

10

Tumors

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Osteolytic metastases and myeloma are the most frequent malignant destructive lesions involving the spine. Affected patients often experience severe back pain and disability related to the vertebral fractures induced by these destructive lesions. The aim of percutaneous vertebroplasty (PV) in these disease processes is to produce pain relief and reinforcement by the injection of acrylic cement. This treatment may be used adjunctively with radiation therapy and chemotherapy.

Percutaneous vertebroplasty is rarely indicated for benign tumors. Spinal osteoid osteoma and aneurismal bone cysts do not need structural reinforcement and therefore are not an indication for PV, although they can be treated by other percutaneous methods (1,2). Fibrous dysplasias, eosinophilic granulomas, and vertebral hemangiomas (VHs) are osteolytic lesions weakening bone, and PV can be used for their treatment (3–5). The most frequent indication for PV in the treatment of benign tumors is VH. This chapter describes the role of PV in the treatment of metastatic lesions, myelomas, and VHs.

Percutaneous Vertebroplasty and Metastatic Lesions

Pathology and Patient Demographics

Patients with cancer eventually present with bone metastases in 27% of the cases (6). The vertebral bodies are the most frequent site of bone metastatic disease (7). The incidence of metastatic lesion to the spine depends on the primary cancer: 80% of patients with prostate cancer, 50% of patients with breast cancer, and 30% of patients with lung, thyroid, or renal cell cancer (8). Breast (30%), prostate (10%), and lung (25%) cancers are the three main etiologies of metastases to the spine (7,8).

The 1-year survival rate after diagnosis of spinal metastases is high for patients with prostate (83%) or breast (78%) cancer (hormonal-dependent cancers) but low for patients with lung cancer (22%) (9). Survival rates for patients with renal cell or thyroid cancer depend on the histologic classification of the tumor cells (9). The detection of spinal

metastasis from the time of primary lesion diagnosis is shortest for patients with lung cancer (3.6–6.1 months) and longest for patients with breast cancer (29.4–33.5 months) (10). About 7.5% of patients present with spinal metastases before the diagnosis of the primary lesion (10). The thoracic spine is the most common site of disease (70%), followed by the lumbar spine (20%), and cervical spine (10%). These data are important to consider when counseling patients for therapy.

Indications and Contraindications

The primary indication for PV is proven metastatic disease to the spine of a patient who is experiencing severe, focal, and mechanical back pain that limits normal activities and requires narcotic medications. Usually there will be a vertebral compression fracture (VCF) associated with the osteolytic metastatic lesion, although the amount of compression may be small.

Inherent in the process of malignant involvement of the spine is destruction of portions of the vertebral body. The greater the destruction, the more chance there is for vertebral collapse and pain. In addition, these lesions present problems for the physician considering PV, because destruction of the cortex of the vertebra, although not a contraindication, increases the possibility of cement leakage. In several studies, 40% of patients treated with PV had partial destruction of the posterior wall (Figure 10.1) (11–13). However, if there is extension of the tumor through the posterior wall (Figure 10.2), PV should be considered only after a multidisciplinary discussion, and a surgical team should be available in case spinal cord decompression is needed. Shimony et al. (14) demonstrated that PV could be performed safely

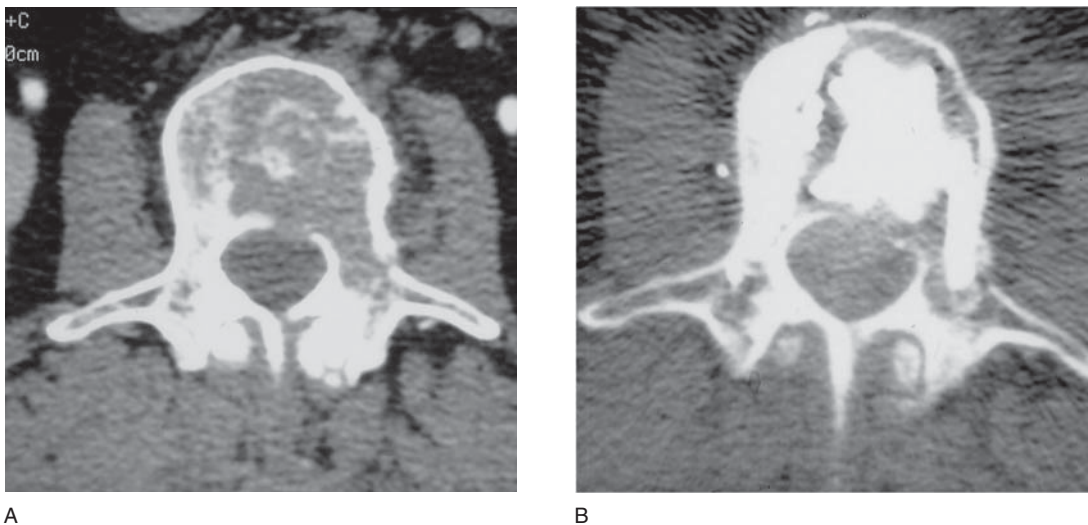


Figure 10.1. Partial destruction of the posterior wall. Axial CT scans before (A) and after (B) injection of cement. Note the injection of both the “normal” part and the osteolytic part of the vertebral body. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

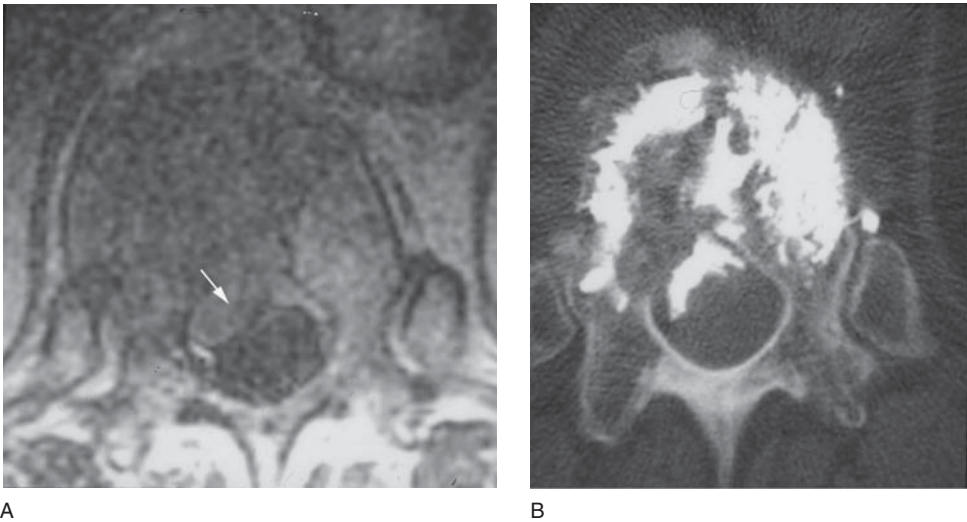


Figure 10.2. Partial destruction of the posterior wall with anterior epidural involvement by the tumor (white arrow in A). **(A)** Axial MR image before PV. **(B)** Axial CT scan after PV. Note the cement in the epidural component of the tumor; there were no neurologic complications. Both the “normal” and the osteolytic parts of the vertebral body were injected. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

and effectively with conscious sedation for patients with epidural involvement without neurologic symptoms. Conscious sedation provides an extra measure of safety because patients are able to tell if any pain, especially radicular pain, develops during the injection of polymethylmethacrylate (PMMA) (14). Clinical signs of compression of nerve roots or cord are contraindications to PV because there is a distinct risk of increasing compression with the injection of cement.

In general, PV is not indicated for asymptomatic lesions of the spine. One should first consider other therapies (radiotherapy, chemotherapy, thermoablation, etc.). Percutaneous vertebroplasty can be performed if other therapies have been exhausted and/or if there is a high risk of vertebral collapse (Figure 10.3).

The presence of multiple spinal lesions with diffuse back pain is not an indication for PV. Percutaneous vertebroplasty for focal pain with multiple lesions is appropriate, but the treatment of several lesions may be required to give adequate pain relief (Figure 10.4). The decision of which vertebra to treat depends on the correlation between the imaging examination and physical findings. Physical examination can be performed by using fluoroscopy to determine which level is symptomatic. No more than three vertebrae should be treated at one session.

Although lesions in the thoracic and lumbar spine are often treated with PV, those in the cervical region can be treated operatively without major surgical exposure. However, based on the situation, patient’s condition, and age, PV may be useful for treating cervical metastatic lesions (Figure 10.5). As in all levels of the spine, metastatic lesions are associated with a high risk of epidural invasion or spinal cord damage in the presence of posterior wall compromise.

