Stereotactic Body Radiotherapy (SBRT) for Spinal Tumors

I. Introduction:

This white paper will focus on spinal radiosurgery - with sections one though five (**I-V**) comprising a general review of spinal tumors from eMedicine.com, more information can be found at http://emedicine.medscape.com/article/1267223-overview (Used with permission from eMedicine.com, 2010). Section six (**VI**) will provide a literature review on stereotactic radiosurgery (SRS) for spinal tumors and section seven (**VII**) (Society Members Only) will provide clinical indications and treatment guidelines on SRS for tumors of the spine.

II. Definition and incidence:

Primary spinal tumors fall into a distinct category because their timely diagnosis and the immediate institution of treatment have an enormous impact on the patient's overall prognosis and hope for a cure. Generally, with spinal pathology, problems that arise are either chronic problems related to degenerative disease or deformity or acute manifestations of traumatic sequelae. When considering tumors of the spine, one must consider the different tissue types around the spinal column. The presence of neural tissue, meningeal tissue, bone, and cartilage make any of these tissue types a possible nidus for neoplastic change. Also, metastatic lesions may spread to the spine from distant primary tumor sites by hematogenous or lymphatic routes.

Primary nonlymphoproliferative tumors of the spine are uncommon and make up less than 5% of bone neoplasms, comprising less than 2.5 to 8.5 primary spine tumors per 100,000 people per year. Metastatic disease of the spine is much more common. Metatstaic spine disease accounts for 10-30% of new cancer diagnosis annually. [1] Approximately 40-80% of patients who die of cancer have bony metastases at the time of death, with the spine being the most common metastatic skeletal location.

III. Differential diagnosis:

Neoplastic disease, however, can present with back pain that is indistinguishable from back pain resulting from more benign causes. Therefore, the physician caring for patients complaining of back pain is faced with the challenge of distinguishing benign causes from those that can be neurologically or systemically devastating and prescribing the appropriate treatment. This sometimes can be difficult because of the complicated architecture of the spine. The physician must consider differential diagnoses of degenerative processes, infections, muscular strains, neurologic impingements, and, finally, neoplastic processes. With thorough history taking, physical examination, and diagnostic imaging, the physician can acquire enough information to efficiently make the correct diagnosis.[2]

The most common clinical presentation associated with all spine tumors is back pain that causes the patient to seek medical attention. Back pain is the most frequent symptom for

patients with either benign or malignant neoplasms of the spine. Neurologic deficits secondary to compression of the spinal cord or nerve roots also can be part of the presentation. The degree of neurologic compromise can vary from slight weakness or an abnormal reflex to complete paraplegia, depending on the degree of encroachment. The loss of bowel or bladder continence can occur from neurologic compression or can be secondary to a local mass effect from a tumor in the sacrococcygeal region of the spine, as occurs in chordomas. Systemic or constitutional symptoms tend to be more common with malignant or metastatic disease than in benign lesions.

For these patients, workup should include a complete blood count and differential, a basic serum chemistry profile, erythrocyte sedimentation rate, or C-reactive protein to help distinguish between neoplastic and infectious processes. Elevations in serum calcium or alkaline phosphatase also can provide evidence for neoplastic bone processes. Specific studies, such as serum electrophoresis or urine electrophoresis, also can be performed to evaluate the likelihood of multiple myeloma or plasmacytoma.

Imaging studies for the workup of spine tumors include plain x-rays, CT scan, MRI, and a technetium bone scan.[3] The first-line imaging study should be plain x-ray to evaluate the trabecular architecture of the spine. Anteroposterior (AP), lateral, and oblique views may be necessary to evaluate the lesion. These studies should be evaluated for what the tumor is doing to the bone and, conversely, for what the bone is doing to the tumor. The blastic or lytic nature of the lesion should be noted. The general location of the lesion within the bone, the integrity of the cortex, and the presence of fractures or soft tissue masses are important findings.

The ultimate way to make the diagnosis and ascertain the specific tumor type is by performing a biopsy of the spine lesion after all radiographic studies have been completed. Biopsies can be performed with open or by percutaneous image-guided technique. Percutaneous needle biopsies may not supply adequate tissue for the diagnosis of a primary tumor of the spine.

The basic principles of biopsy technique also apply to tumors of the spine. The surgeon performing the biopsy should take the most direct route to the tumor, with the least potential to contaminate adjacent compartments. The biopsy tract should be placed in line with the future incision site for surgical resection of the tumor, so that the biopsy tract can be excised with the specimen en bloc. Meticulous hemostasis must be obtained, and a drain must be placed to prevent hematoma formation, which can dissect the soft tissue planes and contaminate adjacent compartments. The drain should exit the skin in line with the incision so that it, too, can be excised with the final specimen.

IV. Pathophysiology:

The spine consists of 33 vertebrae that form the bony spinal column. The spinal column can be divided into the cervical, thoracic, lumbar, and sacrococcygeal regions. Although morphologically distinct, each vertebra in the subaxial cervical, thoracic, and lumbar

spine has a complex architecture, consisting of a vertebral body, pedicles, laminae, and spinous and transverse processes. The bony canal provides protection and support to the fragile spinal cord and nerve roots within the dural sac. The soft tissues surrounding the bony spine vary by location from the thick dorsal paraspinous musculature to the vital organs and vessels within the mediastinal, thoracic, peritoneal, and retroperitoneal spaces. The relevant anatomy discussed above is frequently the limiting factor when determining contraindications to surgical excision of spine tumors. The morbidity of the tumor, the tumor's malignant potential, and the patient's overall prognosis must be compared to the morbidity and potential mortality of radical resection of a tumor near the spinal cord, the aorta, or the heart. The degree of associated blood loss and the overall health of the patient also must be taken into consideration when considering a resection. If the patient is known to have metastatic or systemic tumor involvement, this may be a contraindication to radical resection of a paraspinous tumor, which may render the patient paralyzed.

Weinstein and McLain[4], and Boriani et al[5] have developed a descriptive staging system for spine tumors based on the principles of Enneking staging system for primary bone tumors of the extremities. In their staging system for the spine, the transverse extension of the vertebral tumor is described with reference to 12 radiating zones numbered 1 to 12 in a clockwise order, and to 5 concentric layers A to E, from the paravertebral extraosseous compartments to the dural involvement. The longitudinal extent of the tumor is recorded according to the levels involved. Based on an understanding of the biologic behavior of the tumor, the oncologic staging aids the surgeon to decide what surgical margin provides the best chance for complete tumor resection and possible cure. This system is complex and sometimes difficult to apply clinically.

