The cause of blepharospasm (BSP) is unknown and its pathophysiology, like other focal dystonias, remains poorly understood. Presently there is no cure for the disorder and most oral medications are only minimally helpful. Botulinum toxin injections are often an effective treatment, but this involves discomfort associated with repeated injections and the relief is only temporary. An increased understanding of the pathophysiology of BSP is needed so better treatment options can be developed.

Almost everyone agrees that botulinum toxin is the gold standard for treating the eyelid spasms of benign essential blepharospasm, but very few people would argue that botulinum toxin is a perfect treatment. For example, most studies demonstrate that botulinum toxin does not eliminate the extreme light sensitivity often experienced by blepharospasm patients. Botulinum toxin's limitations occur because it is designed to block excessive lid closure, rather than to treat the underlying brain changes that cause blepharospasm. Botulinum toxin

People with blepharospasm have trouble with too much blinking and with spasms of the muscles that control eye closing. However, the problem does not come from the eyes or the muscles that control eye closure. Instead, the problem comes from the brain, where these muscles are controlled. Most experts believe that the brain is sending incorrect signals to cause the muscles to over-react, leading to too much blinking and spasms. Exactly what part of the brain is doing this, and what is going on there to trigger the problem is not entirely clear.

The 2014 BEBRF Symposium will be held in Pittsburgh, Pennsylvania at the University Club on the University of Pittsburgh Campus with Dr. Raymond Sekula as Program Director. Registration and program details will be printed in the March/April 2014 issue of the BEBRF Newsletter and on the BEBRF website – www.blepharospasm.org.
NEW MEMBER OF THE BEBRF MEDICAL ADVISORY BOARD

We are pleased to announce that Brian Berman, MD, MS, has agreed to serve as a member of the BEBRF Medical Advisory Board. Dr. Berman is an Assistant Professor of Neurology, Psychiatry, and Radiology at the University of Colorado Anschutz Medical Campus. He earned his Bachelor’s degree in Physics from the University of New Mexico followed by a Master’s degree in Medical Physics from the University of Colorado Health Science Center. He went on to receive his medical education at the University of Colorado and then did neurology residency training at the University of California at San Francisco. After a post-doctoral clinical research fellowship with Dr. Mark Hallett at the Human Motor Control section of the National Institutes of Health, he returned to join the faculty at the University of Colorado. His clinical duties include seeing patients with Movement Disorders at the University of Colorado Hospital and the Denver Veteran’s Affairs Hospital. His investigative activities include applying advanced structural and functional imaging techniques to gain a better understanding of the pathophysiology underlying motor and non-motor symptoms in dystonia and Parkinson’s disease. Dr. Berman was a 2013 BEBRF Research Grant Recipient (see page 1).

FEATURED STORY:

We are proud to announce the funding of over $220,000 worth of research into blepharospasm. Read about the projects starting on the front page.

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The opinions expressed in the articles in this newsletter are those of the authors and do not necessarily reflect the view of the BEBRF, publisher of the BEBRF Newsletter.
Support groups are an important part of BEBRF’s mission to our patients. Blepharospasm and Meige are rare disorders that most people have never heard of or knew anyone who had them. When a person is newly diagnosed, there are many questions, not only about the disorder, but also how to live with it. That is one reason why support groups are needed.

Picture this; it is 8:00 pm, the phone rings at the home of an area representative. It is a new patient who has just been diagnosed with blepharospasm. She is afraid, her doctor sent her to a specialist who wants to put botulinum toxin in her eyelids. She can’t see to drive, her head hurts, and she feels panicky. The area rep spends the next hour on the phone, calmly talking about BEB and how it is treated. And now, it just so happens, there is a support group meeting the next week where a local neurologist is going to speak about blepharospasm. Fast forward to the meeting; the new patient comes and is warmly greeted by a group of people wearing sunglasses, visors, and smiles. The doctor is easy to understand and has answered some of her questions personally. She goes home after the meeting with more knowledge about the disorder, a handful of literature, and has met others who have given her lots of ideas to manage her symptoms. She also has the phone and email information of someone to contact when she needs comforting.

This scene plays out many times across the country. Newly diagnosed and those who have had blepharospasm need to hear from others who share their story. They need to hear good speakers explain the disease and the treatments. The Foundation organized a system so that support is localized. The US was divided into four districts; Eastern, Western, Southern, and North Central. Each of these districts has a director who serves as an overseer of states within their district. Many states have a state coordinator who recruits area representatives. The area representatives maintain lists of patients and doctors in their locale and serve as contact people. Many area representatives as well as state coordinators and district directors hold support group meetings once or twice a year, sometimes more often. They also serve as contact people to whom the BEBRF office can refer new patients.

These volunteers who serve from local area representatives to district directors are very valuable assets to the mission of BEBRF. While we have a large number of leaders, there are many states and portions of large states that have no area representative or state coordinator. One reason is there aren’t enough patients or they live so far apart that support groups are not practical. Another big reason is that no one has volunteered to serve as a leader.

Support groups are the personal touch of BEBRF. The meetings might have a wonderful speaker or maybe only meet for care and share over a cup of coffee. Support groups don’t just happen, they need a leader to organize them, to arrange a location, secure a speaker, send announcements, and serve as facilitator. The group may only have 4-6 people or could have a couple of dozen. Numbers aren’t important; what is important is supporting the individual. We need more patients to be local representatives. If you would like to serve other patients in your area, contact the BEBRF office. There are lots of resources to help get you started and other leaders who can share how they do the job.

Thank you to all of our volunteers; you truly are life-lines to many people.
The primary dystonias, where dystonia is a relatively isolated problem, have been suspected to have a genetic basis for more than 40 years. The vast majority of cases are sporadic and emerge in adults, but approximately 15% of cases have an affected family member. This frequency is far higher than expected by chance. There also are rare examples where multiple family members are affected, with a mode of inheritance that seems to be dominant, but with reduced penetrance. Although two genes had previously been identified for early-onset generalized dystonias, it has been very difficult to identify genes for the adult presentations, even though they are far more common.

Finding genes for rare disorders is challenging for several reasons. One obvious challenge is access to large enough numbers of patient samples. Well-powered Genome Wide Association Studies (GWAS) require at least 1000-2000 samples, and collecting this number at a single site is not feasible. Sharing samples across different collection sites often meets obstacles associated with variable quality of the specimen or supporting clinical data, variable criteria used when enrolling subjects, and consent forms that unnecessarily restrict sharing of materials.

