



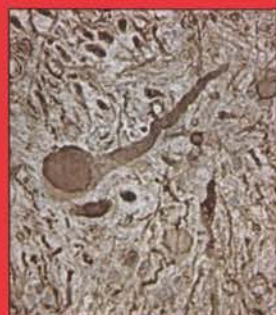
THE CANADIAN JOURNAL OF

Neurological Sciences

LE JOURNAL CANADIEN DES

Sciences Neurologiques

AN INTERNATIONAL JOURNAL / UN JOURNAL INTERNATIONAL



Amyotrophic Lateral
Sclerosis



Neuroimaging Highlight

**37th CANADIAN
CONGRESS OF
NEUROLOGICAL
SCIENCES**

June 18 - 22, 2002

Vancouver,
British Columbia

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Alastair Buchan

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



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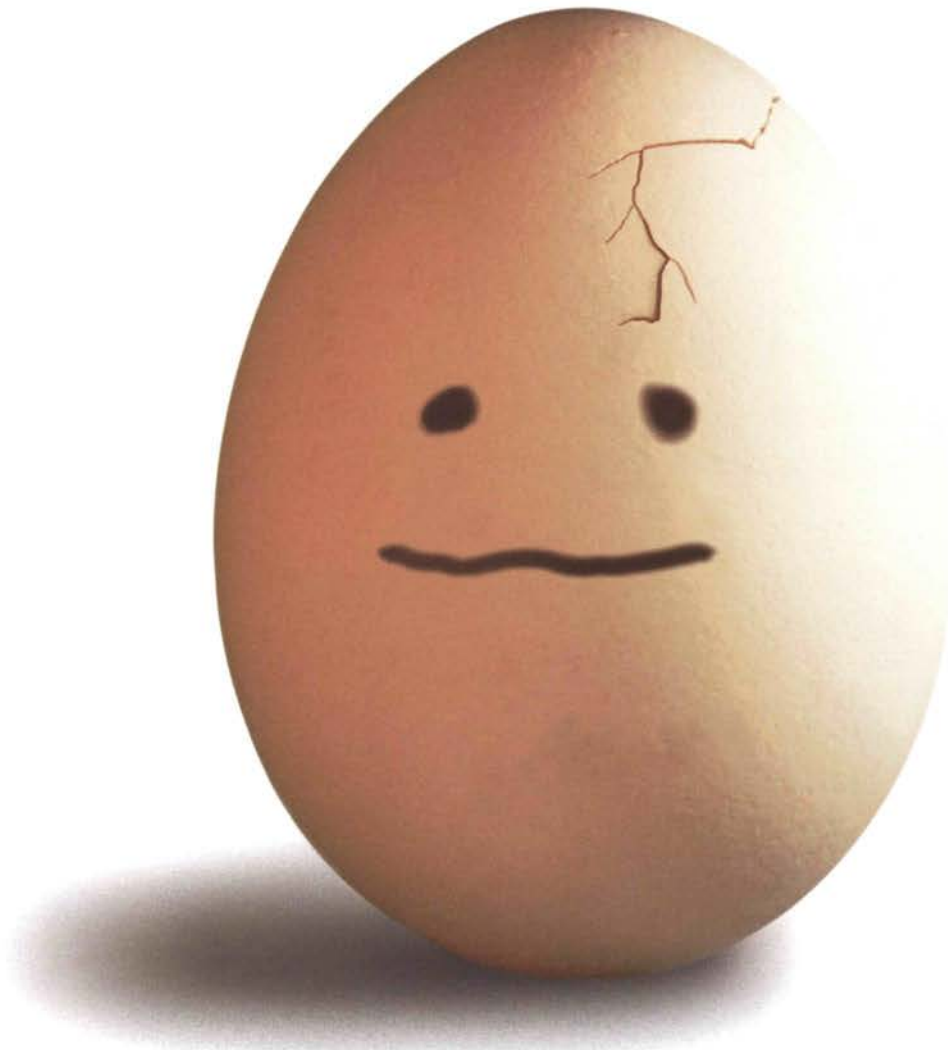
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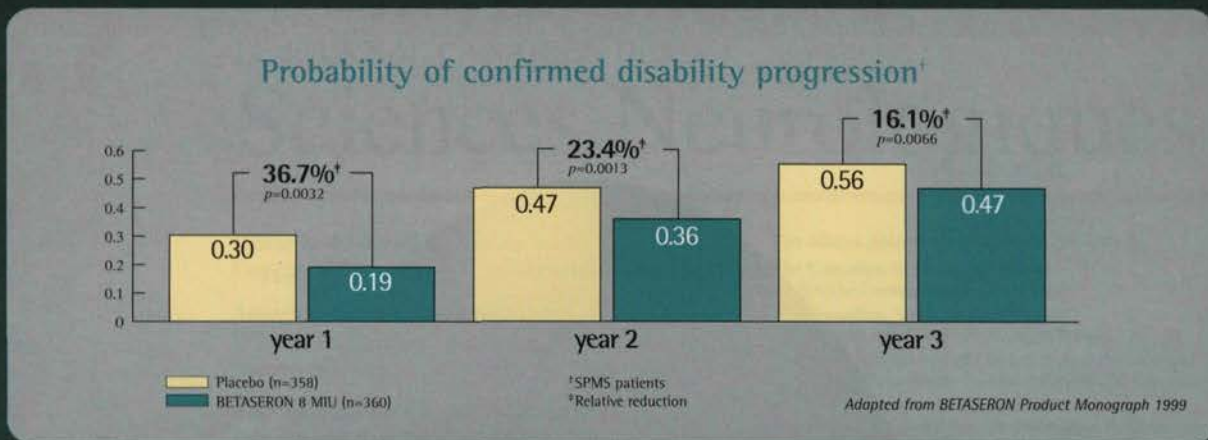