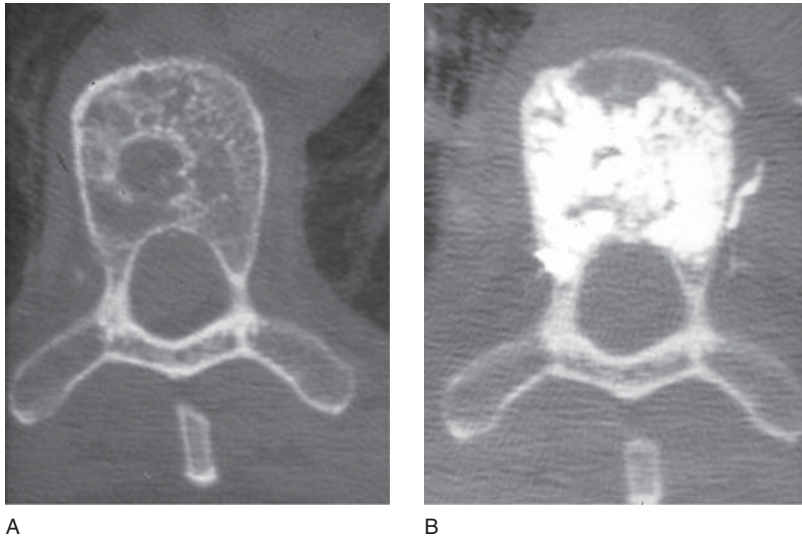


Figure 10.3. Patient with asymptomatic breast osteolysis of T8. (A) Axial CT before PV. (B) Axial CT after PV, which was performed because the extensive tumor placed the vertebral body at high risk for collapse. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

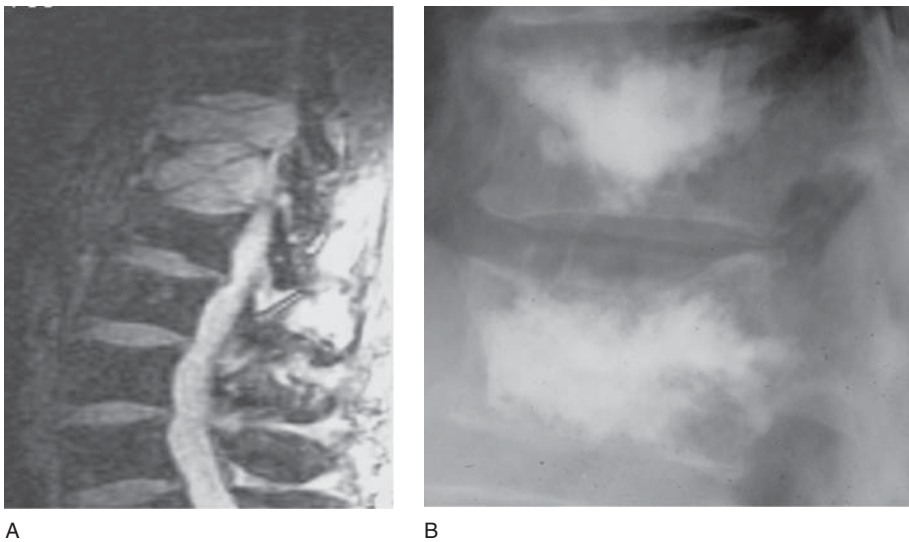


Figure 10.4. This patient presented with severe and focal back pain related to two metastatic lesions of T11–T12. MR image (A) and lateral view (B) after PV at two levels. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

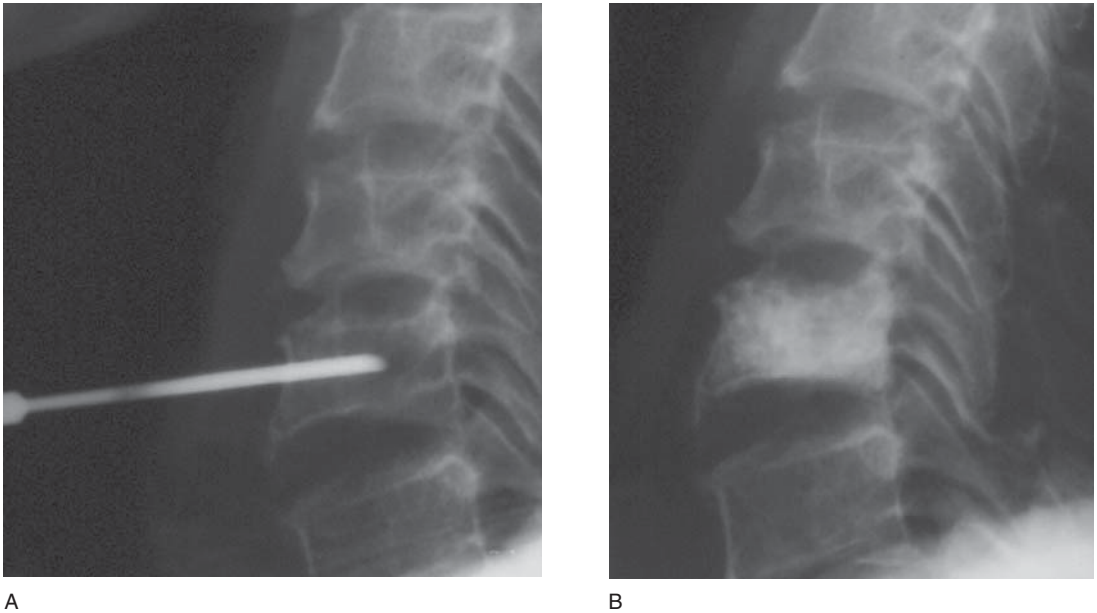


Figure 10.5. This patient presented with severe cervical pain related to a C5 lung cancer metastatic lesion. Lateral views before (**A**) and after (**B**) the injection of cement. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

Contraindications to PV with spinal metastatic lesions include (1) complete collapse of the vertebra (generally there needs to be 25% to 30% of the original height remaining to allow successful PV [12]); (2) pure osteoblastic lesions (a mixed sclerotic and destructive lesion with focal pain and collapse is a good indication for PV) (Figure 10.6); (3) nerve root or spinal cord compression related to epidural or foraminal extension of the tumor; (4) diffuse (nonfocal) back pain and failure to localize symptomatic level(s); (5) general infectious disorders; and (6) coagulation disorders (platelets below 100,000, prothrombin time greater than 3 above the upper limits of normal, and partial thromboplastin time more than 1.5 times normal).

Patient Selection and Evaluation

Generally, patients are referred for three main reasons: known cancer and back pain related to a spinal metastasis, known cancer and a recently diagnosed but asymptomatic spinal lesion, or back pain and suspicious lesions but no known diagnosis. Patient evaluation should consider all available clinical information, and clinical examination should identify the focal pain that correlates to the lesion considered for PV. Back pain usually increases when the patient is standing and decreases when the patient is recumbent. The patient's pain should be severe, altering activities of daily living or requiring substantial use of analgesics. This pain should be documented with measurement instruments such as visual analog scale and a quality-of-life questionnaire.



Figure 10.6. Breast metastatic and mixed osteolytic and osteoblastic lesion at T9 in a patient presenting with severe back pain. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

Back pain described by the patient and detected on the clinical examination should be compared with the findings on plain radiographs, magnetic resonance imaging (MRI), computed tomography (CT), and nuclear medicine scans. These diagnostic studies should be assessed for osteolysis, the degree of collapse, extension of tumor into the epidural space and foramina, compression of the neural tissue, and diffusion of metastatic lesions on the spine and bones. Computed tomography is best for detecting destruction of the posterior vertebral wall and determining whether the lesion is osteolytic and/or osteoblastic. Computed tomography gives the percentage of vertebral body destruction: 50% or more of the vertebral body needs to be destroyed before there is a substantial risk of collapse (Figure 10.3).

Once the patient has been found to meet the criteria indicating a need for PV, the procedure should be completely discussed with the patient and his or her family. This discussion should include the potential benefits of PV, its palliative nature, and the risks associated with the procedure. Finally, the patients should undergo a preanesthetic evaluation: electrocardiogram (ECG) and laboratory screen (complete blood cell count/platelets, electrolytes, prothrombin time/partial thromboplastin time, and blood urea nitrogen/creatinine).

Technique

The technique for PV of malignant lesions is the same as that used for other indications (see Chapter 7). When the primary cancer is not known or if there is a doubt about the cause of the vertebral lesion, a

biopsy should precede the injection of cement. The cannula placed for cement injection will accommodate a 15- to 18-gauge biopsy device. These two procedures can be performed in one session because the presence of malignancy does not preclude PV.

To obtain good structural reinforcement of a partially destroyed vertebral body, both the osteolytic and normal parts of the vertebra should be filled with cement (Figures 10.1 and 10.2). Therefore, a bilateral transpedicular approach is usually required to achieve maximal filling of malignant lesions.

The distribution of cement must be monitored in real time with a high-quality fluoroscope. It is most important to examine the lateral view because this projection reveals leaks that occur posteriorly toward the epidural or foraminal space or anteriorly toward veins. Cement injection should be stopped immediately when the cement approaches the projection of the posterior vertebral wall or fills a vein anterior to the projection of the anterior vertebral cortex. It is not important that the fill be homogeneous in distribution. A partial fill of the vertebral body can provide good pain relief. Pain relief has not been shown to be related to the quantity of cement injected (12,13).

However, if too little cement is injected, there remains the possibility of additional vertebral compression with weight bearing. Belkoff et al. (15) have shown in vitro that 4 to 6 mL of cement is needed to restore initial stiffness to osteoporotic vertebra (without osteolytic destruction). Their study provides an approximation of the minimal volume that may be desired for structural reinforcement at the lumbar level. Another reason to fill the metastatic lesion as much as possible is to try to get the best antitumoral effect of PV. This antitumoral effect could be related to the ischemia or thermal necrosis due to the exothermic polymerization of the cement. The bigger the core of cement, the better will be the antitumoral effect. This is important if the patient is contraindicated for complementary local radiation therapy.

Martin et al. (16) performed PV by using an access route via the lysed pedicle for the treatment of lytic lesions involving the pedicle. If the pedicle was not visible or was partially visible, the position of the pedicle was deduced from the position of the contralateral pedicle and the position of pedicles above and below the level to be treated. After treatment of the vertebral body, the needle is withdrawn stepwise through the pedicle, and the injection of cement can be obtained by introducing the stylet into the needle: 0.7 cc of cement is then delivered. In most of the procedures this amount of cement is sufficient to get a good filling of the osteolytic pedicle. The filling is considered satisfactory if the cement fills the metastasis and extends from the body through the affected pedicle.

The standard needle size varies from 10 to 13 gauge for the lumbar and thoracic spine. A 15-gauge needle is normally used in the cervical region. Smaller 18-gauge needles have been used, but the cement needs to be less viscous (17–19). Although we believe this technique is associated with a higher risk of cement leakage and resultant clinical cervical complications, a technique to reduce the risk of leaks is to insert several needles into different parts of the osteolytic lesion and inject small

amounts of cement through each needle. At the cervical level, insertion of only one 15-gauge needle in the center of the osteolytic lesion using an anterolateral approach permits good filling of the vertebra.

