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Neuroimaging of Scuba Diving Injuries to the CNS

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Diving accidents related to barotrauma constitute a unique subset of ischemic insults to the CNS. Victims may demonstrate components of arterial gas embolism, which has a propensity for cerebral involvement, and/or decompression sickness, with primarily spinal cord involvement. Fourteen patients with diving-related barotrauma were studied with MR imaging of the brain and spinal cord and with CT of the brain. In four patients with presumed cerebral gas embolism, cranial MR was abnormal in three patients while CT was abnormal in only one. Twelve patients had decompression sickness and spinal cord symptoms. MR documented spinal cord abnormalities in three patients. However, scans obtained early in our study were frequently limited by technical constraints.

MR of the brain is more sensitive than conventional CT scanning techniques in detecting and characterizing foci of cerebral ischemia caused by embolic barotrauma to the CNS. Although spinal MR may be less successful in the localization of spinal cord lesions related to decompression sickness, these lesions were previously undetectable by other neuroimaging methods.

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Scuba (self-contained underwater breathing apparatus) diving has enjoyed a tremendous surge in popularity, both in this country and abroad. There are more than three million currently certified scuba divers in the United States alone, and an additional 500,000 individuals are newly certified each year. Although the overall incidence of diving-related CNS barotrauma is small, the increasing number of dives performed each year will result in an increasing number of previously healthy individuals who will be affected by this disorder. Neurologic symptoms may range from minimal dysesthesias to complete quadriplegia, encephalopathy, or death. Divers affected by dysbaric insults to the CNS can be clinically divided into two subsets: cerebral involvement, which is generally believed to result from arterial gas embolism, and spinal cord involvement, otherwise known as spinal cord decompression sickness. Characterization of the site, extent, and origin of these lesions previously has relied on history and physical examination. Neuroimaging traditionally has not played a major role in the assessment of these patients, although an objective imaging method would clearly aid physicians in caring for them as well as aid in defining the pathophysiology of the lesions.

Materials and Methods

From July 1985 to April 1987, 14 patients, 13 men and one woman 16–49 years old (mean, 37 years), were referred to the Duke University Medical Center for diving-related injuries. Two patients had purely cranial signs and symptoms suggestive clinically of arterial gas embolus, while 10 had symptoms of spinal cord dysfunction alone suggesting spinal decompression sickness. Two patients had symptoms consistent with both cerebral and spinal levels of CNS involvement.

MR imaging was initially performed on a GE prototype system operating at 1.5 T. Later scans were obtained on clinical 1.5-T GE systems. Axial head images were obtained with T1-weighted, 500/20–25/2 (TR/TE/excitations), and T2-weighted, 2000–2500/80/2, acqui-

TABLE 1: Dysbaric Diving Injuries in Patients with Clinical Symptoms of Cerebral Involvement

Case No.	Age	Gender	Findings		
			Clinical	Head CT	Head MR
1	40	M	AGE, ophthalmoplegia, ataxia, vertigo	Not performed	Left cerebral peduncle: punctate lesion
2	42	M	AGE (and DCS), lethargy and dysphasia (C4 sensory level), quadriplegia	Negative	Large cortical and centrum semiovale infarcts: bilateral parietooccipital regions
3	30	M	AGE (and DCS), grand mal seizure (T8 sensory level)	Left parietal infarct	Left parietal and right temporal infarcts
4	26	M	AGE vs vestibular barotrauma, vertigo, hearing loss in right ear	Negative	Negative

Note.—AGE = clinical arterial gas embolism; DCS = clinical spinal decompression sickness.

sitions with standard head coils (5-mm slice thickness, 2.5-mm interscan gap, 20-cm field of view, and 128 × 256 matrix). Spinal images were obtained with a 5-in. (12.7-cm) circular surface coil with images obtained in the sagittal (3-mm-thick section and 20-cm-field of view) and axial (5-mm-thick section and 16-cm-field of view) planes. T1- and T2-weighted acquisitions otherwise used the same imaging parameters as the brain images. To reduce total scan time, scans were frequently limited to selected regions of the spine determined on the basis of clinical data.

