3601_e14_p341-361 2/19/02 8:54 AM Page 341

14

Spinal Axis Metastases

RICHARD G. PERRIN, NORMAND J. LAPERRIERE, D. ANDREW LOBLAW,
AND ADRIAN W. LAXTON

Secondary spinal tumors represent an ominous extension of systemic cancer and commonly present as a neurologic emergency. Advances in diagnostic imaging techniques, clarification of the relative benefits provided by radiation therapy and surgery, and refinement in surgical approaches and techniques have all contributed to improve the outlook for patients with spinal metastases (Maranzano et al., 1991; Perrin and McBroom, 1987; Rosenthal et al., 1996; Siegal and Siegal, 1985; Sundaresan et al., 1990, 1991).

CLASSIFICATION AND PATHOLOGY

Spinal tumors are classified by anatomic location (Table 14–1). Extradural metastases account for approximately 95% of secondary spinal tumors. These lesions arise through blood-borne spread of cancerous cells or by direct extension of the primary tumor. Most extradural tumors are metastatic to the vertebral bodies, but some lymphomas and tumors from Hodgkin's disease may occur in the epidural space without bone involvement. Metastatic spinal tumors seldom breach the dura. Intradural extramedullary metastases are uncommon and represent tertiary spread from cerebral secondary sites (Perrin et al., 1982). Intradural extramedullary metastases are transmitted through the cerebrospinal fluid and typically become entangled among the nerve roots of the cauda equina. Intramedullary tumors are rare, comprising approximately 3.5% of spinal metastases (Bruner and Tien, 1998). Intramedullary metastases arise through hematogenous spread.

Spinal tumors are also classified according to the level of the spine involved (cervical, thoracic, lumbosacral). Autopsy studies have shown that the distribution of spinal metastases parallels the bulk of the vertebrae; thus, the lumbosacral spine is most often afflicted, followed by the thoracic and cervical segments (Willis, 1973). Clinically, however, *symptomatic* spinal metastases most often involve the thoracic spine (49%) followed by the lumbosacral (40%) and cervical (11%) segments (Table 14–2).

Spinal tumors most often originate from primary tumors of breast, lung, and prostate, which reflects both the prevalence of these cancers and their propensity to metastasize to bone (Table 14–3). Primary tumors less commonly reported to metastasize to the spine include leukemia, schwannoma, mesothelioma, Merkel's tumor, plasmacytoma, teratoma, as well as basal cell, parotid, nasopharyngeal, laryngeal, esophageal, gall bladder, pancreas, ovarian, endometrial, and urinary bladder tumors (Helweg-Larsen, 1996; Kovner et al., 1999).

As many as 10% of patients with symptomatic spinal metastases present with no known primary lesion (Botterell and Fitzgerald, 1959; Livingston and Perrin, 1978; MacDonald, 1990).

INCIDENCE

Most patients with systemic cancer develop skeletal metastases, and the spine is most commonly involved (Willis, 1973). As many as 30% of all cancer patients develop secondary spinal tumors (Bach et al., 1990;

Table 14–1. Relative Frequencies of Spinal Metastases According to Location of Spinal Involvement

		Extradural		*Intradural Extramedullary*		*Intramedullary*	
Author	*Total*	*No.*	*%*	*No.*	*%*	*No.*	*%*
Rogers and Heard (1958)	17	16	94	1	6	—	—
Barron et al. (1959)	125	123	98	—	—	2	1.6
Edelson et al. (1972)	175	169	97	—	—	6	3.4
Perrin et al. (1982)	200	189	94	10	5	1	0.5

Gomez, 1955). Approximately 18,000 new cases of spinal metastases are diagnosed in North America each year (Gokaslan et al., 1998). Spinal metastases occur 20 times more commonly than primary tumors of the spine.

SYMPTOMS AND SIGNS

Symptomatic spinal metastases produce a characteristic clinical syndrome. Typically, local back or neck pain is followed by weakness, sensory loss, and sphincter dysfunction (Table 14–4) (Botterell and Fitzgerald, 1959; Helweg-Larsen and Sorensen, 1994; Livingston and Perrin, 1978; MacDonald, 1990).

Local back or neck pain is the earliest and most prominent feature in 90% of patients. Palpation or percussion over the posterior spine at an involved level usually elicits local tenderness. Associated radicular pain distribution indicates segmental root irritation and is an especially common symptom among patients with lumbar spine metastases (Helweg-Larsen and Sorensen, 1994). When local back or neck pain is aggravated by movement and relieved by immobility, spinal instability should be suspected (Perrin and Livingston, 1980). If the pain has a severe, burning, dysesthetic quality, intradural extramedullary metastases should be considered (Perrin et al., 1982). Pain caused by spinal metastases may be present for up to 1 year and is often initially attributed to arthritis, back strain, or a slipped disc (Goodkin et al., 1987). Correct diagnosis of spinal metastatic pain is often delayed until more blatant manifestations of spinal cord compromise are manifest. It is axiomatic that *new-onset back or neck pain in a cancer patient means spinal metastasis until proven otherwise.*

Spinal metastases may be the first manifestation of malignancy in 20% or more of patients (Schiff et al., 1997). By the time treatment is initiated, however, only about 2% of spinal metastases are of unknown origin.

Motor weakness is usually manifest after the onset of pain and is especially common in patients with tho-

Table 14–2. Relative Frequencies of Spinal Metastases According to Level of Spinal Involvement

		Cervical		*Thoracic*		*Lumbrosacral*	
Author	*Total*	*No.*	*%*	*No.*	*%*	*No.*	*%*
Sorensen et al. (1994)*	57	3	5	33	58	21	37
Helweg-Larsen (1996)	153	7	4.6	102	66.7	44	28.7
Tatsui et al. (1996)	695	106	15.3	203	29.2	386	55.5
Maranzano et al. (1997)	49	2	4	25	51	22	45
Schiff et al. (1998)	337	33	10	206	61	98	29
Brown et al. (1999)	40	5	12.5	13	32.5	22	55
Khaw et al. (1999)*	160	11	7	123	77	26	16
Kovner et al. (1999)	85	7	8	45	53	33	39
Rompe et al. (1999)*	106	9	8	76	72	21	20
Totals	1682	183	11	826	49	673	40

*In these studies, totals in the lumbosacral column refer to lumbar involvement only.

Table 14–3. Relative Frequencies of Various Primary Tumors Metastasizing to the Spine

Author	*Breast*	*Lung*	*Prostate*	*Kidney*	*Myeloma*	*Lymphoma*	*Colorectal*	*Cervix*	*Stomach*	*Sarcoma*	*Liver*	*Thyroid*	*Melanoma*	*Other**	*Unknown*	*Total*
Sorensen et al. (1994)	34	3	5	1	—	—	6	—	—	4	—	1	2	1	—	57
Akeyson and McCutcheon (1996)	4	8	—	5	—	—	—	1	—	3	—	—	—	3	1	25
Helweg-Larsen (1996)	56	27	43	—	—	—	—	—	—	—	—	—	—	27	—	163
Tatsui et al. (1996)	114	149	59	29	—	—	—	46	28	—	—	—	—	—	—	425
Maranzano et al. (1997)	3	12	11	5	4	—	9	—	—	—	—	—	—	5	—	49
Schiff et al. (1997)	64	58	81	15	23	25	13	—	1	15	3	6	5	—	15	324
Katagiri et al. (1998)	15	19	11	3	10	7	4	—	8	3	9	1	—	5	—	95
Khaw et al. (1999)	18	18	25	6	1	2	7	—	—	—	—	—	1	—	1	70
Kim et al. (1999)	3	18	1	—	17	5	1	—	4	1	2	2	1	3	2	60
Kovner et al. (1999)	28	9	12	—	—	9	2	—	—	—	—	—	—	17	2	79
Rompe et al. (1999)	31	20	6	16	8	—	—	—	—	—	—	5	—	16	4	184
Solberg and Bremnes (1999)	9	11	30	6	5	—	3	—	—	—	—	—	—	16	6	86
Van der Sande et al. (1999)	56	5	9	—	6	8	—	—	—	5	—	—	—	14	—	103
Wise et al. (1999)	18	11	6	6	13	9	—	—	—	8	—	—	—	4	5	80
Chen et al. (2000)	3	12	—	1	—	—	10	1	1	1	9	7	1	1	1	48
Totals	456	380	299	93	87	65	55	48	42	40	23	22	10	112	37	1846
%	26	22	17	5.2	5	3.7	3	2.7	2.4	2.3	1.3	1.2	0.6	6.3	2	100

*Includes leukemia, schwannoma, mesothelioma, Merkel's tumor, plasmacytoma, teratoma, as well as basal cell, parotid, nasopharyngeal, laryngeal, esophageal, gall bladder, pancreas, ovarian, endometrial, and urinary bladder tumors.

Table 14–4. Clinical Presentation of Spinal Metastases

Local back or neck pain (±radiculopathy)
Weakness
Sensory loss (including paresthesia)
Sphincter dysfunction

racic metastases (Helweg-Larsen and Sorensen, 1994). A Brown-Séquard syndrome may occur and is more common among patients with intramedullary rather than epidural metastases (Schiff and O'Neill, 1996).

The rate at which spinal cord compression develops varies. However, once established, weakness, sensory loss, and sphincter dysfunction will progress to complete and irreversible paraplegia unless timely treatment is undertaken (Botterell and Fitzgerald, 1959).

RADIOLOGIC INVESTIGATIONS

Radiologic investigations are conducted to determine the location and extent of spinal metastases. Such data form the basis for management strategies.

Plain Films

Plain radiographs of the spine demonstrate abnormalities in 90% of patients with symptomatic spinal metastases (Helweg-Larsen et al., 1997). Osteoblastic or osteosclerotic alteration may occur, especially with metastases originating from carcinoma of the prostate (Fig. 14–1). The majority of features on plain film, however, predominantly reflect osteolytic changes. Common findings on plain film include pedicle erosion ("winking owl" sign), paraspinal soft tissue shadow, compression fracture (vertebral collapse), and pathologic fracture dislocation (Fig. 14–2).