Results

In 1989, Lapras et al. (17) were the first to report the use of PV for a L1 painful metastatic lesion. This early experience was encouraging because the patient experienced good pain relief and was able to resume walking. This report was followed by that of Kaemmerlen et al. (18,19), who found that 80% of 20 patients experienced substantial pain relief within 48 hours from PV for malignant lesions. In 1996, Weill et al. (12) reported that more than 75% of the patients in their series experienced pain relief and improved quality of life after PV. The results were sustained for 6 months or longer in 73% of the patients. Cortet et al. (20) reported a 97% positive response rate for patients with malignant lesions within 48 hours after PV. Pain relief was complete in 13.5% and substantially improved in 55%. The remaining 30% of patients rated their improvement as moderate. The improvement was unchanged in 75% of the patients 6 months later. Nevertheless, although substantial, the quality and quantity of pain relief after PV for malignant lesions appears to be less than that found for osteoporotic lesions treated by PV.

More recently, Fourney et al. (21) reviewed a consecutive group of cancer patients (21 with myeloma and 35 with other primary malignancies) undergoing vertebro- and kyphoplasty at their institution. Improvement or complete pain relief was noted in 84% of the patients. No patient's pain was worsened by the procedure. Analgesic consumption was reduced at 1 month, and there was a durable analgesic effect at each follow-up interval up to 1 year.

The mechanisms of pain relief in patients with malignant lesions are not completely known. Stabilization of microfractures and reduction of mechanical forces are certainly the main factors. Tumor ischemia induced by the injection of cement into a solid lesion may also play a role. Destruction of the nerve endings in response to chemical (cytotoxic effect of the monomer) and thermal (exothermic reaction of the cement) forces has been postulated, but these mechanisms likely play a relatively minor role (22). The necrotizing effect of the cement on the tumor mass may extend for a short distance beyond the limits of the margins of the PMMA (23) and may be a factor in the low rate of recurrence at the site of the PV even without complementary treatments.

Side Effects and Complications

A more complete description of the potential complications associated with PV is provided in Chapter 13. It is known that the incidence of cement leaks with PV for metastatic lesions is much higher than that associated with osteoporotic fractures. This fact is almost surely attributable to the cortical destruction frequent in metastatic lesions. The rate of complications is about 10% and the incidence of radiculopathy is

about 5% after PV for metastatic lesions (11–13,24). Our personal experience with the most recent 200 patients indicates a complication rate less than 5%, and most of these complications are transient.

Problems that create pain may be transient and amenable to therapy with nonsteroidal anti-inflammatory medications or local steroid injections. Persistent radiculopathy may require surgical intervention to remove cement that might be compressing nerve roots (Figure 10.7). Any side effect (mild or major) should prompt re-examination to determine the cause and initiate the adequate treatment. Usually, a CT scan is the most direct study for identifying a cement leak.

Percutaneous Vertebroplasty and Other Therapies

Radiation therapy alone can give partial or complete pain relief in 75% to 90% of patients (25,26). However, it takes 2–10 days to see improvement in pain following half-body irradiation and 1–2 weeks following external beam radiotherapy (27), and there is little strengthening of the vertebra, which leaves the vertebra at long-term risk for additional collapse and pain. In situations when immediate pain relief is desired, such as intractable pain, or for patients with short life expectancy, vertebroplasty may provide an ideal solution. Percutaneous vertebroplasty does not diminish the positive effects of radiation (28). Percutaneous vertebroplasty can be used to obtain rapid pain relief and to

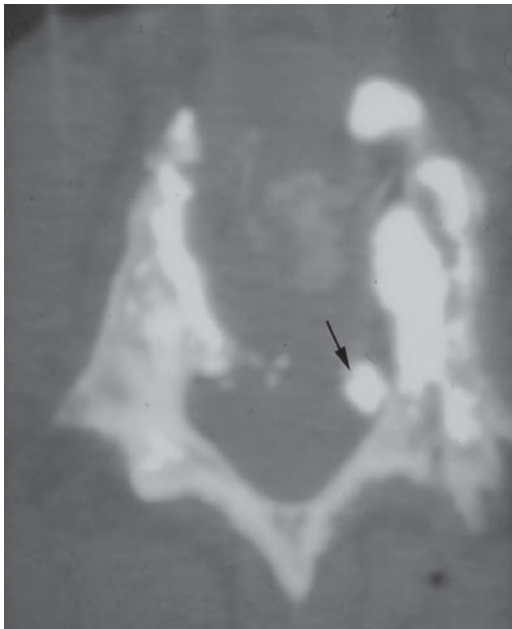


Figure 10.7. L4 breast cancer osteolytic metastatic lesion. Cement leaked into the radicular canal (black arrow), inducing severe radiculopathy that resolved after surgical removal of the extravasated cement. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

induce reinforcement of the involved vertebra. Radiation therapy should help reduce local tumor recurrence. Vertebroplasty and radiation therapy should be considered complementary procedures.

When there are clinical signs of nerve or cord compression in patients with spinal metastases presenting with neurologic symptoms, PV is contraindicated and surgery usually indicated. Surgery may require both anterior and posterior approaches to accomplish corpectomy and place instrumentation for spinal stabilization (29). Analysis of vertebral involvement may occasionally indicate that appropriate use of PV can reduce the amount of surgery needed. Percutaneous reinforcement of involved vertebra may eliminate the need for an anterior approach in some patients (11,12). With the anterior column support provided by PV, a posterior approach can be used for laminectomy to decompress the spinal cord and stabilize the spine with posterior instrumentation. For patients with a shortened expected life span, this less invasive procedure should provide palliative improvement and a shorter period of convalescence.

The main concern when planning PV and chemotherapy is the effect of the chemotherapy on platelets, coagulation factors, and immunization. When possible, PV should precede chemotherapy.

Other local percutaneous therapies for metastatic lesions may be used. Thermal ablation or direct injection of absolute ethanol may be used for small lesions (30). Intraarterial embolization may be used for large and hypervascularized tumors (31). Percutaneous vertebroplasty represents a direct percutaneous embolization of these hypervascular tumors (renal cell or thyroid metastases) and can be combined with transarterial embolization if the amount of cement injected does not fill the volume of the lesion. Percutaneous vertebroplasty must be used or combined with these treatments if structural reinforcement is to be achieved.

Image-guided radiofrequency ablation (RFA) can be a safe modality in the therapy for nonresectable spine tumors (32,33). Using combined multislice CT and fluoroscopic guidance, instrumentation can be precisely placed to cause a controlled ablation. Gronemeyer et al. (32) combined treatment of spinal metastasis with RFA heat ablation and PV with good results. In their experience, PV immediately after RFA during the same procedure is very painful for nonsedated patients and is best performed several days after radiofrequency ablation.

Percutaneous Vertebroplasty and Multiple Myeloma

Pathology and Patient Demographics

Multiple myeloma is a monoclonal proliferation of malignant plasma cells that usually affects the bone marrow (34). The peak incidence occurs during the sixth decade of life. The median survival time is 3 years. This disease is slightly more common in men than in women and affects 3 in 100,000 persons annually (34).

Excessive bone resorption due to an increase of proinflammatory cytokines is a characteristic feature of the disease (35–37). Diffuse osteo-

porosis and focal osteolytic lesions are thought to be potential causes of fractures in patients with multiple myeloma, and such fractures most frequently involve the spine (38–40). Indeed, vertebral compression fractures are present in 55% to 70% of patients with multiple myeloma and represent the initial clinical sign in 34% to 64% of such patients (41–44). Despite major improvements in chemotherapy, bone pain and widespread vertebral collapses are responsible for disability, respiratory restriction, and (sometimes) neurologic complications (45). All of these conditions decrease the quality of life for patients with multiple myeloma.

In approximately 5% of patients with plasma cell myeloma, solitary bone plasmacytoma represents the only disease feature. The diagnosis requires histologic evidence of a monoclonal plasma cell infiltrate in one bone lesion, absence of other bone lesions on skeletal radiographs, and lack of marrow plasmacytosis elsewhere (46). Two thirds of such patients develop multiple myeloma within 3 years after the discovery of a plasmacytoma; one third have no tumor progression for more than 10 years after discovery (46–51). Early progression most likely results from occult generalized disease that was not recognized at diagnosis. Magnetic resonance imaging, which is more sensitive than conventional radiography for the detection of myeloma lesions, may indicate additional foci that represent occult myeloma (46).

Technique

The procedure (guidance for needle positioning, needle route, etc.) for myelomatous vertebral lesions is not substantially different from that for other indications (5,12,52,53). The transpedicular approach, when possible, is preferred. However, it should be remembered that the distribution of cement and the risk of cement leaks depend on the radiologic appearance of the vertebral lesions.

Most of the vertebral collapses in patients with myeloma appear benign on radiographs and MR imaging with a distribution similar to that observed in osteoporotic fractures (36). When PV is performed for such collapses, the distribution of PMMA is frequently homogeneous in the vertebral body, and a single injection of cement may be sufficient (Figure 10.8). The risk of leaks of cement is small, especially if the cement injected is more viscous than that normally used for PV. Venous leaks are commonly observed if cement with a liquid consistency is injected into such lesions (13).

When a lytic lesion is demonstrated on conventional radiographs or CT scan, the degree of lesion filling is more varied and the risk of cement leakage is higher, possibly because of the different texture of this type of lesion. However, a better distribution of cement is usually obtained than in osteolytic metastases (Figure 10.9).

Solitary bone plasmacytoma frequently appears as an osteolytic but trabeculated lesion (54) with cortical osteolysis frequently present only in some places. The quality of the distribution of cement usually is intermediate between the two previously described vertebral lesions (Figure 10.10).

