Trigeminal Neuralgia Association of Canada

September 2012 Newsletter

Support Group

TNAC INTRODUCES CANADA WIDE SUPPORT

No support group near you? Or you are not really a 'group' person even though you would like more information. Or maybe your TN just makes getting to / from a support group not a possibility. Well TNAC has exciting news.

TNAC will be hosting "WEBINARS" starting in 2013. What is a Webinar? A webinar is a seminar or workshop that is hosted on the internet. You register and as long as you have a computer, internet connection, and speaker (audio system on your computer) you can attend and participate.

We are starting with three webinar sessions. They are planned for January, March, and May and will feature Dr. Kaufmann (Winnipeg, MB), Dr. Hodaie (Toronto, ON), and Dr. Honey (Vancouver, BC).

Our first webinar, "An Overview of TN Management," by Dr. A. Kaufmann, will be presented on Saturday January 26, 2013. Final details for registration will be included in our December newsletter as well as on our website and facebook pages.

All webinars will be limited in registration and registration will be open to TNAC members first. If there is still room registration will open to non TNAC members after a pre-registration (members only) time period. TNAC members will be able to register for free. Non members will be able to register for a fee of $10.

Look for registration information and dates in our December newsletter and please let other with TN know about this exciting new venture by TNAC!

Eastern Ontario Support Group

Our group consists of people from Kingston through to Cornwall and north to the hills of Gatineau! We are a group of people who enjoy coming together to encourage and support each other along the journey of TN. We swap stories, laugh, and even cry at times. We share news on where we are at in our TN journeys and what paths we have travelled. Most of all we are always open and looking to welcome new members to our group. Our group is open to individuals with TN as well as their friends and family.

For more information and the time place of our next meeting please contact Jane at cmusicstudio@cogeco.ca or by calling 613.936.6977

Vancouver and Lower Mainland Support Group Update

Coordinator: Ann Hopkins

Vancouver & Lower Mainland Group
Meeting Time: 1.00 – 3.30 pm
G.F. Strong Rehab Centre.
Meeting: Saturday December 1st
Note new room: The Boardroom 109
4255 Laurel St.
(Laurel at W. 26th, one block east of Oak)
Friends, family members and supporters are very welcome.

It's a longish walk to the meeting room so if you need a wheelchair give me a call and I'll organize one. Or if you want to have a chat or have questions please make sure you call or email me.To get in touch: contact Ann Hopkins, email: annhopkins@dccnet.com,
phone: 1 604 741 0662
4945 Laurel Ave, Sechelt, BC VON 3A2

Lethbridge Support Group

Coordinator Marion Guzik
The Lethbridge Support Group meets every second Saturday of the month at 2:00 p.m. in Rm A, Lethbridge Senior Centre, 500 - 11th Street, S., Lethbridge, AB. Coordinator: Marion Guzik, past president / founder TNAC email mguzik@telus.net Phone: 403-327-7668

London Support Group

We have a new support group starting in London, ON
Contact Name: Elizabeth Galbraith **Tel:** 519.471.3439
energyworksnaturally@bell.net
www.tnsupportlondon.ca

PAYPAL

Have you been waiting for paypal to renew your membership? Well it was a lot more work then we ever imagined but … paypal is finally up and working!
To pay your membership by Paypal.

1. Log in to your paypal account (if you do not have one you will have to join paypal and create a personal paypal account of your own first)
2. Select "Send Money" from the tabs at the top of the page
3. Enter info@tnac.org in the "to" or email address box
4. Enter the amount below that and make sure it is set to Canadian funds (US is the default)
5. In the box below this select the "Personal" tab and the select "Other"
6. Next select "Continue"
7. In the next page you will see "Trigeminal Neuralgia Associtiaon Canada 2006 will receive" and the amount listed so you know that you have the correct organzation. TNAC is charged for each transaction. You have the option of whether to pay this fee or not. The choice is yours. Below this is a message that goes to us with your payment. Please change the subject to "Membership renewal" (or TNAC donation if it is a donation) and please put your name, email and full mailing address in the message area so we can be sure to credit you for your renewal and get a receipt to you.
8. Select "Send Money" at the bottom of the page and you are done!

Thank you for your patience as we have set up the Paypal system. It has been a lot more then we anticipated!

PAIN FREE AT LAST

By Joanne Laucius, Ottawa Citizen August 31, 2012

Every day, sometimes a few times a day, Shawnah Roy would suffer two to five minutes of gripping, unrelenting pain. The pain was so severe that if she was outside her home, she would have to sit down and wait it out, with tears springing from her eyes.

"I suffered for five years. It was like torture," she says. "When the pain started, I couldn't even touch my face. I was afraid to go out."

The medical explanation was a condition called severe trigeminal neuralgia, caused by a blood vessel "pinching" the cranial nerve that feeds sensation to the face.

Roy had two choices: pain medication or surgery. Opting for surgery meant numbing her face and leaving half of it paralyzed. "I would only be able to smile with half of my face," she says. Roy, a retired corporate accountant, had been living in British Columbia. When she moved to Cumberland to be close to family, she was treated by Dr. John Sinclair.

He and Dr. Shawn Malone suggested that she try the CyberKnife treatment. A few weeks later, she was on the table in the "bunker," the term used for the CyberKnife room at the Ottawa Hospital's General campus.

