Much of the information on nutrition and dementia is not scientifically well-documented, but there is evidence that proper nutrition can be beneficial.

**BY RONALD DEVERE MD, FAAN**

The public fear of cognitive decline is at epidemic proportions. This fear is mostly associated with the worry of developing Alzheimer’s disease. But despite misconceptions, we know that the current treatment of mild-to-moderate AD can slow decline for a number of years, delay the need to enter a dementia center, and help many of the impaired behaviors that develop. A neurologist’s goal in evaluating cognitively impaired patients is to make the correct diagnosis and offer the best treatments available. Giving all facts will help reduce the caregiver and public fear of the disease. This includes addressing nutritional aspects of cognitive impairment.

Numerous papers have been published on the role of nutrition in the prevention and treatment of cognitive decline, most of which focus on AD. Unfortunately much of this information has not been scientifically well-documented, especially in the treatment of cognitive decline. My goal in this piece is not to do an exhaustive literature review, but to mention a number of pertinent studies that shed some light on the topic and especially suggest the glass is half full, not half empty.

One of the most “upbeat” articles I reviewed on this topic was authored by D.E. Bredesen from UCLA in September 2014. He presented 10 patients with varied diagnosis of subjective mild cognitive impairment (SMCI), amnestic mild cognitive impairment (AMCI) and AD who displayed subjective or objective improvement in three to six months that lasted up to 2.5 years. One case failed due to late stage AD. Six cases who stopped working or had major difficulty cognitively at work prior to treatment returned or improved their work performance. The therapeutic program is shown in Table 1, and as you can see, the list of treatments is very extensive and mostly nutritional related. The article is limited due to lack of controls, detailed information on the cognitive testing, and only showing some pertinent history in three of the 10 cases. Additionally, the authors stated that three of their cases modified the therapeutic program and provide no details about the changes. However, the value in mentioning this article is found in the extensive list of potential nutritional treatment options suggested, and used mostly together concurrently for the possibility of multiple mechanisms to play a role in cognitive impairment.

Currently, as clinicians we know the limitation of the acetylcholinesterase and glutamine inhibitors in the degenerative and vascular dementias. We welcome anything else, especially nutritional information that is helpful in these disorders. There is increasing strong evidence that partaking in aerobic exercise (120 minutes/week) and cognitive and memory therapy can stabilize/slow decline in cognitive function.

**THE EVIDENCE**

Lopes Da Silva et al. did a meta-analysis of published articles on the nutrient status of patients with AD covering 1990-2012. This is the first study of its kind reported. They compared plasma levels of micronutrients and fatty acids in cognitively intact elderly controls and AD patients. The other important feature of their study was to ensure AD patients and controls did not differ in protein/energy nutrients so that malnutrition was not present. All subjects with vitamin supplements were excluded. To be sure there was no malnutrition, they evaluated body mass index, mini nutritional assessment score and albumin levels corrected for different ages. Only 80 articles out of 3397 met their criteria for inclusion.

The researchers required a minimum of eight articles in each nutrient topic for statistical purposes. The B vitamins, antioxidants (vitamin A, C, E and selenium), choline and omega-3 fatty acids (DHE, EPA) have been found to likely play a role in the pathophysiological process of cognitive...
function and decline. For example, antioxidants reduce reactive oxygen species induced damage and stabilize neuronal membranes. The fatty acid DHA affects abnormal protein processing (amyloid Beta and Tau). DHA, choline and uridine modulate neuronal membrane formation. Plasma levels of all these nutrients including iron, zinc, copper and magnesium were reviewed in all the studies.

