

WHAT TO DO ABOUT NEUROPATHY?

READERS SUMMARY:

1. WHAT IS NEUROPATHY?
2. WHAT IS NEUROPATHY ASSOCIATED WITH?
3. HOW IS THIS CONDITION DIAGNOSED?
4. HOW MIGHT ONE CONSIDER TREATING THIS CONDITION?
5. WHAT ARE THE CONVENTIONAL MEDICAL TREATMENTS? DO THEY EVEN WORK?

NERVE PAIN/NEUROPATHY

“Pain is temporary. It may last a minute, or an hour, or a day, or a year, but eventually it will subside and something else will take its place. If I quit, however, it lasts forever. That surrender, even the smallest act of giving up, stays with me. So when I feel like quitting, I ask myself, which would I rather live with?”

I think this perspective, above, is interesting because it allows one to cope when feeling pain but when it comes to chronic peripheral neuropathy that I see most commonly he is dead wrong. I don't think there is anything that slowly destroys a human being more than chronic pain. I see quite a bit of chronic pain in my daily job and decided to write a quick blog about it because of the sheer number of emails I have received to speak on this topic.

Neuropathy is a malfunctioning of a nerve or a group of nerves. It can be caused by many factors. Some of them are

nutritional deficiency, drug toxicity, trauma, vascular damage, poisoning or disease states like diabetes. The one basic premise that is common to all causes is that there is a disruption of normal nerve impulse transmission from the damaged nerve and that information is sent to the central nervous system in the brain and spinal cord for further processing. This malformed message could in fact be perceived by the brain as a sensation associated with pain, numbness, or the muscle innervated by this nerve to be non functional. The most common cause in the USA is diabetic neuropathy. The second most common cause is trauma. This could be physical or iatrogenic.

Neuropathy is generally associated with three sensations.

1. Paraesthesia- which is numbness or tingling
2. Causalgia – burning sensation
3. Dyesthesia- which is an unpleasant burning crawling, itchy, sensation in a numbed or dulled area.

Some patients refer to it as real annoying pain that won't go away. The pain can range from minor to intense. In some cases the pain can begin with a non-painful stimuli touching the affected area. If the motor portion of the nerve is also involved movement may be effected and muscle atrophy or shrinkage will result. Nerves that control autonomic functions like heart rate and blood pressure or gastric emptying can also be effected. When this occurs we generally see changes in tears, salivary flow, erections, urinary retention, respiratory trouble or palpitations.

Diagnosis:

Usually is made by history and physical exam. A MRI to rule out compressive or disc disease is often done. Sometimes we revert to ordering ancillary testing like nerve conduction studies. Often times in tough cases to diagnose we need blood

tests to rule out diabetes or mineral or vitamin deficiencies that can cause neuropathy. There are genetic causes too but they are generally rare. One of the more unknown causes of neuropathy is a recent plane trip. This occurs due to the jet lag and the EMF risk. Read this [hyperlink](#) for further data.

Diabetic neuropathy:

Is a function of the severity of the diabetes and the duration of the disease. The older the patient the more severe the neuropathy seems to be due to reduction in autophagic repair as we get older. The severity is also tied to the HbA1c level, reduction of the HDL level and the elevation of the TG levels. Initially only 8-12% of newly minted diabetics have neuropathy but after 25 years of elevated glucose 60% of diabetics have some form of neuropathy.

How does neuropathy develop?

It begins with a circadian mismatch usually tied to light or the low frequency ELF range. Both alter calcium homeostasis in cells. But excessive EMF is not necessary to be the only cause. Iodine deficiency is a big cause because it causes a loss of myelination of peripheral and central nervous system tissues. A lack of iodine also means the person can not myelinate their nerves because they can not access the ketogenic pathway to myelinate nerves. This has two major effects on nerves. One it decreases the insulation of nerves and can cause a short circuit to develop. The second is more interesting and more significant. It would decrease the the DC current of nerve fibers that has been found experimentally to be tied to tissue regeneration in mammals.

Early humans found them selves in an environment loaded with iodine, DHA, and seawater. Today's modern humans do not.

Sufficient dietary iodine is crucial for proper ketogenesis in our nerves, liver and in our brain. Ketogenesis is critical for myelination of all nerves in humans. These are just three of many tissues that diabetics have massive trouble with.

When iodine is low, estrogen levels tend to be higher in both sexes. This also is associated with elevated SHBG on testing.

Estrogen also blocks the absorption of iodine from the human gut. So any cause of leaky gut can also be an etiology for neuropathy even in a non diabetic. I see this many times a week when some one comes in with a normal MRI and classic radicular pain they think is from compression or from a disc.