"I didn't feel a thing. It's no-pain surgery, " says the 84-year-old Roy, who held down a retail job until recently and still has plans to return to work. "When I got up off the table, they asked me if I could walk. I said 'Sure.' And then I walked out. My daughter was with me. She took me home. But I could have driven myself.

"When you have pain, all of your energy goes to that pain," says Roy."You can't think of anything else. I wouldn't want to go through that again. Life is beautiful when you don't have pain."

LivRelief Nerve Pain Relief Cream

Recently I learned of a new product for treating nerve pain. In fact one of the creator's worked on this to assist in addressing his wife's trigeminal pain. I am currently trying this cream. The active ingredient, capcaicin, is the same as that in zostrix (and the same strength as zostrix HP). Having used zostrix HP for years I figured it was worth a try. So far I can say that the 'heat' factor that turns many off zostrix is much reduced yet I do find similar results in terms of effectiveness, perhaps better results. Their other cream, for aches and pains (non nerve pain) works very well too. If you have never used capccicin note that research shows you need to use it 3 – 4 times a day for approx. 30 days before you know whether or not it is effective. Here is what they say about the nerve pain cream:

Pain – especially nerve pain – can have a profound impact on your life. Whether your nerve pain is caused by shingles, diabetes, pinched nerve, sciatica or any other condition, the pain can seem inescapable and transformative.

LivRelief Nerve Pain Relief Cream provides fast and consistent relief from the agony of nerve pain and

allows you to regain control over the life you wish to live. LivRelief Nerve Pain Relief Cream, is the first nerve pain cream to use *delivra™*, a transdermal delivery system that delivers natural actives deep under the skin's surface. Specifically, the active ingredients are shuttled directly to the target area, blocking the channels that cause nerve pain. You can have the strongest medication in a cream, but without a delivery system like *delivra* there is no telling if active ingredients are being delivered where they need to go. If you are one of the thousands of nerve pain sufferers, you know that gambling on relief is not a safe bet. You expect relief and LivRelief Nerve Pain Relief Cream delivers the active ingredients quickly and consistently right where you need it.

For more information go to:

www.livrelief.com

BONDIBAND

Another product review. This one mainly for the ladies! I can not take hair in my face or wind in my ears. Spring and fall are especially hard when chilly mornings can cause the cold to go down my ears and trigger intense pain. At the same time wearing a winter hat or headband is just a bit silly looking in September or May! I am always looking for something that works for me! Since my MVD I also can not take pressure near where the MVD incision was so headbands do not work and the survivor style of buff also does not work.

Enter Bondibands. I discovered these last weekend at the Montreal Oasis Rock and Roll Marathon Expo. Basically they are a lycra, breathable, wicking wide headband that you can wear low over your ears or just back off your ears. They are light so wearable all year long. They are very soft and did not seem to press on my MVD sensitive spot. So I took it out for a test drive. On Sunday, in 42kph winds with temperatures around 10C, I put it on and went out to run the Montreal half marathon. I was outside for hours in the cold wind but also hot and sweaty at the same time.

The result. I finally found a headband that keeps hair out of my face, wind out of my ears, and no pain or pressure from the headband despite wearing it for twelve hours! They are out of the US (of course) but not expensive so would ship to Canada under the duty charge mark. Anyway just wanted to share in case anyone else out there was looking for something to save their ears from cold in spring and fall! Check them out at http://www.bondiband.com/

Contacting TNAC

For information on membership or general information:
president@tnac.org
613.936.6977
TNAC, 1602 Walton Street
Cornwall, ON, K6H 1W2
For information on support groups:
support@tnac.org
For information on advocacy:
advocacy@tnac.org

Hurt Blocker

The next big pain drug may soothe sensory firestorms without side effects

By Rachel Ehrenberg

Among a small number of related families from northern Pakistan, some individuals never feel pain in any part of their bodies. Scientists studying six such children found that by the age of 4, they all had injuries to the lips or tongue from repeatedly biting themselves. Bruises, cuts and broken bones were common, though fractures were diagnosed only long after the fact, when weird, painless limping or the inability to use a limb called attention to the injury. Tests showed that the pain-free children perceived sensations of warm and cold, tickling and pressure. They could feel the prick of a needle, but it didn't hurt. Two had been scalded — painlessly — by hot liquids. And one boy who performed street theater by putting knives through his arms and walking on hot coals died after jumping off a roof on his 14th birthday.

Besides their inability to feel pain, the Pakistani individuals studied by the scientists had something else in common: mutations in a gene called *SCN9A*. That gene encodes the instructions for a protein that forms a passageway for letting sodium ions into nerve cells. Known as $Na_v1.7$, this particular ion channel sits on pain-sensing nerves; when a nerve is stimulated enough to warrant sending a signal to the brain, a flood of sodium ions rush into the cell.

Among the pain-free Pakistanis, various mutations in *SCN9A* altered the blueprints for $Na_v1.7$ in different ways, but with the same result: The channel didn't work. Muted nerve cells could no longer alert the brain when the body encountered something painful.

