



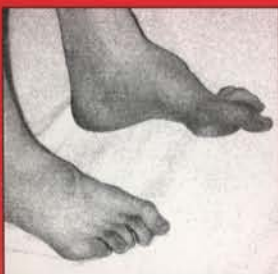
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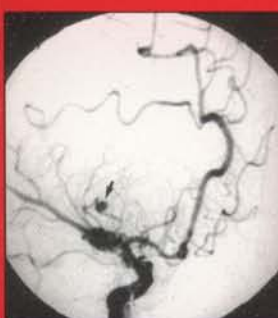
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Sciences Neurologiques

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Charcot-Marie-Tooth
Disease



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¹ PRISMS (Prevention of Relapses and Disability by Interferon B-1a Subcutaneously in Multiple Sclerosis) Study Group (1998). Randomised double-blind placebo-controlled study of interferon B-1a in relapsing/remitting multiple sclerosis. *Lancet* 352:1498-1504.



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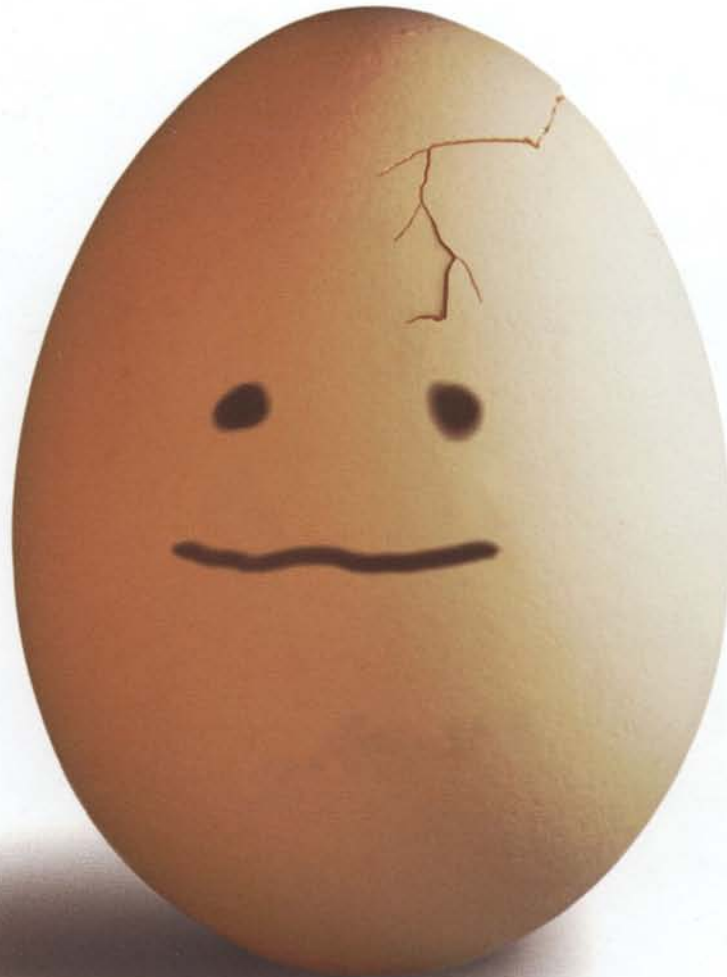
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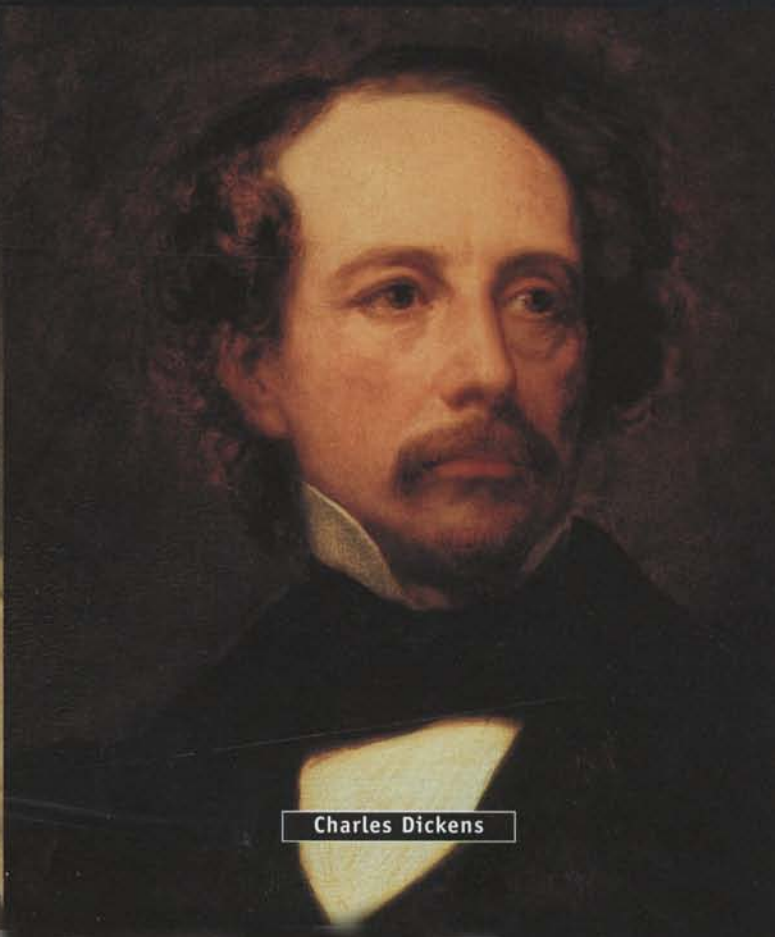


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[‡] Open label trial for children (n=72) treated for ≥ 3 months. Average dose of 10 mg/kg/day.