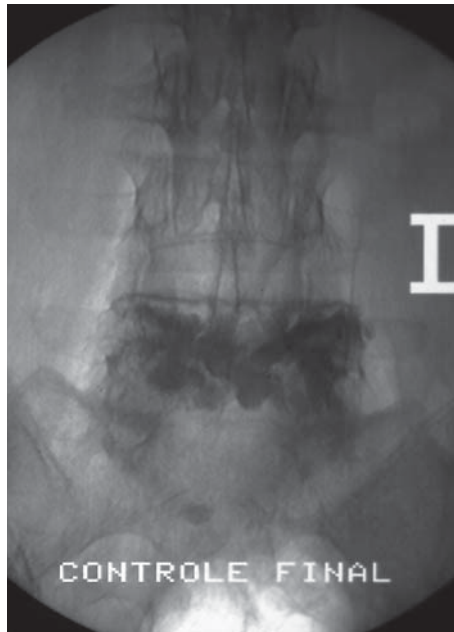


Figure 10.8. Homogeneous distribution of PMMA in the vertebral body of L5. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)



Figure 10.9. Three examples of inhomogeneous cement fill that may occur in myelomatous vertebral bodies. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

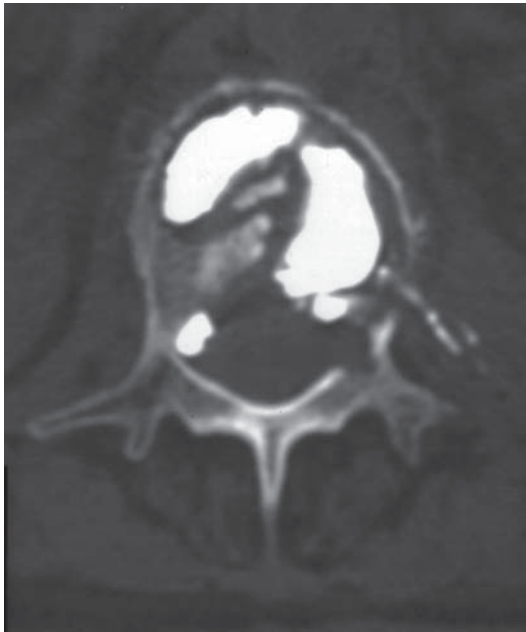


Figure 10.10. Solitary bone plasmacytoma. CT scan showing a small epidural leak. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

Results

As for metastases and osteoporotic vertebral collapses, pain relief after PV for myeloma occurs within hours or days (usually within 24 hours) after the procedure, sometimes after a transient worsening of pain. More than 70% of patients with multiple myeloma experience marked or complete pain relief (5,12,13,21,52,53).

Percutaneous Vertebroplasty and Other Procedures

Vertebrectomy is rarely performed for patients with myeloma because of the multifocal nature of the disease, but radiation therapy, in association with chemotherapy, plays a major role in the management of such patients. Even so, radiation and chemotherapy do not address several treatment issues completely. First, their rapid and highly effective therapeutic effect on epidural involvement and neurologic compression is well documented, and it is of great importance for patients at risk for spinal cord compression, which occurs in 10% to 15% of patients (55). Second, local radiation therapy is effective for solitary bone plasmacytoma because it may prevent tumor growth. However, patients with multiple marrow lesions respond less satisfactorily to local radiation therapy than do the patients with a single lesion, and either type of local tumor may recur. Third, radiation therapy has been associated with a reduced incidence of vertebral fractures and focal marrow lesions (56) and with bone healing, remodeling, and reossification resulting in local reinforcement (57). However,

the bone reconstruction is minimal and delayed (2 to 4 months after the start of irradiation) and sometimes preceded by transitory osteoporosis, which increases the risk of vertebral collapse and consequently of neural compression. Finally, radiation therapy usually results in partial or complete pain relief, with most patients experiencing some relief within 10 to 14 days, but some patients (5% to 10%) may experience insufficient pain relief and may be unable to tolerate additional radiation therapy.

Therefore, PV has an interesting place in the management of focal complicated myeloma lesions: It may provide rapid pain relief and vertebral stabilization when the lesion threatens the stability of the spine. Because such vertebral lesions are of clinical importance to the quality of life of patients with myeloma, PV may prevent some of the morbidity and mortality associated with the disease (45). However, because in this clinical setting PV is a palliative procedure that does not prevent tumor growth, it should be used in conjunction, whenever possible, with radiation and chemotherapy for patients with myeloma.

Percutaneous Vertebroplasty and Benign Lesions

Pathology and Patient Demographics

Vertebral hemangiomas are common abnormalities. They have been found in 10% of spines at autopsy (58) or incidentally discovered on imaging studies. In rare cases, they can be aggressive lesions in terms of clinical and/or radiographic findings.

From a clinical point of view, aggressive VHs can be differentiated as painful VHs and VHs with neurologic symptoms. The most frequent symptom is severe, mechanical back pain that increases with movement, even minimal movement such as shifting position in a chair. The tumor's progression is associated with deterioration in the quality of life. Neurologic signs can be related to nerve root and/or spinal cord compression by the VH invading the neural foramina or the epidural space. These neurologic signs can be acute or progressive.

Asymptomatic VHs can be diagnosed on plain films, CT scan, and/or MRI studies. Plain radiographs show localized and regular vertical striation of the vertebral body affected by the VH (Figure 10.11A). The diagnostic CT scan shows a loss of the trabecular bone and thickening of the remaining vertical osseous network. The hypodense areas that appear surrounding the trabeculae on CT are fatty tissue that has replaced degenerated VH (Figure 10.11B). The fatty component could be evidence of the nonprogressive nature of that part of the lesion (59). Magnetic resonance imaging shows a typical hypersignal on T1-weighted images induced by the fatty stroma of the lesion (Figure 10.11C). All these modalities show a well-demarcated lesion in part or all of the vertebral body, without involvement of the cortical bone. Most of these lesions (which can occur singly or at multiple levels) are asymptomatic and discovered only incidentally.

Aggressive VHs are characterized by the involvement of the whole vertebral body, location (frequently) in the thoracic area, an irregular

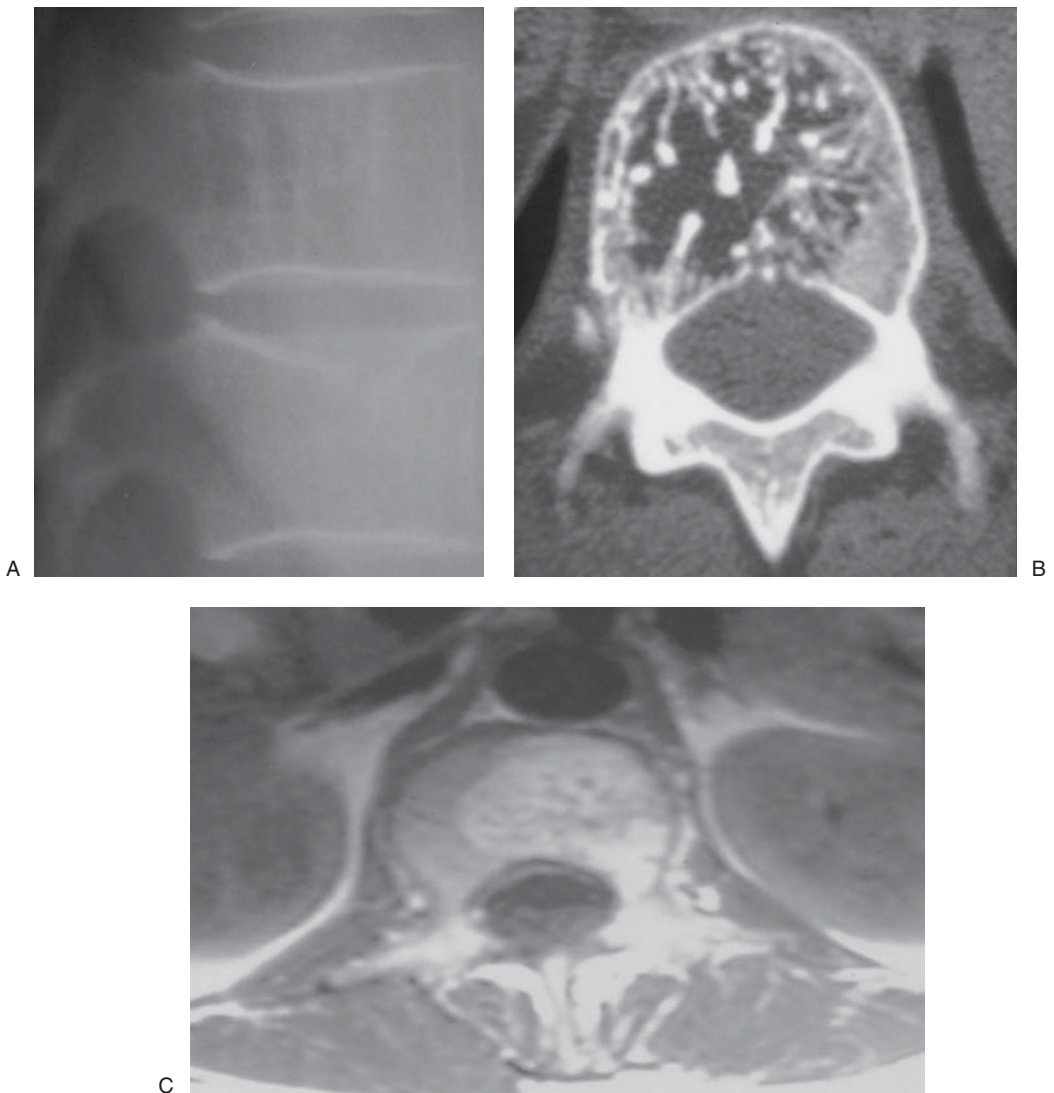


Figure 10.11. Nonaggressive VH. **(A)** Lateral radiograph showing localized and regular vertical striation of the vertebral body. **(B)** Axial CT scan showing loss of the trabecular bone and thickening of the remaining vertical osseous network, containing predominantly fatty stroma. **(C)** Axial T1-weighted MR image showing high signal intensity stroma related to fatty degeneration of a VH. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

honeycomb appearance of trabeculation, an expanded and poorly defined cortical bone, and swelling of the soft tissues (60). On CT scans (Figure 10.12A) and MR images (Figure 10.12B), there is little or none of the fatty component usually seen with nonaggressive VHs. Computed tomography and MR imaging provide the best delineation of the extension of the VH to the paravertebral tissues. The epidural extension is best seen after intravenous injection of contrast (Figure 10.12C). This epidural involvement can induce spinal cord and/or nerve root