In other people, though, changes in the same gene make the channel work too well. Affected nerve cells distort or exaggerate their response, crying wolf when they encounter nonpainful stimuli or even when there is no stimulus at all. In January scientists reported seven $Na_v1.7$-related mutations in some people with unexplained cases of small fiber neuropathy, a condition that typically entails burning pain in the feet with intermittent stabbing, aches and sensations of electric shocks or pins and needles.

Small fiber neuropathy and congenital indifference to pain (the official name for the Pakistanis' pain-free condition) are just two of a handful of human pain disorders that have in the last decade been linked to malfunctioning $Na_v1.7$. Though some of these conditions are relatively rare, a growing body of research suggests that $Na_v1.7$ might play a part in more common persistent pain that follows nerve damage. Such pain can result from a bad burn or traumatic wound, or even accompany diseases such as diabetes.

As more new studies pile up, $Na_v1.7$ is beginning to look like a nerve channel of pharmaceutical company dreams. "Potentially, these channels are just the most amazing drug target," says Geoff Woods of the Cambridge Institute for Medical Research in England, who led the study of the pain-free Pakistani children.

Part of the excitement over $Na_v1.7$ stems from the fact that the channel may offer a solution to an ongoing problem in designing pain drugs: To avoid unwanted side effects, medications should act only on particular targets in particular places. Today's pain drugs aren't so choosy.

For example, lidocaine, an anesthetic that can be applied topically or injected, targets an entire class of sodium channels. It doesn't discriminate between channels on nerves related to pain and channels on other nerves; that's why affected areas feel totally numb. (Because it blocks sodium channels in the heart, the drug is sometimes used to treat irregular heart rhythms.)

Aspirin, ibuprofen and their kin inactivate enzymes that promote inflammation. But these enzymes also help protect the stomach from acid, assist in kidney function and help blood clot, so the drugs can increase the risk of ulcers, kidney failure and certain cardiovascular troubles. The chronic pain drug pregabalin, which is prescribed under the brand name Lyrica for some types of pain and seizures, hits widely distributed calcium ion channels. Because of the effects on channels in the brain, the drug can cause dizziness, drowsiness and other problems.

Then there are opiates, including Vicodin, OxyContin and morphine. These work on cellular machinery in the digestive tract, spinal cord and brain, so they can cause nausea, constipation, dizziness and breathing problems, as well as being highly addictive.

But $Na_v1.7$ is found predominantly in peripheral nerves at work in the outer territories of skin and muscle. So drugs that target this channel shouldn't make people groggy, put them at risk for heart problems or meddle with other organs such as the liver or kidneys. The fact that the pain-free Pakistani children were otherwise healthy suggests that a $Na_v1.7$ blocker wouldn't interfere with other body functions. (The only secondary effect of knocking out $Na_v1.7$ appears to be a reduced or lost sense of smell.)

Drugs aimed at $Na_v1.7$ might provide relief, with few side effects, to people with the kind of pain that persists when peripheral nerves malfunction. Such pain is like an alarm system gone haywire.

Typically pain is protective; it alerts you to impending or actual damage. Nociceptive pain (from the Latin *nocere*: to hurt or injure) delivers a red alert when you touch something dangerously sharp or hot. Nerve cells that sense this type of pain have a pretty high threshold, but once activated, the response is instantaneous: Your withdrawal reflex kicks in and you pull your hand away. Inflammatory pain, stimulated by immune system cells, occurs in response to injury. This pain warns you not to move a broken arm, giving the bone time to heal.

But when physical injury, metabolic disorders such as diabetes, autoimmune diseases or viral infections assault nerve cells, pain is no longer protective. Nerves can become trigger-happy, set off by something as mild as the touch of bedsheets. An estimated 20 million people in the United States experience this kind of pathological pain in their extremities.

"There are a lot of people suffering, and it is really terrible for patients," says Catharina Faber of Maastricht University Medical Center in the Netherlands.

When examining a patient with such pain, Faber and her colleagues first look for an underlying condition that might have attacked the patient's nerves. In many cases, there's no obvious cause.

Research now suggests that genetic changes that alter $Na_v1.7$ may be a culprit. The seven *SCN9A* mutations linked to small fiber neuropathy in a study earlier this year appeared to explain the peripheral pain of nearly 30 percent of study participants. This study — reported by Faber, Maastricht colleague Ingemar Merkies and other collaborators in the *Annals of Neurology* — and other recent studies suggest that much of how pain is experienced is inherited. Many scientists now believe that genetic changes altering $Na_v1.7$, as well as other channels, may be behind a lot of chronic, pathological and mysterious pain.

Stephen Waxman, who directs the Yale Center for Neuroscience and Regeneration Research and is a biomedical researcher with the U.S. Veterans Affairs health care system, has been investigating $Na_v1.7$ and its defects for more than a decade. Working with Faber, Merkies and others from around the world, researchers in Waxman's lab have identified more than two dozen mutations in *SCN9A.* One, reported in *Brain* in February, not only causes burning pain in the extremities, but may also influence development of the hands and feet. The many months of cell studies required to fully understand how each mutation influences $Na_v1.7$'s construction and operation have been carried out for about half.

The first definitive link between mutations in *SCN9A* and a human pain condition

came in 2004. Researchers led by Yong Yang of Peking University First Hospital in Beijing reported two mutations in a family with several members suffering from primary erythromelalgia, a condition wherein $Na_v1.7$ channels open too readily and the body's extremities become red, swollen and burn with pain.