Enneking classification of benign lesions applies to benign spine tumors. Lesions can be latent (stage 1), active (stage 2), or aggressive (stage 3). Stage 1 lesions are usually asymptomatic and are discovered incidentally. Stage 2 lesions usually present with symptoms; most commonly, pain is in the area of the lesion. Stage 3 lesions are locally aggressive and can actually metastasize.[6]

The histologic findings vary according to the tumor types. The following list revisits the primary tissue type associated with some of the tumors of the spine:

Bone producing tumors of the spine:

Enostosis: Also termed a bone island, enostosis is a mass of calcified medullary defects of lamellar compact bone with haversian systems found within the cancellous portion of the bone. Enostosis occurs most frequently in the thoracic and lumbar spine, usually between T1 and T7 and between L1 and L2. Enostosis is one of the most common lesions to involve the spine. Enostoses are usually stage 1 lesions and are discovered incidentally. Most remain stable, but some may slowly increase in size. Resnik et al determined the incidence of enostosis to be approximately 14% in cadavers. [7] Radiographically, enostoses are circular or oblong osteoblastic lesions with a spiculated margin, which gives it the appearance of thorny periphery. An abrupt transition from

normal to the sclerotic bone is exhibited on the x-ray. Bone scan findings are usually normal, and MRI demonstrates low signal intensity with normal surrounding intensity. Enostosis sometimes can be confused with osteoblastic metastatic disease. Enostosis can be differentiated by lack of activity on bone scan, by the normal appearance of adjacent bone, by its thorny margins, and by lack of a primary tumor for metastasis. If the enostosis exhibits an increase in diameter of greater than 25% in 6 months, a biopsy should be performed.

Osteoid osteoma: Benign and locally self-limited. Osteoid osteomas usually present in children aged 10-20 years, with a male predominance. They involve the axial skeleton only 10% of the time. In the spine, 59% of osteoid osteomas are found in the lumbar region, 27% in the cervical region, 12% in the thoracic region, and 2% in the sacral region.[8] Osteoid osteomas are usually stage 2 lesions and are actively symptomatic. Osteoid osteomas can result in painful scoliosis, radicular pain, gait disturbances secondary to pain and splinting, and muscular atrophy. Symptoms usually are relieved or ameliorated by nonsteroidal anti-inflammatory drugs (NSAIDs) or salicylates. In the spine, osteoid osteomas occur 75% of the time in the posterior elements, either the pedicles, facets, or laminae. Osteoid osteomas occur 7% of the time in the vertebral body and 18% of the time in the transverse and spinous processes. On plain x-ray, osteoid osteomas appear as a round or oval radiolucent nidus, with a surrounding rim of sclerotic bone. An area of central calcification may be present, but this classic appearance may be obscured by complex spinal architecture. On bone scan, marked increased uptake by the nidus is demonstrated, and a double intensity pattern may exist. CT scan is the criterion standard for radiographic diagnosis. The nidus is well-defined area of low attenuation with or without central calcification surrounded by an area of sclerosis. The nidus is usually smaller than 1.5 to 2.0 cm, composed of microscopic well-organized trabecular bone with vascular fibrous connective tissue stroma surrounded by reactive cortical bone.

Osteoblastoma: Benign but locally expansile and aggressive. Histologically similar in appearance to the osteoid osteoma, the osteoblastoma is behaviorally very different. Demographically, it occurs in young patients in the second or third decade of life. A 2:1 male-to-female predominance exists. The lesion is distributed equally in the cervical, thoracic, and lumbar segments of the spine. The posterior elements are involved in 55% of cases, but the tumor can extend to the vertebral body in 42% of cases. Patients typically complain of dull localized pain and paresthesias, paraparesis and, if the tumor is large enough and encroaching on the spinal cord, paralysis. Osteoblastomas are expansile lesions with multiple small calcifications and a peripheral scalloped and sclerotic rim. In more aggressive lesions, osseous expansion, bone destruction, infiltration of the surrounding tissue, and intermixed matrix calcification are present. Fifty percent of osteoblastomas are radiolucent, and 20% are osteoblastic. Marked radionucleotide uptake is exhibited on bone scan. CT scan demonstrates areas of mineralization, expansile bone remodeling, and sclerosis or a thin osseous shell at its margins. MRI is nonspecific but is the criterion standard to assess the effect of the tumor on the cord and surrounding tissues. Osteoblastomas are typically larger than 2.0 cm in diameter with histologic features of interconnecting trabecular bone and fibrovascular stroma similar to, but not as

well organized as, osteoid osteoma. They can have an aneurysmal bone cyst component in 10 to 15% of cases.

Osteosarcoma: Malignant spindle cell lesion that produces osteoid. Osteosarcomas of the spine are rare, making up only 0.6 to 3.2% of all osteosarcomas and only 5% of all primary malignant tumors of the spine. They typically present in patients in the fourth decade of life and have a male predominance. Osteosarcomas are found at all levels of the spine but are most common in the lumbosacral segments. Eccentric involvement of the vertebral body with extension into the posterior elements is common. Patients often present with pain and a palpable mass. Neurologic symptoms, ranging from sensory deficits to paresis, are found in 70 to 80% of patients. Serum alkaline phosphatase may be elevated. Plain x-rays of spinal osteosarcomas reveal a densely mineralized matrix, giving rise to the term ivory vertebrae. A loss of vertebral height often occurs, with sparing of the adjacent disc. Purely lytic lesions also have been described. CT scans and MRIs are useful to evaluate the extent of bony and soft tissue involvement. If a large amount of mineralized matrix is present, the lesion may appear with low signal intensity on all MRI sequences. Most osteosarcomas are blastic lesions. They can be osteoblastic, chondroblastic, or fibroblastic. Osteosarcomas can arise primarily or secondarily from an exposure to radiation. Secondary osteosarcomas can have a latency of up to 20 years. Spinal osteosarcomas also have been found in patients with Paget disease.

