

DMT may stimulate visual functions and planning more than memory, but the findings are inconclusive. A benefit was previously observed in the information content of speech output,<sup>5</sup> and this was partly replicated. The behavioral symptoms remained relatively stable, but self-care ability and IADLs seemed to improve slightly.

DMT therefore seems to offer one option in treating dementia, having effects on cognition and self-care abilities.

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## REFERENCES

1. Clare L, Woods RT, Moniz Cook ED et al. Cognitive rehabilitation interventions to improve memory functioning in early stage Alzheimer's disease and vascular dementia. *Cochrane Database Syst Rev* 2003;(4): CD003260.
2. Spector A, Orrell M, Davies S et al. Reality Orientation for Dementia. The Cochrane Library Ed. Oxford: Update Software, 2000.
3. Doody RS, Stevens JC, Beck C et al. Practice parameter: Management of dementia (an evidence-based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology* 2001;56:1154–1166.
4. Vink AC, Birks JS, Bruinsma MS et al. Music therapy for people with dementia. *Cochrane Database Syst Rev* 2003;(3): CD003477.
5. Hokkanen L, Rantala L, Remes AM et al. Dance/movement therapeutic methods in management of dementia. *J Am Geriatr Soc* 2003;5:576–577.
6. Folstein MF, Folstein SE, McHugh PR. "Mini-Mental State." A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 1975;12:189–198.
7. Welsh K, Butters N, Hughes J et al. Detection and staging of dementia in Alzheimer's disease. Use of the neuropsychological measures developed for the Consortium to Establish a Registry for Alzheimer's disease. *Arch Neurol* 1992;49:448–452.
8. Pulliainen V, Hokkanen L, Salo J et al. CERAD Kognitiivinen tehtäväsarja. Käsikirja ja välineistö. [CERAD Neuropsychological Battery. Manual and Test material]. Kuopio: Suomen Alzheimer-tutkimusseura, 1999.
9. Goodglass H, Kaplan E. The Assessment of Aphasia and Related Disorders. Baltimore: Waverly Inc., 1983.
10. Spiegel R, Brunner C, Ermini-Funfschilling D et al. A new behavioral assessment scale for geriatric out- and in-patients: The NOSGER (Nurses' Observation Scale for Geriatric Patients). *J Am Geriatr Soc* 1991;39:339–347.

## A RETROSPECTIVE CHART REVIEW OF THE TOLERABILITY AND EFFICACY OF INTRAVENOUS IMMUNOGLOBULIN IN THE TREATMENT OF ALZHEIMER'S DISEASE

*To the Editor:* Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by insoluble beta-amyloid peptide (A $\beta$ <sub>42</sub>) plaque deposition in the brain.<sup>1,2</sup> Reducing insoluble plaque burden, by decreasing the production of A $\beta$ <sub>42</sub> or by increasing its clearance from the brain, is a goal of several treatment strategies currently being researched.<sup>3</sup>

Some small studies have investigated the efficacy of passive immunization with intravenous immunoglobulin (IVIg) in treating AD.<sup>4–6</sup> Patients diagnosed with AD have low levels of polyclonal anti-A $\beta$  antibodies with possible reduction in A $\beta$  clearance resulting in more plaques and cerebrovascular amyloid deposits.<sup>7</sup> Studies of IVIg treatment have shown an increase in anti-A $\beta$  antibodies in patients' cerebrospinal fluid (CSF), possibly leading to greater clearance of A $\beta$ .<sup>4–6</sup>

Based on these data, a sample of patients with AD was treated with IVIg.<sup>4,5</sup> The tolerability and efficacy of IVIg in the treatment of cognitive loss in this group of 10 patients was retrospectively investigated. It was hypothesized that IVIg would be well tolerated and effective in treating cognitive symptoms in this group.

A total of 18 patients had received IVIg, at 0.4g/kg every 2 weeks, for the treatment of AD. Five patients were cognitively unable to complete a neurocognitive battery

**Table 1. Tests Showing Significant Cognitive Change over Time**

Subtest*	Pretreatment	Posttreatment	Change (post-pre)	P-Value
	Mean ± Standard Deviation			
WAIS verbal scale intelligence	101.50 ± 2.30	99.20 ± 24.18	-2.30 ± 3.80	.09
WAIS full-scale intelligence	97.20 ± 23.11	94.20 ± 23.53	-3.00 ± 5.14	.098
WAIS information	9.10 ± 4.36	8.20 ± 4.16	-0.90 ± 1.52	.095
WMS-III verbal paired associates	4.30 ± 0.82	5.10 ± 1.79	0.80 ± 1.62	.15 <sup>†</sup>
WMS-III logical memory II recall	4.90 ± 2.89	6.00 ± 3.40	1.10 ± 1.10	.09
WMS-III auditory delayed memory	69.89 ± 8.48	74.44 ± 8.92	4.56 ± 7.88	.1
BNT total	46.70 ± 15.86	44.80 ± 17.51	-1.90 ± 3.38	.1

