#7- HOW TO DEAL WITH YOUR BASELINE ("ALWAYS THERE-24/7") PAIN

"MARCH TO 90"*

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WHAT IS BASELINE PAIN?

A good argument can be made that the attempt to treat baseline or constant (24/7) pain resulted in millions of people taking high dose opioids. It has long been recognized that some chronic pain patients only have intermittent episodes of pain while some unfortunate individuals develop pain which is constantly present. In the late 1990's there was a movement to relabel baseline pain and call it "persistent" pain and to label pain flares "breakthrough" pain. Regardless of terminology, in the 1990's it was clear: constant, never-ending pain (even while asleep) occurs in some severe chronic pain patients for unknown reasons.

THE ORIGINAL ANSWER TO CONSTANT PAIN – LONG ACTING OPIOIDS

Since no one at the time knew why constant pain occurs, the simple answer was to symptomatically treat it with a long-acting opioid. Hence, we had the birth of manufactured, extended release (ER) formulations which remain in the blood for longer periods than immediate release (IR) opioids. These formulations include the fentanyl patch (e.g. Duragesic®), oxycodone (Oxycontin®), morphine (MS Contin®), hydromorphone (Exalgo®), and hydrocodone (Zohydro®). The clinical approach was simple and universal. Start treating baseline, constant pain with a low dose of a long-acting opioid. Raise the dose at intervals until the pain is suppressed enough for the patient to physically and mentally function. If there were pain flares, they were labeled "breakthrough pain" and symptomatically control and suppress it with a short-acting opioid. Unfortunately, this approach often force patients into the high and ultra-high opioid dosage range.

WHAT CAUSES BASELINE PAIN?

One of the great scientific discoveries of the past decade is the cause of baseline pain. Of great benefit, is that this discovery has led to measures that almost eliminate the need for long-acting opioids.

The cause of baseline pain is pockets or centers of neuroinflammation inside the brain and/or spinal cord (the Central Nervous System-CNS). This occurs when an injury, disease or chemical agent is introduced to nerves, nerve roots, brain, or spinal cord. This activates cells inside the CNS called microglial cells. These cells are the innate "immune system" of the CNS, and when overly active due to injury, disease or chemical agent, they can over fire and cause auto-immune reactions and resulting in neuroinflammation and damage to the CNS.

After the discovery of microglial activation and neuroinflammation, scientists and clinical researchers went to work and have now identified some therapeutic, non-opioid agents that will greatly suppress and control neuroinflammation in the CNS.

BIGGEST SCIENTIFIC BREAKTHROUGH

After the discovery that neuroinflammation is the basic cause of baseline pain, it was discovered that the CNS makes and uses specific hormones to control and suppress neuroinflammation and the over-active microglial cells that produce it. Some of the hormones made inside the CNS are also made and used outside the CNS and include the well-known hormones estradiol (estrogen), progesterone, testosterone, and cortisone. Other

hormones that the CNS makes and uses to control neuroinflammation are lesser known and include pregnenolone, human chorionic gonadotropin (HCG), and nandrolone.

HORMONE NECESSITY

Be clearly advised. We believe the informed use of some hormones is a critical measure to not only reduce opioid dosage but to possibly attain some permanent healing-neurogenisis.

NEUROINFLAMMATORY AGENTS

Research to find agents that suppress and control neuroinflammation has been an arduous process, but it has paid off. Unfortunately, most common anti-inflammatory agents such as Celebrex®, aspirin, ibuprofen, and hydrocortisone are either too weak or don't cross the blood-brain barrier. Here are the major agents that we have identified that appear clinically effective in suppressing neuroinflammation and reducing opioid dosages:

	WORKS	EXAMPLES THAT DON'T WORK
1.	Ketorolac	Celexcoxib, ibuprofen, diclofenac
2.	Methylprednisolone	Hydrocortisone, Triamcinolone, Prednisone
3.	Dexamethasone	" " "

A few other agents have been identified that will suppress neuroinflammation in some patients: (1) acetazolamide; (2) minocycline, clarithromycin; (3) metformin; and (4) pentoxifylline.

RECOMMENDATIONS TO REDUCE OR ELIMINATE OPIOIDS

Below is our recommended program to significantly reduce neuroinflammation and opioid dosage:

1. Ketorolac – 30 mg as a troche on 3 days a week (cannot take 5 consecutive days)
2. Pregnenolone – 25 to 100 mg on 5 to 7 days a week, oral tablet or capsule
3. Methylprednisolone – 4 mg oral on 3 to 5 days a week in the afternoon, oral tablet or capsule
4. Nandrolone – 25 to 50 mg, taken as a troche on 2-3 days a week, or human chorionic gonadotropin-HCG- (500 units), as a troche or injections on 2-3 days a week

WHAT TO EXPECT

When neuroinflammatory agents are started, do not expect to see immediate pain relief like you get with a potent, symptomatic opioid pain reliever. After about a week you will feel more energized with a better feeling of well-being, and you will feel less need to take opioids. After a week, you should start cutting down on your opioids. We have observed patients who have cut down on their long-acting opioid by as much as 50% after the first week. Many patients totally eliminate their long-acting opioid and some of their short-acting opioid within 4-8 weeks.

Remember, neuroinflammation in your CNS may have been there for years, so it may take a few weeks for your CNS to wipe out enough neuroinflammation and create enough healing to reduce baseline pain. Be patient with the process.

Patient Testimonial: "Thanks to working together with Dr. Tennant, treating neuroinflammation, and tweaking his protocol, with his continued help that I was able to successfully reduce my own opioid dosage by well more than half of where I started when I began with Dr. Tennant in 2015." Denise M. - Arachnoiditis

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