When the Board of Directors of the CMTA began planning for the Third International Conference, one thing was certain: the Crohn family would have to be involved. A research meeting of this magnitude, which will bring together more than 200 leading CMT investigators from around the world, is an incredible financial undertaking. CMT, a rare, nonfatal disorder, does not have substantial “commercial value” to major pharmaceutical companies, which therefore made private support all the more critical. The Crohn family insured the viability of the conference early on with personal contributions of $10,000 each and by securing a generous $15,000 grant from the National Foundation for Jewish Genetic Disease.

Advancing research is part of this family’s legacy. Many readers probably first heard the Crohn name because of Crohn’s disease, discovered in 1932 by Frank and George’s uncle Bur- rill B. Crohn. The Crohn brothers’ personal commitment to advancing CMT research goes back 11 years to the Second International Conference held at Arden House, the meeting facility of Columbia University.

In addition to supporting the international conferences, the brothers also have helped underwrite production costs for the first CMT Facts booklet and the publication of Charcot-Marie-Tooth Disorders: A Handbook for Primary Care Physicians. Not only are the Crohns generous with the CMTA, but both support other philanthropic causes as well. Frank has given generously of his time and resources to the underprivileged children of South Florida, and George founded and serves as President of the National Foundation for Jewish Genetic Diseases. Owing to their long service and various leadership roles within the organization, the Crohn brothers must be credited for enabling the CMTA to sponsor this important research conference.
Begin With Flynn

“...to use the background of long experience for the common good as opportunity arises, and to stand ready to aid and counsel those conducting more youthful enterprises...”

—On the plaque presented to the New York Academy of Sciences upon election to full membership in The 100 Year Association of New York.

I saw the above referenced quote while I was in a meeting room at the New York Academy of Sciences which is co-sponsoring the Third International Conference on CMT Disorders. I chuckled a bit after reading the lofty language of the commemorative plaque and learning that there is an association for 100-year-old associations. It has been said that there is an association for everything. I recall learning from my political science courses that the United States is a nation of joiners, as true today as when first observed by De Toqueville more than 150 years ago. Americans have historically come together forming groups with a common purpose, a shared mission. Whether it is Mothers Against Drunk Drivers, a charter school, or City Cares of America, there is a strongly held belief that the combined energies and talents of many will produce greater value and results for their members than will individuals “going it alone.”

The CMTA is no exception. With significant support from friends like the Crohn Brothers, the National Institutes of Health and Athena Diagnostics, we are making a bold leap forward. Despite our “youthful” 15 years, we have undertaken major initiatives that represent a turning point for the association: the Armington Research Challenge and the Third International Conference on CMT Disorders. Together they signify a new leadership role for the CMTA in advancing research. Board President Diane Frenaney likes to say that we are in business to put ourselves out of business. While this is not imminent, it is a great perspective. Unlike most groups, we certainly do not want to become part of the 100-year association!

Athena Diagnostics—A Vital Partner: When board member Ardith Fetterolf met Vincent Bertolino, Neurogenetic Product Manager for Athena Diagnostics, she knew right away he would be an important partner with the CMTA. Athena has blood tests that can determine CMT 1A, HNPP (hereditary neuropathy with liability for pressure palsy), and CMT X, which makes them a logical corporate partner. Athena’s support of the CMTA dates back to their help in underwriting the publication of the Physician’s Handbook. Vince has insured that the working relationship includes financial backing, $20,000 for the Third International Conference, as well as creative and “physical” support.

Combined marketing efforts are underway that involve direct-mail campaigns, newspaper advertising, speaking engagements, and the distribution of literature by Athena’s sales force. Whenever appropriate, Vince has allowed the CMTA to “piggy-back” our message along with what he sends to medical professionals around the country. This kind of communication is vital to our goal of educating doctors about CMT.