Myelography

Before the development of magnetic resonance imaging (MRI), lumbar myelography was the "gold standard" for determining the level of spinal cord compression by demonstrating a block to the flow of contrast. In addition, characteristics of the myelographic block ("paint brush," meniscus, or "fat cord") provides information concerning the anatomic location of the spinal lesion (extradural, intradural, extramedullary, or intramedullary) (Fig. 14–3). When the level of a complete lumbar myelographic block does not correspond to the clinical localization of the tumor or when multiple levels of involvement are suspected, a combination of lumbar and high cervical myelography may be used to delineate the extent of disease.

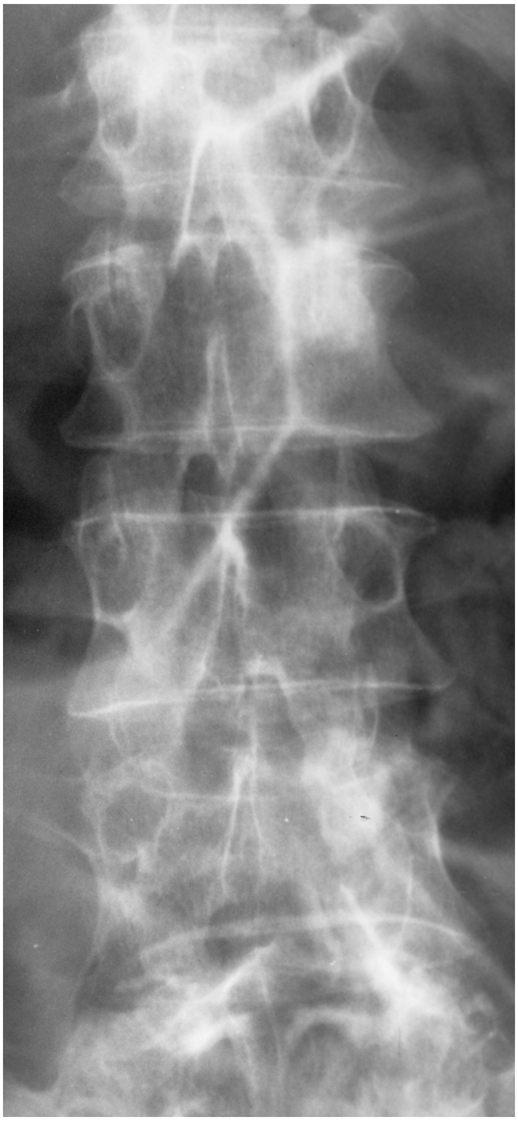

Figure 14–1. Common bone X-ray of osteosclerotic metastasis (pedicles) from prostatic cancer.

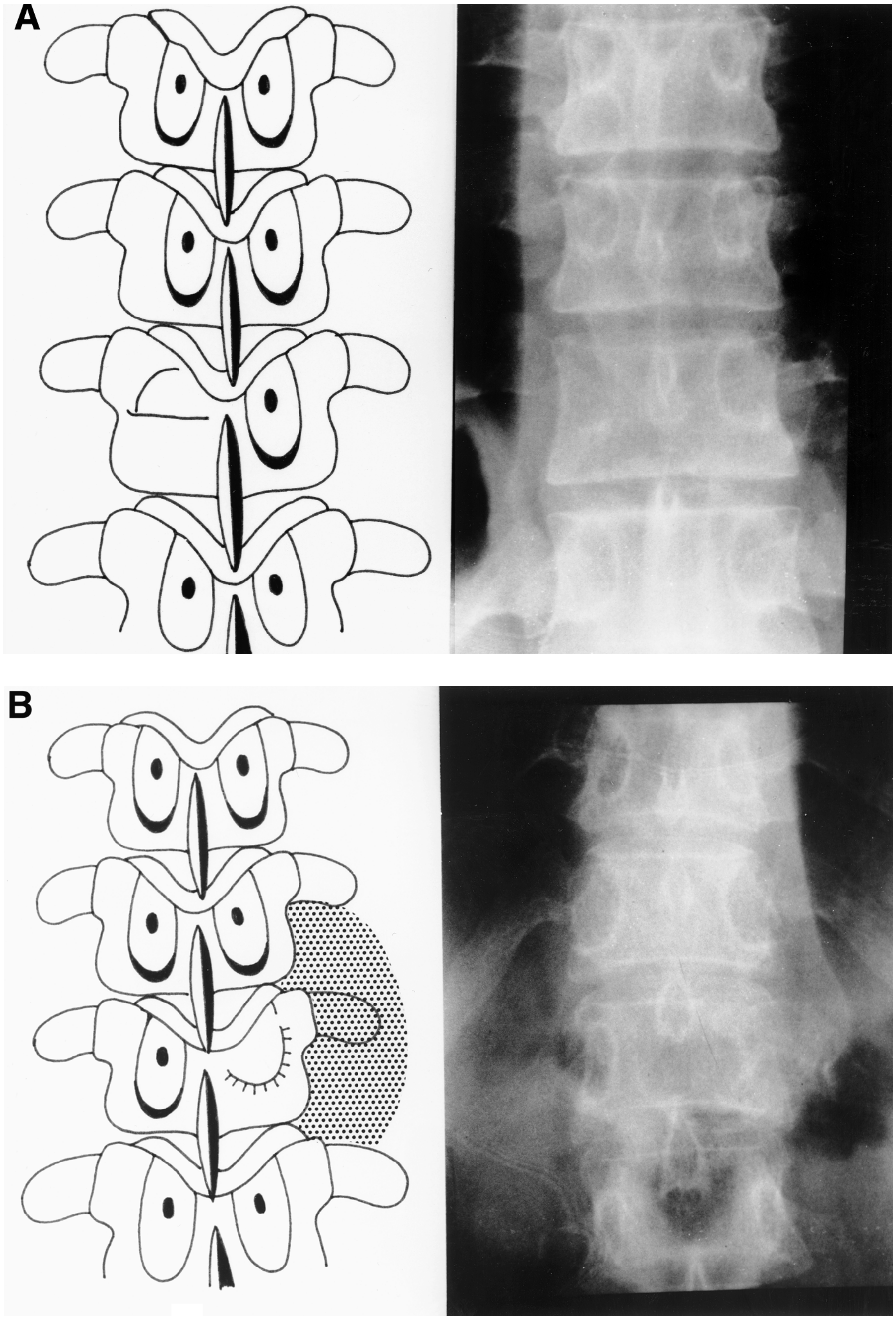

Figure 14–2. Common bone X-rays of **(A)** pedicle erosion producing "winking owl" sign; **(B)** paraspinal soft tissue shadow (with "winking owl"); **(C)** compression fracture (vertebral collapse); and **(D)** pathologic fracture dislocation. (*continued*)

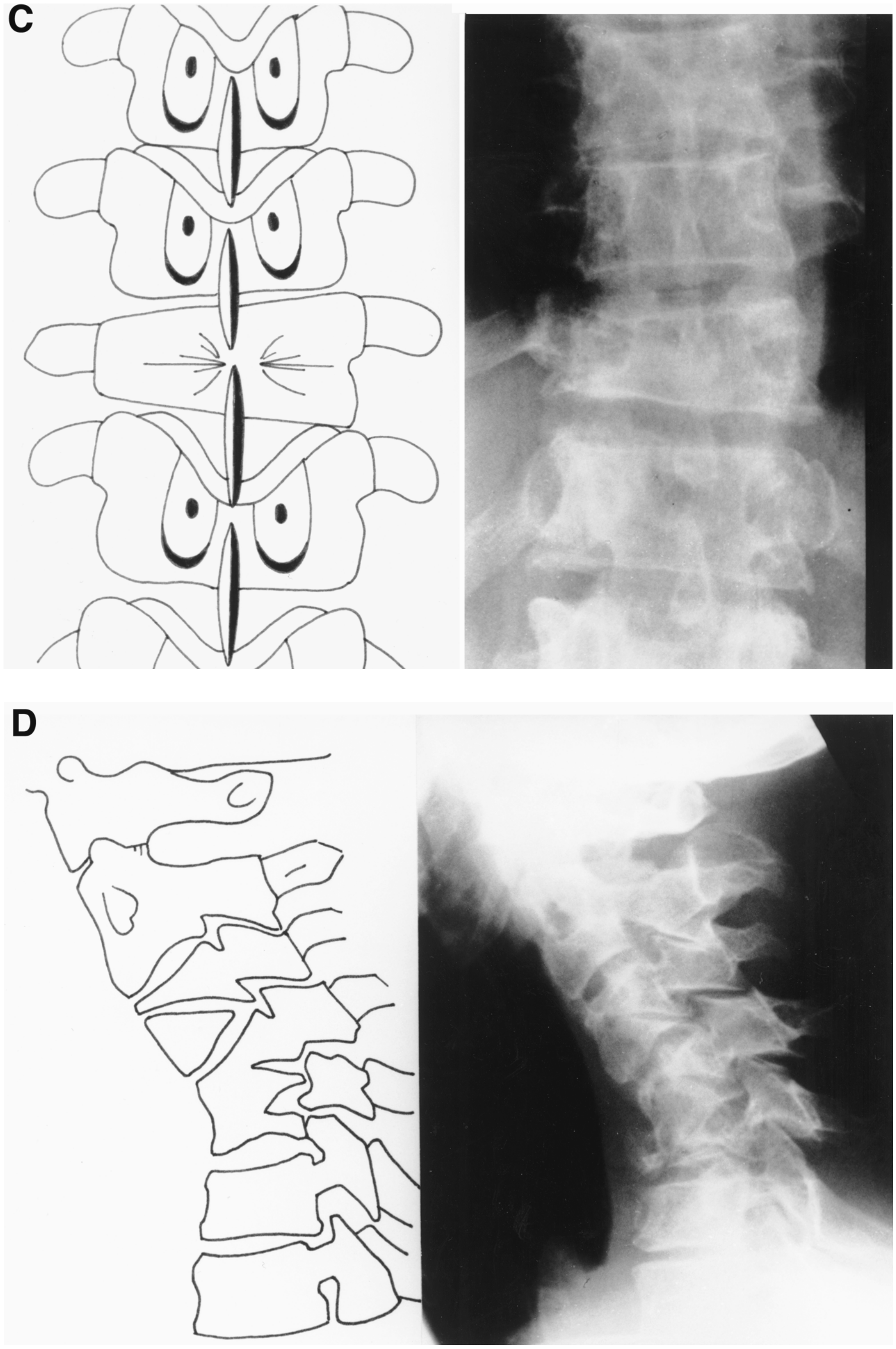

Figure 14–2. (*Continued*)

