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Cystic Necrosis of the Spinal Cord in Compressive Cervical Myelopathy:

Demonstration by Iopamidol CT-Myelography

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Seven consecutive patients with compressive cervical myelopathy were studied with standard water-soluble contrast myelography and immediate CT followed by delayed CT of the spinal canal at 10 to 12 hr. Every case demonstrated findings suggestive of necrosis and/or cavitation of the central portions of the spinal cord on the delayed CT study. Two types of abnormalities were visualized in the form of delayed collections of contrast media inside the cord: (1) bilateral enhancement, a double-barreled "snake-eyes" appearance at or near the level of compression (consistent with central gray matter necrosis and/or cavitation—local syringomyelia ex-vacuo); and (2) longitudinally oriented, "pencil-shaped" central enhancement of variable length distant from the level of compression (consistent either with an enlarged central canal—hydromyelia ex-vacuo—or with necrosis and/or cavitation extending craniad and caudad from the area of maximal compression and located in or near the anterior portion of the dorsal columns—distant syringomyelia ex-vacuo). These findings, supported by previous reports of autopsy specimens, may explain in part: (1) the frequent discrepancy between the levels of maximal cervical compression and the variable neurologic signs; and (2) the frequent lack of improvement in clinical signs after surgical decompression of the spinal cord at this late stage of the illness. We believe similar intramedullary lesions may be present in other cases of chronic compression of varying etiology at any location within the spinal cord.

Compressive lesions of the cervical spinal canal commonly present in three ways: incidental radiographic findings; signs of cervical nerve root compression; and/or signs of cervical spondylotic myelopathy (CSM). This paper deals with the latter phenomenon, which has created much consternation as a result of the following clinical inconsistencies [1–15]. Frequently, the neurologic findings do not correlate with the level of maximal compression as seen on myelography. Often there are variable sensory and motor findings suggestive of a transverse spinal lesion, amyotrophic lateral sclerosis, central cord syndrome, Brown-Sequard syndrome, or syringomyelia syndrome. Atrophy of the hand muscles (whose motor cells are at the C8 and T1 spinal cord levels) is often seen in association with lesions present at higher cervical levels. At surgery, the cord may appear not to be compressed. Surgical decompression often does not improve the clinical signs and occasionally may worsen the neurologic condition. After surgery the myelopathy may continue to progress. Wilson et al. [15] concluded, "Mounting evidence now implicates etiologic factor(s) in addition to, or instead of, compression of neural structures because: (a) levels of dysfunction often do not correspond to the location of vertebral osteophytes; (b) evidence of spinal cord compression by ventral osteophytes is often slight or absent at laminectomy; and (c) myelographic findings do not always correlate with neurologic involvement."

In 1958, Brain and Wilkinson [4] found radiographic signs of cervical spondylosis in 24 out of 52 cases of syringomyelia. They hypothesized that the process of cervical bony joint degeneration was intensified in patients with syringomyelia. A 1981 publication by Lucci et al. [16] suggests, at first glance, a similar phenomenon.

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However, they postulate that two of their cases of CSM had syringes secondary to the cervical spondylosis. Although no CT photographs of these cases were shown, they stated that the nonenchanced CT scans revealed cervical intramedullary cavities. In 1982, Tsuji [17] mentioned a case of cervical canal stenosis and syringomyelia, but gave no details. Another single case was reported in 1983 by Mossman and Jestico [18]. They performed an immediate and a delayed (6 hr) CT scan of the cervical spine after intrathecal injection of metrizamide in a patient who already had a laminectomy for CSM. The delayed scan revealed a large-enhancing cavity or syrinx in the center of the spinal cord.

Despite the recent suggestion that the various forms of secondary syringohydromyelia are exceptionally rare [19], we elected to follow the advice of Quencer et al. [20]: "This case [of spinal cord glioma] points out the need to perform delayed metrizamide CT even in the face of normal myelography when there is a strong clinical indication of spinal cord abnormality." We wished to investigate the possibility that the aforementioned inconsistencies found with CSM may, in fact, be explained by radiographically demonstrable intramedullary lesions (i.e., syringomyelia ex-vacuo) secondary to various causes of cervical cord compression.

Materials and Methods

From July 1984 through January 1985, we investigated seven consecutive patients admitted with symptoms arising from severely stenotic spinal canals of developmental and acquired origin. In each case, the patient presented with a long (months or years) history of progressive myelopathy and with clinical finidngs of sensory, motor and reflex deficits consistent with a cervical myelopathic process.

All the patients in this study had low lumbar punctures with instillation of 10 ml of iopamidol (Niopam 300). The contrast material was then manipulated into the cervical region, and routine filming was done. Subsequently, an immediate CT was performed followed by a delayed CT 10–12 hr later. Earlier (6–8 hr) and later (24 hr) scanning done in these cases showed inadequate contrast-medium penetration at 6–8 hr and poor cord/cavity differentiation (i.e., nearly isodense) at 24 hr. The CT examination was performed both at the level of maximal spinal stenosis as well as above and below the stenotic canal, with the exam extending into the upper cervical and lower thoracic regions, respectively. Narrow window level and width CT filming was performed on the delayed study to electronically enhance the accumulation of contrast medium within the spinal cord at these various levels. In examinations where the sections were contiguous, reformatting was performed in both the sagittal and coronal planes to further elucidate the nature and extent of the lesion.