NIH/NINDS. The CMTA is fortunate to have received Third International Conference funding from the National Institutes of Health and the National Institute for Neurological Diseases and Stroke. In large measure, this financial support was a result of the excellent grant application submitted by Dr. Michael Shy. He and Dr. Robert Lovelace, chairman of the CMTA Medical Advisory Board, were pleased to learn that the application was among the highest ranked submissions in its “class,” scoring in the 90th percentile. Of course, the efforts of Pat Dreibelbis, CMTA Director of Programs, were influential too. Pat had begun “selling” the conference to Captain Steve Groft, director of the Office of Rare Diseases, when they had met nearly 2 years ago at a NORD conference. Pat’s pleasant lobbying efforts helped secure a $10,000 grant.
Le Chantecler:  
A Cloistered Setting

Cloister: a place of religious seclusion; monastery or convent; also, any place where one may lead a secluded life.

—From Webster’s New World Dictionary.

When Dr. Michael Shy began discussing with his colleagues what type of setting would be most appropriate for the Third International Conference, a cloistered environment was deemed best. Removed from the distractions and noise of a city, the somewhat remote venue afforded by Hotel Le Chantecler will provide quiet and peaceful moments for reflection and serious thought. And while this meeting is certainly not monastic in nature, participants who will be immersed in science would nonetheless welcome divine inspiration...

Its name and its emblem were inspired by the popular play “Chantecler,” written by the French dramatist and poet Edmund Rostand. The hero of this lyrical work, which premiered in Paris in 1910, was a rooster. From its beginnings 60 years ago, Le Chantecler has grown into a unique hotel and conference center, offering a rustic and natural setting while at the same time featuring the modern amenities required for staging a conference. Although it has become known for its resort offerings, the special character and location of Le Chantecler should provide the perfect backdrop for a rigorous scientific meeting like the Third International Conference.

The New York Academy of Sciences has an unmatched reputation for its scientific meetings and publications. The CMTA is very fortunate to be teamed with the Academy in sponsoring the Third International Conference. Founded in 1817, the Academy has over 45,000 members worldwide. In addition to conferences and the publication of Annals, the Academy has a varied portfolio of programs, including the publication of a popular science magazine, science education, public policy and human rights.

The Academy is well-known for the Annals of the New York Academy of Science, a series of scholarly books which presents the proceedings of important conferences and emerging research. Published since 1823, the Annals series are subscribed to by more than 750 libraries around the world and ranks among the top two percent of sources cited in scientific publications. Proceedings from the Conference will be published in the Annals.

After the conference proposal was peer-reviewed, the Academy agreed to sponsor the meetings. “The Academy is deeply interested in the application of basic science to clinical problems,” said Dr. Rashid Shaikh, Director, Science and Technology Meetings at the Academy. “Charcot-Marie-Tooth Disorder has long been a puzzling condition and new developments in molecular and structural biology are providing new insights in CMT. The Academy is very enthusiastic about this conference and the Annals expected to result from it.”

The CMTA Board of Directors and the Medical Advisory Board appreciate the prestige that comes from partnering with the NYAS. The Academy “team” is comprised of Rashid Shaikh, Ph.D., Director, Scientific Meetings, and Kathleen Schrader, Administrative Assistant, Science and Technology Meetings (both will be on-site in Quebec), Renee Wilkerson, Senior Meetings Coordinator and Sue Davies, Manager, Meeting Development.

Nestled between the mountain and lake, with guest rooms facing the water, the Hotel Le Chantecler in St. Adele, Quebec, is among the most beautiful and popular hotels in the Laurentian Mountains.
Charcot-Marie-Tooth Disease (CMT) is the most common inherited peripheral neuropathy in humans, with a prevalence of 1 in 2,500. Considerable advances have occurred recently in our understanding of CMT. The genetic causes of most of the demyelinating forms of CMT have been identified. The molecular biology of peripheral nervous system and the function of many of the molecules involved in the pathogenesis of CMT are becoming clear. The development of viral vectors to introduce genes into the peripheral nervous system, and the identification of trophic factors to promote nerve regeneration and remyelination are making gene therapy for CMT a realistic possibility in the future.