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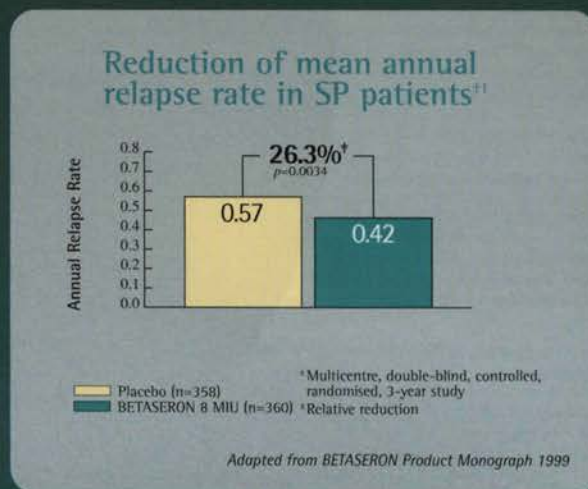
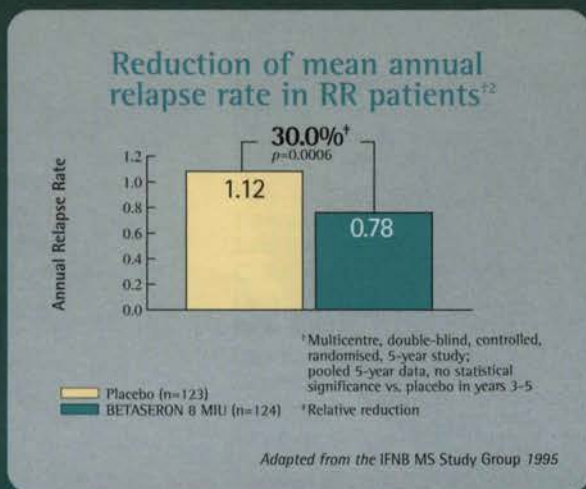
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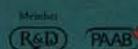
BETASERON has a manageable side-effect profile¹