Cartilage producing tumors of the spine:

Osteochondroma: Benign lesion with cartilaginous cap, osteochondromas make up 4% of all solitary spine tumors. They also are commonly referred to as exostosis. Spinal lesions are encountered in 7 to 9% of patients with multiple hereditary exostoses (MHE). Osteochondromas occur in patients aged 20 to 30 years. Patients with MHE tend to develop the osteochondroma at a younger age; they also tend to experience neurologic deficits and myelopathy more frequently (77% of the time) than the patient with solitary osteochondroma (34%). A male predominance exists. Osteochondromas are more common in the cervical spine, especially at C2. The posterior elements usually are involved. The lesions are believed to arise secondary to trapping of the physeal cartilage outside the growth plate during skeletal development. Making the diagnosis of osteochondroma in the spine on plain radiography can be difficult unless the lesion is large and protruding posteriorly from a spinous process. In fact, 15% of patients with osteochondromas of the spine have normal appearing x-rays. CT scan is the study of choice to detect the exostosis and determine its relationship to the surrounding soft tissue and spinal canal. T1-weighted MRI scans reveal a central area of high signal intensity, which represents yellow marrow. This area has intermediate intensity on T2-weighted images. The cortex of the exostosis has low signal intensity. The hyaline cartilage cap of the exostosis is best evaluated with MRI and appears with low signal intensity on T1 and high intensity on T2. The cartilage cap should be less than 2 cm in adults. Lesions with cartilage caps greater than 2 cm should be suspected of malignant transformation to chondrosarcoma. Qualitatively, the bone composing an osteochondroma is normal. Abnormal bone growth occurs at and as a result of the cartilage cap. A continuity of the

lesion with the marrow and cortex of the underlying bone is present. The exostosis may be sessile or pedunculated.

Chondrosarcoma: Malignant cartilage producing tumors that histologically demonstrate round cellular stroma in a chondroid matrix. Chondrosarcoma is the second most common nonlymphoproliferative tumor of the spine. Chondrosarcomas comprise 7 to 12% of all spine tumors, and the spine is the primary site in 3 to 12% of all chondrosarcomas. Men are affected 2 to 4 times more frequently than women. The mean age of presentation is 45 years. The thoracic spine is the most common site, but chondrosarcomas can occur at all levels of the spine. The most common symptoms are pain, a palpable mass, and neurologic complaints in 45% of patients. Plain radiographs of chondrosarcomas typically demonstrate bone destruction. The lesions may be apparent in the vertebral body 15% of the time, in the posterior elements 40% of the time, or in both 45% of the time. In 70% of patients, the characteristic chondroid matrix in the form of rings and arcs are apparent on x-ray. Cortical destruction with soft tissue extension is best observed on CT scan or MRI. Chondrosarcomas that arise from malignant transformation of osteochondromas are observed as a thickening of the cartilaginous cap. Involvement of the adjacent vertebral levels by extension through the disc is observed in 35% of all lesions. On CT scan or MRI, mineralization is usually apparent in the soft tissue component of the lesion. The radionucleotide uptake by the lesion is intense and has a heterogeneous appearance on bone scan. Chondrosarcomas are relatively low-grade lesions (grade I or II). Most lesions are primary chondrosarcomas rather than secondary chondrosarcomas that arise from the malignant degeneration of osteochondromas as previously noted. Chondrosarcomas have relatively sparse cartilaginous stroma with a surrounding pseudocapsule. Examination under higher magnification reveals atypical nuclei with several mitotic figures per high-powered field.

Lymphoproliferative tumors:

Multiple myeloma: Derived from plasma cell dyscrasias, which histologically appear as sheets of plasma cells. Multiple myeloma is a systemic disease that affects middle-aged people and is characterized by areas of local bone destruction. Multiple myeloma is the most common primary malignancy of bone and the spine. The underlying cell line is the malignant plasma cell, which produces abnormal quantities of immunoglobulins. The presentation of patients with myeloma is similar to that of other spine tumor patients. Patients complain of pain that may be worse at night. The laboratory workup for these patients should include a complete blood count with differential looking for anemia and thrombocytopenia, an elevation of the erythrocyte sedimentation rate, and a decrease in the serum albumin with increased total serum protein. The abnormal production of immunoglobulins can be detected on serum or urine electrophoresis and can be used to confirm the diagnosis. Radiographically, skeletal survey is used to screen for lesions that can occur throughout the skeleton. Bone scans have a high false-negative rate and are not optimal studies for the evaluation of myeloma. Once a lesion is detected in the spine, CT scan, MRI, or both should be performed to assess the destruction of the vertebrae and the effect of this destruction on the surrounding neurologic and paraspinous tissues.

Plasmacytomas: Akin to multiple myeloma as a descendent of plasma cell malignancies, the plasmacytoma is a solitary lesion that usually affects the vertebral body. Plasmacytomas generally affect younger patients than multiple myeloma and are associated with a better prognosis. Plasmacytomas eventually can evolve into multiple myeloma; thus, patients should be monitored for more than 20 years following the original diagnosis of plasmacytoma.

Tumor of notochordal origin:

Chordoma: Identified by the characteristic physaliferous cells. Luschka first described chordoma morphologically in 1856 in Virchow's lab. The discovery of the notochordal nature of the tumor and the coining of the term chordoma is credited to Ribbert in 1894. Chordomas are uncommon tumors comprising 2 to 4% of all primary malignant bone tumors with a prevalence of 0.51 per million. However, they are excluding lymphoproliferative tumors and metastases, the most common primary malignant tumor of the spine in the adult. As Ribbert described, chordomas arise for the notochord remnant. The notochord normally evolves into the nucleus pulposus of the intervertebral discs. Nonneoplastic notochord vestiges also are found at the midline of the sphenooccipital synchondrosis and in the sacrococcygeal regions. The locations in which chordomas occur parallel these vestigial distributions. Regarding chordoma prevalence, 30 to 35% occur in the sphenooccipital region, 50% in the sacrococcygeal region (especially S4-S5), and 15% occur in the other spinal segments. Interestingly, chordomas have not been reported to arise from the intervertebral discs. Chordomas occur most commonly in patients aged 30 to 70 years, with a peak incidence in the fifth to sixth decades of life. Sphenooccipital lesions have equal sex distributions but sacrococcygeal lesions have a 3:1 male-to-female ratio. Presentation of chordomas is often subtle, with a gradual onset of pain, numbness, motor weakness, and constipation or incontinence. Constipation is a uniform finding in most patients with sacrococcygeal lesions. Chordomas are typically slow growing lesions and are often very large at the time of presentation. On plain x-ray, chordomas appear as a destructive lesion of a vertebral body in the midline, with a large associated soft tissue mass. In sacrococcygeal lesions, osseous expansion is frequent and may extend across the sacroiliac joints. Mineralization within the tumor may be observed on the plain x-rays of 50 to 70% of sacrococcygeal lesions. The mineralization is amorphous and predominates in the periphery of the lesion. Lesions in spinal segments above the sacrum are less expansile and demonstrate evidence of calcification in only 30% of cases. They may have areas of sclerosis in 43 to 62% of cases. The intervertebral discs above or below a chordoma may be involved and narrowed in a manner that simulates infection. The lesion can make its way through the intervertebral disc to infiltrate an adjacent level. This occurs in approximately 11 to 14% of cases. CT scan demonstrates both the osseous and soft tissue components of the tumor. Coronal and sagittal reconstructions of the CT scan are helpful in assessing neural foraminal and sacroiliac joint involvement. MRI scans are an important adjunct in the workup of chordomas. The lesions appear with low-to-intermediate signal intensity on T1 images with very high signal intensity on T2 images, reflecting the high water content of chordomas. Enhancement occurs following intravenous contrast on both CT scan and MRI. Chordomas are lobulated neoplasms, which usually are contained within a

