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Treating Pain Without Causing Addiction

Release Version 4-18-15

Forward

I've written this in an informal manner on purpose, as if I were talking to a medical student (there's no better captive audience on earth, poor things) - so I could include personal opinions, examples and funny stories (well, I think they're funny). I do have scientific citations for everything here, and have included a few of them, but the rest will have to wait until the next revision: I want to get this out as a sincere "Thank You" to my kiva lenders ASAP. **Thank you!!**

Yes, it's long – sorry – but my 15 years of *paying attention* just might mean that there's one sentence in here that could change your life or the life of a loved one.

Note: if you wish to skip all my philosophizing in this first long section, just jump to the **13-Part Master Protocol** on page 34.

Introduction

Pain is with us from birth to death. It protects us from harm – well, foreseen harm – and it punishes us most arduously for our failures to foresee the results of incautious behavior. In other words, we learn valuable life lessons from pain, both physical and emotional.

Pain is not always useful, however, nor the result of our own errors in judgment. It can result from accidents, from torture and intentional harm from others (we forget about torture, but in the rest of the world torture is a daily reality for many who dare to speak out), from disease, from unrequited love – perhaps the least avoidable of all of life's blows - and from the aging process, which piles pain upon remorseless aching pain with every year toward wisdom. It distracts us from our purposes in life, it can even take the very joy out of living, and even when minor it dulls our ability to pay full attention to the glories of being alive.

Pain can be variable from similar causes: the person who has just survived a battle or a gruesome wreck with relatively minor injuries, even though some of the injuries might be bullet holes or gashes from flying metal, discounts the pain in the contexts of having survived and the prospect of returning home; the person who stubs his toe on a foot of the bed while innocently changing the sheets is more likely to use warm words and dance about as if he had just had a toe bit off by an alligator.

Pain can even be a preliminary to a reward, or a rite of passage. We all know about indigenous peoples who get some body part lopped off or skewered as a ritual to manhood or womanhood without the slightest wince, but we have plenty of similar rituals in our own culture. The skate boarder who misses a trick and ends up on the pavement spasming in shocking pain soon shakes it off and gets up again, proud as punch, his friends high-fiving him and everyone having a great laugh while he asks, "Did you get that? Oh, man, what a great fail for youtube!" When I was a salesman, lo, these many decades ago, I learned to treasure "no's", which represent, to the uninitiated, a very great emotional pain. I quickly learned that, for the product I was selling, I had to get fifty "no's" a week to get five "yes's"
so "no's" ceased to be painful, and I was happy as a clam at high tide to get twenty "no's" in a row on a Monday; it meant I'd be cruising by Thursday.

Kids that play sports learn quickly to shake off pain if they wish to be admired. Parents accept the pain of carrying sleeping children; anyone who has taken their family to a fair without making provision ahead of time for a cart knows well the aching back and arms they'll have to endure before they make it back to their car, parked, of course, at the very farthest reach of the parking lot – and they accept the pain gladly; it is a badge of parent-hood. And, of course, speaking of children, many women face the prospect of very severe pain indeed in childbirth, and it is only relatively recently that most of the pain can be relieved – if desired. I have noted, without intending the slightest judgment regarding choosing pain control (I am quite certain I would choose it), that – when pain control is not used – the joy of giving birth seems to immediately abolish all the pain. It isn't just that the baby is delivered and the pain relieved – I'm sure there's still a lot of pain going on – it's that the pain is no longer important, except as proof of a rite of passage.

Body piercers, especially those that hang from hooks in their back or chest, might seem extreme – but they also often seem quite happy to me; they must get a psychic payoff for all that pain (and they do: endogenous opioids). Finally, how do you explain people who pile on red hot sauce or jalapeno's? That stuff burns! Obviously, there's a reward that makes it worth it, and there is: the upregulation of endogenous opioid neurotransmitters, just like body piercers (so maybe body piercers aren't so strange, after all, are they?)

However, pain that does ***not*** carry a reward is not welcome, and we naturally desire relief from it. The ability to abolish acute pain in the hospital and the operating room has been one of the most profound advances, both relieving suffering and enabling better care.
It is not so easy to perform meticulous and extensive operations when the patient is screaming and writhing under the knife, even though well strapped down, as surgeons prior to anesthesia would certainly attest; the surgeons considered the best were the fastest; the relative leisure the modern surgeon enjoys contributes to the incredibly high level of surgical mastery that is now commonplace in operating rooms worldwide.

But these are all considerations that apply primarily to **acute pain.** The issue that we have in medicine is that we are challenged with devising good methods for addressing **chronic pain without causing addiction** – and it is chronic pain – **chronic pain without reward** - that wears a person to a nubbin over time, and it is the treatment of chronic pain that often leads to addiction and the subsequent devastation of the patient's life.

Narcotics work wonders for pain – some of the time. But narcotics are not always the best choice for certain types of pain, as we shall see – and narcotics really are dangerous. Many people die from over-doses. Many people enter a living hell of addiction, where they get neither pain relief nor pleasure – it is a hell within which the only goal becomes to avoid withdrawal, and – paradoxically - in that attempt at avoidance accidentally over-dosing is all too frequent, with sometimes fatal results, as they swing from one extreme to another.

Many an overdose fatality goes like this: the patient gets cut off – because they have become addicted. No matter they became addicted *by prescription* – they still get cut off, for one reason or another. They go into withdrawal. Withdrawal, please remember, for the severely addicted, is often *unbearable.* They somehow get their hands on narcotics – pills they steal or street drugs they buy. Dosage is not accurate, especially from the street. However, they also have been in withdrawal, and *their tolerance has changed – what was previously a tolerable dose may now be too much, but they don't know that.* They dose themselves, trying to hit the right dose. The dose might not quickly be enough to get them out of withdrawal – so they dose again. Or, they might miscalculate, especially with a powder from the street. In either case, they accidentally overdose...and they die.

It doesn't take long to get addicted, and it certainly does not take a "drug seeking" attitude to get addicted – far from it. One week of round the clock narcotics will get a person very close to full-blown addiction, and two weeks will guarantee it, and neither attitude nor intent has anything to do with it.

Now, before I go further on this, let me hasten to point out that the potential for addiction is in indirect proportion to the severity of the pain. In very severe pain, such as a burn victim, addiction is not a concern, nor does it come on nearly as quickly. However, even in severe pain, treatment with narcotics for long enough will result in addiction.

Sometimes pain is so severe or so allied with death that we are not overly concerned about addiction. In hospice, it is inappropriate to over-worry about addiction – what matters is relief from agony while a person is dying. Any other considerations are barbaric. The only time I give thought to the escalation of narcotics in hospice is when the patient is likely to take a great many months or even years to die, and then my main consideration is to avoid the unfortunate scenario where the side-effects of the ever-escalating dose of narcotics become worse than the pain.

In patients who are susceptible to **nausea** from narcotics this is a very urgent concern, but – on the other hand – we do have a few fairly good medicines for nausea: the old but still useful

promethazine (Phenergan), the often effective but limited application metoclopramide (Reglan), the occasionally useful scopalamine (Donnatol, etc), the only occasionally useful and dangerous to the elderly and children meclizine (Dramamine, Antivert, etc), the sometimes useful natural agent ginger in high dose combined with a sugar, (here's a nice link for making up your own ginger nausea remedy: http://everydayroots.com/nausea-remedies) – no, I have no connection; I just include the link because the idea of making up a ginger nausea remedy might be too surprising to many readers - the old use of pure Coca-Cola syrup (really only useful for occasional use, but many the throwing-up child has been the grateful recipient of mom's run to the soda fountain), the often useful but by no means guaranteed ondansetron (Zofran; and I abhor the attitude among too many that if ondansetron (Zofran) doesn't work the patient is "out of luck" – no, if the ondansetron (Zofran) doesn't work, promethazine (Phenergan) or something else should be considered at once) - so even nausea can be well relieved...if the doctor is skilled in treating nausea and pain simultaneously and the nurse is dedicated to relieving suffering...which, in my experience, is most often the case with hospice care, but not necessarily in the ED or the hospital. I would also add the note that the suffering from nausea is far more serious in the ill than is commonly appreciated, and that I, for one, take nausea very seriously.

I once jumped overboard from a sailing ship and swam a mile to shore in order to escape sea-sickness. I didn't care if I drowned or got bit by the giant barracuda that lurked nearby, but I sure cared about suffering one more second from nausea.

By the way, you'll note that I did not include any of the anti-cholinergics in this list except the milder meclizine...all the syrups/pills/injections based on diphenhydramine, hydroxyzine, etc, (Benadryl, Atarax, Vistaril, etc), that have a stronger effect and longer half-lives (10 to 30 hours) than meclizine (4 to 6 hours), and which are stronger anti-cholinergics, I left out because I would never use them. There are so many better choices, why use these that can have very nasty side-effects? Yes, promethazine (Phenergan) is a mild anti-psychotic, and can certainly cause the same problems – although it usually doesn't, it just usually causes a welcome drowsiness – but it is prescription, you see – the danger of the anti-cholinergics is that they are (inappropriately) Over the Counter (OTC) – and so are easily available to the patient for continued over-use. Patients are no dummies – if you give them something in the hospital that they can then buy by the bushel OTC, what do you think they'll do when they get home?

The problem is this: a very occasional use of any of the anti-cholinergics is acceptable, but chronic use leads to brain shrinkage – yeah, no kidding – and even occasional use can lead to severe confusion, which can lead to unfortunate diagnostic interventions. In the elderly, the anti-cholinergics can bring on a full-blown confusion which presents as a severe psychoneurosis. Does any of this sound like a good idea to you? Not to me, but I've seen NP's and PA's and Hospitalists, who really should know better, hand them out like candy. I guess because it's easy – but I don't think it's a favor to the patient, because they are instilling the idea that it's OK to use these drugs like candy.

I will tell you a cautionary tale: an elderly woman of my acquantaince was admitted to the hospital with symptoms of a heart attack – but none was found. However, while she was there, she became agitated. She was not normally agitated at home, so I think perhaps the combination of being starved and in a strange, aversive place caused her to become upset and confused. The Hospitalist was called in, and she prescribed risperidone (Risperdol). Really? Wow. WOW. I think crackers and apple juice would have been much more helpful. When that didn't calm the patient down to her satisfaction, she then gave her diphenhydramine (Benadryl). Now the patient was completely out of her mind and wildly agitated – as she might well be; her brain was now deprived of both dopamine and acetylcholine, both required for normal brain function. Then, the Hospitalist wanted to do a

brain CT – for "altered mental status" LOL. ROTFL!! Except it wasn't funny, was it? At that point the daughter said "to heck with this" and took her mom home – over the hospital's protests, of course. It took twenty-four hours of intense attention and care for her mom, but the daughter eventually got her back to normal. What a disaster – and the anti-cholinergic was the "coup de gras" that pushed the poor elderly lady completely over the edge.

I have to wonder: how many nursing home patients are being given anti-cholinergics in order to "calm them" or put them to sleep, and who now have had taken away from them their very last vestiges of sanity?

There are also different narcotics for different situations. Methadone can be marvelous in situations where other narcotics are causing problems due to escalation of dose, and there is the additional consideration that there is no upper limit to methadone dosing, just the appropriate level of dosing for the current situation – and it is a good pain killer without the side effects of very-escalated morphine, dilaudid, fentanyl, oxycodone and hydrocodone.

Fentanyl is to be avoided when respiratory collapse is a concern, and when dose escalation makes the use of fentanyl no longer appropriate; it can, at increasing doses, cause paralysis of the diaphragm, and more than one elderly patient has (silently) suffocated to death by overuse of a fentanyl patch, or old patches left on while new ones were applied, with nobody the wiser except for the patient who suffered greatly before they lost consciousness. Fentanyl patches are often used for convenience, but in actuality extreme care should be exercised; it is hardly commendable to place the patient in danger of suffocation for the sake of convenience.

Morphine can cause over-whelming itchiness in some, and debilitating nausea in others; dilaudid is often a most-welcome alternative with less likelihood to cause these problems.

As a parenthetical note, I have no use for dirty drugs like tramadol, with its many metabolites, ineffectiveness on pain and strange psychological side effects. I also am cynical about combinations with acetaminophen, even though the business of medicine forced me in the past to use them far more frequently than I would like. It never made sense to combine a narcotic with acetaminophen, and it still doesn't make any sense. As far as I can see, it only puts addicts at risk for killing their livers from accidental over-dose; it doesn't take that many; even just five Vicodins or Percocets at once can get you into the liver-toxic range, and ten at once definitely will. Finally, medicine is waking up to the fact that acetaminophen should never have been so promoted in the first place:
http://www.bmj.com/content/350/bmj.h1186
I am grateful for these authors really taking on the topic and helping medicine start divorcing itself from the business of pharmaceuticals and instead pay attention to facts.

In any case, I do not think addicts deserve the death penalty from acetaminophen overdose; combining a narcotic with ibuprofen is far more effective and safer, but those alternate combo's are not covered by low-cost insurance and are so expensive they are out of the reach of most people. The obvious answer is oxycodone IR (instant release) without the acetaminophen, but that has always been in a higher narcotic class and carried more difficult prescribing rules – so most doctors used to stick to Vicodin or Percocet for convenience.

Now that Vicodin and Percoet have been raised to a Level II classification, the same as oxycodone IR (or Oxycontin, morphine, dilaudid, and fentanyl for that matter), it will be interesting to see if Vicodin and Percocet continue to be prescribed. I don't see why they would be – what's the point? The convenience has been eliminated, and the acetaminophen doesn't add anything except toxicity to the liver.

The list of considerations goes on; I only give these to round out the picture a bit, so the reader may get a sense of just how complicated the effective treatment of pain – especially chronic pain – can get.

I think the final point I need to make, which may not be obvious to those who are not familiar with addicted patients, is that the hell that addicts get into is the impossible situation they eventually find themselves in wherein the required dose of narcotic to avoid withdrawal is also the dose that causes unbearable side-effects, such as nausea, dizziness, unbearable itching and anhedonia.

What is anhedonia? It is the loss of interest in the pleasures of life. When you look forward to supper, you are exhibiting hedonia. When you expect to enjoy a movie, you are exhibiting hedonia. When you hope for a kiss – or hope to plant one – you are exhibiting hedonia. One does not have to be a hedonist in the "mortal sins" sense, Goethe's Faust or Marlowe's Faustus, to exhibit hedonia – hedonia is a natural part of a healthy life.

Anhedonia may not seem like a big deal to those who have never been addicted, but it can become a very big deal indeed. Initially, when a person is first overwhelmed by the psychological pleasures of narcotics, they welcome both the psychic pleasure and the relief from pain. **But what happens later is that they realize they are no longer participating in life:** they find themselves unable to enjoy their child's triumphs or sympathize with their child's defeats; intimacy with their partner ceases to be important; they completely forget both family and dear friends; they lose all sense of ambition and whatever purposes they might have nurtured in their heart float away on clouds of indifference – years go by, and they accomplish nothing. All of this creates a profound sense of loss, a deep sadness from inaction, and it becomes a psychic hell.

You may ask: "well, why not stop?"

The answer is withdrawal. Very few people can withstand the rigors of withdrawal unaided. Movies glorify it – and the movie that portrayed Ray Charles withdrawing "cold turkey" was, if anything, under-stated, it really is incredible to me that Ray Charles did that – but in real life, most people truly suffer the tortures of the damned, and will do anything to escape the rigors of withdrawal. Please remember – not to denigrate Ray Charles' courage or endurance, which was remarkable – that, nonetheless, Ray Charles was both highly motivated and stood to gain tremendous admiration from a huge public – or at least those "in the know", and there were plenty of them - and I rather suspect that that was part of why he so stubbornly insisted on going through withdrawal without medical assistance. Your average citizen has no such gains to make, except the personal gains of being clean again. This might seem like plenty, but just wait until you are in the throes of withdrawal, and we'll see just how willing you are to continue without relief. For many people, I beg you to accept the reality that withdrawal is simply unbearable without substantial assistance; anything less is a callous disregard for suffering, and I have no patience for the sociopathic notion that an addict deserves to suffer.