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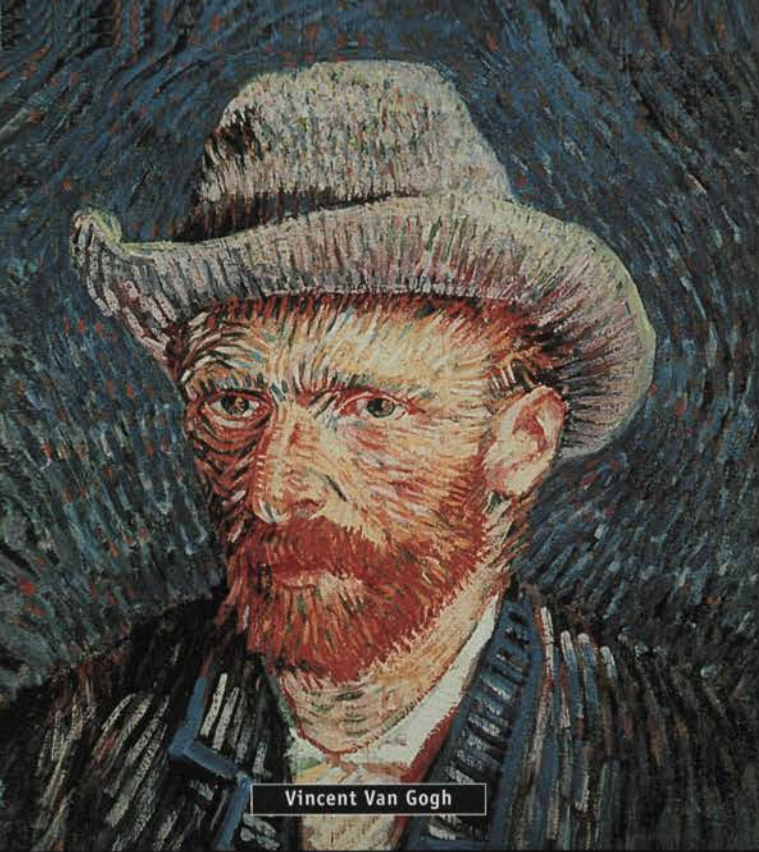
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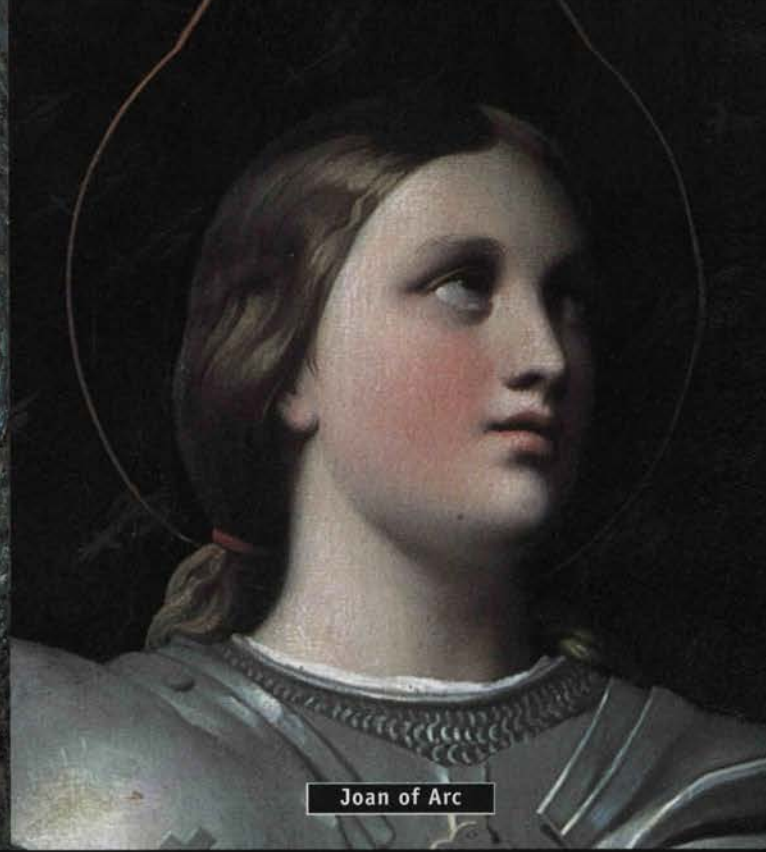
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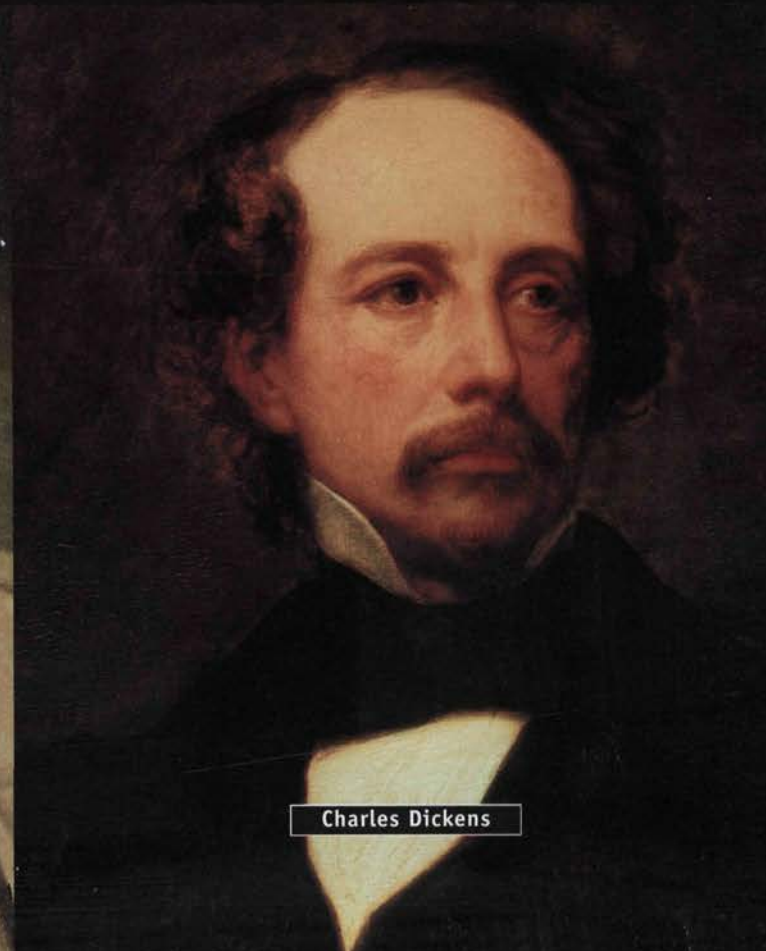