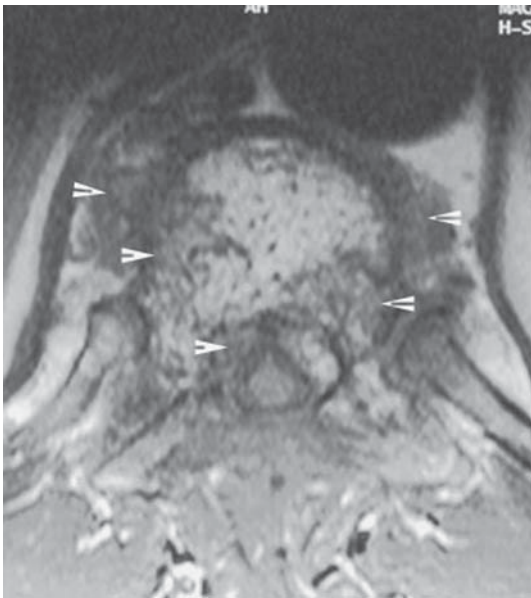
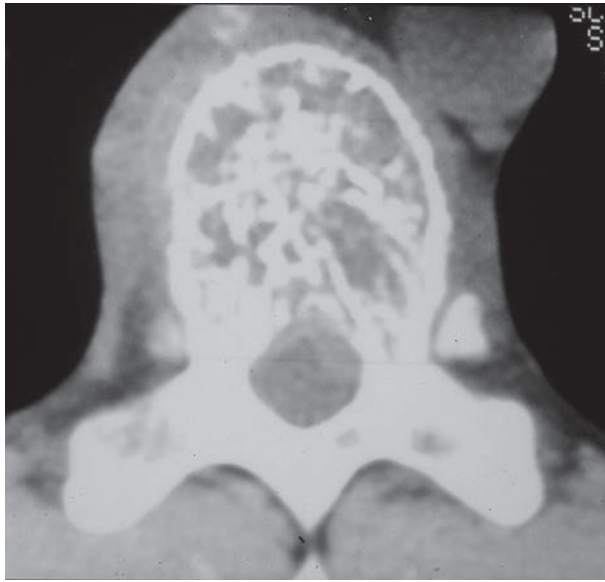


Figure 10.12. Aggressive VH. (A) Axial CT scan showing involvement of the whole vertebra with epidural and paravertebral extension. (B) Axial T1-weighted MR image showing epidural and paravertebral extension. The progressive parts of the lesion appear as an isosignal on noncontrast images (white arrowheads). (C) contrast-enhanced axial T1-weighted MR image showing hyperdense signal of the highly vascularized lesion. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

compression. Vertebral hemangiomas frequently extend to the posterior neural arch, involving the whole vertebra (Figure 10.12A).

Other signs of an aggressive VH include an increase in the size of the VH on two successive radiographic examinations; expansion of the cortical bone, or a periosteal osseous formation that induces a spinal canal stenosis; and a weakened vertebral body and possible vertebral collapse occurring spontaneously or secondary to low-energy trauma (Figure 10.13).

In most cases, VHs with radiographic signs of aggressiveness are symptomatic. Aggressive VHs can occur (singly or at multiple levels) in combination with the nonaggressive form.

Classification and Indications

Vertebral hemangiomas can be classified into one of four groups depending on their clinical and radiographic presentation (53): (1) asymptomatic VH without radiographic signs of aggressiveness (incidental discovery); (2) symptomatic (i.e., severe back pain) VH without radiographic signs of aggressiveness; (3) asymptomatic VH with radiographic signs of aggressiveness (incidental discovery); and (4) symptomatic VH with radiographic signs of aggressiveness. Group 4 can be divided into two subgroups: (a) VH with epidural extension and (b) VH without epidural extension, but inducing severe back pain.



Figure 10.13. Aggressive VH. This axial CT scan of a patient presenting with severe back pain after falling on her back showed a VCF (white arrow). (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

There are no indications for PV treatment for patients in group 1. For patients in group 2, PV is indicated because of severe back pain related to a VH, even in the absence of radiographic signs of aggressiveness. The indication is easier to confirm in the thoracic region when there is only an isolated VH to explain the back pain. It is often difficult to appreciate the role of such a VH in the cervical region and even more so at the lumbar region where associated degenerative disorders can induce the same pain.

Patients in group 3 require close monitoring with annual clinical and MR imaging examinations for progression of the VH. Percutaneous vertebroplasty is indicated only for patients for whom regular, long-term follow-up is not possible or for whom the VH becomes symptomatic or presents an evolution on successive radiographic studies.

Percutaneous vertebroplasty is indicated for all patients in group 4: The technique will vary depending on the progressive nature and severity of the neurologic signs. Patients with an acute myelopathy or cauda equina syndrome should be treated by a combination of PV and surgery (see "Technique," below). Patients presenting with progressive neurologic signs should be treated with PV and percutaneous injection of absolute ethanol (see "Technique," below). For a symptomatic patient with an aggressive VH but without epidural extension, PV alone is the treatment of choice.

Patient Evaluation

In general, patients present for evaluation for one of three reasons: (1) incidental imaging diagnosis of an asymptomatic VH, (2) severe back pain related to a VH, or (3) neurologic signs related to a VH. Evaluation of the patient should include all available clinical information. The clinical examination should elucidate focal pain or neurologic signs that correlate with the lesion in order for the lesion to be considered for PV. Pain should be documented with measurement instruments such as a visual analog scale or quality-of-life questionnaires (e.g., SF36) (61).

The pain described by the patient and detected on the clinical examination should be compared with the findings on plain radiographs, MR imaging, and CT. At the lumbar and cervical levels, particularly in patients with radiographically nonaggressive VH, the clinician should attempt to confirm the relationship between the pain and the VH, that is, exclude degenerative lesions as the possible origin of the pain, for PV to be indicated. The MR images and CT scans should be assessed to differentiate between aggressive and nonaggressive VH, to determine any extension of the VH into the epidural space and neural foramina, and to evaluate for compression of neural tissue.

Once the criteria for PV are met, the procedure should be completely discussed with the patient and his or her family. The discussion should include the potential benefits of PV and the risks associated with the procedure. Finally, the patient should undergo a preanesthetic evaluation, ECG, and laboratory screen (complete blood count/platelets,

electrolytes, prothrombin time, partial thromboplastin time, blood urea nitrogen/creatinine).

Technique

According to the clinical presentation and radiographic signs, PV can be performed with acrylic cement alone or a combined treatment of acrylic cement followed by injection of glue or absolute ethanol (3,53,62,63).

Percutaneous Vertebroplasty

When a VH presents without epidural involvement in patients complaining of pain, the treatment goals are to fill the defect (Figure 10.14), obtain a structural reinforcement of the vertebral body, and provide pain relief. The needle must be inserted into the anterior part of the VH by a transpedicular approach. If the VH involves only a part of the vertebral body, it is possible to fill the whole malformation with only one injection and a single puncture (Figure 10.15). If the whole vertebral body is involved, a bilateral transpedicular approach is usually required to fill the lesion.

Percutaneous Vertebroplasty with Complementary Injection of Ethanol

When a VH invades the anterior epidural space with no or only minor neurologic symptoms, a complementary injection of absolute ethanol

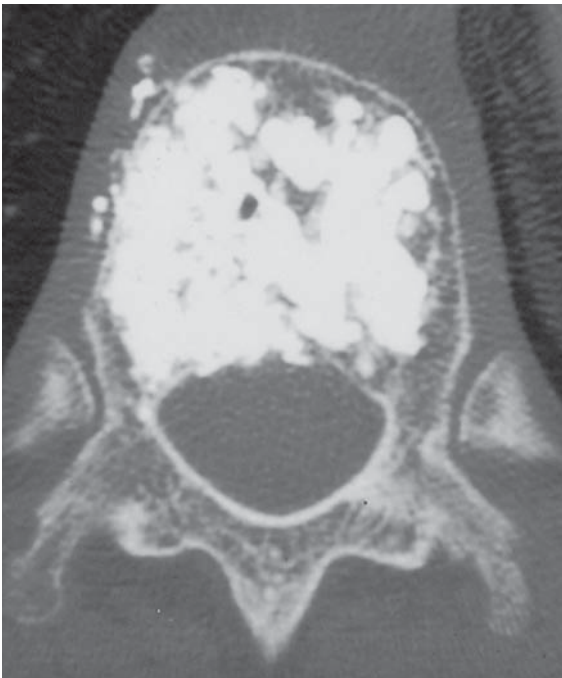


Figure 10.14. Axial CT scan after an injection of acrylic cement into a VH. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

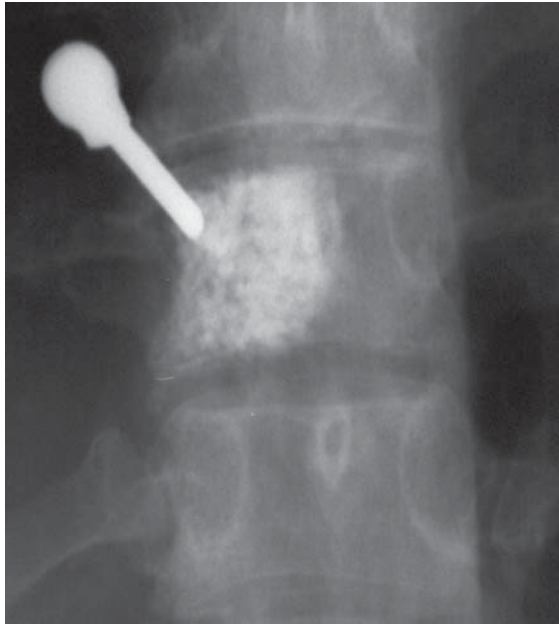


Figure 10.15. This AP radiograph showed a VH involving three quarters of the vertebral body, which was filled with acrylic cement using a unilateral transpedicular approach. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

may be used to sclerose the lesion completely. This procedure is accomplished in four steps. First, the affected vertebral body is injected with acrylic cement via a unilateral or bilateral transpedicular approach. Second, a site is found around or in the vertebral body that has not been injected with cement, and an 18-gauge needle is inserted into it (Figure 10.16). Third, the potential distribution of the sclerosing agent is checked by slow injection of 1 to 4 mL of contrast media; the quantity needed to inject the epidural component is noted and defines the amount to be used for the subsequent injection of ethanol. Fourth, the absolute ethanol, usually no more than 4 mL, is slowly injected. If the VH involves the posterior neural arch, it is possible in the same procedure to puncture that component using one or several needles and to obtain a complete sclerosis of the malformation, injecting no more than 1 mL of ethanol by each needle.