In fact, it pains me to observe – for I can do nothing about it – that we routinely torture addicts in jails; it is commonplace for an addict that has been jailed to suffer withdrawal without effective treatment. Our society condones this torture with the attitude that the jailed addict "deserves to suffer," and I can only say we still have a long way to go before we reach truly humane levels of civilized behavior.

I should also include here that addicts do sometimes die from withdrawal – withdrawal can become fatal from heart arrhythmias - and they die in jails and prisons, without adequate care, from withdrawal.

I am also very suspicious that these mysterious suicides in jail cells are actually secretly addicted victims who choose suicide over continuing to suffer from withdrawal (and the humiliating disclosure to family and friends) – but it gets reported as a suicide only. I wonder.

However, these considerations are, for the purposes of this paper, a bit of a detour, so I make the point, again, only for completeness, sadly reconciled to one more injustice in the world I am powerless to affect.

I need to address my use of the word "addict". I do not employ the term in a perjorative sense. I make no moral judgments, and, indeed, moral judgments of addicts have no place in medicine; this ought to be self-evident. An addict has not "failed", no matter the circumstances of their addiction. Some people become addicted through misadventure. Some become addicted through searching for relief from trauma, sexual trauma, physical trauma, emotional trauma. Some become addicted through simple childishness; it is a condition of human nature that immaturity sometimes leads to mistakes that carry severe repercussions. Some have addiction forced upon them; many a post-surgical patient has been sent home addicted without adequate provision for their followup care. Captives in the sex trades are addicted by force to enslave them. Finally, some become addicted, over time, as a result of care for severe chronic pain; post-Motor Vehicle Accident victims find themselves in this dilemma all too often.

What does it matter how someone became addicted? It does not, at least from a moralistic viewpoint. Certainly there may be considerations arising from the manner of addiction – the person who finds relief in narcotics from abuse will do better if they can access effective therapy for the abuse, certainly; the soldier who is placed on dangerous drugs to treat PTSD will do better if more caution is exercised and an earnest search for better treatments for PTSD is conducted; the addicted sex slave, whether by force or misadventure, deserves the best care available.

Finally, a brief note on the process of addiction itself. **In simplest terms, nature makes us pay for un-earned joy.** If we don't earn our joy, through accomplishment, or through serving others *(actually the more important point)*, we have brain cells that make us pay – because only **earned joy** has survival value for the species.

This is actually why the philosophers caution us to exercise moderation in all things – including happiness. If we allow ourselves to revel in too much happiness, happiness that is out of proportion to what we have earned a right to, depression will follow – nature will attempt to restore balance. A too-happy human is too unobservant of threats such as saber tooth tigers, sheer precipices or credit card debt. Many is the bipolar who, on a happiness binge (mania), has destroyed their family's finances for decades with a wild spending spree that maxed out every credit card they possessed.

Epicurus said, in around 300 BC, *"Nothing is enough for the man to whom enough is too little."*

Ralph Waldo Emerson said, *"Moderation in all things...",* but then he went on to say, perfectly aware of the realities of human nature, *"especially moderation."*

Mick Jagger said: *"Too much is never enough"* – and Mick should know.

These sayings, unfortunately, describe the human condition in all of us – for it is very much a part of human nature to desire more happiness than we have earned – and so the immature (human) individual who gets access to narcotics and goes overboard for too long with the artificial – unearned – happiness that narcotics can imbue, pays a very dear price when the compensatory

neurons impose withdrawal symptoms in the attempt to restore balance. If humans could take Vicodin for three days and then stop, we wouldn't have all these problems – but most humans are not capable of such a degree of self-control, especially not in the face of real pain. Since it is universal, I see no reason to judge. It is a fact of human nature, and it is up to medicine to find ways to reduce the danger without abandoning patients to inadequately treated pain. And, I never forget, Jesus said, "Judge not"...and I think he knew something.

The highlight of *my* week? Second to the service, giving singing lessons on Sunday at church to a small group of children. Now, that is earned joy. I am not at risk of addiction, of course, because there is no un-earned joy – so there is no depression and there is no withdrawal and there is no buildup of tolerance, not if I'm paying attention to the progress of those in front of me – I simply feel a delightful anticipation during the week for the next lesson and my need to prepare for it. This will seem trivial and foolish to you – and yet if you take one thing away from this entire paper, it is this paragraph – it trumps everything. And, if you suffer from pain, find the one thing *you can do, that actually comes from your own talents or desires* to help others, and you will be astonished – astonished – at how, for that hour, you forget some of your pain, and what is left over is less consequential.

However, as I learned long ago from a horse, you can lead 'em to water, but you can't make 'em drink. The time I tested that time with a real horse, I ended up laughing myself silly. It was so true! No matter how hot the day, no matter how hard and dry the workout, no matter how much lather on the horse, no matter how much water you splash into their mouth and over their head (which will get you a very strange look from the horse), if that horse is not ready to drink – they won't. Period. It's truly hilarious.

I will leave the treatment of addiction for another paper; I have rambled too long as it is, but I wanted to "clear the decks" of misconceptions, by presenting the realities of addiction, before I addressed my main topic: how to avoid addiction in the first place. Any physician who treats pain has to confront the problem of the already-addicted patient, and it is a very difficult problem. I can successfully treat such a patient, but it is not easy, and it requires successfully treating the true cause of their pain before treating their addiction is even possible – if I am to be humane, and if I am to gain the patient's trust and retain their compliance. I, like any other physician, have patients where I am obligated to continue narcotics longer than I would wish, in order to get them correctly treated and then have the opportunity to get them free of their addiction – but these are topics for another paper which I shall write; the treatment of already-addicted patients is really difficult, do not underestimate the difficulty of it – for this paper, I will confine myself to the non-addicted patient and how to avoid causing addiction.

The first consideration in avoiding causing addiction when treating pain **is whether a narcotic should be used at all.** A narcotic is not always the best choice.

I should also draw your attention to another class of drugs that are also extremely dangerous: the benzodiazepines: Valium, Restoril, Ativan, Klonopin, Xanax – these drugs are viciously addictive, and a person is often not aware that they have become addicted – until they end up in the ED with hallucinations, having stopped the drug, not realizing they couldn't just stop it cold turkey. Also critically important to keep in mind is that when you add a benzodiazepine on top of a narcotic, respiratory depression becomes much more severe – you really can kill your patient by thoughtlessly adding a benzo on top of a narcotic. Detoxing a patient from a benzodiazepine addiction is not easy either – but I'll leave that for another paper, I just wanted to note that there are two classes of drugs that are viciously addictive, can synergize to kill by respiratory depression, and have to be used with great caution: the **narcotics** (opioids) and the **benzodiazepines.**

I shall first consider methods of treating pain without the use of narcotics. Then I shall consider methods of using narcotics without causing addiction for those kinds of pain where narcotics are essential.

After considering methods of avoiding narcotics individually, I'll talk about how to integrate them into a whole program, with or without the inclusion of narcotics.

Alternatives to Narcotics.

The Glucocorticoids

Glucocorticoids – commonly referred to as "steroids", but not the "steroids" that athletes use. It must be recognized that many types of pain involve inflammatory cells of the immune system. I think this tends to be under-appreciated. We think of pain as arising from mechanical causes – the hammered thumb, the burned finger, the ruptured disc pressing on the nerve root – but in most cases our pain arises not from any mechanical or thermal injury but from the actions of our own immune system. In such cases, a narcotic may partially block the pain from being felt by the brain, but in fact we are treating the wrong thing with the wrong thing.

I will tell a little story as a case illustration. I had an attack of pancreatitis – an autoimmune type of pancreatitis that is not that rare for older men – and was in agony. The pain was unbearable. I went to a local hospital, TMC, where I was treated with surprising rudeness – but I'll leave that out of this paper. Another day. What matters is that I eventually found myself under the care of a young hospitalist whose plan was to give me inadequate doses of narcotics, a saline IV, and nothing else.

When I realized I had only succeeded in putting myself into a "medical prison" and that I was not going to get the help I needed, I told the nurse that if I was going to suffer, I might as well suffer at home, where I could at least treat myself and have my own bathroom.

I then walked out of the hospital against a whole horde of protesting staff. Once my fiance' got me home, with me curled up in the foetal position in the reclined passenger seat, moaning and gasping all the way, and after stopping at the pharmacy (thank God for drive-through pharmacies) to pick up the prescription of dexamethasone I had called in on the way, I crawled into my house and took the first dose of eighteen 4 mg tablets – a dose of 72 mg, which is a 1 mg/kg standard full dose...but by no means excessive. I also started drinking green tea.

Within five hours I went from *agony* to no pain at all...*none*...without any narcotics.

You might ask why I went to the hospital in the first place – why didn't I just stay home and take care of myself properly? Because I was in so much pain initially my thinking was all fuzzed up, and at first I thought I had chest pain (the brain doesn't always map vagal (visceral; abdominal) pain accurately); it was only upon reflection, hours later, in the hospital, after partial pain control from a small bit of IV morphine (thank goodness for ED docs), and finding that the pain had seemed to migrate from my chest to my upper abdomen, that I realized what the real problem was and what my true course should be. And, of course, my labs coming back (including my specific demand to add-on Lipase and Amylase, which nobody at first believed was necessary, and which the blood draw tech from the lab ignored, failing to ask the ED doc to add it on as I requested, so I had to bother the ED doc to get it added, after a great delay that didn't help matters either, thank you very much TMC lab), proving I had pancreatitis, confirmed what I had already figured out – although it didn't change the hospital's old school "do nothing" attitude.

Even doctors fall prey to the mind-stupefying effects of pain; how much more compassion we should have for the lay public, who don't have any understanding at all, when a well-trained doctor can't even think his way through acute pain.

What is the real lesson here? The lesson is that for some painful conditions, a glucocorticoid, such as dexamethasone or prednisone, can be more effective than a narcotic, and far more appropriate for the real cause of the pain. In my case the pancreatitis was autoimmune, so it followed that the pain was mostly due to inflammation, and it followed that stopping the inflammation cold with a high dose of a glucocorticoid (dexamethasone) would stop the pain – and it did. The green tea, containing EGCG (epigallocatechin gallate), which helps block HMGB1-mediated inflammation, helped calm my pancreas, and in another day my labs, which had been sky-high, started to quickly fall back to normal, and I recovered fully, without any need for a narcotic at any time after I got myself back home.

I should note here that there are different types of pancreatitis, and the reader should not assume that dexamethasone and green tea will effect a miracle cure; I was fortunate.

Another example: I inherited a patient who had **ankylosing spondylitis.** He had been treated, most ineffectively, for years with gobs of NSAIDs around the clock. The only good thing I can say is thank Heavens he hadn't been put on Vicodins or Percocets for years; at least I didn't have to treat him for addiction.

I took away his NSAID's for a few days (so they would work again), put him on a very short burst of dexamethasone, upon awakening, and then continued him on very low dose prednisone, again only upon awakening, with high-dose ibuprofen on a more moderate schedule. He improved vastly; he went from "frozen" – literally afraid to move for fear of pain – to living a normal life again.

Did we have to carefully adjust dose and do repeat bursts from time to time? Yes. Did I have to explain to him twice that no, he couldn't hammer gobs of NSAID's all day every day? Yes, but he got it after the second explanation (normal). Was it a vastly better strategy than (only) gobs of NSAID's? Most assuredly. Did he ever get addicted? No, there was nothing in his regimen to get addicted to. Did the steroids cause problems? Well, yes and no. Because I only permitted him to take one dose a day upon awakening and made him take breaks from time to time – both important points for the physiologically correct use of steroids – his side effects were non-existant.

Are there new really great drugs like anti-TNF agents? Yes, but...remember, I'm treating mostly poor people. They can't get these drugs. So, I have to figure out creative ways to make cheap drugs work. Are there supposedly programs where poor people can get fancy, expensive drugs? Sort of. They make you jump through a zillion hoops while you have no clue whether you will succeed, and it's easy to get disqualified. All in all, a ton of work without any assurance there will be benefit – meanwhile, the drug company is making billions, and their programs for poorer patients are mostly, in my opinion, time-wasting window dressings more for marketing than providing benefit. And, can you tell just how much I love working for free to jump through drug company hoops – none of which I get paid to do – while they make billions?

By the way – I should make a note here about biologics. I will not use biologics (drugs that kill immune cells). I leave the use of biologics to rheumatologists, who I presume will use them with appropriate – and extreme - caution. Personally, I consider biologics dangerous. They may cause cancer or other devastating illnesses only rarely – but to the rare patient, it is devastating. I have never created a disaster with anything described in this paper.

Steroids – glucocorticoids – do carry risks, yes, but I've never seen the risks in my patients, and the risks do not include such appalling conditions as cancer. More on the risks later.

Another example: a patient with crippling osteoarthritis, head to foot. He had also been on a very steady course of gobs of NSAID's for years, and the NSAID's no longer seemed to work at all. I took him off the NSAID's temporarily and used a one-day burst of dexamethasone – and then transitioned him to a rotating schedule where he took extremely low dose prednisone upon awakening part of the week (5 mg upon awakening) and high dose ibuprofen once or twice a day also for the other part of the week, convinced him to change his nutrition to anti-inflammatory (see below), and he did marvelously.

I also included clonidine at bedtime (below) and eventually treated the worst places with prolotherapy (below), and all in all, he became better than 80% pain free and stayed that way – and never a narcotic was used.

Now, osteoarthritis is often thought of, and taught as, essentially a mechanical type of arthritis – the result of "wear and tear". Well, it is, initially – but the primary mechanism of pain and stiffness is not mechanical at all, it is inflammatory – so treating inflammation directly is more appropriate and more effective.

I should also note that in his case I did not employ two agents I certainly could have and should have for one of them – fish oil for sure and glucosamine maybe. I now do include them also, or at least the fish oil, so the whole protocol would be a combination of daily fish oil, perhaps daily glucosamine, and rotating steroid and NSAID.

Burst use of glucocorticoids (dexamethasone)

The very short term use of glucocorticoids can be, in my opinion, a game changer for the patient with severe pain – these drugs enable you to completely "reset" the patient's inflammation and do a "start over".

The importance of a truly effective "restart" for many patients cannot be overemphasized.
It enables you to wipe the slate clean and install an entirely new program...everything here!
Are there risks? Using dexamethasone for a one-day burst, the absolute risk for each patient is vanishingly small. More on the risks below.

Other glucocorticoids are certainly used: the medrol dosepak (oral methylprednisolone), oral prednisone, methylprednisolone injectable, and so on. I prefer dexamethasone. A one-day burst gets the job done with almost zero risk.

Methylprednisolone is an injectable alternative, with half the sodium-retaining effect of cortisol, but the half-life is at best 4 hours. For very mild cases where you just want a brief bump, I suppose you could do one injection. Of course, we are talking about pain syndromes here, I'm not discussing autoimmune disorders which require long courses of treatment.

The medrol dosepak, which is prescribed ubiquitously, is not any more effective than a one-day burst of dexamethasone, and now we're into a five to twelve day course of steroid, which carries much higher risk – and prednisone, also used often, is, at higher doses, too much a sodium retainer – so, all in all, I find dexamethasone works better for several reasons. First, while the half life of dexamethasone is admittedly a bit long, up to over 24 hours, that actually is an advantage – a one-day burst carries over into the next day. The additional vast advantage to dexamethasone is that it

has no mineralcorticoid effect at all – one does not have to worry about sodium retention and thus water retention. Methylprednisolone has half the mineral-corticoid effect of cortisol, and so does carry some sodium retention effect, which can lead to edema and volume overload. None of the other glucocorticoids are acceptable: they all have far too much sodium-retaining effect – so, it comes down to dexamethasone and methylprednisolone, and dexamethasone is the clear winner.

