How Anesthesia Sfx Can Mimic Alzheimer's, Parkinson's and More

No surgery is risk-free. But it can be vastly riskier if you're one of the millions of Americans who suffer from undiagnosed B12 deficiency.

October 13, 2018 By Mike Barr

A 69 year old man goes in for a routine surgery for a benign prostate problem. Within two weeks, he finds it increasingly difficult to walk. His legs go numb. Around this time, he develops gallbladder problems. His doctors schedule him for a second surgery. Despite the his strange reaction to the first surgery (a telltale sign of deficient B12 stores or compromised detoxification systems) the surgeon again gives him nitrous oxide. During the post-op recovery he becomes confused. The numbness in his legs grows worse. Within four months he's pretty much paralyzed from the waist down. He can barely use his arms. He became incontinent. The post-op disorientation has morphed into full blown memory loss and was beginning to look like senile dementia. Credit the persistence of his medical team perhaps. (Many would have just stopped here, racked it up to Alzheimer's and shipped him off to the memory unit of Shady Pines.) Still, it took them three full months to figure out what had happened: severe vitamin B12 deficiency--exacerbated by the use of nitrous oxide. They put him on B12 therapy, but it's too late to reverse his symptoms fully. A full year later, he can walk unaided only for short distances.

Millions of patients deteriorate after undergoing cardiac bypass surgery, cancer surgery, neck or back surgery, neurosurgery, or other operations. Among the most common problems observed in post-op (including post-dental surgery) include:

depression
memory loss
confusion
transient ischemic attacks (TIAs)
balance problems/dizziness
tremor
incontinence
fatigue
difficulty walking/falls
neuropathies, numbness or unexplained pains
stroke

Now for a tiny bit of geekiness, sorry: Vitamin B12 aka cobalamin exists in three oxidative states called +1, +2 and +3. For B12 to have any biological activity it needs to be in the "+1" state (as either methylcobalamin or adenosylcobalamin). The B12 we get from food (or even from an IM injection) is not in this active form, and so it needs to be converted by the body. What exposure to nitrous oxide does is exactly the opposite of what the body needs. It oxidizes any "+1" B12 into the unusable +2 and +3

forms. So if you were already kind of just squeaking by with low levels, this blast of massive inactivation, as it were, could tip you over. At least that's what people who spend their lives studying these kinds of things contend.

Vitamin B12 deficiency is much more common than statistics indicate, and it can have devastating consequences. It has been associated with everything from brain fog, cognitive decline, and heart disease to learning disabilities, infertility, and autoimmune disease. When you don't have enough B12, it can affect virtually every system and tissue in the body. Despite this, B12 deficiency is rarely tested for—and even when it is tested, the lab results are problematic.

B12 deficiency has been associated with or can actually cause premature aging or logical disorders that are similar in presentation to MS or even Parkinson's, brain fog, memory problems, cognitive decline; stroke, heart disease, and other vascular problems.

This patient suffered terribly when doctors exposed him to nitrous oxide without discovering his pre-existing B12 deficiency. Yet his doctors did everything "by the book"—because "the book" doesn't say anything about testing patients for B12 deficiency before they undergo surgery. This probably surprises you if you've had surgery, because your doctor undoubtedly ordered an alphabet soup of impressive-looking tests before your operation—CBC, LYTES, BUN, CREAT, PTT, etc

Look at the list, however, and you'll probably find that serum B12, the most basic test for B12 deficiency, isn't on it. A small number of anesthesiologists do give patients single injections of B12 before procedures involving nitrous oxide, to reduce the risk of a dangerous reaction in anyone who might have low B12 stores. However, this "one-shot" approach probably won't prevent complications in severely deficient patients who are exposed to nitrous oxide for several hours. It also fails to identify preoperative B12 deficiencies that will continue to cause insidious damage and may have contributed to the disorders that led to the need for surgery in the first place.

No surgery, minor or major, is risk-free. Surgery can be vastly more risky, however, if you're one of the millions of Americans who suffer from undiagnosed B12 deficiency. In fact, if you're B12-deficient, even the simplest of surgical or dental procedures can turn dangerous or even deadly—and your doctor or dentist isn't likely to recognize that you're at risk until it's too late.

The danger to B12-deficient surgery patients involves a very common anesthetic agent, nitrous oxide (N_2O), used millions of times a year to reduce pain and sedate patients during surgical and dental procedures. You probably know this agent better as "laughing gas," because of its well-known ability to make you feel giggly, an effect that makes it a popular drug of abuse. For people with undiagnosed B12 deficiency, however, there's nothing funny about N_2O's effects. That's because nitrous oxide has an unusual property: It inactivates vitamin B12 in the body.

If you're not B12-deficient, the effects of N_2O typically aren't dangerous, because your body can renew its stores of B12 within a few days. But if your B12 levels are too low, you have an overt deficiency, or you have an undetected B12 malabsorption problem, exposure to niitrous oxide during surgery can have devastating consequences—even if you're young or middle-aged, and even if you appear fine before undergoing your surgery.

