Cervical myelopathies due to a variety of spondylopathic changes are being recognized at an ever increasing rate in large breeds of dogs, probably as a direct result of increased diagnostic awareness stemming from the great number of recent publications on the subject. The as yet unexplained etiology and pathogenesis, assortment of breeds affected, variances in neurologic dysfunction, and seemingly innumerable types of spondylopathies have resulted in many names being given to this syndrome. Attempts at molding the syndrome into the human or equine counterpart have exacerbated the problem. In our attempts to clarify the disorder by each author naming it personally, we have succeeded in muddying the waters further. The original terminology of canine wobbler syndrome was based on a graphic description of the neurologic signs with no claim as to the etiology and pathogenesis and should perhaps have been retained.

The syndrome of cervical spinal cord compression due to abnormalities of the caudal cervical vertebrae, their articulations, or both, in large breeds of dogs has been referred to as progressive cervical spinal cord compression, cervical spondylosis, cervical spondylolisthesis, wobblers, caudal cervical subluxation, cervical vertebral instability, Great Dane ataxia, cervical spondylopathy, caudal cervical spondylopathy, caudal cervical vertebral malformation-malarticulation, cervical vertebral stenosis, cervical myelopathy, midcervical spondylolisthesis, cervical spondylotic myelopathy, caudal cervical spondylomyelopathy, and, most recently, dynamic compression of the cervical spinal cord. Most of these are accurate terms for the one particular type of spondylopathy that the author is describing.

Far more important than the long overdue housekeeping of our medical terminology is the impact of the numerous names for the same disorder on the selection of rational therapy. No one medical or surgical regime is appropriate for all dogs affected with this syndrome. Effective management requires a basic understanding of the pathophysiology and the selection, modification, and individualization of previously described medical and surgical techniques. In spite of our terminology, not all patients present with instability or with developmental stenosis or with true spondylosis or even with concise, localized, singular lesions. Therefore, the literature regarding the efficacy of various therapeutic regimes must be reviewed with an eye
toward the specific spondylopathy in the individual case. Only then can the surgeon rationally and logically select the most appropriate techniques as dictated by the specific lesions in the particular patient.

**Review of the Literature**

In the horse, "wobbles" was first described in 1939 by Dimock and Errington. The cervical vertebral malformations and malarticulations responsible for the ataxia and paresis were later reported by Jones and co-workers, Steel and co-workers, Rooney, Fraser and Palmer, and Prickett. In 1967, the first report of similar cervical vertebral deformation as the cause of spinal cord compression in young male basset hounds was published by Palmer and Wallace. Morgani and Geary described radiographic and postmortem pathologic changes in the cervical vertebrae of dogs, with some comment as to the clinical significance of the lesions. The typical clinical signs of neurologic dysfunction in the dog were first described by LaCroix in 1970 and deLahunta in 1971. In the early cases identified, the prominent pathology appeared to be spondylolisthesis. Subsequently, numerous types of spondylopathies of the caudal cervical vertebrae that resulted in similar neurologic signs of cervical myelopathy were reported. In the 1970s significant effort was devoted to further description of the vertebral column and central nervous system lesions in both the horse and the dog by a number of authors.

Since the first report of surgical intervention for this disorder in the dog by Gage in 1972, numerous therapeutic regimes, frequently based on the author's experience with a single particular type of spondylopathy or even a single case experience, have been presented. Such presentations have resulted in conflicting reports of the most effective technique or techniques owing to the many variations in the types, locations, and severities of the spondylopathies that were subjected to medical or surgical intervention.

**Incidence**

Although reported in various breeds, the disorder has been confirmed by neuropathology most commonly in Great Danes and Doberman pinschers. Originally, the syndrome seemed to be a problem principally of young growing Great Danes and of adult Doberman pinschers. As more data accumulated, the difference in age of onset between these two most commonly affected breeds became less significant, although the average age of onset in Great Danes still remained less than that in Doberman pinschers, possibly as a result of genetic induced differences in life span. Males have been affected more frequently, in a ratio of approximately 2:1.

**Etiology and Pathogenesis**

Although similar vertebral malformations have been reported in other breeds of dogs, the disease appears to be most common in the Great Dane and Doberman pinscher. Many clinical and pathologic features of this syndrome resemble those observed in the horse. The pathogenesis as well as the cause of the syndrome in these two species may be similar. The neurologic dysfunction is due to spinal cord compression from any of the various malformations or malarticulations, or combinations thereof, all of which result in deformity of the vertebral canal. The exact cause of the syndrome is still uncertain, but studies of the various anomalies, their progression, and their equally important sequelae permit reasonable speculation on the pathogenesis.

Clinical and experimental evidence suggests a role of nutrition as well as genetics in the development of this syndrome. The previously cited over-nutrition study involving paired Great Dane littermates supported a role for both. Clinical evidence in this study of excessive feeding and supplementation of some affected dogs and closely related affected dogs from different litters also supports this hypothesis. In addition, breeding affected dogs resulted in a high incidence of this syndrome in the progeny. Rapid growth, which has also been suggested as a cause, may be related to both over-nutrition and genetics. The theory of abnormal vertebral growth due to the disproportion between head and neck size, although feasible in the Great Dane, is difficult to comprehend in the Doberman. Trauma has been ruled out in most reported cases. The determination of predisposition to cervical myelopathy due to a congenital spinal canal stenosis, as occurs in humans, may also
support the theory of inheritance but awaits further confirmation, as does the theory of congenital ligamentous laxity of the vertebral column.\(^{(62)}\)

Osteoarthrosis of the synovial joints is a frequent finding in dogs with caudal cervical instability or ankylosis secondary to malarticulation with instability. This osteoarthrosis may be due to abnormal stresses that result from the abnormal mobility of the synovial joints. The stresses cause an uneven pressure on the articular hyaline cartilage, with loss of the normal pumping action necessary for proper nutrition of the cartilage by synovial fluid. Fibrillation of the cartilage may occur, with subsequent separation of the cartilage from the underlying bone. This results in increased vascularity of the subchondral bone, with eburnation and the formation of marginal osteophytes and osteocartilaginous joint mice.

Even without instability, osteoarthrosis may occur subsequent to abnormal stresses within synovial joints. At surgery and postmortem examination of dogs in various studies as well as in the over-nutrition study, incomplete cartilage coverage and asymmetry of the articular processes was found. The asymmetry has also been observed in some clinically normal dogs. Thus, even without instability, these malformations can result in altered mechanical stresses on the joint cartilage, with subsequent degenerative joint disease. This degenerative joint disease, which may progress to ankylosis, may produce direct or indirect spinal cord compression owing to deformity of the vertebral canal or to attenuating lesions that result from other structures that are altered by the abnormal mobility of the primarily affected articulations.

