW

* In these patients 2 lesions were treated with SLA.

mm axial slice interval). Both MRI and CT studies were transferred to the treatment planning system, fused using a rigid autoregistration tool, and manually verified by the physicist and treating physicians. Planning was performed using Eclipse software, version 8.9 (Varian Medical Systems). The contrast-enhancing volume was contoured as clinical target volume by the treating neurosurgeon (C.C.C.) and radiation oncologist. The lesion was subsequently treated with SRS to the clinical target volume (5 Gy daily × 5 days).

Results

Patient and Tumor Characteristics

There were 7 male and 16 female patients (Table 1). Two patients each underwent 2 SLAs for separate lesions in a single operation, and a third patient underwent 2 serial SLAs for discrete BMs. The mean age of the cohort was 59 ± 11 years. Histological findings in the BMs included the following: breast (n = 6); lung (n = 6); melanoma (n = 5); colon (n = 2); ovarian (n = 1); bladder (n = 1); esophageal (n = 1); and sarcoma (n = 1). All BMs treated involved either the deep gray matter or eloquent cortex.

TABLE 2. Treatment characteristics associated with SLA

Case No.	Ablation Characteristics				Radiographic Response, per RANO Criteria		Surgical Morbidity &/or New Deficits
	Vol (cm ³)	No. of Trajectories	% Tumor Vol Covered by Blue Isotherm Line	Duration of FU (days)			
1	3.4	2	77.4	351	PR	None	
2	4.5	1	74.6	102	PR	Hydrocephalus requiring ventricular drainage	
3	5.5	1	98.2	252	PR	None	
4	3.2	1	92.4	138	PR	None	
4*	1.9	1	100	101	PR	None	
5	6.3	2	96.5	99	SD	None	
5*	5.3	1	63.0	94	PD	Transient rt hemiparesis 4/5†	
6	8.2	3	76.43	109	PD	Transient rt hemiparesis 4/5†	
7	16.9	3	86.45	64	SD	None	
8	3.5	1	97.86	146	PR	None	
9	28.9	1	99.28	83	PR	Acute neurological deterioration requiring hemicraniectomy	
10	2.7	1	100	170	PR	None	
11	2.6	1	100	145	SD	None	
12	2.3	1	NA	433	PD	None	
13	11.6	1	NA	77	PD	None	
14	10.1	1	NA	386	PR	None	
14*	0.4	1	NA	386	PR	None	
15	2.1	1	88.9	80	PD	None	
16	5.5	1	44.6	202	PD	Transient rt hemiparesis 4/5†	
17	12.1	1	73.6	652	PR	None	
18	4.1	1	86.2	338	PD	None	
19	3.7	1	69.3	795	PR	None	
20	9.6	1	67.8	124	PD	None	
21	11.2	1	88.3	323	PR	None	
22	13.1	1	70.1	91	PD	None	
23	4.4	1	88.6	76	SD	None	

CR = complete response; FU = follow-up; NA = not available; PD = progressive disease; PR = partial response; RANO = Radiologic Assessment in Neuro-Oncology; SD = stable disease.

* In these patients 2 lesions were treated with SLA. Patients treated with Visualase and % tumor treated were not calculated by the available software.

† Graded according to the MRC scale (Medical Research Council: *Aids to examination of the peripheral nervous system. Memorandum no. 45.* London: Her Majesty's Stationery Office, 1976).

The SLA Parameters

The median size of the lesion treated was 4.9 cm³ (range 0.4–28.9 cm³). The majority of the lesions were treated using single-trajectory SLA (n = 22, or 85%). Two BMs required 2 trajectories (8%), and 2 BMs required 3 trajectories (8%). On average, 84% of the tumor was ablated using SLA (range 44.6%–100%, Table 2). Complete ablation of the entire tumor volume, defined as 100% coverage of the tumor volume by the blue isotherm line, was achieved in only 3 of the 26 lesions (12%).

Radiographic Follow-Up

All patients were followed with serial MRI studies for at least 2 months. The median follow-up was 141 days (range 64–794 days). Based on the Neurologic Assessment in Neuro-Oncology criteria,¹² disease control was achieved in 17 BMs (defined as stable disease [Fig. 1], or partial response [Fig. 2]). Nine BMs (35%) showed disease progression after SLA. All instances of disease progres-

sion occurred in lesions that underwent < 80% ablation. In contrast, no disease progression was observed in BMs in which ≥ 80% ablation was achieved (Table 2).