This is why women have higher rates of neuropathy, MS, and hypothyroidism than men. It is also why they have less myelin than men in adulthood naturally. Myelination is a proxy regeneration because of the loss of the regenerative DC current below the myelin level and outside the axon of nerve cells.

You might be shocked to learn that artificial blue light in your environment destroys iodine absorption due to poor melatonin signaling in the brain. When iodine is low in nerves over time it causes lower levels of vitamin D and vitamin K2 and atherosclerosis usually develops in the blood vessels that feed the vascular supply to nerves. You might be shocked to learn loss of melatonin signaling in the brain and in nerves also increases estrogen levels and, this in turn, further lowers iodine absorption from the gut in both sexes to cause peripheral neuropathy. This is the most common cause of peripheral neuropathy I see in my medical clinic.

As the disease progresses, blood flow to the nerves decreases because of the development of atherosclerosis from a chronic low vitamin D, vitamin K2 level and iodine. The patients also tend to be dehydrated and have higher BUN/creatinine ratio's as well. This limits the amount of oxygen and nutrients to the nerves cells. This damage occurs at the vasa nervora. Nerves

are fundamentally different in how they transport glucose than other cells in the body.

Membranes of nerves have insulin independent glucose transport mechanisms. They rely on the polyol pathway. This pathway uses polyhydroxyl alcohols. This pathway is the one responsible for many eye diseases like macular degeneration and optic neuritis. These diseases all affect the ability of the eye to absorb electromagnetic signals from sunlight to send to the retina and pineal gland.

Diabetics have constant elevated blood glucose and this allows nerves to absorb the glucose at a higher rate. That glucose is chemically altered to sorbitol by the polyol pathway by an enzyme called aldose reductase. This sugar alcohol is not used by the cell for ATP so it builds up and causes cellular stress to develop. It also limits the amount of ATP a nerve can make and nerves use massive amounts of ATP to transmit messages. When ATP is limited it limits the ability for nerve to use water chemistry for energy transfers. This is why diabetics have serious problems with wound healing and with tissue regeneration. The lack of energy increases overall cellular stress and damages the nerve cells eventually through the action of circadian clock genes on the photoentrainment pigment called melanopsin.

The main effect of this build up of sorbitol is a decrease in a nutrient called myo- inositol. Myo-inositol is responsible for normal nerve conduction to take place. Inositol is a B vitamin compound. The sorbitol causes build up of free radicals, namely peroxides, hydroxyl, and nitric oxide free radicals. In diabetics or in injured nerves, it concentrates these free radicals to make a positive feedback loop and worsen nerve function or pain as time goes on. This in turn depletes nerve cells of taurine. (Hansen 2000) Studies done by Terada et al., in 1998 suggested that there was a close relationship between the unregulated polyol pathway and the depletion of carnitine in the mitochondria that leads to

neuropathy. A “carnitine like” analog called R alpha lipoic acid is thought to balance this depletion and is used as a treatment of diabetic peripheral neuropathy today. It is not as effective as replacing dietary iodine sources in my experience. I also use high dose resveratrol because it blocks the production of nitric oxide in nerves and increases mitochondrial efficiency, even in the face of an unregulated polyol pathway. Resveratrol has massive benefits for diabetics. The dose I like to use in some cases is 500 mgs per day to 10,000 mgs a day based upon the patient’s symptoms and budget. Resveratrol is not cheap, but I believe it is one of the best treatments we have today that is completely under utilized by my profession. I also don’t advocate the use of any sugar alcohols in the diet because this can worsen the neuropathy from any cause. When someone has neuropathy I will immediately tell them to use cold, the Epi-paleo Rx, and avoid non native EMF from their cell phones and from any blue light emitting diode in their environment. The sugar alcohols advocate by many in the low carb community should also be avoided like the plague. I find it disconcerting that many low carb bloggers have sponsors with these chemicals in them. They are not good for people with neuropathy.

Treatment Considerations you must consider ASAP:

1. Strict **Low carb** Epi-paleolithic diet is instituted to lower HbA1c and PUFA content in nerve cell membranes. My suggestion is the read The Epi-paleo Rx book, to see precisely why a ketotic approach is best.

The reversal effect of the diet is not immediate because the lack of fat and iodine is chronic. In my experience, it takes 18-36 months. Interestingly, the Epi paleo Rx is quite high in **carnosine**. Recently, carnosine elevation has shown to increase telomere length and to directly combat glycation from high blood sugars and high PUFA diets. No one is quite sure yet the exact mechanism but since the Epi-paleo Rx contains massive amounts of carnosine it helps tremendously in treatment of

this difficult combination. Another point about diet that must be made: The current American Diabetic Association (ADA) diet is one that favors excessive carbohydrate intake which has a very low iodine content. Also many modern diets call for low sodium, and this further decreases iodine intake. Go to the American Diabetic Association website and look at it. It is appalling in my view as a physician, given what we know today about nerve regeneration.