pseudocapsule. Histology of these lesions reveals long cords of physaliphorous cells. Physaliphorous cells are clear cells containing intracytoplasmic vacuoles with abundant intracellular and extracellular mucin. Sarcomatous chondroid, osteoid, or fibroid elements may be found within the chordoma.

Round cell tumor:

Ewing sarcoma: Malignant tumor of childhood associated with large sheet of homogenous small, round, blue cells. Ewing sarcoma is the most common nonlymphoproliferative primary malignant tumor of the spine in children. Lesions of the spine comprise 3 to10% of all primary sites of Ewing sarcoma. Metastatic foci of Ewing sarcoma involving the spine are more common than primary lesions of the spine. Patients with Ewing sarcoma usually present when aged 10 to 20 years. The most common site of occurrence in the spine is the sacrococcygeal region followed by the lumbar and thoracic segments. Ewing sarcoma rarely occurs in the cervical spine. Lesions are centered primarily in the vertebral body but they can extend into the posterior elements.[9] Plain x-rays reveal permeative bone lysis, osseous expansion, or sclerosis. Diffuse sclerosis is observed in 69% of spinal lesions and is associated with osteonecrosis. CT scans and MRIs demonstrate osseous involvement as well as surrounding soft tissue involvement. However, MRI is nonspecific. Tissue from a Ewing sarcoma is composed of sheets of small, round, blue cells divided by septa, scant cytoplasm, and abundant collagen. Areas of osteonecrosis are found in spinal lesions. These correspond to the sclerotic areas observed on plain x-rays as discussed above. Genetically, patients with Ewing sarcoma are found to have an 11;12 chromosomal translocation.

Metastatic tumors in the spine:

The most common tumors that metastasize to the spine are as follows:

- Prostate carcinoma
- Breast adenocarcinoma
- Lung adenocarcinoma
- Renal cell carcinoma
- Gastric carcinoma

Tatsui et al found that patients with prostate cancer had the highest rate of metastases to the spine.[10] They also found that lung cancer was the most common primary lesion in patients whose spinal metastases were detected before the diagnosis of primary lesions.

The time from diagnosis of the primary lesion to the time the spinal metastasis was detected has demonstrated to be shortest in patients with lung cancer. It was longest in patients with breast cancer. Patients with metastases from breast cancer or prostate cancer had the highest 1-year survival rate, whereas patients with metastases from lung or gastric cancer had the lowest 1-year survival rates.

V. Treatment options:

Treatment varies depending on the tumor type and location. Levine and Crandall offer an excellent summary of the treatment of primary malignant tumors of the spine.[11] For treatment of astrocytomas, a less common spinal tumor, see Minnehan et al. [12]For treatment for nonambulatory patients, see Kondo et al. [13] Palliative surgery for metastatic thoracic and lumbar tumors is presented by Cho and Sung.[14]

Bone producing tumors of the spine:

Osteoid osteoma: Treatment is accomplished by resection of the nidus by open surgical approach or by percutaneous CT scan-guided resection. Recently, percutaneous radiofrequency ablation of the nidus has been performed with acceptable results.[15]

Osteoblastoma: Wide local resection is the treatment of choice whenever possible. This sometimes is limited by the proximity of vital vessels or neural tissue in the spine. A 10 to 20% recurrence rate exists for conventional osteoblastomas. Aggressive osteoblastomas have a recurrence rate of approximately 50% if wide margins are not attained. These tumors are not radiosensitive.

Osteosarcoma: Surgical resection is the rule; however, resection of spine lesions is often incomplete due to the size and location of the tumor at the time of presentation. Adjuvant chemotherapy and radiation therapy often are employed with varying degrees of utility. Spinal osteosarcomas have a dismal prognosis, with deaths usually occurring within the first year of diagnosis. Only a few patients have been reported to survive longer than 2 years.

Cartilage producing tumors of the spine:

Osteochondroma: Complete surgical resection is usually curative. Clinical symptoms improve in 89% of patients following removal of the exostosis. Incomplete resection can lead to recurrence of the lesion.

Chondrosarcoma: Surgical resection by vertebral corpectomy and strut bone grafting sometimes may be necessary for complete excision. Cure is possible when complete resection can be achieved; this is possible 25% of the time. If wide marginal resection cannot be achieved, the tumor recurrence results in death in 74% of cases. The mean survival for all patients with chondrosarcomas is 5.9 years according to Shives et al. [16] Adjunctive treatment with radiation is controversial for these tumors. Chemotherapy is used sometimes to help decrease the size of the mass with high-grade chondrosarcomas and dedifferentiated chondrosarcomas. Metastases of chondrosarcoma depend on the grade of the primary chondrosarcoma. The lungs are the most frequent sites of metastasis.

Lymphoproliferative tumors:

Multiple myeloma: Multiple myelomas are generally sensitive to radiation and chemotherapies. Surgery for stabilization is indicated in myelomas of the spine when destruction of the vertebral body exists to such an extent that collapse and possible

kyphosis with canal compromise could result. Prophylactic posterior stabilization can be carried out with segmental instrumentation in cases prior to fracture. Anterior strut grafting or cage reconstruction may be necessary once fracture and collapse have occurred. Adjuvant radiation therapy may be used postoperatively once healing of the surgical site has been obtained.

Plasmacytoma: The diagnosis is made by biopsy of the lesion, and treatment includes radiation and bracing except in persons with pathologic or impending pathologic fractures. In these individuals, surgical resection and stabilization should be carried out with postoperative adjuvant radiation therapy once 6 to 8 weeks of postsurgical healing has occurred. Patients have greater than 60% 5-year survival.

A recent review summarized current concepts in the management of primary bone tumors of the spine.

