The Magnatherm Model 1000 is a pulse-modulated radiofrequency (RF) diathermy unit used to accelerate tissue healing by local heating. Treatment is applied through two rectangular "inductive treatment heads," which are placed on the tissue of interest. It operates in a frequency range of 27.120 MHz, with the RF carrier pulse-modulated from 700 to 7,000 Hz. At the settings used in our patient, the RF signal was pulsed at 4,000 Hz at 36% of its total power capacity.

The mechanism by which diathermy interacted with the implanted leads or electrodes is believed to be by induction of an RF current and heating of the electrodes. Whether this was induction in the electrode wires en passage in the soft tissues of the neck or directly upon the DBS electrodes in the brainstem is uncertain. The proximity of the clivus to the DBS leads and the fact that the patient's maxilla was being treated with the RF unit suggest that conduction of the RF field through the bony structure of the skull base was the most likely path. The edema surrounding the DBS electrodes in the brainstem suggests that heating of the electrodes by the RF current produced this devastating neurologic result.

Our case illustrates that exposure of the IPG, leads, or electrodes to high-energy electric or magnetic fields poses a danger for tissue damage in the region of the electrodes. Further definition of other potentially dangerous interactions is needed. For now, it is important for the physicians to be aware of this adverse event and to educate their patients to avoid exposure to RF diathermy in the vicinity of the IPGs, leads, and electrodes.

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Progressive brain atrophy on serial MRI in dementia with Lewy bodies, AD, and vascular dementia

Article abstract—The authors determined rates of brain atrophy, as assessed by the boundary shift integral on serial MRI, in patients with dementia with Lewy Bodies (DLB, n = 10), AD (n = 9), vascular dementia (VaD, n = 9), and age-matched controls (n = 20). Mean % ± SD atrophy rates per year were as follows: DLB, 1.4 ± 1.1; AD, 2.0 ± 0.9; VaD, 1.9 ± 1.1; and controls, 0.5 ± 0.7. Dementia subjects had higher rates than controls ($p < 0.001$), but there were no significant differences between the three dementia groups. The authors found accelerating atrophy with increasing severity of cognitive impairment, further emphasizing the need for early diagnosis and intervention in dementia.

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Dementia with Lewy bodies (DLB) is now widely recognized as the second most common cause of degenerative dementia, accounting for up to 20% of all cases.[1] Clinical diagnostic criteria have been proposed and now prospectively validated,[2] but although specificity of the clinical diagnostic criteria is generally high, 17 to 78% of cases may be missed.[3] The potential contribution of neuroimaging in the differential diagnosis of DLB from other dementias remains uncertain, though relative preservation of the hippocampus and temporal lobe is found in DLB compared with AD.[4]

Using a subvoxel registration and subtraction technique, measurement of global cerebral atrophy rates from registered serial MRI scans has been shown to be highly sensitive and reproducible and can contribute both to differential diagnosis and monitoring progression in AD.[5,6] However, there have been no prospective studies of atrophy rates in DLB or vascular dementia (VaD). The aim of this study was to investigate whole-brain atrophy rates by using serial MRI in patients with DLB compared with those with AD, VaD, and age-comparable control subjects.

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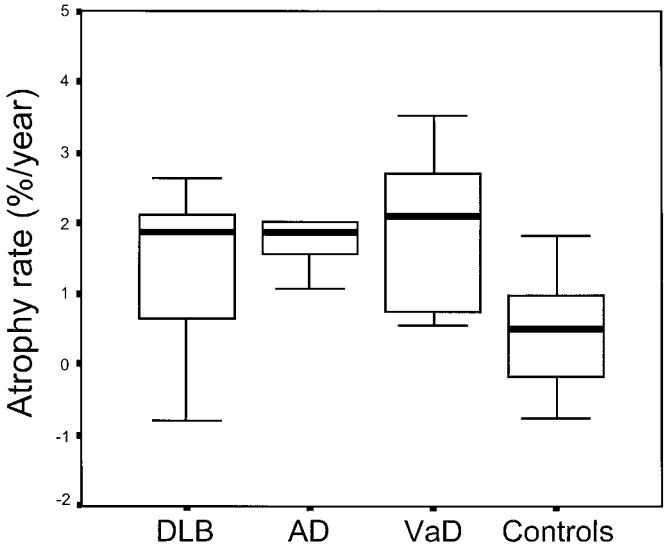

Figure 1. Box-and-whiskers plot of serial volume loss by diagnosis. DLB = dementia with Lewy bodies; VaD = vascular dementia.