In the over-nutrition study, the stenotic cranial orifice of a vertebral foramen, often with asymmetry of the decreased dorsoventral diameter, was thought to occur as a result of hypercalcitonin-induced retardation of osteocytic osteolysis, preventing normal recession of the surfaces of the foramen.\(^{(23)}\) Another plausible explanation for the stenotic cranial orifice would be that it results from remodeling of the vertebra due to stresses on the cervical vertebral column or from altered spinal biomechanics.\(^{(62)}\) Mason's work tends to support the hypothesis that early instability may lead to the osseous and ligamentous changes seen in later life.\(^{(34)}\) Wright's studies, supported by the work of Slijper,\(^{(52)}\) also suggest that an alteration of the normal balance of supporting elements of the cervical vertebral column while the animal is still growing may affect the ultimate size and shape of the vertebrae.\(^{(56-70)}\) This concept is further supported by studies on scoliosis in children following muscular paralysis in which spinal curvature occurs as a result of the imbalance of muscular forces on opposite sides of the vertebrae.\(^{(63)}\) Nisbet and Renwick made similar observations in support of this theory during their work on muscular dystrophy in newborn lambs with what appeared to be secondary vertebral deformations due to abnormal stresses.\(^{(37)}\)

Stresses on the pedicles, such as those resulting from an abnormal symphyseal joint (e.g., degenerated intervertebral disk) may cause stress fractures or gradual remodeling of the pedicles owing to pressure transmitted through the superimposed articular processes. The articular processes, and thus the pedicles, would be subjected to excessive forces. The caudal articular processes of the cranial vertebra would prevent dorsal displacement of the caudal vertebra by exerting ventrally directed pressure on its cranial articular processes. The end result would appear as decreased height of the cranial aspects of the pedicles and, thus, decreased dorsoventral diameter of the cranial vertebral orifice.\(^{(62)}\)

Stenosis at the cranial orifice may also be due to hyperplasia of the interarcuate ligament, joint capsules, dorsal longitudinal ligament, and dorsal annulus fibrosus without appreciable osseous compromise of the vertebral canal. These redundant soft tissues might represent an attempt at stabilization of caudal cervical instability similar to that seen with instability due to osseous malformation or to ligamentous laxity in other synovial joints. The stability of the cervical vertebral column depends primarily on these ligamentous supporting structures.\(^{(62,64)}\) Degenerative and simultaneous regenerative changes in the articular cartilages due to stresses contribute evidence to the theory that joint laxity may be the cause of the hyperplastic supporting soft tissue elements.\(^{(62)}\)

In some cases, lateral spinal cord compression occurs as a result of medial overgrowth of the cranial articular processes. In this type of malformation, the articular processes are often grossly asymmetric and may lack complete cartilage coverage. Both arrested remodeling of the vertebral foramen and remodeling due to stresses on the articulations may be responsible.

Spinal cord compression may occur as a result of impingement of a prominent craniodorsal aspect of a vertebral body. Commonly there is loss of bone from the cranioventral aspect of the involved vertebral body. This type of malformation is probably a more exaggerated form of the malformation that is represented by stenotic cranial orifice due to remodeling of the pedicles as the result of abnormal stresses. The cranial aspect of the vertebral body appears to deviate dorsally, resulting in compromise of the vertebral canal, while the synovial joints remain in fairly normal apposition due to simultaneous resorption of the pedicles. Since there also is concomitant loss of bone from the cranioventral aspect of the vertebral body, the prominent craniodorsal aspect appears as a dorsal "ingrowth" into the vertebral canal, when in fact it represents dorsal angulation of the entire vertebral body, with extensive remodeling in other areas.

As is the case with the synovial joints, degenerative changes due to mechanical stresses also occur in the symphyseal joints of vertebral bodies.\(^{(2,57,62)}\) Intervertebral disk degeneration and, in some cases, extrusion or protrusion have been confirmed
C7 segments. occasional scapular muscle atrophy reflected the chronicity of the lesion, the neuronal loss from the gray matter of the C6 and the spinal cord compression was usually in the caudal cervical region, upper motor neuron (UMN) signs predominated. The ascending proprioceptive tracts (with resultant ataxia) and descending motor tracts (with resultant spastic paresis). Although in protein content of the cerebrospinal fluid. These signs suggested a cervical spinal cord white matter lesion involving the only abnormality revealed by laboratory studies of blood, urine, and cerebrospinal fluid was an occasional slight increase in the neurologic examination, since it can result in iatrogenic spinal cord attenuation.(38,50)

Clinical Examination

The common denominator in this syndrome is the typical neurologic status of paraparesis-tetraparesis-ataxia that results from any one or more of the vertebral malformations-malarticulations (spondylopathies), which produce spinal cord compression in this particular region of the central nervous system (cervical myelopathy). The spinal cord compression, contusion, stretching, or intermittent ischemia most often results in varying degrees of bilateral spastic paresis and ataxia of the pelvic limbs with an awkward swaying movement of the hindquarters-thus, the original designation as wobbler syndrome.(8)

Although most affected animals were presented for varying degrees of paraparesis-ataxia, the neurologic status was quite varied, as was the onset of neurologic dysfunction. In most instances, with the consistent exception of secondary intervertebral disk protrusion or extrusion, the onset of signs was insidious and cervical pain was absent. In young, rapidly growing dogs, the owner often assumed that the poor coordination was normal for a puppy of that age. Conversely, in older dogs, the owner often suspected that the ataxia was due to arthritis, laziness, "rheumatism", or age. Although the principal clinical sign most often noticed by the owner was ataxia of the pelvic limbs, the neurologic deficits ranged from minimal bilateral spastic paresis-ataxia to nonambulatory tetraparesis. In most cases, no thoracic limb deficit was reported by the owner, but in most of these, careful examination revealed mild thoracic limb dysfunction. The signs were usually progressive. There was no history of external injury or medical illness. Neurologic examination was often complicated by the predisposition of these breeds to a variety of musculoskeletal disorders that influenced the gait.

Physical examination most often revealed bilateral paresis and ataxia of the pelvic limbs and occasionally of the thoracic limbs. When the signs were mild, they were most evident as the dogs arose. The dogs were unsteady and tended to overextend a pelvic limb. There was a base-wide pelvic limb stance and often a crouched pelvic limb posture. The onset of protraction was slow, often accompanied by hypermetric movement or by dragging of the limbs on the dorsal surface of the digits. Placement of the limbs was abnormal. Most affected animals had difficulty arising from a recumbent position. The deficits were exacerbated on turning. Hypertonia and hyperreflexia were frequently observed. There was often a stiff appearance to the thoracic limbs. Thoracic limb spasticity was observed more commonly in Doberman pinschers than in Great Danes, as was cervical pain. Scapular muscle atrophy due to lower motor neuron involvement in the caudal cervical spinal cord was noted occasionally and could be confirmed by electromyography. The greatest deficits usually noted were in hopping and proprioceptive positioning. The signs were usually progressive and showed a variable response to previous corticosteroid administration. During walking, and especially in turning, the pelvic limbs often crossed each other, abducted widely, or in more severe cases, tended to collapse. The pelvic limb stride was often longer than normal, and asymmetric, causing the typical awkward swaying movement of the hindquarters. The toenails were often worn dorsally owing to the dragging of the limb on protraction or stepping with the dorsal surface of the paw on the ground.