Adjuvant Hypofractionated SRS Following SLA

Five BMs were treated with SLA, followed by adjuvant SRS (5 Gy daily × 5 days) 1 month later. The median follow-up time for these 5 BMs was 138 days (range 101–351 days), and no tumor growth was observed during follow-up. Twenty-one BMs were treated with SLA without subsequent SRS. With a median follow-up of 145 days, 9 of the 21 SLA-treated BMs showed radiographic evidence of tumor growth (43%). The frequency of disease progression in BMs treated with SLA followed by hypofractionated SRS was significantly lower than that observed in BMs treated with SLA alone (p < 0.05, Student t-test). No evidence of radiation necrosis or progressive FLAIR signal abnormalities was observed in the patients treated with adjuvant, hypofractionated SRS following SLA.

FIG. 1. MR images demonstrating stable BM disease control following SLA. **A:** Pretreatment axial (panel i), coronal (panel ii), and sagittal (panel iii) T1-weighted MR images obtained after administration of contrast and a FLAIR (panel iv) MR image of a deep left-sided BM. **B–E:** Axial (panel i), coronal (panel ii), and sagittal (panel iii) T1-weighted MR images obtained after administration of contrast, and a FLAIR (panel iv) MR image obtained 1 month (B), 3 months (C), 6 months (D), and 12 months (E) post-SLA, demonstrating no local recurrence.

FIG. 2. MR images demonstrating partial treatment response of a BM following SLA. **A:** Pretreatment axial (panel i), coronal (panel ii), and sagittal (panel iii) T1-weighted MR images obtained after administration of contrast and a FLAIR (panel iv) MR image of a left-sided infratentorial BM. **B and C:** Axial (panel i), coronal (panel ii), and sagittal (panel iii) T1-weighted MR images obtained after administration of contrast, and a FLAIR (panel iv) MR image obtained 1 day (B) and 5 months (C) post-SLA, demonstrating a partial treatment response to SLA.

Postoperative Corticosteroid Management

There is significant heterogeneity in dexamethasone management after SLA (Table 3). The highest dose regimen in this case series was 10 mg dexamethasone 4 times daily (QID) tapered over a span of 2 months. The most commonly used regimen was 4 mg dexamethasone 3 times daily (TID) tapered over a span of 2 weeks. In 1 patient a pretreatment MRI revealed minimal peri-BM edema, and this patient tolerated SLA without postoperative dexamethasone treatment.

Posttreatment Complications

Of the 23 SLA-treated patients, 3 suffered transient hemiparesis (13%) that recovered within 1 month of SLA; one of these patients underwent SLA for a BM located in the thalamus, another for a BM in the left frontal lobe, and the remaining patient underwent SLA for a BM located in the left motor strip. One patient with a left thalamic melanoma metastasis adjacent to the ventricle developed hydrocephalus after SLA, requiring temporary ventricular drainage (4%). One patient with a 28.9-cm³ lesion suffered

malignant cerebral edema after SLA, requiring an emergency hemicraniectomy (4%), and remained hemiplegic for the remainder of her life (83 days [Fig. 3]).

Discussion

In this report we provide radiographic follow-up for 26 BMs (in 23 patients) that recurred after SRS and were treated with SLA at 4 different institutions. The results indicate that SLA is an effective treatment option for BMs in which SRS fails. When $\geq 80\%$ of the lesion is ablated, tumor control was uniformly observed. Consideration of the morbidities encountered in this series suggests that resection might be indicated instead of SLA for BMs previously treated with SRS that exceed 20 cm³ in volume. Additionally, careful monitoring of hydrocephalus after SLA for periventricular BMs is warranted. Transient or permanent neurological deficits may be expected after SLA of thalamic and motor-strip lesions.