Joan of Arc

**YESTERDAY, PEOPLE WITH EPILEPSY
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[†] Open label, 20 week trial (n=450 Adults). Optimal dosing was 300-350 mg/day (Average 288 mg/day).

[‡] 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.

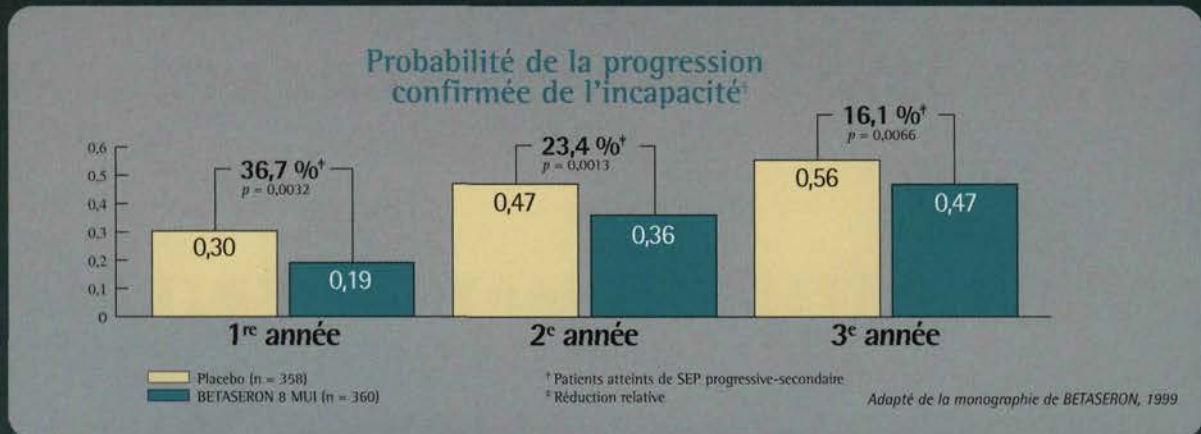
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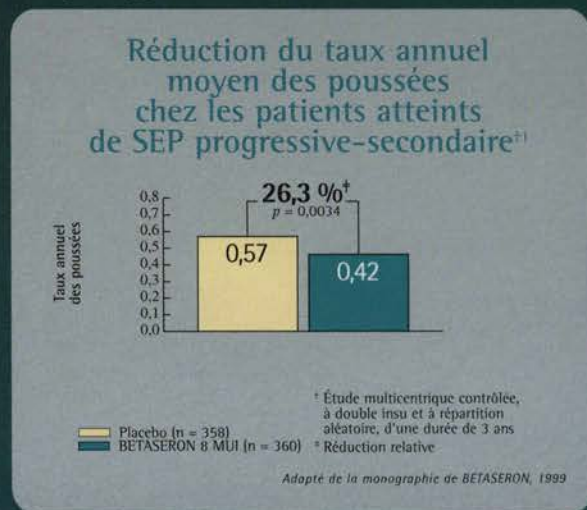
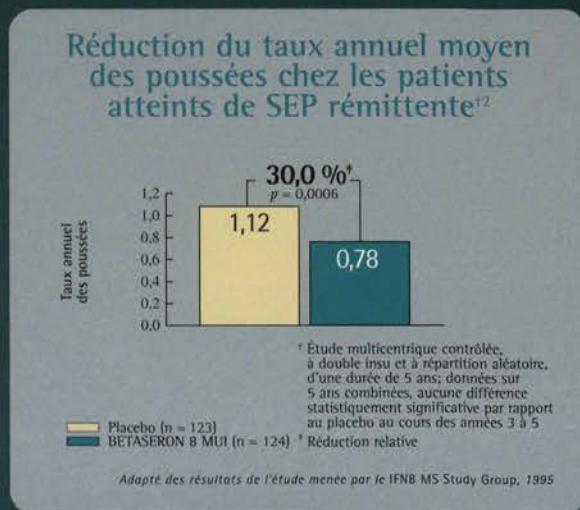
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BETASERON retarde la progression de l'incapacité*¹