All of the myelographic procedures were performed on a standard radiographic tilt table under fluoroscopic control. The CT examination was carried out on a General Electric 9800 scanner (Milwaukee, WI). Slice thicknesses of 3, 5, and 10 mm were obtained depending on the level examined. Our control group of patients included a total of eight individuals examined for reasons other than myelopathy. They had the identical myelographic procedure (the same amount and concentration of contrast medium was administered) and the same type of subsequent CT evaluation. None of these controls demonstrated any evidence of abnormality on the immediate CT examination, nor was there an abnormal accumulation of contrast medium within the spinal cord on delayed evaluation. No patient in either the control group or the tested group had any adverse reaction or clinical deterioration during or after the intrathecal iopamidol injection.

Case Reports

Case 1

This 58-year-old woman was first seen because she had been unable to walk for 1 year on account of weakness. On examination, there was mild touch and pinprick sensory deficit in the C8 and T1 distribution bilaterally and severe hypesthesia (all sensory modalities) below the knees in both legs. The ankle jerks were absent, but knee jerks and upper limb deep tendon reflexes were 3+. Bilateral Hoffmann's reflexes were present. Plantar stimulation caused down-going toe reflexes. There was a positive Romberg test. The upper limbs were moderately weak and spastic throughout. The lower limbs were moderately weak throughout, but the patient could not walk, mainly because of severe sensory ataxia.

The myelogram showed a nearly complete block at the C6–C7 interspace due to developmental stenosis coupled with acquired spondylotic changes as well as severe lumbar stenosis. Immediate CT evaluation at this same cervical level revealed a remarkably atrophied spinal cord surrounded by a moderate-sized subarachnoid space and sagittal narrowing of the spinal canal (Fig. 1A). A repeat CT exam 10 hr later at the C6–C7 level indicated that contrast medium had collected inside the cord and concentrated in areas corresponding to the right and left gray-matter zones (Fig. 1B). This "snake-eyes" appearance was seen only at and near the level of the spondylotic bar. However, extending above (Fig. 1C) and below (Figs. 1D and 1E) this level was a separate, longitudinally oriented, "pencil-shaped" zone of intramedullary contrast collection. This central, longitudinal lesion was well seen only when the window setting of the CT computer console was sufficiently narrow to enhance the accumulated contrast (Figs. 1D and 1E).

The patient underwent a lumbar laminectomy for decompression of the cauda equina. There has been gradual improvement in her lower-limb function. To date, a cervical decompression has not been done.

Case 2

This 40-year-old man was seen in May 1984. He had a 3-year history of electriclike sensations radiating into his back, left thumb, and left leg when he flexed his neck. He also complained of general body weakness. On examination, he was found to have a positive Lhermitte's sign. There was touch and pinprick sensory deficit over the left C8 and T1 zones. His strength was good, but the deep tendon reflexes were 3+ to 4+ throughout and there was a left ankle clonus.

Radiographs of the cervical spine revealed developmental spinal stenosis. A subsequent myelogram demonstrated interruption of contrast medium by a C6–C7 spondylotic process. An immediate CT study at the same level confirmed the narrowed sagittal diameter of the spinal canal, the posterolateral osteophyte, and the associated spinal cord atrophy (Fig. 2A). On delayed CT examination 10 hr later, an intramedullary lesion appearing as a pencil-shaped contrast density was found extending longitudinally below the point of compression and into the thoracic spinal cord (Fig. 2B). The "snake-eyes" lesions were not identified in this patient.