The Dystonia Coalition (DC), of which BEBRF is a member, has addressed these obstacles by establishing a DNA Biorepository that parallels a Natural History study, where 23 centers in North America and Europe collect clinical data with uniform protocols that permit sharing, and they send a blood sample for extraction and validation of DNA samples in a centralized laboratory in the NINDS Biorepository at Coriell. In two years, the DC Biorepository has accumulated 1,029 samples.

With support from the DC, we identified the first gene responsible for adult-onset cervical dystonia last year. This is the most common of the adult-onset focal dystonias. Affected patients have uncontrolled neck muscle spasms that cause neck pain and twist the head into abnormal positions, often with a jerky tremor. We used whole-exome sequencing in a large family where multiple members were affected to identify a sequence variant in the CIZ1 gene, which encodes a Cip1-interacting zinc finger protein. We then performed a case-control study of DNA specimens and found two additional sequence variants in the same gene. The protein product is expressed in brain regions that control motor functions, and plays a known role in DNA replication.

Since our report of the CIZ1 gene, we and others affiliated with the DC have used a similar approach involving exome sequencing of specific families to identify three more candidate genes for adult-onset focal dystonias: ANO3, GNAL, and TUBB4a. The first two also cause cervical dystonia, while the last causes laryngeal dystonia. The discoveries of these new genes may translate into a useful diagnostic tool in the near future, and open up new avenues of investigation to understand the pathogenesis of adult-onset focal dystonias.

However, the significance of these genes for the broader population of patients with dystonia depends on how frequently each is found in other families and in more common sporadic cases. Investing resources to further study a gene that causes dystonia in many families or sporadic cases has broader utility than a gene that causes dystonia in a single family. The DC Biorepository brings GWAS plans within reach for the rare population of dystonia patients, and permits validation of genes found by exome sequencing in individual families. The biorepository is now ready to begin to facilitate solving a problem in dystonia research that was recognized more than 40 years ago.

### The Rare Diseases Clinical Research Network (RDCRN)

The Rare Diseases Clinical Research Network (RDCRN) was established by the National Institutes of Health (NIH) to develop research studies for rare diseases, and to encourage cooperative partnerships among researchers at over 150 clinical centers around the world. This increased cooperation may lead to discoveries that will help treat and perhaps prevent these rare diseases, as well as produce medical advances that will benefit the population in general. The Rare Diseases Clinical Research Network is comprised of a Data Management and Coordinating Center and 17 consortia studying over 100 rare diseases.

### Become a Brain Donor – Advance Research

The Benign Essential Blepharospasm Research Foundation is working with the Harvard Brain Tissue Resource Center (HBTRC) to further research into the cause of blepharospasm through brain donation. If you are interested in participating in this program, please contact the BEBRF for more information – Email: bebrf@blepharospasm.org or Telephone: 1-409-832-0788. Or you may download the forms and the informational letter from this web site: www.blepharospasm.org
Researchers have achieved dynamic, atomic-scale views of a protein needed to maintain the transparency of the lens in the human eye. The work, funded in part by the National Institutes of Health, could lead to new insights and drugs for treating cataract and a variety of other health conditions.

Aquaporins form water channels between cells and are found in many tissues, but aquaporin zero (AQP0) is found only in the back of the eye. The lens is primarily made up of unique cells called lens fibers that contain little else besides water and proteins called crystallins. Tight packing of these fibers and of the crystallin proteins within them helps create a uniform medium that allows light to pass through the lens, almost as if it were glass.

Abnormal development or age-related changes in the lens can lead to cataract—a clouding of the lens that causes vision loss. Besides age, other risk factors for cataract include smoking, diabetes, and genetic factors. Mutations in the AQP0 gene can cause congenital cataract and may increase the risk of age-related cataract.

“The AQP0 channel is believed to play a vital role in maintaining the transparency of the lens and in regulating water volume in the lens fibers, so understanding the molecular details of how water flows through the channel could lead to a better understanding of cataract,” said Dr. Houam Araj, who oversees programs on lens, cataract and oculomotor systems at NIH’s National Eye Institute (NEI), which helped fund the research.

Closing of AQP0 channels is regulated by a calcium-sensitive protein called calmodulin, but the precise mechanism has been unclear. Some models have suggested that calmodulin simply acts as a plug to fill the open channel. The new study, published in Nature Structural and Molecular Biology, reveals a more nuanced process in which calmodulin essentially grasps the open channel and forces it to close.

The research was a collaboration between investigators at the University of California, Irvine, and the Janelia Farm Research Campus in Ashburn, Va., a part of the Howard Hughes Medical Institute (HHMI). Drs. James Hall and Douglas Tobias led the effort at UC Irvine. Dr. Tamir Gonen led the effort at Janelia Farm.

In prior studies, Dr. Gonen had examined the atomic structure of the AQP0 protein by X-ray crystallography, which involves crystallizing a protein and bombarding it with X-rays. But X-ray crystallography does not work well for large groups of proteins or for proteins in motion. So in the new study, the researchers first used electron microscopy to view AQP0 and calmodulin bound together. Then they combined their microscopy and crystallography data to generate computerized models of how the two proteins interact and to identify the most critical amino acids (the building blocks for proteins) within AQP0. To test their models, they neutralized those amino acids one by one in the actual AQP0 channel.

The AQP0 channel is made up of four identical barrel-shaped units, bundled together side by side. The researchers found that in the presence of calcium, calmodulin binds to one unit and then another, as if grabbing a pair of reins. This makes the channel twist slightly, which causes just a few amino acids within each unit to slide into the channel’s core and block the flow of water.

“Calmodulin essentially throws a molecular switch that moves in and out of the water pore, like the gate valve of a plumbing fixture,” Dr. Hall said.

This new view of AQP0 could help lead to new approaches for treating cataract, Dr. Hall said. Cataracts are the most common cause of blindness worldwide. In the United States, they affect about 1 in 6 people over age 40 and half over age 80. Congenital cataracts (present from birth) affect about 1 in 5,000 American children.

Cataracts can be successfully treated with surgery, in which the cloudy lens is removed and replaced with an artificial plastic lens. But the new findings “may be a step toward learning how to prevent or delay cataracts,” said Dr. Hall.