Then in 2006, an international team linked mutations that alter $Na_v1.7$ to paroxysmal extreme pain disorder. Formerly known as familial rectal pain, this lifelong condition is characterized by attacks of excruciating pain in various parts of the body, including the rectum, genitalia, eyes, jaw and limbs (the name was changed in part because the pain is not confined to the rectum). That same year, the team led by Woods reported the mutations causing an inability to feel pain in the families from northern Pakistan; additional mutations with the same effect have since been discovered worldwide.

These genetic studies cemented $Na_v1.7$'s prominent role in human pain perception. They also offered an unusual treat: Rarely do geneticists find a tidy and direct link connecting changes in a single gene with what scientists call a loss of function and its counterpart, a gain of function. Often the picture is much more complicated, with many genes contributing to a trait or disease.

Yet *SCN9A* offered a textbook example: A single mutation in a single gene could cause a person to lose the ability to sense pain, while another mutation in that same gene could amp up that ability, making people feel pain even when they should not.

"The genetic demonstration was very clear. It was so clean. It's usually not that clean," says Simon Halegoua, director of the Center for Nervous System Disorders at Stony Brook University in New York. "That's when multiple drug companies jumped on it."

Halegoua saw $Na_v1.7$'s potential earlier. In 1997, he and colleagues published research describing a sodium channel found predominantly in the peripheral nervous system. Since it was the first such sodium channel, the researchers called it peripheral nerve type 1. Later, it became known as $Na_v1.7$ (Na for sodium and v for voltage-gated channel). Earlier studies in rats and human cells had hinted that some sodium channels favored certain body regions over others. Some did their stuff primarily in heart muscle, for example, and others acted mainly in the brain. But this was the first description of a sodium channel used mostly by peripheral nerves, at the front line of the body's interactions with the world.

"That was the breakthrough," says Halegoua. "It opened the door to an approach which would target those peripheral pain-sensing neurons specifically. It was a paradigm shift in the way of thinking about how to target pain. Instead of targeting it at the brain like opiates do, you would target it at the source — the first nerve that gets excited."

The efforts of nearly two decades of research on $Na_v1.7$ are now bearing fruit. London-based Convergence Pharmaceuticals has developed a compound that blocks the channel only when nerves are firing like crazy. Normal pain thresholds, such as those experienced when touching a hot stove, aren't altered by the $Na_v1.7$ blocker, says Simon Tate, Convergence's chief scientific officer. The company's compound is now in Phase II clinical testing — the stage that looks at dosage and effectiveness — for a painful condition of the lower back and limbs called lumbosacral radiculopathy. Convergence has also started a Phase II trial testing the same $Na_v1.7$ blocker as a treatment for trigeminal neuralgia, a chronic pain condition that entails recurring brief episodes of intense, stabbing facial pain.

In January the Canadian company Xenon Pharmaceuticals reported success with an $Na_v1.7$ blocker for treating erythromelalgia, often referred to as "man on fire syndrome." And Pfizer and its subsidiary Icagen are working with Waxman and Yale colleague Sulayman Dib-Hajj to investigate a blocker for treating the same disorder.

Some conditions under investigation — lumbosacral radiculopathy and **trigeminal neuralgia**, for example — aren't caused by inherited mutations, but appear to result from nerve damage. If an $Na_v1.7$ blocker works in such cases, the target may prove useful for treating long-term debilitating pain more broadly. Good candidates may include the 60 to 70 percent of people with diabetes who have nervous system damage that can cause daily pain. People with traumatic injuries for which the healing time is long, such as a severe burn, may also benefit. Studies by Waxman and colleagues have found that mice without working $Na_v1.7$ channels don't develop the hypersensitivity to heat that typically follows a burn injury.

Beyond offering new tricks for treating pain when it strikes, studying the channel may also help doctors understand their patients' personal pain profiles. Recent research led by Woods found that some versions of *SCN9A* in people without known pain disorders can confer increased sensitivity to pain. Such studies may help explain why one solider who has been shot through the arm experiences chronic post-trauma pain, while another soldier with the same wound might not. Or why some amputees have phantom limb pain and others don't, says Waxman.

"It's very exciting to us," he says. "If you go into a room full of people who all look normal, 30 percent may have a lower threshold for pain and an increased likelihood of developing pain after nerve injury."

As scientists continue to investigate the nuances of $Na_v1.7$, some are turning to its channel brethren as well. $Na_v1.8$ and

$Na_v1.9$, also sodium channels, are less understood than $Na_v1.7$. But scientists do know that these channels also play a role in generating pain, and therefore may be promising targets too, says Michael Costigan, a specialist in the genetics of chronic pain at Boston Children's Hospital. Changes in the behavior of $Na_v1.8$ have been implicated in inflammatory pain, for instance.

Other pain-related channels being studied include transient receptor potential, or TRP, channels, which play an important role in detecting nociceptive pain, warm and cold, and the fire of wasabi and chili peppers. Some TRP blockers are under investigation for treating migraines, postsurgical pain and pain from nerve damage. And Costigan and colleagues recently reported that people with a particular version of a potassium channel gene are more likely to develop chronic pain after an injury.