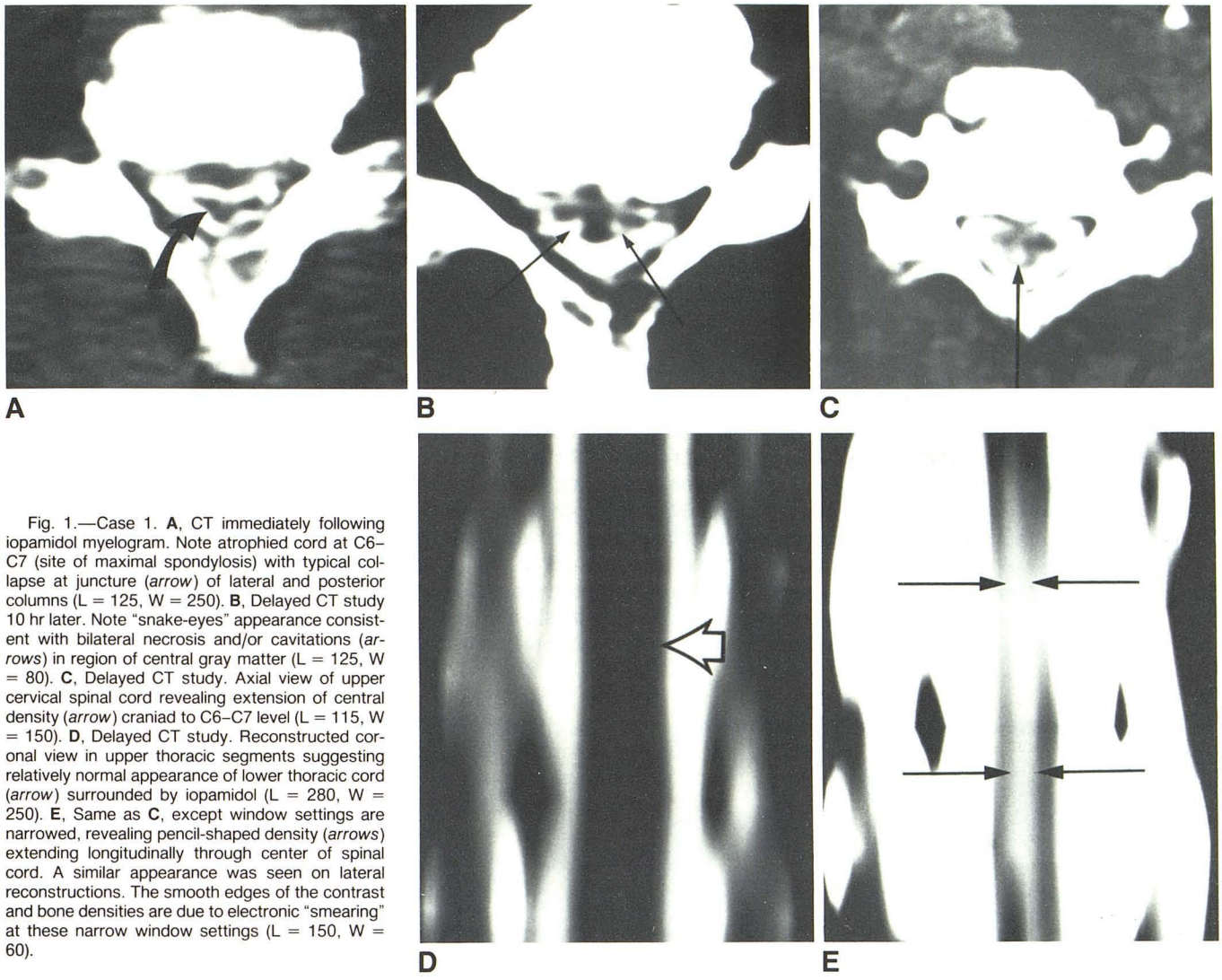

Fig. 1.—Case 1. **A**, CT immediately following iopamidol myelogram. Note atrophied cord at C6–C7 (site of maximal spondylosis) with typical collapse at juncture (*arrow*) of lateral and posterior columns (L = 125, W = 250). **B**, Delayed CT study 10 hr later. Note "snake-eyes" appearance consistent with bilateral necrosis and/or cavitations (*arrows*) in region of central gray matter (L = 125, W = 80). **C**, Delayed CT study. Axial view of upper cervical spinal cord revealing extension of central density (*arrow*) craniad to C6–C7 level (L = 115, W = 150). **D**, Delayed CT study. Reconstructed coronal view in upper thoracic segments suggesting relatively normal appearance of lower thoracic cord (*arrow*) surrounded by iopamidol (L = 280, W = 250). **E**, Same as **C**, except window settings are narrowed, revealing pencil-shaped density (*arrows*) extending longitudinally through center of spinal cord. A similar appearance was seen on lateral reconstructions. The smooth edges of the contrast and bone densities are due to electronic "smearing" at these narrow window settings (L = 150, W = 60).

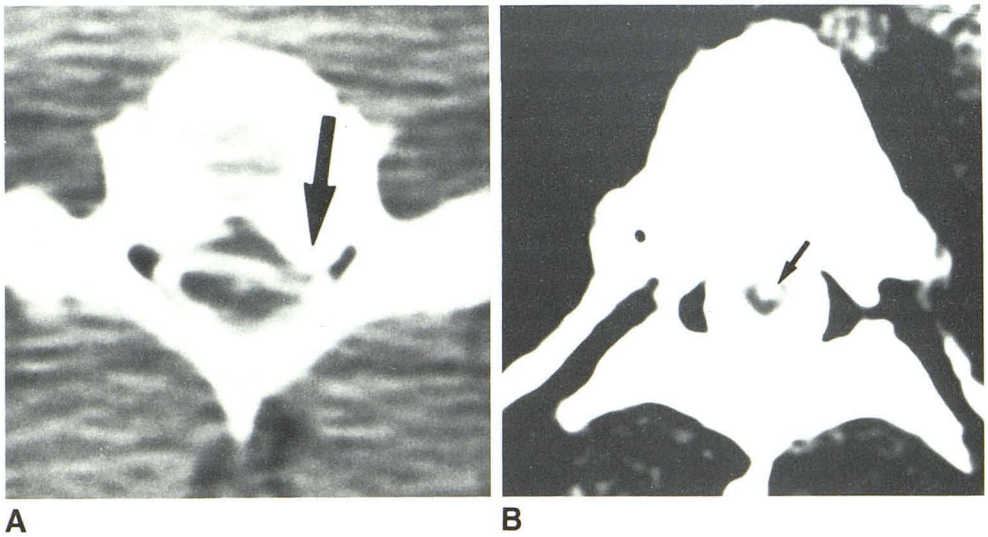

Fig. 2.—Case 2. **A**, Immediate postmyelographic CT study demonstrating C6–C7 osteophyte formation (*arrow*) and cord atrophy with flattening (L = 75, W = 350). **B**, Mid-thoracic cord axial CT examination 10 hr later. The centrally enhancing lesion (*arrow*) is seen when narrow window settings are used (L = 85, W = 60).

