Volume 28 Number 1 February 2001

THE CANADIAN JOURNAL OF
Neurological Sciences
LE JOURNAL CANADIEN DES
Sciences Neurologiques

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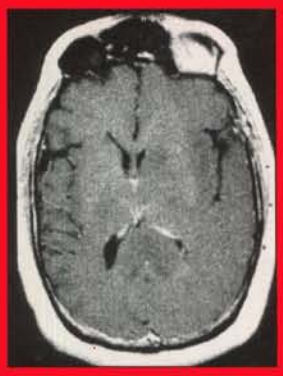
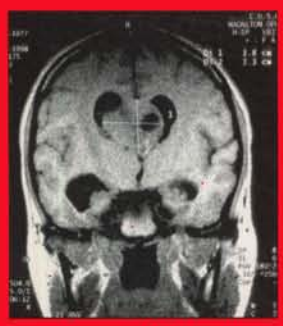


Neuroimaging Highlight



Neuropathological Conference

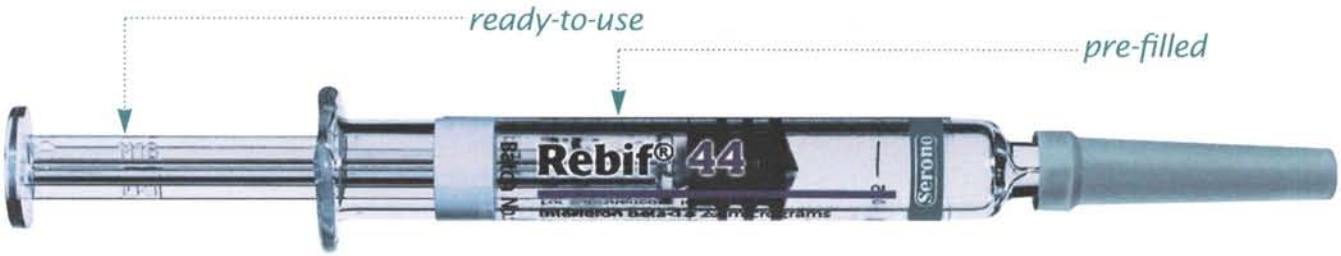
36th CANADIAN CONGRESS OF NEUROLOGICAL SCIENCES
June 12 - 16, 2001
Halifax, Nova Scotia

The official Journal of: The Canadian Neurological Society, The Canadian Neurosurgical Society, The Canadian Society of Clinical Neurophysiologists, The Canadian Association of Child Neurology

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 ß-1a Subcutaneously in Multiple Sclerosis) Study Group (1998). Randomised double-blind placebo-controlled study of interferon ß-1a in relapsing/remitting multiple sclerosis. Lancet 352:1498-1504

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For brief prescribing information see pages A-36, A-37

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THE CANADIAN JOURNAL OF Neurological Sciences

LE JOURNAL CANADIEN DES Sciences Neurologiques

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La Société Canadienne de Neurologie

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The permanent secretariat for the four societies and the Canadian Congress of Neurological Sciences is at/
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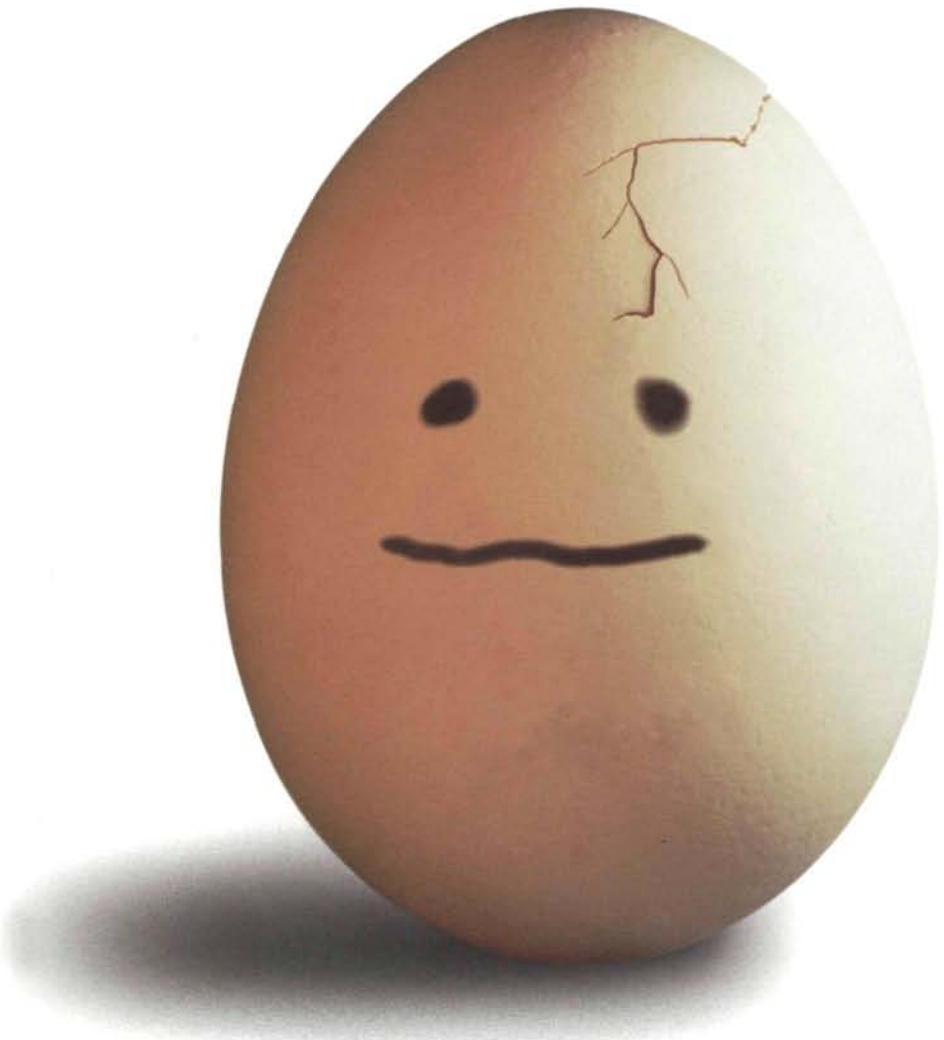
Are you using ASA for the prevention of a second stroke?