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Effets indésirables pouvant être pris en charge¹

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Les symptômes pseudo-grippaux et les réactions au point d'injection peuvent être pris en charge et diminuent de façon marquée avec le temps¹.

* Il a été démontré que BETASERON retarde la progression de l'incapacité chez les patients atteints de SEP progressive-secondaire.
L'efficacité et l'innocuité de BETASERON dans la SEP progressive-primaire n'ont pas été évaluées.
On ne dispose pas de données probantes sur l'efficacité du traitement dans la SEP rémittente au-delà de deux ans, ni de données sur l'efficacité et l'innocuité du traitement dans la SEP progressive-secondaire au-delà de trois ans.
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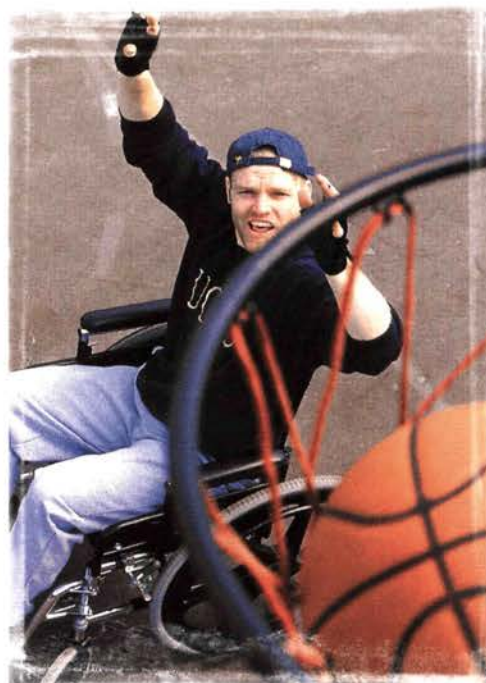
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McGeer PL, McGeer EG. Amino acid neurotransmitters. In: Siegel GJ, Albers RW, Agranoff BW, Katzman R, eds. *Basic Neurochemistry*. Boston: Little, Brown & Co., 1981: 233-254.

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- **Tables** Type tables double-spaced on pages separate from the text. Provide a table number and title for each. Particular care should be taken in the preparation of tables to ensure that the data are presented clearly and concisely. Each column should have a short or abbreviated heading. Place explanatory matter in footnotes, not in the heading. Do not submit tables as photographs.
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Zanaflex is effective first-line therapy for patients with spasticity associated with disorders and conditions such as *Multiple Sclerosis, stroke, cerebral palsy, spinal cord injury and traumatic brain injury*.^{1,2,3} The **dual mechanism of action**, targeting both the locus ceruleus and polysynaptic pathways, reduces hyperactivity of spinal motor neurons.^{2,4}

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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%).²