CT of the brain was performed on a GE 9800 scanner. Scans were obtained without IV contrast material. The posterior fossa was evaluated with 5-mm-thick contiguous slices and the remainder of the brain with 10-mm contiguous slices.

The interval between the diving accident and the initial neuroimaging study ranged from 1 day to 3 months. Excluding the one patient (case 1) with the 3-month interval between injury and presentation, the mean time from accident to initial study was 7 days. MR and CT studies, when obtained in the same individual, were performed within 48 hr of one another. A xenon CT regional cerebral blood flow study was performed in one patient on the second hospital day. This examination was performed during inhalation of a 32% nonradioactive xenon/oxygen mixture with commercially available data processing and blood-flow map generation.*

Neurologic examinations were performed by a staff neurologist to characterize the extent of clinical compromise. Patients were treated with therapeutic hyperbaric recompressions and auxiliary therapy at the F. G. Hall Center by staff of the departments of medicine and anesthesiology and the Hall Center.

An autopsy including detailed neuropathologic examination of the brain and spinal cord was performed on one patient in this series (case 2) who died of a massive pulmonary embolus.

Results

Cerebral Involvement

Four patients had clinical symptoms and signs indicative of intracranial injury (Table 1). CT identified one lesion in one patient, was negative in two patients, and was not performed in the fourth. The single lesion identified on CT (case 3, Fig. 1A) was a poorly defined area of low attenuation in the subcortical white matter of the left parietooccipital region. This lesion was also demonstrated by MR (Fig. 1B), but a second lesion in this patient, identified by MR in the right temporal lobe, was obscured on the CT examination by beam-hardening artifacts.

MR identified four lesions in three patients, but was negative in one patient. The location of two MR lesions corresponded to the neurologic deficits diagnosed by physical examination, but did not correspond for the other two lesions. The one patient in whom no lesion was identified was believed to have had hearing loss and severe vertigo on the basis of vestibular barotrauma.

On the MR examinations, abnormal areas were of increased signal intensity on T2-weighted images. All four lesions involved predominantly the subcortical white matter, although the large bilateral lesion in case 2 also extended to the overlying occipital cortex. One lesion consisted of a punctate focus of prolonged T2 in the left cerebral peduncle.

In addition, a single punctate lesion in the frontal centrum semiovale and a small patchy lesion in the right cerebellar white matter (Fig. 2) were detected incidentally in two other divers with spinal symptoms but no evidence of cerebral dysfunction. CT of the patient with the cerebellar lesion was negative; in the other patient, brain CT was not performed.

Spinal Cord Involvement

Twelve patients had clinical symptoms and signs referable to spinal cord decompression sickness. MR demonstrated patchy areas of increased signal intensity in the white-matter tracts on T2-weighted images in three patients, in each case corresponding to an area of the cord believed to be clinically involved (two cervical, one low thoracic). The MR abnormali-

* Xenon package, General Electric Medical Systems, Milwaukee, WI.