\*Wechsler Adult Intelligence Scale (WAIS) and Wechsler Memory Scale (WMS)-III Primary Index normal distribution  $100 \pm 15$ , scaled score normal distribution  $10 \pm 3$  (range 1–19) (Wechsler D. Wechsler Adult Intelligence Scale (3rd Ed). San Antonio: The Psychological Corporation, 1997); Boston Naming Test (BNT) (range 1–60) (Kaplan E, Goodglass H, Weintraub S. The Boston Naming Test. Philadelphia: Lea and Febiger, 1983).

<sup>†</sup>Approaching trend at the  $P < .1$  level.

(Wechsler Adult Intelligence Scale (WAIS) III; Wechsler Memory Scale (WMS) III; Bushke Selective Reminding Test (BSRT); 60-item Boston Naming Test (BNT)) before or after IVIg treatment and were therefore not included in the analysis. Three of the patients completed their first neurocognitive battery more than 6 months before they began receiving IVIg treatment and were also excluded. The remaining 10 patients (5 men, 5 women; mean age  $\pm$  standard deviation  $74 \pm 7.5$ ) were analyzed in the retrospective analysis. Seven of these patients had been diagnosed with probable AD and three with possible AD according to the National Institute of Neurological and Communicative Disorders and Stroke/Alzheimer's Disease and Related Disorders Association criteria.<sup>8</sup> Eight of the patients completed 6 months of treatment; two completed 3.5 months of treatment. Two patients developed a pruritic, maculopapular, generalized rash, resolving with appropriate treatment, but both continued with IVIg.

The institutional review board of Lenox Hill Hospital in New York, New York, approved the retrospective chart review. Outcome variables on the neurocognitive battery before and after IVIg treatment were evaluated using Student paired *t*-tests. Nonparametric tests were used to test for differences between sexes.

Patients showed stability on neurocognitive scores overall, with trends toward decline on their WAIS verbal scale and full-scale intelligence scores ( $P < .1$ ), as well as on the WAIS information ( $P < .1$ ) subtest and the BNT ( $P = .1$ ). Patients showed trends toward improvement on the WMS logical memory II recall ( $P < .1$ ), WMS verbal paired associates ( $P = .15$ ), and the WMS auditory delayed memory test ( $P = .1$ ). These results are summarized in Table 1. Because of the small number of patients included in the analysis ( $n = 10$ ) and the resulting low power of the study, a significance level of  $P < .1$  was used to indicate trends toward improvement or decline in this group.

Stratifying the data according to sex showed that the mean proportion was higher and variability lower for men ( $1.05 \pm 0.07$ ) than women ( $0.93 \pm 0.10$ ). Nonparametric tests showed differences approaching significance between the sexes. Significance values for each of these tests were as follows: analysis of variance,  $P = .05$ ; Wilcoxon rank sum,  $P = .07$ ; Kruskal-Wallis,  $P = .08$ ; and Median Score,  $P = .07$ . This difference may be attributed to differential

plaque clearance between the sexes. Various studies have shown an existing disparity in A $\beta$  plaque load between men and women, with women having an overall higher plaque burden.<sup>9,10</sup>

Overall, results from this retrospective chart review appear promising, notwithstanding a few limitations. It was found that IVIg was well tolerated and effective in this sample, with patients showing stability on neurocognitive test scores and trends toward improvement in some areas. However, this study was relatively small. A larger study of IVIg treatment of cognitive loss in this cohort would be ideal, as well as future research into potential differences between the sexes. Researchers and practitioners are currently awaiting the results of a Phase II Trial of IVIg for the treatment of cognitive loss in AD conducted by Elan Pharmaceuticals, Inc. Results of this larger trial may have important implications for the direction of future IVIg and Alzheimer's research. Meanwhile, smaller studies such as this retrospective chart review continue to support the use of IVIg for treating cognitive loss in AD, both through neurocognitive test measures and clinical improvements.