Computed Tomography

Computed tomography (CT) is useful for showing the disposition of spinal tumors by demonstrating vertebral destruction and paraspinal extension in transverse sections (Helweg-Larsen et al., 1997) (Fig. 14–4A). Computed tomography scans performed in conjunction with and following myelography are particularly valuable for displaying the degree of displacement of the dural sac and its contents (O'Rourke

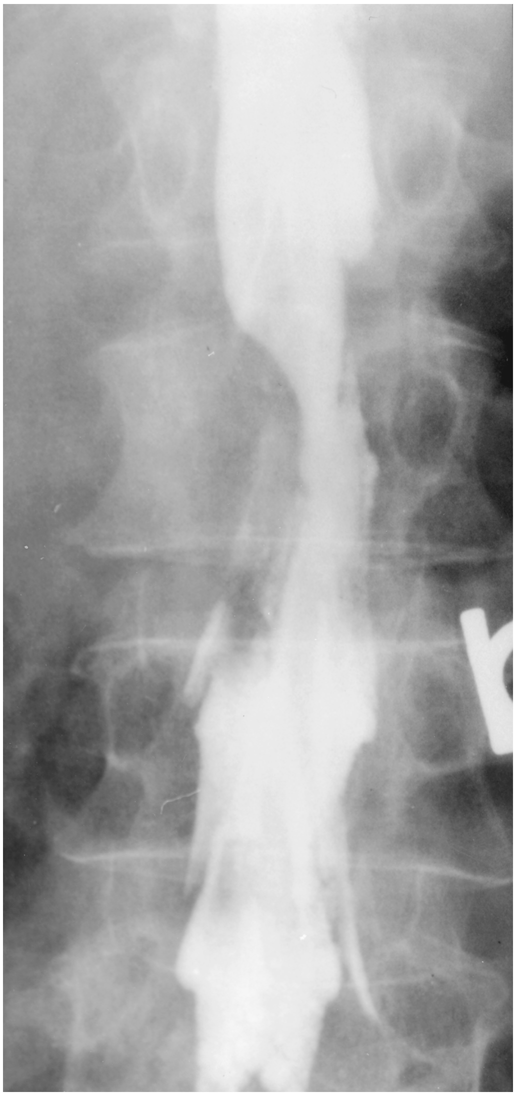

Figure 14–3. Extradural metastasis causing pedicle erosion ("winking owl") and myelographic block.

et al., 1986; Redmond et al., 1984) (Fig. 14–4B). However, CT scanning is effectively limited to transverse representations; coronal and sagittal reconstructions with this imaging method are less exact.

Magnetic Resonance Imaging

Magnetic resonance imaging is the imaging modality of choice for spinal tumors, including spinal metastases (Berenstein and Graeb, 1982; Jaeckle, 1991; Khaw et al., 1999; Markus, 1996; Schiff et al., 1998; Sze, 1991). The spine may be evaluated in various planes, and the entire spinal column can be visualized in sagittal cross sections. Patterns of extradural metastases can be identified, including an isolated level of focal disease, multiple levels of contiguous involvement, or multiple, noncontiguous levels of tumor foci (Fig. 14–5).

Magnetic resonance imaging with gadolinium enhancement permits identification of intradural extramedullary "drop" metastases typically found along the cauda equina nerve roots. Gadolinium-enhanced MRI will also delineate intramedullary spinal metastases.

Coronal, sagittal, and transverse reconstructions from MRI provide important information concerning the location, multiplicity, and geometry of secondary spinal tumors and demonstrate the degree of bony integrity at adjacent vertebral levels, all essential parameters for planning an optimal treatment.

MANAGEMENT

Rationale

Treatment of patients with spinal metastases is undertaken to relieve pain and preserve or restore neurologic function. Cancer patients do not die of spinal metastases per se (Table 14–5). Rather, they succumb to infection, organ failure, infarction, carcinomatosis, and hemorrhage (Inagaki et al., 1974). Morbidity from spinal metastases can increase a cancer patient's susceptibility to various complications, thereby reducing life expectancy. Morbidity is generally lessened if the diagnosis is made and treatment initiated before significant neurologic or functional disability has developed (Bilsky et al., 1999; Helweg-Larsen, 1996; Kovner et al., 1999). Relief from pain and preservation or restoration of neurologic function contribute immeasurably to the quality of remaining life and reduce the burden of care (Weigel et al., 1999).

Radiation Therapy Versus Surgery

Therapeutic irradiation and surgery are complementary treatment modalities for spinal metastases. Response to radiation depends on the primary histology and volume of disease. Complete response to radiation therapy for spinal cord compression is achieved

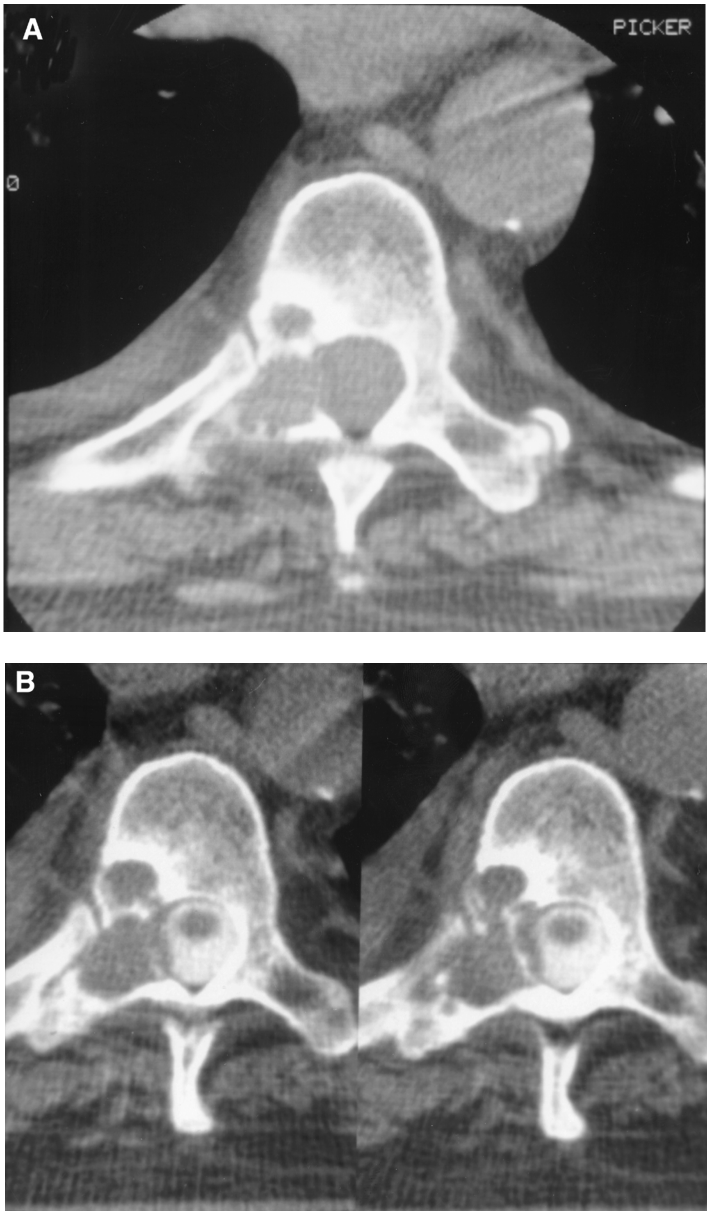

Figure 14–4. (A) Plain CT scan showing pedicle destruction. **(B)** CT scan following myelography showing compression of dural sac.

in 30% of patients with all types of tumors, including those with breast cancer and malignant melanoma (Leviov et al., 1993). Radiosensitive tumors, such as lymphoproliferative malignancies, multiple myelomas, and germ cell tumors, provide an exception; 77% of patients with these tumors achieve a complete response to irradiation alone.

Of patients with spinal metastases, 80% respond to radiation therapy alone. Improvement of motor dysfunction occurs in 49%, and stabilization of the clinical status occurs in an additional 31% of cases (Maranzano et al., 1991).

Due to irreversible spinal cord injury, patients presenting with a complete spinal cord block generally