[§] CNS adverse events: Somnolence (30.1%), dizziness (28.3%), ataxia (21.2%), speech disorders (16.8%), psychomotor slowing (16.8%), nystagmus (15.0%), paresthesia (15.0%), nervousness (15.9%), difficulty with concentration/attention (8.0%), confusion (9.7%), depression (8.0%), anorexia (5.3%), language problems (6.2%) and mood problems (3.5%). In an audit of 1446 adults and 303 children, there appeared to be a similar pattern of adverse events.

^{**} The long-term effects of weight loss in pediatric patients are not known.



^{††} Limited use benefit: Ontario, Nova Scotia, New Brunswick, PEI. Full benefit: Quebec, Saskatchewan, British Columbia, Alberta, Manitoba.

Please refer to the TOPAMAX Prescribing Information for complete prescribing details.

REFERENCES: 1. TOPAMAX[®] topiramate Tablets and Sprinkle Capsules Product Monograph, May 11, 1999. 2. Kamin M, Kraut L, Olson W. Dose optimization of topiramate as add-on therapy in adults with treatment-resistant partial-onset seizures *Neurology* 1999;52 (Suppl 2):A525-526. 3. Glauser TA, Elterman R, Wyllie E et al. Open label topiramate in paediatric partial epilepsy *Epilepsia* 1997;38 (Suppl 3):94. 4. Rosenfeld WE et al. Topiramate and concomitant weight loss. *Epilepsia* 1997;38 (Suppl 8):98.

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In Canada,

IVIg is

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5 indications.^{1,2,3*}

Why are

Doctors using it

for approximately 100?⁴

* Idiopathic thrombocytopenic purpura,^{A,B} primary humoral immune deficiency^{A,B,C} and secondary immune deficiency,^C allogeneic bone marrow transplant,^A pediatric AIDS^B and B-cell chronic lymphocytic leukemia.^B A Gamimune[®] B Gammagard[®] C Iveegam Immuno[®]

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39052/05-2002

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In making the decision to use IVIG (Intravenous Immune Globulin) in specific indications, Canadian physicians rely on tools such as the published literature and/or available guidelines. The development of guidelines for the appropriate use of IVIG was sponsored by Bayer, who provided an unrestricted educational grant, including extensive literature review and specialist consultation.^{4,5}

A recent survey found that over 80% of IVIG use in Canadian hospitals is in keeping with these consensus guidelines for appropriate use.⁶ But 80% is not 100%.

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And because physicians are the key, we also sponsor two Immunology Fellowships (one in co-operation with CIHR) worth \$250,000, and we develop and offer CME promoting appropriate use through Canadian hospitals. For more information about the Appropriate Use of IVIG, contact Bayer at gamimune.canada@bayer.com



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1. Gamimune Prescribing Information, 2001. 2. Gammagard Prescribing Information, 2001. 3. Iveegam Prescribing Information, 2001. 4. The Consensus Working Group. Present and Future Uses of IVIG: A Canadian Multidisciplinary Consensus-Building Initiative. *Canadian Journal of Allergy & Clinical Immunology* 1997; 2(5): 176-208. 5. Brill V, Allenby K, Midroni G, O'Connor PW, Vajsar J. IVIG in Neurology – Evidence and Recommendations. *Can J Neurol Sci* 1999;26(2): 139-152. 6. Hanna K, Poulin-Costello M, Preston M, Maresky N. Intravenous Immune Globulin (IVIG) Utilization in Canada. Submitted. BP080-0601E

25 Years Ago in the Canadian Journal of Neurological Sciences

Quebec Cooperative Study of Friedreich's Ataxia Phase One: A Prospective Survey of 50 Cases

Organized and Edited by André Barbeau

DESIGN OF THE INVESTIGATION

A. Barbeau

SUMMARY: The general outline of the complete prospective study of 50 cases of spinocerebellar degeneration is given. The general protocol followed, the criteria for inclusion and the mode of analysis are described. The aim of this study was to establish a base of clinical, physiological and biochemical facts upon which a logical and systematic approach to pathogenesis and treatment of Friedreich's ataxia could be attempted.

