Literature Review & Commentary
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L-Arginine for dementia
Sixteen elderly patients (mean age, 79 years) with cerebrovascular disease who had been living in a nursing home for 2-4 years received 1.6 g/day of L-arginine for 3 months. Cognitive function was determined by a revised version of Hasegawa’s Dementia Scale (which is comparable to the Mini-Mental State Examination). Thirty is a perfect score and less than 20 is considered to reflect dementia. The mean score improved from 16 at baseline to 23 (p < 0.0001) at the end of the treatment period. However, 3 months after L-arginine was discontinued, the score had fallen to 17. In general, patients showed more expressive faces and quicker responses while receiving L-arginine. No side effects were seen.

Comment: Arginine is a precursor to nitric oxide, which has been reported to function as a neurotransmitter that plays a role in learning and memory. In addition, nitric oxide functions as a vasodilator and might, therefore, promote increased blood flow to the brain. Tissue concentrations of nitric oxide and arginine decline with age, suggesting that arginine deficiency may be a contributory factor to age-related mental decline. This open trial suggests that supplementation with a relatively small amount of L-arginine (a typical diet contains approximately 3 times that amount) improved cognitive function in patients with cerebrovascular disease. Controlled trials are needed to confirm this promising study.


Unexplained dialysis dementia/encephalopathy caused by thiamine deficiency
Ten patients on dialysis (9 hemodialysis) with altered mental status, the cause of which was unidentified after an initial work-up, were studied. Manifestations included confusion, chorea, acute visual loss, rapidly progressive dementia, myoclonus, convulsions, and coma. Of 7 patients in whom serum thiamine concentrations were measured, all had subnormal levels. All 10 patients received an intravenous injection of 200 mg of thiamine, followed by 100 mg/day intravenously until they could consume a normal diet. The neurological deficits resolved dramatically in 9 of the 10 patients; the other patient failed to respond because of a delay in treatment. Five of the 10 patients had been receiving oral B vitamin supplements before developing thiamine deficiency.

Comment: Patients with end-stage renal disease undergoing regular dialysis are at risk of developing encephalopathy, the cause of which is often unclear. Dialysis patients are also at risk of developing thiamine deficiency, which can mimic many of the complications of uremia, including encephalopathy. Although peritoneal dialysis patients are routinely given supplemental thiamine, thiamine supplementation of hemodialysis patients is controversial. The results of the present study suggest that, in regular dialysis patients, unexplained encephalopathy is due primarily to thiamine deficiency (e.g., Wernicke’s encephalopathy).


Dementia and Alzheimer’s disease: environmental illnesses?
A total of 2,459 community-dwelling Yoruba residents of Ibadan, Nigeria, without dementia, and 2,147 community-dwelling African-American residents of Indianapolis, Indiana, without dementia (all aged 65 years or older) were followed prospectively for a mean of 5.1 years and 4.7 years, respectively. The age-standardized annual incidence rates were significantly lower among Yoruba than among African-Americans for dementia (1.35% vs. 3.24%) and for Alzheimer’s disease (1.15% vs. 2.52%).

Comment: The results of this study indicate that the incidence rates for dementia and Alzheimer’s disease are significantly lower among individuals from a non-industrialized country than among those from an industrialized country. That finding suggests that environmental factors play a role in the development of dementia and Alzheimer’s disease. There is evidence that exposure to aluminum can increase the risk of Alzheimer’s disease; however, not all studies agree. It is likely that other environmental toxins, or chronic consumption of modern processed foods, make it difficult for old brains to remain healthy.


Choline, parenteral nutrition, and cognitive decline
Eleven patients who had received total parenteral nutrition (TPN) for more than 60% of their nutritional needs for at least 12 weeks were randomly assigned to receive their usual TPN regimen (n = 6; mean age, 34.0 years) or their usual TPN regimen plus 2 g/day of choline chloride (n = 5; mean age, 37.3 years). The following neuropsychological tests were administered at baseline and after 24 weeks: Weschler Adult Intelligence Scale-Revised (WAIS-R, intellectual functioning), Weschler Memory Scale-Revised (WMS-R, 2 subtests, verbal and visual memory), Rey-Osterrieth Complex Figure Test (visuospatial functioning and perceptual organization), Controlled Oral Word Association Test (verbal fluency), Grooved Pegboard (manual dexterity and motor speed), California Verbal Learning Test (CVLT, rote verbal learning ability), and Trail Making Parts A & B (visual scanning, psychomotor speed and set shifting). Compared with the placebo group, significant improvements were seen in the choline group in the delayed visual recall of the WMS-R (p = 0.028), and borderline improvements were seen in the List B subset of the CVLT (p = 0.06) and the Trails A test (p = 0.067).

Comment: This study demonstrates that both verbal and visual memory may be impaired in patients receiving long-