The patient underwent a cervical laminectomy. Postoperatively, his neurologic exam has been unchanged, but the electric shocks he experienced during neck flexion have disappeared.

Case 3

This 64-year-old man had a 2-year history of stiffness and weakness in both legs, with his legs often giving way while he was walking. On examination, he had good extremity strength and sensation, but his tone was moderately spastic in all four limbs. Deep tendon reflexes were 4+ in his arms and legs. He had positive Babinski and Hoffmann signs. He walked slowly with a spastic gait. The Romberg test was normal.

The conventional radiographs and myelogram revealed developmental stenosis and a prominent spondylotic bar at C3–C4. Bar and cord atrophy were confirmed on an immediate CT study (Fig. 3A). The typical "Napoleonic hat" appearance (upside down in the CT view) of the spinal cord in cross section was seen. Immediate CT examinations above and below the primary, compressive lesion showed moderate but less severe atrophy. At the C3–C4 level a 10-hr delayed CT scan (Fig. 3B) demonstrated the "snake-eyes" appearance resulting from an apparent intramedullary collection of contrast medium in the right and left central gray matter. Above (Figs. 3C and 3D) and below the C3–C4 spondylosis, proper settings of window levels allowed presentation of centrally located intramedullary contrast enhancement, the contrast medium that was presumably lying in central zones of necrosis and/or cavitation.

The patient underwent a decompressive cervical laminectomy. To date, his neurologic examination remains as it was before surgery.

Case 4

This 57-year-old man had had a ventriculoperitoneal shunt inserted 4 years earlier for normal pressure hydrocephalus, which was diagnosed as a cause of his gait disturbance. In the same year, a metrizamide myelogram was attempted by a C1–C2 level puncture, but without success. The patient's gait disturbance accelerated into a progressive quadriparesis and spasticity of unclear origin (i.e., from normal pressure hydrocephalus, cord injury during attempted myelography, or cervical spondylosis). A zone of suspended, painful dysesthesia from C4 to T4 levels evolved. On examination, there was mild loss of temperature and pinprick sensation bilaterally