Heiss et al. (64) were the first to report the use of absolute ethanol (up to 50 mL) for the sclerosis of aggressive VH. However, they did not use an accompanying injection of acrylic cement. Two years later, they reported that two of seven patients had additional VCFs, presumably related to focal vertebral osteonecrosis secondary to the injection of a large amount of ethanol (40 and 50 mL, respectively) (65). Use of PV before the injection of ethanol prevents such a complication by providing structural reinforcement of the vertebral body and by decreas-

ing the amount of alcohol needed for sclerosing the VH (no more than 4 mL in our experience).

Percutaneous Vertebroplasty with Complementary Injection of Glue

In the presence of VH associated with an epidural component and acute clinical signs of compression of the spinal cord or cauda equina, the goal of PV is to reinforce the vertebral body and to make laminectomy and surgical excision of the epidural hemangioma easier by completely devascularizing the VH. This goal is accomplished by combining a PV procedure accompanied by an injection of N-butyl cyanoacrylate glue (opacified) on day 1 and surgery on day 2 (53,63).

The PV with glue procedure has five steps. First, the vertebral body invaded by the VH (Figure 10.17A) is injected with acrylic cement via a unilateral or bilateral transpedicular approach. Second, an 18-gauge needle is inserted into the remaining VH that has not been injected with acrylic cement (Figure 10.17B). Third, the predictable distribution of the glue is checked by the slow injection of up to 4 mL of contrast media. Fourth, after having carefully washed the needle with a nonionic solution (glucose serum) to avoid the early polymerization of the glue in the needle, 3 to 5 mL of the glue mixture is slowly injected under fluoroscopic control to fill the compressive epidural component of the lesion (Figure 10.17C). Fifth (if necessary), the percutaneous embolization of the remaining component of the VH is completed by injecting glue via one or several needles inserted into the posterior neural arch (Figure 10.17D,E). Laminectomy and surgical excision of the epidural component of the VH (simplified by the PV) is usually planned for the following day (Figure 10.17F). (Editor's note: Thus far this aggressive therapy has only been reported in France).

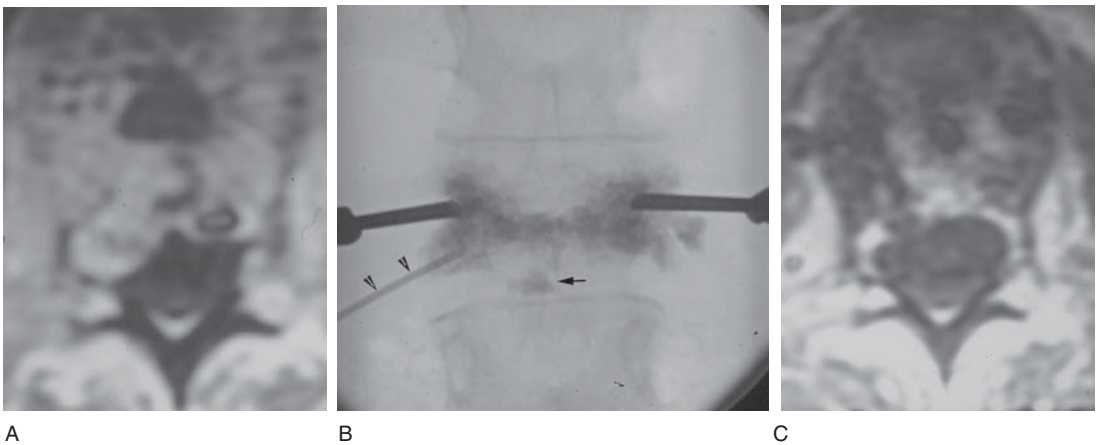


Figure 10.16. This patient had an aggressive VH with an epidural component. **(A)** Preoperative T1-weighted MR image. **(B)** Anteroposterior view showing injection of the vertebral body part of the VH with acrylic cement. An 18-gauge needle was inserted into a part of the vertebral body that was not injected with cement (black arrowheads), and alcohol was injected into the remaining part of the VH. Note the leakage of cement into the adjacent discs (black arrow). **(C)** Resolution of the epidural VH 3 months after PV. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

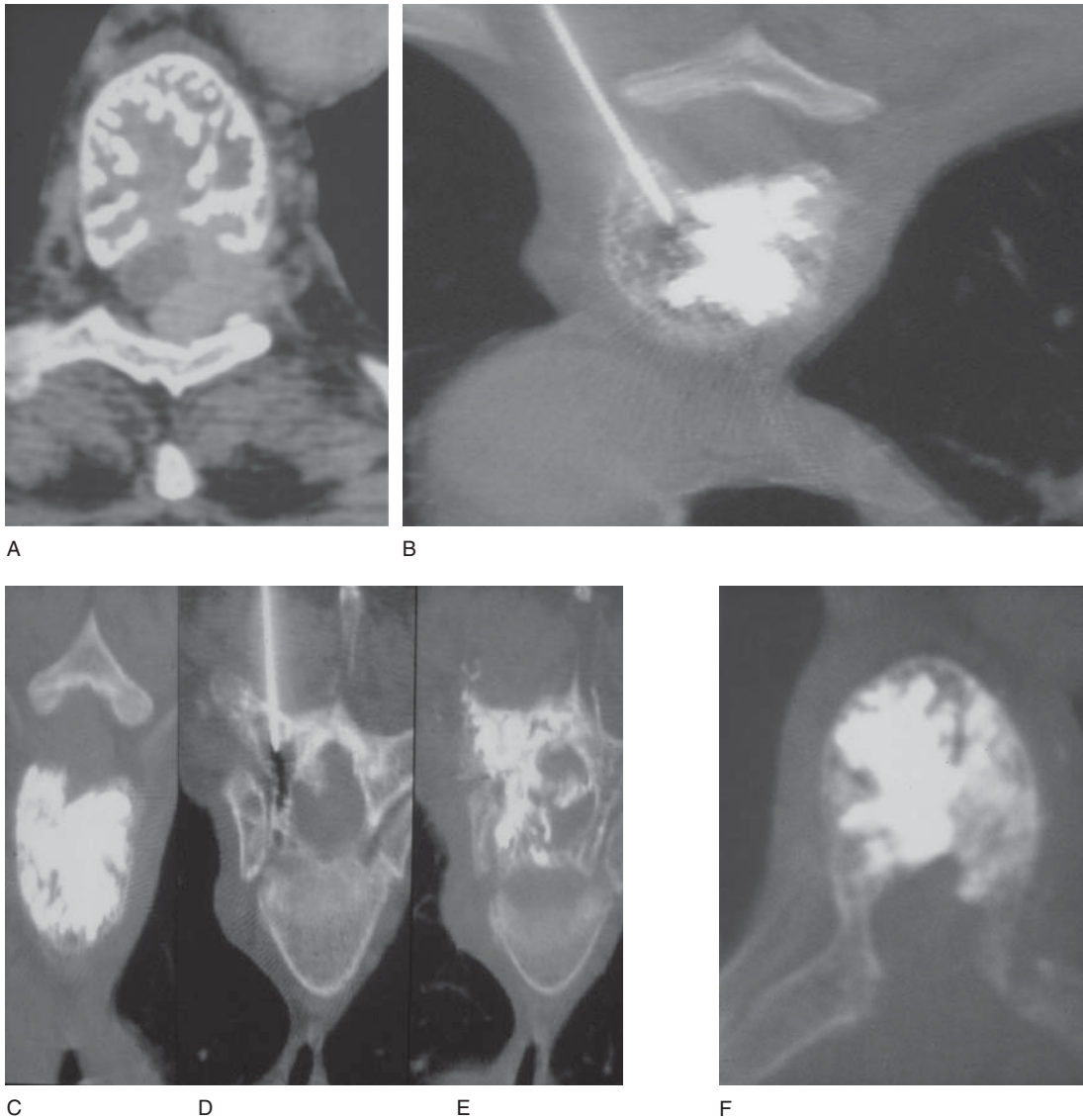


Figure 10.17. This patient presented with an acute spinal cord compression related to an aggressive thoracic VH. **(A)** Axial CT scan before PV. **(B)** Axial CT scan of the injection of acrylic cement into three quarters of the vertebral body part affected by the VH. Under CT guidance, an 18-gauge needle was inserted into the portion of the vertebral body lesion not injected with cement. **(C)** Axial CT scan showing the distribution of glue in the remaining part of the vertebral body but without injection of the epidural component. **(D)** Axial CT scan showing the insertion under CT guidance of an 18-gauge needle into the posterior neural arch invaded by the VH. **(E)** Axial CT scan showing the distribution of the glue into the posterior neural arch and the epidural component of the VH. **(F)** Axial CT scan after surgical laminectomy and excision of the epidural VH. (From J.M. Mathis, H. Deramond, and S.M. Belkoff [eds], *Percutaneous Vertebroplasty*. New York: Springer, 2002, with permission.)