Thoracic limb signs, when present, were similar but usually less remarkable. These dogs occasionally stumbled, with the carpus flexed, such that the dorsal surface of the paw struck the ground. The limbs frequently crossed and in some instances had a restricted motion and appeared to be rigid. This spastic gait, with limited joint flexion, gave the appearance that the thoracic limbs tended to float on protraction or were hypermetric.

Although manipulation of the neck did not usually elicit pain, extension of the neck often appeared to exacerbate the neurologic deficits in dogs examined early in this series. Extension of the neck is no longer performed as a portion of the neurologic examination, since it can result in iatrogenic spinal cord attenuation.(38,50)

The only abnormality revealed by laboratory studies of blood, urine, and cerebrospinal fluid was an occasional slight increase in protein content of the cerebrospinal fluid. These signs suggested a cervical spinal cord white matter lesion involving ascending proprioceptive tracts (with resultant ataxia) and descending motor tracts (with resultant spastic paresis). Although the spinal cord compression was usually in the caudal cervical region, upper motor neuron (UMN) signs predominated. The occasional scapular muscle atrophy reflected the chronicity of the lesion, the neuronal loss from the gray matter of the C6 and C7 segments.
Differential Diagnosis of Neurologic Dysfunction

The differential diagnosis should include other diseases that cause progressive cervical spinal cord lesions. Myelitis, most commonly due to canine distemper virus, will usually result in other abnormalities that cannot be explained solely by a lesion in the cervical spinal cord. Although it is not unusual for a dog with encephalomyelitis caused by canine distemper virus to have signs of spinal cord disease, the disseminated central nervous system lesions, often coupled with typical abnormalities in the cerebrospinal fluid, would not be compatible with a diagnosis of compressive myelopathy. Space-occupying lesions such as intradural or extradural neoplasms (neurofibroma, meningioma, neuroepithelioma), epidural abscesses, vertebral osteomyelitis, and progressive primary cervical intervertebral disk protrusions not associated with vertebral malformation-malarticulation can usually be diagnosed on the basis of plain or positive-contrast neuroradiography. Cervical pain is also a more consistent sign in these disorders. In the early stages of progressive cerebellar cortical abiotrophy, the ataxia may be mistaken for that caused by a cervical spinal cord lesion.

When the onset is acute, there should also be included in the differential diagnosis spinal cord contusion from vertebral fractures and luxations, spinal cord infarction from fibrocartilaginous embolization of spinal cord vasculature, and acute intervertebral disk extrusions, either primary or secondary to preexistent vertebral abnormalities or malarticulations.

Since pelvic limb signs are usually far more evident than thoracic signs, thoracolumbar disorders that commonly result in paraparesis-ataxia may also be included. Those with a progressive course include intradural and extradural neoplasms, idiopathic senile myelopathy, progressive intervertebral disk protrusions, and congenital mesodermally derived malformations of the thoracolumbar spine. Those with an acute onset include intervertebral disk protrusion or extrusion, traumatic intervertebral disk extrusion, vertebral column fracture or luxation, and thoracolumbar ischemic myelopathy.

The differential diagnosis is further complicated by the predisposition of the giant breeds to a variety of musculoskeletal disorders that commonly influence the gait. The clinician must then determine whether the patient is not willing to perform a function because of skeletal disease or is unable to perform it due to neurologic dysfunction. In some instances the dog may have both neurologic disease and skeletal disease, but careful testing should reveal the neurologic deficit. Coxofemoral dysplasia, osteochondrosis dissecans (shoulder, elbow, or stifle), hypertrophic osteodystrophy, panosteitis, genu valgum, ununited anconeal process, and fragmentation of the coronoid process are frequently observed in the giant breeds, especially if their predisposition to these disorders is aggravated by over-nutrition.

Radiographic Examination

Line drawings to illustrate the basic radiographic features of this syndrome (Figs. 63-1 and 63-2) include the appearance of the normal vertebral column in unflexed (Fig. 63-1, A) and in flexed (Fig. 63-2, A) positions, diagnostic changes observed on plain films of the unflexed cervical vertebral column (Fig. 63-1, B through E), and the changes that are most pronounced when the region is flexed (Fig. 63-2, B through E).

Proper positioning of the anesthetized patient is critical, as are radiographic exposures in both the ventrodorsal and lateral projections to delineate stenotic lesions in the transverse and sagittal planes, respectively. In contrast to earlier work,(62) myelography is now considered to be essential for the identification and localization of the site or sites of spinal cord compression due to soft tissue or osseous changes. Originally, myelography was used predominantly for those animals that showed no significant evidence of lesions on the plain films. With more experience, however, the value of myelography in elucidating soft tissue changes of the interarcuate ligament, joint capsule, dorsal longitudinal ligament, dorsal anulus fibrosus, and extruded or protruded intervertebral disks and in accurately identifying all sites of spinal cord compression and their relative significance became obvious. Frequently, lesions that appeared to be significant on plain films were excluded as causes of spinal cord compression by myelography. In these, the frequently bizarre osseous changes did not influence vertebral canal diameter and were often located at spaces well removed from the site of spinal cord attenuation.

Lateral views in normal extension and in moderate flexion are exposed first. Only after ruling out spinal cord compression of a dynamic nature,(38) in which cord compression is exaggerated by either flexion or extension, should hyperextended and fully flexed views be taken. In animals with elongated or redundant dorsal laminae or hypertrophy, hyperplasia, or relative redundancy of the dorsal longitudinal ligament and dorsal anulus fibrosus, forced hyperextension may significantly increase spinal cord attenuation during radiographic positioning.(38,50) The patient's preoperative posture may raise the index of suspicion of these dynamic types of spinal cord compromise. Dogs with hypertrophy of the dorsal longitudinal ligament or dorsal anulus fibrosus or with dynamic dorsal laminar lesions usually resist extension of the neck by the examiner and frequently carry their heads low. They appear to observe the clinician by rolling their eyes dorsally without
raising the head. By maintaining the cervical spine in a partially flexed position, the patient is actually decreasing the amount of spinal cord attenuation.

