Drenth: Genetic Research Puts EM in Spotlight

Editor’s Note: Joost P H Drenth, M.D., Ph.D., wrote this report for the members of TEA, who sponsored a $35,000 grant for his 2006 research. His laboratories are at Radboud University, Nijmegen, The Netherlands.

As most of you know to find a physician who knows something about EM is very hard. Most of the physicians out there have never heard of EM, no less how to diagnose it. The same holds for research into this rare disorder. More than once I have heard people asking me why bother with such a small disease that affects so few? Well, what I tell them is that no matter how “small” the disease is, we can learn a lot from it. Moreover, seeing patients with EM in such pain and without a drug that is able to treat you fueled my determination to take this on.

The EM Gene
We decided to take an in-depth look into the genes. We knew that in some of you EM runs in the family. That was a strong argument that a variation in one of the 30,000 human genes was to blame. We looked at different European and North American families and found the gene (Nav1.7, a sodium channel in nerves) that causes EM in these families.

Hunt for a second gene
We were funded by TEA to look into why variations in one gene explain EM in a lot of patients with inherited disease, but some families have EM and a normal Na,1.7 channel. We opened the hunt for other genes and were partially successful. Let’s compare our project to a map of the United States and finding a gene is like finding a city on the map. At first we had no clue where to look, but now, after the research TEA made possible, we not only obtained the map, but we also were able to zero in on one state—let’s say Ohio. But now that we are collaborating with Professor Waxman’s lab at Yale, we will even be able to take that final step and sort out whether the gene is in, let’s (Continued on page 2)

Dr. Cohen’s Revised Guidelines on Web

A revised version of what TEA members have dubbed the “treatment guidelines” is now available on TEA’s Web site. Jay Cohen, M.D., chairman of TEA’s Medical Advisory Committee, updated this comprehensive summary of the wide range of therapies used to treat EM, which he last revised in 2005.

Re-titled “Treatment of Erythromelalgia,” the article is intended as a reference for (Continued on page 2)
TEA members and cites findings from TEA’s 2003 survey. It is listed first in the “Treatment” group in TEA’s “Articles” section on the members-only side of the Web site www.erythromelalgia.org. (Also see Articles list starting on p. 9.)

“This version has more detailed drug information,” says Dr. Cohen, who was disabled by EM in the 1990s. He helped found TEA in 1999.

Included is a list of 89 references and the medical journal articles cited are footnoted throughout.

“Many treatments exist, but none is highly effective in a majority of people with EM,” he says. Treatment involves finding a doctor willing to help the person with EM try various therapies until one or a combination provides benefit.

As in 2005, Dr. Cohen suggests the “first line drug for treating EM” is Effexor (venlafaxine) because it can produce major benefit or complete remission.

Calcium antagonist therapies may help about 25 percent of people with EM, he says. Treatment involves finding a doctor willing to help the person with EM try various therapies until one or a combination provides benefit.

For the first time, his discussion of causes includes a new theory: EM is a dysfunction of the body’s hyperthermia mechanism—a hypothesis that warrants further study.

**FootSteps**

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Lidocaine Skin Patches Alleviate Pain of EM

By Jean Jeffery

Two papers about the treatment of EM with lidocaine patches have been written by Drs. Mark Davis and Paola Sandroni at the Mayo Clinic in Rochester, Minnesota. Both papers can be found in TEA Articles (Treatment section). The first paper describes treatment of a teenager with EM; the second paper reports on the treatment of 33 patients with severe EM.

The Lidocaine Patch

The “Lidoderm” 5% lidocaine patch is soft and pliable and has an adhesive layer containing the anesthetic lidocaine. The patch is applied to a painful area of intact skin and the lidocaine penetrates the skin to reach the damaged nerves, but does not enter the bloodstream to cause any adverse effects. Each patch measures 10x14 cm but can be cut into smaller pieces as needed. Three patches can be worn for up to 12 hours within any 24-hour period.

A 15-year-old girl severely disabled by EM was seen at the Mayo Clinic. At rest her feet were cool with a bluish discoloration, but after walking for only ten minutes her feet became red, hot and extremely painful. She had not been able to take part in any exercise, sport or physical education classes at school for one year. Several drugs including aspirin, gabapentin, nortriptyline, and mexiletine had failed to relieve her EM. Detailed nerve studies showed the presence of small-fiber neuropathy (damaged nerves) with lack of sweating in her lower legs and feet.

