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MR Findings in the Brains of Compressed-Air Tunnel Workers: Relationship to Psychometric Results

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Cranial MR imaging was performed in 30 subjects who had been involved in compressed-air tunnel projects in and around Milwaukee, WI. Nineteen of these subjects had been exposed to various degrees of hyperbaric air as calculated by an exposure index (average PSI pressure gauge multiplied by the number of years exposed), while 11 of the subjects were age-matched controls who belonged to the same labor union but had not been exposed to hyperbaric air. All MR scanning was done on a 1.5-T unit, and axial, sagittal, and coronal T1, proton density, and T2-weighted images were obtained. Ventricular size was measured objectively. Foci of increased T2 intensity within deep white matter tracts were evaluated as to number and location, and psychometric testing was performed on both groups to exclude preexisting organic brain disease. The 19 subjects in the experimental group had a statistically higher number ($p = .05$) of white matter lesions (more than 152) than the control group (22 lesions), and 37% of the experimental group had more than 20 white matter lesions each (seven of 19 subjects) while only 18% of the control group had 10 or 11 lesions each. The experimental group had a five times higher risk than the control group of having high-grade lesions, and a high statistical correlation ($p = .02$) was found between the number and severity of lesions in the experimental group as compared with the control group when linear trend analysis was performed. No correlation was found between exposure index, MR grade or number, or aseptic necrosis. Ventricular size was normal in all subjects. The distribution of white matter lesions was 100% in the centrum semiovale and 50% in the optic radiations. Additionally, three subjects in the experimental group had two internal capsule lesions and one basal gangliar lesion.

Our results suggest that a much higher risk of cerebral injury exists among workers who have been exposed to compressed air than among age- and occupationally matched control subjects.

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A number of recent reports have described the correlation between pathologic findings and the number of patchy white matter lesions (PWML) detected by MR imaging in the aging brain [1-5]. Other reports have described the sensitivity of MR in detecting CNS abnormalities in scuba divers [6]. In 1967, Rozsahegyi [7] reported that nearly half of a group of Hungarian caisson workers had abnormal electroencephalograms even though they had never experienced neurologic decompression sickness. Similarly, it has been reported that scuba divers who had been treated for pain-only decompression sickness had abnormal findings on electroencephalograms, psychological tests, and CT scans despite normal neurologic examinations (Gorman et al., paper presented at Ninth International Symposium on Underwater Physiology, Kobe, Japan, 1986). Because a large population of compressed-air workers resides in the community around our hospital, we have accumulated a considerable amount of experience in observing the radiologic manifestations of decompression trauma as they relate to dysbaric osteonecrosis [8]. Thus, we saw an opportunity to objectively evaluate the MR findings in compressed-air workers. The experimental design was that of a case control study

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in which MR and psychometric testing were used to evaluate the extent and possible effects of compressed-air exposure.

Materials and Methods

The experimental group consisted of 19 male caisson workers, 30–67 years old (mean age, 55 years), with well-documented dysbaric exposure to compressed air. The control group, with no exposure to compressed air, consisted of 11 men, 38–65 years old (mean age, 52 years), who were employed as tunnel miners and muckers in the same labor union. Both groups were asked for extensive medical and social histories, and all subjects had psychometric testing and MR imaging of the brain. During the period in which they had worked in a compressed-air environment, all workers were given yearly neurologic examinations, all of which were normal. Neurologic examinations were not performed at the time of the study. Relative exposure indexes were calculated from work records by multiplying the average PSI pressure gauge by the number of years exposed. The experimental protocol was submitted and approved by our hospital institutional review board.

All MR scans were obtained on a 1.5-T unit (Gyrosan, Philips Medical Systems, Inc., Shelton, CT) using spin-echo (SE) multisection imaging sequences. Axial, sagittal, and coronal T1-weighted, 650/25/2 (TR/TE), images were obtained, and T2-weighted sequences, 2000/20,70, were obtained in a single acquisition using cardiac gating. All studies were done with standard 23-cm field of view head coils (5-mm slice thickness, 2.5-mm interscan gap, 256 × 256 matrix, and in-plane resolution of 0.8 to 1.0 mm). All MR scans were blindly read by two experienced radiologists who graded the PWMLs according to a 0 through 4 grading system initially proposed by Brant-Zawadzki et al. [1]. A score of 0 was given when no PWMLs were present on T2-weighted images. A score of 1 indicated punctate foci of high signal intensity in the white matter at the tip of the frontal horns of the lateral ventricles. A score of 2 denoted foci of high signal intensity elsewhere but confined to the immediate subependymal region of the ventricles. A score of 3 indicated paraventricular as well as separate discrete deep white matter foci of high T2 signal. A score of 4 was reserved for large coalescent deep white matter foci of increased signal. The white matter lesions were further characterized as to number (up to 20 and greater than 20), and location (basal ganglia, centrum semiovale, optic radiations, etc.). Ventricular size was measured according to the method of Nagata et al. [9].