Oddly enough, it appears that a high dose given only once, by IV or IM injection, actually carries less risk than an oral taper over days – and there's no question that risks are very minimized by a one day burst vs chronic therapy over weeks and months. Risk of avascular necrosis of the femoral head appears to be much more associated with duration of steroid use over many weeks and months than peak blood level for one day. So, to really minimize risk, it is actually better to give the patient an IM injection than to write them an Rx – or even send them home with a syringe to do their own IM injection in the morning. Yes, some people can handle this, with a little instruction (I can hear all the nurses hitting the floor after fainting at the idea).

I must emphasize that the majority of risks from steroid use are from ***long-term use,*** not burst doses – and especially not ***one-day*** burst doses. I have searched, and continue to search, the literature on this, and the data all points to the longer the use the higher the risk, with extremely small risk for a one-day dose, and even lower – vanishingly lower - if injected – so high dose for one day does not appear to be the issue; chronic dosing over weeks and months is the primary issue.

So, this brings up the possibility of repeat burst dosing. How frequently would be too frequent? I think once a week is definitely too frequent, once a year is fine, and once a month is probably completely sustainable. If the patient is doing everything else here, it is hard for me to imagine situations where a once a week burst therapy is really needed, but there are situations where this can transform the patient's life. Please keep in mind that we are considering strategies to avoid full-blown addiction; it is not a favor to a patient with severe pain issues to get them hopelessly addicted just to try to avoid a small amount of risk from burst-therapy dexamethasone; when the day comes that the patient must deal with the addiction, their life will become a hell on earth, and while we minimize this in medicine, pretending this is not a big deal, it is most assuredly not minimal for the patient.

For short-term use, ***in low-risk populations,*** the risk from short-term use is very low – **essentially one case per thousand patients treated with short-term glucocorticoids:**
http://www.ncbi.nlm.nih.gov/pubmed/15943727
And please note that these patients were being treated for a month or longer – these were not just one-day treatment courses.

Finally, it is actually not at all clear that most cases of avascular necrosis of the femoral head are due to the steroid alone – the risk is vastly elevated when there is a serious underlying condition, such as Systemic Lupus Erythmatosis (SLE).
http://www.ncbi.nlm.nih.gov/pubmed/11770392

NSAIDs – ibuprofen, naproxen

Now, you ask, don't NSAID's also treat inflammation? Not well. They interfere with part of the inflammatory cascade, sure, but they are not wholly effective COX-2 inhibitors, and they do nothing to directly inhibit the action of inflammatory cells – macrophages, neutrophils, T-cells, even B-cells, which infiltrate osteoarthritic joints and proceed to merrily message each other with

inflammatory cytokines that are far "upstream" from prostaglandin synthesis (the target of a COX-2 inhibitor).

And, because the body is perfectly capable of upregulating "treatment evasion" pathways to defeat the anti-inflammatory effect of NSAID's – and does, it does – the constant administration of NSAID's only succeeds in causing "treatment evasion" to occur; it only takes two weeks. The textbook says that tolerance does not occur with NSAID'S. Maybe so – but I can assure you that "treatment evasion" does occur. COX-2 is not the only inflammatory pathway: there is HMGB-1, there is LOX-5, and I'm sure there are more.

By the way, you may have noted by now how many changes in the human body seem to take "two weeks". That's because it takes about two weeks for changes in the expression of genes from our genome (DNA) to become noticeable – so it often seems to take about two weeks for something to change, to appear, to wear off – no matter what it is.

For example, when I did some research on the effect of curcumin on the expression of VEGF, I found that – sure enough – it took two weeks for curcumin to start to cause VEGF to fall, and two weeks – after stopping curcumin – for VEGF to start to rise again.

An added problem is that there is a "ceiling" to NSAID effectiveness – so taking more, all day long – isn't going to help with pain, but it **is** going to increase risk of GI bleed.

You'll see doctors rotate NSAID's – ibuprofen to naproxen, then to lodine, then to indomethacin or sulindac or something else – but it's not very effective; once "treatment evasion" is complete, no NSAID will work well. To use NSAID's effectively, they must be used less than constantly, but dosed high when used, and rotated with something else – and there's not the slightest point in using anything but either ibuprofen (800 mg at once, once or twice a day) or naproxen (generic 500 mg, either two once a day or one twice a day).

The differences between ibuprofen and naproxen?

Naproxen is more selective for COX-2 than ibuprofen and thus a bit more effective against pain than ibuprofen – but ibuprofen usually works just fine.

However, naproxen has a significantly higher risk of causing GI bleeds than ibuprofen.

The risk for cardiac events is a bit lower for naproxen than ibuprofen, but neither ibuprofen nor naproxen are high-risk for cardiac events compared to other NSAID's – so long as they are not high-dosed around the clock.

Naproxen is only modestly more expensive than ibuprofen, so all in all it's a toss-up – naproxen is a bit more effective, ibuprofen overall is a bit safer. But, used in a patient where you take care to bring down their inflammation and consider comorbidities, as well as limit dosing frequency as much as feasible, in fact both are really very low risk.

NSAIDs and GI Bleeds

There is no question that risk of bleeds goes up with more NSAID usage. Here's a nifty little study from Turkey that clearly shows the association between increased usage and increased bleeds: http://www.ncbi.nlm.nih.gov/pubmed/17891690

However, what is the actual risk for each patient using ibuprofen?

The incidence of GI bleeds from chronic over the counter use of ibuprofen is actually very low. In this review, the incidence of hospitalizaton due to a GI bleed was 0.2% - so, that meant that for every 1,000 people hammering NSAID's frequently, 20 would end up in the hospital. But this is actually a bit misleading, because in that statistic we are not taking into account time of exposure: converted to a cumulative-risk model, the range – depending upon which studies were included in the meta-analysis – ranged from 0 to 3 hospitalizations for GI bleed per 1,000 ***patient years*** of chronic use of ibuprofen:
http://www.unboundmedicine.com/medline/citation/22017233/Over-the-counter_ibuprofen_and_risk_of_gastrointestinal_bleeding_complications:_a_systematic_literature_review.

This is vanishingly low. But it depends upon the patient population. Healthy patients with pain are not at much risk for bleeds. Elderly, ill patients with other factors ARE at greatly increased risk for bleeds. Otherwise healthy pain patients who also have *H. pylori* gastritis are ALSO at increased risk for bleeds.

So, a serious consideration is additional disease. We know, for example, that if a patient is much older and has significant artery disease, such as Peripheral Arterial Disease (PAD) or Coronary Arterial Disease (CAD), or gastritis from *H. pylori* infection, the risk of a bleed from NSAID use goes up a lot. The risk in high risk populations can be as high as 10% per 6 months use, so it can be grave. On the other hand, adding a Proton Pump Inhibitor may lower the risk, lowering inflammation can greatly lower the risk, and intelligent supplementation also lowers the risk. Finally, using a model where the ibuprofen is used on an "anti-PRN" basis – skipping doses as feasible – lowers the risk.

Here's a brand new article that demonstrates – ***at the lower dosage levels I'm advocating for ibuprofen*** – that the risk of a GI bleed is almost zip:
http://www.ema.europa.eu/ema/index.jsp?curl=pages/news_and_events/news/2015/04/news_detail_002306.jsp&mid=WC0b01ac058004d5c1

How to screen for *H. pylori*? First, as a clinician I take a history and if they have a history of a lot of stomach sensitivity, I have a great deal of clinical suspicion to start with.

H. pylori continues to be a much greater problem than appreciated. New research has dispelled myths and revealed a much more worrisome reality.

Over half the cases of *H. pylori* gastritis in adults *started in childhood.* Wow, what a surprise. Pediatric *H. pylori* is a very big problem – who knew?

It also appears that most *H. pylori* spread is not from flies – huh, another big surprise – but spread from contact (gastro-oral) with other...humans! Family members, friends – or the kid who projectile vomits in the lobby, inoculating everyone sharing the same air (droplets). Delightful thought, eh? If more people knew this, when a kid up-chucks in public, you'd see a lot more people high-tailing it for the door LOL.

H. pylori should not be left untreated, because not only does *H. pylori* gastritis greatly elevate the risk of GI bleeds from NSAIDs, it is also a very significant risk factor for developing stomach cancer...and stomach cancer will kill you deader than a door-nail if not caught very early – and it's almost never caught very early. So why leave *H. pylori* untreated?

The problem with screening tests is that they often have unacceptably high false negative rates – meaning sometimes the test says there is no *H. pylori* when there actually is.

The breath test for *H. pylori* is a total pain in the neck – and there are all these restrictions that invalidate the test, so I've given up on it...and the false negative rate is at least 10%, maybe 20%, so that makes it pretty much useless – why would you want to miss 1 out of 5 cases of gastritis that need treatment?

The only other practical alternative I know of is blood testing, and that's pretty good actually – test for IgM and IgG for *H. pylori* antibodies, and if there's a problem, you'll see it.

Yes, I know, there are a bunch of new, fancy *H. pylori* tests – I'll research them for the next revision of this paper. In the meantime, I'm sticking with my old-fashioned method, which I admit is imprecise and has too much of my grandmother's common sense for medical administrators:

Here's the algorithm I use for whether or not to treat for *H. pylori*:

If **IgM is elevated,** that means acute or active infection – treat.

If the **IgG is elevated** (past exposure) but the **IgM is not** – AND they haven't been recently treated – AND they ARE having or have recently HAD gastritis symptoms – THEN I also treat – there's no point in not treating; the risks of leaving the gastritis untreated are too high.
Here's the take-home point: *H. pylori* is perfectly capable of "hanging out" – not elevating IgM (acute infection) but elevating IgG (past infection) – but also perfectly capable of firing back into action at any time, including from NSAID use. So...this is a case where I would rather just treat than assume (ignorantly) that if IgM is not elevated there's nothing there. Textbook rules can really fool you, you have to think deeper than numbers. Corporate medicine hates this kind of thinking, because they want everything to be just numbers for their business model.

If IgG is elevated and IgM is not, AND they have ZERO history of recent gastritis, AND they apparently WERE successfully treated in the past (with cessation of gastritis symptoms), THEN they are probably safe to leave untreated.

Finally, if IgG is elevated, and they've never been treated – then treat. In this case, it doesn't matter whether IgM is elevated or not, and it doesn't matter if they have gastritis or not – if they've had past exposure and they were never treated, they must be treated in order to avoid greatly elevated risk of a GI bleed from NSAID use – as well as to lower their risk for stomach cancer – because how do you know there isn't a latent *H. pylori* infection "hanging out", "waiting for something to do?". You don't – and neither does the textbook. Yes, you can refer the patient to a gastroenterologist for endoscopy so that biopsy samples can be taken – but doesn't that seem like enormous time, trouble and expense (and some risk; endoscopies are invasive, even in the best of hands) when you might just as well treat anyway? Add to this the fact that the false negative rate even for endoscopic biopsies can be as high as 5% to 10%, and I see no reason for endoscopic biopsies unless there are other, more important reasons to do endoscopy.

Here is a great link that explains *H. pylori* testing:
http://www.nlm.nih.gov/medlineplus/ency/article/007501.htm
You'll note they don't agree with me on how to interpret blood testing, but I don't care – there's "by the book" medicine that inflicts a lot of suffering on patients while we chase some number of doubtful actual practical value, and there's practical clinical medicine where you demonstrate

concern for the patient. If I'm going to put someone on ibuprofen or naproxen for life, I want to make sure I eliminate *H. pylori* as a risk factor.

I'll tell you a cautionary tale. You can skip this, it's not relevant to pain. When I was a med student, one time I got assigned to an elderly doc who was...well, not all there. We got a park ranger with diarrhea. The poor guy was suffering. This doc ordered a zillion tests, both blood and stool, I mean crazy stuff, for even I as a med student could see what was wrong: the park ranger had picked up a case of *Giardia*. He had been building a little bridge across a beaver stream, had gotten a dose of the water which was infested with *Giardia* eggs from beavers, and now had raging diarrhea. I suggested treating the guy but the doc over-ruled me. Luckily for the ranger, when the ranger came back a week later for followup I had a different mentor. I asked the new mentor if he minded if I just went ahead and treated the poor guy, and the doc said "for God's sake, please do." So I did. And the ranger's diarrhea stopped, and he went back to work ten pounds lighter. And all those lab values we got? Not one of them was positive for anything, including – as I knew would happen – the stool exams.

Dosing NSAIDs

1. Dosing: for ibuprofen, 800 mg total once or twice a day.
2. Dosing: for naproxen, 500 mg total once or twice a day OR 1,000 mg once a day (I still prefer the BID (twice a day) schedule for flexibility; BID allows "anti-PRN").
3. Whether to dose regularly or on an as-needed basis (PRN) or on a "skip as feasible" basis - (anti-PRN):
    1. If a condition which irretrievably degenerates, Anklyosing Spondylitis being a perfect example, stick with regular dosing: intermittent dosing just lets the damage steadily increase at a quicker rate – so, ibuprofen 800 mg BID or naproxen 500 mg BID.
    2. I do not yet know which conditions are best treated with regular dosing and which would benefit from "anti-PRN" – I am working on it right now. I will include this information in the next revision. For now, I am comfortable with the notion that for osteoarthritis and trauma-related (ligamento-osseus enthesopathy), the "anti-PRN" is a neat idea and for everything else that tends to progress inexorably without the use of biologics (which I personally won't use), such as rheumatoid, MS, anklylosing spondylitis, etc, the "anti-PRN" idea might not be appropriate.
    3. If trauma or osteoarthritis related, however, my experience so far is that the patient does do better with what I call "anti-PRN" dosing – have a regular BID schedule, BUT the patient is definitely encouraged to skip doses if they don't actually need it. This greatly lowers risks and at the same time reserves the use of the NSAID for those times when the patient really needs some pain relief – and NSAID's seem to work better that way; the problem of "treatment escape" is minimized ("treatment escape" is not the same thing as "buildup of tolerance" before all the Pain Management guys start yelling at me). There is an important difference in attitude with "anti-PRN" dosing: if PRN, the patient is always asking themselves, "do I feel pain yet and should I take a dose" – but if it's a BID schedule with "anti-PRN", the patient asks themselves, "it's time for my dose, but do I really need it?".

Celocoxib (Celebrex)

This is the only practical selective COX-2 inhibitor available right now. It works great; the effect on pain is far better than any of the non-selective NSAID's (Ibuprofen, Naproxen, Lodine, etc).

Cost is high, but I only use it once or twice a week in a program, so it's about $2-and-change online for once a day (200 mg) or $5 for the day at twice a day. If used only once a week, that's only $20 a month for twice a day, not bad.

There's no doubt there are dangers – risk of GI bleeds, surprisingly, and heart attacks, are very significantly higher than ibuprofen or naproxen. However, for one or two days a week pain relief is very good, and at one or two days a week I think the risks are very moderate.

I can't prove this yet, there is no good comprehensive data – I'm very willing to work with any research funded institution that would like to test the idea – but sometimes in clinical medicine we have to make a choice: we do what we think is really right for the patient, or we do nothing that hasn't been nailed down for years and let the patient kind of...twist in the wind, frankly.

