

STEREOTACTIC RADIOTHERAPY FOR THE TREATMENT OF ACOUSTIC NEUROMAS

Clinical White Paper

Acoustic neuromas (AN) have an annual incidence of approximately one per 100,000 people and may account for up to 8% of all new tumors presenting to a neurosurgical referral practice¹. Acoustic neuromas are benign tumors arising from Schwann cells from the vestibular branch of the eighth cranial nerve. Nevertheless, they can pursue a potentially aggressive course, with uncontrolled local growth resulting in compression of the brainstem and fourth ventricle, cranial nerve and other neurological deficits².

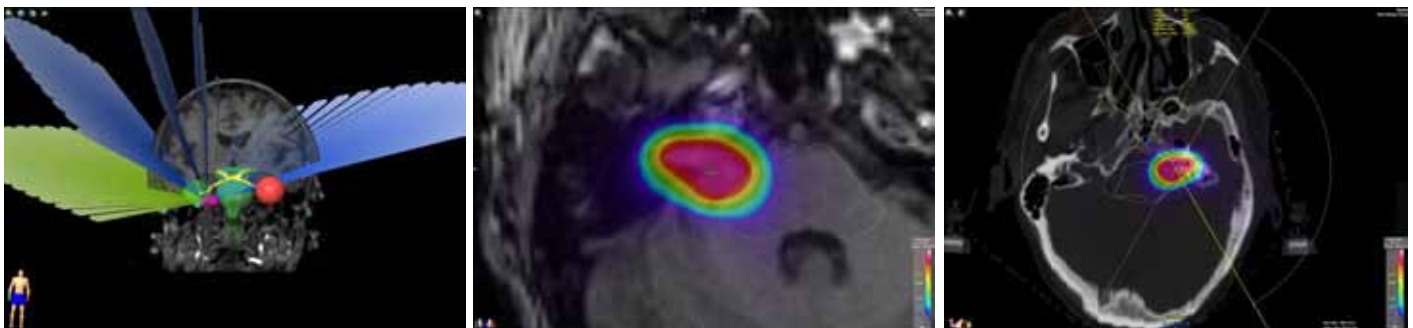


Figure: Details of a typical acoustic neuroma treatment plan

Acoustic neuroma growth rates are variable and inconsistent³. An observational wait-and-see approach advocated by many authors, if patients follow up with regular MRI scans and clinical review, can be maintained.

Surgical excision has been the standard treatment for AN, should this become necessary⁴. In recent years, an increasing number of clinical publications regarding stereotactic radiosurgery (SRS) and stereotactic radiotherapy (SRT) have established these techniques as a safe, efficient and minimally invasive alternative⁵⁻¹¹.

Furthermore, stereotactic irradiation has gained increased acceptance with the publication of long-term follow up data suggesting high rates of long-term control¹². SRS is being used increasingly on younger patients¹³. Concern over the additional surgical risks, should progressive tumor growth occur after SRS, remains a controversial but legitimate issue¹⁴⁻¹⁶, although descriptions of salvage stereotactic irradiation, as an alternative strategy, are described¹⁷.

While microsurgery and SRS/SRT achieve similar success rates, no randomized controlled trial exists to guide management. Surgical removal of AN, even with experienced surgeons and modern microsurgical techniques, may be associated with morbidity, including loss of useful hearing, temporary or permanent facial and trigeminal nerve dysfunction^{18,19}.

Combined approaches may be useful for larger tumors to reduce morbidity, as well as the risk of recurrence¹⁶. Therefore, in the case of a large tumor resulting in brainstem compression and progressive neurological signs, a combined strategy of microsurgical resection followed by SRS or SRT can often be very helpful and the treatment of choice to preserve cranial nerve function and limit the recurrence rate.

For both microsurgery and SRS, the risks of facial, trigeminal and auditory dysfunction are proportional to the size of the treated AN^{20,21}. Cochlear dose also appears a significant factor in hearing preservation after SRS/SRT^{22,23}. SRT results suggest that some cranial nerve dysfunction may be avoided, even with larger tumors⁶.

SRT of an AN is typically performed with a dedicated linear accelerator equipped with a micro-multi-leaf collimator, which improves dose conformity over conical collimators. When comparing treatment details, the dynamic arc technique showed a statistically significant increase in the minimum dose to the target and a better conformity index over fixed fields¹.

A summary table of some important recent results concerning the SRT treatment of an AN is presented below. In these studies, results are described in terms of local control and hearing preservation.

While local control rates may be overestimated for small tumors, many of the series listed, describe AN with dimensions greater than 18 mm. Here, tumors have been shown to have enhanced proliferative activity, suggesting a meaningful response to SRT treatment²⁴.

Hearing preservation was typically determined using the Gardner-Robertson hearing classification scheme²⁵. Useful or serviceable hearing was defined as Gardner-Robertson Class I or II after treatment.