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## REFERENCES

1. Yaari R, Corey-Bloom J. Alzheimer's disease. *Semin Neurol* 2007;27:32–41.
2. Hyman BT. New neuropathological criteria for Alzheimer disease. *Arch Neurol* 1998;55:1174–1176.
3. Kennedy GJ, Golde TE, Tariot PN et al. Amyloid-Based interventions in Alzheimer's disease. *CNS Spectr* 2007;12:1–14.
4. Dodel RC, Du Y, Depboylu C et al. Intravenous immunoglobulins containing antibodies against beta-amyloid for the treatment of Alzheimer's disease. *J Neurol Neurosurg Psychiatry* 2004;75:1472–1474.
5. Relkin NR, Younkin L, Younkin S et al. Decreased plasma beta amyloid levels in Alzheimer patients treated chronically with Intravenous immunoglobulin (IVIg) [Abstract]. *Alzheimer Dement* 2006;2:S590.
6. Weksler ME. The immunotherapy of Alzheimer's disease. *Immun Ageing* 2004;1:2.
7. Dodel R, Hampel H, Depboylu C et al. Human antibodies against amyloid peptide: A potential treatment for Alzheimer's disease. *Ann Neurol* 2002;52:253–256.
8. McKhann G, Drachmann D, Folstein M et al. Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA work group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. *Neurology* 1984;34:939–944.
9. Kraszpulski M, Soininen H, Helisalmi S et al. The load and distribution of  $\beta$ -amyloid in brain tissue of patients with Alzheimer's disease. *Acta Neurol Scand* 2001;103:88–92.
10. Schafer S, Wirths O, Multhaup G et al. Gender dependent APP processing in a transgenic mouse model of Alzheimer's disease. *J Neural Transm* 2007;114:387–394.

## COCKCROFT-GAULT FORMULA FOR DIAGNOSING MODERATE KIDNEY FAILURE

*To the Editor:* The article by Giannelli et al.<sup>1</sup> concerning specificity and sensitivity of various laboratory methods of estimating glomerular filtration rate (GFR) may be of practical importance relative to Medicare D reimbursement for the use of erythropoietin (EPO) stimulators.

One of the approved diagnoses for using EPO stimulators is when anemia is associated with moderate kidney failure, defined as GFR  $< 60$  mL/min per  $1.73$  m<sup>2</sup>. The laboratory at the Bergen Regional Medical Center conventionally employs the Modification of Diet in Renal Disease 4 equation which is reported as greater than 60 when a lesser degree of renal failure is present, insufficient to meet Medicare criteria for using EPO stimulators. However, when GFR was recalculated for four such anemic patients using the older and apparently more-sensitive Cockcroft-

Gault formula, in each case, the GFR was  $< 60$  (52.8, 55.9, 46.5, and 32.8).

We suggest that, when confronted with anemic elderly patients whose diagnosis is uncertain and whose renal function appears to be normal according to conventional methods, GFR should be recalculated using the Cockcroft-Gault formula. Clinicians may be surprised to find that moderate kidney failure exists and that EPO stimulation might be worth a try—and may even be compensable.

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## REFERENCE

1. Giannelli SV, Patel KV, Windham BG et al. Magnitude of underascertainment of impaired kidney function in older adults with normal serum creatinine. *J Am Geriatr Soc* 2007;55:816–823.

## SOUND-ALIKE SYNDROMES: FIRST REPORT OF MORVAN'S, AND NOT MARFAN'S, SYNDROME

*To the Editor:* “Can you handle a case of Marfan's syndrome?” was asked of our 720-bed subacute and long-term care facility in the Bronx, New York.

“Sure, why not?” came the immediate reply, with thoughts of Abraham Lincoln and the connective tissue genetic disease affecting the limbs, valves, and aorta. Life expectancy for this disease has improved with better treatments, and it would not be surprising that a patient with this syndrome would require nursing home placement.

“Sound-alike” medications have been a notable concern for the Joint Commission on Accreditation of Healthcare Organizations (JCAHO), pharmacies, physicians, and patients, but this example of “sound-alike” syndromes was new to us, as was this specific syndrome, which has yet to be reported in the geriatric or long-term care literature.

## CASE REPORT

Mr. M, a 67-year-old man from Bangladesh, was admitted to Kings Harbor Multicare Center from a referring hospital. He had been in his usual state of health until January 2007, when he developed personality changes including excessive crying and banging his head against the wall, as well as visual hallucinations, insomnia, apathy, lethargy, muscle twitching, inability to walk, and seizures. An extensive examination included a computed tomography scan of the head showing old ischemic changes and lacunar infarcts, a magnetic resonance imaging (MRI) scan revealing increa-