Tumor of notochordal origin:

Chordoma: Surgical resection is the rule. Adjuvant postoperative radiation therapy, proton beam therapy, and brachytherapy all have been used with varying results. The prognosis depends on whether the tumor can be resected completely. The location of the lesion and the size at presentation often necessitate incomplete resection. Persons with sacrococcygeal tumors often have improved survival because the surrounding structures are relatively more expendable and allow a more complete resection. Persons with sacrococcygeal lesions typically have 8 to10 years survival as opposed to 4 to 5 years survival for persons with chordomas in other spinal sites. Death usually is related to local recurrence and invasion rather than metastatic disease. Chordomas can metastasize. The most common sites of metastases are the liver, lungs, regional lymph nodes, peritoneum, skin, and heart.

Round cell tumor:

Ewing sarcoma: Before the advent of chemotherapy, the survival rate for patients with Ewing sarcoma was dismal because of the inability to completely resect these lesions, especially in the axial skeleton. Radiation and chemotherapy are the current mainstays of treatment of Ewing sarcoma in the spine, achieving almost 100% local control with an 86% long-term survival rate in patients with spinal Ewing nonsacral sarcomas. Sacral tumors have a 62% local control rate and only 25% long-term survival rate because of the tendency for delayed clinical presentation and larger tumor size. The most important prognostic indicator for survival of Ewing sarcoma is the tumor's response to chemotherapy.

Complications:

Complications associated with spine tumors can be divided into the following 2 groups:

Complications associated with the tumor, its recurrences, or its metastases:

- Neurologic complications include radicular pain or focal weakness from impingement on a nerve root and complete or incomplete paraplegia from direct pressure on the spinal cord.

Complications associated with the surgical, radiation, or chemotherapeutic treatment of the tumors:

- Complications that result from the treatment modality employed may be related to structures sacrificed during the surgical resection to obtain clear margins, structures in the path of radiation therapy, or the systemic effects of chemotherapy.

VI. Literature review on stereotactic radiosurgery for spinal tumors

This section reviews the literature on treatment of spinal lesions with high dose radiation delivered in 1 to 5 fractions. The role of stereotactic radiosurgery (SRS) for the treatment of intracranial lesions has been well established but its use for the treatment of spinal lesions has been more limited. Surgical extirpation remains the primary treatment option for most benign spinal tumors however SRS has demonstrated clinical benefits of treatment. The major potential benefits of SRS for spinal lesions are short treatment time in an outpatient setting, rapid recovery and symptomatic response. Indeed, this procedure offers a successful alternative therapeutic modality for the treatment of a variety of spinal lesions not amenable to open surgical techniques. In addition, SRS can be performed in medically untreatable patients, lesions located in previously irradiated sites, or as an adjunct to surgery. Local tumor control and long-term palliation for both primary and metastatic tumors may be achieved with SRS while reducing the risk of spinal cord toxicity associated with conventional radiotherapy techniques.

In 1995 Hamilton et al [17]reported on one of the earliest prototype devices that delivered SRS with a modified linear accelerator, to metastatic lesions of the spine in five patients. All patients had failed to previously respond to spinal cord tolerance doses delivered by standard external beam radiation therapy (EBRT) to a median dose of 45 Gy (range, 33 to 65 Gy/11 to 30 fractions). The tumors were then treated with single-fraction SRS with a median number of isocenters of one (range, one to five) and a median single fraction dose of 10 Gy (range, 8 to 10 Gy). There was a single complication of esophagitis of a tumor involving the C6-T1 segments of the spine; which resolved with medical therapy. Median follow-up time on average was 6 months (range, 1 to 12) with no radiographic or clinical progression of the treated tumor in any patient. The authors concluded that extracranial SRS may be suitable for the treatment of paraspinal neoplasms after EBRT, even in the face of spinal cord compression.

A few years later in 2000 Murphy et al[18] reported on the use of frameless SRS to treat an arterovenous malformation (AVM) in the cervical spine, a recurrent schwannoma of the thoracic spine, and a metastatic adenocarcinoma of the lumbar spine. This was one of the first experiences to demonstrate the feasibility of using frameless SRS outside the cranium. In 2001 the same group reported on the technical feasibility of frameless SRS

for treatment of unresectable spinal vascular malformations and primary and metastatic spinal tumors.[19] In this study, 16 patients with spinal lesions including hemangioblastomas, vascular malformations, metastatic carcinomas, schwannomas, a meningioma, and a chordoma were treated. The total treatment dose of 11 to 25 Gy was given in 1 to 5 fractions. At 6 months follow-up, patients demonstrated no progression of disease and no patients experienced complications as a result of SRS. The authors concluded that frameless SRS for previously untreatable spinal lesions was both safe and feasible.

In 2002 Gerszten et al[20] first reported one of many studies on single fraction frameless SRS for treatment of spinal lesions . Fifty-six spinal lesions in 46 consecutive patients were treated using single-fraction SRS to 11 benign and 45 metastatic lesions. Tumor volume ranged from 0.3 to 168 ml (mean 26.7 ml). Thirty-one lesions had previously received EBRT with maximum spinal cord doses. Dose plans were calculated based on CT scans and tumor dose was maintained at 12 to 18 Gy to the 80% isodose line. Spinal cord lesions receiving greater than 8 Gy ranged from 0 to 1.3 ml (mean 0.3 ml). All patients tolerated the procedure in an outpatient setting and there were no acute radiation-induced toxicity or new neurological deficits occurred during the follow-up period. In addition, axial and radicular pain improved in all patients who were symptomatic prior to treatment.

In another study by this same group, 125 spinal lesions in 115 consecutive patients were treated with a single-fraction SRS technique[21]. Tumor volume ranged from 0.3 to 232 ml (mean 27.8 ml) and dose was maintained at 12 to 20 Gy to the 80% isodose line (mean 14 Gy). The spinal cord lesions receiving greater than 8 Gy ranged from 0.0 to 1.7 ml (mean 0.2 ml). No acute radiation toxicity or new neurological deficits occurred during the follow-up period of 3 to 24 months. The same group then evaluated single- fraction frameless SRS for the treatment of sacral spinal lesion[22]. Eighteen patients were treated with single-fraction SRS. Tumor dose was maintained at 12 to 20 Gy to the 80% isodose line (mean 15 Gy). Tumor volume ranged from 23.6 to 187.4 ml (mean 90 ml). The volume of the cauda equina receiving greater than 8 Gy ranged from 0 to 1 ml (mean 0.1 ml). There were no acute radiation toxicities or new neurological deficits during the mean follow-up period of 6 months. Pain improved in all 13 patients who were symptomatic prior to treatment. No tumor progression was found on follow-up imaging. SRS was found to be feasible, safe, and effective for the treatment of both benign and malignant sacral lesions.