"Every patient who has experienced an atherothrombotic … stroke or TIA and has no contraindication should receive an antiplatelet agent regularly …"

- Fifth ACCP Consensus Conference on Antithrombotic Therapy[1]

Reassess your options...

For brief prescribing information see pages A-42, A-43

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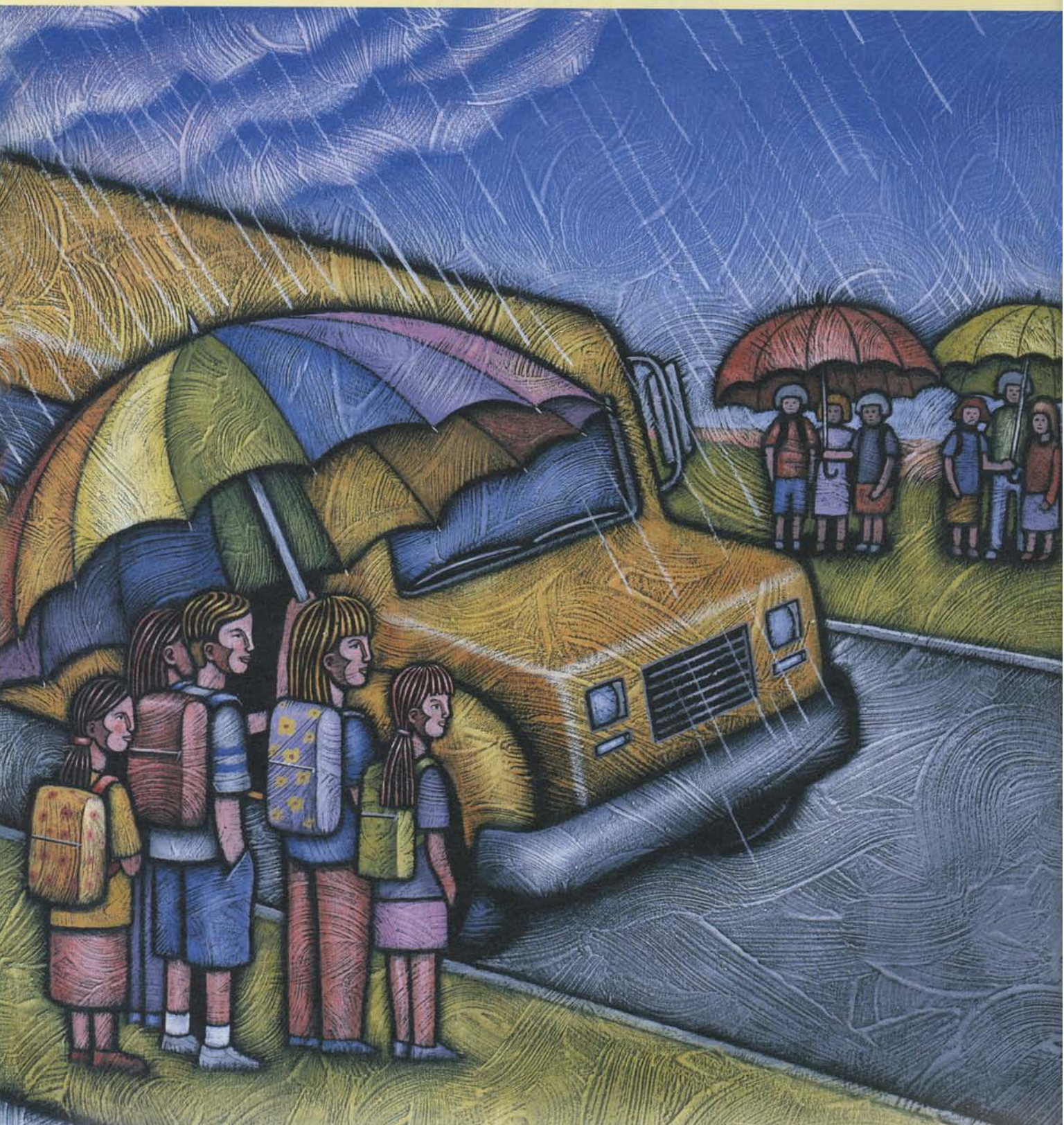
New in Lennox