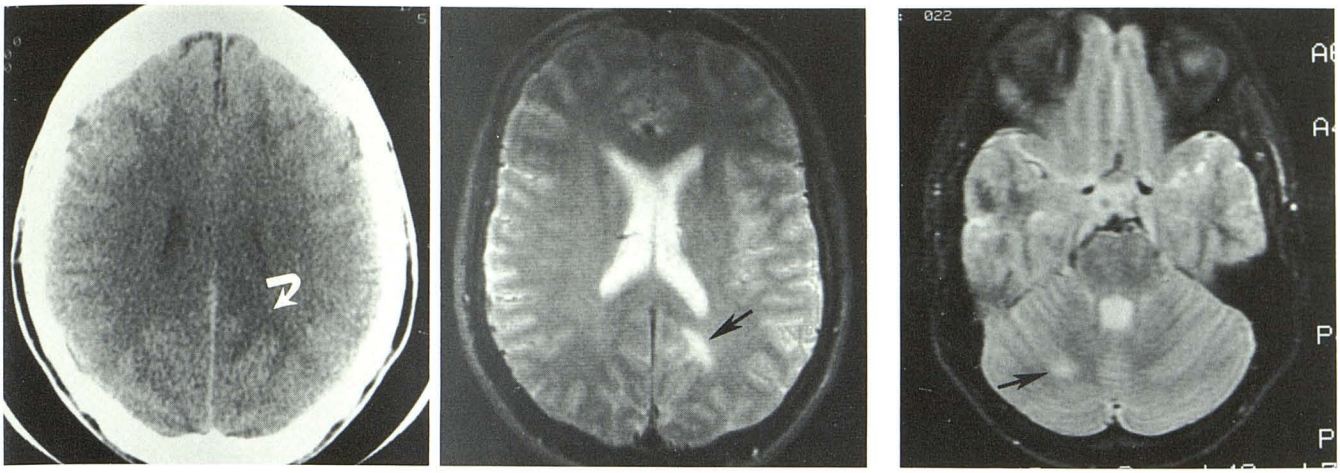

Fig. 1.—Case 3.
***A*, Unenhanced CT scan shows subcortical hypodensity in left parietooccipital region (*arrow*).**
***B*, T2-weighted MR image, 2000/80. Corresponding infarct is of increased signal intensity. A second lesion seen in right temporal lobe on MR was obscured on CT by beam-hardening artifacts.**

Fig. 2.—Case 5. MR image, 2000/80. Asymptomatic small patchy lesion in deep white matter of right cerebellar hemisphere (*arrow*) in patient with decompression sickness without signs or symptoms of cerebral dysfunction.

ties were predominantly confined to the dorsal aspect of the cord in each of these three cases.

Representative Case Reports

Case 2: Cerebral Arterial Gas Embolism and Spinal Decompression Sickness

A 42-year-old experienced male diver made a dive to 18 m seawater for 40 min. On surfacing, he became unconscious and had a major motor seizure. Despite rapid recompression, he remained confused, totally quadriplegic, and manifested a C4 sensory level. A CT scan on admission to Duke 2 days after the injury was normal (Fig. 3A), but MR examination of the brain revealed extensive areas of prolonged T2 involving the parietal white matter and cortical portions of the parietooccipital lobes (Figs. 3B and 3C). MR of the cervical spine also showed areas of increased T2 signal in the dorsal columns of the spinal cord at C4–C6 levels (Fig. 3D). A xenon regional cerebral blood flow study on the third day after the accident demonstrated large areas of diminished perfusion corresponding to the areas of MR abnormality (Fig. 3E).

Six days after the accident, the patient died from a massive pulmonary embolus. Gross inspection of the fixed brain showed only subtle foci of hyperemia consistent with recent cortical infarction. Histologic examination revealed changes of cortical anoxic injury as well as pallor of the myelin of the centrum semiovale, corresponding to the areas of MR abnormality (Fig. 3F). Sections of the cord also revealed focal areas of myelinolysis at the C4–C6 levels, corresponding to the abnormalities seen on MR (Fig. 3G), as well as patchy involvement of the white matter in the upper thoracic cord. These changes were most pronounced in the dorsal columns, but also involved the anterior and lateral columns to a lesser extent.