The study showed statistically significantly lower plasma levels of folate, vitamin B12, C and E in the AD patients (15 to 30 percent) vs. controls. Vitamin A showed a trend toward lower levels but was not statistically different. DHA and EPA plasma levels and part of the cholesterol moiety showed

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<th>GOAL</th>
<th>APPROACH</th>
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<td>Optimize diet: minimize simple CHO</td>
<td>Patients given choice of low glycemic, low grain diets</td>
<td>Minimize inflammation and insulin resistance, Minimize inflammation</td>
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<td>Enhance Ketogenesis</td>
<td>Fast 12 hours each night including 3hrs prior to bedtime</td>
<td>Reduce insulin levels; Reduce A beta</td>
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<td>Reduce Stress</td>
<td>Personalized: yoga, meditation or music.</td>
<td>Reduction of cortisol &amp; Stress axis</td>
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<td>Optimize Sleep</td>
<td>8 hrs. sleep per night, melatonin 0.5mg, To 2mg @ hs, Tryptophan 500mg 3x/wk. Exclude sleep apnea</td>
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<td>C-reactive protein &lt; 1.0, A/G &gt; 1.5</td>
<td>Gluten free diet; curcumin; DHA/EPA</td>
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<td>Vitamin D25OH-D3= 50ng/ml or &gt;</td>
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<td>Increase Nerve Growth Factor</td>
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<td>Citicoline, DHA</td>
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<td>Optimize antioxidants</td>
<td>Mixed Tocopherols, Selenium, Blueberries, NAC, ascorbate, a-lipoic acid</td>
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<td>Optimize Mitochondrial Function</td>
<td>CoQ, a-lipoic acid, NAC, Acetyl-L- carnitine, selenium, zinc, reservatrol, Ascorbate, thiamine.</td>
<td>52</td>
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<td>Increase focus</td>
<td>Pantothenic acid</td>
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<tr>
<td>Exclude Heavy Metal Toxicity</td>
<td>Evaluate Hg, Pb, Cd; chelate if indicated</td>
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significant lower levels in AD vs. controls. Vitamins B1 and B6 levels (only two papers found) were significantly lower in AD vs. controls. Plasma levels of calcium, selenium, magnesium (three papers) did not show lower levels than controls. Putting together all published studies provides unequivocally that folate, vitamins C, E, and B12 (and possibly vitamin A), DHA, EPA and Choline, are significantly low in healthy, not malnourished AD patients. Studies have shown that three percent of mild to moderate AD patients have evidence of malnutrition, increasing to 50 percent in severe AD.\textsuperscript{7,8} The exact cause of these lower plasma nutrients is unclear but clearly not due to low protein and energy metabolism. Altered feeding behavior, nutrient absorption and metabolism are possible causes, according to the authors.

The latter two processes have been implicated in the pathophysiology of AD.\textsuperscript{9} Vitamin A, C, E, and selenium are antioxidants that help to protect these lipid precursors from peroxidation and resultant neuronal membrane damage. Bourdel-Marchasson, et al.\textsuperscript{10} measured lipid oxidation in AD vs. controls. They found that low plasma levels of antioxidants were due to enhanced brain consumption due to excessive production of free radicals. This may be another reason why vitamins and fatty acids were found to be low in the meta-analysis study. Reduced DHA and EPA may also be due to impaired liver synthesis, which converts dietary alpha linolenic acid to DHA and EPA. The B vitamins (B6, B12 and folate) are also important in methylation capacity of the cell and are responsible for converting toxic homocysteine to methionine or cysteine.

There are two randomized control studies of a liquid nutrient (Souvenaid) containing DHA, EPA, UMP, choline, folate, vitamins B6, 12, C, E, and selenium. The studies showed improved memory formation in mild AD and preserved functional connectivity, important for synaptic function.\textsuperscript{11,12} To further support the use of multiple nutrients in cognitive function, Fotuhi, et al.\textsuperscript{13} studied 3,376 elderly residents in Utah with MMSE testing. They carefully monitored by checking their medicine bottles and history, their intake of vitamins C, E and NSAIDs. The residents had to be on these nutrients and NSAIDs at least four times a week for at least a month. Vitamin C (500mg) and E (400 IU) could also be taken in multivitamins tablets. Controlling for age, sex, education, history of stroke/diabetes and the presence of APOE4, users showed a slower rate of cognitive decline than nonusers over eight years. NSAIDs are not micronutrients and could cloud the study.