I do the former approach, and I explain what I'm doing, I explain that I'm doing what I *think* is best for them, and I let them decide how risk averse they are. I'm happy with that. A clear conscience is more important to me than worrying about lawsuits, it really is. I've never been sued (knock on wood), but I don't really care; doing what is right is what matters to me. For many patients, the reward of much superior pain relief once or twice a week may be more than worth the unknown risk. At the cost level of four to eight doses a month, the cash cost from online pharmacies is sustainable.

As I said, my patients do not have good insurance, the whole thing of their insurance paying for celocoxib (Celebrex) is irrelevant and I don't have time to fool with it. Yes, I'm sure there's some fancy program for the poor, but how am I going to employ yet another full-time staff member to chase all that paperwork?

Dosage: 200 mg once or twice a day as needed – again, have the patient choose two days a week they really want to be active and pain free, and then put them on 200 mg BID "anti-PRN" dosing – skip a dose if they don't need it. Yes, there is 100 mg dosing also, but I tend, as you may have noticed, to have a typically aggressive "doc" approach – if you're going to take it, take enough to get the job done. However, for some people, 100 mg BID on an "anti-PRN" basis might be fine, although I am not a fan of minimal dosing except with narcotics; as I said in other places, I prefer to dose up to what I know will get the job done. The problem with "creeping up" on the dose is the patient is at risk for two errors: falling too far behind on intervening and never quite getting full relief in that day, or over-compensating in an effort to catch up and ending up actually over-dosing.

Anti-Inflammatory Nutrition

Is an anti-inflammatory diet a cure-all? No. Does it help? Oh, yeah. Big time. It can even be a game-changer in rheumatoid arthritis or multiple sclerosis or cancer – **and for simpler pain syndromes, it can be *transformative*** - so one should never discount the potential of nutrition to truly change the patient's life.

The anti-inflammatory nutrition approach is actually simple – and should be used as only part of a whole program that includes NSAID's used properly, COX-2 inhibitors, perhaps narcotics when needed, prolotherapy, and so on, but this nutritional intervention **is** amazingly powerful.

The protocols give here rely on anti-inflammatory nutrition as the foundation – I won't treat a patient who won't get serious about this – life is too short; I can't spend my time shoveling the sand against the tide if a patient is not willing to do their part.

The fact is that animal flesh, all animal flesh, including fish, and including eggs, causes inflammation. Period. There is no argument about this. **In like manner, casein protein in milk also causes inflammation. Again, period.** So, all these Hollywood types with milk moustaches are doing the public a grave disservice. No adult should be drinking cow's milk. Frankly, no kids should be drinking cow's milk either, but I can't argue with millions of parents who are convinced of the wrong information. Casein is inflammatory, that's all there is to it, and inflammation is the cause of most of our diseases, and most of our pain.

And so are eggs. And now we have Kevin Bacon promoting eggs. Sigh. Truly great actor, complete dummy about nutrition. Doesn't he already have more money than he needs? Doesn't he already have validation as a great actor? Why would he do such a stupid thing?

Here's what I tell patients: "You will cause inflammation, and make whatever inflammatory process you already have going far worse if you eat the flesh of anything that flies in the air, swims in the sea, runs on the ground or burrows underneath, and that includes their milk and eggs." Period.

T. Colin Campbell, PhD, demonstrated in his China Study that farmers on almost-vegan diets, no dairy at all (no milk, cheese, etc), and a small amount of meat only once a week (a very little chicken or maybe pork on a Sunday, that's all), did not have our diseases. They lived into their 90's in very good condition, without cancer, heart disease, stroke or arthritis...or **pain.** Hmmmmmmmmm.

Why? They had hardly any inflammation caused by nutrition. Simple as that. Powerful as that.

Let me tell you a brief story. I am subject to severe upper and lower back pain, from carrying way too many canoes by myself on my shoulders over way too many long portages in the Quetico back when I got the wonderful opportunity to work as a wilderness guide for the Boy Scouts out of Ely, Minnesota. My back is seriously messed up and I pay for all that fun anytime I let inflammation get out of control. Someday I have to get prolotherapy for my own back, but it's the old story of the cobbler's shoes.

When I transitioned to a vegan diet, I was amazed at how much less pain I had – even with the damage to my spine. Then came the day when I gave in to temptation. I drove into a What-A-Burger joint – fabulous burgers - overcome by a craving for meat. It happens. Twenty minutes after I indulged in that glorious burger, I had to pull off the road and lie down on the ground, flat on my back, unable to move, barely able to breathe – I was so overcome by back pain.

What happened? The meat caused an inflammatory flare; my back pain went from pretty much almost zero to pretty much a screaming +1,000 in twenty minutes flat. It was excruciating, and what a lesson for me; it only took two more similar mistakes for me to get it *thoroughly* through my *thick skull* that I really *couldn't* indulge in meat cravings LOL.

You might say, well, I eat meat, and I don't get flares like that. Yes, you don't – you just have constant inflammation, more or less constant pain at a lower level, and your brain is used to it and dealing with it – sort of. **But the damage is occuring, nonetheless, the internal corrosion is going on anyway, and you're going to have a heart attack, stroke or cancer – pretty much guaranteed – and your chronic pain level is much higher than it needs to be, and the day *will* come when your pain shoots through the roof.**
Here is the best article I've ever seen on the subject:

http://www.lef.org/Protocols/Health-Concerns/Chronic-Inflammation/Page-01?utm_source=eNewsletter&utm_medium=email&utm_term=Articles&utm_content=Update&utm_campaign=2015Wk14

When I write up the White Paper on Anti-Inflammation Nutrition and Supplementation, I'll be reviewing this article in detail. The Life Extension Foundation is a fount of useful information, they're the *only* supplement company I trust information from, but I've also found over the years that I have to temper somewhat their information according to both practical considerations and what I've proven out in my own experiece. One simply cannot take a thousand pills a day and spend $10,000 a month. Well, I can't.

In any case, when I became vegan, my inflammation went so low that when I did indulge, the difference between almost zero inflammation and a sudden assault of inflammation was huge.

Now, everyone carries on about sugar, it's the new devil, but sugar is only inflammatory when there is already a ton of inflammation going on. Not that sugar is a good idea, it is not – but sugar is not the prime culprit. **I will say it again: flesh, dairy (casein) and eggs are the culprits.** And there's nothing you can do to change that. It is true, however, that meat or casein with sugar is really an inflammatory atomic bomb – ribs, slathered in barbecue sauce, that luscious combination of meat and sugar, have done in more than one person by sending their pain through the roof or actually precipitating a heart attack.

I remember a 20-year-old young woman who presented in a rural urgent care with an upset stomach half an hour after eating ribs. In this case, the patient was seen by an NP first, who was unsure what she was seeing. She came to me and asked me to take a look at the patient...and thank Goodness she did that.

I walked into the exam tent after noting the patient's rapid pulse rate on the chart, crossed to the patient, noted her weight and the sweat on her forehead and the wet spots on her T-shirt on her upper chest, arm pits and back (diaphoresis – that is, cold sweat in the absence of fever), felt her forehead to check for fever or heat-stroke (none), also noted her skin color (a bit on the gray side) and her breathing (very slightly labored, but not in grave respiratory distress...yet), listened to her heart and lungs very briefly with my stethoscope (pulse rapid but regular; lung fields clear) and asked her one question: "are you diabetic?" She said she was. I said, "Well, let's get you a little more comfortable", grabbed a pillow (back then we had pillows handy), laid her down, and then said, "I'll be right back". I went to the NP and said, "Your 20 year old diabetic overweight female patient with atypical chest-pain, diaphoresis, mild respiratory distress but clear lungs, rapid pulse but otherwise regular rhythm, is having a heart attack."

(I included all that information for the NP for teaching purposes. Yeah, I chose to leave the patient for a brief minute; I thought hearing me say all this to the NP might just kill her)

I continued: "I would suggest, in this order: oxygen, two regular aspirins chewed up with a sip of water, cardiac monitor, check rhythm and call me back in at once if abnormal, start an IV with saline - wide open for starters, check blood sugar, correct if needed, then sublingual nitro, give a push of morphine if the nitro doesn't answer, consider metoprolol so long as the patient is not bradycardic or hypotensive from the nitro and morphine or hypoglycemic or, God forbid, on a calcium channel blocker, get a 12 lead EKG fast, but don't let that get in the way of everything else first, and as soon as the EKG shows the heart attack, call the EMT's for transfer to the E.D. And...get all the nurses and techs on this all at once; everything needs to happen pretty much at the same time." (I didn't ask for cardiac labs because we couldn't get labs)

Did you notice how we now have two examples of mis-mapped pain? In the first example, the brain mis-mapped vagal pain from the pancreas to the chest. In the second, the brain mis-mapped vagal pain from the heart to the stomach. One must always interpret reports of pain in context; the map is not the territory; the word is not the thing.

In the case of back pain, as you'll see later, just because the pain seems located at the sacro-iliac, for example, does not mean it's sacro-iliac pain. In like manner, just because the pain seems located at the exit of nerve roots from the spinal canal near a ruptured disc does not mean the pain is being caused by the ruptured disc...hard as that might be to imagine. It all seems so logical, but the fact is that the brain does not map pain well from areas it doesn't often pay attention to. Your finger-tips? Yes, the mapping is perfect. Your butt? No, the mapping is pretty much like throwing a dart in the dark with a few beers onboard and after having been spun around; you're lucky if you even hit the same wall the dart board's on. Many a disc surgery has been done that missed the true cause of the pain; many are the back pain patients with steel rods in their spines who are not one bit better.

It does appear to be true that if you can get truly wild-caught – *not* fake farm-raised-but-wild-released-for-two-weeks-fake-wild-caught – salmon, and limit yourself to a small portion (4 to 6 ounces) – you can get away with fish once a week without causing much problem. Otherwise? Foggedaboutit. **Fish flesh is actually very highly inflammatory:** it is chock-full of trimethylamines, which are the direct precursor for trimethylamine N-oxide, AND fish is chock-full of trimethylamine N-oxide ***itself,*** which is the chemical that actually causes all the havoc. **TMAO.** The only reason truly wild-caught salmon is not so directly inflammatory is that all the omega-3 oils counter-act the trimethylamines and trimethylamine N-oxide....so it's a zero sum food, but it's not actually good for you. You're better off eating veggies and taking your fish oil caps. http://en.wikipedia.org/wiki/Trimethylamine_N-oxide#Health_issues

Vegetables and fruits – in the absence of meat and dairy - do not cause inflammation!!! Not at all!!! Never!!! So...eat what you want, but if you hammer meat, fish and dairy don't expect to avoid the corrosive effects of inflammation.

Another thing you should keep in mind: while a vegan diet is no sure protection against cancer, I can tell you your risks for cancer sky-rocket when you eat meat and dairy (casein). Inflammation is not only a serious risk factor for cancer, but eating meat and dairy causes the upregulation of an enzyme in the liver that actually transforms dietary and metabolic toxins into very carcinogenic poisons...in the vegan, the liver simply doesn't do that.

Also, and just as important, inflammation drives cancer...big time. There are three stages to cancer: initiation, progression, proliferation. We know some causes of initiation, but we don't know them all, not by a very long shot. However, cancer that is initiated doesn't cause disease **if it does not progress! And cancer doesn't usually cause death unless it proliferates (lungs, liver, brain, etc).**

Now, a century ago, some cancer patients did die of cachexia – which is a wasting away condition while the "mother" tumor grows to gargantuan proportions. Which is a ... long story and very important, but I'll leave it for the White Paper I will write on natural supportive strategies for the patient with cancer.

However, guess what causes cancer to progress – and progress at increasingly faster rates, until it is growing like *crazy?* Nutritional inflammation, that's what: flesh, dairy (casein), eggs. What continues to drive it, and ultimately helps cause it to proliferate...proliferate *like crazy?* Yeah, you got it: nutritional inflammation: flesh, dairy (casein) and eggs. Are there other factors

involved? Sure. But does removing nutritional inflammation help? **Yes.** I will write a White Paper on this subject; it's too complex to be included here, but I couldn't leave this subject without at least mentioning it, it's too important.

Finally, it's important to distinguish between casein and whey protein. Both are in dairy; only whey protein is actually good for you (and safe for cancer). If you get whey protein to use in shakes, etc, be sure to get pure whey protein, not one of those awful mixes in giant plastic bottles. Look out for bogus ingredients in whey powders like casein, "milk protein" (casein), aspartame, sucralose – none of which are good for you or any other beast on earth. The protein powders that are useful in an anti-inflammatory diet are whey protein **(pure),** pea protein, soy protein and hemp protein.

Final note: soy protein is perfectly good for you. Huge epidemiologic studies clearly demonstrate that people who live on a lot of soy in any form live a lot longer and have half the disease rate – including cancers - of meat eaters. So, eat your soy yogurt and don't worry about soy, and stop eating dairy-based yogurts – none of them are good for you and casein is inflammatory. Period. Yogurt or kefer does not magically transform casein into something benign; it's still a metabolic poison and the equivalent to pouring gasoline on your immune system. And it doesn't matter if the yogurt is probiotic or Greek or Hungarian or Bulgarian or Martian or Venusian – if it's dairy, it's casein, and if it's casein, it's inflammatory. Someone should invent a yogurt based on whey protein with zero casein – now, THAT would be a great product.

Anti-Inflammation Supplements

Yes, the following work. No, I do not have any commercial interests or connections.

Vitamin C, cheapest possible, Walmart, vitacost, no fancy stuff – not Ester-C or Airborne, either, for Heaven's sake, have them save their money for something that matters – 500 mg to 1,000 mg two or three times a day. Make it a religion. Too many reasons to count not to. I could write a book. Maybe I will. But Linus Pauling already did; there's no need for me to, and he was a ton smarter than I am – two Nobel Prizes, and he deserved a third? I think that counts. For your convenience: http://www.vitacost.com/vitacost-vitamin-c-1000-mg-250-capsules

Note: just plain old ascorbic acid in capsules. Not fancy C with bioflavenoids, etc, because there's no need, it costs more, and you don't want any C that has calcium. See below, Magnesium, for my warnings about taking calcium.

Vitamin D3, 5,000 iu gelcap, two once a day with a meal, any meal; Walmart has them for pennies. 10,000 iu per day (two caps). You can test blood levels if you want, but I stopped because I never, ever got a blood test that wasn't low if the patient wasn't taking D3, and I've never, ever gotten a blood test that was too high if the patient was taking 10,000 iu per day. 20,000 iu per day, yes, that can lead to blood levels a bit on the high side, but I've never seen 10,000 iu per day (two of the 5,000 iu gelcaps) do so. No, you can't get enough D3 by sun exposure and not taking showers, not unless you're going to move to central Africa, bake in the sun all day long for months and stink to high Heaven (hey, one bath a year is really all you need, so go for it).

Why D3? Many reasons (like vit C), but for this protocol, sufficient D3 vastly lowers the risk of osteoporosis whether or not glucocorticoids are used. Since using glucocorticoids is a very important strategy for treating pain without undue recourse to narcotics and thus risking addiction,

it makes sense to include it – especially since including it lowers risk of cancer and depression and undoubtedly other things, so why not?