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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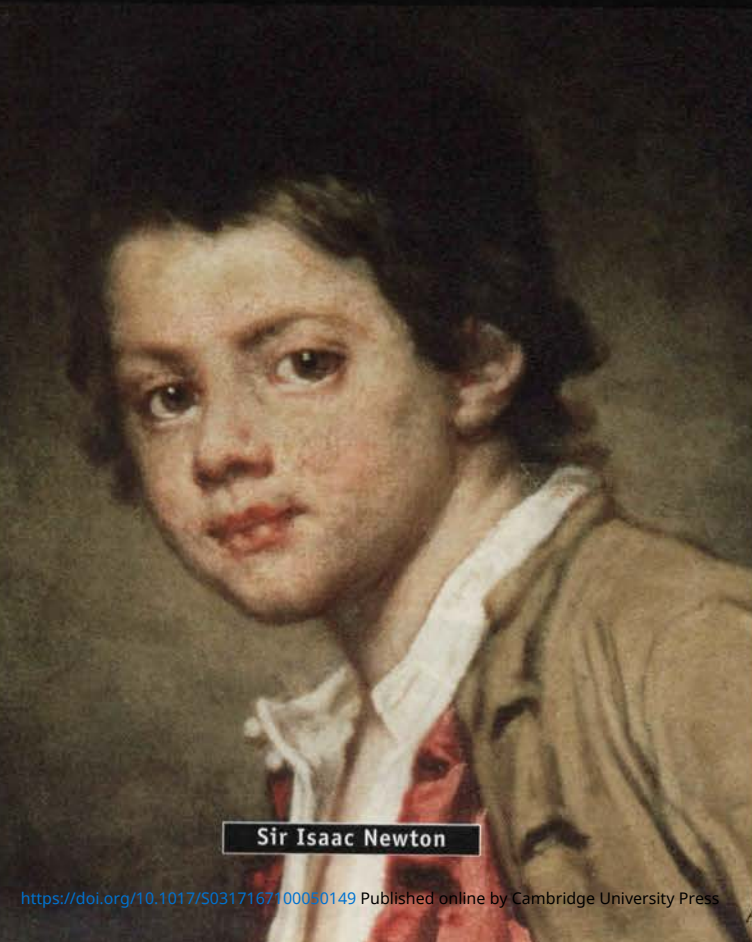


Vincent Van Gogh

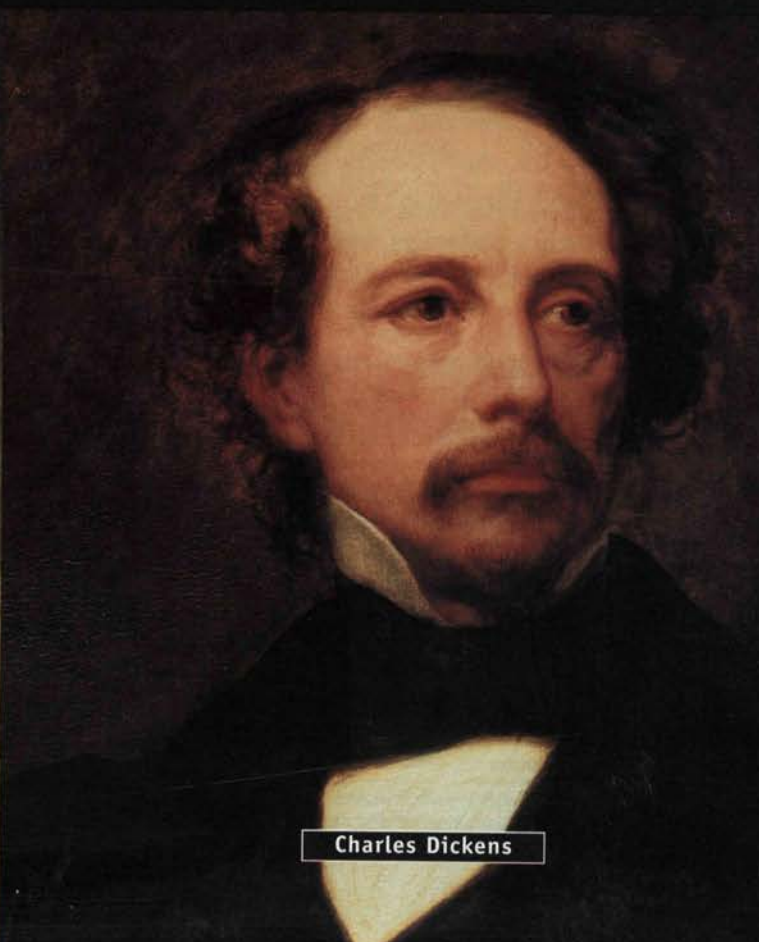


Jeanne d'Arc

**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^{4,1}

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^{5,1}

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^{††}

**MAINTENANT
OFFERT EN CAPSULES
À SAUPOUDRER**



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topiramate

**MAINTENANT
INDIQUÉ
CHEZ L'ENFANT**

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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.