Another study\textsuperscript{14} that many may remember was a randomized, double blind placebo controlled trial in Alzheimer’s from 23 sites. Vitamin E at 2000 IU/day increased median survival, needed less supervision and had reduction in bad behaviors. Dysken et al.\textsuperscript{15} used a randomized clinical trial of older veterans with AD and MMSE scores of 12-26 (mild to moderate) and receiving acetylcholinesterase inhibitors. They were assigned to four groups; vitamin E plus memantine placebo; memantine and vitamin E placebo; vitamin E plus memantine; vitamin E and memantine placebo. Vitamin E was given 1000 IU bid. Some of the problems with this study included moderate medication adherence and greater than optimal loss-to-follow-up. The primary outcome was activities of daily living scales and secondary outcomes were MMSE scores and ADAS-cog.

The best results in these scales were in the vitamin E only group, who had a 19 percent lower reduction in the ADL scales. The other groups were the same as placebo. The MMSE and ADAS-cog scores were no different in all groups. There was no surprise the memantine group didn’t change because memantine has not been shown to be effective in mild-to-moderate AD. Why vitamin E and memantine together showed no changes from placebo is not clear. No adverse effects in this trial were noted. Other vitamin E trials in MCI and people with normal cognition were not effective.\textsuperscript{16} In Miller et al.’s\textsuperscript{17} meta-analysis of 19 randomized studies, vitamin E in doses greater than 400 IU was associated with higher all cause mortality. This vitamin E dose is astronomical; the institute of medicine recommends doses no higher than 22.4 IU/day. Studies using lower doses are underway.

Blasko et al.\textsuperscript{18} studied effect of folate and B12 in 81 cases of MCI. Serum levels of homocysteine, B12 and folate were measured and detailed history of supplement use was taken. The group was followed for five years. Individuals who reported use of folate and B12 corroborated by blood levels and caregiver information were less likely to develop dementia. Higher levels of folate correlated with lower conversion rate of MCI to dementia compared to nonusers.

**PHOSPHATIDYLSTERINE PLUS OMEGA-3FATTY ACIDS (VAYACOG)**

Phosphatidylserine (PS) is a naturally occurring phospholipid present in the inner leaflet of mammalian plasma membranes. In humans it is most concentrated in the brain where it makes up 15 percent of the total phospholipid pool. It has shown to play a role in neuron membranes such as signal transduction, secretory vesicle release and cell to cell communication.\textsuperscript{19} Crook et al. in 1991\textsuperscript{20} used PS extracted from bovine cortex, which contains high levels of omega-3 long chain polyunsaturated fatty acids, attached to its backbone. This combination was shown to improve learning and memory in subjective MCI and to improve cognitive performance in AD patients. Because of safety concerns in using this product regularly and risk of developing bovine spongiform encephalopathy prions, an alternative product such as soy-derived PS has been developed. Vakhapova et al.\textsuperscript{21} studied this alternative product in non-demented elderly (ages 50-90) with memory complaints. They evaluated 157 subjects randomized to receive PS-DHA/EPA (3x 100mg tabs/d)
or placebo for 15 weeks followed by an additional open label extension of 15 weeks with a smaller dose (100mg/day). All conditions that could produce cognitive impairment including use of medications were not allowed in the study. At the end of the study they verified that the product was safe, well-tolerated and did not produce any major side effects except some minor GI discomfort, which can be reduced by taking the medication with food.

The clinical benefit of PS-DHA/EPA was reported by Vakhapova et al., for 15 weeks but not open label afterwards. Cognitive measures were evaluated, which included Clinical Global Impression of change and various cognitive tests including the Rey auditory verbal learning test, etc. One hundred thirty-one out of 157 completed the study and dropouts were equal in the active and placebo groups. They also studied a subgroup that might be differently responsive to treatment. This subgroup had a MMSE score >26, delayed recall trial above the mean score, academic education >12 years. This group had 78 participants (38 placebo, 40 PS-DHA/EPA). The key outcome of this study showed that this treatment improved verbal immediate memory, subjectively and objectively. The subset population with higher cognitive status prior to treatment in addition showed improvement in long-term memory and learning ability. The authors’ explanation for this is that the subset group likely had a better cognitive performance in their earlier years and actually suffer from a deep age-related cognitive decline and are therefore more likely to respond to the treatment.