Case 1: Cerebral Arterial Gas Embolism

A 40-year-old professional diver worked at 27 m seawater for 90 min. Shortly after surface decompression, he noted severe rotary vertigo. These symptoms continued intermittently over the next 3 months. When seen at Duke, neurologic examination revealed dysmetria of eye movement with intranuclear ophthalmoplegia and ataxia. Clinically, he was thought to have had a cerebral air embolus. MR of the brain revealed a small focus of increased signal intensity in the left cerebral peduncle (Fig. 4). Although this lesion did not correspond to his clinical symptoms, the presence of a documented MR abnormality suggested the possibility of a previous cerebral injury and a possible increased risk for diving-related paradoxic embolus. A two-dimensional echo microcavitation (shunt bubble) study subsequently revealed microcavitations in the left atrium spontaneously and with Valsalva maneuver, compatible with a right-to-left shunt at the atrial level. It was thought that the cause of his cerebral dysfunction was a previously unrecognized intermittent right-to-left shunt, with paradoxic arterial gas embolism.

Case 6: Spinal Cord Decompression Sickness

A 44-year-old man was diving at 37 m seawater for 25 min. He surfaced without decompression stops, and noted weakness in his thighs, which shortly progressed to paraplegia without bladder control. Examination at Duke revealed total paralysis below the waist with some weakness of his lower abdominal musculature. A sensory level was noted at T10–T12. MR scans of the brain revealed no abnormality, but MR scans of the spinal cord revealed areas of increased signal within the dorsal columns at approximately the T10 level (Figs. 5A and 5B). In addition, multiple focal areas of increased signal were noted within several thoracic and upper lumbar vertebral bodies (Fig. 5C), possibly representing bone infarcts. Plain radiographs of the spine and a radionuclide bone scan were negative. After a course of hyperbaric recompressions, he was able to stand and walk a short distance.

Discussion

The pathophysiology and mechanism of injury in scuba diving accidents is still somewhat controversial and has been