Can. J. Neurol. Sci. 1976;4:271

NICOLAUS FRIEDREICH AND DEGENERATIVE ATROPHY OF THE POSTERIOR COLUMNS OF THE SPINAL CORD

F. Andermann

SUMMARY: A short outline is given of the pioneer efforts of Nicolaus Friedreich in the description of the spinocerebellar degeneration which now bears his name.

Can. J. Neurol. Sci. 1976;4:275

CLINICAL DESCRIPTION AND ROENTGENOLOGIC EVALUATION OF PATIENTS WITH FRIEDREICH'S ATAXIA

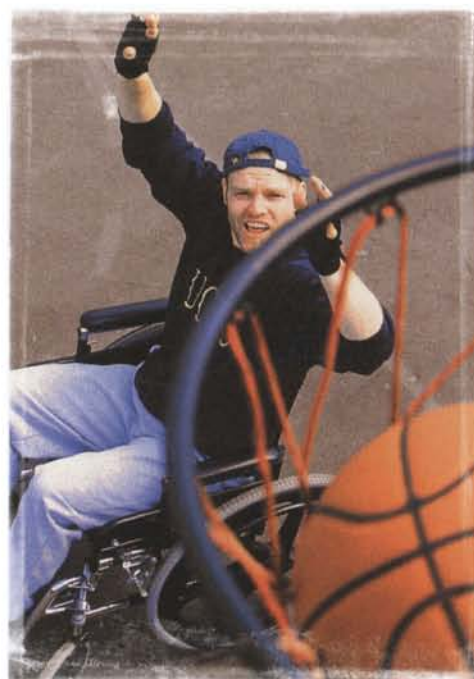
G. Geoffroy, A. Barbeau, G. Breton, B. Lemieux, M. Aube, C. Leger and J.P. Bouchard

SUMMARY: The 50 patients in this survey were classified by a panel of neurologists into four clinical subgroups: Group Ia ("typical" Friedreich's ataxia, complete picture), Group Ib ("typical" Friedreich's ataxia, incomplete picture), Group IIa ("atypical" Friedreich's ataxia, possible recessive Roussy-Levy syndrome), Group IIb (heterogeneous ataxias). The clinical symptoms and signs were analyzed for each of these groups. A constellation of signs constantly present in Friedreich's ataxia and obligatory for diagnosis was described. Other important symptoms, such as the Babinski sign, kyphoscoliosis and pes cavus were found to be progressive, but not essential for the diagnosis at any given time. Finally, a host of other symptoms can only be called accessory. The progression of scoliosis was found to be an important tool in the differential diagnosis of ataxias. Our study also indicates, in contrast to the opinion of some authors, that absent deep tendon reflexes in the lower limbs and early dysarthria are essential in "typical" Friedreich's ataxia.

Can. J. Neurol. Sci. 1976;4:279

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In multiple-dose, placebo-controlled studies, the most frequently reported adverse events included dry mouth (49%), sedation/somnolence (48%), asthenia (weakness, fatigue and/or tiredness) (41%) and dizziness (16%).⁴ The most common adverse events leading to discontinuation of therapy were asthenia (3%), somnolence (3%) and dry mouth (3%).⁵ Sedation may be additive when Zanaflex is taken in conjunction with drugs or substances that act as CNS depressants. Caution is advised when treatment is used in patients who have a history of orthostatic hypotension or are receiving concurrent antihypertensive therapy. Monitoring of aminotransferase levels is recommended during the first six months of treatment, and periodically thereafter, based on clinical status.

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e n t ê t e

Pour passer à la monothérapie après une polythérapie, dans le traitement de diverses crises épileptiques.

Lors d'essais cliniques, on a pu constater que le passage de la polythérapie à la monothérapie par LAMICTAL peut maintenir ou même améliorer la maîtrise d'un vaste éventail de crises épileptiques*. La monothérapie par LAMICTAL a été généralement bien tolérée¹. Les effets indésirables les plus souvent associés à l'abandon de la monothérapie par LAMICTAL ont été l'éruption cutanée (6,1 %), l'asthénie (1,1 %), la céphalée (1,1 %), la nausée (0,7 %) et les vomissements (0,7 %).

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LAMICTAL est le premier et le seul parmi les nouveaux antiépileptiques[†] qui est indiqué comme traitement d'appoint chez les enfants et les adultes atteints du syndrome de Lennox-Gastaut. LAMICTAL offre une maîtrise significativement supérieure[‡] d'un vaste éventail de crises épileptiques associées au syndrome de Lennox-Gastaut[§], y compris les crises majeures, les effondrements épileptiques et les crises tonico-cloniques. LAMICTAL a démontré peu d'effets indésirables^{¶**††} sur le SNC. Il a été associé à une amélioration de la fonction neurologique et des facultés cognitives, comme le comportement, la parole et la communication non verbale[‡].

* Veuillez consulter la monographie du produit afin d'ajuster les doses de LAMICTAL selon l'antiépileptique concomitant faisant l'objet du retrait.

† Lamotrigine, gabapentine, vigabatrine et topiramate (à distinguer des antiépileptiques standards).

‡ Comparativement au placebo.

§ À l'exception des absences épileptiques atypiques.