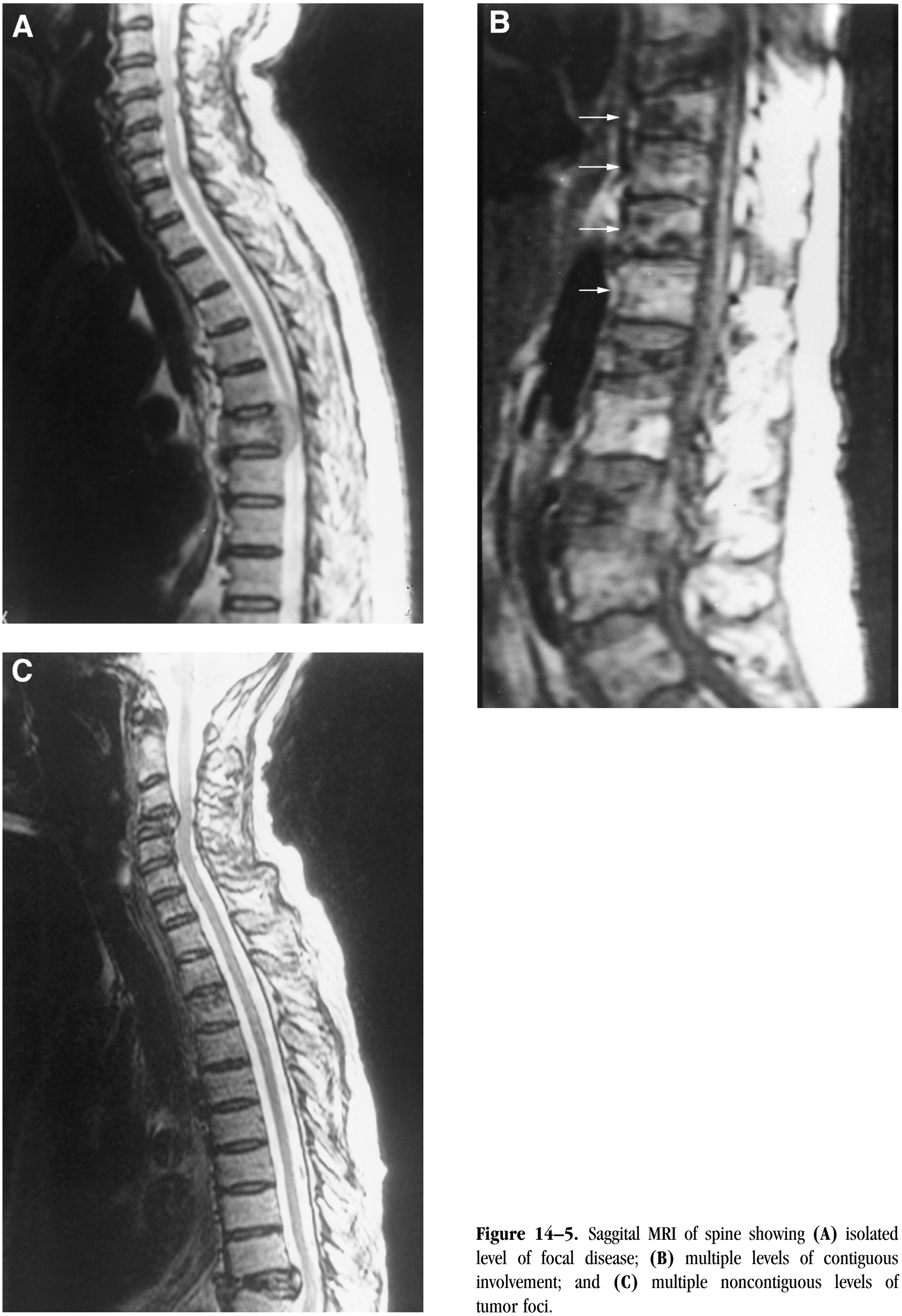

Figure 14–5. Saggital MRI of spine showing **(A)** isolated level of focal disease; **(B)** multiple levels of contiguous involvement; and **(C)** multiple noncontiguous levels of tumor foci.

Table 14–5. Causes of Death in Cancer Patients

Cause	%
Infection	47
Organ failure	25
Infarction	11
Carcinomatosis	10
Hemorrhage	7

have greater residual neurologic impairment after radiation therapy than those with a partial block (Boogerd and van der Sande, 1993). Approximately 20% of patients with epidural spinal cord compression will have an associated paravertebral mass (Kim et al., 1993; Turner et al., 1993). Radiation therapy is less effective when epidural spinal cord compression is associated with a paravertebral mass because of the large tumor burden. In these cases, surgical resection, followed by radiation therapy, has been suggested as a way to improve functional outcome (Kim et al., 1993). Moreover, Schiff et al. (1998) have identified surgical intervention as a favorable prognostic factor associated with survival among patients with epidural spinal metastases.

Radiation therapy has traditionally been the initial treatment of choice for most cases of spinal cord compression because no overall difference in the neurologic outcome has been observed when patients are treated by either radiation therapy alone or surgery with radiation therapy (Byrne, 1992; Young et al., 1980). Recent refinements in surgical strategies, including elaboration of posterolateral, anterior, and endoscopic approaches for spinal decompression, together with the evolution of spinal stabilization procedures, have improved the outcome for patients undergoing surgery for spinal metastases. Such improvement has, in turn, lent support to the concept of de novo surgery for secondary spinal tumors. Furthermore, operating on spinal metastasis before applying radiation minimizes the risk of wound complications, which can be as high as 30% with surgery through an irradiated tissue bed. Clarification of the relative roles of radiation and surgery (or a combination of these modalities) requires an appropriate prospective randomized trial.

RADIATION THERAPY

Traditional indications for radiation therapy are summarized in Table 14–6. Radiation therapy is generally delivered to an area that incorporates at least one to two vertebral levels above and below the known sites of spinal cord compression. Radiation is administered via a direct posterior field with dose specified at a depth of 5 to 8 cm. Occasionally the field length is adjusted to incorporate adjacent vertebral bodies involved by metastases but which are not causing spinal cord compression. The width of the field is usually 2 to 3 cm wider than the width of the vertebral body, but would be increased in situations where there exists a paraspinal mass. Doses on the order of 2000 to 3000 centigray (cGy) in 5 to 10 fractions are generally delivered once a day, but the range of published experiences extends from 1500 cGy in 5 fractions to 4000 cGy in 20 fractions per day. There is accumulating evidence that short-course, low-dose radiotherapy may be as effective as longer, higher dose regimens, but with fewer and less severe side effects (Maranzano et al., 1997; Tombolini et al., 1994).

When patients present with total paraplegia of several days duration, 800 cGy in one fraction is an acceptable alternative approach in an effort to minimize pain when there is no realistic prospect of neurologic recovery. Occasionally, when spinal cord compression occurs as a result of direct extension, rather than from hematogenous spread, and represents the initial manifestation of cancer, a potentially curative approach incorporating surgery, chemotherapy, and high-dose, localized radiation therapy might be appropriately considered depending on the histologic type of tumor.

Table 14–6. Indications for Radiation Therapy as the Initial Management of Spinal Metastases

Patients with radiation-sensitive tumors in the absence of any indications for surgery (i.e., lymphoma, multiple myeloma, small cell lung carcinoma, seminoma of testes)
Life expectancy of 3 months or less
More than one level of simultaneous spinal cord compression
Patients with paraplegia of greater than 12 to 24 hours duration
Co-morbid conditions that preclude surgery

The most common acute toxicities associated with radiation are nausea and vomiting, which occur as a direct result of the exit beam through the epigastrium in cord compression of the distal thoracic and proximal lumbar spine. This is usually most pronounced with the first two to three fractions and can generally be controlled with various antiemetics. Radiation esophagitis may occur 1 to 2 weeks following completion of treatment for an upper to midthoracic cord compression, but is usually mild and resolves within 1 week. The most important late toxicity is radiation myelopathy, but this is a rare event with the usual dosages quoted above and is only occasionally seen in the setting of re-treatment of the spine with a second or third course of irradiation (Wong et al., 1994).

Treatment of spinal cord compression involves a delicate balance between delivering a dose of radiation sufficient to kill the tumor and not injuring the spinal cord further. A ceiling of response, defined as maintaining the pretherapeutic level of ambulation and motor function, is considered to be 80% with radiation alone (Leviov et al., 1993). This is particularly true in patients with extensive tumor burdens, such as spinal cord compression associated with a paravertebral mass, which require high doses of radiation to achieve local control and may achieve little functional improvement after irradiation alone (Kim et al., 1993).

Table 14–7 summarizes the reported outcomes associated with radiation therapy for spinal metastases in a number of recent studies. Improvement or stabilization in patients' functional status occurred in 73% of cases following radiation therapy. Furthermore, whereas 49% of patients were able to walk before radiation therapy, 53% were able to walk after radiation therapy. These results suggest that radiation therapy is an appropriate and effective treatment for many patients with spinal metastases.

INDICATIONS FOR SURGERY

Indications for surgery in patients with symptomatic spinal metastases are listed in Table 14–8 (Botterell and Fitzgerald, 1959; Dunn et al., 1980; Gilbert et al., 1978; Perrin et al., 1982; Perrin and Livingston, 1980; Perrin and McBroom, 1990).

Table 14–7. Outcomes Following Radiation Therapy for Spinal Metastases

Author	*N*	*Dosage** *(cGy)*	*Global Rating*† *Good*	*Global Rating*† *Poor*	*Ambulatory Pre*	*Ambulatory Post*	*Mortality*‡
Sorensen et al. (1994)	57	2800	41	16	36	34	6
Tombolini et al. (1994)	103	Varied	60	43	—	—	—
Helweg-Larsen (1996)	153	2800	—	11	60	54	4
Schiff and O'Neill (1996)	35	3000	34	1	—	—	4
Maranzano et al. (1997)	53	1600	31	22	23	31	5
Katagiri et al. (1998)§	101	4000	67	33	73	75	10
Brown et al. (1999)	35	3000	31	4	21	18	4.1
Kovner et al. (1999)	79	3000	75	4	23	39	2
Solberg and Bremnes (1999)	58	Varied	—	—	—	—	3.3
Totals	674		339	134	236	251	$M = 4.8$
%		73	22	49	53		

*Median total dose.

†For global ratings, good outcomes are those in which functional or neurologic status either stabilized or improved following treatment, and poor outcomes are those in which further deterioration occurred following treatment.

‡Median total dose.

‡Median or mean survival for cohort in months.

§Sixty-two of these patients also received chemotherapy.

Table 14–8. Indications for Surgery in the Management of Spinal Metastases

Failed radiation therapy
Uncertain diagnosis
Pathologic fracture dislocation
Rapid progression or advanced paralysis

Failure of Radiation Therapy

Given the common practice of administering therapeutic radiation as the initial treatment for spinal metastases, the most common indication for surgical intervention for patients with symptomatic spinal metastases is failure of radiation to stop the spread of disease. Characteristically, symptoms persist or recur during or after radiation therapy. Surgical intervention is then indicated to relieve pain and to preserve or restore neurologic function.

Uncertain Diagnosis

Surgical intervention is indicated if it is suspected that a cancer patient's pain and neurologic dysfunction are due to disc extrusion, epidural abscess, hematoma, or some pathologic cause other than spinal metastasis. Approximately 10% of patients with symptomatic spinal metastases present without a known primary tumor. In such instances, spinal decompression may be diagnostic as well as therapeutic.

Pathologic Fracture Dislocation

Pathologic fracture dislocation occurs in approximately 10% of patients with symptomatic spinal metastases. In this circumstance, compression of the spinal cord and nerve roots by the tumor mass is compounded by distortion of the dural sac and its contents due to malalignment of the spine. Surgical intervention is required to restore alignment of the spine, to decompress the spinal cord and nerve roots, and to stabilize the spinal column.

Rapidly Progressing or Far-Advanced Paraplegia

Rapidly progressing or far-advanced paraplegia represents a neurosurgical emergency. Complete and irreversible spinal cord injury might supervene before the benefits of therapeutic radiation are manifest. Surgical decompression is indicated to provide prompt and effective decompression of the spinal cord and nerve roots.

SURGICAL STRATEGIES

Treatment for spinal metastases must ensure both decompression of the spinal cord and nerve roots and stabilization of the spinal column. Spinal instability may already have occurred at the time of clinical presentation or may be precipitated during the course of surgical decompression. In either case, appropriate spinal reconstruction must be carried out.