Our study further suggests that when $< 80\%$ of a BM is ablated by SLA, the efficacy of disease control may be

TABLE 3. Preoperative and postoperative steroid treatment regimens in patients who underwent SLA

Case No.	Preop Dexamethasone Regimen	Postop Dexamethasone Regimen
1	4 mg QID	4 mg TID × 4 d; 3 mg TID × 4 d; 2 mg TID × 4 d; 1 mg TID × 4 d; DC
2	4 mg QID	4 mg TID × 4 d; 3 mg TID × 4 d; 2 mg TID × 4 d; 1 mg TID × 4 d; DC
3	4 mg QID	4 mg TID × 4 d; 3 mg TID × 4 d; 2 mg TID × 4 d; 1 mg TID × 4 d; DC
4	4 mg QID	4 mg TID × 1 d; 3 mg TID × 1 d; 2 mg TID × 1 d; 1 mg TID × 1 d; DC
4*		4 mg TID × 1 d; 3 mg TID × 1 d; 2 mg TID × 1 d; 1 mg TID × 1 d; DC
5	4 mg QID	4 mg TID × 6 d; 4 mg OD & 2 mg BID × 6 d; 2 mg TID × 6 d; 2 mg BID until DC
6	2 mg BID	4 mg TID × 6 d; 2 mg BID × 6 d; 2 mg TID × 6 d; 2 mg BID × 6 d; DC
7	4 mg BID	4 mg BID × 5 d; 2 mg TID × 5 d; 2 mg BID × 5 d; DC
8	None	10 mg QID × 5 d; 6 mg QID × 5 d; 6 mg TID × 5 d; 4 mg QID × 5 d; 4 mg TID × 5 d; 4 mg BID × 5 d; 2 mg TID × 5 d; 2 mg BID × 20 d; DC
9	4 mg BID	10 mg QID × 5 d; 6 mg QID × 5 d; 6 mg TID × 5 d; 4 mg QID × 5 d; 4 mg TID × 5 d; 4 mg BID × 5 d; 2 mg TID × 5 d; 2 mg BID × 20 d; DC
10	4 mg BID	4 mg QID × 3 d; 4 mg TID × 4 d; 2 mg TID × 7 d; 1 mg BID × 7 d; DC
11	None	4 mg TID × 5 d; 4 mg BID × 5 d; 2 mg TID × 4 d; 2 mg BID × 4 d; DC
12	None	None
13	4 mg BID	4 mg QID × 2 d; 4 mg TID × 4 d; 2 mg BID × 4 d; 2 mg QD × 2 d; DC
14	4 mg BID	4 mg BID × 3 d; 2 mg BID × 3 d; 2 mg QD × 3 d; DC
15	4 mg BID	4 mg BID × 3 d; 2 mg BID × 3 d; 2 mg QD × 3 d; DC
16	4 mg BID	10 mg QID × 3 d; 6 mg TID × 3 d; 4 g BID × 3 d; 2 mg BID until postop visit in 2–3 wks
17	4 mg BID	10 mg QID × 3 d; 6 mg TID × 3 d; 4 g BID × 3 d; 2 mg BID until postop visit in 2–3 wks
18	4 mg BID	10 mg QID × 3 d; 6 mg TID × 3 d; 4 g BID × 3 d; 2 mg BID until postop visit in 2–3 wks
19	4 mg BID	10 mg QID × 3 d; 6 mg TID × 3 d; 4 g BID × 3 d; 2 mg BID until postop visit in 2–3 wks
20	4 mg BID	10 mg QID × 3 d; 6 mg TID × 3 d; 4 g BID × 3 d; 2 mg BID until postop visit in 2–3 wks
21	4 mg BID	10 mg QID × 3 d; 6 mg TID × 3 d; 4 g BID × 3 d; 2 mg BID until postop visit in 2–3 wks
22	4 mg BID	10 mg QID × 3 d; 6 mg TID × 3 d; 4 g BID × 3 d; 2 mg BID until postop visit in 2–3 wks
23	4 mg BID	10 mg QID × 3 d; 6 mg TID × 3 d; 4 g BID × 3 d; 2 mg BID until postop visit in 2–3 wks

BID = twice daily; d = day(s); DC = discontinued; OD = once daily; QD = daily.