This conference will focus on the latest developments in research and clinical aspects of CMT. It will be of interest to geneticists, molecular biologists, clinicians, morphologists and physiologists, as well as young investigators and other scientists interested in CMT. A strong effort will be made to integrate basic and clinical research and to highlight important areas for future research.
Preliminary Schedule

Wednesday, October 21, 1998 5:00 PM – 8:30 PM
Special Lecture
- The Role of the Axon in Inherited Demyelinating Neuropathy, John Griffin

Thursday, October 22, 1998
7:30 AM – 10:30 PM
Introduction and Welcome
Robert E. Lovelace

Session I. Clinical and Pathological Review of What Constitutes CMT
- Overview of CMT 1A, P. K. Thomas
- Historical and Clinical Aspects of CMT 1B, Thomas D. Bird
- Overview of Hereditary Neuropathy with Liability to Pressure Palsies, Phillip F. Chance
- Molecular Mechanisms for the CMT 1A Duplication and HNPP Deletion, James R. Lupski
- PMP22 Polymorphisms in the African-American Population, Lisa Baumbuch
- X-Linked Charcot-Marie-Tooth Disease, Kenneth H. Fischbeck
- Overview of CMT 2, Jeffery M. Vance
- Recressive CMT 4B, Antonio Gambardella
- The Autosomal Recessive Form of CMT Linked to Sq, Eric LeGuern
- Molecular Genetics of Distal Hereditary Motor Neuropathy Type II, Vincent Timmerman
- Autosomal Recessive Motor Neuropathy: Clinical Aspects and Molecular Genetics, Lefkos T. Middleton
- A Clinical Review of CMT, Carlos A. Garcia
- Clinical and Pathological Features of Hereditary Sensory Neuropathies, Peter J. Dyck

Session II. Pathogenesis of Demyelination in CMT
A) Schwann Cell-Axonal Interactions and the Program of PNS Myelination
- The Schwann Cell Program of Myelin Gene Expression: Its Relevance to CMT, John Kamholz
- Autosomal Dominant Form of Juvenile Amyotrophic Lateral Sclerosis, David Cornblath
- Schwann Cell Precursors and the Development of Myelinating Schwann Cells, Kris Jessen
- Role of Extracellular Matrix and Integrins in PNS Myelination, Maria Laura Feltri
- The Role of Ras Signaling in Schwann Cells, Gihan Tennekoon
- A Novel Molecular Phenotype in Schwann Cells of PO Knockout Mice, Daniela Menichella

B) Molecules Involved in the Pathogenesis of CMT 1
- PMP22 as a Schwann Cell Myelin Protein, B. Jackson Snipes
- Structural Studies on Adhesion Molecules in Myelination, David R. Colman
- Correlation of CMT 1B-Associated Mutations with Deficits in the In Vitro Adhesiveness/Functioning of PO Protein, Maria T. Filbin
- Connexin 32 in the PNS Functional Analysis of CMTX Mutations and Implications for the Disease, Roberto Bruzzone
- Location and Function of PLP and DM-20 in Schwann Cells, Wendy Macklin

C) Schwann Cell Cultures and Studies of Myelination
- Signals that Regulate Schwann Cell Development and Myelination, Rhona Misky
- Lessons on Myelination from Mouse Schwann Cell Mutations, Nancy Ratner
- IL-8 Secretion by Human Schwann Cells and its Role in Leukocyte Recruitment, J. Lynn Rutkowski
- The Biology of Chronically Denervated Schwann Cells, Susan Hall
- Nodes, Paranodes and Incisures: From Form to Function, Steven Scherer

D) Mouse Models in Studies of CMT
- Heterogenous Phenotypes Associated with Mutations of the PLP Gene: Implications for PMP22-Related Disorders, Ian Griffiths
- Transgenic Mouse Models of CMT 1A and HNPP, Ueli Suter
- PMP-22 Overexpression in Rat, Klaus-Armin Nave
- Trembler and TremblerJ As Mouse Models of CMT 1A?, Bertrand Garbay
- Nerve Conduction Abnormalities and Neuromyotonia in Genetically Engineered Mouse Models of Human Hereditary Neuropathies, Klaus V. Toyka
- Progressing Peripheral Neuropathy in PO-Deficient Mice, Rudolf Martini
- PO Gene Dosage and Mouse Models of CMT 1B, Lawrence Wrabetz
- Characterization of Targeted Connexin32 Deficient Mice—A Model for CMTX, Klaus Willecke