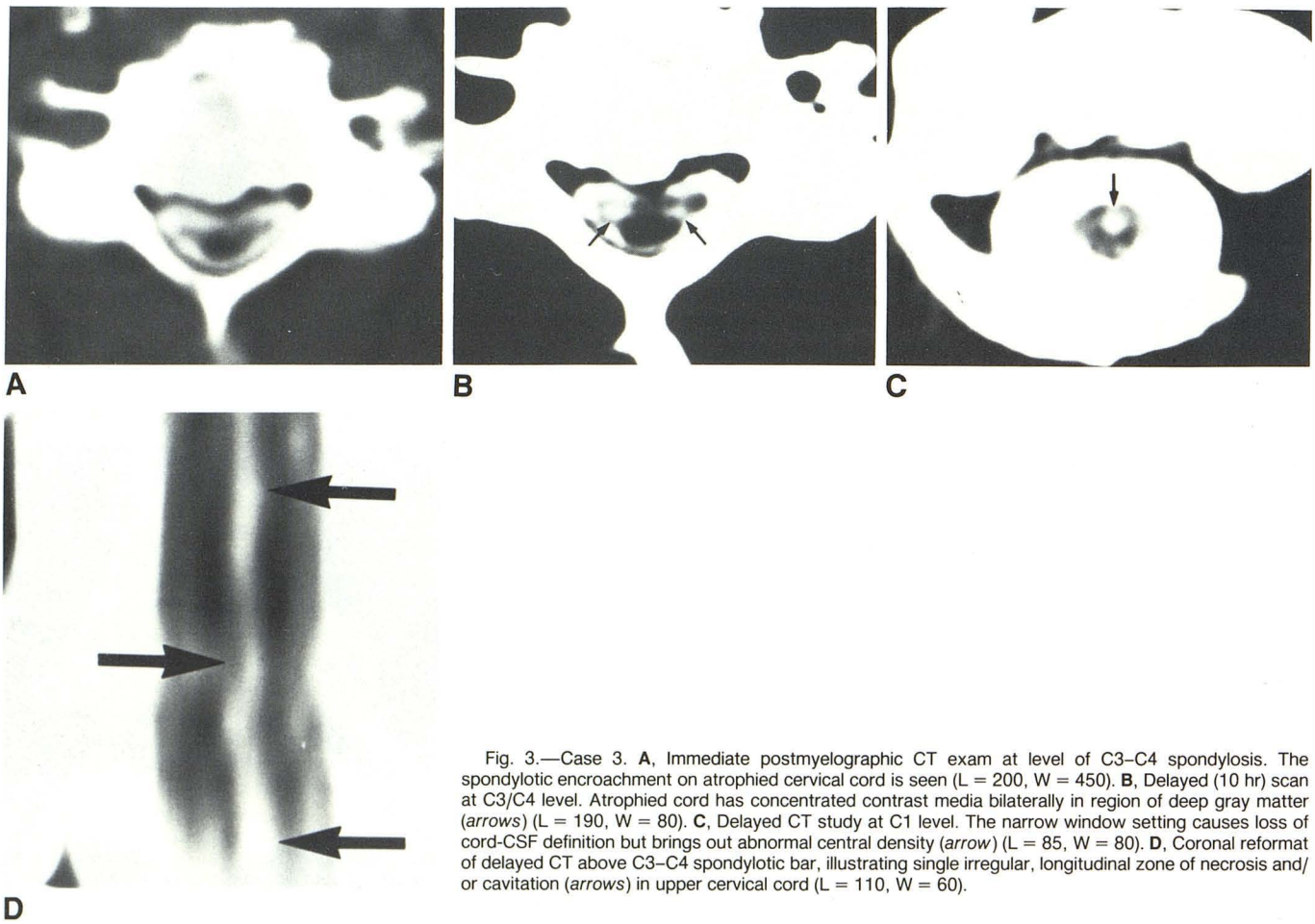

Fig. 3.—Case 3. **A**, Immediate postmyelographic CT exam at level of C3–C4 spondylosis. The spondylotic encroachment on atrophied cervical cord is seen (L = 200, W = 450). **B**, Delayed (10 hr) scan at C3/C4 level. Atrophied cord has concentrated contrast media bilaterally in region of deep gray matter (*arrows*) (L = 190, W = 80). **C**, Delayed CT study at C1 level. The narrow window setting causes loss of cord-CSF definition but brings out abnormal central density (*arrow*) (L = 85, W = 80). **D**, Coronal reformat of delayed CT above C3–C4 spondylotic bar, illustrating single irregular, longitudinal zone of necrosis and/or cavitation (*arrows*) in upper cervical cord (L = 110, W = 60).

from C4 to T4, inclusively. He had a mild spasticity of all limbs, but his strength was good. There were bilateral, positive Hoffmann's reflexes and a normal response to plantar stimulation. Deep tendon reflexes were 3+ throughout.

The myelogram revealed a developmentally narrow canal with spondylotic bars at C4–C5 and C5–C6. A CT scan done immediately thereafter demonstrated a severely atrophied cord surrounded by a moderate-sized subarachnoid space. The delayed study failed to suggest any pencil-shaped central cavitation, but the classical double-barreled "snake-eyes" abnormality was seen at the level of the spondylosis (Figs. 4A and 4B). These intramedullary lesions appeared to lie within the central gray matter.

The patient has been followed in the neurology clinic. No cervical decompression has been performed, and his neurologic status remains unchanged.

Case 5

This 48-year-old man was seen for a prolactinoma that was well controlled by bromocriptine. His chief complaint was difficulty in walking and occasionally falling during the past year. On examination, there was slight, bilateral optic atrophy and reduced visual acuity. His strength was good, but severe spasticity was present in all four limbs. Deep tendon reflexes were 4+ throughout, and Hoffmann and Babinski signs were positive bilaterally. The sensory examination was within normal limits.

The axial CT and sagittal reformat showed ossification of the posterior longitudinal ligament at C5 and C6 levels (Fig. 5A). Marked focal cord atrophy was present at these same levels. A delayed (10 hr) CT exam at the level of cord compression again illustrated the abnormal bilateral contrast collections in the central gray-matter zones (Fig. 5B). Above and

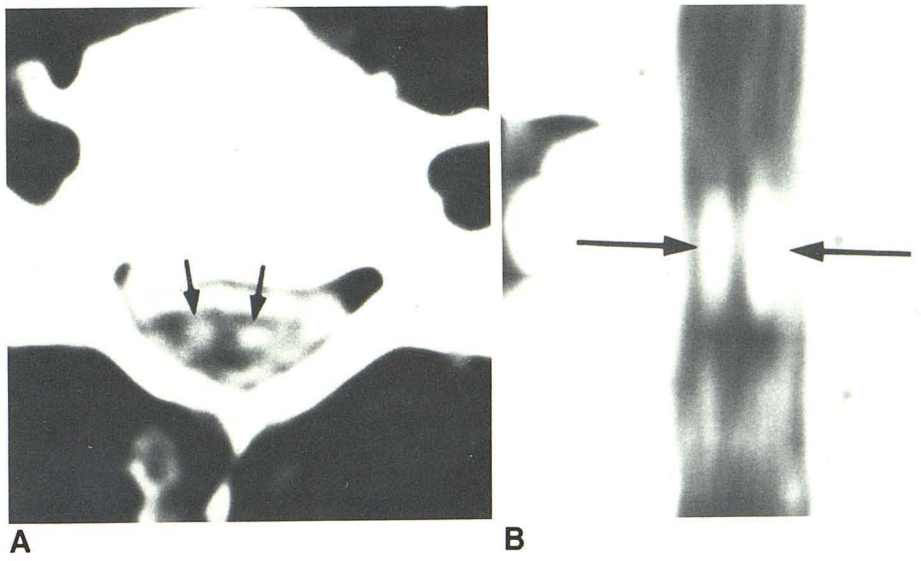

Fig. 4.—Case 4. **A**, This delayed postmyelographic CT scan (10 hr) at C5 illustrates bilateral collection of contrast media in central gray zones (*arrows*) giving typical "snake-eyes" appearance at level of spondylosis (L = 120, W = 150). **B**, The double-barreled, 1-cm-long lesion (*arrows*) is well seen on coronal reformat (L = 120, W = 150).