There were a number of limitations to the above study. No primary endpoint of the participants was predetermined. The study population was not homogeneous, and the inclusion criteria did not correspond to a single clinical entity. They believed, however, that the participants qualified to be included under subjective MCI or amnestic MCI. The exact mechanism of PS has not been established.

More research with this product in a larger but well-defined cognitive impaired population including mild dementia should be done, since it is safe and suggests a benefit. Its only current limitation in its use is it is considered a medical food (prescription only) and is not covered by health insurance.

**VITAMIN D**

In a recent edition of *Practical Neurology*, I reviewed the cognitive consequences of vitamin D deficiency. I stated that there was evidence that vitamin D was important in the health of the cerebral and systemic vasculature, and low levels increased cerebral infarcts and secondary cognitive decline. Since my review, there has been a number of further published articles that show a strong correlation between cognitive decline and low vitamin D levels. The big question that has not been definitively answered is whether treating low vitamin D reverses or slows cognitive decline. This has been suggested. More studies are certainly needed. I suggested that measuring vitamin D levels in the cognitive impaired elderly and treating low levels <30ng/ml with vitamin D3 2000 IU/day (one capsule) was cost effective and could possibly reduce strokes and delay/improve cognitive decline. Definitive studies could take a long time, and there is no risk and nothing to lose with vitamin D supplements.

**CAROTENOIDs**

Carotenoids are fat-soluble organic plant pigments and antioxidants found in fruits and vegetable in western diets. They include: alpha carotene, beta carotene, lycopene, zeaxanthin, and beta cryptoxanthin. Most studies have been done in animal models of Alzheimer’s, showing that in combination with vitamins they may reduce levels of oxidative stress. Retinoic acid, a metabolite of vitamin A that reduces a-beta proteins and tau hyperphosphorylation in the mouse hippocampus, improved spatial learning vs. controls. Large controlled studies in humans with cognitive decline are needed.

**POLYPHENOLS**

Polyphenols, abundant in our diet, have been shown to provide anti-inflammatory, anti-tumerogenic, antimicrobial and antioxidant effects. Curcumin is a powerful polyphenol commonly found in Indian food, shown to neutralize free radicles and protect cortical neurons against cell death induced by a-beta peptides. Polyphenols in red wine (0.4ml) significantly reduced cortical degeneration and accumulation of a-beta neuropathology in a transgenic AD mouse model. This equates to 5oz. of red wine in humans. They found that the type of grape works differently in the mouse model of AD. Cabernet Sauvignon promotes non-amyloidogenic alpha secretase activity, while muscadine wine interferes with accumulation of a-beta peptide into high molecular weight oligometric a-beta species in the brain. Both of these effects are positive in reducing neurodegeneration in the AD mouse model.

The authors of this paper discussed the problems with the use of polyphenols in possible treatment for cognitive concerns: 1) Brain levels of polyphenols in animal brain models are very low, 2) most foods are not eaten in isolation, making it difficult to isolate the effects specifically related to polyphenols, 3) future research needs to determine how diets rich in polyphenols work in combination with lifestyle factors to provide neuro-protective properties.

**CALORIC RESTRICTION**

Studies have suggested that excessive caloric intake and sedentary lifestyle are associated with a higher risk of cognitive impairment in later life and developing AD. High calorie diets have shown to reduce Brain Derived Neurotrophic Factor (BDNF), which impairs hippocampal activity. Diets that
include caloric restriction result in less oxidative stress.32 Witte et al.,33 studied 49 people, divided into three groups: 1) Caloric restriction (min. 1,200 calorie diet or 30 percent calories reduction) over three months, 2) high polysaturated fatty acid group, 3) controls (normal diet and increase polysaturated fatty acids 20 percent). Criteria for study entrance were: age 50-80, BMI>21, no drug or alcohol dependence, no severe medical disorders, no psychiatric medications, and MMSE <26.