The new findings also provide inroads to understanding how calmodulin interacts with a variety of protein channels, and thus could open doors to new drugs for other common health conditions. In addition to aquaporins, our bodies rely on a vast menagerie of channels, many of which are regulated by calmodulin. For example, calmodulin helps control the gating of ion channels, which allow the passage of ions (charged particles) in and out of our cells and are essential for nerve cell firing, muscle contraction, and the rhythmic beating of the heart. This study provides the first structural model of calmodulin bound to any complete protein channel.

Drs. Daniel Clemens and Steve Reichow were co-first authors on the study. NIH support for the study came from NEI (grants EY005661, EY018768), the National Institute of General Medical Sciences (NIGMS grant GM079233), a joint program on “Making Sense of Voltage Sensors” co-funded by NIGMS and the National Institute of Neurological Disorders and Stroke (grant GM086685), and the National Library of Medicine (grant LM007443). Additional support came from HHMI, the National Science Foundation, and the German Academy of Sciences.
The Centers for Medicare and Medicaid Services (CMS) have set the Medicare premiums, deductibles and coinsurance amounts to be paid by Medicare beneficiaries in 2014.

MEDICARE PART A (HOSPITAL INSURANCE)
Medicare Part A helps cover inpatient hospital care, inpatient care in a skilled nursing facility, hospice care, and home health care services.

- **Part A Monthly Premium**: Approximately 99 percent of Medicare beneficiaries do not have to pay a premium for Part A services because they have at least 40 quarters of Medicare-covered employment (or are the spouse or widow(er) of such a person). However, some enrollees who have fewer than 40 quarters of coverage obtain Part A coverage by paying a monthly premium. Those with less than 40 quarters of covered employment can buy Part A coverage by paying a monthly premium of up to $426 in 2014.

- **Part A Deductible and Coinsurance**: For each benefit period, Medicare pays all covered costs except the Part A deductible during the first 60 days and coinsurance amounts for hospital stays that last beyond 60 days and no more than 150 days. (A benefit period starts the day a patient is admitted and ends when the patient has been out of the hospital for 60 days in a row). In 2014, for each benefit period, you pay:
  - A deductible of $1,216 and no coinsurance for a hospital stay of 1-60 days.
  - $304 per day for days 61-90 of a hospital stay.
  - $608 per day for 91-150 of a hospital stay and all costs for each day beyond 150 days.
  - Skilled Nursing Facility coinsurance is $152 per day for days 21-100 and all costs for each day after day 100 in a benefit period.

MEDICARE PART B (MEDICAL INSURANCE)
Medicare Part B helps cover medically necessary physicians’ services, outpatient care, home health services, durable medical equipment, and other medical services. Part B also covers many previous services.

- **Part B Monthly Premium**: As a result of cost controls implemented in the Affordable Care Act, most Medicare beneficiaries will not see a Part B monthly premium increase in 2014. Most beneficiaries will continue to pay the 2013 premium of $104.90.
  - **Part B Income Related Adjustments**: while the majority of Medicare beneficiaries will pay the standard Medicare Part B monthly premiums of $104.90 in 2014, some will pay more based on their annual income. Specifically, if a beneficiary’s “modified adjusted gross income” as reported on their IRS tax return from two years ago is above $85,000 ($170,000 filing jointly), the beneficiary is responsible for a larger portion of the cost of their coverage. These premium adjustments range from $42 to $230.80 more per month for Medicare Part B.
  - **NOTE**: The Affordable Care Act also requires Part D enrollees whose incomes exceed these same thresholds to pay a monthly adjustment amount in addition to their plan premium. These enrollees will pay the regular plan premium to their Part D plan and will pay the income-related adjustment to Medicare.

- **Part B Deductible and Coinsurance**: Costs for Part B services depend on whether you have Original Medicare or are in a Medicare health plan. For some services, there are no costs, but you may have to pay for doctor visits. If the Part B deductible applies, you must pay all costs until you meet the yearly Part B deductible before Medicare begins to pay its share.
  - In 2014, the Part B deductible remains unchanged from 2013 and will continue to be $147.
  - After your deductible is met, you typically pay 20% of the Medicare-approved amount of the service.
  - If you join a Medicare Advantage Plan (like an HMO or PPO) or have other insurance (like a Medigap policy, or employer or union coverage), your costs may be different.

**SO KNOW YOUR MEDICARE COSTS!**

MAIL BAG – LETTER TO THE FOUNDATION

Dear BEBRF,

During this season of giving thanks, it is more than appropriate to let each other know why we are thankful for them. The information and resources I have received from BEBRF has provided such encouragement, or maybe I should say courage, to keep pressing forward through the disability effects of blepharospasm.

The network of support groups is great and the work you do to orchestrate the yearly Symposiums is outstanding! I was fortunate to have friends who paid for my plane flight to Columbus so I could attend the 2012 Symposium. Having that experience gave me strength to endure. I equally enjoyed attending the BEBRF Symposium in San Diego this year.

Thank you one and ALL... Matt
Prior electrophysiological studies suggest that the trigeminal nerve system—a brain system that transmits sensory information from the eye to the facial nerves that trigger blinking—may be abnormal in patients with BSP. To date, however, an exploration into trigeminal nerve system function together with an examination of activity that occurs throughout the rest of the brain during blinking in BSP has not been performed.

While a wide variety of imaging studies help support the presence of basal ganglia dysfunction in focal dystonia, the sensorimotor cortex, thalamus and cerebellum may also play a role in its pathophysiology. Indeed, a network model with abnormal interactions between the basal ganglia and cerebellar circuits has increasingly been proposed as a central problem in dystonia. An investigation into brain circuit functioning during individual dystonic muscle spasms in BSP, however, has not previously been conducted.

In this BEBRF research project, we aim to fill in gaps in our knowledge of the brain mechanisms that drive blinking and underlie dystonic muscle spasms in BSP using functional MRI (fMRI). We will first test the hypothesis that spontaneous and reflexive blinking is associated with an increase in activity of the trigeminal nerve system in BSP patients relative to healthy controls. Second we will test the hypothesis that basal ganglia circuit dysfunction leads to the triggering of dystonic spasms while cerebellar circuit dysfunction determines the severity of spasms.

Fifteen adult patients with BSP will be recruited. Patients must not be on medications that might alter brain activity. Additionally, patients need to be symptomatic so they must not have been injected with botulinum toxin within the prior 3 months. Fifteen matched healthy controls without neurologic or psychiatric disease will be recruited from patient spouses and the community at large.