In 2007 this same group reported on a prospective nonrandomized, longitudinal study evaluating the clinical outcomes of single-fraction SRS as part of the management of metastatic spine tumors[23]. A cohort of 500 cases of spinal metastases underwent radiosurgery. Ages ranged from 18 to 85 years (mean 56). The maximum radiation dose ranged from 12.5 to 25 Gy (mean 20). Tumor volume ranged from 0.20 to 264 mL (mean 46). Long-term pain improvement occurred in 290 of 336 cases (86%). Long-term tumor control was demonstrated in 90% of lesions treated with SRS as a primary treatment modality and in 88% of lesions treated for radiographic tumor progression. Twenty-seven of 32 cases (84%) with a progressive neurologic deficit before treatment experienced at

least some clinical improvement. These results indicated the potential of SRS to treat patients with spinal metastases, especially those with solitary sites of spine involvement, to improve long-term palliation.

In 2004 Benzil et al[24] reported their initial experience with SRS of the spine with attention to dose, efficacy, and toxicity. Patients treated between December 2001 and January 2004 included a total of 31 patients (12 men, 19 women; mean age 61 years, median age 63 years) with 35 tumors which included twenty six metastases and nine primary tumors. Rapid and significant pain relief was achieved after SRS in 32 of 34 treated tumors. In patients treated for metastases, pain was relieved within 72 hours and remained reduced at 3 months. Pain relief was achieved with a single dose as low as 50 Gy. Two patients experienced transient radiculitis (both with a biological equivalent dose (BED) > 60 Gy). No patient experienced other organ toxicity. The authors concluded that SRS of the spine is safe at the doses used and provided effective pain relief. In this study, BEDs greater than 60 Gy were associated with an increased risk of radiculitis.

Also in 2004 Blisky et al reported[25] on the initial clinical experience in treating 16 paraspinal tumors with SRS using a linear accelerator mounted with a multileaf collimator. During a 30-month period, 16 patients underwent SRS for metastatic and primary spine tumors. Eleven patients were treated for symptomatic recurrences after undergoing surgery and prior EBRT. Patients with metastatic tumors were administered a median dose of 20 Gy in four to five fractions and a maximum spinal cord dose of 6 Gy in addition to the full tolerance dose administered in previous radiation treatments. Of the 15 patients who underwent radiographic follow-up, 13 demonstrated either no interval growth or a reduction in tumor size in a median follow-up period of 12 months (range, 2 to 23 months). Two patients showed tumor progression 1 year after undergoing SRS. Pain symptoms improved in 11 of 11 patients, and 4 of 4 patients had significant improvement in their functionally significant radiculopathy and/or plexopathy. No patient showed signs or symptoms of radiation-induced myelopathy, radiculopathy, or plexopathy with a median follow-up of 18 months.

In 2005 Bhatnagar et al[26] evaluated the feasibility, toxicity, and local control of patients treated with the frameless SRS for benign extracranial lesions. A total of 59 benign tumors in 44 patients were treated. All patients were treated in a single fraction except for 3 lesions which were treated in a fractionated manner. Twenty three of the lesions underwent initial surgical resection. Ten lesions received prior EBRT with a median dose 48 Gy (range 40 to 54 Gy), and 1 lesion received two prior SRS treatments for a total dose of 32 Gy to the 80% isodose line. The median follow-up was 8 months (range 1 to 25 months). Acute and late toxicity was graded using the National Cancer Institute Common Toxicity Criteria (CTC) scale. The median tumor dose delivered was 16.5 Gy to the 80% isodose line (range 10 to 31 Gy). The median tumor volume was 4.3 cc (range 0.14 to 98.6 cc). The median spinal cord volume receiving more than 8 Gy was 0.035 cc (range 0 to 2.5 cc) and the median maximum spinal cord dose 11.5 Gy (range 0 to 19.8 Gy). There were no patients that suffered a significant acute toxicity and there was no observed late toxicity. Seventy-eight percent of patients experienced an improvement of their pre-treatment symptoms while only 1 patient experienced symptom progression. Of

the 26 patients who underwent follow-up imaging, the local control rate was 96%. This study suggests that SRS is a safe and efficacious treatment modality for benign tumors, even for those patients with recurrent previously irradiated or SRS treated lesions.
Also in 2005 Degen et al[27] reported on the results of a study to assess safety, pain, and quality of life outcomes following SRS for treatment of spinal tumors. Fifty-one patients with 72 lesions (58 metastatic and 14 primary) were treated. The mean follow-up period was 1 year. Pain was improved, with the mean VAS score decreasing significantly from 51.5 to 21.3 at 4 weeks ($p < 0.001$). This effect on pain was durable, with a mean score of 17.5 at 1 year, which was still significantly decreased ($p = 0.002$). Quality of life was maintained throughout the study period and after 18 months, physical well-being was 33 (initial score 32; $p = 0.96$) and mental well-being was 43.8 (initial score 44.2; $p = 0.97$). Adverse effects included self-limited dysphagia (three cases), diarrhea (two cases), lethargy (three cases), paresthesias (one case), and wound dehiscence (one case). The authors concluded that SRS both improved pain control and maintained QOL in patients with spinal tumors and adverse events were infrequent and minor.

In 2005, Yamada et al[28] reported on the use of SRS to treat lesions within close proximity to the spinal cord. Patients were either previously irradiated or prescribed doses beyond conventional spinal cord tolerance (54 Gy in standard fractionation) and had unresectable gross disease involving the spinal canal. Patients were followed up with serial MR imaging every 3 to 4 months, and no patients were lost to follow-up. A total of thirty five patients (14 primary and 21 secondary malignancies) underwent treatment with a median follow-up of 11 month. The median dose previously received was 30 Gy in 10 fractions. The median dose prescribed for these patients was 20 Gy in five fractions (20 to 30 Gy). In previously unirradiated patients, the median prescribed dose was 70 Gy (59 to 70 Gy). More than 90% of patients experienced palliation from pain, weakness, or paresthesia; 75% and 81% of secondary and primary lesions, respectively, exhibited local control at the time of last follow-up. No cases of radiation-induced myelopathy or radiculopathy were encountered.