The surgical approach to spinal metastases may be from the front (anterior or anterolateral) or from behind (posterior or posterolateral). Each avenue has its place, and neither is always applicable (Perrin and McBroom, 1987). Because the surgical strategies must achieve both decompression of the neural elements and stabilization of the vertebral column, the optimal approach is based on a number of interrelated factors (Table 14–9).

Tumor Within the Dural Sac

Spinal metastases occurring within the dural sac are generally best approached from behind through a wide laminectomy. Occasionally, extradural spinal metastases involve only the posterior elements (Fig. 14–6), and, in these cases, decompression through a wide laminectomy is most appropriate. More often, however, spinal cord compression results from an anteriorly or laterally located extradural tumor mass or collapsed bone (Fig. 14–7; see also Fig. 14–5A). In such circumstances, adequate decompression may best be achieved through an anterior or anterolateral approach and vertebral corpectomy.

Table 14–9. Factors Determining the Optimal Surgical Approach for the Treatment of Spinal Metastases

Tumor location
Spinal level
Extent of the tumor
Bony integrity
Degree of debility

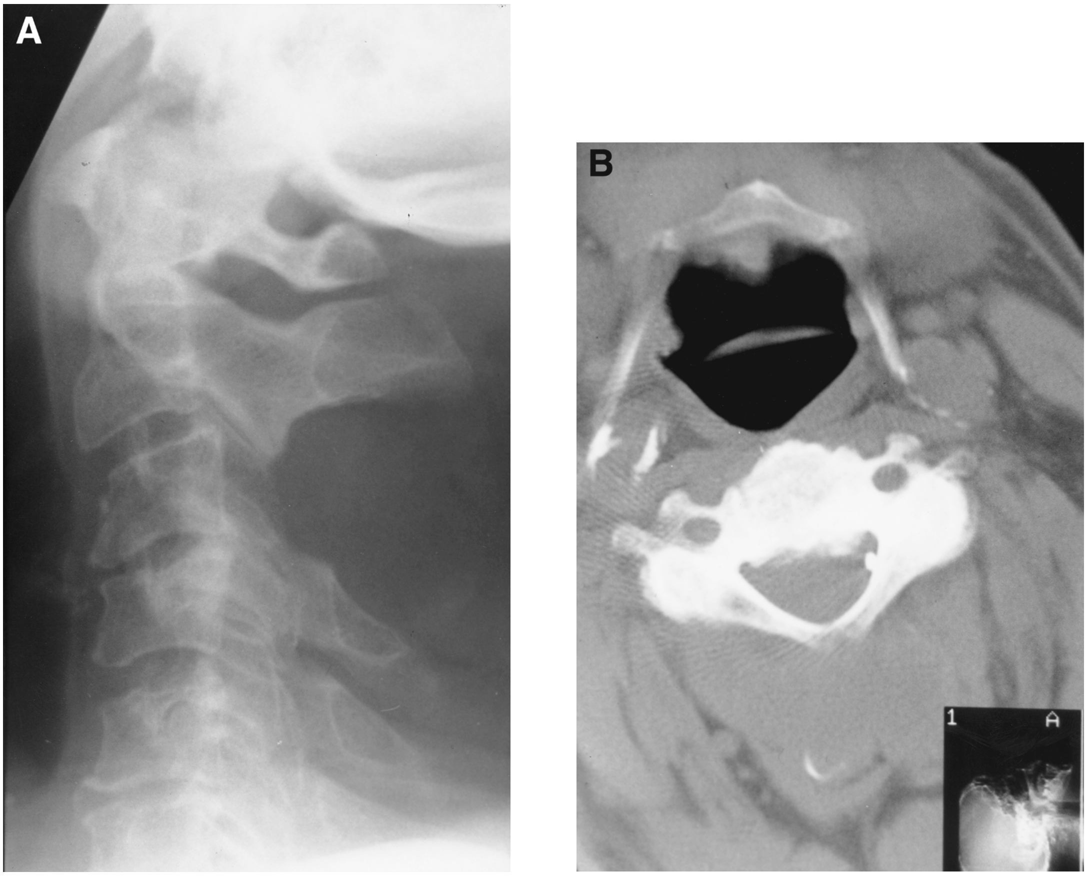

Figure 14–6. Spinal metastasis involving posterior elements, as shown by **(A)** plain film and **(B)** CT scan.

Tumor at Ends of Vertebral Column

Spinal metastases at the rostral and caudal ends of the vertebral column represent a particular challenge. Adequate anterior decompression at the craniocervical junction may be achieved through transoral and mandible-splitting exposures. However, the associated morbidity and lengthy postoperative convalescence is not in keeping with the intended palliation of a cancer patient who has a limited life expectancy. Even if adequate spinal decompression were achieved through an anterior approach at the rostral or caudal extremes of the spinal column, anterior spinal reconstruction at these levels poses an enormous technical challenge. Therefore, extradural metastases at the craniocervical and lumbosacral junctions are initially best approached from behind.

Extradural Tumor

Extradural metastases occurring anteriorly or anterolaterally and involving one or two contiguous levels are best approached from the front. The anterior (anterolateral) avenue provides direct access to the compressing mass. Furthermore, an anteriorly applied reconstruction device is biomechanically most effective. Anterior decompression (corpectomy) involving three or more contiguous segments is not impossible; however, fixation of an anteriorly applied prosthesis spanning three or more segments becomes tenuous, at best. Consequently, in such cases, posterolateral decompression and posterior fixation may be more appropriate. If vertebral corpectomy extending across three segments is undertaken, it is advisable to reinforce an anteriorly applied reconstruction prosthesis with posterior fixation.

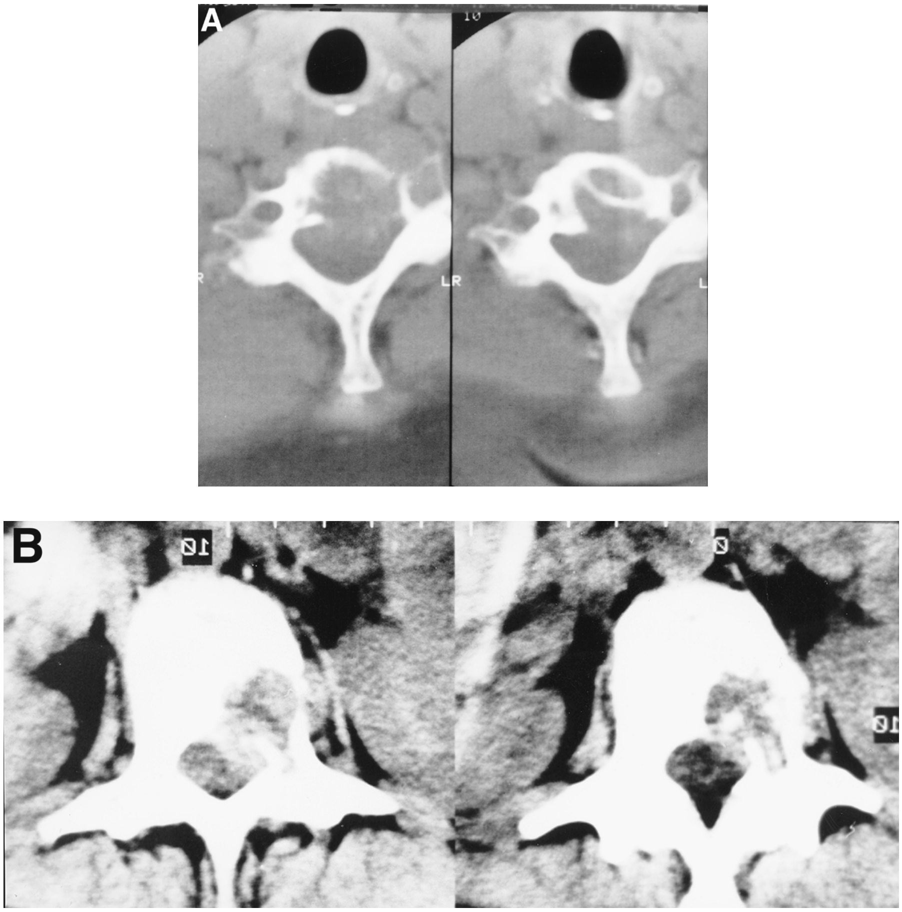

Figure14– 7. Spinal metastasis located **(A)** anteriorly and **(B)** laterally.

Integrity of Vertebral Bone

The bony integrity of vertebrae adjacent to a decompressed segment must be adequate to accept and anchor a prosthetic construct. When it is anticipated that an anteriorly applied reconstruction apparatus cannot be adequately anchored in place, it may be preferable to proceed with posterolateral decompression secured with sublaminar wires at several levels above and below the decompressed segment (see Fig. 14–5B).

Degree of Debility

The optimal surgical approach may be dictated by local and systemic factors. The anterior approach through an irradiated neck poses increased risk of tracheoesophageal perforation and associated consequences. By the same token, lengthy spinal procedures performed from behind in the thoracolumbar region with a midline incision through radiation-saturated skin in a cancer patient with impaired immunity and compromised nutrition carries a high risk of wound healing complications. On the other hand, the patient in the advanced stages of systemic cancer may be too debilitated to tolerate a transthoracic or thoracoabdominal operation.

SURGICAL APPROACHES

Preoperative Embolization

Metastases arising from thyroid and renal cell carcinoma are notoriously vascular. Catastrophic blood loss may occur unless preoperative embolization is undertaken to reduce tumor vascularity before direct surgical intervention (Bhojraj et al., 1992; Roscoe et al., 1989; Soo et al., 1982). Preoperative embolization greatly decreases intraoperative blood loss and has thus been found to allow for more complete tumor resection (Hess et al., 1997).

Posterior (Posterolateral) Decompression and Stabilization

Simple laminectomy is inadequate or inappropriate surgical treatment for all but a few patients with secondary spinal tumors. Laminectomy generally permits adequate exposure for intradural metastases and may also suffice in the uncommon event that extradural metastases involve only the posterior elements (e.g., Fig. 14–6). Most patients, however, require a wide laminectomy with posterolateral resection of the tumor-destroyed elements, which, in turn, permits excavation of the tumor-destroyed vertebral body (Fig. 14–8). Such posterolateral decompression, applied bilaterally, enables effective circumferential decompression of the dural sac and its contents (Akeyson and McCutcheon, 1996; Bauer, 1997; Perrin and McBroom, 1990; Rompe et al., 1999; Tomita et al., 1994).