¶ Les effets indésirables fréquemment signalés étaient : pharyngite (14 %), infection (13 %), vomissements (9 %) et éruptions cutanées (9 %).

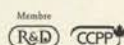
** Dans de rares cas, des éruptions cutanées graves, y compris le syndrome de Stevens-Johnson et l'épidermolyse nécrosante suraiguë (syndrome de Lyell), ont été signalées. Bien que la majorité des patients se soient rétablis après le retrait du médicament, certains ont présenté des séquelles irréversibles et il y a eu de rares cas de décès associés.

†† NE PAS DÉPASSER la dose initiale de lamotrigine ni l'augmentation posologique graduelle qui sont recommandées. Une augmentation plus rapide de la dose initiale a été associée à une fréquence accrue de réactions dermatologiques graves.

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COPAXONE® has a side effect profile that compares to placebo and is an excellent choice to start with.

In clinical trials, only 8% of 844 patients discontinued treatment due to an adverse event.[†] The most commonly observed adverse events associated with the use of COPAXONE® in controlled clinical trials which occurred at a higher frequency than placebo were[†]:

Adverse Event	Non-interferon COPAXONE® [†]	Placebo
Injection Site Reactions [†]	2.4% - 66.4%	0.0% - 36.5%
Asthenia	64.8%	61.9%
Hypertonia	35.2%	29.4%
Vasodilatation	27.2%	11.1%
Back Pain	26.4%	22.2%
Chest Pain	26.4%	10.3%
Arthralgia	24.8%	17.5%
Nausea	23.2%	17.5%
Pain (Neck)	12.8%	7.1%
Infection (Vaginal Moniliasis)	12.8%	7.1%
Agitation (Anxiety)	5.6%	3.2%

[†] Depending on reaction.



COPAXONE® is an excellent choice for the long-term (2 years), too.

COPAXONE® is supported by long-term evidence of up to 2 years and efficacy that's been demonstrated in 6 clinical studies.²⁻⁷ A correlation between a reduction in attack frequency and a decreased risk of future disability remains to be established. The safety and efficacy of COPAXONE® beyond two years have not been adequately studied in placebo-controlled trials.

COPAXONE® is an antigenic substance and thus, it is possible detrimental host responses can occur with its use.

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Shared Solutions™ helps support patients through early treatment and in the long-term (January 1999-March 2000 data).¹¹ With Shared Solutions™, 93% of COPAXONE® patients stayed on therapy.⁸

¹¹n=1377

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Open to all senior neurology residents,
PGY3 to PGY5.

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October 20-21, 2001**

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at Queen Square, London, UK
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Supported by an unrestricted
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Arrangements for this course, including travel and lodgings, can be made through the CCNS Secretariat. Tel: (403) 229-9544 Fax: (403) 229-1661 E-mail: brains@ccns.org

4TH ANNUAL NEUROLOGY RESIDENTS HEADACHE COURSE

Friday October 19, 2001

19:30-22:00 Welcome Reception

Saturday October 20, 2001

07:00 Breakfast

07:30-08:00 Pretest

08:00-08:30 Headache Classification and
Epidemiology

08:30-08:40 Questions

08:40-09:25 Imaging Headache – State of the Art
2001

09:25-09:40 Questions

09:40-10:00 Coffee Break

10:00-10:45 Headache History Taking – Patient
Based

10:45-12:15 Case-Based Session (Migraine)

12:15-12:45 Resident Summaries of Case Discussions

12:45-13:45 Lunch

13:45-15:45 Chronic Daily Headache and Medication
Induced Headache

15:45-16:00 Break

16:00-17:30 Case-Based Session (Secondary
Headaches)

17:30-17:55 Resident Summaries of Case Discussions

18:00 Adjourn

19:00 Reception and Dinner

Sunday October 21, 2001

08:00-08:20 Diagnosis/Investigation

08:20-08:30 Questions

08:30-09:15 Migraine Therapy – Acute and
Preventative

09:15-09:35 Tension Type Headache Therapy

09:35-09:45 Questions

09:45-10:00 Break

10:00-10:40 Case-Based Session (Trigeminal
Autonomic Cephalalgias)

10:40-11:00 Therapy of Trigeminal Autonomic
Cephalalgia

11:00-11:30 Posttest

11:30-12:00 Burning Issues

12:00 Certificate Ceremony

Notes:

- This course will combine primary and secondary headaches in one year.
- We will emphasize imaging this time but this of course will include some neurobiology.
- The headache history will be new and could be a case on video or a live patient.
- There will be more case based sessions, and feedback from the residents.
- There will be a pretest and a posttest again.



For more information please contact the Canadian Congress of Neurological Sciences
Tel: (403) 229-9544; Fax: (403) 229-1661; E-mail: brains@ccns.org

25 Years Ago in the Canadian Journal of Neurological Sciences

GENETIC AND FAMILY STUDIES IN FRIEDREICH'S ATAXIA

E. Andermann, G.M. Remillard, C. Goyer, L. Blitzer, F. Andermann and A. Barbeau

SUMMARY: This study consists of two parts: 1. A detailed genetic analysis of 35 sibships in which 58 individuals were affected with Friedreich's ataxia; and 2. Clinical and laboratory examinations of parents and siblings, in an attempt at carrier detection and diagnosis of the preclinical state.