In 2006 Dodd et al[29] published a prospective study on preliminary experience using SRS for treatment of selected benign spinal neoplasms. SRS was used to manage 51 patients (median age, 46 yr; range, 12 to 86 yrs) with 55 benign spinal tumors (30 schwannomas, 9 neurofibromas and 16 meningiomas). Total treatment doses ranged from 16 to 30 Gy delivered in one to five fractions. Tumor volumes varied from 0.136 to 24.6 cm. Less than 1 year post SRS, 3 of the 51 patients in this series required surgical resection of their tumor because of persistent or worsening symptoms. After a mean follow-up of 36 months, all of the remaining patients were either stable (61%) or smaller radiographically (39%). Two patients died from unrelated causes. Radiation-induced myelopathy appeared 8 months post SRS in one patient.

In 2006 Rock et al[30] reported on outcomes in 18 postoperative patients who received SRS treatment of their residual spinal tumors. Of the patients, ten had metastatic carcinomas, three had sarcomas, four had multiple myeloma/plasmacytomas and one was a giant cell tumor. SRS doses ranged from 6 to 16 Gy (mean, 11.4 Gy) prescribed to the 90% isodose line. The volume of irradiated spinal elements receiving 30, 50, and 80% of the total

dose ranged from 0.51 to 11.05, 0.19 to 6.34, and 0.06 to 1.73 cm, respectively. Follow-up ranged from 4 to 36 months (median, 7 months). Only 1 patient in this series developed progressive symptoms possibly attributable to a toxic effect of SRS. Of those patients initially presenting with neurological deficits, 92% either remained neurologically stable or improved. The authors suggest that SRS as prescribed in this series of postoperative patients with residual spinal tumor is well-tolerated and associated with little to no significant morbidity.

In 2006 Muacevic et al[31] describe the initial clinical feasibility of using a fiducial-free alignment system with frameless SRS which eliminates the need for implantation of either radiographic markers or fiducials prior to any spinal treatment. The authors performed 10 tests of accuracy using a spine phantom and then treatment of 50 spinal lesions in 42 patients. The procedure was successful in all patients with treatment doses between 2 to 25 Gy to the median prescription isodose of 65% (40 to 70%), and tumor volumes ranged between 1.3 and 152.8 cm3. The mean spinal cord volume receiving greater than 8 Gy was 0.69 ± 0.35 cm3. Phantom tests produced an overall mean targeting error of 0.52 ± 0.22 mm. No short-term adverse events were noted during the 1 to 7 month follow-up period and axial or radicular pain was relieved in 14 out of 15 patients treated for pain. The authors concluded that fiducial-free tracking is a feasible, accurate, and reliable tool for SRS treatment of the entire spine.

The same authors also presented preliminary clinical results and phantom accuracy measurements for fiducial-free frameless sacral SRS[32]. Fifty-one lesions in 38 patients were treated using fiducial-free spinal tracking of the L5 vertebra. The tracked targets were up to 17 cm away from the treated tumor. Phantom tests produced an overall mean targeting error of 1.43 mm (± 0.47mm). The treatment doses were 12 to 25 Gy with a median prescription isodose of 65% (40 to 70%) and tumor volumes were between 1.3 and 152.8 cc. Median patient follow-up was 12.7 months and local tumor control was 95%. No short-term adverse events were noted during the follow-up period. Thus, the authors concluded that fiducial-free tracking of the lower lumbar vertebrae is a feasible, accurate, and reliable tool for SRS of sacral and pelvic tumors.

This same group evaluated both the targeting precision and patient movement during fiducial-free, single-fraction frameless SRS in 2010.[33] Accuracy was tested using two phantoms and the position was registered with an accuracy of <0.2 mm for translational and <0.3 degrees for rotational directions. The region of the spine did not influence the overall targeting error, which was <1 mm for more than 95% of treatments (median, 0.48 mm). In the worst case scenario with maximum motion, target coverage decreased by 1.7% (from 92.1% to 90.4%) for the 20 Gy prescription isodose and spinal cord volume receiving more than 8 Gy increased slightly, from 2.41 to 2.46 cm. The authors noted submillimeter targeting precision was obtained for fiducial-free spinal SRS despite patient motion.

In 2008, this same group assessed the ideal patients for fiducial-free frameless spinal SRS.[34] A consecutive series of 102 patients with a total of 134 malignant spinal tumors

were selected for single-fraction, fiducial-free frameless SRS with a maximum of 2 tumors. Patients with spinal cord compression or evidence of myelopathy were excluded. The endpoints studied were sequential neurologic status and tumor-associated spinal pain recorded with the visual analogue scale (VAS). Of 102 individuals, 22 (21.6%) died due to progression of systemic disease. Mean survival after treatment was 1.4 years. Median survival after initial tumor diagnosis was 18.4 years. Two (2%) patients suffered complications after SRS; a tumor hemorrhage occurred in one patient, and another developed spinal instability which were stabilized by kyphoplasty. Neurotoxicity or myelopathy was not observed. Local tumor control 15 months after SRS was 98%. Tumor-associated pain was observed in 52 (51%) patients. In these patients the median pre-treatment pain score of VAS = 7 was significantly reduced to VAS = 1 ($P < 0.001$) within 1 week after SRS. Analysis of variance identified that the initial pain score as the only significant variable to predict pain reduction after SRS ($P < 0.03$). Pain recurrence in correlation with tumor recurrence was observed for 3 (6%) patients. The authors concluded that the best benefit of the treatment can be expected in patients with good to excellent clinical condition and patients with severe tumor associated pain.

In 2007 Chang et al[35] reported on the safety, effectiveness, and patterns of failure in a Phase I/II study of SRS for spinal metastatic tumors. Sixty-three patients underwent SRS with National Cancer Institute Common Toxicity Criteria 2.0 assesments used to evaluate toxicity. The median tumor volume of 74 spinal metastatic lesions was 37.4 cm3 (range 1.6 to 358 cm3). No radiation induced neuropathy or myelopathy was observed during a median follow-up period of 21.3 months (range 0.9 to 49.6 months). The actuarial 1-year tumor progression-free incidence was 84% for all tumors. Pattern-of-failure analysis showed that the two primary mechanisms of failure were recurrence in the bone adjacent to the site of previous treatment, and recurrence in the epidural space adjacent to the spinal cord. The authors thus concluded that it may be wise to routinely treat a wide margin in spine patients including the pedicles and posterior elements of the spine while using a wide bone margin posterior to the diseased vertebrae because of the possible direct extension into these structures as patterns of tumor recurrence.