Teen’s Dramatic Response

A lidocaine patch was placed over the top of each foot where the girl’s pain from EM was the most severe. She experienced almost immediate relief from her pain and continued to be pain-free for the 12 hours she wore the patches. However her symptoms of redness and heat persisted.

Improvement was dramatic: within 2 weeks of wearing the lidocaine patches each day she was able to walk for one hour and take part in school sports again. The girl’s pain has been well controlled for 3 years and the lidocaine patch is the only treatment she uses for her EM.

17 Patients Improve

Lidocaine patches were used to treat 33 patients severely affected by EM. The group included 27 women and 6 men aged between 17 and 84 years; 25 of them had small fiber neuropathy. All of them had previously tried many treatments without success.

The patients placed the patches on their skin where the pain from EM was the most severe: 32 on their feet and one on her hands, and removed the patches 12 hours later.

Each person reported back on how effective the lidocaine patches were for their EM. 17 (52%) reported an improvement in symptoms of their EM. Four of the 17 recorded an improvement of 80-90%, 10 of 20-55%, and 3 of 5-15%.

Eleven of the 33 patients reported no benefit with lidocaine and 5 people stopped using the patch due to adverse effects: 4 experienced “claustrophobic sensations” on their feet, and one had blisters after applying the patch.

A few patients with the most severe and longest duration of EM found the patches helpful when used alongside or instead of some of their oral pain medication.

Overall, the Mayo team consider the lidocaine patch to be a safe and useful treatment for EM.

Note for UK members: the “Lidoderm” patch is not available in the UK. However a new product called the “Versatis” 5% lidocaine plaster was licensed in the UK in January 2007 and should be available on prescription.

EM Hits Mainstream Science Media

“The Pain Gate,” an article featuring EM appeared in the April/May issue of Scientific American Mind, a highly regarded brain science magazine. Written by author and journalist David Dobbs, the article uses the life experience of TEA member Pam Costa, Ph.D., to humanize the science of neuropathic pain.

While recounting Pam’s lifelong struggle with EM, Dobbs describes the role inherited EM has begun to play in pain research.

We learn that almost all chronic pain is “neuropathic pain.” And that Stephen Waxman, M.D., Ph.D., and his group of pain researchers at Yale had zeroed in on the mechanism within neurons that sparks pain signals that cause pain.

The researchers knew the gene (sodium channel Na1.7) involved in the process, and they even had the tools to manipulate and observe its behavior in a laboratory dish.

All they needed was a genetic mutation that would link Na1.7 to neuropathic pain.

That’s where inherited EM came in. Once they discovered the rare disease, TEA provided a large family—

Pam’s—willing to supply blood samples to Yale researchers for investigation.

And these samples revealed a mutation in just the gene that Waxman’s group had surmised—sodium channel Na1.7.

Read It Yourself

To read the whole fascinating story yourself, until mid-July, you’ll have to find a copy of the magazine itself. After July, the story will be posted on TEA’s Web site. (The magazine legally requires this delay.)

Notes from Yale

Phone Number Changes
People with inherited EM whose first symptoms appeared either in early childhood or in adulthood are still being sought as volunteers by Yale researchers.

If you fit that description and want to volunteer, call Tanya Fischer, M.D, at a new phone number—(203) 464-8729.

Yale Doctor Takes EM Referrals
People with non-inherited EM looking for a physician may now ask their doctors to refer them to neurologist Steven Novella, M.D., at Yale School of Medicine, in New Haven, CT., U.S.

Physicians may refer patients by contacting Dr. Novella at steven.novella@yale.edu.

Q and A By Gayla Kanaster
Send answers and new questions to Gayla, 2532 N. Fremont St., Tacoma, WA, USA 98406, or GaylaKanaster@aol.com

Q “What food and drinks, including caffeine, have an effect on EM?” (Submitted by Nicki Greer, Aurora, OH)

A Linda Reger, Walla Walla, WA, U.S.: “I don’t know if anyone has heard of tyramines, but my doctor and I both avoid them. She, because of migraines, and I do because they cause flare-ups. Spinach is my worse culprit. Also, alcohol, red wine and beer.”