The increased parental consanguinity, the lack of affected individuals in other generations, and the lack of significance of extrinsic etiological variables, all suggested an autosomal recessive mode of inheritance, and this was confirmed by formal genetic analyses, employing several different methods.

Associated abnormalities in our series of 58 patients included cardiomyopathy (51.7%), diabetes mellitus (19.0%), optic atrophy (5.2%), nerve deafness (5.2%) and congenital malformations (6.9%). The incidence of diabetes mellitus, congenital malformations, and epilepsy and/or febrile convulsions was elevated in first degree relatives of patients with Friedreich's ataxia.

Examinations in first degree relatives revealed an increased frequency of neurological and skeletal abnormalities (26.3%), but no abnormalities on neuro-ophthalmological examination. Frequent EMG abnormalities were noted in parents (56.3%), but not in siblings; and these could usually be attributed to extrinsic causes. There was an increased incidence of ECG abnormalities in both parents (50.0%) and siblings (25.0%) and some of these abnormalities may represent cardiomyopathy. An increased frequency of EEG abnormalities was also recorded in parents (14.3%) and siblings (27.2%), but these were not specific. None of these examinations resulted in a practicable method of carrier detection or preclinical diagnosis.

Since carrier detection is still not feasible, genetic counselling remains the only possible means of prevention of Friedreich's ataxia.

Can. J. Neurol. Sci. 1976;4:287

FRIEDREICH'S ATAXIA: PRELIMINARY RESULTS OF SOME GENEALOGICAL RESEARCH

A. Barbeau, M. Le Siege, G. Breton, R. Coallier and J.P. Bouchard

SUMMARY: A preliminary genealogical investigation of all the known ancestors from the year 1608 of four apparently unrelated French Canadian kindreds with Friedreich's ataxia reveals that the original ataxia gene in the province of Quebec was present within a core of no more than 10 families living in Quebec City in the mid-1600s.

Can. J. Neurol. Sci. 1976;4:303

FRIEDREICH'S ATAXIA: OBSERVATIONS WITH Q AND G BANDING OF HUMAN CHROMOSOMES

M. Cadotte, A. Barbeau and G. Breton

SUMMARY: No chromosomal anomaly was found in 15 cases of typical Friedreich's ataxia and three cases of atypical recessive ataxia studied with Q and G banding techniques. No difference in frequency of chromosomes gaps or breakages was noted amongst patients with Friedreich's ataxia and controls.

Can. J. Neurol. Sci. 1976;4:307

Now we can celebrate the long-term benefits in the treatment of Alzheimer's disease with once-a-day Aricept*.



There's cause for celebration—because Aricept* has been shown to result in improvement or stabilization in 80% of Alzheimer's disease patients over 6 months of treatment.^{1‡} And long-term data shows that Aricept*-treated patients continued to show treatment benefits up to 3 years on cognition and global functioning compared to data expected from untreated patients.^{2§} What's more, Aricept* has demonstrated long-term safety and tolerability profiles.^{2†} All of which means there's even more reason to make Aricept* your standard of care.³

Aricept* does not change the underlying course of the disease. Aricept* is indicated for the symptomatic treatment of patients with mild-to-moderate dementia of the Alzheimer's type.

† With appropriate dose escalation 5 mg/day dose, 10 mg/day dose and placebo were shown to have comparable adverse events. Most common adverse clinical events with Aricept*: diarrhea, nausea, insomnia, fatigue, vomiting, muscle cramps and anorexia. These events are usually mild and transient, resolving with continued Aricept* treatment without need for dose modification.

‡ In a 24-week, double-blind, placebo-controlled study, 473 mild-to-moderate AD patients were randomized to receive Aricept* 5 mg/day, 10 mg/day or placebo. The mean difference for Aricept*-treated patients (10 mg/day) vs. placebo was -2.87 ± 0.63 ($p < 0.0001$) units in ADAS-cog, 0.47 ± 0.11 ($p < 0.0001$) units in CIBIC-plus, and 0.59 ± 0.17 ($p = 0.0007$) units in CDR-SB.

§ In a 162-week, multicentre, open-label extension study, 579 patients who had previously completed a randomized, double-blind, placebo-controlled study with Aricept* were treated with Aricept* 5 mg which could be increased to 10 mg between weeks 6 and 24, as per clinician's judgement. At study endpoint, ADAS-cog declined 15.57 points (95% CI, 12, 19.2) vs. the estimated decline of 6-12 points per year in untreated patients.