Posterior spinal stabilization can be achieved with bone struts (when bony arthrodesis is anticipated in patients with prolonged life expectancy), steel rods (Harrington rods, Luque rods, or rectangle), or molded methyl methacrylate. The suitable struts are secured with sublaminar wires at a minimum of two levels above and two levels below the decompressed segment. Table 14–10 lists the variety of materials and methods described to secure spinal stabilization following posterior (posterolateral) decompression.

Anterior (Anterolateral) Decompression and Stabilization

Decompression from the front involves vertebral corpectomy. The approach is directly anterior in the cer-

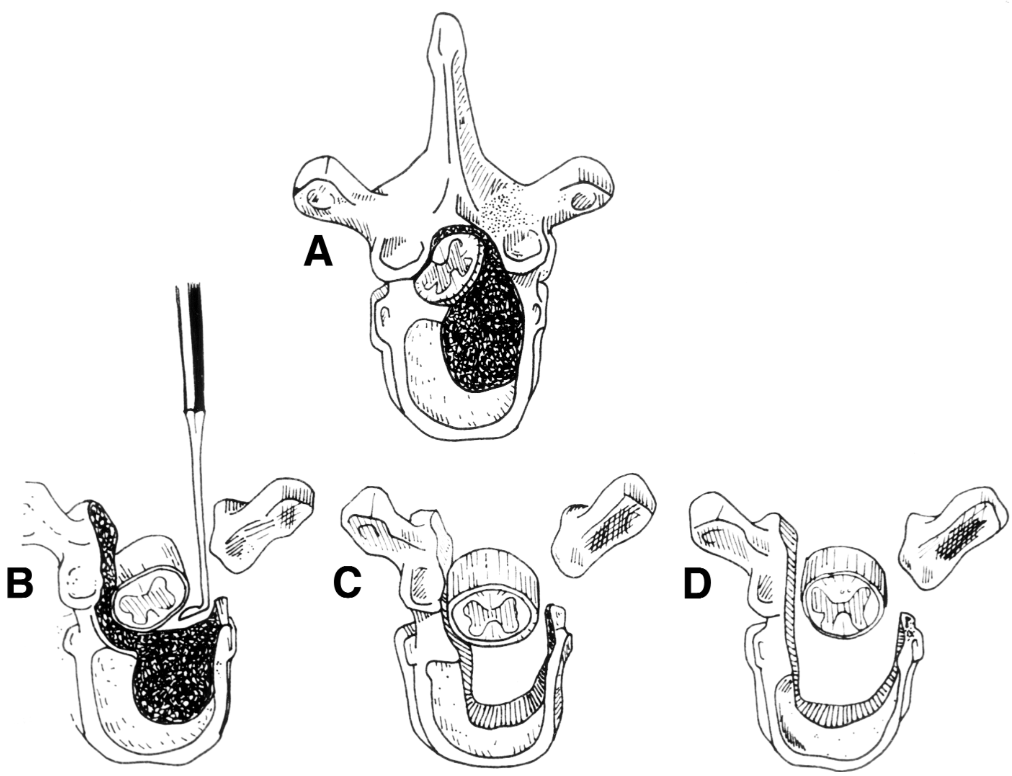

Figure 14–8. **(A)** Anterolaterally disposed extradural tumor. **(B)** Wide laminectomy with posterolateral access to vertebral body. **(C)** Posterolateral excavation of vertebral body. **(D)** Posterolaberal decompression, bilaterally.

Table 14–10. Posterior Spine Stabilization

Author	*Technique*
Rogers (1942)	Interspinous wiring
Robinson and Smith (1955)	Posterolateral facet fusion
Roy-Camille et al. (1976)	Roy-Camille plates
Livingston and Perrin (1978)	Rib struts
Perrin and Livingstone (1980)	Methylmethacrylate and sublaminar wiring
Holness et al. (1984)	Halifax clamp
Harrington (1984)	Harrington rods
Davey et al. (1985)	Dewar procedure
White et al. (1986)	San Francisco system
Krag et al. (1986)	Vermont system
Steffee et al. (1986)	Variable spine plating
Luque (1986)	Luque rods/rectangle
Ellis and Findlay (1994)	Contoured luque
Tomita et al. (1994)	En bloc spondylectomy stabilized with Cotrel-Dubousset instrumentation; vertebral reconstruction with apatite-wollastonite vertebral spacer supported by allograft bone
Akeyson and McCutcheon (1996)	Spondylectomy with posterior fixation using Luque rectangles and sublaminar cables and reconstruction with methylmethacrylate
Bauer (1997)	Wide decompression followed by stabilization without bone grafting using Cotrel-Dubousset instrumentation
Rompe et al. (1999)	Decompression and stabilization with Cotrel-Dubousset instrumentation
Weigel et al. (1999)	Laminectomy or hemilaminectomy and stabilization with titanium implants
Wise et al. (1999)	Decompression followed by autograft or allograft bone and instrumentation

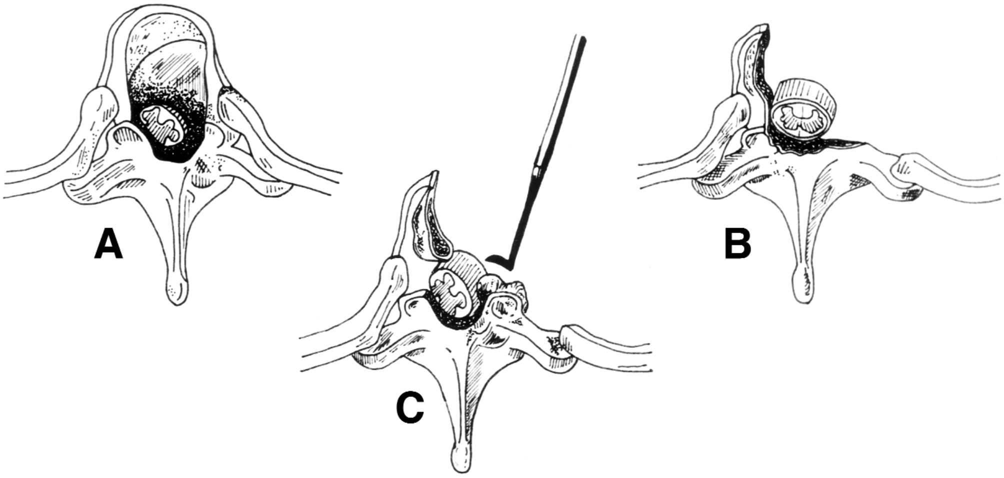

Figure 14–9. (A) Anterolaterally disposed extradural tumor. **(B)** Anterior decompression (corpectomy). **(C)** Anterolateral decompression.

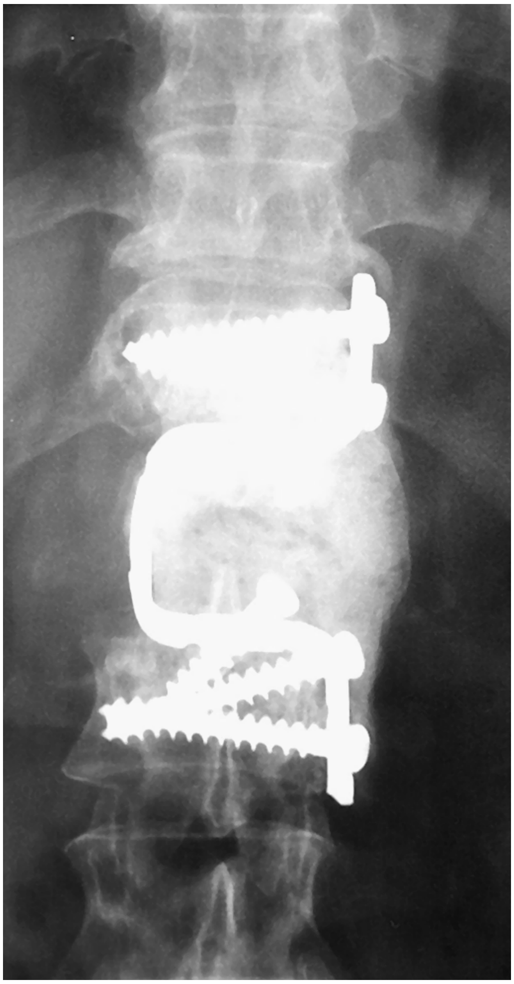

Figure 14–10. Anterior reconstruction "U"-shaped plate and methylmethacrylate (Wellesley wedge).

vical and lower lumbar regions and anterolateral in the thoracic (transthoracic avenue) and thoracolumbar (thoracoabdominal access) segments. The anterior (anterolateral) approach permits decompression under direct vision of approximately two-thirds of the dural sac circumference. When the approach is anterolateral (transthoracic and thoracoabdominal), the contralateral root sleeves are hidden from view and cannot be safely decompressed (Fig. 14–9) (Perrin and McBroom, 1994; Siegal and Siegal, 1985; Sundaresan et al., 1985).

Anterior spinal reconstruction can be achieved by means of a bone graft, with or without a plate, or with various prosthetic devices (Fig. 14–10) (Chen et al., 2000; Gokaslan et al., 1998; Perrin and McBroom, 1994). Table 14–11 lists the variety of materials and methods described to achieve spinal stability following anterior (anterolateral) decompression procedures. Because of the wide range of cervical spine mobility, if more than two vertebral segments are spanned, or when the integrity of the bone adjacent to the decompressed interval is insufficient to provide secure fixation, then supplemental stabilization with an apparatus applied from the back may be advisable.

Microsurgical endoscopic techniques provide a promising variation of anterior decompression surgery. Endoscopic surgery has been found to achieve adequate decompression and stabilization, but with a reduction in surgical trauma, postoperative pain, and postoperative hospitalization (Rosenthal et al., 1996).