Q “Does anyone else wake up with pain and redness in their feet and legs after sleeping a short time? (Submitted by Pam Costa, University Place, WA, U.S.)

Carol Kanter, Sparrowbush, NY, U.S.: 

A “Definitely! I sometimes get very sleepy around 3 p.m. I find that as soon as I begin to doze I have a hand and face flare. For some reason this doesn’t happen when I fall asleep at night.

A Stacy Wilensky, Freeport, NY, U.S.: "I first learned that I had EM when I started having burning attacks after about an hour or so of sleep. For me, the EM pain is at its worst during my sleeping hours or if something wakes me up suddenly. If I stand up, my feet are bright red and really sore as if some-
New Support Group Meets, Raises $933 for TEA

“It was a time of interpersonal connection and spiritual healing as everyone was able to share about the struggles we have all had,” said Jon Rue about the first Southern California support group meeting May 19.

“We were able to joke and have some fun, too. Everyone got along great,” he said.

Held at a restaurant, the dinner drew eight people with EM and a ninth with a peripheral neuropathy.

Group members also “passed the hat” and collected $933, which they donated to TEA.

“We also compared feet. It was really funny because everyone looked so distinguished and professional and then we all started comparing feet,” said Rue.

Rue recently founded the Southern California Intergroup following the lead of a Northern California group that had met once.

“It was my idea that if we made a commitment to meet at least once quarterly the group could remain a permanent thing. These intergroups are autonomous and not officially affiliated with TEA.”

A TEA member, Rue used TEA’s online directory to find other people with EM in California.

“We also now have a network and the opportunity to be of service to anyone new. We hope that our effort will inspire people in other cities to do the same,” Rue said.

Fundraiser Planned

In the planning stages is the 1st Annual EM/RSD “Cure for Pain” Hollywood Red Carpet Fundraiser Event, report Jon and Cher Rue.

The Rues are arranging an October event packed with exciting entertainment and fundraising activities like auctions.

They hope to raise money to help erythromelalgia and reflex sympathetic dystrophy.

They plan to donate 25 percent of the proceeds to TEA and another 25 percent to the Yale University Department of Neurology for EM research. The other 50 percent will go to RSD groups.

They invite TEA members, family and friends to join together to promote awareness of EM and RSD and help fund research for pain.

For more information about attending or submitting items for the silent auction, please e-mail Jon at jrueri@yahoocom or call 949-573-3180.

Southern California Support Group member Berta Summers demonstrates her scooter driving skills for other group members Doreen Irish (left) and Jon Rue, group founder. Another member Deb Hoffman (not pictured) owns the scooter. The fun followed the first dinner meeting of the newly formed support group.
Meg Lombardi writes: I am a 45-year-old female from Wallingford, CT. I led a very active life until I was first diagnosed with plantar fasciitis in both feet in June of 2004. I had physical therapy, feet taped and then two injections of cortisone at the same time in both feet.

Right after having the cortisone injections, I stood up and felt a strange “squishy” feeling under my feet; I compare it to a tire being deflated. This strange squishy feeling was my fat pads atrophying, due to the cortisone injections. I was later diagnosed with “fat pad atrophy.”

Immediately after my fat pads were destroyed, I started to experience unbearable burning pain. I could no longer stand in one place for any length of time, even sitting with legs down. I would experience severe pain, with the only relief being when I would elevate my legs under a ceiling fan.

One night while kneeling over giving my daughter a bath, I was in severe pain and looked at the soles of my feet. They were the brightest red I had ever seen. I knew something was drastically wrong with me. Then came the hard part—trying to find a diagnosis.

I was as relentless as my pain was in searching for an answer. I luckily came upon TEA’s website and saw some of the pictures of feet with symptoms. I said out loud to my husband, “Look, those are my feet!”

Immediately after researching, I booked a flight for the Mayo Clinic in Rochester, Minnesota. After a grueling week of a battery of tests back to back, which included peripheral vascular testing, neurophysiology autonomic testing, electromyography, blood work, and neurophysiology sweat tests, I was diagnosed by Dr. Mark Davis with EM.