Magnesium. Like D3, there are a million reasons to take magnesium, and no reasons not to. D3 and magnesium together build bone and help prevent bone loss from glucocorticoids. Magnesium deficiency can be deadly (see articles below). Magnesium also lowers cancer risk and helps lower inflammation overall, so what's not to like? Just do it, please. And don't take calcium. Calcium does not build your bones, it does calcify your arteries and raise your risk for heart attack and stroke. It was a dumb idea to start with decades ago, but like so many dumb ideas that get pig-piled by the supplement manufacturers, it takes forever to make it go away. It needs to go away. And stop drinking milk, too, the calcium from milk just calcifies your arteries, that's all it does – and heart attack or stroke will follow, sure as shooting.

http://www.lef.org//Magazine/2014/12/Magnesium-The-Missing-Link-To-A-Healthy-Heart/Page-01
http://www.lef.org//Magazine/2008/5/Magnesium-Widespread-Deficiency-With-Deadly-Consequences/Page-01
http://www.lef.org/Vitamins-Supplements/item01682/Magnesium-Citrate?q=magnesium

Quick little story: one time, I had a patient in the ED in a country hospital with a life-threatening arrhythmia. I tried every drug that was supposed to work: nothing helped; he was going to die. Then I tried IV magnesium. His potentially fatal rhythm immediately disappeared (but it wasn't an arrhythmia that magnesium was officially approved for). I admitted him to the hospital and kept him on the magnesium drip. The nurses, of course, were freaking – because what I was doing was not in their nursing handbook. The Internal Med doc who took over the case was a very cool guy from NJ who had seen it all, and I do mean seen it all. He kept the patient on the mag drip, even though we both agreed we were going to get some serious sh*t from the hospital. Sure enough, three days later the Chief of Medicine for the hospital ordered the mag drip stopped, to the nurses' great relief. The patient died within twelve hours, pretty violently. We never did figure out what the heck was wrong with the patient, but nowadays, once they die, we seldom ever do figure anything out. Isn't modern medicine fun?

Cytokine Suppress from www.lef.org, one to two caps twice a day. I take two caps twice a day, but that's a lot; I don't think one cap a day is sufficient, however. It is a very important supplement because it contains both EGCG and Mung Bean Coating Extract, which work together synergistically to down-regulate the HMGB-1 pathway. You can get it here (my patients get it through me at wholesale):
http://www.lef.org/Vitamins-Supplements/item01804/Cytokine-Suppress

Alternatives to Cytokine Suppress

These don't work as well, but are cheaper and might suit your lifestyle better; **what is important about Cytokine Suppress is it contains two ingredients that are synergistic against the HMGB-1 inflammatory pathway - an extremely important inflammatory pathway that is completely ignored by NSAID's or, for that matter, narcotics.**

A cup of green tea in the AM – but it needs to be made from a teaspoon of "matcha" or "macha" powder, tea-bags won't cut it.

Or any green tea extract that contains lots of EGCG (epigallocatechin gallate). Cytokine Suppress from LEF contains 300 mg of EGCG per capsule, and my experience is that at least two caps a day

are required, so that would be 600 mg a day of EGCG you would need, regardless of the source.

Then, to get the full effect, you'll need to consume mung beans. You can get mung beans at Lee-Lee's Oriental Grocery, a chain. Incredibly cheap, I buy the ones from Korea. I don't trust any food item from China (and neither do the Chinese, if they have a choice), so I guess it's Korea or nothing for now. Dunno how much you'll need to eat, but my guess is a small bowl of mung bean soup a day will do the trick: it is good to know that in traditional Chinese medicine, mung bean soup is revered for helping the invalid recover from serious illness. A word to the wise? It's hard to imagine over a billion Chinese over at least three thousand years being wrong about something so fundamental and universally known.

Fish oil caps. Since you won't be eating fish – not even that fakey-so-called-wild-caught-baloney-artificially-pinked-up-salmon (how easy it is to fool the public, and do you really think the restaurant salmon special is truly wild-caught?) – you'll need the caps. One or two twice a day: breakfast and supper. There is still a lot of debate about EPA vs DHA, my take for now is just take plenty of both and don't stress out about it. I no longer recommend getting fish oil in a bottle and dosing by the spoonful; nobody I know, including me, can keep that up – so caps are the practical way to go. For convenience, here are a number of choices from VitaCost; there are many more, and I don't yet know which I would specifically recommend over any others:

http://www.vitacost.com/vitacost-omega-3-super-dha-500

http://www.vitacost.com/vitacost-ultra-pure-omega-3-lemon-flavor-800-mg-120-softgels-3

http://www.vitacost.com/vitacost-mega-efa-omega-3-epa-dha-fish-oil-2100-mg-240-softgels-3

http://www.vitacost.com/vitacost-norwegian-salmon-oil-100-wild-caught-2200-mg-per-serving-240-softgels-4

http://www.vitacost.com/carlson-elite-omega-3-gems-fish-oil-professional-strength-bogo-lemon-1250-mg-90-softgels

Provinal. These are the omega-7 fatty acids, and it is the omega-7's that appear to complement the omega-3's in helping to block inflammation. For your convenience:
http://www.lef.org/Vitamins-Supplements/item01812/PROVINAL-Purified-Omega-7

Nanocurcumin. This is controversial, and I am of two minds.

On the one hand, I know from my own research that nanocurcumin hammers VEGF down. Why is this potentially really important? Growing tumors have to attract new blood vessels constantly in order to grow, because they destroy their previous blood supply as they grow. Tumors trick the body into giving them new blood vessels by putting out VEGF (vasculoendothelial growth factor). Nanocurcumin blocks this wonderfully, helping slow down tumor growth. Nanocurcumin does not, however, in my experience, interfere with wound healing, another process that includes the expression of VEGF; nanocurcumin, in my studies, appears to block only **abnormal** expression of VEGF (i.e. Cancer). You say you don't have any tumors? How do you know? A tumor less than a million cells cannot be imaged by CT or MRI or PET.

In curcumin's favor, the people of India have been eating gobs of it every day, two or three times a day, for...oh, at least three thousand years. They don't absorb much, but they absorb enough. The reason they absorb it is they fry it in oil at very high temps (part of curry) – far higher temps than

we customarily use in the U.S. Now, if you take turmeric, or the concentrated curcumin from turmeric, as a supplement, or as an ingredient in a curry or soup that hasn't had the heck fried out of it, you simply won't absorb enough to benefit. The absorption is pretty much zero.

So, what about liposomal curcumin – there are some products out there? Yes and no – do you probably absorb a lot more? Yes. But is liposomal anything a good idea? Probably not. Consider: what do they use to make liposomes? Lecithin; phosphatidylcholine. When phosphatidylcholine hits the gut bacteria, what gets made? TMAO. What happens then? Inflammation. Does all of that sound like a good idea to you? I hope not.

What other choices do we have? The only choice I know of right now for absorbing lots of curcumin is a nanocurcumin product online. Can I vouch for it completely? No. I don't know what the heck is in that little bottle, except that I'm sure it doesn't contain lecithin or phosphatidylcholine (it's not liposomal, it's nano) but I CAN tell you from experience that the curcumin (the nanocurcumin) in that solution does absorb – for sure. Absorbs quite well. So, this is what I use. But, again, I only use it because I just don't know yet of any good option – I cannot vouch for it's safety, you'll have to make up your own mind. I'm just giving you ideas and resources, you still have to decide for yourself, or follow your doctor's advice.
https://curcumin100.com/shop/curcumin/quantum-serum-1-month-supply-copy/\

So, is nanocurcumin anti-inflammatory? Yes, apparently so. There are zillions of articles demonstrating it's profound anti-inflammatory properties. Here is just one of them:
http://www.ncbi.nlm.nih.gov/pubmed/12676044

So, what's the problem? I'll just warn you that curcumin – well, nanocurcumin, at least – can cause the heartburn from Hell. It can also cause GI tract irritation – or, rather, probably more accurately, a big up-tick in GI motility. I use nanocurcumin with cancer patients, and it appears to be essential to the process of restoring well-being (remember VEGF), but I'll also tell you that if the patient has tumors involved with the small or large intestines, curcumin can definitely cause increased pain by causing the gut to work against the tumors. So, I take it in smaller doses several times a day with food, and I also take – for now, I know it's not a great idea – a proton pump inhibitor (omeprazole) – and I keep Tums handy LOL. Not a great situation, I'm not pretending it is – and you may not have a problem with it, I'm just warning you that if you chug a full dose in some tea in the morning without food, don't be surprised if twenty minutes later you're frantically searching the house for antacids because your stomach feels like you filled it kerosene and then swallowed a lit flare (think of that great scene from Water World where the Mariner (Costner) drops the lit flare into the crude-oil hold of the ship, and the poor prisoner Depth Gauge, when he sees the lit flare about to hit the crude, says "Oh, thank God").

Glucosamine: I know everyone says to take glucosamine, but my take is different: if you stop flesh and dairy and get serious about anti-inflammation, you may not need glucosamine. I'm no longer thrilled about glucosamine, because glucosamine is flesh – well, cartilage, but it's from the same animals that fly in the air, swim in the sea, run on land and burrow underneath – so I'd just as soon leave it out. If you need it, start at 1,500 mg per day for two months then go to 1,000 mg a day. I am not convinced at all that chondroiten adds anything. In any case, for your convenience:
http://www.vitacost.com/vitacost-triple-strength-glucosamine-sulfate-1500-mg-180-capsules

Sleep

Sleep agents in the treatment of pain

People in chronic pain often have tremendous difficulties sleeping. Poor sleep makes the pain far worse! Really calming down the pain can be accomplished by a **day / night approach:** reduce causes of inflammation in the day – and improve sleep during the night.

I've worked with a huge variety of drugs and supplements for sleep, and I can tell you that, from my experience, all the choices boil down to a very few things that actually help – and everything else is dangerous, harmful or ineffective – if good quality sleep is what you want.

The problem with sleep drugs such as the old benzodiazepines (temazepam (Restoril), clonazepam (Klonopin), lorazepam, alprazolam (Xanax)), or the older tri-cyclics (Amitryptiline), or anti-psychotics (promethazine (Phenergan), Compazine, Seraquel, etc), or anti-cholinergics (Benadryl, Sominex, Atarax, Vistaril) , or more extreme anti-noradrenergics (Trazadone) or the newer drugs (hypnotics) like Ambien or Lunesta, is that the deleterious hang-over effects on memory, cognition and psychological stability – as well as actual sleep quality – are significant, and the potential for serious, permanent harm very high – from movement disorders (tardive dyskinesia, loss of coordination (ataxia)) to brain shrinkage (early-onset senility), to car accidents. I usually don't use them, but, as you'll see, sometimes they are useful.

Sometimes these drugs are useful as "knockout" sleep drugs, especially in withdrawal – **used briefly** – promethazine (Phenergan) can be useful – but only very briefly and with caution. I would half-dose it (12.5 mg oral) only, but you must be aware that in some patients, especially if they are unknowingly inhibiting liver enzymes (grapefruit juice, cimetidine, etc), promethazine (Phenergan) can be dangerous, even fatal, in combination with narcotics. Only an experienced physician should even consider this. Trazadone will ensure sleep – but block dreaming – so it is useful only very briefly, with the caution that if the patient cannot sleep as late as needed in order to permit breakthrough dreaming to run its course, don't use it, not even briefly – and never long-term. And the same cautions apply: there is real risk of fatality if used improperly in the wrong patient. The benzodiazepines can, again, be used briefly – but beware; they destroy memory consolidation, and they are viciously addictive. Perhaps a tiny bit of Xanax at bedtime to fall asleep; perhaps a week of Restoril – but I'll caution you very seriously – if you start these drugs, you may pay the devil trying to convince your patients to stop them.

All docs who have taken care of elderly patients know well the little old lady who has been taking temazepam (Restoril) for a decade – and you don't stand a chance of stopping it (not that you necessarily need to, not at that late stage in life, but it's still a cautionary thing to know, and the temazepam (Restoril) is probably the reason she has no memory function at all).

There are no really great solutions for sleep yet. There are acceptable agents that are reasonably safe and don't carry the addictive potential or extreme risks of permanent harm of pretty much everything I listed in the paragraphs above.

Here's what I often use – but patients are quite individual, again, there is no one-size-fits-all remedy for sleep issues.

First, sleep hygiene must be addressed. Lights must be extinquished, little LED's on all sorts of devices covered with black tape or turned off. The TV must be turned off an hour before bedtime. No, it is not a good idea to fall asleep with the TV on! If you want noise, get sleep earphones and a sleep CD. Using your laptop, tablet or cell phone to cruise the internet or respond to email is

verboten, for an hour before sleep. Alcohol does not help, not even red wine. Have wine with supper, don't have it near bedtime. Eating at bedtime, especially sugary – or caffeinated (chocolate) deserts - such as ice cream or chocolate cake, must stop (that can be a toughie). Finally, one or two cups of coffe in the AM before noon are fine, and are even associated with increased longevity, but not a drop of caffeinated coffee after noon-time – or any energy drink or tea with caffeine – for caffeine is a severe sleep disruptor; you want all of it metabolized and gone well before bedtime.

Second, I do recommend melatonin universally. It is a brain anti-oxidant, it is a circadian cycle normalizer, and I see no reason not to use it. The only question is dose. For many people, less is more: the 300 mcg dose should be tried first. Some people do better with the higher doses: 3 mg, 5 mg, even 9 mg – but it's individual, and I still think that giving the lowest dose, 300 mcg, a good try first is best. However, melatonin is not a "knock-down" drug, and people who struggle to fall asleep often find melatonin, by itself, inadeaquate.

Third, I have come to rely on **clonidine.** It's not a perfect drug, but it does seem to be very helpful with sleep – not only helping achieve falling asleep, but preventing awakening later – and the side effects are usually minimal. The dose usually ends up at 0.2 mg for most people. You may wish to start as low as possible; start at 0.1 mg or even half that (0.05 mg) and work up from there, but you'll probably end up at 0.2 mg. Getting up into the 0.4 or 0.6 mg range on a chronic basis is necessary for a few patients here and there, but I don't go that high right from the beginning - except for the occasional "narcotic-free" night (see later). It is an anti-noradrenergic agent, but far milder and without all the incredible receptor interactions that the heavy-hitter Trazadone has.

Clonidine helps maintain sleep by blocking "fight or flight" influences on the brain. We are programmed to wake up if a saber tooth tiger enters the cave; in modern life, we no longer have these kinds of dangers – our modern disruptors of sleep maintenance are worries, anxieties, drama's, troubles of all sorts – and clonidine gives us blessed relief from these while asleep, along with a small blocking of pain influences as well – and even restless leg! What's not to like? It works. Now, it has to be used correctly – only at bedtime. Lowest effective dose. Once a day – at bedtime. Period. No exceptions. If used this way, it is quite safe and will not cause rebound hypertension (which many clinicians fear – but if you use it correctly, you don't have to worry about rebound hypertension, that won't happen). In fact, one nice Japanese study showed that clonidine at (only) bedtime blocked the morning hypertension spike that some suffer from, and that is possibly part of the cause of early morning strokes or heart attacks – without rebound, and without hypotension.
http://www.ncbi.nlm.nih.gov/pubmed/12658028

Fourth, L-theanine is very helpful, as part of a whole regimen, for falling asleep. It is non-toxic and non-addictive. I have used it in huge doses with cancer patients, and it was a blessed relief for them. It is a supplement, an amino acid of sorts – even at heroic doses for cancer patients, it caused no harm. It is cheap and useful.

It is essentially a stimulator of GABA production in the brain – GABA binds to "benzodiazepine" receptors and is anxiolytic, causing relaxation – but using L-theanine is very safe because it's almost impossible to overdose because the production of GABA in the brain, even when stimulated by L-theanine, is self-limited – your brain has two layers of built-in protection that prevent over-production. In addition, L-theanine blocks glutamate-receptor-mediated over-excitation of neurons, so it also protects against neurotoxicity. Good stuff, all around. Here's a nice forum discussion on the use of theanine:
http://www.reddit.com/r/Nootropics/comments/16gqr7/ltheanine_increases_gaba_but_by_how_much_alcohol/

Does it bother you that this isn't a scientific citation? It doesn't bother me – sometimes I find the most useful "real world" information in forum discussions just like this one.