The sheer number of ion channels now known to play some role in pain and the overlap of those roles highlight how important pain is for safety and survival — a fact not lost on the researchers who are trying to beat it.

There's potential danger that comes with the promise of a superior pain drug. Total pain blockers with few side effects could be abused by athletes or others who want to ignore an injury, allowing them to do even more damage. Such drugs might also quiet warnings of a new and serious condition, such as an intestinal obstruction or a stroke. As with the Pakistani children, living pain-free might even result in severe trauma and early death.

"If we have a really effective block, it could be dangerous," Halegoua says. "We need pain.

Donations

TNAC survives on the donations of our members. Please consider donations to TNAC. Ways you can help include:

1. Instead of birthday or Christmas presents ask people to make a donation to TNAC in your name!
2. Remember TNAC in your will or make a donation to TNAC in memory of a friend.
3. Do you have a grandchild, niece or nephew needing to do some volunteer work for school? Ask them to consider a yard sale, car wash, or small bake sale with the proceeds going to TNAC

Your membership fees and donations help us to support TN research across Canada. Funds also go towards supporting people across Canada such as the webinar series we are hosting.

TNAC is 100% volunteer run. All board members and even the doctors for the webinars are giving their time to support people across Canada coping with TN. We thank you for your support so that we can help others!

Unraveling Trigeminal Neuralgia—In Depth Doctor's Interview

Robert Goodman, MD, PhD., Chairman, Department of Neurosurgery at St. Luke's-Roosevelt Hospital in New York, discusses a way to treat face pain.

We're here to talk about trigeminal neuralgia, I don't think a lot of people have heard of it.

Robert Goodman: It's a fairly rare condition. It's not unusual for people to have pain in their face or pain in their mouth and their teeth. But only a small percentage of people that have pain in their face actually have trigeminal neuralgia. Most people that have pain have their pain from other causes, not trigeminal neuralgia. Unfortunately it's often very difficult for them to find out that they have trigeminal neuralgia because almost everybody that has pain in their mouth has it from something wrong with their tooth or something else going on and not from this nerve problem. So they often see dentists or doctors that don't recognize that it's actually trigeminal neuralgia, that it's actually this nerve problem. I It's a pretty rare condition, but trigeminal neuralgia basically is a short circuit in the trigeminal nerve. The trigeminal nerve is a sensation nerve that just carries sensation from the face into the brain. If you lost that nerve, your face would be completely numb but you would still be able to move your face because the nerve that controls the muscles of the face is a separate nerve and that would still be working. The nerve has two kinds of sensation nerves in it, there are about a hundred and forty thousand nerve fibers in the trigeminal nerve and most of them actually are there to send normal messages, sensation messages, from the face and to the brain. I If something touches your face you would have a message sent into your brain so your brain would know something touched your face. It's not pain messages those are just regular sensation messages. And then there is a different kind of sensation nerve that travels in the trigeminal nerve that's in the same bundle these are nerves that are specialized only to send messages if something painful happens in your face. They're only supposed to send messages if you get poked by a needle, or cut, or burned, but they travel in the same bundle with these other nerves. So all the time you're having messages sent through the regular nerves and almost never getting messages sent through the pain nerves. And they're supposed to be insulated so they don't bother each other so that when one nerve is sending messages the other nerves don't even know about it. But the trigeminal nerve if it's damaged in a certain way can develop a problem with the insulation so then the nerves are bothering each other. So the normal nerves are sending their messages, electrical messages, and they irritate the pain nerves which are sort of trying to ignore those messages, but if they get bothered too much then the pain nerves will be triggered to go berserk for a short time, basically they have a burst of activity. So all of a sudden they'll send a

burst of messages. Of course the brain will think its something painful happening in the face when that happens. And it's triggered by regular stimulation of the face like if wind touches the face and those regular nerves are sending messages they can then trigger the pain nerves to go berserk. They usually won't go berserk for more than a few seconds, they might go berserk for thirty seconds or a minute but they get tired out and they have to rest. Usually, they have to rest for a while before they could then be ready to be irritated again. So basically what it is is these pain nerves are having a seizure. It's just like if you had a seizure in the upper part of the brain when one little area of the brain can go berserk and affect the rest of the brain and make people have a convulsion. This is a similar kind of thing but it's only happening in these pain nerves that are from the face. So when people get this burst of activity, the seizure, it's only making them feel like they have this horrible pain all of a sudden in their face. And it can be any part of the face, it might just be the jaw or the cheek or they might feel like it's in the lower teeth or the upper teeth for ninety percent of people it's in the either upper or lower teeth area. For ten percent of people it actually primarily affects the branch that goes to the eyes. So they may feel a sudden burst of pain in their eye and get even tearing or their eye might turn red during the time that they have this burst of activity. —I It has to be triggered by the regular nerves, in fact we know that if we totally got rid of the regular nerves and left the pain nerves intact (still functioning), you would never get a pain attack. Because they don't spontaneously have this burst of activity, it only happens if they're irritated by the regular nerves. But if you lost all of your regular nerves your face would feel funny like thick or wooden and that's not a great solution for people. And it turns out the reason we really know that this is a burst of activity like a seizure is because the only medicine that can stop these attacks is the same medicine that works to stop seizures. And in fact the medicine that stops what we call focal seizures, seizures that occur in one part of the brain is the medicine that works the best to stop trigeminal neuralgia attacks. It's Tegretol or its carbamazepine, the generic name. And there's another form of it that's very similar called Trileptal it's a different chemical form but it works the same way as Tegretol.