At the time of MR imaging, all patients underwent psychological testing by a board-eligible psychiatrist or clinical psychologist. The psychometric testing consisted of the Shipley Institute of Living scale, the peg board test, the digit symbol test, Trailmaking, versions A and B, and letter cancellation [10, 11]. The peg board test consisted of a tray of small metal pegs, all of which had to be fitted into holes in a wooden block using one hand, either the left or the right. Subjects were graded according to the time it took to fill all the holes. The digit symbol test is a timed test to determine how many symbols, coded to different numbers from 1 through 9, can be matched with the correct numbers in rows of boxes. Trailmaking A requires use of a pencil to place randomly distributed numbered circles in consecutive order. This is a timed test and errors were recorded. Trailmaking B is a variant of Trailmaking A in which letters are alternated with numbers; for example, 1-A-2-B-3-C, etc. The letter cancellation test, which consists of several rows of random letters, requires the subject to draw a line through the letter C each time it occurs. The test is timed and errors recorded. The Shipley scale is a rough measure of IQ as reflected in word recognition.

A statistical analysis of the two groups was performed using the Wilcoxon two-sample test [12], the Kruskal-Wallis test, and linear trend analysis [13].

Results

In the experimental group, the educational level averaged 12 years (range, 5–15 years) while the control group average was 11 years (range, 9–12 years). The medical histories of both groups were negative for hypertension, multiple sclerosis, diabetes, or known cardiovascular disease. Alcohol abuse was significant in both groups.

Eight of the 11 subjects in the control group and 18 of the 19 subjects in the experimental group had psychometric testing. It was necessary to eliminate all the psychometric test results in one experimental subject because of underlying Parkinson disease and illiteracy. Similarly, the Shipley IQ test results were eliminated in three additional experimental subjects and in one control subject because of illiteracy.

The mean peg board scores were 88 sec for the right hand and 94 sec for the left hand in the experimental group, and 82 sec for the right hand and 88 sec for the left hand in the control group. Digital symbol mean time was 43 sec with no errors in the experimental group and 40 sec with no errors in the control group. Trailmaking A mean time was 40 sec in the experimental group with 0.2 errors, and 34 sec with no errors in the control group. Trailmaking B mean time was 113 sec with two errors in the experimental group and 104 sec with one error in the control group. Letter cancellation mean time was 145 sec with five errors for the experimental group and 148 sec with three errors for the control group. The average Shipley IQ was 31 in the experimental group and 30 in the control group. The apparent differences in psychometric testing results between groups was not statistically significant.

In the control group, a total of 22 PWMLs were found as opposed to more than 152 in the experimental group. Both groups demonstrated considerable skewing of the numerical distribution of PWMLs. Nine (47%) of the 19 experimental subjects had no lesions while seven (37%) had greater than 20 PWMLs each. Three (16%) of the 19 had one, three, and eight PWMLs, respectively.

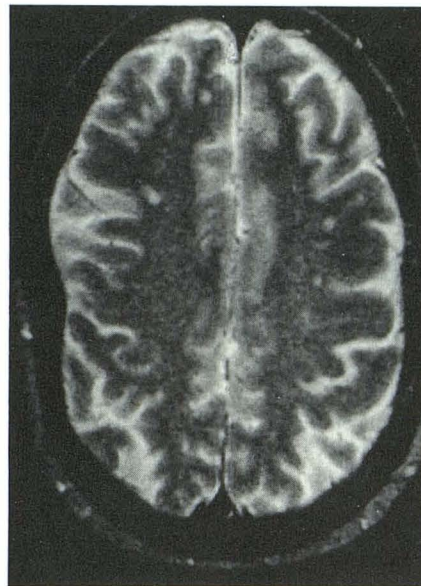
In the control group, nine individuals (82%) failed to demonstrate any MR abnormalities while two individuals (18%) demonstrated 10 and 11 PWMLs, respectively.

When the PWML grading system was applied, a similar bimodal distribution occurred in both groups. Among the 19 experimental subjects, nine (47%) were shown to have stage 0 lesions; another nine (47%) were judged to have stage 3 lesions (Fig. 1); and a single subject (5%) (Fig. 2) was shown to have stage 4 lesions. In the 11 control individuals, nine (82%) were classified as having stage 0 lesions while two (18%) were shown to have stage 3 lesions. All 12 individuals who were given scores of 3 or 4 (10 in the experimental group and two in the control group) had PWMLs in the centrum semiovale either unilaterally or bilaterally. Of these, six (five experimental subjects and one control subject) had lesions within the optic radiations. Two experimental subjects had lesions in the internal capsule and one manifested a single basal ganglion lesion. Another demonstrated a wedge-shaped area of decreased T1 and increased T2 signal at the right frontal pole consistent with a prior cerebrovascular accident.

Ventricular size was judged to be within the normal range according to the nomograms developed by Nagata et al. [9].