In 2007 Sahgal et al[36] reported on a retrospective review of 16 consecutively treated patients with 19 benign spinal tumors (11 neurofibromas, 4 chordomas, 2 hemangiomas, and 2 meningiomas). Three patients had Neurofibromatosis Type 1 (NF1). Only one tumor had been previously irradiated. The median total dose, number of fractions, and prescription isodose was 21 Gy (10 to 30 Gy), 3 fractions, 80% isodose (42 to 87%). The median tumor volume was 7.6 cc (0.2 to 274.1 cc). Three tumors progressed at 2, 4, and 36 months post-SRS (n=18), two of these tumors were neurofibromas (both in NF1 patients), and the third was an intramedullary hemangioblastoma. Local failure, for the remaining 18 tumors, was based clinically on symptom progression and/or tumor enlargement based on imaging. Median tumor follow-up at 25 months (range 2 to 37) based on imaging showed 2 tumors with MRI documented progression, 3 tumors with regression and 13 tumors were unchanged (n=18).

In 2008 Gerszten et al[37] published a study evaluating the clinical efficacy of SRS treatment for benign tumors of the spine. Seventy-three benign intradural extramedullary

spinal tumors were treated with SRS and prospectively evaluated. Tumor type included 25 neurofibromas, 35 schwannomas, and 13 meningiomas. Twenty-one cases were associated with neurofibromatosis Type 1 (NF1), and 9 patients had neurofibromatosis Type 2 (NF2). Nineteen tumors (26%) had previously undergone open surgical resection, and six tumors (8%) had previously been treated with EBRT. Patient ages ranged from 18 to 85 years (mean age, 44 yr); the follow-up period was 8 to 71 months (median, 37 months). Similar radiation doses were prescribed for all 3 tumor type histopathologies. The maximum intratumoral dose was 15 to 25 Gy. Tumor volume ranged from 0.3 to 93.4 cm (mean, 10.5 cm; median, 4.11 cm). SRS was used for the treatment of postsurgical radiographic progression in 18 cases and used as primary treatment in 14 cases; and it was used for the treatment of postsurgical residual tumor in 2 cases. Long-term pain improvement occurred in 22 out of 30 cases (73%). Long-term radiographic tumor control was demonstrated in all cases however three patients experienced new symptoms attributed to radiation-induced spinal cord toxicity 5 to 13 months after treatment. The authors concluded that single fraction SRS was a clinically effective treatment modality for benign extramedullary spinal neoplasms.

In 2008 Yamada et al[38] reported on tumor control and toxicity for patients treated with SRS for spinal metastases. A total of 103 consecutive spinal metastases in 93 patients without spinal cord compression were treated with SRS to doses of 18 to 24 Gy (median, 24 Gy) in a single fraction. The maximal spinal cord dose was limited to 14 Gy. The patients were prospectively examined every 3 to 4 months with clinical assessment and cross-sectional imaging, for median follow-up of 15 months (range, 2 to 45 months). The overall actuarial local control rate was 90% and local failure developed in 7 patients. The median time to local failure was 9 months (range, 2 to 15 months). The median overall survival was 15 months and 37 of 93 patients died. In all cases, death was from progression of systemic disease and not local failure. The histologic type of tumor was not a statistically significant predictor of survival or of local control however the radiation dose was a significant predictor of local control ($p = 0.03$). Acute toxicity was mild with no cases of post radiation radiculopathy or myelopathy. The authors concluded that SRS is a safe and effective palliation for patients with spinal metastases which had minimal negative effects on quality of life and a high probability of tumor control.

In 2009 Nelson et al[39] reported on 32 patients with 33 spinal lesions who underwent SRS in 3 SRS fractions (range, 1 to 4) of 5 to 16 Gy. Median cord and target BED were 70 Gy and 34.3 Gy, respectively. Mean previous radiotherapy dose in 22 patients was 35 Gy, and the median interval was 17 months. Twenty-one patients were alive at 1 year (median survival, 14 months) with a median follow-up was 6 months for all patients (7 months for survivors). Thirteen patients reported complete and 17 patients reported partial pain relief at 1 month. There were 4 failures at a mean of 5.8 months where MR imaging showed evidence of in-field progression however there were no dosimetric parameters that were predictive of failure and there was no treatment-related observed toxicity. The authors concluded that spinal SRS is effective in the palliative/re-treatment setting.

Also in 2009 Gibbs et al[40] reported on the incidence of spinal cord injury after SRS. A total of 1075 patients with benign or malignant spinal tumors were treated by SRS at two centers. Patients were followed prospectively with clinical and radiographic assessments at 1 month intervals. A retrospective review identified patients who developed delayed radiation-induced myelopathy. Six patients (5 women, 1 man) with a mean age of 48 years (range, 25 to 61 years) developed delayed myelopathy at a mean time of 6.3 months (range, 2 to 9 months) where 3 cases involved previous radiation therapy. Dose volume histograms and clinical and dosimetric factors were analyzed for factors predictive of spinal cord injury but specific factors could not be identified, although half of the patients with myelopathy received spinal cord BED exceeding 8 Gy. The authors concluded that delayed myelopathy after SRS is uncommon and radiation injury to the spinal cord occurred over a spectrum of dose parameters. The authors concluded by recommending a limit to the volume of spinal cord treated above an 8 Gy equivalent dose.

Kim et al reported on the safe and efficacious use of helical tomotherapy for fractionated spine SBRT.[41] Another recent Phase 1/2 trial from MD Anderson Cancer Center used single fraction 10-24 Gy for primary treatment of spine metastatsis and showed excellent imaging local control rate of 88%.[42]

In the setting of prior radiation therapy, SBRT has been proven to be an effective and safe retreatment modality.[43] In a retrospective single institution study, Mahadevan et al[44] utilized a flexible hypofractionated regime based on the extent of disease for re-irradiation of recurrent spine metastasis.

The American College of Radiology, Canadian Association of Radiation Oncology and other expert groups have endorsed the role and drafted guidelines for appropriateness and safe practice of spine SBRT.[45] [46] [47]As clinical experience and new data are emerging, important safety parameters are being established in relation to radiation myelopathy and vertebral compression fractures. Probabilities of radiation myelopathy based on dose volume parameters serve as helpful guidelines for safe practice.[48] Unintended vertebral compression fractures after SBRT are increasingly being recognized.[49]

This literature review for the treatment of both benign and metastatic spinal lesions clearly shows the feasibility, safety and efficacy of SRS. The radiosurgical dose tolerance for organs at risk and the spinal cord must always be taken into consideration. SRS for spinal lesions is emerging as a new therapeutic standard of care to help maintain or improve patient's quality of life.

VII. Clinical indications and treatment guidelines on stereotactic radiosurgery for spinal tumors

This section is accessible only to society members – for more information about the Radiosurgery Society, go to www.therss.org

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