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

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

[§]Manifestations indésirables liées au SNC : Somnolence (30,1 %), étourdissements (28,3 %), ataxie (21,2 %), troubles de la parole (16,8 %), ralentissement psychomoteur (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.

^{**}Les 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, I.-P.-É. Remboursement intégral : Québec, Saskatchewan, Colombie-Britannique, Alberta, Manitoba.

Veillez vous 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 8):98.

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- 37% reduction in the probability of disability progression over two years (21.9% vs. 34.9%; $p=0.02$)^{¶1,2}
- 32% reduction in the annual exacerbation rate over two years (0.61 vs. 0.90; $p=0.002$)^{*1,2}
- 38% of patients remained relapse free at two years ($p=0.03$)^{®1,2}
- 55% reduction in brain atrophy progression during the second year of therapy (-0.233 vs. -0.521; $p=0.03$)^{#3}
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- AVONEX[®] is indicated for the treatment of relapsing forms of MS.[!]

AVONEX[®] is generally well tolerated. The most common side effects associated with treatment are flu-like symptoms (muscle ache [myalgia], fever, chills, and asthenia). Please see product monograph for important patient selection and monitoring information.[!] AVONEX[®] should be used with caution in patients with depression and in patients with seizure disorders. AVONEX[®] should not be used by pregnant women. Patients with cardiac disease should be closely monitored. Routine periodic blood chemistry and hematology tests are recommended during treatment with AVONEX[®]!

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IM Injection

¶ Kaplan-Meier methodology. AVONEX[®] n=158, placebo n=143.

* AVONEX[®] n=85, placebo n=87.

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As measured by brain parenchymal fraction in the second year of treatment. AVONEX[®] n=68, placebo n=72.

† AVONEX[®] n=44, placebo n=44. The exact relationship between MRI findings and clinical status is unknown.

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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%.

We're still looking.

We spend \$8M in Research & Development, which includes IVIG use as a priority, and we are committed to sponsoring Canadian clinical trials to investigate possible new indications.

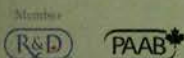
And because physicians are the key, we also sponsor two Immunology Fellowships (one in co-operation with CIHR) worth \$250,000, and we develop and offer CME promoting appropriate use through Canadian hospitals. For more information about the Appropriate Use of IVIG, contact Bayer at gamimune.canada@bayer.com



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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

ELECTROENCEPHALOGRAPHIC FINDINGS IN FRIEDREICH'S ATAXIA

G. Remillard, F. Andermann, L. Blitzer and E. Andermann

SUMMARY: Electroencephalographic tracings of 50 patients who presented the classical features of Friedreich's ataxia were reviewed. Mild nonspecific abnormalities were found in 33% and consisted of:

- a) Abnormal slow or irregular background rhythms in 15 patients (30%).
- b) Intermittent paroxysmal rhythms, considered to be projected from diencephalic or upper midbrain structures, in four patients (8%).
- c) Unilaterally absent driving responses in two affected siblings (4%).

There was no response to intermittent photic stimulation in 60% of the patients. This finding is not considered a definite abnormality, and its significance remains unclear.

Four patients (8%) had epileptic seizures, but of these, only two had interictal epileptic abnormalities.

There was no correlation between the duration and severity of the disease and the presence of electroencephalographic abnormalities.

Friedreich's ataxia is mainly a spinal disorder. Involvement of supraspinal and, in particular, brain stem or diencephalic structures may be more extensive in those patients who show electrographic abnormalities. This would require confirmation with comparative data based on pathological observations.

Impaired function of brain stem inhibitory mechanism may be responsible for the slightly raised incidence of seizures in patients with Friedreich's ataxia and other cerebellar degenerations

Can. J. Neurol. Sci. 1976;4:309

NERVE CONDUCTION STUDIES AND ELECTROMYOGRAPHY IN FRIEDREICH'S ATAXIA

J.M. Peyronnard, L. LaPointe, J.P. Bouchard, A. Lamontagne, B. Lemieux and A. Barbeau

SUMMARY: Twenty-six of 50 patients were investigated with nerve conduction studies and electromyography using a standard protocol and were compared to the findings in 50 normal control subjects. Almost all cases of typical Friedreich's ataxia had absent sensory action potentials (SAP) in the digital (92%) or sural (96%) nerves. The others had markedly decreased SAPs. In these same patients, motor conduction velocities were either normal or only slightly decreased. In the second, atypical group of nine patients, the motor conduction velocities were considerably decreased.