A



B

Fig. 1.—A and B, Patient with stage 3 patchy white matter lesions. Axial T2-weighted images (2100/70) at the midventricular level (A) and 2 cm cephalad (B) show variable-sized foci of increased T2 signal (arrow in A) within deep white matter tracts and in centrum semiovale.

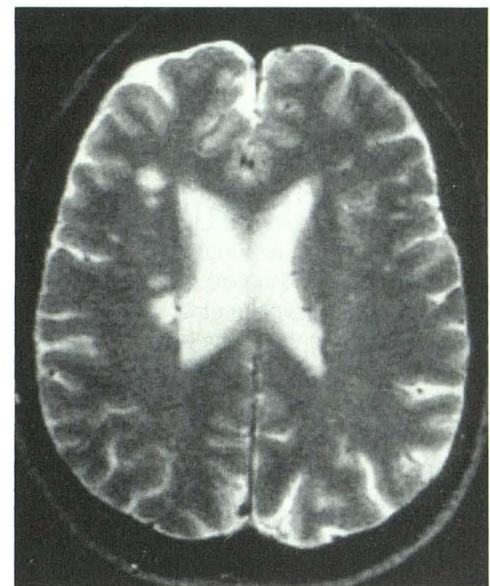


Fig. 2.—Axial T2-weighted MR image (2100/70) of the single patient with grade 4 patchy white matter lesions clearly shows the coalescent focus of high signal in the right anterior centrum semiovale.

Statistical analysis of our data revealed that the exposed (experimental) group had a statistically significant increase in the number of PWMLs as compared with the control group (Kruskal-Wallis test, $p = .05$). An odds ratio was also calculated, which showed a fivefold increase in the likelihood of an exposed individual having a grade 3 or 4 lesion compared with an age-matched control subject. The 90% confidence interval for the odds ratio ranged from an increased risk of 1.13 times to 22.2 times. When linear trend analysis was applied, a high statistical correlation ($p = .02$) was found between the number of PWMLs and their severity (grades 3 or 4), indicating that individuals who had both large numbers of PWMLs and high-grade lesions were most probably in the experimental group. In neither group, no correlation could be shown between the MR findings and exposure index, degree of osteonecrosis, psychometric testing, or ventricular size.

Discussion

Because of the availability of two hyperbaric chambers at our institution, and because of the relatively extensive use of large sewage tunnel and drainage systems in the nearby area, we are a tertiary referral center for decompression-related sickness. In the present study, nine of the experimental subjects were in the original group of patients with occupational avascular necrosis related to compressed-air work described by Nellen and Kindwall in 1972 [8]. Since that time, there has been an increasing awareness of the grave consequences of occupationally related dysbaric osteonecrosis [6–8, Gorman et al.]; however, little is known about the potential neurologic sequelae of decompression sickness in the setting of compressed-air tunnel work.

Because of the exquisite sensitivity of MR to subtle changes in brain anatomy, we chose to use this imaging technique for an initial investigation of the possibility of neurologic sequelae in dysbaric exposure to compressed air.

In the spinal cord, the pathophysiology of decompression-related diseases is fairly well known [5, Gorman et al.]. It is postulated that microbubbles form in the venous plexus around the cord, and capillary blockade results in decreased tissue perfusion and venous infarction. More recent work [10] implicates autochthonous bubble formation within the substance of the cord during decompression. MR has been useful in evaluating air emboli in the spinal cord of scuba divers [5].

Initially, we believed that the severity of exposure would be proportional to the number of deep white matter lesions with resultant diffuse neurologic abnormality. We also postulated that a threshold probably existed for the neurologic sequelae as it did with bone abnormalities. Little or no framework existed for the grading of white matter lesions on MR except for the description by Brant-Zawadzki et al. [1] of white matter changes in cases of non-Alzheimer dementia. Some of these MR changes have since been shown to be normal sequelae of the aging brain [2–4]. The diffuse abnormal EEG changes and nonspecific psychometric testing abnormality noted by Rozsahegyi [7] did not point to any definite focal areas of abnormality to focus the attention of MR imaging.

The striking bimodal distribution of our results tends to confirm the hypothesis that the grade 0, 1, and 2 stages of Brant-Zawadzki et al. are probably normal sequelae of brain aging and that the grade 3 and 4 lesions should be considered more indicative of deep white matter abnormalities, as might be expected with dysbaric exposures. Our data suggest that the standard pathophysiologic mechanisms postulated for

dysbaric osteonecrosis in caisson workers do not explain cerebral injury. It is with great concern that we note that a much higher risk of cerebral injury exists in the caisson worker population than among age- and occupationally matched unexposed control subjects.

In our study, psychometric testing was administered in part to exclude organic brain pathology in the experimental group, which might have skewed the interpretation of the MR findings. Interestingly, although there was no statistical significance, the exposed individuals scored lower than the control subjects in all psychometric examinations. There was no significant difference in the ages or psychosocial or medical histories of the two groups. The psychometric testing did not point to any focal abnormalities pathognomonic for any disease process specifically.

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