Friday, October 23, 1998
8:30 AM – 10:00 PM
Session II. Pathogenesis of Demyelination in CMT (Cont’d)

E) Human Studies on CMT 1 Biopsies
- Schwann Cell Regulation and Targeting of PMP22—New Vistas on Pathomechanism, Hans Werner Mueller
- Immuno EM Studies in Nerve Biopsies of Hereditary Sensory-Motor Neuropathies, Jean-Michel Vallat

Session III. Genotype/Phenotype Correlation in CMT
- Clinical and Electrical Distinctions Between Inherited and Acquired Demyelinating Neuropathy, Austin J. Sumner
- Clinical and Neurophysiological Features of CMT 1A, J.E. Hoogendijk
- Pathological Features in Human Nerve Biopsies Caused by Different Mutational Mechanisms of PMP22: Duplication, Deletion, Missense Mutations, Frame Shift Mutations, Anneke Gabriëls-Festen
- Phenotypic Variability in Patients with Hereditary Neuropathy with Liability to Pressure Palsy (HNPP), Gareth J. Parry
- Demyelinating Peripheral Neuropathy Due to Proteolipid Protein Gene Mutations, James Garbern
- Genotype/Phenotype Correlations in CMTX, Angelika F. Hahn

Session IV. Diagnosis and Therapy of CMT
- Appropriate Genetic Testing in Suspected CMT Patients, Garth Nicholson
- Diagnostic Testing in CMT: Approaches and Results, Peter De Jonghe
- Introducing Genes into Schwann Cells with Viral Vectors, Michael Shy
- Potential Use of Trophic Factors in the Treatment of Hereditary Neuropathy, Anthony Windelspecht
- Role of IGF-1 in Peripheral Nervous System Regeneration, Eva L. Feldman
- Abnormal Schwann Cell-Axon Interactions in CMT Neuropathies: Effects of Mutant Schwann Cells on the Axonal Cytoskeleton and Regeneration Associated Myelination, Zarife Sahenk
- Aberrant Expression of Trophic Factors in Trembler Schwann Cells, Garth Bracey

Saturday, October 24, 1998
8:30 AM – 5:00 PM
Session II. Pathogenesis of Demyelination in CMT (Cont’d)

Session V. Pathology of CMT
- Structural Studies on Adhesion Molecules in Myelination, Correlation in CMT, Jean-Michel Vallat
- Schwann Cell Regulation and Targeting of PMP22, H. Martini

Session VI. Diagnosis and Therapy of CMT
- Appropriate Genetic Testing in Suspected CMT Patients, Garth Nicholson
- Diagnostic Testing in CMT: Approaches and Results, Peter De Jonghe
- Introducing Genes into Schwann Cells with Viral Vectors, Michael Shy
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October 21–24, 1998, Hotel Le Chantecler, Sainte-Adele, Quebec, Canada

ATTENTION READERS:
Unlike our patient/family conferences which are open to members and guests, this is a scientific meeting specifically planned for CMT researchers.
Looking Back: Second International Conference on CMT Disorders

June 28–July 1, 1987, Arden House, NY, The International Conference Center of Columbia University

Research highlights from the conference are presented below. (Editor’s Note: Hereditary motor and sensory neuropathy [HMSN] and CMT are used interchangeably.)

Anita Harding, M.D.: There are two main groups of cases, one in which motor nerve conduction velocity (MNCV) is less than 38 m/s and another in which MNCV is normal or only slightly reduced. These two groups are referred to as hereditary motor and sensory neuropathy (HMSN) types I and II. Ataxia, areflexia, weakness of the upper limbs, and skeletal deformity are more common in type I than type II.

Ludwig Gutmann, M.D.: Marked slowing of motor nerve conduction velocities (MNCV) is the physiological hallmark of HMSN-I. The maximal slowing of MNCV occurs by age 3–5 years and appears to remain stable thereafter despite progression of the illness. Decrease in muscle action potential amplitude more accurately reflects the progression of clinical symptoms and signs.