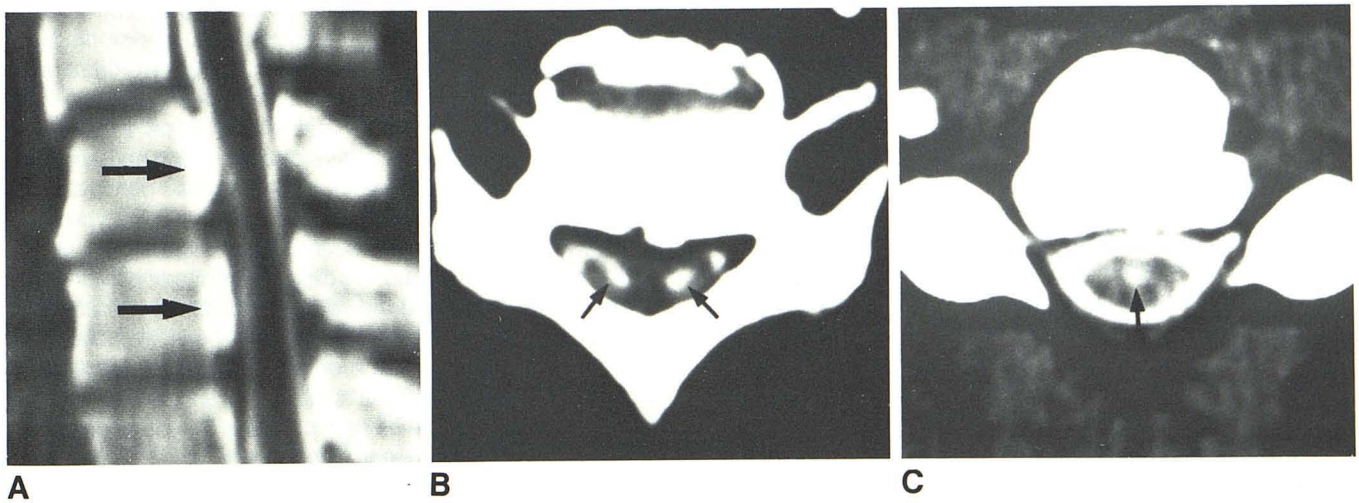

Fig. 5.—Case 5. **A**, Lateral reformat of immediate CT myelogram at midcervical level. Ossification at the posterior longitudinal ligament at C5 and C6 is seen (*arrows*) and adjacent cord is atrophied (L = 330, W = 850). **B**, Delayed (10 hr) CT scan at C5 level. Window levels set to show bilateral, intramedullary collection of contrast media (*arrows*) (L = 160, W = 150). **C**, Delayed CT scan at C3 with narrow window settings revealing abnormal central intramedullary enhancement (*arrow*) (L = 100, W = 100).

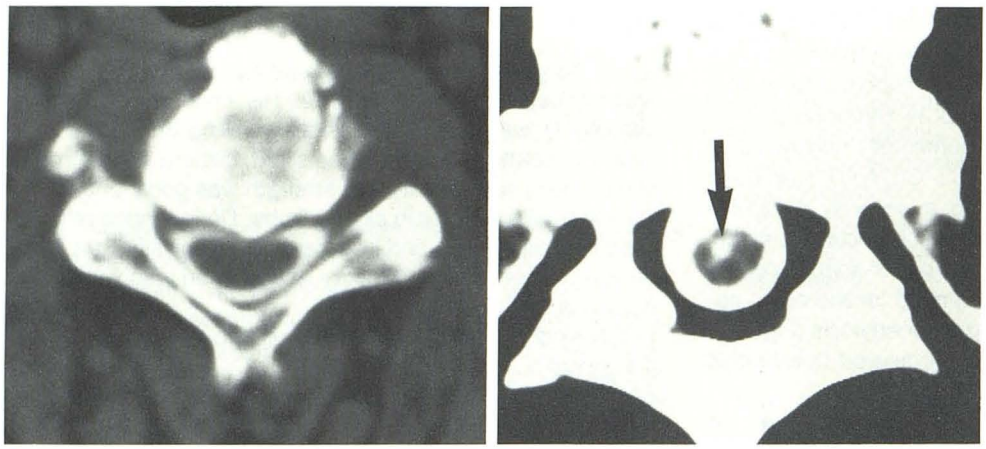

Fig. 6.—Case 6. **A**, Initial CT section at level of cervical spondylosis immediately after iopamidol myelogram. Note moderate flattening of spinal cord (L = 70, W = 650). **B**, Delayed (12 hr) CT scan in lower thoracic region. Iopamidol concentrated (*arrow*) within spinal cord is noted at narrow window settings (L = 75, W = 60).

below the levels of ossification of the posterior longitudinal ligament, the spinal cord appeared relatively normal at wide window settings. However, at narrower window levels (Fig. 5C), these same sites now were seen to have centrally located, abnormal contrast collections within the spinal cord.

A decompressive cervical laminectomy was done. Postoperatively, there has been minimal improvement in the patient's gait and spasticity.

Case 6

This 65-year-old man had a history of cervical injury. He complained chiefly of pain in the left thumb and index finger that had been present for several months. He had a 3-month history of electriclike sensations radiating into both arms and legs during neck flexion. On examination, his reflexes, strength, and tone were normal. Touch and pinprick sensations in the thumb and index finger were reduced bilaterally. A positive Lhermitte's sign was present.