Other B12-deficient patients lose their ability to walk, suffer excruciating pain, become paralyzed or incontinent, or even develop full-blown dementia after exposure to nitrous oxide. Nitrous oxide's ability to wreak havoc on the mind and body of a B12-deficient patient isn't a new discovery. Doctors first reported the phenomenon more than thirty years ago, and dozens of case studies are described in the medical literature. Moreover, it's not just a handful of patients who are at risk. Neurosurgeons Kathryn Holloway and Anthony Alberico say, "Nitrous oxide is very dangerous in the B12-deficient patient. Because B12 deficiency is not uncommon and nitrous oxide use is ubiquitous, the potential exists in every [surgical] practice for this complication to occur." Thus, they stress, "The surgeon should look for evidence of B12 deficiency in every patient." Unfortunately, most surgeons don't, and the consequences of this failure can be kind of horrific.

7% -- or 68?

If you use the relatively insensitive serum B12 as the only measure of B12 rather than some of these newer tests, statistics suggest that only 7 percent of vegetarians and 52 percent of vegans are deficient in B12. But in a more recent study using the more sensitive markers that are capable of detecting B12 deficiency at an earlier stage, a whopping 83 percent of vegans were B12 deficient along with 68 percent of vegetarians. But what about so-called omnivores?

In clinical practice folks reporting finding B12 deficiency to be much more common. One of the reasons for that is that deficiency of a certain nutrient doesn't just come down to how much of that nutrient you're getting from your diet. It comes down to how well you are absorbing that nutrient in the gut and also what the demand for that nutrient is because of certain physiological processes in the body. Thirdly, genetic polymorphisms (mutations in certain genes you've inherited from your parents) can affect your utilization of the nutrient.

A tiny bit more of geekiness..

MTRR and MTR are both genes that are connected to B12 metabolism. If you have single nucleotide polymorphisms (SNPs which folks pronounce as "snips") in those genes, this can create a greater need for B12 than you would have if you didn't have these genetic mutations

And then there are conditions like bacterial overgrowth in the small bowel that have been shown to decrease absorption of B vitamins in the small intestine because the bacteria in the small intestine can actually utilize B vitamins as well. They'll take them for themselves and you will get less of them. Low stomach acid (hypochlorhydria), a major cause of gastric reflux, is a common condition that can also decrease the absorption of B12. Even if someone is consuming sufficient B12, if s/he has low stomach acid, small intestine bacterial overgrowth or genetic polymorphisms that affect her/his ability to absorb B12, then these people can also be deficient even though they're not vegetarian or vegan.

What Can You Do?

There are more sensitive markers for B12 deficiency that are now available including methylmalonic acid (MMA), which can be measured both in the serum and urine. Another marker called holotranscobalamin II, or holoTC, that's in fact the most sensitive marker for B12 deficiency. It is capable of detecting B12 deficiency at the earliest stage, stage one. These markers are much more sensitive to B12 deficiency than serum B12, which means they'll go out of range at an earlier stage of B12 deficiency. HoloTC is just beginning to become more available in the US but has been used in Europe and other parts of the world for some time.

Then we also have homocysteine, which is a marker of B12 deficiency, although it's not exclusively related to B12. Homocysteine can be high in cases of folate deficiency or even B6 deficiency as well. Homocysteine is more sensitive than serum B12, but it doesn't only reflect B12 deficiency. If you see it high, that tells you that either B12, folate, or B6 is low, and you need to do some additional testing with these other markers to determine whether the cause of the elevated homocysteine is related to B12 or if it's related to folate or B6. The upper limit of normal for homocysteine at most labs typically goes up to 13, 14, even 15, but there's a significant body of research suggesting that B12 or folate deficiency can be present at homocysteine levels above eight. Certainly a homocysteine level above 9 would warrant, in many peoples' minds, some additional investigation.

Some of the leading thinkers in this area describe the "four stages" of B12 deficiency-- and note that even if your doctor or NP is thorough enough to tick off the cobalamin box on your blood work slip, this run of the mill serum B12 test will only pick up deficiency once you've gotten to "stage 3"

or 4.

In stage one and two, there will be no observable … usually no signs or symptoms of B12 deficiency. In other words, it's not measurable using any other markers. It doesn't start to cause anemia at that level and it's not going to show up in other markers and it may even be completely asymptomatic. In fact, B12 deficiency doesn't cause macrocytic anemia until stage 4, so that's in the very last stage of B12 deficiency. Unfortunately, some of the more serious effects of B12 deficiency such as nerve damage are irreversible. We have this really tricky situation where B12 deficiency is under diagnosed because it's not being adequately tested for. The symptoms and clinical signs can take years to become evident, but some of the more serious effects of B12 deficiency can actually be irreversible if the B12 deficiency has progressed to an advanced stage and has gone on for long enough.

The Final Little Wrinkle

You may be aware that the conventional lab ranges that are used for these markers and many other lab markers are often not what we really want to see for ideal health. Conventional ranges are typically designed to detect frank disease rather than an optimal level, and serum B12 and even homocysteine are no exception. Most labs define B12 deficiency at less than 200 pg/mL, but it is well documented in the scientific literature that many people experience signs and symptoms of B12 deficiency at levels between 200 and 350. In Japan and Europe, the lower end of the range is 400 to 500. I can't recall. Mental health folks I know who report incredible results with their nutritional investigative work and corrections see any serum B12 under 500 pg/mL as a red flag.

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