Study participants will have their brain activity recorded during four fMRI scans while they lie comfortably with their eyes open and gaze on a fixation point. During all four fMRI scans, spontaneous blinks in all participants and eye muscle spasms in patients will be recorded using electrodes attached to the skin around the eye that can detect the electrical activity in the underlying muscles. During two of the four scans, reflexive blinks will be elicited using air puffs delivered to an eye. The occurrence of all blinks and spasms will be verified by comparing muscle electrode recordings to video recordings that are made with an eye-tracker system inside the MRI scanner.

By characterizing the abnormal brain signals associated with spontaneous and reflexive blinking, a better understanding of how blinking mechanisms are affected in BSP will be gained. This knowledge in turn could lead to an imaging biomarker that can improve our ability to diagnose BSP and monitor brain responses to treatment interventions. Identifying the brain mechanisms associated with dystonic spasms in BSP could help improve emerging therapies that alter circuit function. For example, it might help direct the selection of the best target brain region for transcranial magnetic stimulation, which has shown some potential to improve symptoms in BSP. Further investigations will be needed, however, to try and link abnormal brain activity and circuit dysfunction to the underlying molecular or cellular disturbances that may be present in BSP.

Several studies have attempted to figure out what is going on in the brain using brain scans. These studies have been very important for identifying regions that may not be working normally. They have pointed to problems in several different regions. They include many regions involved in controlling movement, such as the basal ganglia, brainstem, cerebellum, cerebral cortex, and thalamus. Which of these regions is most important for causing the problem in blepharospasm is not yet known, because these scans provide only an indirect window to look into the brain.

To figure out what is going on in the brain, it is helpful to look at the brain directly. The field of Neuropathology is devoted to looking directly at brain tissue, using a microscope so that individual nerve cells (neurons) and other types of cells can be seen directly. Because these methods require cutting the brain into thin sections, they cannot be applied during life. These methods can only be applied to brains that were donated for evaluation after death.

Only a few prior neuropathology studies have examined the brains of people who had blepharospasm. Experts looking at these brains could not see any obvious abnormalities, and there is broad agreement that there are no glaring brain defects in blepharospasm. However, all of these studies included only one or two brains. Most of these studies were conducted several decades ago with relatively limited techniques for revealing the neurons and other cells. Examining additional brains more carefully is important to see if any subtle changes can be found anywhere.

There are several reasons why so few brains from people with blepharospasm have been studied so far. First, blepharospasm is a rare condition, so collecting more than a few brains is not easy. Having one or two brains is good, but ten or twenty brains is better if the changes are subtle. Second, blepharospasm is not a lethal condition. Most people with blepharospasm have normal life spans, and their brains cannot be evaluated directly during life. Asking their families to donate their brains after death is not an easy thing to do, so there are not many brains available to evaluate.
FOCUS ON

NEW MEXICO: A state wide meeting was held recently in Albuquerque, New Mexico. Al Deguio, State Coordinator, facilitated the meeting.

MINNESOTA: A State wide meeting was held recently in the Minneapolis area. Dr. Andrew Harrison was the speaker and Virgil Koski, BEBRF State Coordinator conducted the meeting.

L-R: Dr. Andrew Harrison, Ann Loch, Virgil Koski, Jackie Moe, Dianne Badalich, Jo Ann Arnold, Sandy Cavallin, Marion Michalik, Liz Anderson, Kathy Henry, Julie Brown, Deb Stokes, Sharon Shunk, Reonne Nelson, Grace Goodall, Wanda Strobush and Mary Leduc.

LONG BEACH, CALIFORNIA: A small group meeting was held in Long Beach, California in November. Janice Dominguez is the Area Representative. Stephanie Kallay, Merz Pharmaceutical Representative provided lunch. L-R: Brita Goldsmith, Stephanie Kallay, and Janice Dominguez

NEW ORLEANS, LOUISIANA: A November meeting was held at the Ochsner Medical Center in New Orleans Dr. Georgia Lea, neurologist, was the speaker. The meeting was facilitated by Brenda Hopkins, State Coordinator. Sitting L-R: Peter Kahan, Dr. Georgia Lea, Frances Stevens and Brenda Hopkins. Standing L-R: Betty Trosclair, Tom Wonderlich, Sal Bernadas, Miriam Azuru, Avis Richard-Griffin, Kathleen Meadows, Winston Ricks, Dominque Thomas, and Linda Stevens.

GREENVILLE, SOUTH CAROLINA: Dr. Kathleen V. Woschkolup spoke at a fall meeting in Greenville, South Carolina. Pat Miller is the State Coordinator and Judy Duncan is the Area Representative.

AUSTIN, TEXAS: A meeting of the Austin Area BEB group met last October. Zoe Fallgren, Texas State Coordinator facilitated the meeting.
WEST SAN FERNANDO VALLEY, CALIFORNIA: A well-attended meeting was held at Northridge Hospital and Medical Center in Northridge, California last October. Mark Sheeler, Area Representative for the last fifteen years, facilitated the meeting. This was his last meeting. Beverly Conradson will be leading the group starting in the spring. Dr. David Samimi was the guest speaker.

ALEXANDRIA, LOUISIANA: A productive and interesting support group meeting was held in Alexandria, Louisiana recently. The meeting was organized by Brenda Hopkins, BBRF State Coordinator. Dr. Karren Laird-Russo was the speaker. Kneeling: Donna McMickens, Dr. Laird-Russo and Brenda Hopkins. Standing: L-R: Gladys Bagley, Elizabeth Ehler, Dr. Ray Beurlet, Jr., Anna Normand, Ronnie Lemoine, Daisy Lasyone, Debra Allen, Donnie Lasyone, Joyce Pierce, Gloria Rogers, Linda Stevens and Frances Stevens.

PITTSBURGH, PENNSYLVANIA: A fall meeting was held in Pittsburgh and facilitated by Marion Maniet, Area Representative. L-R: Tom Kudlawiec, Bob Davis, Cheryl Kudlawiec, Debi Hall, Marion Maniet and David Maniet.

DALLAS, TEXAS ARA: Parker County was the place for a support group meeting in the Dallas area in October. Ena Wilmot, BBRF Area Representative, conducted the meeting. Those attending were: Nancy and Don Good, Kathryn King, Ann and Walt Piasecki, Paula Roland, Linda Corley and Laurie and Ena Wilmot.