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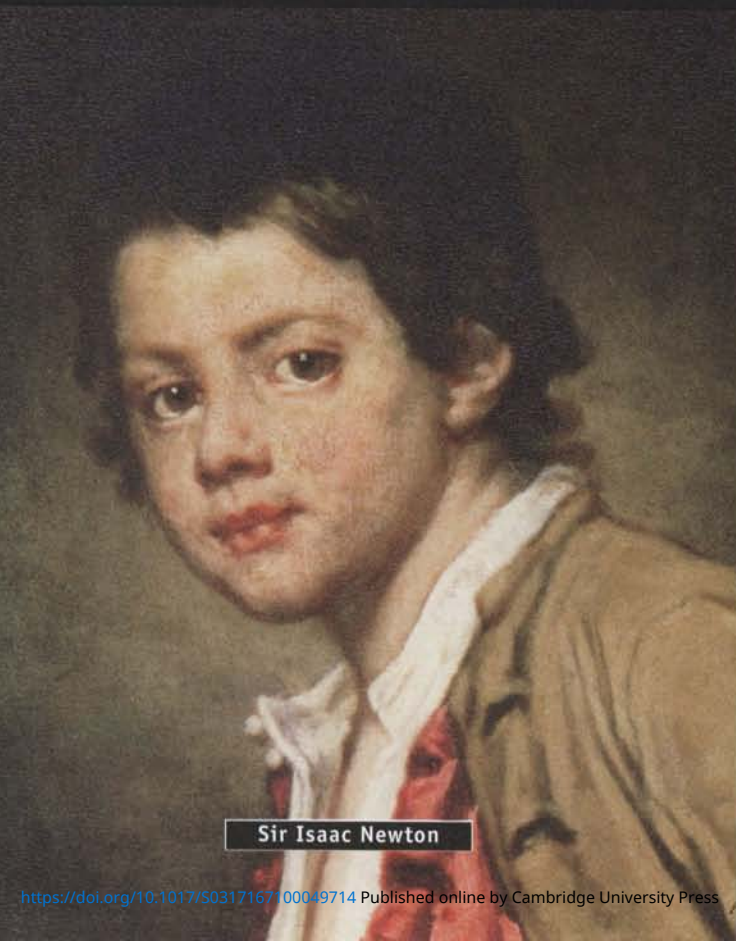


Vincent Van Gogh

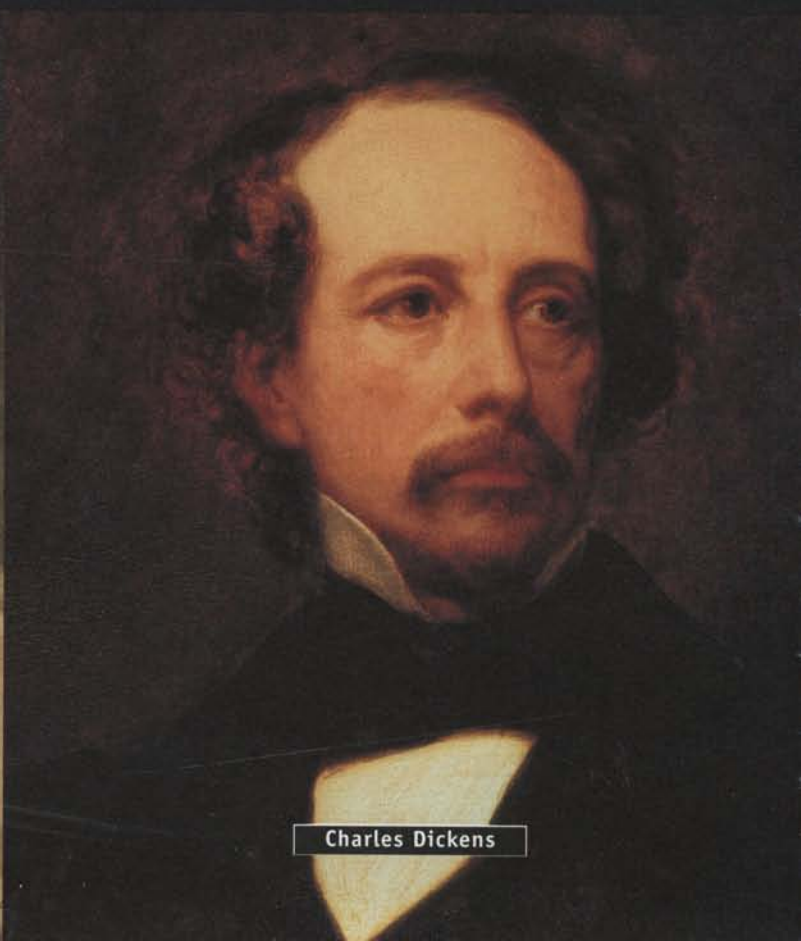


Jeanne d'Arc

**AUPARAVANT, LES PERSONNES ÉPILEPTIQUES DEVAIENT
SE MONTRER EXCEPTIONNELLES POUR RÉUSSIR.**



Sir Isaac Newton



Charles Dickens

EFFICACE CONTRE UN GRAND NOMBRE DE TYPES DE CRISES.