I should note here that L-theanine is the only "fall asleep" supplement I have ever found that works reliably, night after night. It doesn't appear to have an addictive effect, such as a benzodiazepine would have, either. I have tried, and observed patients trying, everything else on the natural food store shelves: lemon balm, hops extract (yuck), passion flower, valerian, skullcap, chamomile, kava kava (which actually has extremely complex interactions with many brain receptors, and is tough on the liver, so I'm not a fan of kava kava, even though it's "natural"), and so on. Patients usually have great enthusiasm for these, and for various combinations of these, at the beginning...but I've never known one who was happy with any of these for restorative sleep over the long run.

Fifth, CBD is extremely useful – it is a "sleep normalizer". CBD stands for cannabidiol. CBD is a component of hemp – as well as some marijuana. Please note that hemp and marijuana are not the same thing: hemp does not contain any psychotropic agents – no THC. THC is what causes the pot "high" – and THC is not the same thing as CBD. CBD does not cause the pot "high".

Cannabidiol appears to have multiple advantages. It is a true, natural anti-cancer supplement; I always insist my cancer patients take it, it's far too powerful a tool to not use it. It also appears that it may have some anti-schizophrenia properties without being an anti-psychotic drug. This is a complex area, but the initial hints are most intriguing. The list goes on. As with any new natural agent that appears to have mutiple advantages, there is the tendency among marketers to start touting it as a miracle cure-all. It's not that, of course, but I must say it's the most promising new natural agent I can remember seeing.

I believe cannabidiol was first discovered due to a paradox: pot smokers didn't (used to) get lung cancer as much as they should – not by a long shot. Researchers found that pot smoke had the same tars in it as cigarette smoke, so they assumed that pot smokers would be at the same risk as cigarette smokers. Wrong. So, what the heck was in pot that prevented lung cancer? CBD.

Now, this has changed somewhat, because there are now so many hybrids, bred for "stoning effect", where the THC has been bred sky-high, and the CBD levels have been bred down to virtually zero. This is not a good thing!!! If you're going to smoke pot (I don't recommend it, you'll lose a lot of your memory and cognitive function – pot definitely makes you stupid over time), for Heaven's sake make sure you're smoking a strain that has lots of CBD in it.

But for you, who just wants CBD, you don't want pot anyway. **You want CBD from hemp oil, which has no THC.** It is hard to find affordable CBD, but prices are coming down, you can order it from here, $70 for a month's supply of thirty 25 mg capsules:
http://pluscbdoil.com/product/30-count-pluscbd-oil-capsules/
I include this link because it's so hard to find.
Or, I have set up so my patients can get it wholesale through me, for $50 per bottle of thirty 25 mg capsules plus $10 UPS ground shipping anywhere continental U.S., no matter how many bottles you order.
If you wish to take advantage of this, without being my patient, you can, the tiny bit of profit I make just gets passed on to my patients. You must order two or more bottles, at $50 per bottle, plus $10 shipping.
So, it works out like this: if you order two bottles from the link above, it will cost you $140 including shipping. If you order two bottles through me (well, through TIMC, LLC), it will cost you $110 including shipping UPS ground continental U.S.

If you order four bottles, you save more - $210 as opposed to $280.
If you wish to order, just email me how many bottles you'd like and your shipping address and I'll send you an invoice to remit by check, paypal or credit/debit card: aztimc@gmail.com

Narcotics for sleep and pain

Sixth, a vastly under-rated but extremely effective sleep drug is a narcotic. However, very low dose only, and not as an extension of all-day narcotics. It is true that there is a lot of pain-management literature that supports the use of all-day extended release narcotics with an additional dose at bedtime to carry through the night – but this is where, I think, we get into serious risks of causing addiction. I have seen too many pain-management "rejects" who were addicted, strung-out, in withdrawal hell and in terrible pain, all at the same time, who were maintained for years on extended release narcotics **day and night...until the honey-moon was over.**

There is virtually no clinical research on the use of narcotics for sleep, but over several thousand years of the use of them for sleep (and depression) nevertheless teaches some lessons. First, if dosed quite moderately, they do appear to confer blessed sleep. Second, they are best used in an either-or way: either during the day, or at night, but not both. And, take note: too high a dose will really wreck sleep quality or abolish sleep altogether – this is another situation **where using the lowest dose possible really matters.**

The ideal application is to use all the tools above, and add a touch of narcotic. This is a very low dose – just 1.25 mg or 2.5 mg an hour or two before bedtime. That's one-quarter or one-half a 5 mg oxycodone or hydrocodone tablet, with or without acetaminophen.

I always used to prescribe a quarter or half a 5 mg Vicodin or Percocet, but now that both of those have been reclassified as Schedule II drugs, there's no point – I now prescribe Oxycodone IR or Hydrocodone IR, without any acetaminophen. Since they're all Schedule II now, why use the acetaminophen? It just puts your liver at risk, and the acetaminiphen is useless for pain, so why bother with it?

However, acetaminophen or no, the reality is that most patients will insist on going to the full 5 mg dose, whichever you prescribe. It really is too much for good sleep, though, so I use a workaround: I have them take it four or five hours before bedtime. This way, they have something to look forward to in the evening, and a better reason to tolerate pain more during the day, and enough if the dose wears off before bedtime that they can sleep. Ideal scheduling would be take the dose at **4 or 5 pm** and go to bed at **9 pm,** or take it at **5 or 6 pm** and go to bed at **10 pm.** Not after 10 – every hour after ten o'clock that you're not asleep is a loss of an hour of rejuvenative sleep. **No, you can't fool Mother Nature – you are hard-wired to get your best quality sleep starting at 9 or at the latest 10 pm; anything later, and you're just cheating yourself.**

It might seem counter-intuitive to forbid daytime narcotic, but the patient who is willing to work through pain during the day in order to get that blessed reward in the evening will do very, very well...and you can sleep at night too, knowing you're not putting that patient at high risk for full-blown addiction.

Put that way, it seems almost evil, though, doesn't it? We have this crazy, puritanical bent in our national character that medicine should never be any fun. However, the wise clinician will do everything possible to ensure harmless bliss and pleasure: the patient will improve (vastly; the body loves bliss and pleasure), they will love you (as well they might) and – last but not least, not by a long shot – they'll actually follow your instructions!!! (compliance).

You'd be amazed, though, at what some patients do, without the slightest thought, regarding directions. One time, not apropos to this paper, but instructive nonetheless, I had an older gentleman who was sort of a woodsman in Appalachia. He had an infected elbow, and I drained it and put him on amoxicillin/clavulanate (Augmentin), 875, twice a day. I saw him a week later, and while he was improved, he was not as improved as I expected. Somehow, the conversation got onto his daily schedule of pills, and he told me this – with great pride, by the way: "Oh, I just takes 'em all in the morning, all at once." "What do you mean, 'em all?" I asked suspiciously. "Oh, all my pills – what you give me, and blood pressure, and diabetes, and arthritis, and all the rest – I just fill my hand up, it fills my whole hand, you know, Doc, (shows me a huge hand, covered in calluses) and gulp! They all go down together, and then I don't have to worry about remembering to take them during the day! They're already taken!"

However...does the use of narcotic, low dose, only at night, completely avoid the risk of addiction? No. There will be the very slow buildup of tolerance, leading to inevitable addiction. But – it's slow, and there's a long period of grace, and the addiction, if it appears, is low-level...and clonidine actually helps prevent the buildup of tolerance.

Another possibililty is the use of memantine (Namenda) or dextromethorphan added for sleep and prevention of buildup of tolerance.

However, the main strategy to avoid the buildup of tolerance – leading to addiction – is taking breaks – **OR working through a little bit of withdrawal.**

Patients on nightly narcotics will eventually succumb to a **low level of addiction;** this will manifest in most patients as a brief episode, during the afternoon, where they experience a sudden onset of sweating (cold sweat, but not excessive, and not overly long) with some anxiety or restlessness. If they can simply work through this – and physical work or activity is best – then they can continue their narcotic use for evening relief and blissful sleep.

It's a continuing process they get used to: evening relief and blissful sleep, alternating each day with a brief hour or so of low-level withdrawal in the afternoon. **Many can maintain this pretty much forever, so it's a very workable strategy.**

There will be patients who cannot bear the brief withdrawal symptoms each day. For them, the process is more difficult. You may have to enforce periodic breaks in order to "reset" them. Thus, one night narcotic-free a week, or three or four nights in a row narcotic-free once a month.

Will they suffer from insomnia on the narcotic-free nights? Maybe. So, you might decide to treat them for those nights only – just those nights, no more – with knockout drugs: promethazine (Phenergan), or Trazadone, or perhaps Restoril, lorazepam (Ativan) or alprazolam (Xanax). Ambien or Lunesta are weak hypnotics and not useful for this purpose. However, the above strategies are not risk-free – **there are real dangers, even fatal dangers!** - so, please see next below:

Another option, which I prefer, is simply increase the dose of clonidine for only those nights that are narcotic-free. Thus, if the patient is normally on 0.1 mg or 0.2 mg (the most common dose), you might bump it up to 0.4 mg for just the narcotic-free night.

Trying to alleviate the afternoon brief withdrawal symptoms by adding an additional narcotic dose is a **terrible idea,** and will only increase their addiction risk. Trying to alleviate the symptoms with a day-time dose of a short-acting benzodiazepine (i.e. Alprazolam (Xanax)) is another **terrible**

idea, although if the patient is really cooperative (compliant) you can use this strategy – but beware; benzo's are viciously addictive, and if you get talked into continuing a benzo for more than a very few days, the patient will get into trouble. Also critically important to keep in mind: respiratory depression is a real danger and a real cause of death in over-medicated patients, and....when you add a benzodiazepine to a narcotic, this combination causes the respiratory depression to get much more severe – you really can kill your patient by giving them a benzo on top of their narcotic!!! All in all, I don't find that trying to mask symptoms of withdrawal during the day is a good idea.

Having said that, however, a glucocorticoid is another option that can help a great deal; dexamethasone, especially, has been noted to moderate withdrawal symptoms – so, if the patient is on a rotating schedule with some nights narcotic-free, it is often best to schedule the narcotic-free nights before a day where a burst of dexamethasone is going to be used.

So, for the night and following day of a narcotic-free night, a great combo is to bump up the clonidine dose for the narcotic-free night and do a burst dose of dexamethasone early the next morning. And, if they didn't sleep that night, the dexamethasone will get them through the day fine. But this is only for special cases where you're going to use dexamethasone on a repeating basis; for everyone else, just the bump the clonidine.

If all of this seems awfully complicated, that's because the ***proper*** use of narcotics ***is*** complicated, and the use of dangerous drugs to counter-act the effects of the buildup of tolerance *is also complicated,* and that's the way it is. The simple solution of using narcotics night **and** day just leads to **disaster,** so in this case complicated is not only unavoidable it is beneficial.

Preventing tolerance: consider NDMA inhibitors & Clonidine

Are there drugs or supplements that prevent the buildup of tolerance? Yes, sort of. Perfect drugs for this are not yet available, but we do know that both anti-noradrenergic drugs and anti-NDMA drugs do help prevent tolerance – NDMA-antagonists include ketamine, memantine (Namenda) and dextromethorphan. Ketamine is simply not practical for chronic use – injectable only, far too short a half-life, and far too much psychological effect. Both memantine (Namenda) and dextromethorphan, however, might turn out to be very good options for chronic use. See here for a beautiful little study from Iran on combining clonidine and dextromethorphan:
http://www.ncbi.nlm.nih.gov/pubmed/15124977
This is one of the reasons I include clonidine for sleep (see above), and now I'm considering the use of memantine (Namenda) or dextromethorphan in addition.

But there's a lot more to consider, so these are just hints for now; please wait until I have the "Treating Addiction" White Paper ready, which will include "re-setting" the addicted patient so they can continue appropriate use of narcotics at a lower dosage level.

To summarize the combination I use most frequently for helping the pain patient obtain blessed sleep:

Melatonin at bedtime, start at 300 mcg, may go up to 3 mg, 5 mg or 9 mg if needed.

L-theanine at bedtime, dose range 400 mg to 1,000 mg. (200 mg is too low to bother with)

Clonidine at bedtime, 0.05 mg, 0.1 mg, 0.2 mg, exercise caution with going higher, although there

is the occasional patient who does benefit from 0.3 mg, 0.4 mg, 0.5 mg or even 0.6 mg – and you can bump it to 0.4 mg for narcotic-free nights as needed; never had a problem.

Strongly recommend: CBD, 25 mg two or three hours before bedtime. Cannabidiol; sleep normalizer; can get from either of these sources for $70 for thirty 25 mg pills, a month supply:
http://pluscbdoil.com/product/30-count-pluscbd-oil-capsules/
http://www.thecbdsource.com/Hemp-CBD-Capsules-s/107.htm
I include these links because they're so hard to find. There is a lot of gouging going on from the fancy products – but I don't see the need for the fancier products, like "CBD gold", I think all the cannibinoids in the "green gunk" hemp oil CBD are beneficial. You might find that squirting half the contents into an empty capsule, thus cutting the nightly dose to 12.5 mg might work for you, and cut the cost in half. Personally, I really like the whole 25 mg for sleep...and I even go to 50 mg at bedtime sometimes.
If you're a patient of mine, I can pass on to you wholesale savings, and then the price drops to $42 per bottle of thirty 25 mg caps (as of the date of this paper), with shipping (UPS ground, not much). I make no profit. I probably should. Shrug.

Hydrocodone 5 mg or Oxycodone 5 mg, A QUARTER OR HALF a tablet one to two hours *before* bedtime. It is important that the patient get pain relief in the evening before bedtime, so they get a reward to look forward to and a "winding down" period where they can relax, in anticipation of sleep.
If **the whole 5 mg,** which really is too much for good sleep, then dose **four or even five hours before bedtime.**

Sleep Disruptors

Before I leave this topic, I would just list well-known drug classes that are known sleep disruptors – if the above suggested combination isn't working well for sleep, there could be another drug or food that is interfering. Drugs that can interfere with good quality sleep (restorative sleep) include:

Alcohol (a glass of wine with dinner OK, but not any later)
Nicotine
Caffeine (coffee, tea, chocolate, energy drinks; ***after noon-time*****)**
Caffeine-containing drugs (Acetaminophen/Butalbital/Caffeine: Fioricet, Fiorinal, etc)
Glucocorticoids (if taken too late in day; if taken "upon awakening" only, usually OK)
Anti-Cholinergics (Benadryl, Sominex, Atarax, Vistaril – even though used for sleep)
Hypnotics (Ambien, Lunesta – even though used for sleep)
Benzodiazepines (Restoril, Ativan, Klonopin, Xanax – even though used for sleep)
Statins (Crestor, Zocor, Lipitor)
Ace inhibitors (if they cause coughing at night; Lisinopril, Captopril, benazepril, etc)
Tricyclics (Amitriptyline, amoxapine, clomipramine – even though used for sleep)
Anti-Psychotics (Risperdol, Haldol, Abilify – even though used for sleep)
Anti-Noradrenergics (too strong for chronic: Trazadone. OK for chronic use: clonidine)
SSRI anti-depressants (citalopram (Celexa), escitalopram (Lexapro), fluoxetine (Prozac)

Prolotherapy

The vast majority of patients with severe pain I've seen over the past decade were complelely or

largely treatable with prolotherapy, with great improvement, almost all of them at least 80% pain free within 60 days, at an overall success rate approaching very close to 100% - ***no matter their presenting diagnosis.***

There are two reasons for this. First, many of the presenting diagnoses were wrong. In fact, the patient might have had a ruptured disk but the real cause of pain was not the disc or a nerve root compression, but enthesopathy of the ligamento-osseus junctions in the relevant area surrounding the area of injury. Second, because prolotherapy is actually a stem cell therapy, there are many conditions that improve even though it doesn't seem at first that prolotherapy should be applicable.