Conventional spine films revealed mid- and lower-cervical developmental spinal stenosis, Forestier's disease (idiopathic skeletal hyperostosis) of the cervical spine, and an old fracture of the hyperostotic mass at C6. Myelography with CT demonstrated moderate cord atrophy (Fig. 6A). A delayed CT scan failed to show any necrosis/cavitation in the cervical region, but an intramedullary density coursed through the thoracic spinal cord (Fig. 6B).

This patient's signs and symptoms have remained unchanged without surgical intervention.

Case 7

This 63-year-old man complained of neck pain, loss of strength in all limbs, and difficulty urinating for 8 months. On examination, he had a wide-based, spastic gait. There was marked spasticity and mild weakness in all four limbs. Sensation was intact. Deep tendon reflexes were 4+ throughout. There was ankle clonus on the right, and bilaterally positive Babinski and Hoffmann reflexes.

The iopamidol myelogram demonstrated a prominent C4–C5 spondylotic bar with partial obstruction to contrast flow in cervical extension. The subsequent CT demonstrated a markedly atrophic cervical spinal cord (Fig. 7A). "Double-barrel" necrosis/cavitation was seen in the mid-cervical region at 10 hr, and contrast material had collected centrally within the thoracic cord (Fig. 7B), but no abnormal accumulation could be identified craniad to C4–C5.

A C4–C5 diskectomy by the anterior approach was carried out. Since then, there has been little improvement in the patient's clinical symptoms and signs, although his clinical course seems to have stabilized.

Discussion

It appears that special techniques of CT examination of the spinal cord 10–12 hr after water-soluble contrast (iopamidol) myelography permit demonstration of intramedullary lesions at and distant from levels of known spinal cord compression. These studies, while placing additional demands on time and scheduling, provide rather solid evidence, not heretofore demonstrated in a consecutive series of patients, that spinal cord compression can and does create both local and remote intramedullary spinal cord zones of destruction and apparent cavitation. The occurrence of such remote, pencil-shaped lesions (whether they are necroses, cavitations, or hydromyelia ex-vacuo) are frequently seen in severe cases and clearly explains some of the puzzling clinical aspects of compressive cervical myelopathy and the surgical results mentioned in the introduction. Certainly a patient with mid-cervical spondylosis and secondary necrosis and/or cavitation (syrinx) extending up into the upper cervical spinal cord and down into the thoracic spinal cord can be expected to show signs of muscle atrophy of the hands; electromyographic signs of fasciculations in the arms and legs [8, 21–23]; and misinterpreted signs of motor neuron degeneration, demyelinating disease, amyotrophic lateral sclerosis, or primary syringomyelia [2, 5, 8, 22].

Secondary, usually noncommunicating syringomyelia has

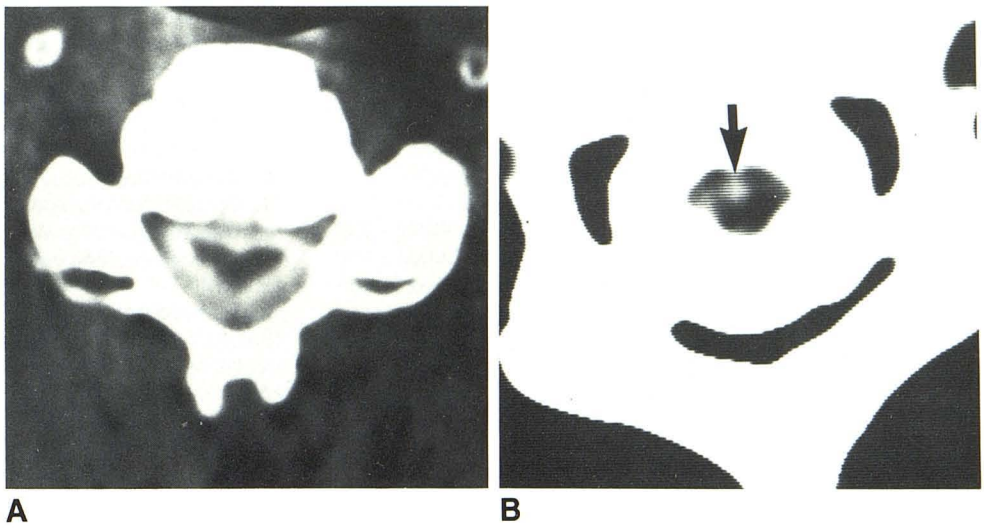

Fig. 7.—Case 7. **A**, Initial CT section near level of cervical spondylosis immediately after iopamidol myelogram. Note severe flattening and atrophy of spinal cord (L = 65, W = 200). **B**, Delayed (10 hr) CT scan in lower thoracic region. Iopamidol concentrated (*arrow*) within spinal cord is noted at narrow window settings (L = 150, W = 60).

been reported in association with spinal cord neoplasms, Pott's disease (tuberculosis) of the spine, spinal trauma, foramen magnum tumors, arachnoiditis (from infection, hemorrhage, trauma, or other inflammatory causes), vascular disorders and infarcts, intramedullary hematomas, malformations of the central nervous system, extramedullary tumors, vertebral body tumors, myelitis, and spondylotic compression of the spinal cord [16, 18, 20, 24–33]. Brain and Wilkinson [4] in 1958, McRae and Standen [28] in 1966, and Smith [12] in 1968 suggested the possible coexistence of syringomyelia and cervical spondylosis. As previously noted, Lucci et al. [16] in 1981 and Mossman and Jestico [18] in 1983 related instances of CSM in which CT had shown signs of intramedullary cavitation.