The calorie-restricted group had improved memory performance that correlated with decreased fasting insulin compared to the other two groups. Levels of BDNF and insulin growth factor were no different in any of the groups. Reduced fasting insulin is due to lower insulin resistance and increased insulin sensitivity, which may lead to better synaptogenesis.33 Caloric restriction led to weight loss and reduced BMI. The authors recommend similar studies with more participants, less restrictive caloric diet, and subjects with and without cognitive decline.

DISCUSSION
What can we take away currently as clinical neurologists treating our normal patients who ask how they can best keep their brain from cognitive impairment and our obviously cognitively impaired patients with SMCI, MCI or dementia?

1. AD patients clearly have shown to have impaired systemic availability of several nutrients even in the absence of malnutrition (vitamins C, E, B12, folate, A, DHE, EPA, choline and uridine). This has not been shown in a similar meta-analysis in SMCI or MCI.

2. Replacing these nutrients in the above cognitive disorders along with some caloric restriction (30 percent) and physical exercise (120 minutes/week) suggests this may be helpful in reducing cognitive decline. More aggressive diets suggesting more complex carbs, polysaturated fats, low salt and perhaps less gluten seem reasonable but will require major discipline and support from family members who may not decide to make their own dietary changes. These dietary changes will help control and help reduce systemic and cognitive complications that occur in diabetes, high cholesterol, and hypertension. This program with or without general vitamins and supplements has not shown at this time to prevent AD.

3. Existing reviews of the literature have suggested that there is insufficient evidence for many vitamins and supplements that mitigate normal cognitive decline in humans.

4. Definite exceptions are folic acid, vitamin B12 and in the correct clinical situation vitamin B1 (Wernicke/Korsakoff, chronic GI disorders, Hyperemesis of pregnancy) and B6 (Pellagra suspicion).

5. Vitamin D deficiency has some support to be diagnosed and treated to reduce stroke risk and may reduce or delay cognitive decline.

6. Elevated homocysteine has been shown to increase risk of stroke and cognitive impairment by promoting vascular disease and beta-amyloid 42 production (both increasing cognitive decline). Lowering high homocysteine levels has not been proven to delay or improve cognitive decline at this time. It is still not unreasonable to lower levels by B12/folate supplements or Cerefolin with NAC until definitive studies are conclusive.

7. Current research in cognitive disorders is focusing on pre-symptomatic diagnostic markers so that earlier treatment interventions can be instituted before nervous system damage occurs or is still reversible. This would be a perfect time to institute the above suggested nutritional changes which are low cost and have low side effects. More research is needed to further support this treatment program.

8. More research is needed to investigate the changes in AD specific eating behavior, nutrient metabolism, causes of low nutrient levels in AD, and when they start to lower.

9. The therapeutic system described in Table 1 by Bredesen in 2014 is derived from basic studies of the role of APP signaling and proteolysis in plasticity and the imbalance in this receptor proteolysis that reproducibly occurs in cognitive decline especially in AD. There are numerous parameters that feed into this balance such as hormones, trophic factors, glucose metabolism, inflammatory mediators, ApoE gene status, sleep related factors, and exercise related factors. The major side effect of this treatment system is the difficulty of patients to follow the program as evidenced in the small study. However, the positive result is a lower BMI and improved health in general.

Let us assume that Dr. Bredesen’s multiple therapeutic program as outlined in Table 1 has proven to stabilize cognitive impairment for 2.5 years or more in SMCI, MCI, and AD. How exactly can this program be instituted in our patients/public? How easily can this be done? My answers are hypothetical but based on a long history of treating cognitively impaired patients and their caregivers. (Note: I included only those nutrient and supplements and changes that three of his patients incorporated [the only ones reported in his paper] which does not cover all his treatment suggestions in Table 1.)