FIG. 63-1 Diagrammatic representations of radiographs of the caudal cervical vertebral column of a dog, with the neck in the normal extended position. In most cases, there is little if any change in the normal position of the vertebral arches. Therefore, on radiography, the dorsal bony limits of the vertebral canal usually appear smooth and continuous, without obstructing or projecting changes. (A) The normal extended caudal cervical vertebral column. (B) Dorsal displacement of the cranial aspect of the body of C7. (C) Ventral exostosis has appreciably altered the shape of the body of C7. Displacement of the vertebral body is minimal. (D) Combination of B and C: dorsal displacement of the cranial aspect of the body of C7 with ventral exostosis. Immobilization of the C6-7 symphysis (ankylosis) has occurred, with subsequent development of malarticulation at C5-6. (Trotter EJ, deLahunta A, Geary JC et al Caudal cervical vertebral malformation-malarticulation in Great Danes and Doberman pinschers. J Am vet Med Assoc 168:917-930, 1976)

FIG. 63-2 Diagrammatic representations of radiographs of the caudal cervical vertebral column of a dog, with the neck flexed. Note stenosis of the cranial orifices of the vertebral foramina in all illustrations, a fairly consistent feature of this syndrome. (A) The normal flexed caudal cervical vertebral column. (B) An unstable malarticulation, which is best appreciated on flexion. Wedging of the intervertebral disk space and displacement of disk material can be visualized. (C) Similar to B, but with pronounced loss of bone from the cranioventral aspect of the vertebral body. This remodeling change can be recognized with the neck extended, but the amount of displacement is best appreciated with flexion. (D) An unstable malarticulation with obvious exostosis but without ankylosis. When the neck is flexed, the displacement appears more prominent. (E) Similar to Figure 63-1, E, but without the obvious ankylosis of the C6-7 symphysis. However, the effective immobilization of this joint is demonstrated when radiographs of the flexed and normally extended neck are compared. (Trotter EJ, deLahunta A, Geary JC et al Caudal cervical vertebral malformation-malarticulation in Great Danes and Doberman pinschers. J Am vet Med Assoc 168:917-930, 1976)

In contrast, dogs with severe vertebral instability may maintain the neck in a rigidly extended position, since flexion tends to increase the spinal cord compression subsequent to subluxation of the vertebral bodies.

Hyperextended views rarely contribute significant information in this disorder, and, as mentioned, can be extremely hazardous owing to the iatrogenic exaggeration of spinal cord compression during forced extension. Moderate extension, cautiously produced under fluoroscopy, and "traction views" during normal extension in patients with hypertrophy of the dorsal longitudinal ligament or dorsal anulus fibrosus are, however, essential not only for accurate diagnosis but for patient safety and the selection of the most appropriate surgical techniques. (Trotter EJ, deLahunta A, Geary JC et al Caudal cervical vertebral malformation-malarticulation in Great Danes and Doberman pinschers. J Am vet Med Assoc 168:917-930, 1976)
extension of the cervical spine.(38)

An additional problem with radiographs exposed extreme flexion or extension is the creation of artifactual lesions. Spurious subluxations or vertebral "tilting" lesions are frequently created with flexed views. As suggested by Wright, there is significant individual variation in the amount of vertebral angulation during flexion and extension of the cervical spine. (68,69) This normal variation in vertebral angles with changes in the position of the neck can easily lead to misinterpretation and an erroneous diagnosis of vertebral slippage. In some cases, however, unstable malarticulation (subluxation), with or without vertebral deformation or predisposing vertebral canal stenosis, is, in fact, the cause of spinal cord compression. This can be confirmed by myelography, with comparison of the normally extended and flexed views. Further studies confirmed the value of sagittal diameter measurements in the diagnosis of cervical spinal stenosis, as utilized in humans.(70)

Comparison of interpedicular dimension and symmetry of the articular processes, with and without contrast radiography, is important in dogs with vertebral canal compromise in the transverse plane. Frequently, visualization of the lesions is significantly improved by removal of the endotracheal tube during exposure of these ventrodorsal views.

The neurologic dysfunction is due to spinal cord attenuation from any of the various malformations or malarticulations, or combinations thereof, that result in deformity of the vertebral canal. The abnormalities that do not result in spinal cord compression are nevertheless significant, since they represent "footprints" of disease that have probably resulted from altered spinal biomechanics, that is, from abnormal stresses on the bony and ligamentous structures of the caudal cervical spine. These plain radiographic changes, without evidence of compromise of the subarachnoid space on myelography, include the following:

Exostoses, which are most often on the cranioventral aspects of the vertebral bodies

Degenerative periarticular osseous changes typical of arthritis

Degenerative changes of the associated intervertebral disks reactive changes on the dorsal spines

Degenerative or reactive changes of the ligamentous supporting structures of the ventral aspect of the vertebral column

Loss of, or production of, bone at the ventral aspects of these vertebrae

Ankylosis of symphyseal joints

Thus, in many affected dogs, multiple abnormalities may be visualized on plain films of the normally extended cervical spine. With myelography, one (most commonly) or multiple sites and types of spinal cord compression may be confirmed. There is frequently a discrepancy between the lesions that appear to be most significant on examination of plain films and those that are found on myelography. The bizarre and often extensive secondary bony changes representing the body's response to abnormal forces are not often indicative of the exact site or sites of spinal cord compromise. Vertebral ankylosis demonstrates this concept well. The completely collapsed intervertebral space, exostoses, and reactive changes visualized on the ventral aspects of the vertebrae in these cases are only sometimes associated, at least directly, with spinal cord compression. Most frequently, the spinal cord compression is present at the interspace either cranial or caudal (or both) to the ankylosed or previously arthrodesed interspace (symphyseal joint). This domino effect probably occurs as the result of the transfer of, or induction of excessive, abnormal mechanical stresses to the contiguous intervertebral articulations. This may then result in compressive lesions at these locations.

The spondylopathies in the caudal cervical region of large breeds of dogs that cause spinal cord compression, contusion, ischemia, or stretching, and the resultant neurologic signs of wobbler syndrome, are as follows:

Stenotic cranial orifice of a vertebral foramen, probably as a result of delayed osteocytic osteolysis(23) or of continual bony remodeling of both the vertebral body and lamina, resulting in static or dynamic spinal cord compression. This may occur with or without deformity of the craniodorsal or cranioventral aspects of the vertebral body.

Chronic intervertebral disk disease in the nonchondrodystrophoid breeds with chondroid or fibroid metaplasia in the disk. This probably occurs as a result of abnormal stresses induced by vertebral instability and thus altered spinal biomechanics or by the domino effect of excessive force transfer subsequent to either spontaneous or surgical fusion of adjacent symphyseal (interbody) joints.

Dorsoventral overgrowth or malformation of the articular processes. This may include one or both of the cranial and/or caudal articular processes on one or both sides.
Mediolateral overgrowth or malformation of the articular processes. This may include one or both of the cranial and/or caudal articular processes on one or both sides.

Dorsal anulus fibrosus (predominantly) and longitudinal ligament hypertrophy or relative redundancy, with or without collapse of the intervertebral disk space and decompression during traction applied to the vertebral column.

Interarcuate ligament and joint capsule hyperplasia or hypertrophy. This often results in the typical "hour glass" appearance on myelography owing to almost circumferential spinal cord compromise. Unstable malarticulation or vertebral subluxation without vertebral malformation (evident on comparison of normal extended and flexed views).

Unstable malarticulation or vertebral subluxation with exacerbation of spinal cord compression by malformation or remodeling of the vertebral body and/or arch. Instability again is evident as a change in position of the malformed vertebral body in the comparison of flexed and normally extended views.

Stable vertebral malarticulation without vertebral malformation (evident on normally extended and flexed views).

Stable vertebral malarticulation with exacerbation of spinal cord compression by malformation or remodeling (evident on normally extended and flexed views) of the vertebral body or arch, or both.