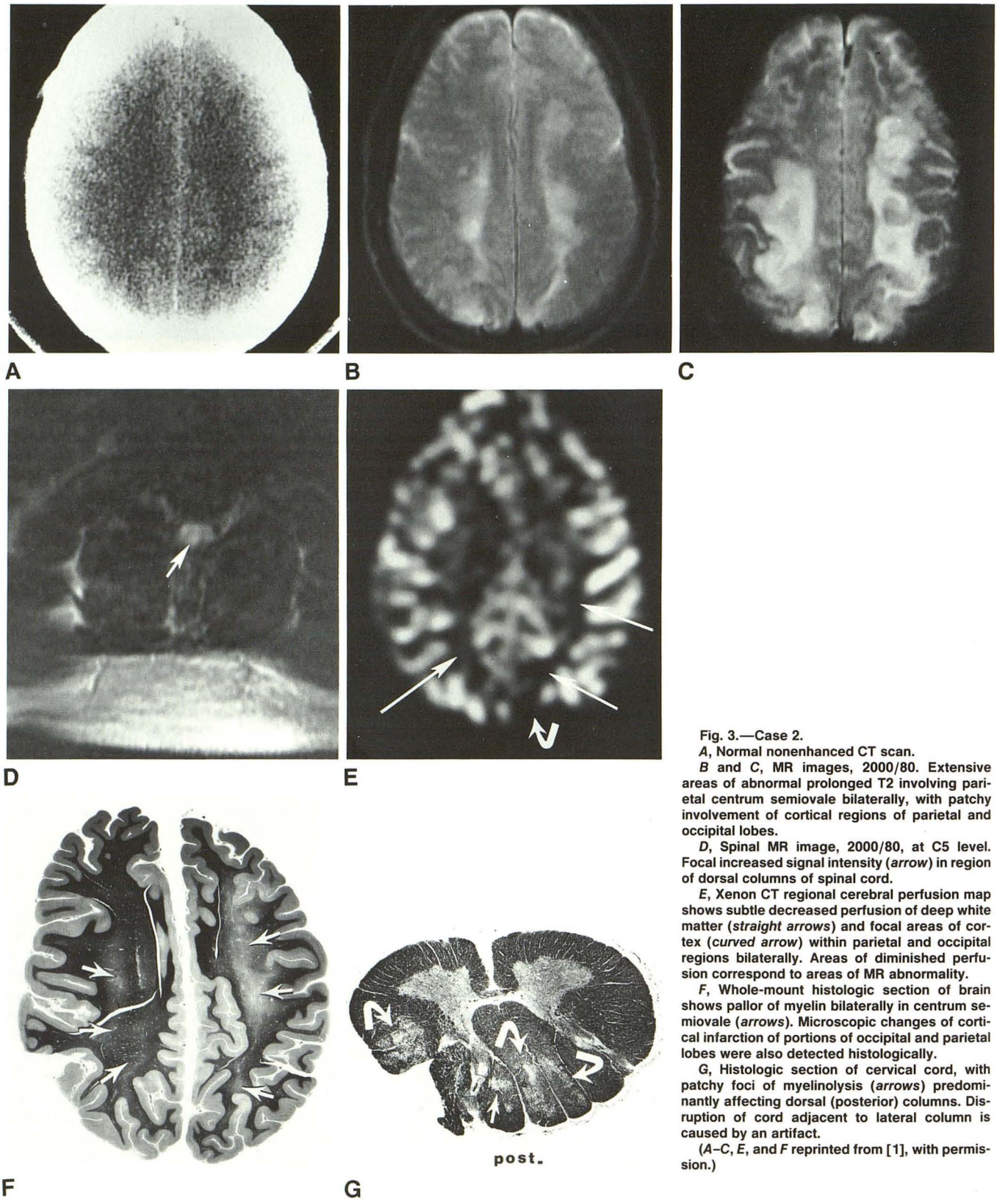

Fig. 3.—Case 2.

A, Normal nonenhanced CT scan.

B and *C*, MR images, 2000/80. Extensive areas of abnormal prolonged T2 involving parietal centrum semiovale bilaterally, with patchy involvement of cortical regions of parietal and occipital lobes.

D, Spinal MR image, 2000/80, at C5 level. Focal increased signal intensity (*arrow*) in region of dorsal columns of spinal cord.

E, Xenon CT regional cerebral perfusion map shows subtle decreased perfusion of deep white matter (*straight arrows*) and focal areas of cortex (*curved arrow*) within parietal and occipital regions bilaterally. Areas of diminished perfusion correspond to areas of MR abnormality.

F, Whole-mount histologic section of brain shows pallor of myelin bilaterally in centrum semiovale (*arrows*). Microscopic changes of cortical infarction of portions of occipital and parietal lobes were also detected histologically.

G, Histologic section of cervical cord, with patchy foci of myelinolysis (*arrows*) predominantly affecting dorsal (posterior) columns. Disruption of cord adjacent to lateral column is caused by an artifact.

(*A–C*, *E*, and *F* reprinted from [1], with permission.)