PARTS OF THERAPEUTIC PROGRAM ARE RELATIVELY EASY TO INCORPORATE
A, b, c and I depend on treating physician lab testing, unless refused, but also requires help and support from spouse and/ or caregivers to stay on medications/vitamins and C-pap machine, if required. Caregiver may have to observe the patient take their medications/supplements to verify intake.

a. We must still insist that if an individual or his/her family member(s) notice cognitive changes, a full neurological evaluation be done to determine the level of cognitive impairment and possible cause. This may include neuropsychological testing, MRI/CT scan and standard lab work, which usually includes...
CBC and complete metabolic profile often done by referring physician, plus TSH, B12 and folate. I would also include vitamin D (>50ng/ml), homocysteine blood levels (<9) and a methylmalonic acid level if B12 is low or low normal. Based on Table I, consider getting additional lab tests for: CRP (normal<1.0), heavy metals if clinically suspected (Hg, Pb, Cd), hormone levels of testosterone, progesterone and cortisol.

b. Be sure to exclude sleep disorders (sleep apnea, periodic leg movements, REM sleep behavior disorder) and treat as needed.

c. If vitamin D is low, treat with vitamin D3 2000 IU capsules/day. If homocysteine is high, treat with vitamin B12 and folate supplements over the counter (though they may not be well absorbed in the elderly) or Cerefolin with NAC (an active form of vitamin b12 and folate and more effective in lowering homocysteine) that is only available by prescription and not covered by insurance.

d. Optimize sleep. Assuming a sleep disorder is diagnosed and treated, encourage 8 hours sleep/night. There must be good sleep hygiene, and be sure there are no urinary/prostate disorders. Add melatonin 0.5-2mg/night if needed.

e. Help reduce a-beta and lower CRP (if elevated). Start curcumin 400mg/day.

f. Optimize mitochondrial function. Start CoQ 10 (200mg/day), alpha lipoic acid (100mg/day).

g. Optimize antioxidants: Mixed tocopherols (vitamin E 400mg/day), blueberries.

h. Provide synaptic structural components; citicoline (500mg bid), DHA 320-700mg and EPA 180-500mg bid.

i. Optimize hormonal balance. Low testosterone, estrogen and progesterone should be treated to the normal range.

j. Increase middle chain triglycerides—a non-carbohydrate source of brain energy and metabolism by forming a ketone (beta hydroxybutyric acid): Coconut oil one teaspoon bid.

k. Cognitive enhancement. Use herbs Bacopa monniera 250mg and ashwagandha 500mg each day.

m. Enhance autophagy and ketogenesis. Fast 12 hours every night including 3 hours prior to bedtime.

n. Exercise 120 minutes or more per week (30 minutes, 4 times a week). The safest activities for the elderly—not necessarily requiring good balance and normal strength—would include pedaling an indoor bicycle or pedaling a machine that sits in front of a chair.

o. Reduce stress; Incorporate meditation, yoga, music, etc.

p. Encourage increasing brain stimulation, some preferably with family members, such as: games, puzzles, crosswords, Scrabble, cards, computer games, reading regular books or books on tape (easier for memory impaired), arts and crafts, bingo, family photographs, socializing, etc.

CONCLUSION

As you can see, the current scientific nutrient and supplemental information on cognitive decline as it relates to normal aging, SMCI, MCI and dementia (AD) is not quite ready for prime time. However two important provisos must be included in the practical and clinical use of this information: 1) we are dealing with cognitive disorders of the brain, many which invariably progress to severe impairment in quality of life and loss of independence; 2) the majority of recommended nutrient and supplemental information is without significant side effects and harm to the individual.

The most difficult part of this information is implementation. It requires discipline and a good memory, which in most individuals, will require loving assistance from spouses, family or other caregivers. In this proper setting I believe a good part of this program can be implemented. As I stated in my introduction, the fear of cognitive decline especially memory loss and dementia is epidemic across the world. We all grab at anything we hear that may be beneficial and safe (and sometimes not safe) even if not scientifically proven. There is some reasonable evidence in the nutritional and supplemental research literature that interventions can help improve, slow and may delay—but not prevent—cognitive decline.

Other recommended changes regarding various supplements and herbs are still not clear. Waiting for more definitive studies in cognitively impaired and pre-symptomatic individuals will take a lot more time. We as clinicians, who are aging or have cognitive problems, as well as our patients, will want to consider part or the whole therapeutic program. This is a real challenge and we should give our patients and caregivers the information and let them decide for themselves. Remember: “the glass is half full, not half empty.”

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