It has been very difficult to accept my diagnosis. I have a beautiful 4-year-old daughter. She was just two years old then and I could not believe that my dreams of doing all the things a “normal” mother could do with her child, I could no longer do. The hardest thing in coping is how I think it affects my daughter and husband, and how it has changed their lives as well. I used to work as an administrative assistant, which I can no longer do because of my daily debilitating pain.

John Langford writes: I am 77 years old and a widower, my wife having died last year. In my first few years of life I had many illnesses and was in and out of the hospital, ... An abscess I developed in my ear left me deaf in that ear. A number of infections caused partial deafness in the other one. Fortunately hearing aids became available. ...

In my early 40s gout began to plague me in my heels, big toes and knees. This was soon followed by Raynaud’s phenomenon in my hands and feet, which I still have. Then, I was diagnosed with ME (myalgia encephalomyelitis) by two private doctors associated with Action for ME in the UK. However, doctors in the UK National Health Service did not agree with that diagnosis or that I had anything medically wrong. According to them, I was “a very fit man.”

In my early 50s I began to have severe itching in the soles of my feet. No amount of scratching would help for it was deep inside. What did help was immersing my feet in cold water. The itching came and went, mainly in the summer. Leaving off socks and walking on cold surfaces also helped. Doctors could do nothing except give me
The itching later became a burning sensation, which worsened over time. This affected my feet and legs and to some extent, my hands, arms, shoulders and neck. ... Still, nothing was done for me. Later, I insisted on some kind of answer and he (my G.P.) diagnosed diabetic neuropathy. He apparently had tested me for diabetes, but still offered no help.

I continued seeing different doctors until one finally recognized my symptoms as EM and referred me to a rheumatologist, who started me on gabapentin. He said the EM was related to the Raynaud’s. ... He also said that he was limited in treatment options and further medication would cause side affects and that I should just try to cope as well as possible.

One of my favorite pastimes is reading, mostly spiritual and biographical books. I also am interested in books on alternative and complementary medicine, which is understandable with my having so many illnesses for which there are no cures and little in the way of palliatives. I also am involved with the deaf and hard-of-hearing community. I used to draw and colour faces and street scenes ... while my wife was very good at flowers. She exhibited locally and went to college for art classes. We spent many an afternoon drawing and colouring.

My immediate family is spread over a large area, so we don’t visit often. Our usual contact is by letter—no phone or Internet. I live alone and eat out a lot, not being a particularly good cook.

After my erythromelalgia diagnosis, I joined TEA, hoping for some help in this worsening condition.

(Continued from page 6)

William Blaha writes: In 1992 I wore boots all day because I was working in the woods for the U.S. Forest Service. When I came home, I had to cool my feet in cool water. I also noticed that the soles of my feet felt swollen when I walked on a hard surface.

That fall the symptoms came on with a vengeance. I couldn’t sleep. As soon as I got horizontal, my feet became swollen, bright red and so hot I could not believe it.

I made an appointment with a podiatrist and he informed me that I had a rare condition called erythromelalgia. He put me on aspirin therapy, which did not help. I spent the next 10 years going to different doctors and trying drugs such as propranolol, amitriptyline, nortriptyline, and others that I have forgotten. Most doctors had no clue as to what my condition was—I had to tell them.

In 2002 I heard of a treatment called Bariatric Therapy, which is used to treat wounds on the feet of diabetics. I called a chiropractor in Chico, CA who administers the treatment, but he didn’t think it would help my condition. However, he said he had a paper from a doctor in Japan who had treated two ladies with EM with cyproheptadine, with very good results.

I immediately called my G.P., who prescribed it for me. Although it hasn’t cured my condition, I would say it is 75 percent better.

To date, I am unable to wear shoes, only socks and sandals.

I have gotten a lot of good information from TEA.
Complete Citations Added To TEA Articles

Thanks to TEA member and scientist Jean Jeffery, TEA’s collection of medical journal articles now are “cited” properly.

TEA has a Web-based library of articles about EM that have been published in medical journals by physician scientists.