Is that always the first line of defense?

Robert Goodman: It is, well it should be. The neurologists typically are the doctors that would treat patients with trigeminal neuralgia and not all neurologists agree on what they think is the best first drug to use. I happen to think Tegretol or Trileptal is really the best. In fact I think it's the only medicine that should really be tried before thinking about the surgical options.

Damaging the insulation to the nerve how does that happen?

Robert Goodman: Why does that happen, that's a good question. Unfortunately in medicine we don't know the answer to why questions as well as we would like to. We have ideas and speculation and we know that in many cases it's caused by an artery that damages the nerve. And in fact in some cases people have an abnormal artery. One of the patients I operated on the other day happened to have compression by the basilar artery, which is a normal artery. Everyone has a basilar artery but it's usually very far away from the trigeminal nerve but in some people it actually gets elongated and can actually move off to the side and touch the nerve. And it's rare for that to happen but that's one of the ways you can get it. If it's a large artery and it's pressing up against the trigeminal nerve clearly it causes damage to the nerve and causes this condition to develop. But in most people it seems to occur because of a normal size, a smaller artery that is normally in the vicinity of the trigeminal nerve. But in some cases as it gets elongated over time, you know as we get older our arteries get a little longer. They also get firmer, they get harder. And so the arteries can get wedged up against the nerve and every time we have a heartbeat the arteries that are sitting in the fluid around the nerve and around the brain those arteries jump actually, they move significantly. And if it's wedged up against the nerve and with every heartbeat basically pounding the nerve it can cause damage to the nerve. I It probably has to be going on for maybe even years before it causes damage enough for this short circuit to develop the trigeminal neuralgia problem. So that's the way we think that probably ninety percent of people get this but there's a tiny percentage of people that have trigeminal neuralgia because of multiple sclerosis. They have a problem that's actually in the brain stem and the pons which is where the trigeminal nerve enters the brain. And if they have a problem with the insulation, that's what multiple sclerosis is; it affects the insulation around nerves. And so if it happens right in the wrong spot or the right spot in where the trigeminal nerve enters the brain, it can make people get a short circuit, get trigeminal neuralgia. It looks exactly the same as regular trigeminal neuralgia except that it's harder for us to get that under control with medications. It's usually more refractory than the standard kind of trigeminal neuralgia. But only three percent of people that have trigeminal neuralgia have it because of MS. One other thing I should tell you though, about one percent of people that have trigeminal neuralgia actually have a tumor that's pressing on the nerve. That's rare but if people that have trigeminal neuralgia if they get an MRI of their brain we know if they have MS we see that right away, we know if they have a tumor we can see that clearly. In fact we can even do an MRI in a very special way to see the blood vessels and whether the blood vessels are touching the nerve or pressing on the nerve. So we usually have a pretty good idea in each individual patient what is the cause of their trigeminal neuralgia, at least I think so. There's still some

controversy and some people are not convinced that the patients that don't have an abnormal artery touching the nerve or don't have a tumor and don't have MS, some neurologists that we understand why those people have trigeminal neuralgia.

How many people are helped enough that you don't have to do surgery with the medicine?

Robert Goodman: You know that's a difficult question because what I think has helped enough is different than what a lot of other people think has helped enough. I happen to think that the goal of the treatment should be for a patient to have no pain attacks at all, be able to eat normally, talk normally and not to be in fear that they're going to have a sudden attack of pain. And be able to function without side effects that are bothersome from their medication. And that is not achieved in probably not in the majority of patients. But a majority of patients seem to be satisfied with the treatment that they have. Usually that means living with pain here and there, sometimes maybe acting up quite a bit at certain times. Maybe acting up for a week or two at a time and then subsiding. Or they have their medication at a level that makes them feel a little groggy or having trouble concentrating. In order to avoid the pains they will keep themselves at a level of medication, sometimes taking two or three medicines together and they may not even realize how much the medication is affecting their functioning. Because they would only know if they were able to decrease the medicine and find out that they actually would be clear headed and more awake and functioning better. So I think that if you cannot take carbamazepine (Tegretol) or Trileptal (the most effective medications) if you can't take that one medication at a dose so when you take it every day you are able to function normally and have no pain, then I think you should be considering one of the two surgical procedures that I do because those surgical procedures have an excellent chance to eliminate the pain completely and a very low risk of causing morbidity or causing side effects.

Tell me about the surgeries.