* In this patient 2 lesions were treated with SLA.

augmented by adjuvant hypofractionated SRS (5 Gy daily × 5 days). Because SLA is typically preferred when the location of a BM involves regions of deep gray matter or eloquent cortex, incomplete ablation may be necessary to minimize the risk of neurological deficits. Our results indicate that hypofractionated SRS may augment the local control of these incompletely ablated tumors. Although the risk of radiation necrosis related to repeat SRS is a consideration,⁵ it is likely to be mitigated by SLA ablation of the tumor mass.^{10,18} Supporting this hypothesis, we did not observe radiographic evidence of radiation necrosis in patients treated with adjuvant, hypofractionated SRS following SLA. Nevertheless, the number of patients treated with combined SRS and hypofractionated SRS was limited. Validation of these results in future studies is warranted.

Temporary increases in cerebral edema after SLA is a well-described phenomenon.¹⁴ For this reason, most of the patients in this series were treated with corticosteroids after SLA. Although there are significant differences in surgeon preference regarding corticosteroid management, the most common regimen involved 4 mg dexamethasone TID tapered over a 2-week period. In cases in which the treating surgeon was concerned about the risk of malignant edema, 10 mg dexamethasone QID tapered over a 2-month period

was used. Given that 1 patient tolerated SLA-related edema without postprocedure corticosteroids, this treatment may not be necessary in select patients with minimal peri-BM edema.

The findings reported here are subject to all limitations inherent to case series in which there are a small number of patients and heterogeneous clinical practices. Admittedly, most patients in this series were followed for a limited duration, but this follow-up (median 141 days) must be taken in the context of the poor survival expectation of patients with Stage IV cancer with BMs in which SRS has failed.⁷ That said, assessment of radiation necrosis in patients who underwent adjuvant, hypofractionated SRS after SLA will require longer follow-up than that carried out in this study. Despite these limitations, we believe that our results offer pilot data for guiding future studies of SLA as a treatment for BMs in which SRS has failed.

Conclusions

Stereotactic laser ablation is an effective treatment option for patients suffering BM recurrence after SRS. Ablation of ≥ 80% of BMs is desirable to minimize risk of dis-

FIG. 3. Malignant cerebral edema following SLA for a large BM. **A:** The SLA trajectory for a 28.9-cm³ thalamic BM. **B:** Coronal T1-weighted MR image obtained after administration of contrast following hemispherectomy for symptomatic cerebral edema post-SLA.

ease progression. The efficacy of SLA may be augmented by adjuvant hypofractionated SRS.

References

1. Aoyama H, Shirato H, Tago M, Nakagawa K, Toyoda T, Hatano K, et al: Stereotactic radiosurgery plus whole-brain radiation therapy vs stereotactic radiosurgery alone for treatment of brain metastases: a randomized controlled trial. **JAMA** **295**:2483–2491, 2006
2. Carpentier A, McNichols RJ, Stafford RJ, Itzcovitz J, Guichard JP, Reizine D, et al: Real-time magnetic resonance-guided laser thermal therapy for focal metastatic brain tumors. **Neurosurgery** **63** (1 Suppl 1):ONS21–ONS29, 2008
3. Grimm SA: Treatment of brain metastases: chemotherapy. **Curr Oncol Rep** **14**:85–90, 2012
4. Hawasli AH, Kim AH, Dunn GP, Tran DD, Leuthardt EC: Stereotactic laser ablation of high-grade gliomas. **Neurosurg Focus** **37**(6):E1, 2014
5. Kwon KY, Kong DS, Lee JJ, Nam DH, Park K, Kim JH: Outcome of repeated radiosurgery for recurrent metastatic brain tumors. **Clin Neurol Neurosurg** **109**:132–137, 2007