Acute secondary subluxations, often with intervertebral disk extrusion at the symphyseal joint cranial or caudal (most often C7-T1) to the site of a spontaneous or surgical fusion, once again demonstrating the domino effect of transfer of abnormal stresses due to "block vertebrae"-induced instability with adjoining segments.

Various combinations of any of the above described first or second-degree lesions at the same or multiple locations in the caudal cervical vertebral column.

Radiographs of affected dogs illustrate the various malformations and malarticulations and combinations thereof (Figs. 63-3 through 63-10).

FIG. 63-3 A 15-month-old Great Dane with pronounced changes in both the shape and position of the body of C7. Even the vertebral arch has been altered in position. (See Fig. 63-1, D) (Trotter EJ, deLahunta A, Geary JC et al: Caudal cervical vertebral malformation-malarticulation in Great Danes and Doberman pinschers. J Am Vet Med Assoc 168:917-930, 1976)


FIG. 63-5 A 7-year-old Great Dane with dorsal displacement of the body of C7, with resulting narrowed canal. Remodeling of the vertebral body accounts for its unusual shape. Dorsal canal limits are reasonably normal. The appearance is similar to that of Figure 63-1, D, but without the "rough" contour of active exostosis. (Trotter EJ, deLahunta A, Geary JC et al: Caudal cervical vertebral malformation-malarticulation in Great Danes and Doberman pinschers. J Am Vet Med Assoc 168:917-930, 1976)
Gross Pathology

Postmortem examinations of affected dogs have confirmed the plain radiographic and myelographic findings. The exact site or sites of spinal cord compression have been confirmed either grossly or by histopathologic examination for wallerian
Degeneration. These sites correlated well with the radiographic findings.

Following disarticulation, the spinal cord was in some instances observed to be compressed dorsoventrally. This occurred as a result of stenosis of the cranial orifice of a vertebral foramen. The disparity in diameter of the cranial and caudal orifices of the vertebral foramen was apparent on removal of the spinal cord segments. The vertebral foramen was often funnel-shaped, with the smaller orifice at the cranial end. In some, this stenosis was markedly exacerbated by extensive bony remodeling or malformation of the vertebral body, which had resulted in the projection of a prominent craniodorsal aspect of the vertebral body into the vertebral canal (Fig. 63-11).

Enlarged interarcuate ligaments and joint capsules, sometimes in association with redundant dorsal longitudinal ligaments and dorsal anulus fibrosus, resulted, in some cases, in circumferential spinal cord compression (Fig. 63-12).

Marked asymmetry in position, size, and shape of the articular processes with or without instability was noticed in many of the cervical vertebrae and in some instances had resulted in either mediolateral or dorso-ventral spinal cord compression (Fig. 63-13). Osteochondritic and osteoarthritis changes were also observed in many of these abnormal symphyseal joints.

Hypertrophy or redundancy of the dorsal longitudinal ligament and/or dorsal anulus fibrosus that had resulted in spinal cord compression from the ventral aspect could not always be reduced by means of traction during postmortem examination. This hypertrophy occurred both with and without collapse of the involved disk space and/or simultaneous hypertrophy of the interarcuate ligament or joint capsules. Chondroid metaplasia of the intervertebral disks in nonchondrodystrophoid breeds was also confirmed in some, and, as suspected with the other previously mentioned secondary degenerative changes, was due to the transfer or induction of abnormal stresses.


**FIG. 63-12** Cranial view of C5 of a 4-month old female Doberman pinscher. Note the compressed spinal cord at the cranial orifice, with hyperplastic interarcuate ligament on its dorsal surface. (Trotter EJ, deLahunta A, Geary JC et al: Caudal cervical vertebral malformation-malarticulation in Great Danes and Doberman pinschers. J Am Vet Med Assoc 168:917-930, 1976)


**Histopathology**

Microscopic studies reveal focal spinal cord injury, usually limited to the sixth or seventh cervical spinal cord segments, or both. Occasionally, the fifth segment is involved. Variable degrees of degeneration of the gray and white matter in these segments involve most funiculi of the white matter (Fig. 63-14). At the focal spinal cord lesion, there is frequently a paucity of neurons in the gray matter, with an abundance of hypertrophied astrocytes. In some cases, this correlates well with the scapular muscle atrophy due to the lower motor neuron involvement, that is, neurogenic atrophy. In some dogs, the degeneration is limited to the white matter but involves both the ascending and descending pathways. In spinal cord segments cranial to the lesion, the white matter degeneration is limited to the ascending tracts in the dorsal and dorsolateral funiculi.
Caudal to the focal lesion, white matter degeneration is limited to the descending spinal cord tracts in the ventral funiculi and deep portions of the lateral funiculi. This pattern of noninflammatory degeneration of ascending neurons cranial to the spinal cord injury and descending neurons caudal to the injury is explained by wallerian degeneration of the axon and its myelin that occurs in the segment of the neuron separated from its cell body.

Transverse sections at the site of the lesion usually stain lightly and have a spongiform appearance due to the degeneration of myelin. The enlarged empty spaces that represent demyelination sometimes coalesce into small cavities and occasionally contain swollen axons or lipid-filled macrophages or both. Hypertrophied astrocytes (gemistocytes) are often scattered throughout the degenerate white matter. Occasionally, a focus of necrosis is found in the white matter adjacent to gray matter containing lipid-filled macrophages. The degeneration of myelin and axons is often more obvious in longitudinal sections of the affected funiculi. Chains of continuous myelin degeneration chambers often contain swollen or granulated axonal debris or lipid-filled macrophages or both.

Myelin degeneration is usually more prominent than axonal degeneration. It has been assumed and partially substantiated by experimental studies(27) that the spinal cord degeneration occurs secondary to interference with the blood supply to this region of the spinal cord. Satisfactory response from surgery can thus be anticipated on the basis of elimination of further injury to the spinal cord and on remyelination of those axons that are still intact.

Treatment

Rational medical, surgical, and adjunctive medical therapies have evolved from the study of all aspects of this syndrome. Owing to the variety of malformations, malarticulations, their static or dynamic nature, secondary changes, and existence of multiple lesions, a single surgical procedure obviously cannot be recommended for all cases. The therapy utilized must be that best suited to the successful control of signs or to the correction of the animal's particular spondylopathies and must be further modified by the surgeon's previous experiences with the various techniques. Accurate and complete diagnosis, predominantly by myelography, cannot be overemphasized in the selection of the most appropriate surgical techniques. The information gained is critical to the determination of the advisability and necessity of decompression, either ventrally or dorsally, and the need for stabilization, either ventrally or dorsally, in a single or in multiple locations. Surgical judgment is then further influenced by the surgeon's previous experiences with the morbidity, mortality, and postoperative responses and care inherent to the various surgical techniques in patients of a variety of ages with a variety of neurologic deficits. Further consideration must also be given to the financial impact of surgery and the often extended postoperative care.