- TOPAMAX est efficace contre les crises partielles initiales, les crises tonico-cloniques primaires généralisées et les crises associées au syndrome de Lennox-Gastaut¹
- Des résultats souhaitables avec absence totale de crises chez 19 % des adultes¹ et 22 % des enfants¹ atteints de crises partielles initiales^{2,3}

AUCUN SIGNE D'EFFETS SECONDAIRES CAPABLES DE MENACER LE PRONOSTIC VITAL.

- Comme pour la plupart des antiépileptiques, les effets secondaires le plus fréquemment signalés relèvent du SNC et sont généralement légers à modérés et de nature passagère^{5,1}

IL EST POSSIBLE QUE LES PATIENTS ADULTES SUBISSENT UNE PERTE DE POIDS.

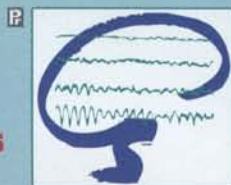
- 73 % (n = 52) des patients ont subi une perte de poids de 5,97 lb en moyenne (Analyse provisoire. Durée moyenne de 60 jours)⁴
- 96 % des enfants traités dans le cadre des essais cliniques pendant au moins un an et ayant subi une perte de poids ont repris du poids au cours de la période d'exécution des essais^{6,1}

AUJOURD'HUI, IL Y A TOPAMAX.

UNE POSOLOGIE BIQUOTIDIENNE POUR TENIR COMPTE DU PATIENT.

- Le traitement par TOPAMAX peut être commencé et ajusté selon la réponse clinique quel que soit le traitement anticonvulsivant en cours
- Les comprimés sont inscrits au formulaire^{††}

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À SAUPOUDRER**



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Comprimés et capsules à saupoudrer [®]TOPAMAX* (topiramate) : indiqués comme traitement adjuvant chez les patients (adultes et enfants âgés de deux ans ou plus) atteints d'épilepsie dont l'état n'est pas maîtrisé de façon satisfaisante avec le traitement traditionnel. Les renseignements sur l'emploi du topiramate en monothérapie sont encore limités¹.

¹Une étude ouverte d'une durée de 20 semaines (n = 450 adultes). Posologie optimale : 300 à 350 mg/jour (moyenne : 288 mg/jour).

²Étude ouverte portant sur des enfants (n = 72) traités pendant au moins 3 mois. Posologie moyenne : 10 mg/kg/jour.

³Manifestations indésirables liées au SNC : Somnolence (30,1 %), étourdissements (28,3 %), ataxie (21,2 %), troubles de la parole (16,8 %), nystagmus (15 %), paresthésie (15 %), nervosité (15,9 %), difficulté à se concentrer/troubles de l'attention (8 %), confusion (9,7 %), dépression (8 %), anorexie (5,3 %), problèmes de langage (6,2 %) et troubles de l'humeur (3,5 %). Une évaluation de 1 446 adultes et 303 enfants a indiqué que ces deux groupes semblent présenter des profils de manifestations indésirables similaires.

⁴Les effets à long terme d'une perte de poids chez les enfants ne sont pas connus.



^{††}Médicament à usage limité : Ontario, Nouvelle-Écosse, Nouveau-Brunswick, L.-P.-É. Remboursement intégral : Québec, Saskatchewan, Colombie-Britannique, Alberta, Manitoba.

⁵Vous devez reporter aux Renseignements thérapeutiques sur TOPAMAX pour les détails thérapeutiques complets.

RÉFÉRENCES : 1. Monographie des comprimés et capsules à saupoudrer TOPAMAX* (topiramate), 11 mai 1999. 2. Kamin M, Kraut L, Olson W. Dose optimization of topiramate as add-on therapy in adults with treatment-resistant partial-onset seizures *Neurology* 1999;52 (Suppl 2):A525-526. 3. Glauser TA, Elterman R, Wyllie E et al. Open label topiramate in paediatric partial epilepsy *Epilepsia* 1997;38 (Suppl. 3):94. 4. Rosenfeld WE et al. Topiramate and concomitant weight loss. *Epilepsia* 1997;38 (Suppl 6):98.

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**37th Meeting of the
Canadian Congress of Neurological Sciences
VANCOUVER, BRITISH COLUMBIA**

with the Australian Association of Neurologists

**Scientific Themes
in Vancouver**

June 18-22, 2002

Tentative Scientific Program



TUESDAY JUNE 18, 2002

Pre-Congress Courses

Neurobiology Review Course
ALS Strategies for Quality of Life and
Quality of Care
Movement Disorder Video Session
Epilepsy Video Session

WEDNESDAY JUNE 19, 2002

Course Day

Spinal Surgery Course
Evidence Based Neurology
CSCN EMG
Update on Radiosurgery
Introduction of Design and Analysis of
Clinical Research
CSCN EEG
Imaging in Neurocritical Care
Welcome Reception

THURSDAY JUNE 20, 2002

Plenary Session I

Platform Sessions
Lunch, Poster Viewing, Exhibit viewing
Interactive/CPC Neurology/Neurosurgery
Wine and cheese/Poster viewing