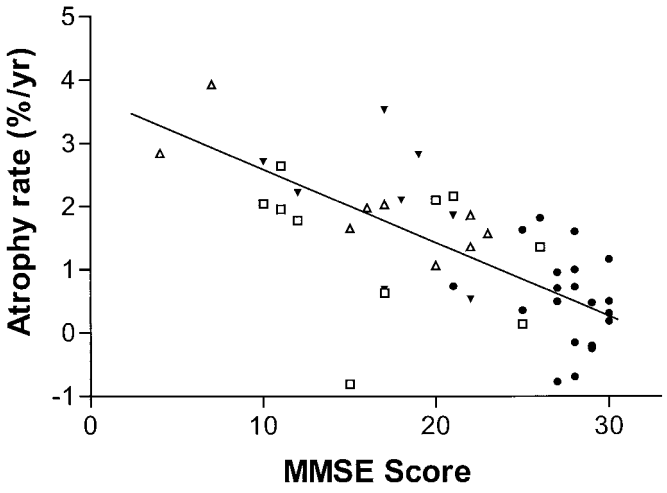

Figure 2. Plot of atrophy rates against cognitive test score for all subjects. Open squares = dementia with Lewy bodies (DLB); open triangles = AD; inverted filled triangles = vascular dementia (VaD); filled circles = controls. Correlation for all subjects r = −0.68, p < 0.001, *for dementia only* r = −0.49, p = 0.008. *MMSE = Mini-Mental State Examination.*

Methods. All subjects who had taken part in a cross-sectional MRI study[4] and who were willing and able to undergo a repeat scan 12 months later were included. In all, 28 subjects over the age of 60 years who fulfilled *Diagnostic and Statistical Manual, 4th rev.* criteria for dementia were recruited from a community-dwelling population of patients with an informant in regular contact. Twenty age-matched controls were recruited from among spouses and friends of dementia subjects. Cognitive function was measured using the Mini-Mental State Examination (MMSE) and stage of dementia with the clinical dementia rating scale (CDR).

Diagnoses of AD, VaD, and DLB were made in accordance with National Institute of Neurological and Communicative Disorders and Stroke/AD and Related Disorders Association (NINCDS/ADRDA), National Institute of Neurological Disorders and Stroke (NINDS)/AIREN, and DLB Consensus criteria, respectively, by consensus agreement between three experienced raters (J.O.B., C.B., I.M.K.) blind to all MRI scan findings (CT findings were used to assess the presence or absence of infarcts and other vascular changes necessary for the application of each of the diagnostic criteria). We included 10 subjects with consensus criteria for probable DLB (male:female ratio [M:F], 8:2; age, 74.4 ± 6.1 years; MMSE, 16.8 ± 6.0), nine subjects with NINCDS/ADRDA AD (probable, n = 8; possible, n = 1; M:F, 3:6; age, 74.3 ± 5.3 years; MMSE 16.2 ± 6.7), and nine subjects with NINDS/AIREN VaD (probable, n = 6; possible, n = 3; M:F, 8:1; age, 76.4 ± 6.7 years; MMSE, 17.4 ± 4.0). Twenty control subjects (M:F, 10:10; age, 75.8 ± 4.7 years; MMSE, 28.0 ± 1.6) were also recruited.

Baseline and follow-up scans were performed on the same 1.0 T Siemens Magnetom Impact Expert MRI Scanner (Siemens Medical, Erlangen, Germany), using an identical set-up and imaging protocol and the same experienced radiographer. T1-weighted 3D MPRAGE (magnetization-prepared rapid acquisition gradient echo) turbo flash sagittal sequence was used to acquire whole-brain images (repetition time = 11.4 ms, echo time = 4.4 ms, inversion time = 400 ms, delay time = 50 ms, matrix 256 × 256; slice thickness = 1 mm). Standard head positioning was used. Images were transferred to a Sun Ultra 30 workstation (Sun Microsystems Inc., Mountain View, CA) and analyzed by using MIDAS software (Dementia Research Group, London, UK).[6] All analysis was performed by the same operator, who was not aware of the patient's name or diagnosis. After segmentation and registration, quantification of atrophy rates was performed by measuring the change in the brain/CSF boundary shift between scans as previously described.[6,7] Full methodologic details are provided in the on-line version of this article (go to www.neurology.org).

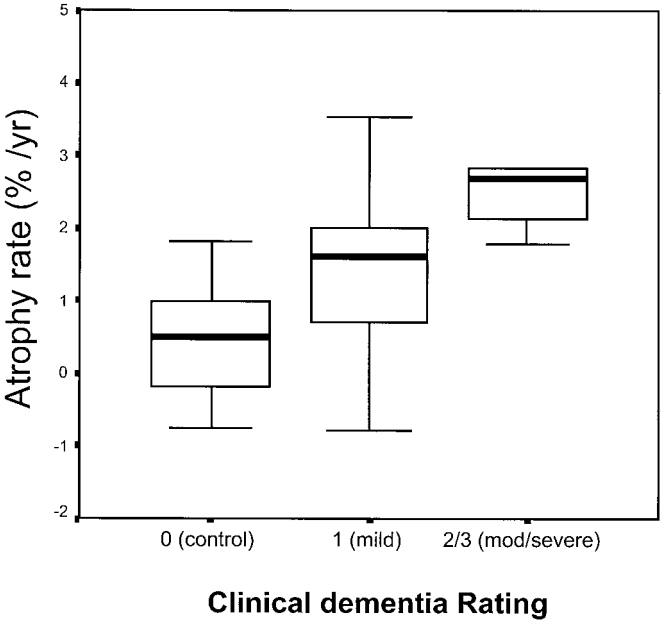

Figure 3. Box-and-whiskers plot of serial volume loss by severity of dementia.