Robert Goodman: Alright there are two and I usually go in this order which is the first one is not really a surgery. The first one is more appropriately called a procedure probably because it doesn't involve cutting open the patient. It involves beams of radiation that are focused on the nerve actually. We can deliver radiation just to the trigeminal nerve and nowhere else in a very precise way so that radiation is able to damage the nerve. What we do is actually kill a certain percentage of the nerve fibers. We don't know exactly what percentage we kill and it could be different in each patient but we give the same exact amount of radiation to each patient very precisely. And this has been done now almost twenty years and has actually replaced an old treatment that we used to do regularly (and still some people do) which is to use a needle. We used to do a procedure with a needle and I still do rarely where we put a needle through the cheek and it actually is able to go through one of the openings in the skull where one branch of the trigeminal nerve is exiting to come out of the skull and go into the face. We can actually put the needle up through that hole and get the tip of the needle up to where the three branches of the trigeminal nerve are coming together into one bundle. And we can use the needle to damage the nerve there with a variety of ways. One is heat or we can inject alcohol or we can even use a balloon to put pressure on the nerve and to kill part of the nerve and that procedure is very effective and it used to be used very commonly until we figured out that we could damage that same part of the nerve much more precisely and consistently and more safely if we used this focused radiation. So this machine that was actually developed or they actually hoped it was going to be used to make lesions in the brain to help people that had maybe Parkinson's or--.

So use the gamma knife?

Robert Goodman: Exactly, the gamma knife is one way to do it. The Gamma Knife delivers what is called stereotactic radiosurgery. But it can be done with the Gamma Knife. It can be done with a Cyber Knife. It can be done with linear accelerator machines that are designed to be able to deliver this radiation to a tiny target and outside of that target almost no significant radiation is delivered. And the trigeminal nerve is only a few millimeters in diameter in the area that we're aiming this radiation and around it is just fluid. We can deliver the radiation so that it only hits the nerve and the fluid right around it and doesn't go anywhere else and we can give the same exact dose every time. And since it's been done for a long time now people have tried some different doses. We have very good experience to know what to expect from that treatment and we know—I started off using a treatment of eighty gray it's called and after a few years I had two patients that developed a problem from it which is numbness. Because if we give too much radiation it can cause too much damage to the nerve and if it kills the nerve too much it makes people numb in half their face permanently. So what we want is to be able give an amount of radiation that kills a percentage of the nerves but preserves enough nerve fiber so that you still have basically normal sensation in the face. But if we reduce the percentage of nerves enough it can eliminate trigeminal neuralgia because you need a certain number of nerves to be able to have the attacks of pain. And so after that experience with two patients after four or five years I switched to a slightly lower dose, seventy five gray is what I've used since then. That's now eight years. And with that dose I have not had any patient that has developed complete numbness and still a high percentage of patients are very effectively treated by this. I it's terrific

it's like magic because the procedure is done in just a few hours; it's basically a painless procedure. We have to put a holder on a patient and get an MRI done and they have to be holding still for awhile but it basically is like a magic procedure because they go home right afterwards and the next day they're behaving normally. It takes a little while for the radiation to have its effect, usually it takes a few weeks and it actually takes months for it to have its full effect on the nerve. But the problem is that the thing that caused, the problem that caused the trigeminal neuralgia to develop in the first place is not changed by this procedure. And the nerve can still be affected over time, the residual nerve. So patients who have this procedure often get very good results initially, eighty percent of patients do very well for the first six months, a year or two years but over time they have a significant risk of having their neuralgia come back again and even five years later, ten years later. And then they might need either stronger medication or possibly even another procedure down the road. But it's a terrific procedure especially for people who are older and may not be expected to be around for ten or fifteen years or twenty years and many patients that have trigeminal neuralgia are over seventy. And so for patients that are older or have medical problems that would make it difficult for them to handle a surgery typically the radiation treatment would be the first choice, although both procedures are excellent choices for almost everybody. The real surgery, the microvascular decompression surgery is a whole different thing, it's a real surgery and the idea is to actually cure trigeminal neuralgia. It has an excellent chance of curing it, it has an excellent chance of eliminating the need to take the medication and it lasts much longer, it has a much better chance of curing the problem forever and not having to come back again in the future. And that means for the people that have a blood vessel that's touching the nerve or pressing on the nerve having a surgery which is done to move the blood vessel away from the nerve and to put a cushion so that it can't touch the nerve again. Basically that's all we do once we get to the nerve. Obviously, to do that we have to do a surgery with the patient under general anesthesia, asleep, in the operating room. It takes usually between two and three hours to do the surgery; a lot of the surgery is actually getting to the nerve. Which means having to get down to the bone behind the ear, we have to make a small opening in the bone behind the ear, it's about a one inch diameter opening and then having to open the sac that holds the fluid around the brain. And with the microscope we can work through the fluid space next to the brain, I barely touch the brain at all for this surgery because we can work in the fluid space and see the nerve very easily with a microscope. And once we get to the nerve and we see the blood vessels there, sometimes there are veins also that are affecting the nerve, that are touching the nerve. We have extra veins. We don't need all of our veins. And veins we can't really move safely. If we see a vein that's causing trouble we actually can sacrifice that vein. We can cauterize it and get rid of it safely. Arteries though we can't sacrifice (you need the arteries). Luckily usually arteries are very easy to move. And we can usually move the artery easily away from the nerve and then there's a space between the artery and the nerve and I can put a cushion in between that is a permanent sponge or Teflon (we can use what we call shredded Teflon felt) which is kind of like a fuzzy material, a soft material that we can put in that is permanent that would prevent the blood vessel, the artery from getting back to where the nerve is, to touching the nerve. And then obviously we have to close things up. Actually where the bone has been removed I put in artificial bone, a hard plastic that feels just like bone. And obviously there's muscle there that has to be closed and skin.