Alcohol or glue? In our experience, we think it might be possible to avoid surgery by using the PV with ethanol procedure, and using a sclerosing agent could allow a progressive and complete improvement of the neurologic signs and avoid the need for surgery.

Percutaneous Vertebroplasty with Computed Tomography

Gangi et al. (66) described the technique for PV using CT: The needle is placed precisely and safely under CT guidance, and the cement injection is performed under real-time fluoroscopic control. Most of the time, a good biplane fluoroscopy unit allows a fast and safe procedure for PV. However, when a complementary injection of absolute ethanol or glue is needed for the treatment of VHs, checking the distribution of the acrylic cement into the vertebral body and setting the 18-gauge needle into a part of the vertebral body not injected with cement or into the posterior neural arch requires the use of CT (Figure 10.17).

Results

In the first published cases, PV was used to treat VHs (3,62). Of those first 11 patients, 10 had complete relief of pain after the PV procedure. The literature documents substantial pain reduction in more than 80% of patients whose VHs were treated by PV (53,64,67,68).

Deramond et al. (53) have treated 61 patients with symptomatic VH. With a long-term follow-up period (up to 15 years), structural reinforcement was obtained in all patients, there was no change in the shape of the vertebral body, and relief of severe back pain was obtained by more than 90%. Only once did evolution of the epidural part of the VH occur. In that case, PV was conducted at the C2 level, and acrylic cement alone (with no sclerosing agent) was injected into the vertebral body. Early results were good, but after 3 years the epidural component suddenly increased, and the growth continued despite radiation therapy. The patient died 4 years later from neurologic complications (56).

A review of the results in terms of the classification groups described above shows the following: group 2 (38 patients; treated with PV), complete pain relief in more than 90% (35 patients), with no recurrence of the lesion; group 4a-i (12 patients; all treated with PV, five also treated with ethanol injection), all had cessation of progressive neurologic signs, no evolution (3 to 7 years of follow up) or recurrence of the epidural component (except for the first patient already described), and the epidural component disappeared in two of the five treated with ethanol; group 4a-ii (four patients; treated with PV, glue, and laminectomy), no evolution (3 to 7 years of follow up) or recurrence of epidural component, and disappearance of acute neurologic signs; group 4b (seven patients; treated with PV), complete relief of back pain in all patients and no change in the lesion.

Side Effects and Complications

In the first group of 54 patients with VH treated by PV, there were only two complications: both were intercostal neuralgias that healed after

local injection with steroids and anesthetic (53). These complications were related to leakage of cement into foraminal veins and occurred among the first patients treated. One patient had been injected with cement having a low radio-opacity; the method was subsequently improved by adding tantalum powder (62). In the second patient, intercostal neuralgia was related to a leakage of cement along the track of a needle inserted via an intercostal posterolateral approach (Craig technique) (69), which irritated the adjacent nerve root. The transpedicular approach avoids this complication.

Percutaneous Vertebroplasty and Other Therapies

Radiation Therapy

Radiation therapy alone with fractionated doses under 4,000 cGy has been used to treat VH. (70,71). With these low doses, the risk of complication is low, but the rate of recurrence is approximately 50% (72). These considerations, combined with the efficacy of PV, have led us to believe that radiation therapy is no longer indicated for the treatment of VHs.

Laminectomy and Surgical Excision

Laminectomy and surgical excision of the epidural component of the lesion was the classic treatment for VH with neurologic signs (73,74). However, this surgery is often difficult because of the vascular nature of the lesion. In our experience, PV before surgery makes the excision easier and less risky. In addition, we think that for most patients with acute neurologic signs, PV combined with ethanol injection may obviate surgery.

Transarterial Embolization

Transarterial embolization (75) provides excellent short-term results for aggressive VHs. However, evolution and recurrence of the VH is frequent. It is the classic treatment before surgery, with the goal of decreasing preoperative bleeding, but it has variable efficacy. Moreover, transarterial embolization can be impossible or dangerous, with the risk of spinal cord infarction when a common artery supplies the VH and the spinal cord. In the early days of PV treatment, embolization was performed before PV (62,63), but it quickly became evident that that procedure was unnecessary because PV provides a far more efficient in situ filling of the vascular malformation.

References

1. Parlier-Cuau C, Champsaur P, Nizard R, et al. Percutaneous removal of osteoid osteoma. *Radiol Clin North Am* 1998; 36(3):559-566.
2. Gladden ML, Jr, Gillingham BL, Hennrikus W, et al. Aneurysmal bone cyst of the first cervical vertebrae in a child treated with percutaneous intralesional injection of calcitonin and methylprednisolone. A case report. *Spine* 2000; 25(4):527-530.
3. Galibert P, Deramond H, Rosat P, et al. [Preliminary note on the treatment of vertebral angioma by percutaneous acrylic vertebroplasty.] *Neurochirurgie* 1987; 33(2):166-168.

4. Cardon T, Hachulla E, Flipo RM, et al. Percutaneous vertebroplasty with acrylic cement in the treatment of a Langerhans cell vertebral histiocytosis. *Clin Rheumatol* 1994; 13(3):518–521.
5. Cotten A, Boutry N, Cortet B, et al. Percutaneous vertebroplasty: state of the art. *RadioGraphics* 1998; 18(2):311–323.
6. Abrams HL, Spiro R, Goldstein N. Metastases in carcinoma. Analysis of 1000 autopsied cases. *Cancer* 1950; 3:74–85.
7. Malawer MM, Delandy TF. Treatment of metastatic cancer to bone. In *Cancer: Principles and Practice of Oncology*, 8th Ed. VT DeVita, S Hellman, SA Rosenberg (eds). Philadelphia: JB Lippincott Co, 1989:2298–2317.
8. Bontoux D, Azais I. Cancer secondaire des os. Clinique et epidemiologie. In *Cancer Secondaire des Os*. D Bontoux, M Alcalay (eds). Paris: Expansion Scientifique Francaise, 1997:19–27.
9. Tubiana-Hulin M. Incidence, prevalence and distribution of bone metastases. *Bone* 1991; 12(Suppl 1):S9–S10.
10. Tatsui H, Onomura T, Morishita S, et al. Survival rates of patients with metastatic spinal cancer after scintigraphic detection of abnormal radioactive accumulation. *Spine* 1996; 21(18):2143–2148.
11. Deramond H, Depriester C, Toussaint P. [Vertebroplasty and percutaneous interventional radiology in bone metastases: techniques, indications, contra-indications.] *Bull Cancer Radiother* 1996; 83(4):277–282.
12. Weill A, Chiras J, Simon JM, et al. Spinal metastases: indications for and results of percutaneous injection of acrylic surgical cement. *Radiology* 1996; 199(1):241–247.
13. Cotten A, Dewatre F, Cortet B, et al. Percutaneous vertebroplasty for osteolytic metastases and myeloma: effects of the percentage of lesion filling and the leakage of methyl methacrylate at clinical follow-up. *Radiology* 1996; 200(2):525–530.
14. Shimony JS, Gilula LA, Zeller AJ, Brown DB. Percutaneous vertebroplasty for malignant compression fractures with epidural involvement. *Radiology* 2004; 232(3):846–853.
15. Belkoff SM, Mathis JM, Jasper LE, et al. The biomechanics of vertebroplasty: the effect of cement volume on mechanical behavior. *Spine* 2001; 26(14): 1537–1541.
16. Martin JB, Wetzel SG, Seium Y, et al. Percutaneous vertebroplasty in metastatic disease: transpedicular access and treatment of lysed pedicles. Initial experience. *Radiology* 2003; 229:593–597.
17. Lapras C, Mottolèse C, Deruty R, et al. [Percutaneous injection of methylmethacrylate in osteoporosis and severe vertebral osteolysis (Galibert's technic).] *Ann Chir* 1989; 43(5):371–376.
18. Kaemmerlen P, Thiesse P, Bouvard H, et al. [Percutaneous vertebroplasty in the treatment of metastases. Technic and results.] *J Radiol* 1989; 70(10):557–562.
19. Kaemmerlen P, Thiesse P, Jonas P, et al. Percutaneous injection of orthopedic cement in metastatic vertebral lesions [letter]. *N Engl J Med* 1989; 321(2):121.
20. Cortet B, Cotten A, Boutry N, et al. Percutaneous vertebroplasty in patients with osteolytic metastases or multiple myeloma [see comments]. *Rev Rhum Engl Ed* 1997; 64(3):177–183.
21. Fourney DR, Schomer DF, Nader R, et al. Percutaneous vertebroplasty and kyphoplasty for painful vertebral body fractures in cancer patients. *J Neurosurg Spine* 2003; 98(1):21–30.
22. Deramond H, Wright NT, Belkoff SM. Temperature elevation caused by bone cement polymerization during vertebroplasty. *Bone* 1999; 25(Suppl 2):17S–21S.