Sometimes useful hearing was subjectively defined as the unaided ability to discriminate normal speech and use a telephone with the affected ear.

From the recent literature, it can be concluded that SRT, using advanced imaging and highly conformal field shaping, achieves high rates of tumor control and preservation of useful hearing from 57 to 93%. Because this minimally invasive technique produces only low rates of damage to the facial and trigeminal nerves, SRT can be considered a low-risk and highly effective treatment option.

Overview of the recent clinical literature on SRT for acoustic neuromas

Author	Institution	Year	# Patients	Tumor Diameter (mm)	Dose (Gy)	# Fractions	% IDL Covering PTV	% Local Control	% Hearing Preservation	% Nerve Toxicity Trigeminal / Facial
Williams ⁶	John Hopkins University, Baltimore	2002	125	20	25	5	80	100	64 at 2 years	0 / 0
Meijer ⁵	VU Medical Center, Amsterdam	2003	80	25	20 - 25	5	80	94	61 at 5 years	2 / 3
Selch ⁹	David Geffen School of Medicine, Los Angeles	2004	48	22	54	30	90	100	93 at 3 years	2 / 2
Chung ¹⁰	BC Cancer Agency, Vancouver	2004	27	16	45	25	90	100	57 at 2 years	7 / 4
Combs ⁸	University of Heidelberg	2010	172	N/A	58	32	90	96	78 at 5 years	3 / 2
Meijer ⁷	VU Medical Center, Amsterdam	2008	20	25	20 - 25	5	80	100	N/A	0 / 0
Andrews ¹¹	Thomas Jefferson University, Philadelphia	2009	46	N/A	46.8	26	90	100	79 at 3 years	0 / 0

Hearing preservation was typically determined using the Gardner-Robertson hearing classification scheme. Useful or serviceable hearing was considered if patients qualified as Gardner-Robertson Class I or II after treatment. Sometimes useful hearing was subjectively defined as the unaided ability to discriminate normal speech and use a telephone with the affected ear.

References

- | | |
|---|--|
| <p>[1] Perks J.R. et al., Int J Radiat Oncol Biol Phys 57, 1450, 2003</p> <p>[2] Charabi S. et al., Otolaryngol Head Neck Surg 113, 5, 1995</p> <p>[3] Shirato H. et al., Int J Radiat Oncol Biol Phys 44, 545, 1999</p> <p>[4] Eldridge R. et al., Neurosurg 30, 962, 1992</p> <p>[5] Meijer O.W.M. et al., Int J Radiat Oncol Biol Phys 56, 1390, 2003</p> <p>[6] Williams J.A. et al., Int J Radiat Oncol Biol Phys 54(2), 500, 2002</p> <p>[7] Meijer O.W.M. et al., Neurosurg 62, A37, 2008</p> <p>[8] Combs S.E. et al., Int J Radiat Oncol Biol Phys 76(1), 193, 2010</p> <p>[9] Selch M.T. et al., J Neurosurg 101, 362, 2004</p> <p>[10] Chung H.T. et al., Int J Radiat Oncol Biol Phys 59(4), 1116, 2004</p> <p>[11] Andrews D.W. et al., Int J Radiat Oncol Biol Phys 74(2), 419, 2009</p> <p>[12] Kondziolka D. et al., Neurosurg 53, 815, 2003</p> <p>[13] Lobato-Polo J. et al., Neurosurg 65, 294, 2009</p> | <p>[14] Pollock B.E., Neurosurg 58, 241, 2006</p> <p>[15] Roche P.H. et al., Prog Neurol Surg 21, 152, 2008</p> <p>[16] Iwai Y. et al., Neurosurg 60, ONS75, 2007</p> <p>[17] Yomo S. et al., Neurosurg 64, 48, 2009</p> <p>[18] Samii M. et al., Neurosurg 40, 248, 1997</p> <p>[19] Samii M. et al., Neurosurg 40, 684, 1997</p> <p>[20] Wiet R.J. et al., Otolaryngol Head Neck Surg 124, 645, 2001</p> <p>[21] Flickinger J.C., Int J Radiat Oncol Biol Phys 17, 879, 1989</p> <p>[22] Lasak J.M. et al., Otol Neurotol 29, 1179, 2008</p> <p>[23] Thomas C. et al., J Neurosurg 107, 917, 2007</p> <p>[24] Bedavanija A. et al., J Neurosurg 98, 807, 2003</p> <p>[25] Gardner G. et al., Ann Otol Rhinol Laryngol 97, 55, 1988</p> |
|---|--|

Europe | +49 89 99 1568 0 | de_sales@brainlab.com
 North America | +1 800 784 7700 | us_sales@brainlab.com
 Latin America | +55 11 3355 3370 | br_sales@brainlab.com

Asia Pacific | +852 2417 1881 | hk_sales@brainlab.com
 Japan | +81 3 3769 6900 | jp_sales@brainlab.com