NEW YORK, NEW YORK: St. Luke’s Roosevelt Hospital in New York City was the meeting place for a BEB support group. Dr. David Swope, Neurologist, was the speaker. The meeting was facilitated by Lee Ann De Berry, State Coordinator.
Q: I have been getting BOTOX® injections since the mid 1980s (I participated in clinical trials in California). I am now 87 years old and about two years ago, I moved to live near my daughter. My new doctor injects less sites and uses more BOTOX® in each site. He also insists on using EMLA (a topical anesthetic) before the injections, which I’ve never needed before. The injections used to last 4 months but now they are only lasting about 3 months. The last couple of times after the injections, I’ve developed headaches that seem to start around my eyes. Could the BOTOX® be causing this? What about the EMLA? I’ve never had this problem before.

A: EMLA cream is a local anesthetic cream that can decrease the pain of injections. I have not found it necessary for most patients. One study found that using the EMLA cream may decrease the efficacy of botulinum toxin injections. It is unlikely that the EMLA cream is causing your headaches. I would recommend that you find an experienced injector who would be willing to try your old injection pattern that worked well for you in the past. There are many causes for headaches, and it is possible that your current injections are a trigger for you.

Andrew Harrison, MD, Director, Oculoplastic and Orbital Surgery, Department of Ophthalmology, University of Minnesota, Minneapolis, Minnesota

Q: I have had blepharospasm for many years. Along with the spasms of the muscles around the eyes, and the sensation of heavy eyelids, is the problem of constant blinking. The blinking seems to be triggered by the stinging sensation on the surface of the eye itself. The use of artificial tears and dark glasses only slightly help. I have noticed though, when having my eyes dilated during an eye exam, the constant blinking sensation stops and I get some relief. Is there a medication that numbs the surface of the eye without dilating it?

A: The reason the blinking stops is because before dilating drops are instilled in the eye, a numbing drop is used. This probably decreases the stinging. You should NEVER use numbing drops on a regular basis since they are very toxic to the eye and can cause loss of the eye. The stinging likely is due to dry eye. Please consult a Cornea/Dry Eye specialist. This needs vigorous treatment, not the occasional drop of artificial tears.

Peter J. Savino, MD, Division of Neuro-Ophthalmology, Shiley Eye Center, University of California, San Diego

Q: I am in the process of completing an application for Long-Term Care Insurance. There are many questions including do I have a neurological disorder and/or visual disorder. When completing medical forms for insurance purposes, in what category does blepharospasm fall?

A: Blepharospasm should be classified as a Neurological Disorder, particularly, a focal dystonia.

Mark Stacy, MD, Director, Parkinson’s Disease and Movement Disorders, Duke University Medical Center, Durham, North Carolina

Q: For many, dry eyes are a part of BEB. Are there any advantages to installing punctual plugs as opposed to the frequent use of eye drops?

A: Dry eye is not only visually disabling, but it can also be extremely painful and threaten the very survival of the eye. It is also considered a trigger in benign essential blepharospasm (BEB), as you so correctly identified. Your question about punctal plugs is a very good and valid one and, in order to address it fully, I will first briefly mention some key points about tears.

Tears are made of mostly water but there is also a small but vital component in tears that is comprised of oils. This oil component is essential for decreasing tear evaporation and, when absent, causes the eye to dry out too fast. Certain conditions such as rosacea and chronic eyelid inflammation contribute to dry eyes by resulting in decreased oil in the tear film and can also decrease the aqueous or water component as well. These situations result in not only decreased tear production but also the tears that are present are poor “quality” and lubricate the surface of the eye inadequately. In these situations we advise tear supplementation as well as other measures to improve, over a period of time, the quality of tears.

Now, coming back to your question about punctal plugs. Absolutely, if tear quality is normal or optimal but the amount is decreased due to various factors, punctal plugs can certainly play a role in keeping the eye lubricated by virtue of preventing them from draining out of the eye and forming a longer-lasting tear lake.

However, some people with eyelid inflammation may develop a condition known as “toxic tear syndrome.” This is a problem where the thick, poorly diluted and rancid oils in someone with a poor quality tear film cause problematic irritation and inflammation of the eye surface that in turn could trigger BEB. In these situations, placement of punctal plugs often exacerbates the problem as the irritating oils are then unable to drain away and merely further concentrate on the surface of the eye as the water from the tear film evaporates.

So, the treatment of dry eyes in BEB depends largely on the quality and quantity of tears made by the individual. In deciding if someone would benefit from punctal plugs so that they can make use of their own tears versus constantly relying on artificial tears, your ophthalmologist should take all these factors into account such as tear production, quality of tears, eyelid inflammation and skin disorders in order to formulate the best treatment plan.

Mirwat S. Sami, MD, Plastic Eye Surgery Associates, Houston, Texas
Q: I was diagnosed with benign essential blepharospasm in 1987, however, my eyelids do not involuntarily clamp shut in both eyes. Initially I started having spasms in the left eye with pain alongside of my nose. The spasms with pain eventually moved to the right eye with similar symptoms. I have had botulinum toxin injections but they were unsuccessful in controlling the spasms. I do have a problem falling asleep and staying asleep and my neurologist believes it is due to the spasms. My problem sounds more like what you foundation describes as hemifacial spasm. I have been on trazodone and neurontin for sleep and take it nightly in order to fall asleep and sometimes it keeps me awake. Have you known of anyone that has hemifacial spasm and is dealing with pain as well?

A1: Hemifacial spasm does not involve pain. Patients do, however, complain of fatigue in their face.

Raymond Sekula, MD, Department of Neurological Surgery, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

A2: Without personally examining you I am unable to determine whether you have blepharospasm, which often starts asymmetrically, or whether you have bilateral hemifacial spasm, which is extremely rare (Tan EK, Jankovic J. Bilateral hemifacial spasm: A report of 5 cases and a literature review. Mov Disord 1999;14:345-349). I suggest you consult a movement disorder neurologist who can differentiate these two conditions or diagnose another disorder that would explain your symptoms. It is extremely unusual for a patient with either blepharospasm or hemifacial spasm not to obtain benefit from botulinum toxin injections.