FRIDAY JUNE 21, 2002

Meet the Expert Breakfast:
Neurosurgery

Plenary Session II

Platform Sessions
Lunch, Poster Viewing, Exhibit viewing
CLAE/CEC Epilepsy
Update on peripheral nerve surgery
Neurosonology or Ultrasound in
Neurology/Neurosurgery
Neuroscience Challenge and Social
Night

SATURDAY JUNE 22, 2002

Plenary Session III

Child Neurology Day
Stroke Course: Neurovascular
Endovascular Surgery
MS Course

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Rebif®

Interféron bêta-1a



Les effets secondaires les plus fréquemment observés sont les réactions au point d'injection et les symptômes pseudo-grippaux (asthénie, pyrexie, frissons, arthralgie, myalgie et céphalées). Leur fréquence et leur intensité tend à diminuer avec la poursuite du traitement. Veuillez consulter la monographie du produit pour les renseignements posologiques complets. Les données portant sur l'innocuité et l'efficacité proviennent d'observations sur 2 ans seulement.

* Rebif® est indiqué pour le traitement de la sclérose en plaques rémittente chez des patients dont la cote EDSS se situe entre 0 et 5,0, afin de réduire le nombre et la gravité des poussées cliniques, de ralentir la progression de l'invalidité physique, et de réduire les besoins de corticothérapie et le nombre de séjours à l'hôpital pour le traitement de la sclérose en plaques.

RÉFÉRENCE :

¹ Groupe d'étude PRISMS (Prevention of Relapses and Disability by Interferon B-1a Subcutaneously in Multiple Sclerosis), 1998. Randomised double-blind placebo-controlled study of Interferon B-1a in relapsing/remitting multiple sclerosis. *Lancet*, 352:1498-1504



FAIRE PLUS AVEC PLUS

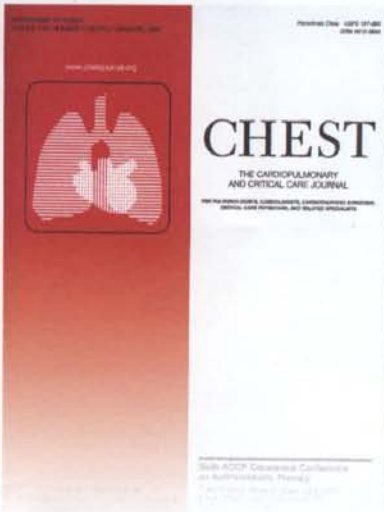
Introducing...

New **Aggrenox**[®]

**Prevented twice
as many strokes
vs. ASA^{2,3,4*}**



New American College of Chest Physician Stroke Guidelines state¹:



“Aggrenox is more effective than ASA alone for the prevention of [secondary] stroke” (grade 1A evidence)

Consider switching your ASA patients to Aggrenox:

- 22.1% additional stroke protection over ASA ($p=0.008$)^{3†§}
- 36.8% additional stroke protection over placebo ($p<0.001$)³
- Proven safety and tolerability profile^{3††} (Most common adverse events vs. ASA alone and vs. placebo: headache 39.2%, 33.8%, 32.9%; nausea 16.0%, 12.7%, 14.1%.)
- One capsule B.I.D.³

The overall discontinuation rate for Aggrenox was 27.8%, 23.2% for ASA and 23.7% for placebo.³

Aggrenox is indicated for the prevention of stroke in patients who have had a previous stroke or transient ischemic attack (TIA).³

Aggrenox is contraindicated in patients with hypersensitivity to dipyridamole, ASA, or any of the other product components. Due to the ASA content, Aggrenox is also contraindicated in patients with known allergy to nonsteroidal anti-inflammatory drug products and in patients with the syndrome of asthma, rhinitis and nasal polyps.³

Due to the ASA component, Aggrenox should be avoided in patients with severe hepatic insufficiency or severe renal failure, used with caution in patients with inherited/acquired bleeding disorders or who consume three or more alcoholic drinks every day, and avoided in patients with a history of active peptic ulcer disease. Aggrenox should not be used in pediatric patients or during the third trimester of pregnancy.³

Aggrenox has a vasodilatory effect and should be used with caution in patients with severe coronary artery disease (e.g., unstable angina or recently sustained myocardial infarction).³

* For every 1,000 patients treated for two years, Aggrenox prevented 58 strokes vs. only 29 for ASA, compared to placebo.^{2,3}

† Percentage of patients experiencing a stroke within two years: Aggrenox 9.5%, ASA 12.5%, placebo 15.2%.³

§ Randomised, double-blind, placebo-controlled trial, 6602 patients with history of TIA or ischemic stroke, mean age 66.7 years, 58% male, 42% female.³

†† When headache occurred, it was particularly evident in the first month of therapy. 8.9% of patients discontinued due to headache, 66% of these discontinued within the first month.³

Full Product Monograph is available upon request.

*Aggrenox is a registered trademark of Boehringer Ingelheim (Canada) Ltd.



Aggrenox[®]

ASA/Extended Release Dipyridamole
Helping to maximize stroke protection