Every article now is listed with a full citation, which means its listing includes name, authors, date of publication, name of journal, page numbers, and more.

Jeffery, who lives in Nottingham, UK, also wrote brief descriptions of each article’s content, which appear at the end of every listing.

And the articles are sorted into four categories—EM/Raynaud’s, General, Research, and Treatment—to help members more easily find subjects of interest.

You can find the list by clicking on “Articles” on the home page on the members side of TEA’s Web site. (www.erythromelalgia.org) Also, see pages 9, 10, and 11 of this issue for the complete list.

Networking Without the Web

TEA still offers the Networking Program, a service for people who don’t have easy access to the Web site.

Program co-chairperson Judy Reese will give you the names of other members in the Networking Program who live in your geographic area. And you can order copies of TEA’s Articles sent to you by mail.

Just fill out the form (above) and send it to Judy, 1155 E. Wild Duck Lane, Salt Lake City, Utah, USA 84117.

I want to participate in the TEA Networking Program.

Signature ____________________________
Name (Please print) _______________________
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Not computer savvy? Use this form to join TEA’s Networking Program.

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Mail orders to: Gayla Kanaster
2532 N. Fremont St., Tacoma, WA, USA 98406

Members with computers should print articles from the Web site
EM / Raynaud's Articles

Coexistence of Raynaud's syndrome and erythromelalgia.
Two letters with brief discussion on association of Raynaud’s with EM.

Treatment of Raynaud’s phenomenon with the selective serotonin reuptake inhibitor fluoxetine.

Temperature-associated vascular disorders: Raynaud’s phenomenon and erythromelalgia.
Belch JJ. A textbook of vascular medicine Lowe GD, Tooke JE. 1996, chapter 22:329-352. Long thorough account of all aspects of EM and Raynaud’s including cause, diagnosis, and treatments for different types of EM.

Serotonin reuptake inhibitors, Raynaud’s phenomenon and erythromelalgia.

Coexistence of erythromelalgia and Raynaud’s phenomenon.

Pharmacotherapy of Raynaud’s phenomenon.
Belch J, Ho M. Drugs 1996, 52:682-695. Abstract only. Cause of Raynaud’s and drugs for treating it.

General Articles

Erythromelalgia: a clinical study of 87 cases.

Erythromelalgia – A condition caused by microvascular arteriovenous shunting.

Erythromelalgia – a mysterious condition?

Erythromelalgia caused by platelet-mediated arteriolar inflammation and thrombosis in thrombocythemia.

Erythromelalgia: symptom or syndrome?

Hot feet: erythromelalgia and related disorders.

Poxviruses isolated from epidemic erythromelalgia in China.

A refractory case of erythromelalgia involving the ears.

Erythromelalgia: an under recognized manifestation of small-fibre neuropathy.

Natural history of erythromelalgia: presentation and outcome in 168 patients.

Research Articles

Neurobiology: a channel sets the gain on pain.
Waxman SG. Nature 2006, 444(7121):831-832. Different mutations of sodium channel Nav1.7 can trigger intense pain of EM or prevent all pain sensation.

Skin blood flow in adult human thermoregulation: how it works, when it does not, and why.

Reduced skin capillary density during attacks of erythromelalgia implies arteriovenous shunting as pathogenetic mechanism.

A single sodium channel mutation produces hyper- or hypoeexcitability in different types of neurons.

(Continued on page 10)
Research Articles (Continued from page 9)


Impaired skin vasomotor reflexes in patients with erythromelalgia.

Skin perfusion in patients with erythromelalgia.

Microvascular arteriovenous shunting is a probable pathogenetic mechanism in erythromelalgia.

A comment by Mark Davis and reply by Cato Mork follow this article.

Erythromelalgia: studies on pathogenesis and therapy.

Erythromelalgia: a hereditary pain syndrome enters the molecular era.

Gain-of-function mutation in Na+, 1.7 in familial erythromelalgia induces bursting of sensory neurons.

Genetic heterogeneity and exclusion of a modifying locus at 2q in a family with autosomal dominant primary erythermalgia.

SCN9A mutations define primary erythermalgia as a neuropathic disorder of voltage gated sodium channels.