Results. Groups were well matched for age, interval between scans, and, for dementia subjects, cognitive test score. Mean ± SD volume changes per year were as follows: controls, 5.2 ± 8 mL; DLB, 15.8 ± 12.3 mL; AD, 19.9 ± 7.9 mL; and VaD, 19.4 ± 10.9 mL. Atrophy rates were different between groups ($F = 7.63$, $p < 0.001$), and post hoc Bonferroni tests showed differences between controls and AD ($p = 0.02$), VaD ($p = 0.004$), and DLB ($p = 0.037$). Atrophy rates in percentage ± SD per year (i.e., normalized for initial brain volume) were as follows: controls, 0.5 to 0.7%; DLB, 1.4 ± 1.1%; AD, 2.0 ± 0.9%; and VaD, 1.9 ± 1.1% (figure 1). Again, results were different between groups ($F = 8.7$, $p < 0.001$), with controls differing from those with AD ($p = 0.001$), VaD ($p = 0.002$), and DLB ($p = 0.071$). There were no differences in atrophy rates between any of the three dementia groups.

Stepwise linear regression was used to examine the relationship between atrophy rate, diagnosis of dementia, and other factors (age, sex, MMSE score, and APOE status). The only variable entered into the analysis was MMSE score (beta, −0.68; $t = -6.23$, $p < 0.001$), which alone accounted for 46% of the variance in atrophy rates ($R^2 = 0.462$) (figure 2). Consistent with this finding, atrophy rates were associated with severity of dementia as assessed by CDR rating ($F = 20.65$, $p < 0.001$ for normalized atrophy rates; $F = 19.49$, $p < 0.001$ for raw volume changes), with rates (mean ± SD) as follows: CDR 0 (controls, n = 20), 0.5 ± 0.7%; CDR 1 (n = 20), 1.4 ± 0.9%; and CDR 2/3 (n = 8), 2.6 ± 0.7% (figure 3).

Discussion. This study is the first to examine brain atrophy rate as determined from serial MRI scans in patients with DLB and VaD in comparison with those with AD and normal controls. Our main finding was that atrophy rate was increased in all three dementia groups compared with control subjects, with no significant differences between AD, DLB, and VaD. Although there was some suggestion that atrophy rate in DLB (1.4%) may be slightly less than in AD (2%) and VaD (1.9%), these differences were not significant. Rates of atrophy found in this study for subjects with late-onset AD (mean age, 74.3 years; atrophy rate, 2.0% per year) were similar to those reported by Fox et al.[8] (2.4% per year for AD with early-onset disease; mean age, 65 years). Our control rates (0.5% per year) also paralleled the 0.4% per year reported for controls a decade younger using the same technique. The finding of similar rates in DLB and VaD could potentially be attributable to clinical misdiagnosis of AD cases (though we have established good accuracy for clinical diagnosis in this cohort[2]), overlapping neuropathology, or the fact that different pathologic changes (vascular or neurodegenerative) may produce the same final result of global brain volume change. In the future, it will be important to perform quantification of serial regional volume changes and to correlate atrophy rates with neuropathologic changes in different diagnostic groups. However, our results suggest that serial volume loss on MRI may be useful for monitoring disease progression in late-onset AD, DLB, and VaD, as has been advocated for early-onset AD.

By far the strongest predictor of rate of atrophy, irrespective of diagnosis, was baseline severity of dementia as measured either by MMSE score or CDR stage (see figures 2 and 3). Rates increased threefold, from 0.5% per year in controls (CDR 0), to 1.4% per year in those with mild dementia (CDR 1), and fivefold, to 2.6% per year, in those with moderate to severe dementia (CDR 2/3). Two important observations can be made. First, even in mild dementia (CDR 1), atrophy rates are three times higher than those of normal controls. This implies that even in those with early dementia, serial measurement of atrophy by MRI may be useful in monitoring course and also that volume loss can be detected early in the illness. Secondly, the rapid increase in atrophy rates with increasing severity of dementia provide important new data supporting the view that dementia follows an accelerating course, as has been suggested by neuropsychological studies.[9,10] Our results further recommend the need for early diagnosis and, when available, early administration of disease-stabilizing or disease-modifying therapies in all three subtypes of dementia.

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