For example, I have successfully used prolotherapy to dramatically improve rheumatoid arthritis in hands and fingers, and to really slow down or stop the process of irreversible damage. Why? Because stem cells *control their hormonal (cytokine) mileu.*

What is prolotherapy, how does it work? Briefly, by injecting a lidocaine(epi)/dextrose solution into the area of each damaged ligamento-osseus junction, a micro-inflammation area is created *on purpose,* and stem cells from all over the body (so called fibrocytes) migrate to the site of "intentional injury" and set up shop to repair collagen – they transform into *fibroblasts* and start laying down new collagen. *They also control their hormonal mileu, and the patient's pain changes very quickly from an unbearable quality to a very bearable less bothersome quality.*

Now, this change in the nature of the pain is a God-send to the patient for the two-month process of the prolotherapy, but this is not what eventually abolishes most of their pain – what gives the patient their permanent relief is they now have repaired ligaments. Once the ligaments are repaired – actually, the ligamanento-osseus junctions – the irritation of the tiny, exquisitely sensitive nerve fibers in the ligamento-osseus junctions stops, and then the pain syndrome that the patient has been suffering from disappears. If you wish to read some links about prolotherapy, please go here:
http://tomravinmd.com/index.php
http://tomravinmd.com/practice.php
http://tomravinmd.com/prolotherapy.php

Position of Ease

Humans have this strange reflex where we want to stretch *into* pain – which just makes the pain worse. Finding the "position of ease" is just the opposite – positioning yourself *away from* the pain. It's really as simple as that – best done lying down, one moves to different positions and relaxes until one finds the position where the pain is the least or even disappears. Then one relaxes into that position, with deep breathing, until one either falls asleep or rests easily or...the pain starts to come back. When the pain starts to come back, one then tries to find a new "position of ease", which is usually a little *farther* in the same direction of posture that relieved the pain before.

What is happening here? You are finding a position where the nerve fibers in the ligamento-osseus junction are no longer being stretched. This allows them to quiet down and stop firing. The more you can find this position, the more relief you'll get.

Does it fix the fundamental problem? No, but at least now you know how to find a position you can get some rest in, and that's not to be sneezed at. Sometimes, however, when you fall asleep in the "position of ease", you'll wake up remarkably better – because, in your sleep, you actually "fixed" some of the joint somatic dysfunction (in chiropractic terms, known as "subluxation"). It's not a permanent fix of the ligamentous structures, but it is a relief of previously abnormal strain on the

joint, and the relief can be dramatic.

Note we are not talking about a joint dislocation. That's a very gross dislocation of the joint that is a very different matter; no, this is a tiny misalignment of the joint, not seeable on an xray or MRI, where parts of the cartilage inside the joint actually kind of get "stuck" together. This "stickiness" of the cartilagenous surfaces causes a "bind" or "strain" across the joint – the joint is no longer moving freely – and this then translates into pain both at the cartilagenous surfaces that are "sticking" together and in the ligamento-osseus fibers of the ligaments surrounding the joint that are now taking a constant, abnormal strain. I am also not talking about great joints – humeral head in the shoulder socket, for example – I'm talking here about tiny joints, such as the facets between adjacent vertebrae in the spine or the joint between the end of the transverse process of a vertebra and the proximal rib.

Another note: when you receive Osteopathic Manipulation – or a chiropractic adjustment, for that matter – what is happening is these tiny misalignments in small joints, such as facets in the spinal column – are being released, so that the "stiction" between cartilage faces is relieved and the facet surfaces can move easily again. You'll feel a "pop" in some techniques – this is a little vacuum bubble that formed in the synovial fluid and then collapsed with a "pop" during manipulation when the "stiction" between cartilage surfaces was suddenly freed; it doesn't hurt a thing, and there is usually great relief instantly. However, Osteopathic Manipulation has many modalities, many subtleties, and there are many ways of restoring ease of motion without necessarily needing the "pop".

Position of ease applies only to musculoskeletal pain; it won't work for pain that is purely inflammatory in origin, such as cancer pain.

Self-Administered Muscle Energy Osteopathic Manipulation

A recent discovery I made that has me excited is teaching the patient to do their own muscle energy technique to temporarily relieve a particular cause of pain. I will include information on how to do this for the most common conditions in the next revision. For now, I am just noting that this can be done, and the patient can "reset" their painful "part" very successfully on their own. The relief lasts for a day or up to a several days – but since it only takes three minutes to do a "reset", and there is zero risk of harm, it's a great technique!!!

It works really well for low-back pain. OK, I will describe one of the interventions, but please keep in mind that without a drawing and maybe the help of your DO or chiropractor, you might not get this right.

If you have back pain, especially really low down (lumbar on sacrum), and especially if it's mostly one-sided, you can try this:

lay on your back. Bring your leg up on the same side as the pain, with your knee bent. Grab your thigh with both hands. Pull against your hands – try to push your foot away from you. Don't do it very hard; it isn't necessary to strain, and if you over-power your hands, it won't work anyway. Just for a few seconds. Then relax, and with your hands – with your hands, not your leg muscles – pull your leg toward your head, *taking up only what your hip joint will easily give you* – do not strain. Then, repeat. Then, repeat...and keep going until you've gotten your leg to come way up...but don't strain your hip. That is, don't overdo. When you think you've gone far enough – with practice, you'll "tune into" what is just enough – you slowly lower your leg, and then get up sideways, being

careful not to fall. You may find your low back pain is gone or greatly reduced. Legal: yeah, don't do this LOL. Only do it with the help of your doctor and with your doctor's permission. There, now my lawyer is happy...well, as happy as he ever gets, which, with me, isn't very happy.

12-Part Master Protocol

12-Part Master Protocol for pain treatment – this builds, from #1 to #12, in layers. ***Clinical correlation is a must – if you're a patient, you must work with your physician*****:**

1. **Consider special causes for the pain that might be easily curable.** See "Special Considerations" section below. **This is not trivial!!!** You must make sure you know what the pain really is, and if you ignore the special conditions, you will miss many causes of pain that are easily curable. (click here to go to Special Considerations)

2. **Give careful consideration to whether prolotherapy** needs to be included. It probably does...but, you may have to first get the pain under control and the addiction prevented or treated before you can make progress with fixing the real source of the pain. (click here to go to Prolotherapy)

3. **Insist on anti-inflammatory nutrition.** (click here to go to Anti-Inflammatory Nutrition)

   I have no desire to shovel sand against the tide; if the patient is too emotionally addicted to inflammatory foods, I refer them back to the Pain Management Specialist for the drugs-only approach, and wish them the best of luck. I do give them, however, three months to make the conversion; it's not easy – but it can be done; it only takes 30 days to change the taste buds and 30 more days to create a permanent habit and 30 more days to establish the commitment permanently. It's worth it, and you're dramatically lowering their risk of cancer, heart disease, stroke, obesity, diabetes (or greatly improving their diabetes), liver disease, kidney disease and uncontrollable hypertension all at the same time, as well as cutting out a lot of expense from the grocery budget. What's not to like?

4. **Improve sleep.** (click here to go to Sleep) The better the sleep, the better the pain control during the day. Just as importantly, the lower the risk of disease.

5. **Teach the "position of ease" for self-relief, and provide Osteopathic Manipulation.** (click here to go to Position of Ease)

6. **If appropriate, teach self-muscle-energy technique for self-relief** (click here to go to Self-Administered Muscle Energy Osteopathic Manipulation)

7. **Begin a comprehensive anti-inflammation supplement program.** (click here to go to Anti-Inflammation Supplements) In order of importance, although they really do form a synergistic whole, so please only "pick and choose" if financially you no choice but to select only a few:

   Vitamin C
   Vitamin D3
   Magnesium
   Cytokine Suppress (or green tea plus mung bean soup)
   Fish Oil caps
   Provinal
   Nanocurcumin
   perhaps Glucosamine

8. **Ibuprofen or naproxen.** (click here to go to NSAIDs – Ibuprofen, Naproxen; click here

to go to NSAID's and GI Bleeds; click here to go to Dosing NSAIDs

Either, it really doesn't matter – naproxen is a bit more effective, ibuprofen is a bit safer; I personally use ibuprofen.

1. Ibuprofen: 800 mg QD or BID, or BID PRN (or BID anti-PRN, but the pharmacist will not understand anti-PRN, so just tell the patient what to really do, and keep the Rx simple. Life is too short.

2. Naproxen: 500 mg QD or BID (and PRN or anti-PRN).

9. **Consider Substituting Celebrex for the NSAID one, two or three days a week.** (click here to go to Celocoxib (Celebrex))

Celebrex: 200 mg QD or BID (PRN or anti-PRN), one, two or three days of the week; no NSAID (ibuprofen, naproxen) on Celebrex days.

10. **Consider adding a narcotic on a minimum-use basis (oxycodone or hydrocodone, with or without acetaminophen).** (click here to go to **Narcotics for sleep and pain)**

My favorite strategy for patients, in order to avoid full-out addiction, is **one dose each evening only;** it is sustainable forever if the patient can work through the brief bit of mild withdrawal each afternoon.

Lowest dose: 1/4 or 1/2 of a 5 mg tab one or two hours before bedtime.

Practical for many: one 5 mg tab four to five hours before bedtime.

An additional fairly low-dose strategy is – either in *addition to* the once-an-evening strategy or *instead of* it – **one, two or three days a week on a narcotic** with or without (it depends!) the NSAID or Celebrex.

If you go to more than three days of narcotic a week, you *will* eventually addict the patient. The beauty of the three day a week strategy is that they only get up to the edge of addiction; if they can then work through the fourth day (a great day for Celebrex) and – especially if they still have their evening dose to look forward to on day four – ***they can sustain this schedule forever also.***

You can also split the narcotic days up instead of three in a row. This works for a few people – it all depends on how badly they react to each "off narcotic day" – if badly, then you want to minimize their "bad day" to once a week, otherwise most will eventually cheat on you.

Please note that real addiction is not the same as mild craving. As humans, we all wish we could have more on the days off, especially the first day off. That is not addiction. Addiction results in withdrawal symptoms: significant anxiety, bothersome to unbearable restlessness, cold sweats, nausea, vomiting, psychic pain, rapid pulse, high blood pressure, desperate craving with increasingly risky drug-seeking behavior.

For those whose pain is so severe they cannot – or will not (the distinction is important – it is the "will not" patients who become problems) tolerate "narcotic free" days, addiction

will happen.

If truly severe pain, or conditions such as cancer or other terminal illness, including advanced age, we accept the addiction. Try to minimize it, but we don't torture the patient by making it more important than the patient's pain. Methadone can always be resorted to if necessary.

If the pain is treatable, *treat.* This is especially relevant with prolotherapy (please see text), which can be life changing. However – and this is important – during the prolotherapy period you can't use the NSAID's or Celebrex nearly as much, so you may be committed to a temporary period of greatly increased narcotic use. One strategy I use when I need to use prolotherapy is to take them off NSAID and celocoxib (Celebrex) and not use a glucocorticoid (dexamethasone) three days prior, do the injections, put them on a solid week of narcotic, then put them back onto their regular schedule of NSAID and Celebrex, etc, for two or three weeks, then repeat the cycle – up to six or even eight times, although for many four of these cycles will get them to the 80% pain-free level.

All I can really say about the use of "round the clock" narcotics is that the patient will become completely addicted sooner or later. Period.

11. **Consider adding an anti-NDMA inhibitor** at night to the clonidine to slow down the addiction process. (click here to go to Preventing tolerance: consider NDMA inhibitors & Clonidine)

This is still investigational among the Pain Management specialists, but I think it can be considered with appropriate informed consent and careful explanation of the known risks and benefits. There are two current possibilities worth considering: **dextromethorphan or memantine (Namenda)** – in addition to **clonidine** for sleep, which also helps slow down addiction (being an anti-noradrenergic agent):

1. If **dextromethorphan,** 15 to 30 mg at bedtime with possibly a second 15 to 30 mg dose mid-sleep cycle. I believe you have to have this compounded; however, a compounding pharmacy can make up a 90 day supply, at two per night, for a very modest cost, under $30 a month.

2. If **memantine (Namenda),** 5 mg with supper to start. Can go to 10 mg with supper. Daytime dosing not recommended (by me) unless there are other reasons or there is urgency to prevent or treat addiction.

12. **Consider a burst – or a repeating schedule of bursts - of glucocorticoid (dexamethasone).** (click here to go to The Glucocorticoids; click here to go to **burst use of glucocorticoids (dexamethasone**)

This can be a one time only to "reset" the patient aggressively, or it can be on a repeating basis. It is an aggressive approach with significant risks, so, especially if it is contemplated on a repeating basis, the benefits vs risk must be considered carefully.

If a single burst, always dose "upon awakening" if you can, and you can go to 1 mg/kg – 72 mg (eighteen 4 mg tablets) – for that one dose. Range is from 4 mg to 72 mg, but if the dose is too low, the risk becomes much greater than the benefit.

One can consider other glucocorticoids, such as methylprednisolone injectable or a medrol dosepak or prednisine, but in my opinion dexamethasone is the drug of choice, especially for one-day bursts (please see text in discussion).

Special Considerations

Varicella

Sometimes pain is caused by the varicella virus. That's Chicken Pox the second time around, as shingles – or not as shingles, but still causing pain. There is the mistaken notion that varicella can only cause shingles the second time – not true.

There is the U.S.-chauvinistic idea that "shingles without the rash" – ***Herpetes sin Zoster,*** as it is called in Europe - does not exist. It does.

Either immune-suppression from many causes, or hyper-inflammation from many causes, can allow the varicella virus to cause pain. It's usually at one spinal level, but can be up to three spinal levels, and always on one side only – now THAT, the one-side-only rule, does appear to be true in my experience.

But the rash is not mandatory; I've seen plenty of patients with severe pain localized to one or a few spinal levels, on one side only, that had no rash – but responded completely to acyclovir or valacyclovir (Valtrex). Since there's nothing else these drugs work on but varicella – or a few other viruses in the same herpes family - I don't know what else the pain could be caused by except ***Herpetes sin Zoster*** – shingles without the rash. The Europeans don't have any problem with this diagnosis, it is irrational that there is a problem with this diagnosis in the U.S.

My clinical suspicion – and this is a diagnosis that requires substantial clinical suspicion; it won't put up a neon sign – is usually aroused not only by pain at one or a few spinal levels on only one side, but the complete lack of relief from osteopathic manipulation or prolotherapy or anything else, except some mild relief from glucocorticoids, but with worsening later from rebound.

Let me tell you the story of the first time I discovered this condition, although I'm sure I missed it for years prior to wising up. I had an Asian patient with unremitting pain in the left side of her neck at C4. Nothing I or anyone else did helped: osteopathic manipulation made it worse, chiropractic adjustments made it worse, prolotherapy injections made it worse, massage made it worse, hot packs made it worse, even glucocorticoids only made it a little better...and then it got worse later, presumably rebound. What to do? Xrays showed nothing; CT showed nothing; no real history of trauma to explain it. No rash, of course. Then I remembered an article from Europe I had read on the subject of ***Herpetes sin Zoster.*** Ah hah!! Maybe. **I put her on Acyclovir, 800 mg, five times a day, and three days later she came back, happy as a clam at high tide and singing like a lark at dawn. No pain. No stiffness. All gone. Neck supple. Wow. I had her finish the 10 days, then continue on 800 mg twice a day for life.** ***Her neck pain never returned.***

I know the textbook says 400 mg twice a day acyclovir for maintenance, but in my experience that's not enough – 800 mg acyclovir twice a day always works (unless the patient is for some reason hyperinflammatory or severely immunocompromised). 500 mg twice a day valacyclovir (Valtrex) would be the dose I would use for valacyclovir (Valtrex) – either half a 1,000 mg tablet twice a day or the 500 mg tablet size.