Erythromelalgia: vasculopathy, neuropathy, or both? A prospective study of vascular and neurophysiologic studies in erythromelalgia.

Autosomal dominant erythromelalgia.

Mutations in SCN9A, encoding a sodium channel alpha subunit, in patients with primary erythermalgia.

Histopathological findings in primary erythermalgia show a decrease in small nerve fiber density.

Thermoregulatory sweat testing in patients with erythromelalgia.
Davis MD, Genebriera J, Sandroni P, Fealey RD. Archives of Dermatology 2006, 142(12):1583-1588. Absence of sweating and damaged small nerve fibers are prevalent in EM.

Treatment Articles

The Treatment of Erythromelalgia.
Cohen Jay S. Feb 2007 An article for the express use as a reference for TEA members; not yet published.

Lidocaine patch for pain of erythromelalgia: follow-up of 34 patients.

(Continued on page 11)
A way to understand erythromelalgia.

Erythromelalgia pain managed with gabapentin.

Nitroprusside treatment of erythromelalgia in an adolescent female.

Refractory idiopathic erythromelalgia.

Erythromelalgia.

Erythromelalgia.

High-dose oral magnesium treatment of chronic, intractable erythromelalgia.

Treatment of refractory primary erythromelalgia in a child using a continuous epidural infusion.

Unexpected healing of cutaneous ulcers in a short child (with erythromelalgia).

Erythromelalgia: response to serotonin reuptake inhibitors.

Treatment of erythromelalgia with a serotonin/noradrenaline reuptake inhibitor.

Lidocaine patch for pain of erythromelalgia.

Resolution of refractory symptoms of secondary erythromelalgia with intermittent epidural bupivacaine.

Erythromelalgia: an endothelial disorder responsive to sodium nitroprusside.

Treatment of primary erythromelalgia with cyclosporine.

Aspirin-responsive painful red, black toe, or finger syndrome in polycythemia vera associated with thrombocytopenia.

The prostaglandin E1 analog misoprostol reduces symptoms and microvascular shunting in erythromelalgia – a double-blind, crossover, placebo-compared study.

Alleviation of erythromelalgia with venlafaxine.

Combination gel of 1% amitriptyline and 0.5% ketamine to treat refractory erythromelalgia pain: a new treatment option?

Pregabalin and gabapentin for neuropathic pain and CRPS/RSD.
Stacey BR, Campbell P. Oregon Health and Science University. Published by Reflex Sympathetic Dystrophy Association 2006. Comparison of pregabalin and gabapentin for pain relief.

A case of inherited erythromelalgia.
Q and A (Continued from p. 4)

one were squeezing them. The good news, though—my doctor recently prescribed a beta-blocker called Lopressor (metopolol). It has practically eliminated the burning and pain during sleep. I cannot even begin to tell you what a relief this little pill has provided. I take 25 mg about 30 minutes before bedtime and another 25 mg when I wake up. I've also added 25 mg of amitriptyline at bedtime and that has helped tremendously.

A Russell Jarrett, Buckhannon, WV, U.S.: "Yes! Just like all the other crazy symptoms of this bizarre blood vessel disease, I thought it was just me. Every night after trying to sleep, I look down and my feet look completely different—swollen, tight skin, very hot and pressure. I try to re-think what I did or ate an hour before so I can prevent this. But, nothing ever changes these bedtime flares. I recently bought a chillow, which helps with some of my symptoms."

Q "Has anyone noticed the effect smoking has on EM?" (Submitted by John Forbush, Austentown, OH, U.S.)

A Isabelle Davis, West Bloomfield, MI, U.S.: "EM gave me the negative reinforcement I needed to finally quit smoking 13 years ago. That’s when EM really hit me. And every drag caused almost immediate burning in my feet and legs.

A Linda Reger: “I smoked for over 40 years and quit some 13 months ago. Beware—your feet may get worse because the nicotine has to clear out of the veins. I haven’t noticed my feet getting better, but I’m very glad I quit.”

Q "Does humidity affect other people with EM?" (Submitted by Karen Kimble, South Bend, IN, U.S.)

A Linda Reger: "I had to return early from my vacation in the Savannah area. The humidity caused the worst flare-up I’ve ever had and I couldn’t tolerate the pain."