Since America is a country of frying chefs, be very careful of frying any carbohydrates. Think french fries or funnel cakes! Carbohydrates subjected to frying in PUFA oils form the chemical acrylamide, and this compound is strongly linked to the development of peripheral neuropathy as well. Changing your diet to an Epi-paleo Rx and controlling glucose and PUFA intake is the most important and best way to reverse neuropathy. This must be done with a heavy intake of reverse osmosis or spring water daily.

2. Use of iodine and iodide supplements, R alpha lipoic acid, Resveratrol, PQQ, Magnesium and CoEnzyme Q 10 to decrease cellular stress.

3. Fatty acid balance to reduce peroxide generation. The goal is to decrease omega 6 fats and increase omega 3 fats and also increase gamma linolenic acid (GLA). Borage oil has copious amounts of GLA. So does black currant oil and evening primrose oil. These oils tend to increase blood flow in the vasa nervosa to decrease the cellular stress.

4. Very liberal replacement of B complex vitamins especially B1 and B12. Optimization of vitamin D and E levels due to their immune modulating and antioxidant effects in nerves. Use of inositol is also a treatment choice.

5. Zinc and Magnesium replacement. This only works if you live in a low EMF environment. Zn with Mg are two of the most common mineral deficiencies in neuropathic pain due to the

unregulated polyol pathway. Don't go crazy with zinc supplementation because it can cause a secondary copper metabolic problem but I can not emphasize enough if you are a diabetic you must supplement with zinc, but only when your inflammation is first controlled with the Epi-paleo Rx. It helps wound healing out tremendously. In fact, in my surgical patients who are diabetic I usually have them on three key supplements for wound healing pre op and post op. Those are magnesium, zinc, and vitamin K2. The doses vary based upon the disease severity they have and the type of surgery I am planning.

6. Supplementation with NAC and Acetyl- L- Carnitine. NAC is a precursor for glutathione to rid the nerve of all the free radicals produced by the polyol pathway. The carnitine is replaced because it is depleted by the polyol pathway. Taurine replacement can also be done, but has never been shown to be effective in trials. (Franconi et al 1995.) I rarely have use taurine myself because of the trial data. I find iodine and water are far better choice for peripheral neuropathy of any cause.

7. Optimization of thyroid hormone function is critical. Most diabetics have high cortisol and altered leptin signaling. If you have not read my leptin prescription post I would suggest a peak at it now. This predicts that their thyroid function will be altered or completely shut down physiologically as shown in any textbook of physiology or biochemistry. This has huge implications for the nerve fiber. In order to a neuron to work, it first must be excitable to a stimulus. This excitability requires sufficient amounts of T3. Remember from my leptin series that leptin resistance cause a spike in production of reverse T3. Reverse T3 is a DIRECT INHIBITOR of T3! This means that no matter how good your T3 level is reverse T3 inactivates it directly. So one can see how devastating LR is to nerve function. Generally, the best replacement of T3 in these cases is Cytomel, which contains

peripherally active T3. You can also use over the counter T2 supplements if your doctor will not Rx meds for you.

8. Consider the use of acupuncture. In severe cases that were unresponsive to the above I have seen decent successes with this alternative treatment. The effect however takes 48-106 hours to begin.

9. Talk to your pain physician about placing peripheral nerve stimulators on the effected nerve to increase its blood flow by increasing signal transduction. This can work but I only recommend it in cases where nothing else works. This is rare unless the patient is completely non compliant with the above advice.

Conventional Medical treatment you likely will get today in an office:

1. Neurotin or other antiepileptic medicines, a blood thinner to decrease platlet clumping and decrease blood viscosity to increase flow, and one or several antidepressants for the chronic pain that walks hand and hand with this condition. The side effects really limit their effectiveness. Sleepiness and weight gain and a feeling like your drunk or hungover are common ones I hear.

2. Pain medications that will lead to secondary effects over time. Addiction and chronic pain development due to hyper-excitability of satelllite astrocytes in the CNS. I will post a future blog about what opiates to to chronic pain and the how the affect a leaky gut. Safe to say these drugs do little and they cause many secondary problems that are often harder to deal with than the nerve pain they are supposed to treat.

CITES:

Goldberg RJ, Katz J. 2007. A meta-analysis of the analgesic effects of omega-3 polyunsaturated fatty acid supplementation for inflammatory joint pain. Pain 129(1-2):210-23.