Because of the absence of sensory action potentials in Friedreich's ataxia, and that the absence was noted in our very mild cases, it is proposed that this measure be used to facilitate early diagnosis.

Can. J. Neurol. Sci. 1976;4:313

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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For brief prescribing information see page A-23

June 18-22, 2002
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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 Course
- Update on Radiosurgery
- Introduction of Design and Analysis of Clinical Research
- CSCN EEG Course
- Imaging in Neurocritical Care

Welcome Reception

Thursday June 20, 2002

- Meet the Expert Breakfast: Neurosurgery
- **Plenary Session I: Neurogenetics**
- Platform Sessions
- Grand rounds
- Creutzfeldt-Jacob Disease Course

Wine and cheese Poster viewing

Friday June 21, 2002

- **Plenary Session II: Neuroimaging**
- Platform Sessions
- What's New in Epilepsy?
- Update on Peripheral Nerve Surgery
- Ultrasound in Neurology

Neuroscience Challenge and Social Night

Saturday June 22, 2002

- **Plenary Session III: Neuroinflammation, Good and Bad**
- Child Neurology Day
- Stroke Course: Neurovascular / Endovascular surgery
- MS Course

37th meeting of the

Canadian Congress of Neurological Sciences

Special joint meeting with the
Australian Association of Neurologists

Introducing...

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

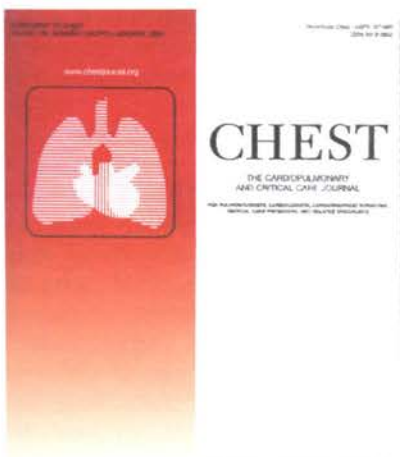


New American College of Chest Physician Stroke Guidelines state¹:

“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.

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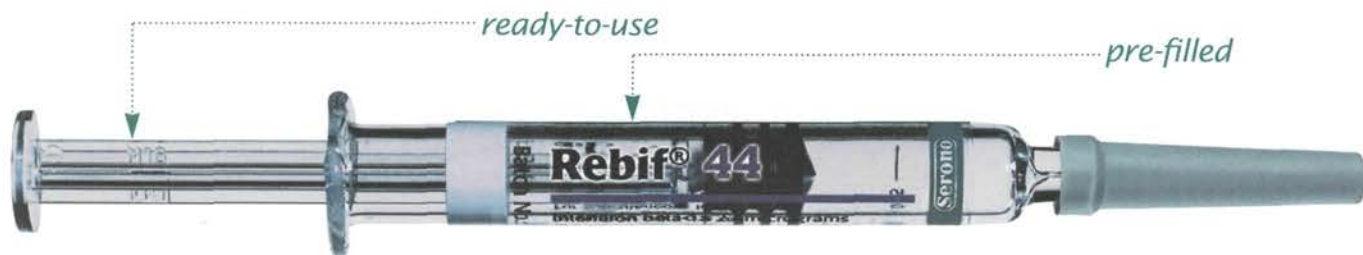
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Helping to maximize stroke protection

Rebif®. Dose-dependent Efficacy in Relapsing MS^{1*}



Rebif®

Interferon beta-1a



The most common reported adverse events are injection-site reactions and flu-like symptoms – e.g., asthenia, pyrexia, chills, arthralgia, myalgia, and headache. These tend to decrease in frequency and severity with continued treatment. Please see product monograph for full prescribing information. Evidence of safety and efficacy derived from 2-year data only.

* Rebif® is indicated for the treatment of relapsing-remitting multiple sclerosis in patients with an EDSS between 0 and 5.0, to reduce the number and severity of clinical exacerbations, slow the progression of physical disability, reduce the requirement for steroids, and reduce the number of hospitalizations for treatment of multiple sclerosis.

REFERENCES:

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



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