MEDICAL

Dogs with minimal to mild signs of paraparesis-tetraparesis-ataxia may often be managed by the oral administration of corticosteroids alone. After a 48- to 72-hour trial to determine corticosteroid responsiveness at a dosage of 1 mg to 2 mg/kg bid, the loading dose of prednisone (Deltasone)* is decreased gradually over a 5-day period to a dosage of 0.5 mg to 1 mg/kg on an alternate-day basis. Further decreasing doses of corticosteroids are used depending on the response of the animal. In dogs with a classic history and neurologic signs and a satisfactory response to the corticosteroid administration, no further diagnostic workup is performed. In many patients, the administration of corticosteroids results in sufficient improvement to allow return of functional status as a companion animal. Only rarely are signs completely relieved. The response in some cases, however, even with relatively severe neurologic deficits, is dramatic. Often, corticosteroid administration may be discontinued after a few months without deterioration in neurologic status. In some, intermittent exacerbations remain corticosteroid-responsive. Some patients have been managed with this regime for periods as long as 5 years, possibly as a result of spontaneous resolution from remodeling with maturity, compensation, or ankylosis. In other patients, usually those with more severe deficits, the response to corticosteroid administration is either unsatisfactory or temporary. In these animals, complete diagnostic evaluation and surgery are often indicated.
As is the case with most neurosurgical patients, significant spinal cord compression requires decompression, and significant vertebral instability requires stabilization. The techniques of decompression and stabilization are determined by the particular types, severities, and locations of the compressions or instabilities. Therefore, decompression can vary from minimal facetectomy to decompressive laminectomy of most of the cervical spine. Stabilization varies from simple arthrodesis of one of the pairs of synovial joints to extensive ventral interbody fusion of the majority of the cervical spine.

Three basic surgical techniques are used, with minor variations dependent on the animal's combination of malformations, malarticulations, and their singular or multiple sites of spinal cord compression.

1. Dorsal decompressive laminectomy with or without fusion of the articular processes (synovial joints)
2. Ventral decompression with or without fusion of the vertebral bodies (symphyseal joints)
3. Ventral distraction and fusion

The selection of the most appropriate surgical techniques for the individual patient is based predominantly on the myelographic findings of spinal cord compression and is often modified further by observations made during surgical intervention.

With either static or dynamic dorsal spinal cord compression due to a stenotic cranial orifice of a vertebral foramen, laminectomy of the malformed vertebral lamina, plus portions or all of the lamina of the next cranial vertebra, is performed. If instability is present also, arthrodesis of the synovial joints is indicated.

With ventral spinal cord compression due to acute or chronic intervertebral disk protrusion or extrusion, ventral decompression with mass removal is performed. Instability at this or adjacent interspaces may require arthrodesis of the symphyseal joints.

With dorsal or dorsolateral spinal cord compression due to acute or chronic intervertebral disk protrusion or extrusion, laminectomy with mass removal is performed. Instability at this or adjacent interspaces may require arthrodesis of the synovial joints.

With spinal cord compression in either the sagittal or transverse plane due to dorsoventral or mediolateral overgrowth or malformation of the cranial or caudal articular processes, laminectomy and partial facetectomy is performed. Fusion of the remainders of the articular processes may be necessary if instability also exists.

With ventral spinal cord compression due to real or relative redundancy of the dorsal anulus fibrosus and dorsal longitudinal ligament that reduces when traction is applied to the vertebral column, ventral distraction with fusion is performed. If traction fails to eliminate the cord compression, ventral decompression, with or without fusion of the symphyseal joint is indicated.

With "hour glass" compression of the spinal cord due to hypertrophy of the interarcuate ligament and joint capsules, laminectomy with excision of all impinging portions of the ligaments and capsular structures is followed by pedicle fat grafting into the laminectomy defect. Instability, if present, is corrected by arthrodesis of the synovial joints.

With spinal cord attenuation due to unstable vertebral malarticulation (subluxation) without attenuation due to vertebral malformation, stabilization by means of fusion of the articular processes following reduction of the subluxation and laminectomy is preferred.

With ventral spinal cord compression due to a prominent, impinging, craniodorsal aspect of a vertebral body, ventral decompression is necessary. With instability, ventral fusion is also performed.

With spinal cord compression due to stable malarticulation without exaggeration of attenuation by bony vertebral deformity, laminectomy is preferred.

With spinal cord compression due to stable malarticulation and vertebral remodeling or malformation, the choice of ventral or dorsal decompression and excision of lesions depends on the location of the impinging masses.
Multiple lesions may require any combination of the above described techniques, in a variety of locations, and with decompression of varying extent and length.

**Dorsal Decompressive Laminectomy**

The preoperative and intraoperative use of corticosteroids has been advocated. The dog is positioned in sternal recumbency, with the head slightly elevated in a head restraining apparatus. This technique is utilized to avoid compression of the external jugular veins and subsequent shunting of venous return through the internal vertebral venous plexuses. The dorsum of the neck is prepared for aseptic surgery.

A dorsal midline skin incision is made from the occiput to the spine of the first thoracic vertebra (T1). Following incision of the superficial fascia of the neck and the midline attachments of the cleidocervicalis, trapezius rhomboideus, and splenius muscles, the ligamentum nuchae is retracted laterally to allow subperiosteal elevation of the muscles of the transversospinalis system to the level of the articular processes of the involved vertebrae. Moistened lap tapes and self-retaining retractors (Downing laminectomy retractor, Zimmer USA, Warsaw, IN) are used to maintain exposure and to allow for dissection under tension.

The spines of the involved vertebrae are excised with bone rongeurs and saved in a sterile stainless steel cup for use later as bone grafts. By means of a high-speed air drill (Air drill 100, Surgical Products Division, 3M Co., Santa Barbara, CA) with an egg-shaped bur, (A-507 4-mm carbide bur, 3M Co., Santa Barbara, CA) the proposed laminectomy defect is outlined by cutting grooves in the outer compact bone of the vertebral laminae. The width of the defect is limited by the medial aspects of the caudal articular processes of the cranial vertebra (Fig. 63-15). The length of the defect is a minimum of two vertebral arches, from interarcuate ligament to interarcuate ligament in those animals with a single lesion. In others, more extensive laminectomy is necessary, and in some may extend from C3 to C7. When the drilling of the grooves has continued through the middle layer of cancellous bone of the vertebral arches to the inner layer of compact bone, a small, round carbide-tip bur (A-508 2.3-mm and 1.6-mm carbide-head burs, 3M Co., Santa Barbara, CA) is substituted to allow greater visualization within the depths of the grooves. Incision of the interarcuate ligaments and joint capsules permits en bloc resection of this isolated portion of the vertebral arches of the involved vertebrae. Hyperplastic interarcuate ligaments and portions of hyperplastic joint capsules, if present, are removed with the vertebral arches. Impinging portions of cranial or caudal articular processes are excised by continued drilling or by means of small bone rongeurs. A 5-0 silk suture is placed in the dura mater to allow minimal atraumatic retraction or "rolling" of the spinal cord. This permits safer undercutting of the laminectomy edges. The middle layer of cancellous bone and the inner layer of compact bone of the vertebral pedicles are hollowed out or excavated to the level of the internal vertebral venous plexus. During this undercutting procedure, any additional medial ingrowth of the cranial articular processes or joint capsules is resected. The undercut edges of the laminectomy defect provide sufficient exposure for the removal of protruded or extruded intervertebral disk material from the lateral aspects of the vertebral canal. Removal of ventral midline masses with this approach can be accomplished only with hazardous spinal cord manipulation and is not recommended. In severe cases, the dura mater is incised on the dorsal midline for the full length of the laminectomy to provide more spacious decompression of the spinal cord and to permit direct inspection of the spinal cord, spinal cord vasculature, and cerebrospinal fluid flow.