So, something to know, to always keep in mind. And a trial of Acyclovir or valacyclovir (Valtrex) is hardly likely to hurt (see below, however) – and when it abolishes the pain, it's a gift from Heaven for the patient, because this is a condition that *nothing else will help.*

I've also seen ***Herpetes sin Zoster*** a lot in American Indians. I think, perhaps, although it gets complicated, that the American Indians were not very immunocompetent to varicella when first encountered, brought by the whites. I have gone back and done some reading, and in fact there were many deaths among native tribes from Chicken Pox; it wasn't just Small Pox and Yellow Fever doing them in. In any case, I noticed, while working on a temp doc gig at a Navajo reservation, a lot of patients with back pain – back pain that was: localized to one or a few spinal levels, close to the spine, one side only, without a rash, and unrelieved by all measures – **but quickly and completely abolished with acyclovir.**

Finally, there is some concern about kidney damage with prolonged high-dose acyclovir. I think this is mostly in combination with nephrotoxic drugs such as cyclosporin during transplant or cancer treatment, but if the patient can be prescribed valacyclovir (Valtrex) instead, this concern is probably eliminated. This little study suggests that valacyclovir (Valtrex) is probably safer for the kidneys than acyclovir – although I hasten to add I've never had a patient develop renal abnormalities (increased creatinine) on acyclovir.
http://ndt.oxfordjournals.org/content/15/3/442.long

Fibromyalgia or Chronic Fatigue Syndrome.

Sorry, I don't believe this is a correctly-named condition. I usually find that what it really is, is ligament damage – enthesopathy of ligamento-osseus junctions, with tremendous pain and fatigue exacerbated by terrible sleep due to...the pain. A vicious cycle. Treatment? Prolotherapy plus treatment to restore good sleep. However, it does sometimes get more complicated: I always also test for viremia and spirochetal infections: Epstein-Barr, Cytomegalovirus, HIV, Hep C and B, Lyme's disease, ehrlichiosis (especially Oklahoma and surrounding areas), babesiosis, and so on. Usually the only one that pops, if any do, is EBV. Treatment? If just EBV, **intravenous vitamin C (Riordan Clinic protocol),** along with acyclovir or valacyclovir (Valtrex) - (yeah, I know, they're not supposed to help – they do), 2 to 4 grams of L-lysine divided daily, and arginine-rich food avoidance. If other, then tx more specific: if Lyme's or ehrlichiosis, IV ceftriaxone, with IVC's also. Put all those together, and I find Fibromyalgia/Chronic Fatigue Syndrome usually surrenders. And they don't need gabapentin (Neurontin) or aripiprazole (Abilify) once you get them on the road to recovery. Link for the Riordan Clinic (they have an Affiliate Program your doc can join), a truly wonderful place with great research integrity:
https://riordanclinic.org/what-we-do/high-dose-iv-vitamin-c/

Note: most naturopathic or integrative medicine physicians doing high dose intravenous vitamin C are using sodium ascorbate solution to make up the IV's. This is not, in my view correct, although it's better than nothing – far better. The correct method is to make up the solution using ascorbic acid, and then buffer it with sodium bicarbonate. Yes, I know, everything in medicine is supposed to be done to perfect-world standards, but that just denies care to the poor; Klenner and Cathcart provided wonderful care over entire life-times using a "DIY" approach, one really can make up IV's from technical powders – 99.9% pure ascorbic acid powder and 99.7% pure sodium bicarbonate powder...but not in the U.S. unless you can get and maintain a compounding license. Just FYI, if you want the details, I'm happy to send them to you.

Migraines

I cannot remember the last time I saw a patient who had a true migraine. I've seen a TON of patients who came to me with the **diagnosis** of migraines – but not one of them actually was having migraines. This has to be the worst garbage-can diagnosis on the face of the earth: all these patients who think they have migraines, and they...don't. What is the headache really, over 80% of the time in my experience? Suboccipital nerve spasms, that's what. Can I prove it? Yes, without a shadow of a doubt.

I block the suboccipital nerves low on the occiput (back of the head) with lidocaine/marcaine/a tiny bit of epinephrine (to shrink the blood vessels in the area so the lidocaine and marcaine don't "wash away"), and about three minutes after I do the blocks, the sun comes up. The patient's face clears from an expression of misery and abject suffering to an expression of complete and utter ***amazement*** – and then it turns to pure joy; their face ***glows*** with happiness. The pain is gone. The so-called migraine is *gone.*

Then I explain that what is really causing the suboccipital nerve spasming is some degree or form of cervical instability syndrome, and to permanently abolish their headaches, I need to treat them with prolotherapy for their cervical spine. Which they usually do. And their so-called migraines go away....forever.

Now, you say, well, how does that explain that sumatriptan (Imitrex) makes the headache better, and how about visual disturbances during a migraine, and pain deep in the temples (the pterygopalatine fossa)? I answer, well, smarty-pants, you're treating the tail of the donkey....which, in a patient who thinks they have a migraine, hurts a lot – the tail of the donkey is hurting the donkey a lot, but it's still the tail. In fact, all this headache pain is actually "outside to in", but medicine can be so ignorant, stuck in a stupid idea that was wrong to start with. The "business of medicine" causes these wrong ideas to be perpetuated for decades – early research found these engorged veins in the brain, and it seemed to originate from the pterygopalatine fossa, etc, etc, and then the "medical business suppliers" all pig-piled on with their drugs and injections, and now we have – once again, there are dozens of these messes in medicine – ensconsed in our profession this completely mistaken misunderstanding of what these headaches really are, with all this pig-pile of wrong drugs, wrong injections, wrong treatments – oh, there's hundreds of wrong treatments for migraines, because...as I hope you understand by now...I'm not sure there even ***is*** such a thing as a migraine!

The suboccipital nerve comes up over the outside of the skull on both sides – there's a left-sided one and a right-sided one - all the way to the forehead and even to the eyebrows, on both sides, and it also spreads down sideways to the temples, where fibers penetrate the pterygopalatine fossa (soft spot in the front part of your temples where you often rub yourself when you're feeling a headache). When the suboccipital nerve is firing, it can irritate the heck out of everything in the pterygopalatine fossa and right inside the brain from the posterior portal of the pterygopalatine fossa: the trigeminal nerve ganglion and all three branches – V.1, V.2 and V.3, the optic nerve, and the sympathetic fibers that invest upon the trigeminal and optic nerves traversing the fossa (yes, information traffic goes both ways in a nerve; it's not a one-way street).
http://www.wikidoc.org/index.php/File:Gray790.png
(not a great drawing, but at least it shows the branches that go all the way to the forehead – most drawings of the Greater Suboccipital nerve show only the part on the back of the head – on the occiput – which is vastly misleading. It is a *plexus, a grand plexus,* and it involves just about the entire head.

The result is a storm of effects, radiating pain outwardly via the trigeminals, radiating pain back into the brain via the trigeminals and sympathetics, radiating pain into the eyes via the sympathetics upon the optic nerves, causing visual disturbances via direct irritation of the optic nerves, and even transfer of the storm from the sympathetics to vagal pathways (parasympathetic) causing nausea and even vomiting, as well as abdominal pain...wow – and that's just off the top of my head, I'm sure there's a whole bunch more going on, such as the interactions between the sympathetics and the blood vessels in the brain (which gave rise to the whole engorgement of brain veins theory of migraines – still treating the tail, now the tip of the tail).

So, yeah, you can inject sumatriptan into the fossa and calm that storm down and get some relief, but if you instead stop the suboccipital spasming, you stop all of it – everything both inside and outside, including the storm in the fossa and the interactions between the sympathetics and parasympathetics. And sumatriptan is super dangerous! That drug has potentially devastating toxicities! Here's a link for this seemingly great, really informed article on migraines...and it's all wrong. Completely wrong. All this erudition about the donkey's tail, it's hilarious, really, I laughed myself silly. Is the data presented correct? Sure. But it's all inside-out, and that's backwards – it's outside-in, you ninnies. But, read for yourself, and now that you understand what a migraine really is, you'll see precisely what I mean: http://www.migrainesurvival.com/understand-migraine-pathophysiology-allodynia

Blocking the suboccipital nerve is safe, ridiculously safe – your main concern is to not accidentally inject a dural fold, so you don't inject into the cerebral spinal fluid – not a disaster, but the patient will not be happy with you for half an hour of severe dizziness – or into the nerve itself, which could leave them with a numb half of their scalp for six months until the nerve regenerates – but this is all just anatomy + technique, not a big deal. And, no, fluoroscopy is not necessary, although it would probably give you more profit by adding on the fluoroscopy charge to Medicare (ugh).

In some patients, simply repeated blocking of the suboccipitals will abolish the headaches for years. In most, however, prolotherapy on the cervical spine (and upper thoracics, often) really is necessary to stop the suboccipitals from repeatedly spasming. Osteopathic manipulation can also be useful, but prolotherapy is still required to fix the root problem: cervical instability (which, by the way, often includes the sternocleidomastoid tendinous attachment to the mastoid process, one side or both sides, depending, which is extra-cervical, but whatever caused the damage to the cervical spine often also damaged the sternocleidomastoid tendino-osseus junction and fibers in the adjacent sternocleidomastoid tendon).

Also, for this condition, it's not just the cervical spine, the attachments of the suboccipital muscles onto the inferior and superior nuchal ridges on the occiput are also important to treat with prolotherapy injections. Imagine little muscles and tiny tendons attaching to this huge bowling ball of a head, precariously balanced on two tiny fingers (the Atlas; the atlanto-occipital facets), and then imagine what happens to those suboccipital tendino-osseus junctions on the occiput while the skull is bouncing and rotating all over the place, (car accident, whip-lash, soccer, head-banging dancing, falling onto head, domestic abuse, carrying children for years in one arm or the other, or just plain aging...so many ways), this big, heavy bowling ball of a head doing it's best to fall right off the atlanto-occipital facets, with the suboccipital muscles desperately working to keep it from falling off...yeah, the tendino-osseus junctions invested upon the superior and inferior nuchal ridges of the occiput need to be included in the prolotherapy injections.

Finally, and very important, there are quite a few conditions that can mimic a migraine – and many of these are extremely serious, so it's important to catch them while you're evaluating whether your patient actually is having true migraines:

As mentioned above, there can be a variety of presentations of **frank shingles or *herpetes sin zoster*** (both from varicella virus) in the head that can mimic the pain of a migraine: zoster outbreaks in the (not comprehensive, just some examples):
the facial nerve
http://www.stritch.luc.edu/lumen/meded/grossanatomy/h_n/cn/cn1/cnb7c.htm
http://www.stritch.luc.edu/lumen/meded/grossanatomy/h_n/cn/cn1/cnb7.htm
chorda tympani
https://dnbhelp.files.wordpress.com/2011/10/herpes-zoster-oticus-ramsay-hunt-syndrome.jpeg
the optic nerve
http://www.meddean.luc.edu/lumen/MedEd/grossanatomy/h_n/cn/cn1/cn2.htm
any or all of the branches of the trigeminal nerve CN V;
http://www.meddean.luc.edu/lumen/MedEd/grossanatomy/h_n/cn/cn1/cn5.htm
v.1 ophthalmic
http://www.meddean.luc.edu/lumen/MedEd/grossanatomy/h_n/cn/cn1/cnb1.htm
v.2 maxillary
http://www.meddean.luc.edu/lumen/MedEd/grossanatomy/h_n/cn/cn1/cnb2.htm
v.3 mandibular
http://www.meddean.luc.edu/lumen/MedEd/grossanatomy/h_n/cn/cn1/cnb3.htm

– and while it's true these are all one-sided outbreaks, there is enough communication between sides of the head in sympathetic nervous ganglia that the pain can appear to be inside both sides – albeit one side worse than the other – and it's easy to mistake a zoster outbreak for a migraine...and, contrary to myth, zoster outbreaks can last – especially *herpetes sin zoster* – for years and years, and swing from worse to better and back – so it's not that hard for a patient – or their doctor – to mistakenly assume it's a migraine when it's not remotely related to a migraine.

Sinusitis can cause tremendous pain and can mimic a migraine.

Glaucoma can cause severe eye pain and mimic a migraine.

Temporal (giant cell) arteritis is an inflammation of the temporal artery that can mimic a migraine.

Any infection causing fever can cause a headache that mimics a migraine, including meningitis, viral or bacterial, chronic or acute. And, there are infections that can last a very long time and present as a migraine.

A Chiari Malformation, a genetic malformation of the brainstem can cause severe headaches often misinterpreted as migraines.

Hypertension or dissecting carotid aneurysm can either mimic migraine.

Of course, a brain tumor can mimic migraine, although this is probably the rarest mimic.

Nitrates and nitrites, such as the chemicals used to cure meats (just don't eat these, cured meats are truly toxic, stop poisoning yourself), can cause raging headaches that perfectly mimic migraines, due to their vasodilating actions on brain vessels.

Caffeine withdrawal is a well-known migraine mimic, and it gets worse – because, in some people, caffeine can temporarily relieve a headache, a vicious cycle can get going – headache, caffeine, caffeine withdrawal, worse headache, caffeine, caffeine withdrawal....

Lumbar puncture is another well-known cause of severe headache, although I'll admit I'm not sure how one would mistake it for a migraine...

Behcet's disease, also known as *Silk Road Disease,* because long ago many of the people involved in moving goods along the Silk Road were genetically predisposed to Behcet's disease, causes painful sores in the mouth – and sometimes the inflammation and pain can get up into the upper nasal passages and even our old friend, the pterygopalatine fossa, and into the brain as well, causing a ***meningoencephalitis*** – all or any of which can be misinterpreted as migraine.

Drug addictions that cause painful conditions

Cocaine
If you get a patient who has: arthritis of their fingers, itchiness of their fingers and wrists and forearms (always scratching, no obvious reason), constantly running nose (and always claiming seasonal allergies), very skinny body habitus, bouts of depression – think cocaine addiction. Not always, but...cocaine causes an early *hyperinflammatory condition* with resulting arthritis that almost looks like rheumatoid arthritis, it's so bad, along with itchiness of the fingers and wrists and forearms. Ask them – they might tell you the truth.

Methamphetamine
Get a patient who talks a mile a minute, who seems amazingly happy and bright and very high-energy BUT they don't actually *make complete sense,* very skinny, eyes shining, pulse fast, maybe irregular, fast breathing (although this might be subtle), unrelenting energy....yeah, think speed. Methamphetamine. (But also consider Bipolar Disorder – *never forget Bipolar mania as a possibility).* Just a side note, contrasting methamphetamine with cocaine: meth addicts don't usually have pain problems until late stage; methamphetamine does not cause the *early* hyperinflammation and arthritis that cocaine does. On the other hand, once methamphetamine addiction gets to a late stage, out of control itching and obsessive scratching that can draw blood – to the point of complaining about "bugs under the skin" – does present – but by then, the patient looks so much older-than-real-age and just...broken down - that there's no doubt about the condition. They also "pick" – sores on their face from picking are common. Finally, the **methamphetamine** addiction causes **brain inflammation,** and that leads to all sorts of mental health presentations.

Quick After-Word: There is a lot more to say, but I have to stop here for now. I will add a summary later, and a section on the use of nicotinamide ribonucleoside for neuropathic pain.

If you've made it to here, CONGRATULATIONS!

Wow, what a long-winded paper, huh?

Thanks so much for taking the time to read it, and I do hope you found some one thing in here that really helps you.

If you'd like to contact me, you can email me at: aztimc@gmail.com,

Very Best Regards, In Christ's Love, Dr Knouse