Joseph Jankovic, MD, Director, Parkinson’s Disease Center and Movement Disorders Clinic, Baylor College of Medicine, Houston, Texas

Q: It is 30 days from successful botulinum toxin injections. At what point in the 90 day period, is it recommended to get reading glasses? The ophthalmologist said I had mild astigmatism, or blurriness from improper use of eye drops. I will step up my eye drops, and go in at 60 days from the injections for another exam, and get reading glasses, and computer tinted glasses. I have just been using drugstore readers for 5 years, because I thought it was futile, with my eyes changing during the 90 days.

A: For most patients with BEB who are well-controlled with botulinum neurotoxin injections (BoNT), there should be very little difference in refraction (i.e. change in the power of glasses needed) throughout a treatment cycle. If your BEB is not well-controlled and your eyelids are squeezing hard, the pressure of the eyelids can distort the shape of the eye and change the measured refraction. Prying or holding the eyes open in an attempt to obtain a refraction can also put pressure on the eye, confounding the examiner’s measurements.

Everyone’s response to BoNT is different. Not everyone is on a 90 day cycle. In fact, most of our patients are not. If you chart your BoNT effect, you will find a "Lag Time" (the time for the BoNT to have their full effect) and a “Duration” (the time that your BoNT lasts before your spasms start again). For most people with BEB who are not significantly changing their diet and who receive the same toxin provided by the same experienced injector with an individualized treatment pattern, Lag Time and Duration is pretty consistent from treatment to treatment for that individual, but these metrics are different from person to person. If you receive your injections at a Frequency = (Duration - 1/2[Lag Time]), then there should be minimal spasms and minimal eye drying at the start of your treatment cycle, and then it doesn’t really matter
when you get a refraction (exam to check for the power of glasses needed). If you find that your vision is more blurry shortly after receiving BoNT, then your eyes are probably drying out when your injections are most effective, and you need to lubricate more. If you find that your vision is most blurry just before the next BoNT injections, then you are probably trying hard to keep your eyes open, getting an incomplete blink, and thus causing eye drying, and you might wish to decrease the time between your treatments. It is difficult for an examiner to provide you with a good refraction if your eyes are dry, since drying distorts the optical surface of the eye. Short answer: You can obtain a refraction any time if your BEB is well-controlled. If your BEB is not well-controlled, then get a refraction in the middle of your cycle when your eyes are most open, least dry, and have the best vision.

Charles N.S. Soparkar, MD, PhD, Plastic Eye Surgery Associates, Houston, Texas

Q: I recently had my first BotoX® injections around my eyes, but I chose not to have them around my nose. My right nostril constantly feels like it has dust in it and is twitching and causing my right eye to twitch, too. Will botox help this nostril problem? Is that part of blepharospasm, too?

A: Without personally examining you it is difficult to be certain about the nature of your nose twitching. It is, however, possible that it is related to the blepharospasm as most patients with blepharospasm also have involuntary muscle contractions of other facial muscles, including the paranasal muscles. These facial (and mouth and jaw contractions) are a manifestation of cranial dystonia. Blepharospasm is considered one component (categorized as focal dystonia).

Q: After 20 years of having blepharospasm, I have recently been diagnosed with fortification spectrum. Is there any connection?

A: Fortification spectra, also known as scintillating scotoma, is a medical term that is used to describe visual disturbance that is often experienced before the onset of a migraine headache as part of “migraine aura.” It typically starts as a tiny flickering of light that moves to the peripheral vision of one or both eyes where it expands and often assumes different shapes and patterns such as zig zag lines resembling fortification of a castle. The symptoms typically last only a few minutes, rarely more than one hour, before the migraine headache emerges. Although patients with blepharospasm often complain of increased light sensitivity (photophobia) there is no evidence that fortification spectra (or migraine headaches) are related to blepharospasm.

Joseph Jankovic, MD, Director, Parkinson’s Disease Center and Movement Disorders Clinic, Baylor College of Medicine, Houston, Texas

Q: I have had focal dystonia for over a decade now. Over many years I have tried many, many medications and therapies, none of which helped much. BOTOX® shots into my tongue have been beneficial, but the results are often inconsistent from month to month. Quite by accident I tried Ambien or (zolpidem) for some sleep issues I was having, and my dystonia improved quite dramatically. I tried it again with the same results, my focal dystonia in my jaw and tongue diminished by around 70 to 80 percent. I did some research online and found some case studies where Ambien had been used to treat focal dystonia too. In combination with BOTOX® shots - it helps me enormously and I feel compelled to share this information with as many people as possible who have this seemingly intractable disease.

A: There have been a number of reports of individual patients with different forms of dystonia who have responded to zolpidem (Ambien is the brand name). The drug is being tried now for other patients; it is likely that a proper trial is warranted. Zolpidem may also have severe side-effects of sleepiness and interfering with ability to think clearly, so it must be used carefully. It is primarily used as a sleeping pill! There does seem to be a deficit of inhibition in dystonia and zolpidem might improve this.

Q: A few weeks ago, the BEBRF office sent me a list of “Drugs That May Cause Dystonia”. I am wondering if the BEBRF doctors would recommend an antidepressant drug that will not make my eyes worse.

A: It is difficult to give generic advice about drugs since patients do seem to differ significantly one to another. If patients have some experience with certain drugs that cause problems, then they might try drugs of a different class. The patient’s personal physician should guide him/her.

Q: I was diagnosed with Meige in 2006 and endured the full blown symptoms affecting my eyelids, mouth, and neck muscles. I have been getting BOTOX® injections as well as oral medications. During the past 2 years, my symptoms have gradually become more controllable. I voluntarily discontinued my BOTOX® injections last April, but I am still on one oral medication (I plan to try to get off that as soon as possible). There is no question that BOTOX® was the first line of help for me as I would have never improved without it. I do not feel my Meige is in remission as the symptoms are still present. However, I can suppress the abnormal symptoms. My eyelids are well under voluntary control, my breathing and speech muscles are in need of improvement, but they are improving.

Since I ran track/cross country in college, I was able to easily start running and lifting weights again and also incorporate Pilates classes for balance and flexibility. I learned how to use diaphragmatic breathing and some biofeedback training to influence stress levels. When I run, I am constantly thinking about each step and adjusting as necessary for balance, breathing and what my body tells me. Running is not an automatic thing for me now like my younger days. Breathing while running is still a limiting factor, but it is improving. We already know that patients can use sensory tricks to temporarily help their situation. Maybe there is more to this technique than we know. Would it not be good research to investigate the effect of exercise and cuing systems on these movement disorders?