Peter James Dyck, M.D.: Epidemiologic surveys have probably underestimated the prevalence of inherited neuropathy because many cases remain incorrectly diagnosed or undiagnosed. It remains the leading cause of undiagnosed neuropathy referred to this investigator and may be the third most frequent cause of neuropathy in the USA after mechanical injury, compression and entrapment, and diabetes. More satisfactory classification must await much more intensive gene localization and characterization studies now in progress.

Phil Chance, M.D.: Studies of genetic linkage in 14 families with classic HMSN type I showed strong evidence for genetic heterogeneity in the disorder. We concluded that there is one form of HMSN type I linked to the Duffy locus on chromosome 1 and one or more mutations producing the same phenotype, but not linked to Duffy. The type linked to Duffy may be less common.

Roger Lebo, Ph.D.: The CMT syndrome loci are in at least three locations: near the centromere on chromosome 1, on the proximal long arm of the X chromosome, and on another autosome. In order to further characterize CMT disease, we are beginning to study families with the disease locus linked to the Duffy blood group locus on human chromosome 1.

Kenneth Fischbeck, M.D.: X-linked hereditary neuropathy has been reported for nearly 100 years but has not been completely accepted until recently. A long-term follow-up study of a large North Carolina family found no male-to-male transmission in 28 chances, making autosomal dominant transmission exceedingly unlikely. We did a linkage analysis of this family and seven others with X-linked neuropathy to localize the gene defect on the X chromo-

some. Nerve conduction velocities are about 25–40 m/sec, intermediate between the type I and II forms of hereditary neuropathies in the traditional classification.

Stanley Myers, M.D.: There is no uniform management of patients with HMSN. Patients must be evaluated and treated on an individual basis. A rationale for management is possible. There is little in the literature today that scientifically evaluates the results of therapeutic physical means of treatment in patients with CMT. Studies have been done on patients with other neuromuscular disorders assessing the beneficial and/or harmful effects of exercise; energy sparing effects of dynamic bracing; and the value of orthotics in preventing contractures. Some carry over from these studies can be made to those patients with CMT disorders.

Robert Clark, M.D.: The neuropathic foot is a typical deformity consisting of clawed toes, high arch, and a varus position of the heel. Also known as pes cavus, this characteristic deformity may be seen in CMT disease, Friedreich’s ataxia, Roussy-Levy syndrome, spinal muscular atrophy, etc. In most HMSNs, the muscles initially affected are the intrinsic muscles of the foot. With these muscles absent, the long flexors and long extensors can manifest their pure functions. As these muscles are strongest in the great toe, their actions will be exaggerated in this digit. The result is a bowstringing effect that combines to depress the first ray of the foot. In the non-weight-bearing position, this will tend to raise the arch by approximating the forefoot to the heel. This will result in a contracture of the planter fascia. In the weight-bearing position, it will tend to shift the weight to the lateral aspect of the foot and rotate the heel into the varus position. Initially flexible, the deformity eventually becomes fixed and the bones deformed. Early measures may prevent fixed deformity.

John Hsu, M.D.: Young CMT patients have minimal functional disability, but can have a relatively early manifestation of ankle and foot weakness leading to pes cavus, equinus, equinovarus, or other foot deformities. Treatment is needed to prevent the formation of permanently fixed deformities, pain, and early loss of function. Soft tissue procedures include tendon lengthenings, tendon transfers, and plantar fascia releases. Bone operations consist of triple arthrodesis, extra-articular subtalar arthrodesis, calcaneal-cuboid fusion, and dorsal wedge osteotomies. Surgical intervention may be necessary to prevent or treat fixed deformities, relieve pain and improve function.

* “Duffy” refers to a region on chromosome 1 where linkage was found for a large family group. For some time, researchers believed all type I CMT might locate there, but only a small percent of cases do locate there, causing the version called CMT type IB, which results from mutation of the PO gene.
Q&A with Dr. Michael Shy

CMTA: Dr. Shy, the goal of the Second International Conference on CMT Disorders was “to provide a current evaluation of the CMT group of hereditary neuropathies” and to provide a follow-up of the first conference held in France. As a co-chair for the meeting, what are your goals for the Third International Conference?