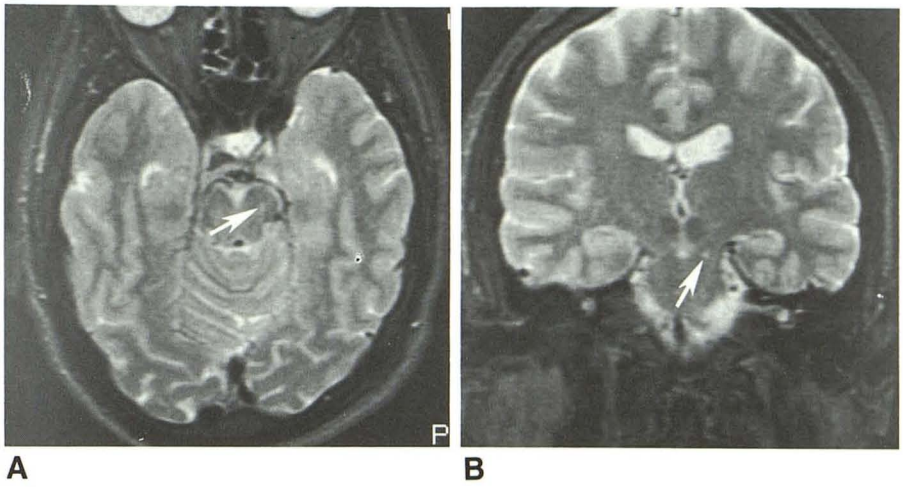

Fig. 4.—Case 1: ophthalmoplegia and vertigo. Axial (*A*) and coronal (*B*) MR images, 2000/80, show punctate lesion in left cerebral peduncle (*arrows*). Lesion does not localize to site of clinical involvement, but may help document presence of intracranial injury.

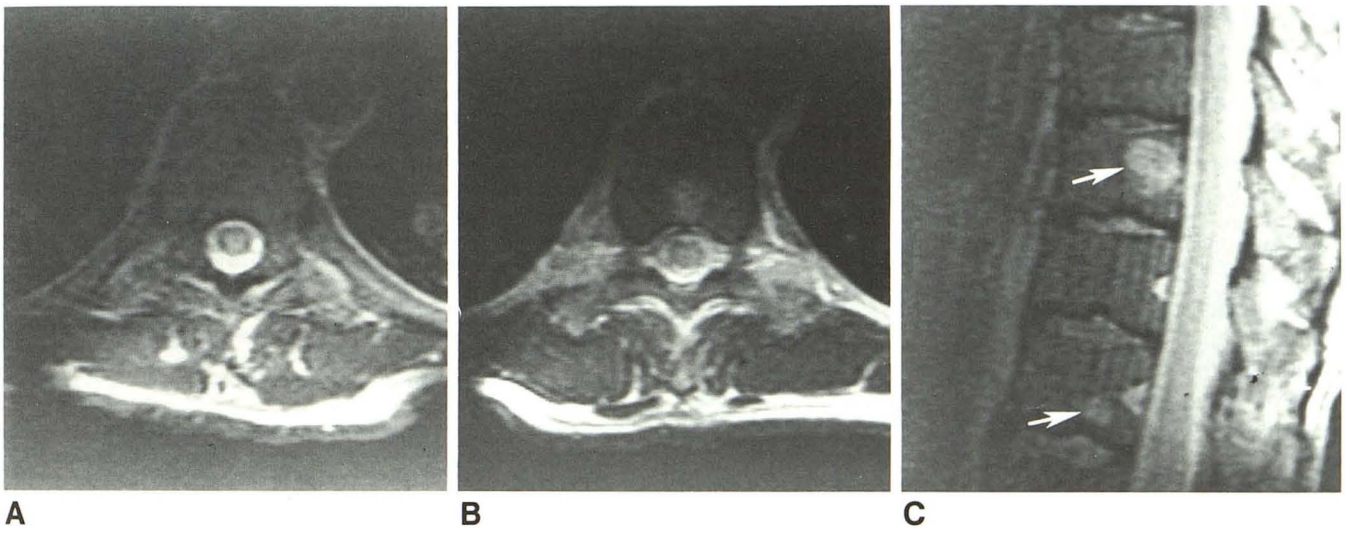

Fig. 5.—Case 6.
***A* and *B*, Axial T2-weighted images, 2000/80, of the spine.**
***A*, Focal abnormal increased signal intensity in central portion and dorsal columns of cord at T10 level.**
***B*, Scan at higher level (T8) shows normal cord anatomy and signal intensity.**
***C*, Sagittal image of lower thoracic spine shows two foci of abnormal increased signal intensity within vertebral bodies (*arrows*) presumed to represent bone infarcts. Cord lesions are believed to be obscured by truncation artifact.**

reviewed in detail [2–5]. Cerebral injuries likely are due to arterial gas embolization, either from pulmonary barotrauma with direct rupture into the arterial system or from paradoxic embolization through previously unrecognized right-to-left shunts (that may only be open during abnormal pressure conditions found during diving). Cerebral air embolism generally occurs within minutes of surfacing, with cognitive dysfunction the predominant feature [6]. Up to 41% of patients may be unconscious when first seen.