Is it relief immediately?

Robert Goodman: Yes. The patients after that surgery wake up, obviously go to the intensive care unit or the recovery room and the first night may be a little groggy. Many patients have a trigger point that they know will make them get a pain. That they know they have to avoid touching a certain place on their face because they'll get a pain if they touch that place. Very, very often as soon as the patient is awake enough, and sometimes within an hour of the surgery, I can have them touch that spot, and they discover that it doesn't cause the pain and that's usually an excellent sign that the problem is gone.

Is it always successful?

Robert Goodman: No it's not always successful and sometimes it takes a day or two for the nerve to settle down. So if they have pains the first day or two it's not surprising, it doesn't mean that it's not going to be successful. But for over ninety percent of people it does eliminate the pain problem and for those people it usually stays gone. They have a tiny chance that it can come back later on and for the people that it does come back in very often it responds to medication much more readily than it did in the first place. It's not as severe of a trigeminal neuralgia as it was in the beginning. And most of the patients that have this, we call it MVD surgery because it's hard to say microvascular decompression all the time. But people that have the MVD surgery typically are able to get off of their medication within weeks. I try to get people to go slowly getting off of that medication because it can be dangerous to get off too quickly but many patients just stop it on their own because they realize they don't need to be taking it to get rid of their pain anymore and for most patients, it's really a cure for them and they don't have to deal with the problem again the rest of their life.

What is the most severe case that you've seen?

Robert Goodman: I don't know about the most severe case. There are some patients, many patients, I can't say that it's just one but there are many patients that have pains that are almost continuous. They are miserable. In fact for many patients they cannot eat or swallow because they know every time they try to eat or swallow liquids they're going to have a terrible pain. And so for those patients it can be an emergency to cure them, to get rid of their pain. Sometimes we can do that by basically raising their medication to a very high level which we know will cause side effects but at least usually will get their pain under control. That's not a long term solution but it's usually a good temporary solution to be able to figure out which of these other treatments we're going to use, the surgeries.

I thought this was a really alarming fact that trigeminal neuralgia is one of the leading causes of suicide.

Robert Goodman: I'm not shocked. It's funny that you said that, I haven't seen that but over a hundred years ago brain surgery was extremely dangerous. When people had brain surgery they actually had almost a fifty percent chance of dying from the surgery. Certainly more than a twenty percent chance even with the best neurosurgeons. So people had to have a pretty bad problem in order to have a brain surgery back then and it was usually brain tumors or some kind of bad stroke or something. But one of the most common surgeries back then was for people with trigeminal neuralgia because they knew that if they lifted up the brain, actually the temporal lobe they could get to the nerve and cut it and it would make peoples face numb but it would completely eliminate their neuralgia. And people were—many patients with trigeminal neuralgia were willing to have that surgery even though they knew there was more than a twenty percent chance they were going die during the surgery rather than to have to keep living with the pain attacks.

That's amazing.

Robert Goodman: And the big problem that most people say, actually that have this, most of the time they're not having pain. The pains, for almost everybody that has trigeminal neuralgia, the pain attacks are only a very tiny percentage of the day of their time that they're awake, and it can happen at night too and wake people up. But they spend all the rest of their time worried about when the next attack is going to happen and many patients know that certain things they do are likely to make them get an attack. So they'll avoid doing things like avoid talking, avoid being outside, avoid having any wind touch their face, avoid eating, avoid swallowing because they're afraid they're going to get an attack if they do these things.

Abe and the two people upstairs I think they're both women, did they all have the surgery or the other procedure?

Robert Goodman: They all had the surgery, in fact if the radiosurgery procedure is done they don't stay overnight in the hospital. The radiation, the stereotactic radiosurgical treatment is an ambulatory outpatient procedure, people go home right afterwards. They're both great solutions and I, what I typically do is I explain to patients if they are a candidate for the MVD surgery, meaning that they have a blood vessel that we see on the MRI and they're in reasonable medical shape so they can handle the surgery with a low risk of having a complication then I present both of these options to every patient. And of course every patient asks me which do I think is the better treatment.

You go with the MVD.

Robert Goodman: No I actually don't, some surgeons do. Unfortunately, there are many neurosurgeons that take care of patients with trigeminal neuralgia that only do one of these treatments. They only do either the radiation or they do the MVD surgery but they don't have experience doing both. They don't offer both. And so if you see one of those doctors you can be pretty sure what they're going to recommend their treatment. And I really disagree with that approach because I believe both of these are extremely effective very safe successful treatments. I just think that the patient really should decide which of these two is better for that patient, for this person. I can help them because I have some idea of which I think is better for each individual patient but I don't rule out their choosing the other one and I try to give them as realistic an idea as possible of the difference between the two. And in general what I do is I say, if you're old you should have the stereotactic radiosurgery and if you're young you should have the MVD surgery. Of course every patient asks me what's old and what's young and I happen to think pretty much people that are over seventy five are old, I may change my mind in a few more years. But if you're over seventy five you're old and if you're under sixty five you're young, pretty much. There are some people that are in between sixty five and seventy five and for them I say, if you feel like you're under sixty five then you're young and if you feel like you're over seventy five you're old.

END OF INTERVIEW