Table 14–12 summarizes the outcomes associated with surgery in a number of recent studies. Improvement or stabilization of functional status occurred in 86% of cases following surgery. Whereas 68% of patients were able to walk before surgery, 85% were ambulatory after surgery. In addition to functional status, improvements have been reported in patients' quality of life following surgical treatment for spinal metastases (Weigel et al., 1999). These results suggest that, under the appropriate circumstances, surgery is an effective treatment option for many patients with spinal metastases.

CONCLUSION

The management of spinal metastases continues to pose a controversial challenge. Early diagnosis and prompt remedy are the cornerstones of treatment. Radiation and surgery constitute complimentary therapeutic modalities. Given the multiplicity of variables involved, it is impossible to critically compare reported series. In general, however, the outcomes for patients with spinal metastases depend on a number of factors, including the degree of deficit, speed of onset, culpable primary tumor burden (local and systemic), tumor location, and treatment technique (Table 14–13).

Optimal management of patients with spinal metastases involves multidisciplinary collaboration among specialists in neurology, oncology, radiation therapy, neurosurgery and orthopedics.

Table 14–11. Anterior Spine Stabilization

Author	*Technique*
Robinson and Smith (1955)	Smith-Robinson approach
Cloward (1958)	Cloward
Scoville et al. (1967)	Pins/methylmethacrylate
Cross et al. (1971)	Methylmethacrylate
Ono and Tada (1975)	Metal prosthesis
Fielding et al. (1979)	Corpectomy/iliac crest
Raycroft et al. (1978)	Corpectomy/tibia
Conley et al. (1979)	Corpectomy/fibula
Sundaresan et al. (1984)	Double K-wire/methylmethacrylate
Harrington (1984)	Knodt rods/methylmethacrylate
Perrin and McBroom (1988)	Wellesley wedge
Rosenthal et al. (1996)	Microsurgical endoscopic decompression, reconstruction with polymethylmethacrylate and plating for stabilization
Bauer (1997)	Decompression with Zielke instrumentation and cement
Gokaslan et al. (1998)	Transthroracic vertebrectomy with methylmethacrylate reconstruction and stabilization with locking plate and screw constructs
Weigel et al. (1999)	Screw fixation of the dense axis; corpectomy stabilized with titanium mesh cage filled with polymethylmethacrylate cement and titanium implants
Wise et al. (1999)	Decompression with autograft or allograft bone plus instrumentation or cement plus instrumentation
Chen et al. (2000)	Corpectomy and reconstruction using methylmethacrylate plus fixation with Zielke instrumentation

Table 14–12. Outcomes Following Surgery for Spinal Metastases

			*Global Rating**		*Ambulatory*		
Author	*No.*	*Approach*	*Good*	*Poor*	*Preop*	*Postop*	*Mortality*
Tomita et al. (1994)	20	Posterior	18	2	10	16	9
Akeyson and McCutcheon (1996)	25	Posterior	23	2	13	18	—
Rosenthal et al. (1996)	28	Anterior	28	0	—	28	—
Schiff and O'Neill (1996)	5	N/A‡	4	1	5	4	8
Tatsui et al. (1996)	1	Anterior	1	—	0	1	7.8
Tatsui et al. (1996)	15	Posterior	9	—	0	9	18.7
Bauer (1997)	67	Posterior	57	10	41	57	6
Gokaslan et al. (1998)	72	Anterior	69	3	59	68	>12
Rompe et al. (1999)	106	Posterior	98	8	73	88	19.2
Scarrow et al. (1999)	1	Posterior	1	—	1	1	—
Solberg and Bremnes (1999)	28	Posterior	—	—	—	—	10.1
Weigel et al. (1999)	96	Ant./post./both	58	28	76	90	13.1
Wise et al. (1999)	88	Ant./post./both	78	10	71	78	15.9
Chen et al. (2000)	60	Anterior	60	0	29	40	6–12
Totals	612		504	64	378	498	$M = 11.7$
%		86	11	68	85		

*For global ratings, good outcomes are those in which functional or neurologic status either stabilized or improved following treatment and poor outcomes are those in which further deterioration occurred following treatment.

†Median or mean survival for cohort in months.

‡Information on surgical approach was not available.

Table 14–13. Factors Determining Outcome

Degree of deficit
Speed of onset
Culpable primary tumor
Tumor burden (local and systemic)
Tumor location
Treatment technique

REFERENCES

Akeyson EW, McCutcheon IE. 1996. Single-stage posterior vertebrectomy and replacement combined with posterior instrumentation for spinal metastasis. J Neurosurg 85: 211–220.

Bach F, Larsen BH, Rohde K, et al. 1990. Metastatic spinal cord compression: Occurrence, symptoms, clinical presentations and prognosis in 398 patients with spinal cord compression. Acta Neurochir (Wien) 107:37–43.

Barron KD, Hirano A, Araki S, Terry RD. 1959. Experiences with metastatic neoplasms involving the spinal cord. Neurology 9:91–106.

Bauer HC. Posterior decompression and stabilization for spinal metastases: Analysis of 67 consecutive patients. 1997. J Bone Joint Surg Am 79:514–522.

Berenstein A, Graeb D. 1982. Convenient preparation of ready-to-use particles in polyvinyl alcohol foam suspensions for embolization. Radiology 145:846.

Bhojraj SY, Dandawate AV, Ramakantan R. 1992. Preoperative embolisation, transpedicular decompression and posterior stabilization for metastatic disease of the thoracic spine causing paraplegia. Paraplegia 30:292–299.

Bilsky MH, Lis E, Raizer J, Lee H, Boland P. 1999. The diagnosis and treatment of metastatic spinal tumor. Oncologist 4:459–469.

Boogerd W, van der Sande JJ. 1993. Diagnosis and treatment of spinal cord compression in malignant disease. Cancer Treat Rev 19:129–150.

Botterell EH, Fitzgerald GW. 1959. Spinal cord compression produced by extradural malignant tumours. Can Med Assoc J 80:791–796.

Brown PD, Stafford SL, Schild SE, Martenson JA, Schiff D. 1999. Metastatic spinal cord compression in patients with colorectal cancer. J Neurooncol 44:175–180.

Bruner JM, Tien RD. 1998. Secondary tumors. In: Bigner DD, McLendon RE, Bruner JM (eds), Russell and Rubinstein's Pathology of Tumors of the Nervous System, 6th ed. London: Arnold, p 441.

Byrne TN. 1992. Spinal cord compression from epidural metastses. N Engl J Med 327:614–619.

Chen LH, Chen WJ, Niu CC, Shih CH. 2000. Anterior reconstructive spinal surgery with Zielke instrumentation for metastatic malignancies of the spine. Arch Orthop Trauma Surg 120:27–31.

Cloward RB. 1958. The anterior approach for removal of ruptured cervical discs. J Neurosurg 15:602–614.

Conley FK, Britt RH, Hanberry JW, Silverberg GD. 1979. Anterior fibular strut graft in neoplastic disease of the cervical spine. J Neurosurg 51:677–684.

Cross GO, White HL, White LP. 1971. Acrylic prosthesis of the fifth vertebra in multiple myeloma. Technical note. J Neurosurg 35:112–114.

Davey JR, Rorabeck CH, Bailey SI, Bourne RB, Dewar FP. 1985. A technique of posterior cervical fusion for instability of the cervical spine. Spine 10:722–728.

Dunn RC Jr, Kelly WA, Wohns RN, Howe JF. 1980. Spinal epidural neoplasia: A 15-year review of the results of surgical therapy. J Neurosurg 52:47–51.

Edelson RN, Deck MD, Posner JB. 1972. Intramedullary spinal cord metastases. Clinical and radiographic findings in nine cases. Neurology 22:1222–1231.

Ellis PM, Findlay IM. 1994. Craniocervical fusion with contoured Luque rod and autogenic bone graft. Can J Surg 37:50–54.

Fielding JW, Pyle RN, Fietti VG. 1979. Anterior cervical vertebral body resection and bone-grafting for benign and malignant tumors. J Bone Joint Surg Am 61:251–253.

Gilbert RW, Kin JH, Posner JB. 1978. Epidural spinal cord compression from metastatic tumor: diagnosis and treatment. Ann Neurol 3:40–51.

Gokaslan ZL, York JE, Walsh GL, et al. 1998. Transthoracic vertebrectomy for metastatic spinal tumors. J Neurosurg 89:599–609.

Gomez JAO. 1955. The incidence of vertebral body metastases. Int Orthop 19:309–311.

Goodkin R, Carr BI, Perrin RG. 1987. Herniated lumbar disc disease in patients with malignancy. J Clin Oncol 5:667–671.

Harrington KD. 1984. Anterior decompression and spinal stabilization for patients with metastatic lesions of the spine. J. Neurosurg 61:107–117.

Helweg-Larsen S. 1996. Clinical outcome in metastatic spinal cord compression. A prospective study of 153 patients. Acta Neurol Scand 94:269–275.

Helweg-Larsen S, Johnsen A, Boesen J, Sorensen PS. 1997. Radiologic features compared to clinical findings in a prospective study of 153 patients with metastatic spinal cord compression treated by radiotherapy. Acta Neurochir (Wien) 139:105–111.

Helweg-Larsen S, Sorensen PS. 1994. Symptoms and signs in metastatic spinal cord compression: a study of progression from first symptom until diagnosis in 153 patients. Eur J Cancer 30A:396–398.

Hess T, Kramann B, Schmidt E, Rupp S. 1997. Use of preoperative vascular embolisation in spinal metastasis resection. Arch Orthop Trauma Surg 116:279–282.

Holness RO, Huestis WS, Howes JW, Langille RA. 1984. Posterior stabilization with an interlaminar clamp in cervical injuries: technical note and review of the long term experience with the method. Neurosurgery 14:318–322.

Inagaki J, Rodriguez V, Bodey GP. 1974. Causes of death in cancer patients. Cancer 33:568–573.

Jaeckle KA. 1991. Neuroimaging for central nervous system tumors. Semin Oncol 18:150–157.

Katagiri H, Takahashi M, Inagaki J, et al. 1998. Clinical results of nonsurgical treatment for spinal metastases. Int J Radiat Oncol Biol Phys 42:1127–1132.

Khaw FM, Worthy SA, Gibson MJ, Gholkar A. 1999. The appearance on MRI of vertebrae in acute compression of the spinal cord due to metastases. J Bone Joint Surg Br 81: 830–834.