A: There are remissions, both full and partial, in patients with focal dystonias including Meige (cranial dystonia) and blepharospasm. Why and how this happens is not known. Remissions may only be temporary and it is difficult to predict what would happen in future. Perhaps the exercise is useful; we just don't know. As to the botulinum toxin, it is only symptomatic, and there is no problem in discontinuing it (temporarily or permanently).

Mark Hallett, MD, Chief, Human Motor Control Section, NINDS, NIH, Bethesda, Maryland
NEUROPATHOLOGY OF BLEPHARO SPASM

Continued from page 7

Fortunately, the Benign Essential Blepharospasm Research Foundation (BEBRF) has partnered with other dystonia foundations to create a brain bank, called the Dystonia Brain Collective. Those with blepharospasm can sign up by contacting the BEBRF at bebrf@blepharospasm.org. This program allows people with blepharospasm to sign up during life, so that their family knows about their wish for brain donation when the time comes. People with blepharospasm can discuss the possibility of brain donation with their family in advance, so decisions can be made early. Several brains have already been collected in the Dystonia Brain Collective, and this growing resource makes it possible to plan better neuropathological studies for blepharospasm.

We recently completed a study that included the brains of six people who had cervical dystonia. This condition is thought to be similar to blepharospasm, except that it involves jerking and spasms of the neck muscles. Some people have both blepharospasm and cervical dystonia combined. In our study, we found some subtle changes in one brain region, called the cerebellum. This brain region is known to be involved in the automatic control of many different types of movements, so it makes sense that it could be the source for abnormal movement control. In this region, there seemed to be fewer neurons of a specific type called Purkinje neurons. Purkinje neurons also looked a bit unusual. A picture of a typical Purkinje neuron is shown in the figure to the right. These changes in Purkinje neurons were found in people who had cervical dystonia, not blepharospasm. Because cervical dystonia and blepharospasm are thought to be related to each other, there is a possibility that Purkinje neurons of the cerebellum in patients with blepharospasm will show these same changes. If they do, it will be the first finding of a consistent change in a specific brain region in people with blepharospasm.

Why do we care what brain region and neurons look different in blepharospasm? The main reason is that the information provides an important clue for which brain region may be responsible for causing the blinking and spasms associated with blepharospasm. If we can pinpoint the exact region responsible for sending the wrong signals to the muscles of eye closure, then we may be able to direct further studies at these neurons to figure out what they are doing and maybe even correct the incorrect signals.

A NOVEL TREATMENT FOR BENIGN ESSENTIAL BLEPHARO SPASM

Continued from front page

weakens the connection between the brain and eyelid closing muscle so that it is not possible to produce lid spasms even though the brain is still trying to generate them. Botulinum toxin treatment is akin to giving someone a pain killer when they have a broken leg. There is invaluable relief from the pain, but the pain killer doesn't directly help the leg to heal. Like many investigators studying blepharospasm, the goal of my research is to identify the underlying brain changes that cause blepharospasm and use that knowledge to develop a procedure to treat those brain changes. We believe that we may have found a better treatment. To understand the basis for the novel treatment we're testing, it's important to know something about what previous research has found out about the neural basis of benign essential blepharospasm.

For reasons that are currently unclear, blepharospasm causes the trigeminal system to become hyperexcitable. This technical phrase means that the portion of your brain responsible for sensations from your face and eyes and initiating blinks, the trigeminal system, becomes overactive. This overactivity can create a sensation of dry eye or eye irritation, make your eyes more sensitive to light, produce excessive blinking, as well as generate spasms of eyelid closure. My lab works from the hypothesis that trigeminal over activity results from an exaggeration of motor learning created by abnormal interactions among the trigeminal system, and two other brain regions, the cerebellum and the basal ganglia. To understand trigeminal learning, we developed a procedure in humans in which we delivered high frequency stimulation to a trigeminal nerve that evokes a blink. By adjusting the amplitude of trigeminal reflex blinks (http://mysbfiles.stonybrook.edu/~levinger/mao2001.pdf). Consistent with our hypothesis of enhanced motor learning in the trigeminal system with blepharospasm, other investigators used our procedure to increase blink amplitude in individuals with blepharospasm and found that the motor learning was exaggerated compared to control subjects (http://www.ncbi.nlm.nih.gov/pubmed/16407569). Another BEBRF grantee, Dr. Kranz, used our learning procedure to depress the trigeminal system in patients with blepharospasm. He and his colleagues reported that a single presentation of trigeminal depression paradigm produced “…a subjective, but not objective, improvement…” (http://www.ncbi.nlm.nih.gov/pubmed/23401198). We decided to use an animal model to investigate whether multiple
presentations of the procedure might produce a long lasting effect and objectively, as well as subjectively, improve the symptoms of blepharospasm.

Our studies demonstrated that repeating the high frequency stimulus procedure to reduce blink amplitude once a day for ten days created long-term depression of the trigeminal system. First, the trigeminal system became less active with repeated treatments. Second, the high frequency stimulus procedure became more effective at depressing trigeminal activity each time it was applied. Thus, repeating the high frequency stimulation paradigm caused a long-term reduction in the activity of the trigeminal system and the high frequency stimulation procedure became more effective at depressing the trigeminal system each time it was used. This reduction of trigeminal activity seen in the animal model was what was needed to diminish the trigeminal over activity in benign essential blepharospasm. Given that our animal model exhibited the same changes in blinking with diseases such as Parkinson’s disease, dry eye, and blepharospasm as occur in humans, we requested support from BEBRF to investigate whether repeated high frequency stimulation treatments reduced or eliminated spasms of lid closure, excessive blinking, and extreme sensitivity to light in blepharospasm patients.

The experimental procedure is straightforward. We will perform an objective evaluation of blepharospasm symptoms when patients come to the clinic for botulinum toxin injections. If the patient agrees to participate in the experiment, they will forego their botulinum toxin injection for two weeks. During this two week period, the subject will receive ten days of high frequency stimulation requiring approximately 30-40 minutes each day. At the end of the two weeks, the subject will be reevaluated and receive their botulinum toxin injection. To avoid placebo effects in which the patient feels an artificial improvement in response to receiving a treatment that they believe will improve their condition; we will employ two high frequency stimulation procedures. The experimental procedure will depress trigeminal activity and the control procedure will not alter trigeminal system activity. Patients will be randomly assigned to receive either the depressing or the ineffective procedure for the first two weeks. When the patient returns for later botulinum toxin injections, we will ask them to participate in the experiment one more time and use the high frequency stimulation procedure that they did not receive in the first two weeks. We believe that repeating the experimental high frequency stimulation procedure will lengthen the time between botulinum toxin injections, reduce the extreme sensitivity to light and sensations of eye irritation, and decrease the excessive blinking. Thanks to the generous support of the BEBRF, we’ll be able to test this potential new treatment in the coming year.