6. Liew DN, Kano H, Kondziolka D, Mathieu D, Niranjan A, Flickinger JC, et al: Outcome predictors of Gamma Knife surgery for melanoma brain metastases. Clinical article. **J Neurosurg** **114**:769–779, 2011
7. Marshall DC, Marcus LP, Kim TE, McCutcheon BA, Goetsch SJ, Koiso T, et al: Management patterns of patients with cerebral metastases who underwent multiple stereotactic radiosurgeries. **J Neurooncol** **128**:119–128, 2016
8. Minniti G, Clarke E, Lanzetta G, Osti MF, Trasimeni G, Bozzao A, et al: Stereotactic radiosurgery for brain metastases: analysis of outcome and risk of brain radionecrosis. **Radiat Oncol** **6**:48, 2011
9. Mohammadi AM, Hawasli AH, Rodriguez A, Schroeder JL, Laxton AW, Elson P, et al: The role of laser interstitial thermal therapy in enhancing progression-free survival of difficult-to-access high-grade gliomas: a multicenter study. **Cancer Med** **3**:971–979, 2014
10. Rahmathulla G, Recinos PF, Valerio JE, Chao S, Barnett GH: Laser interstitial thermal therapy for focal cerebral radiation necrosis: a case report and literature review. **Stereotact Funct Neurosurg** **90**:192–200, 2012
11. Rava P, Sioshansi S, DiPetrillo T, Cosgrove R, Melhus C, Wu J, et al: Local recurrence and survival following stereotactic radiosurgery for brain metastases from small cell lung cancer. **Pract Radiat Oncol** **5**:e37–e44, 2015
12. Reardon DA, Nayak L, DeAngelis LM, Wen PY, Brandes AA, Soffietti R, et al: The Neurologic Assessment in Neuro-Oncology (NANO) Scale: A tool to assess neurologic function for integration in the radiologic assessment in neuro-oncology (RANO) criteria. **Neuro Oncol** **16** (Suppl 2):ii76, 2014 (Abstract P15.13)
13. Regine WF, Huhn JL, Patchell RA, St Clair WH, Strottmann J, Meigooni A, et al: Risk of symptomatic brain tumor recurrence and neurologic deficit after radiosurgery alone in patients with newly diagnosed brain metastases: results and implications. **Int J Radiat Oncol Biol Phys** **52**:333–338, 2002
14. Schwabe B, Kahn T, Harth T, Ulrich F, Schwarzaier HJ: Laser-induced thermal lesions in the human brain: short- and long-term appearance on MRI. **J Comput Assist Tomogr** **21**:818–825, 1997
15. Sloan AE, Ahluwalia MS, Valerio-Pascua J, Manjila S, Torchia MG, Jones SE, et al: Results of the NeuroBlate System first-in-humans Phase I clinical trial for recurrent glioblastoma: clinical article. **J Neurosurg** **118**:1202–1219, 2013
16. Soliman H, Das S, Larson DA, Sahgal A: Stereotactic radiosurgery (SRS) in the modern management of patients with brain metastases. **Oncotarget** **7**:12318–12330, 2016
17. Sugiyama K, Sakai T, Fujishima I, Ryu H, Uemura K, Yokoyama T: Stereotactic interstitial laser-hyperthermia using Nd-YAG laser. **Stereotact Funct Neurosurg** **54-55**:501–505, 1990
18. Torres-Reveron J, Tomasiewicz HC, Shetty A, Amankulor NM, Chiang VL: Stereotactic laser induced thermotherapy (LITT): a novel treatment for brain lesions regrowing after radiosurgery. **J Neurooncol** **113**:495–503, 2013
19. Yomo S, Hayashi M: Salvage stereotactic radiosurgery with adjuvant use of bevacizumab for heavily treated recurrent brain metastases: a preliminary report. **J Neurooncol** **127**:119–126, 2016

Disclosures

Dr. Chen is a consultant for Monteris Medical and has consulted for MRI Interventions. Drs. Mohammadi and Sloan are consultants for Monteris Medical, Inc. Dr. Smith is a consultant for Monteris Medical, Inc.; Medtronic, Inc.; and OsteoMed.

Author Contributions

Conception and design: Chen. Acquisition of data: Chen, Hamelin, Chang, Lemkuil, Sharma, Barnholtz-Sloan, Myers, Barnett, Smith, Mohammadi, Sloan. Analysis and interpretation of data: Chen, Ali, Carroll, Rennert, Hamelin, Chang, Lemkuil, Sharma, Barnholtz-Sloan, Myers, Barnett, Smith, Mohammadi. Drafting the article: Chen, Ali, Carroll, Rennert, Sloan. Critically revising the article: Chen, Ali, Carroll, Rennert, Sharma, Barnholtz-Sloan, Barnett, Smith, Mohammadi, Sloan. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Chen.

Correspondence

Clark C. Chen, Department of Neurosurgery, University of California, San Diego, 3855 Health Sciences Dr. #0987, La Jolla, CA 92093. email: clarkchen@ucsd.edu.