Dr. Shy: The goal of the meeting is to bring together distinguished investigators from different disciplines of CMT-related research and place them in a cloistered setting to promote meaningful interactions between presenters and other conference participants. Since 1987, there have been few, if any, extended meetings in which geneticists, molecular biologists, physiologists, and clinicians have had the opportunity to converse and exchange ideas.

CMTA: You alluded to the “setting” in your last response. Can you comment on why the meeting is taking place in a somewhat remote location?

Dr. Shy: The Chantecler Hotel in Quebec, Canada, was chosen for a variety of reasons. The meetings of the American Neurological Association are taking place October 18–21 in Montreal. Since some presenters will be attending those meetings, it made sense to hold our conference afterwards. We’re moving to a more “cloistered” setting to create an environment where participants will be immersed in the subject matter without the distractions of a big city.

CMTA: Please tell us about some of the scientists who will be making presentations during the first session: Clinical and Pathological Review of What Constitutes CMT.

Dr. Shy: Professor P. K. Thomas has made a career studying peripheral nerve disease and, along with Anita Harding, wrote the seminal work on distinguishing the phenotypes of CMT I and CMT II. Dr. Thomas Bird was the first to identify families with what is now known as CMT I. Dr. Philip Chance was the first to demonstrate the deletion on chromosome 17 associated with hereditary neuropathy with liability for pressure palsy (HNPP), and Dr. James Lupski not only identified the duplication on chromosome 17 causing CMT IA, but also demonstrated that the duplication takes place at a recombination hotspot located near a mariner transposon-like element. Dr. Kenneth Fischbeck's group identified mutations in connexin-32 as the cause of CMTX. Dr. Jeffrey Vance’s lab is close to identifying the gene causing at least one form of CMT II. Finally, Dr. Peter Dyck is an international authority on peripheral nerve pathology, as is Dr. Jack Griffin, who has done pioneering work on axonal changes secondary to Schwann cell abnormalities.

CMTA: Can you tell us about some of the foreign investigators?

Dr. Shy: Dr. Ueli Suter, of the Swiss Federal Institute of Technology in Zurich, Switzerland, was the first to identify peripheral myelin protein (PMP)-22, and a mutation in PMP-22 as the cause of the Trembler mouse model of CMT. He has now generated a transgenic rat which over-expresses PMP-22 and develops a neuropathy that is morphologically quite similar to CMTIA. He will be presenting in the session on mouse models in studies of CMT.

Dr. Christine Van Broeckhoven, University of Antwerp, Belgium, was one of the first to identify the duplication in chromosome 17 as the cause of CMTIA and her lab is pursuing new genetic tests for CMT. She also founded the HMSN (hereditary motor/sensory neuropathy) European Consortium to bring together researchers to share their findings. She is presenting in the “Diagnosis and Therapy” session of the conference.

There are many other renowned scientists from around the world who will make presentations as well, and their work will be published in the *Annals of the New York Academy of Science*.

CMTA: How many researchers are you expecting for the conference?

Dr. Shy: We’ve designed the meeting to be small—not more than 225 investigators. In addition to the presenters and other senior investigators, a special effort is being made to encourage younger investigators to contribute posters and participate in the meeting. It’s essential that we promote the work of junior researchers and cultivate their interest in studying CMT.

CMTA: We know that you have invested an incredible amount of time in organizing this meeting. What do you hope will be gained from this gathering of investigators?

Dr. Shy: While there will be untold value from the networking alone, there are two primary expectations. First, that the multidisciplinary context of the meeting will engender “cross-fertilization” in the research approach of investigators. Secondly, that the resulting publication from the New York Academy will serve as the premier resource on state-of-the-art CMT research.
Armington Research Challenge Met: $151,119 Raised!!!

Special thanks to the hundreds of members and friends who contributed to the successful completion of the $150,000 Armington Research Challenge for 1998. We are very grateful for the leadership support of the those donors who contributed $500 or more toward this effort. We are pleased to acknowledge those individuals listed below.

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The CMTA Report

Information on Charcot-Marie-Tooth Disorders from the Charcot-Marie-Tooth Association

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