Kim HJ, Ryu KN, Choi WS, Choi BK, Choi JM, Yoon Y. 1999. Spinal involvement of hematopoietic malignancies and metastasis: Differentiation using MR imaging. Clin Imaging 23:125–133.

Kim RY, Smith JW, Spencer SA, Meredith RF, Salter MM. 1993. Malignant epidural spinal cord compression associated with a paravertebral mass: its radiotherapeutic outcome on radiosensitivity. Int J Radiat Oncol Biol Phys 27:1079–1083.

Kovner F, Spigel S, Rider I, et al. 1999. Radiation therapy of metastatic spinal cord compression: multidisciplinary team diagnosis and treatment. J Neurooncol 42:85–92.

Krag MH, Beynnon, BD, Pope MH, Frymoyer JW, Haugh LD, Weaver DL. 1986. An internal fixator for posterior application to short segments of the thoracic, lumbar, or lumbosacral spine. Design and testing. Clin Orthop 203:75–98.

Leviov M, Dale J, Stein M et al. 1993. The management of metastatic spinal cord compression: a radiotherapeutic success ceiling. Int J Radiat Oncol Biol Phys 27:231–234.

Livingston KE, Perrin RG. 1978. The neurosurgical management of spinal metastases causing cord and cauda equina compression. J Neurosurg 49:839–843.

Luque ER. 1986. Interpeduncular segmental fixation. Clin Orthop 203:54–57.

MacDonald DR. 1990. Clinical manifestations. In: Sundaresan N, Schmidek H, Schuller A (eds), Tumors of the Spine. Philadelphia: WB Saunders, Philadelphia, pp 6–21.

Maranzano E, Latini P, Checcaglini F. 1991. Radiation therapy in metastatic spinal cord compression. A prospective analysis of 105 consecutive patients. Cancer 67:1311–1317.

Maranzano E, Latini P, Perrucci E, Beneventi S, Lupattelli M, Corgna E. 1997. Short-course radiotherapy (8 Gyx2) in metastatic spinal cord compression: an effective and feasible treatment. Int J Radiat Oncol Biol Phys 38:1037–1044.

Markus JB. 1996. Magnetic resonance imaging of intramedullary spinal cord metastases. Clin Imaging 20:238–242.

Ono K, Tada K. 1975. Metal prostheses of the cervical vertebra. J Neurosurg 42:562–566.

O'Rourke T, George CB, Redmond J 3rd, et al. 1986. Spinal computed tomography and computed tomographic metrizamide myelography in the early diagnosis of metastatic disease. J Clin Oncol 4:576–583.

Perrin RG, Livingston KE. 1980. Neurosurgical treatment of pathological fracture-dislocation of the spine. J Neurosurg 52:330–334.

Perrin RG, Livingston KE, Aarabi R. 1982. Intradural extramedullary spinal metastasis. A report of 10 cases. J Neurosurg 56:835–837.

Perrin RG, McBroom RJ. 1987. Anterior versus posterior decompression for symptomatic spinal metastasis. Can J Neurol Sci 14:75–80.

Perrin RG, McBroom RJ. 1988. Spinal fixation after anterior decompression for symptomatic spinal metastasis. J Neurosurg 22:324–327.

Perrin RG, McBroom RJ. 1990. Surgical treatment for spinal metastases: the posterolateral approach. In: Sundaresan N, Schmidek H, Schiller A, Rosenthal A (eds), Tumors of the Spine. Philadelphia: WB Saunders, pp 305–315.

Perrin RG, McBroom RJ. 1994. Metastatic tumors of the spine. In: Rengachary SS, Wikins RH (eds), Principles of Neurosurgery. St. Louis: Wolfe Publishing, pp 37.1–37.32.

Raycroft JF, Hockman RP, Southwick WO. 1978. Metastatic tumors involving the cervical vertebrae: surgical palliation. J Bone Joint Surg Am 60:763–768.

Redmond J 3d, Spring DB, Munderloh SH, George SB, Mansour RP, Volk SA. 1984. Spinal computed tomography scanning in the evaluation of metastatic disease. Cancer 54:253–258.

Robinson RA, Smith GW. 1955. Anterolateral cervical disc removal and interbody fusion for instability of the cervical spine. Bull Johns Hopkins Hosp 96:223–224.

Rogers L, Heard G. 1958. Intrathecal spinal metastases (rare tumours). Br J Surg 45:317–320.

Rogers WA. 1942. Treatment of fracture-dislocation of the cervical spine. J Bone Joint Surg 24:245–258.

Rompe JD, Hopf CG, Eysel P. 1999. Outcome after palliative posterior surgery for metastatic disease of the spine—evaluation of 106 consecutive patients after decompression and stabilisation with the Cotrel-Dubousset instrumentation. Arch Orthop Trauma Surg 119:394–400.

Roscoe MW, McBroom RJ, St. Louis E, Grossman H, Perrin R. 1989. Preoperative embolization in the treatment of osseous metastases from renal cell carcinoma. Clin Orthop 238:302–307.

Rosenthal D, Marquardt G, Lorenz R, Nichtweiss M. 1996. Anterior decompression and stabilization using a microsurgical endoscopic technique for metastatic tumors of the thoracic spine. J Neurosurg 84:565–572.

Roy-Camille R, Saillant G, Berteaux D, Salsado V. 1976. Osteosynthesis of thoraco-lumbar spine fractures with metal plates screwed through the vertebral pedicles. Reconstr Surg Traumatol 15:2–16.

Scarrow AM, Colina JL, Levy EI, Welch WC. 1999. Thyroid carcinoma with isolated spinal metastasis: case history and review of the literature. Clin Neurol Neurosurg 101:245–248.

Schiff D, O'Neill BP. 1996. Intramedullary spinal cord metastases: clinical features and treatment outcome. Neurology 47:906–912.

Schiff D, O'Neill BP, Suman VJ. 1997. Spinal epidural metastasis as the initial manifestation of malignancy: clinical features and diagnostic approach. Neurology 49:452–456.

Schiff D, O'Neill BP, Wang CH, O'Fallon Jr. 1998. Neuroimaging and treatment implications of patients with multiple epidural spinal metastases. Cancer 83:1593–1601.

Scoville WB, Palmer AH, Samra K, Chong G. 1967. The use of acrylic plastic for vertebral replacement or fixation in metastatic disease of the spine: technical note. J Neurosurg 27:274–279.

Siegal T, Siegal T. 1985. Treatment of malignant epidural cord and cauda equina compression. Prog Exp Tumor Res 29:225–234.

Solberg A, Bremnes RM. 1999. Metastatic spinal cord compression: diagnostic delay, treatment and outcome. Anticancer Res 19:677–684.

Soo C, Wallace S, Chuang VP, Carrasco CH, Phillies G. 1982. Lumbar artery embolization in cancer patients. Radiology 145:655–659.

Sorensen S, Helweg-Larsen S, Mouridsen H, Hansen HH. 1994. Effect of high-dose dexamethasone in carcinomatous metastatic spinal cord compression treated with radiotherapy: a randomised trial. Eur J Cancer 30A:22–27.

Steffee AD, Biscup RS, Sitkowski DJ. 1986. Segmental spine plates with pedicle screw fixation. A new internal fixation device for disorders of the lumbar and thoracolumbar spine. Clin Orthop 203:45–53.

Sundaresan N, DiGiacinto GV, Hughes JE, Cafferty M, Vallejo A. 1991. Treatment of neoplastic spinal cord compression: results of a prospective study. Neurosurgery 29:645–650.

Sundaresan N, DiGiacinto GV, Krol G, Hughes JE. 1990. Complete spondylectomy for malignant tumors. In: Sundaresan N, Schmidek H, Schiller A, Rosenthal A (eds), Tumors of the Spine. Philadelphia: WB Saunders, Philadelphia, pp 438–445.

Sundaresan N, Galicich JH, Bains MS, Martini N, Beattie EJ. 1984. Vertebral body resection in the treatment of cancer involving the spine. Cancer 53:1393–1396.

Sundaresan N, Galicich JH, Lane JM, Bains MS, McCormack P. 1985. Treatment of neoplastic epidural cord comprssion by vertebral body resection and stabilization. J Neurosurg 63:676–684.

Sze G. 1991. Magnetic resonance imaging in the evaluation of spinal tumors. Cancer 67:1229–1241.

Tatsui H, Onomura T, Morishita S, Oketa M, Inoue T. 1996. Survival rates of patients with metastatic spinal cancer after scintigraphic detection of abnormal radioactive accumulation. Spine 21:2143–2148.

Tombolini V, Zurlo A, Montagna A, et al. 1994. Radiation therapy of spinal metastases: results with different fractionations. Tumori 80:353–356.

Tomita K, Kawahara N, Baba H, Tsuchiya H, Nagata S, Toribatake Y. 1994. Total en bloc spondylectomy for solitary spinal metastases. Int Orthop 18:291–298.

Turner S, Marosszeky B, Timms I, Boyages J. 1993. Malignant spinal cord compression: a prospective evaluation. Int J Radiat Oncol Biol Phys 26:141–146.

Van der Sande JJ, Boogerd W, Kröger, Kapelle AC. 1999. Recurrent spinal epidural metastases: a prospective study with a complete follow up. J Neurol Neurosurg Psychiatry 66:623–627.

Weigel B, Maghsudi M, Neumann, Kretschmer R, Muller FJ, Nerlich M. 1999. Surgical management of symptomatic spinal metastases: postoperative outcome and quality of life. Spine 24:2240–2246.

White AH, Zucherman JF, Hsu K. 1986. Lumbosacral fusions with Harrington rods and intersegmental wiring. Clin Orthop 203:185–190.

Willis RA. 1973. The Spread of Tumors in the Human Body, 3rd ed. London: Butterworths, 429 pp.

Wise JJ, Fischgrund JS, Herkowitz HN, Montgomery D, Kurz LT. 1999. Complication, survival rates, and risk factors of surgery for metastatic disease of the spine. Spine 24:1943–1951.

Wong CS, Van Dyk J, Milosevic M, Laperriere NJ. 1994. Radiation myelopathy following single courses of radiotherapy and retreatment. Int J Radiat Oncol Biol Phys 30:575–581.

Young RF, Post EM, King GA. 1980. Treatment of spinal epidural metastases. Randomized prospective comparison of laminectomy and radiotherapy. J Neurosurg 53:741–748.