Owing to the traumatic exposure and spinal cord manipulation in this procedure, all available mechanisms for the protection of the vital and sensitive nervous tissue elements are utilized. Selective regional spinal cord hypothermia, according to a previously described technique, is often used in addition to corticosteroid administration and hyperventilation to decrease the effects of iatrogenic spinal cord trauma.

Secondary compression of the spinal cord (16, 59) by healing of the laminectomy defect has not usually been a problem with cervical laminectomy, except in those animals with hypertrophy or hyperplasia of the joint capsules and interarcuate ligaments. In these, a pedicle or free fat graft is placed in the laminectomy defect to prevent the occurrence of constrictive fibrosis. (21, 30, 31, 33) In most other patients, absorbable gelatin sponge (Gelfoam, Upjohn Co., Kalamazoo, MI.) is "tented" over the laminectomy defect prior to layered closure of the surgical wound with suction drainage. (Snyder Hemovac, Zimmer USA, Warsaw, IN.) If, however, removal of bone of the vertebral pedicles below the level of a dorsal tangent to the spinal...
if stabilization is indicated either by radiographic or surgical findings of instability, the articular processes of the involved vertebrae (synovial joints) are fused bilaterally. The joint capsules are incised and reflected to provide exposure of the articular surfaces. In most animals with instability, the joint capsules and interarcuate ligaments are hypertrophied, redundant, and incapable of maintaining, as usual, some stability of the cervical spine. By means of a nitrogen-powered drill, (K-100 Mini-Driver, Surgical Products Division, 3M Co., Santa Barbara, CA) a tunnel approximately 1 mm in diameter is drilled through the cranial and caudal articular processes at the midpoint of their articulations (Fig. 63-16). The hyaline cartilage, which is often abnormal, is removed from the articular surfaces with the high-speed drill. A cancellous bone graft, obtained from the previously excised dorsal spines or from the greater tubercle of the humerus of the same patient, is packed into the articulations. Eighteen-gauge orthopaedic wire (Suture wire (malleable), Richards Manufacturing Co., Memphis, TN) is passed through the tunnels and twisted tightly to immobilize and compress the articulation. Orthopaedic screws, (Cortical bone screws, 4.5-mm diameter, Synthes Ltd., Wayne, PA) placed in accordance with the lag principle, have been reported to achieve superior fixation to that achieved with orthopaedic wire.(56) In either case, some caution must be exercised during drilling of the tunnels to prevent inadvertent damage to the cervical nerves or vertebral arteries. The surgical wound is closed with suction drainage, described above. A heavily-padded neck bandage is applied postoperatively in animals with midcervical lesions. Application of neck bandages, however, in caudal cervical lesions is not recommended unless excessive "levering" due to the weight of the external support can be avoided. Normally, fusion is radiographically evident at approximately 6 to 8 weeks. Corticosteroids are administered postoperatively according to a previously described regiment Analgesics are often also indicated following dorsal cervical laminectomy. Broadspectrum antibiotic therapy is continued for at least 3 days after corticosteroid therapy has been discontinued.

Ventral Decompression (Fig. 63-17)

Ventral decompression has been described by various authors (22,43,44,49,54,55,62) The dog is positioned in dorsal recumbency, with the neck slightly extended over a sandbag. Following preparation of the ventral cervical region for aseptic surgery, the skin is incised on the ventral midline, from the manubrium to the level of the caudal angle of the mandible. The paired midline insertions of the sternohyoideus muscles are incised prior to retraction of the trachea and esophagus to the left of midline. Moistened lap tapes and a self-retaining retractor (Gelpi retractor, Zimmer USA, Warsaw, IN) are used to maintain exposure. The longus colli muscles are elevated from the ventral aspects of the involved vertebrae and the intervertebral disk. By means of a high-speed air drill, a defect, centered over the intervertebral disk and including approximately the caudal one-third to one-half of the cranial vertebra and approximately one-third to one-half of the cranial aspect of the caudal vertebra, is created (Fig. 63-17). The width of the defect is approximately one-half of the width of the intervertebral disk. When the ventral cortical and middle Cancerous bone has been removed with the large egg-shaped bur, a small round carbide-tip bur is substituted for the removal of the dorsal layer of cortical bone. During drilling, the angulation of the defect being created should occasionally be compared with the angulation of the intervertebral disk on preoperative radiographs to ensure penetration of the vertebral canal at the center of the synphseal joint. Hemorrhage from the internal vertebral venous plexus is controlled with Gelfoam or temporary packing of the defect with gauze sponges. Hemorrhage from the basivertebral vessels is controlled with bone wax. This bone wax must be completely removed prior to placement of a bone graft in those animals in which fusion is desired.

An impinging vertebral body malformation, usually the craniodorsal aspect of the caudal of the two involved vertebral bodies, may be excised by means of the high speed drill under direct visualization with minimal hazard of inadvertent spinal cord laceration. Protruded or extruded intervertebral disk material may be removed from the ventral aspect of the canal by means of small rongeurs, tartar scrapers, hemostats, and other small instruments normally used for ophthalmologic surgery. Excision of the relatively tough fibers of redundant dorsal anulus fibrosus and dorsal longitudinal ligament frequently requires a combination of blunt and sharp instrumentation.

Following inspection of the vertebral canal for the effectiveness of decompression and the completeness of removal of attenuating masses, fusion of the synphseal joint is performed in those animals with significant instability. Even without
bone grafting, fusion will occur spontaneously in this type of ventral decompressive defect. However, the importance of the immediate stability achieved by firm placement of a bone graft is obvious in those patients with instability at this articulation. Most other means of ventral stabilization of the cervical spine have not been highly satisfactory. A bone graft, previously obtained from the iliac crest, is shaped to fit the defect in the vertebral bodies (Fig. 63-17). The graft is contoured by means of the high-speed drill to be approximately 1 mm to 2 mm wider and 2 mm to 4 mm longer than the decompressive defect. With widening of the symphyseal joint produced by strong distraction of the pelvis and head as a result of linear traction applied to the vertebral column, the graft is forcibly tapped into place with a bone tamp (Kiene bone tamp, Zimmer USA, Warsaw, IN.) to afford immediate stabilization. The drilling process itself results in the vertebral defect being somewhat longer and wider on the ventral aspect of the vertebral bodies than on the dorsal. This in itself effectively prevents migration or inadvertent placement of the graft into the vertebral canal. For stabilization only, iliac grafts have proven sufficient. However, if significant distraction of the vertebrae is to be achieved, a stronger graft more resistant to collapse, such as a full tibial cortical allograft, obtained from a bone bank should be used. In either case, fusion is usually radiographically evident in approximately 8 weeks. Postoperative radiographs are evaluated for the positioning of the graft and the completeness of removal of any attenuating masses. Corticosteroids and antibiotics are continued in the postoperative period as described for dorsal laminectomy. The same cautions should be exercised with external fixation devices to prevent a lever arm effect. Analgesics are necessary less frequently than following dorsal laminectomy.