More common (77% of CNS diving insults) is the syndrome of spinal decompression sickness [2]. Decompression sickness usually causes progressive sensory or motor loss in the extremities, without loss of consciousness or cognitive disturbance. Symptoms may appear after a delay of 10 min to hours after surfacing. This illness may be related to venous stasis in the large epidural venous channels surrounding the cord, with subsequent venous infarction. Elevated central venous pressure, local microbubble formation, and activation of the coagulation cascade have been implicated. In experimental animals subjected to diving, patchy light and electron microscopic changes involving cord myelin have been reported [7]. Degeneration of the posterior, lateral, and anterior white-matter columns has also been observed in human diving accident victims [8].

Although Kizer [9] described the use of brain CT in the diagnosis of diving-induced neurologic dysbarism syndromes, these illnesses have not been investigated extensively with neuroimaging methods. Imaging has not played a major role in patient management, primarily because of the poor sensitivity of lesion detection with conventional cerebral CT scan-

ning as documented in this series. In addition, imaging of spinal cord ischemic lesions, which constitute the majority of diving-related injuries, has heretofore not been feasible. Thus, until recently physical examination has remained the only objective method of patient evaluation. Our study supports the feasibility of MR imaging for both cerebral and spinal diving-related injuries.

In the four cases of suspected cerebral arterial gas embolism, MR detected four lesions in three patients, whereas CT detected only one lesion. In cases 2 and 3, significant focal areas of presumed infarction were demonstrated. In the one case of arterial gas embolism with negative MR findings (case 4), the clinical complaints of vertigo and hearing loss were probably related to vestibular barotrauma as opposed to ischemic injury. In the fourth case of arterial gas embolism (case 1) as well as in two divers with decompression sickness who had no clinical findings suggestive of cerebral involvement (cases 5 and 7), we noted small focal or punctate abnormalities on cerebral MR. These lesions did not correspond to the patients' presenting neurologic symptoms. Divers are typically extremely healthy individuals, so there is little likelihood of concurrent nondysbaric neurologic disease. One of these patients was shown to have a previously unknown right-to-left shunt by an ECG microcavitation study. We believe that this may be evidence for asymptomatic cerebral injury related to dysbarism, which can be documented by MR, but not shown by CT. The presence of asymptomatic lesions may help to document intracerebral injury, although the observed lesion may not localize to the anatomic site of clinical injury. These findings may prompt further investigation, such as an ultrasound bubble study (as in case 1), to further delineate risk factors for paradoxic emboli.

Of interest is the predominant involvement of subcortical white matter in our series (Fig. 4). Cortical infarcts have been reported with experimental air embolisms [10] as well as with clinical air embolisms [9, 11]. However, some white-matter involvement was present in the cases reported by Kizer [9] and Jensen and Lipper [11], as well as in the experimental studies of De la Torre et al. [12]. Air emboli may be propelled by hydrostatic forces to watershed or distal zones of the cerebral vasculature [13]. After injection of solid emboli, Swank and Hain [14] demonstrated involvement of gray matter with large emboli but white-matter involvement with emboli of less than 17 μm in size. In addition, the size and location of lesions may be influenced further by vasoconstriction and vasodilatation responses of arterial vessels to gas emboli [13].

Spinal cord lesions were detected in only three (25%) of 12 cases of presumed decompression sickness. However, imaging the small detailed patchy pathologic changes of spinal decompression sickness would be expected to be difficult. In addition, detailed imaging of the spinal cord by MR may be additionally hampered by physiologic motion artifacts. Continued progress in surface-coil technology and image resolution has enabled us to improve our sensitivity in this difficult area.

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