Thanks to the generous support of the BEBRF, we’ll be able to test this potential new treatment in the coming year.

IF YOU WOULD LIKE TO START A SUPPORT GROUP IN YOUR AREA, CONTACT YOUR DISTRICT DIRECTOR OR THE BEBRF OFFICE FOR ASSISTANCE.

MARK’S RAMBLINGS
Mark Sheeler, Woodland Hills, California

1. Did you know that the typewriter was invented before the fountain pen? Now some youngsters will ask, “What’s a fountain pen?” Mine was a Waterman!

2. A vulture carrying two dead raccoons boards an airplane. The flight attendant looks at him and says, “I’m sorry, sir, only one carrion allowed per passenger!”

3. Two fish swim into a concrete wall. One turns to the other and says, Dam?

4. A person sent ten puns into a contest with the hope that one of the puns would win a prize. No pun in ten did!

5. In filling out an application where it says, “In case of emergency notify,” I put Doctor!

6. I was always told by my parents to respect my elders. However, I’m having a heck of a time now finding an “elder.”

7. I used to have a handle on life, now I find it’s broken!

8. In our advancing years, I find my wife has gotten jealous because the voices only talk to me!

9. It’s scary when your stomach starts to make the same sounds as your coffee maker.

10. I’ve learned that people are as happy as they want to be! I’d better quit while I’m still behind!

MS
**Support Group Meetings**

**To get your support group meeting in the next issue of the newsletter,**

*Please notify the foundation office, before February 3, 2014, the next newsletter deadline.*

**New Area Representative**

Peoria, Illinois Area  
Linda Buck  
1211 W Poplar Woods Ct  
Dunlap, IL 61525  
Tel. (309) 981-9722  
Email: lbuck1234@comcast.net

Terre Haute, Indiana Area  
Bernie Reece  
1824 E 44th Dr  
Terre Haute, IN 47802  
Tel. (812) 243-2560  
Email: breece@frontier.com

**Contact Person**

Wyoming  
Norman Garrey  
Rock Springs, WY 82901  
Email: whitetiger97@hotmail.com

**New Email**

Traverse City, Michigan Area Representative  
Carol Taberski  
ctaberski@yahoo.com

**Support Group Meetings**

**South**

Blepharo-Buddies Awareness Support Group  
Sunday, January 26, 2014 and Sunday, April 27, 2014; 1 – 4 p.m.  
Speaker: To Be Announced  
Contact: Linda Webb… (256) 723-2661

**Mississippi Statewide Meeting**

Wednesday, February 12, 2014  
11:00 a.m. - 1:00 p.m.  
Home of Brenda Hopkins, 317 Edmond Jones Rd, Lumberton, MS 39455  
General Meeting & Pot Luck Luncheon, Please RSVP  
Contact: Brenda Hopkins… (601) 796-3741, Email: brenda_hopkins@att.net

**Dallas, Texas Area**

Tuesday, January 21, 2014 and Tuesday, March 18, 2014  
12:00 Noon – 3:00 p.m.  
Dallas Baptist University, 110 Grapevine Hwy, Hurst, TX 76054  
Contact: Ena Wilmot… (817) 488-0445 (home), (469) 964-1428 (cell), Email: enamwa@hotmail.com

**West**

Berkeley/Oakland, California Area  
Sunday, January 26, 2014; 2 – 4 p.m.  
Home of Herdis Pelle, 2607 Grant St, Berkeley, CA 94703  
Please RSVP  
Contact: Herdis Pelle… (510) 649-9812, Email: herdispelle@pacbell.net

**Colorado**

Saturday, February 1, 2014; 1 - 3 p.m.  
Location to be determined.  
Speaker: Dr. Brian Berman  
Contact: Linda Peterson… (303) 940-9409, Email: lindabebrf@msn.com

**New Mexico**

Saturday, January 18, 2014; 1 p.m.  
Pathway Room-Lower Level, Presbyterian Urgent Care Center, 5901 Harper NE, Albuquerque, NM  
Contact: Al Deguio… (505) 298-6129, Email: deguio@comcast.net

**Seattle, Washington**

Sunday, February 23, 2014; 2 p.m.  
Classroom B, Education Center, James Tower 1st floor, Cherry Hill Campus, Swedish Hospital, 500 17th Ave, Seattle, WA 98122  
Speaker: Dr. Steven Hamilton  
Contact: Peter Bakalor… (206) 219-9053, Email: pbakalor@gmail.com

**Thanks to all the BEBRF Volunteers**

These individuals are serving as District Directors, State Coordinators, Area Representatives and Telephone Contact Persons for the Benign Essential Blepharospasm Research Foundation. We appreciate their hard work and efforts on behalf of the foundation and BEB, Meige and hemifacial spasm patients.

Claudia A Adams  
Peter Bakalor  
Pauline M Barnes  
Shirley Barr  
Barbara Beckett  
Barbara Benton  
Kathy Berg  
Mary Berlow  
Mildred Blackwell  
Sara Jane Brouchoud  
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Renee Capen  
Helen Rose Chestnut  
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Lynn Yarbrough  
Sande Young  
Marie C Zehnder
It is our editorial policy to report on developments regarding BEB/Meige and related disorders but do not endorse any of the drugs or treatments in the Newsletter. We urge you to consult with your own physician about the procedures mentioned.

YOUR INPUT AND PARTICIPATION IS IMPORTANT TO US!

PLEASE CONSIDER:

Volunteering to be a phone contact person in your state.

Starting a new support group in your area.

Writing your story for an issue of the BEBRF Newsletter.

Sending us a “helpful hint” that you would like to share with other patients.

Sending us the contact information on treating physicians that you would recommend to others.

Asking your physician or other professionals to submit an article of interest to BEB/Meige or hemifacial spasm patients for publication in the BEBRF Newsletter.

Telling us about topics you would like to have discussed at a BEBRF Symposium.