Sotaniemi et al. [34] observed, "When an intramedullary cavitation filled with contrast medium is demonstrated, the diagnosis of syringomyelia seems clear." However, at this point we cannot confirm whether the abnormal intramedullary collections of contrast material demonstrated in our cases of CSM represent actual cavities (syringomyelia) or whether they are necrotic tissue retaining contrast medium. Stevens et al. [32] found that 15% of their operated cases of posttraumatic syringes diagnosed by delayed CT examinations after myelography did not have intramedullary cavities. Instead, necrotic zones appeared to be present. Certainly, liquifaction necrosis often precedes cavitation.

The use of delayed CT examinations after subarachnoid injection of water-soluble contrast material has been in common use for demonstrating posttraumatic syringes [31, 34], classical syringomyelia or hydromyelia [19, 35–39], syrinx secondary to arachnoiditis [27], and an occasional cystic spinal cord glioma [20]. Many of the cerebrospinal fluid (CSF)-enhanced CT studies nicely showed the syringes after a delay of only 4–8 hr. The fact that our lesions were seen best with iopamidol at 8–12 hr suggests one or more of the following: (1) there may be differences between the transparenchymal spinal cord diffusion rates of metrizamide (used mostly by others) and iopamidol (used in our cases); (2) the cavities in our cases are much smaller, requiring a longer diffusion and collection time; and (3) some or all of the lesions in our cases represent a form of central cord necrosis (without cavitation), which more slowly concentrates the inwardly diffusing contrast media.

We expect that in the future MR studies will reveal these lesions as spinal MR technology improves. The MR studies of cervical spondylosis to date have been somewhat useful for evaluating spinal stenosis and spondylosis [40, 41], but spinal cord anatomic details have been limited. Such imaging has been much more useful for classical syringomyelia [19, 38, 40–42].

Up to now, most CT examinations of cases of CSM have been performed either without contrast enhancement or with CSF-contrast enhancement studied without delay [16, 43, 44]. The same is true for cases of ossification of the posterior longitudinal ligament [45] and the developmentally narrowed cervical canal [46]. Whereas Lucci et al. [16] apparently were the first to relate syringomyelia causally to cervical spondylotic cord pressure, Mossman and Jestico [18] in 1983 were the first to report delayed CT scanning after intrathecal injection of contrast medium in one postoperative case of CSM and thus demonstrate the presence of a large syrinx. While one possibly may assume that these were incidentally coexisting lesions in their three cases, our findings confirm a significant relationship between the chronic spinal cord pressure and intramedullary spinal cord cavitations.

CSF-enhanced contrast medium may enter a classical (Arnold-Chiari type) hydromyelic cavity by one of two postulated routes: downward reflux via the fourth ventricle or transmyelon migration [37]. In our cases, we believe that the iopamidol crossed the pia and passed through the parenchymal, extracellular spaces of the white matter and then into the lesions. This is consistent with the theories and observations of other investigators regarding metrizamide: water-soluble contrast material in the CSF penetrates the brain and spinal cord through extracellular spaces communicating directly with the CSF [47–49]. Isherwood et al. [48] noted it took 3–6 hr for intrathecally administered metrizamide to achieve an equilibrium between the spinal cord parenchyma and the surround-

ing CSF of patients. It is possible that iopamidol has a different equilibrium time than does metrizamide, one factor that may account for the longer (10–12 hr) delay to reach optimum visualization of the lesions in our cases. As a control, we have studied eight cases without evidence of spinal cord compression. The focal intramedullary contrast collections were not seen at 10 or 12 hr.

We cannot give precise measurements of the lesion demonstrated on our delayed CT scans. Observed absolute and relative sizes of the imaged lesion, spinal cord, and surrounding CSF vary depending on the operator-selected window-density settings of the CT console [50]. We can state that: (1) the lesions shown are smaller than most previously demonstrated syringomyelic cavities; (2) no communication with the fourth ventricle has been proven; and (3) two separate types of lesions have been demonstrated. On the basis of our radiologic studies, we cannot confirm the histologic classification of the two types of lesions. We can postulate that the focal "snake-eyes" lesions at the cervical site of compression represent zones of repeated traumatic and/or ischemic damage leading to necrosis with almost certain subsequent deep gray-matter cavitation, or local syringomyelia ex-vacuo [11, 51–54]. The somewhat smaller, pencil-shaped lesions extending caudal and/or cranial to the primary compressive site may represent similar liquifaction necrosis and cavitation near the anterior part of the dorsal columns [3, 11, 14, 51, 55–57] or distant syringomyelia ex-vacuo. Alternatively, this may represent a dilated central canal, or hydromyelia ex-vacuo, secondary to severe atrophy of ascending and descending spinal cord pathways. This combination of changes explains why, outwardly, the spinal cord exhibits the markedly severe focal atrophy at the site of maximal cervical compression while the remainder of the cord only shows moderate, diffuse shrinkage. At the same time, it may well be the key to understanding and interpreting the medical and surgical dilemma of patients manifesting compressive myelopathy.

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