Ventral Distraction

In those patients with dynamic spinal cord compression due to a relative redundancy of the dorsal anulus fibrosus and dorsal longitudinal ligament that is exaggerated by cervical hyperextension and reduced by cervical flexion or traction, a ventral distraction with fusion procedure is recommended. Initially, attempts were made to retain the vertebrae in distracted position by ventral plating of the vertebral bodies in order to maintain the hypertrophied soft tissue structures in a stretched or reduced position on the ventral aspect of the vertebral canal. Although success was achieved in some cases, loosening of the implants has been experienced. More recently, a ventral distraction procedure with bone grafting has met with a higher degree of success. The ventral defect created here is not, in fact, a decompressive defect, since the vertebral canal is not invaded. Preoperative, intraoperative, and postoperative care is identical to that with ventral decompression and fusion. The defect created in the ventral aspects of the bodies of the two adjacent vertebrae, however, is shallower than in ventral decompression and is frequently made to be longer in the depths of the defect than at the ventral surface of the vertebral bodies. This is an attempt to prevent ventral migration of the bone graft by undercutting of the edges of the defect in the ventral cortical bone. A full tibial cortical allograft, which is somewhat wider and considerably longer than the defect created in the vertebral bodies, is placed during linear traction on the cervical spine so as to maintain the adjacent vertebrae in a distracted position. This results in stretching or flattening of the hypertrophied dorsal anulus fibrosus and dorsal longitudinal ligament and therefore continued spinal cord decompression. As with ventral decompression and fusion, changes in the adjacent symphyseal joints (2,57,62) (intervertebral disks) and synovial joints (articular processes) may create problems in the future as a result of altered stresses induced by this functional block in a normally dynamic region of the cervical spine.

Advantages, Disadvantages, and Complications

As with any operative procedure, the dorsal and ventral surgical techniques for the correction of cervical myelopathies have inherent advantages and disadvantages. Rarely does the surgeon have a free choice of approach to the problem, since the specific locations of the lesions usually mandate either a ventral or dorsal approach. Although the consensus is far from unanimous, most surgeons, if given the choice, prefer ventral approaches to the cervical spine in spite of their disadvantages, which include limited visualization of the spinal cord with limited decompression; limited exposure for the inspection of osseous, ligamentous, and neural lesions at this and other locations; increased risk of iatrogenic spinal cord trauma due to the poor visualization within the depth of the decompressive defect; prolonged operative time in those cases in which it is necessary to obtain a bone graft from the ilium (although this can be eliminated by the use of cortical grafts from a bone bank); significant hemorrhage from the internal vertebral venous plexus, its anastomotic branches, and the basivertebral vessels; and the limited techniques available for ventral stabilization.

Cervical dorsal laminectomy, however, especially if extensive, is associated with high morbidity. Most patients, in spite of all
ancillary techniques used to protect the nervous tissue, have a more severe neurologic deficit in the early postoperative period. Aftercare of a functionally tetraplegic 90-pound dog can be traumatic for the patient, owner, and surgeon. Despite a somewhat "rocky" postoperative course, dogs who regain functional status do so within 6 weeks. Early discharge from the hospital to a cooperative and enthusiastic owner appears to shorten the recovery period for both patient and surgeon. Postoperative care of slightly ataxic to nonambulatory tetraparetic patients is similar to, but usually more extensive than that previously described for dogs with paralysis due to thoracolumbar intervertebral disk protrusion or extrusion.(58,60) The use of large water beds, both in the hospital and after discharge, has proven invaluable in the prevention of decubiti in large dogs. Exercise carts for nonambulatory patients can be constructed with a little ingenuity from discarded aluminum lawn furniture or hospital gurneys.

As more animals are followed on a long-term basis, more problems have been seen with secondary lesions that appear to have been induced by the initial surgical procedures. The domino effect previously described appears to be responsible. The fusion-induced alterations in spinal biomechanics with transfer of excessive forces to adjacent articulations often result in early degenerative joint disease of both the synovial and symphyseal articulations at either or both ends of the functional vertebral block. In some cases, this has resulted in signs of spinal cord compression of varying degrees at locations (usually C7-T1) adjacent to the initial lesion or lesions. In a small number of patients, a "vicious cycle" of additional disk protrusions, extrusions, or subluxations at either or both ends of the initially fused vertebrae has necessitated sequential fusion of the entire cervical spine. As in humans, alternate techniques, usually involving less rigorous attempts at fusion, may be indicated. Some have advocated intervertebral disk fenestration for its stabilizing effect on the cervical spine.(34) Others rely on postoperative external support and fibrosis of soft tissues surrounding the vertebral column.

Prognosis

The prognosis in affected animals is as varied as their vertebral column lesions and neurologic deficits. More extensive lesions have a less favorable prognosis, as do soft tissue lesions as compared with osseous lesions (with the exception of hypertrophy of the dorsal anulus fibrosus and dorsal longitudinal ligament). Patients with single lesions have a more favorable prognosis than those with multiple lesions, which require more extensive surgery. As with thoracolumbar spinal cord lesions, lower motor neuron involvement necessitates a more guarded prognosis owing to direct involvement of the cell bodies rather than the axons. Recovery is dependent on the reversibility of the spinal cord lesion or lesions. If all of the lesion at the injured segment is one of axonal disruption, surgical therapy should not be expected to be followed by improvement except for what compensation may occur. Myelin degeneration is the most prominent lesion in most instances and may precede axonal disruption caused by progressive compression and possibly by stretching of the spinal cord at the site of the malformation-malarticulation. Experimental studies support the observation that the focal myelopathy results from interference with the blood supply to this region of the cervical spinal cord(28) Postsurgical improvement depends on the elimination of further injury to the spinal cord and remyelination of those axons that are still intact.

In general, the more severe the neurologic dysfunction, the less favorable is the prognosis. In my experience, the acuteness of onset has not warranted a poorer prognosis as consistently as it has in paralysis due to thoracolumbar disk disease. In those animals with severe tetraparesis, especially nonambulatory tetraparesis, recovery is rarely complete. Frequently, proprioceptive deficits remain. In spite of this, these dogs may be returned to the status of a functional companion animal.

References

7. Conrad C: Motion of the canine cervical vertebral column in the median plane: A radiographic method of analysis.
34. Mason TA: Cervical vertebral instability (wobbler syndrome) in the dog. Vet Rec 104:142, 1979
52. Slijper EJ: Kon Med Akad Vet Verb (Tweede Sectie) 42:1, 1946

All rights reserved. This document is available on-line at www.ivis.org. Document No. B0064.0685.