23. San Millan RD, Burkhardt K, Jean B, et al. Pathology findings with acrylic implants. *Bone* 1999; 25(Suppl 2):85S–90S.
24. Chiras J, Depriester C, Weill A, et al. [Percutaneous vertebral surgery. Techniques and indications.] *J Neuroradiol* 1997; 24(1):45–59.
25. Shepherd S. Radiotherapy and the management of metastatic bone pain. *Clin Radiol* 1988; 39(5):547–550.
26. Salazar OM, Rubin P, Hendrickson FR, et al. Single-dose half-body irradiation for the palliation of multiple bone metastases from solid tumors: a preliminary report. *Int J Radiat Oncol Biol Phys* 1981; 7:773–781.
27. Chow E, Holden L, Danjoux C, et al. Successful salvage using percutaneous vertebroplasty in cancer patients with painful spinal metastases or osteoporotic compression fractures. *Radiother Oncol* 2004; 70(3):265–267.
28. Murray JA, Bruels MC, Lindberg RD. Irradiation of polymethylmethacrylate. In vitro gamma radiation effect. *J Bone Joint Surg* 1974; 56A(2):311–312.
29. Riley LH, Frassica DA, Kostuik JP, et al. Metastatic disease to the spine: diagnosis and treatment. *Instr Course Lect* 2000; 49:471–477.
30. Gangi A, Guth S, Dietemann JL, et al. Interventional musculoskeletal procedures. *RadioGraphics* 2001; 21(2):520.
31. Meder JF, Reizine D, Chiras J, et al. Apport de l'artériographie dans le diagnostic et la traitement des tumeurs du rachis. *Rachis* 1992; 4(4):215–228.
32. Gronemeyer DH, Schirp S, Gevargez A. Image-guided radiofrequency ablation of spinal tumors: preliminary experience with an expandable array electrode. *Cancer J* 2002; 8(1):33–39.
33. Schaefer O, Lohrmann C, Markmiller M, et al. Technical innovation. Combined treatment of a spinal metastasis with radiofrequency heat ablation and vertebroplasty. *Am J Roentgenol* 2003; 180(4):1075–1077.
34. Longo DL. Plasma cell disorders. In *Harrison's Principles of Internal medicine*, 12th Ed. JD Wilson, E Braunwald, KJ Isselbacher, et al (eds). New York: McGraw-Hill, 1991:1412–1416.
35. Bataille R, Chappard D, Klein B. Mechanisms of bone lesions in multiple myeloma. *Hematol Oncol Clin North Am* 1992; 6(2):285–295.
36. Lecouvet FE, Van de Berg BC, Maldague BE, et al. Vertebral compression fractures in multiple myeloma. Part I. Distribution and appearance at MR imaging [see comments]. *Radiology* 1997; 204(1):195–199.
37. Salmon SE, Cassady JR. Plasma cell neoplasms. In *Cancer: Principles and Practice of Oncology*, 4th Ed. VT DeVita, S Hellman, SA Rosenberg (eds). Philadelphia: JB Lippincott Co, 1993:1984–2025.
38. de Gramont A, Benitez O, Brissaud P, et al. Quantification of bone lytic lesions and prognosis in myelomatosis. *Scand J Haematol* 1985; 34(1):78–82.
39. Kanis JA, McCloskey EV. Disorders of calcium metabolism and their management. In *Myeloma: Biology and Management*. JS Malpas, DE Bergsagel, RA Kyle (eds). New York: Oxford University Press, 1995: 375–396.
40. Kapadia SB. Multiple myeloma: a clinicopathologic study of 62 consecutively autopsied cases. *Medicine (Baltimore)* 1980; 59(5):380–392.
41. Kyle RA. Multiple myeloma: review of 869 cases. *Mayo Clin Proc* 1975; 50(1):29–40.
42. Carson CP, Ackerman LV, Maltby JD. Plasma cell myeloma: a clinical, pathologic, and roentgenologic review of 90 cases. *Am J Clin Pathol* 1955; 25:849–888.
43. Riccardi A, Gobbi PG, Ucci G, et al. Changing clinical presentation of multiple myeloma. *Eur J Cancer* 1991; 27(11):1401–1405.
44. Spiess JL, Adelstein DJ, Hines JD. Multiple myeloma presenting with spinal cord compression. *Oncology* 1988; 45(2):88–92.

45. Lecouvet FE, Malghem J, Michaux L, et al. Vertebral compression fractures in multiple myeloma. Part II. Assessment of fracture risk with MR imaging of spinal bone marrow [see comments]. *Radiology* 1997; 204(1):201–205.
46. Mouloupoulos LA, Dimopoulos MA, Weber D, et al. Magnetic resonance imaging in the staging of solitary plasmacytoma of bone. *J Clin Oncol* 1993; 11(7):1311–1315.
47. Knowling MA, Harwood AR, Bergsagel DE. Comparison of extramedullary plasmacytomas with solitary and multiple plasma cell tumors of bone. *J Clin Oncol* 1983; 1(4):255–262.
48. Chak LY, Cox RS, Bostwick DG, et al. Solitary plasmacytoma of bone: treatment, progression, and survival. *J Clin Oncol* 1987; 5(11):1811–1815.
49. Frassica DA, Frassica FJ, Schray ME, et al. Solitary plasmacytoma of bone: Mayo Clinic experience. *Int J Radiat Oncol Biol Phys* 1989; 16(1):43–48.
50. Dimopoulos MA, Goldstein J, Fuller L, et al. Curability of solitary bone plasmacytoma. *J Clin Oncol* 1992; 10(4):587–590.
51. Holland J, Trenkner DA, Wasserman TH, et al. Plasmacytoma. Treatment results and conversion to myeloma. *Cancer* 1992; 69(6):1513–1517.
52. Murphy KJ, Deramond H. Percutaneous vertebroplasty in benign and malignant disease. *Neuroimaging Clin North Am* 2000; 10(3):535–545.
53. Deramond H, Depriester C, Galibert P, et al. Percutaneous vertebroplasty with polymethylmethacrylate. Technique, indications, and results. *Radiol Clin North Am* 1998; 36(3):533–546.
54. Huvos HG. Multiple myeloma including solitary osseous myeloma. In *Bone Tumors: Diagnosis, Treatment, and Prognosis*. Philadelphia: WB Saunders Co, 1992:653–676.
55. Plowman PN. Radiotherapy of myeloma. In *Myeloma: Biology and Management*. JS Malpas, DE Bergsagel, RA Kyle (eds). New York: Oxford University Press, 1995:314–321.
56. Lecouvet F, Richard F, Vande Berg B, et al. Long-term effects of localized spinal radiation therapy on vertebral fractures and focal lesions appearance in patients with multiple myeloma. *Br J Haematol* 1997; 96(4):743–745.
57. Hoskin PJ. Radiotherapy in the management of bone pain. *Clin Orthop* 1995; 312:105–119.
58. Schmorl G, Junghans H. *The Human Spine in Health and Disease*, 2nd Ed. EF Besemann (ed, tr). New York: Grune & Stratton, 1971.
59. Laredo JD, Assouline E, Gelbert F, et al. Vertebral hemangiomas: fat content as a sign of aggressiveness. *Radiology* 1990; 177(2):467–472.
60. Laredo JD, Reizine D, Bard M, et al. Vertebral hemangiomas: radiologic evaluation. *Radiology* 1986; 161(1):183–189.
61. Ware JE, Jr., Snow KK, Kosinski M, et al. *SF-36 Health Survey. Manual and Interpretation Guide*. Boston: The Health Institute, 1993.
62. Deramond H, Darrason R, Galibert P. [Percutaneous vertebroplasty with acrylic cement in the treatment of aggressive spinal angiomas.] *Rachis* 1989; 1(2):143–153.
63. Cotten A, Deramond H, Cortet B, et al. Preoperative percutaneous injection of methyl methacrylate and N-butyl cyanoacrylate in vertebral hemangiomas. *Am J Neuroradiol* 1996; 17(1):137–142.
64. Heiss JD, Doppman JL, Oldfield EH. Brief report: relief of spinal cord compression from vertebral hemangioma by intralesional injection of absolute ethanol [see comments]. *N Engl J Med* 1994; 331(8):508–511.
65. Heiss JD, Doppman JL, Oldfield EH. Treatment of vertebral hemangioma by intralesional injection of absolute ethanol [letter; comment]. *N Engl J Med* 1996; 334(20):1340.

66. Gangi A, Kastler BA, Dietemann JL. Percutaneous vertebroplasty guided by a combination of CT and fluoroscopy. *Am J Neuroradiol* 1994; 15(1): 83–86.
67. Ide C, Gangi A, Rimmelin A, et al. Vertebral haemangiomas with spinal cord compression: the place of preoperative percutaneous vertebroplasty with methyl methacrylate. *Neuroradiology* 1996; 38(6):585–589.
68. Martin JB, Jean B, Sugiu K, et al. Vertebroplasty: clinical experience and follow-up results. *Bone* 1999; 25(Suppl 2):11S–15S.
69. Craig FS. Vertebral-body biopsy. *J Bone Joint Surg* 1956; 38A(1):93–102.
70. Yang ZY, Zhang LJ, Chen ZX, et al. Hemangioma of the vertebral column. A report on twenty-three patients with special reference to functional recovery after radiation therapy. *Acta Radiol Oncol* 1985; 24(2):129–132.
71. Pavlovitch JM, Nguyen JP, Djindjian M, et al. Radiotherapy of compressive vertebral hemangiomas. *Neurochirurgie* 1989; 35:296–298.
72. Nguyen JP, Djindjian M, Pavlovitch JM, et al. Vertebral hemangiomas with neurologic symptoms. Treatment. Results of the “Societe Francaise de Neuro-Chirurgie” series. *Neurochirurgie* 1989; 35:299–303.
73. Nguyen JP, Djindjian M, Gaston A, et al. Vertebral hemangiomas presenting with neurologic symptoms. *Surg Neurol* 1987; 27(4):391–397.
74. Nguyen JP, Djindjian M, Badiane S. Vertebral hemangiomas with neurologic symptoms. Clinical presentation. Results of the “Societe Francaise de Neurochirurgie” series. *Neurochirurgie* 1989; 35:270–274.
75. Picard L, Bracard S, Roland J, et al. [Embolization of vertebral hemangioma. Technic-indications-results.] Embolisation des hemangiomes vertebraux. Technique-indications-resultats. *Neurochirurgie* 1989; 35(5):289–293.