*Refers to lamotrigine, gabapentin, vigabatrin, and topiramate, to be distinguished from standard AEDs.
†With the exception of atypical absence seizures.
‡Statistical significance not reported.
§**Rarely, serious skin rashes, including Stevens-Johnson syndrome and toxic epidermal necrolysis (Lyell syndrome), have been reported. Although the majority recover following drug withdrawal, some patients experience irreversible scarring and there have been rare cases of associated death.**
¶Frequently reported adverse events were pharyngitis, fever, infection, and rash (p = not significant).[1]
**For detailed information about dosing in adult and pediatric patients with LGS, please refer to the full prescribing information for LAMICTAL.
Dosage of add-on LAMICTAL in Motte *et al.* and Mullens *et al.* studies ranged from 50 to 400 mg/day, after escalation.
DO NOT EXCEED the recommended initial dose and subsequent dose escalations of LAMICTAL. More rapid initial titration has been associated with an increase incidence of serious dermatological reactions.

Product Monograph available to health care professionals upon request.

Gastaut Syndrome

Lamotrigine

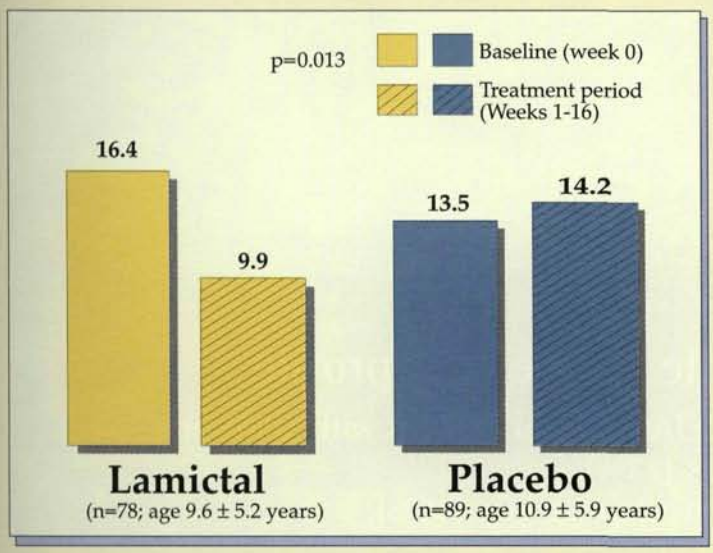
Lamictal®

LAMICTAL is the first and only of the newer* antiepileptic drugs (AED) indicated as adjunctive therapy for pediatric and adult patients with Lennox-Gastaut syndrome (LGS).[1] LAMICTAL is also the first and only of the newer* AEDs indicated for monotherapy after polytherapy in adults.

Significantly superior *control over the wide range of seizure types* associated with Lennox-Gastaut syndrome†

- Add-on LAMICTAL significantly reduced the number of all major seizures, all drop attacks, and all tonic-clonic seizures in patients with LGS.[1]

MEDIAN NUMBER OF ALL MAJOR SEIZURES/WEEK



A double-blind, randomised, placebo-controlled trial in patients from 3 to 25 years of age

Low CNS side-effect profile maintained in patients with Lennox-Gastaut syndrome aged 3-25

- Low withdrawal rate compared to placebo:‡[1,2] group taking LAMICTAL 3.8% (mostly due to rash§) vs. placebo group 7.8% (mostly due to deterioration of seizure control).

- No significant difference in the incidence of adverse events between LAMICTAL and placebo except for cold or viral illness (LAMICTAL 5% vs placebo 0%; p=0.05).¶[1]

Improved neurological function and cognitive skills[2,3]

- A greater proportion of LGS patients (age 3 to 25 years) treated with add-on LAMICTAL (n=79) vs add-on placebo (n=90) had a **clinically significant improvement in neurological findings** across the 16 week treatment period for: behaviour (30.4% vs. 14.4%); speech (11.4% vs. 2.2%); and non-verbal communication (11.4% vs. 7.8%).‡[3]

LAMICTAL offers superior control over the seizure types associated with LGS and a low CNS side-effect profile. You may also improve the neurological function and cognitive skills of your patients.[2,3] Add LAMICTAL** as soon as the diagnosis of LGS is suspected.[4]

GlaxoWellcome
Glaxo Wellcome Inc.

Lamotrigine

Lamictal®

For a Brighter Future

PMAC PAAB CCPP

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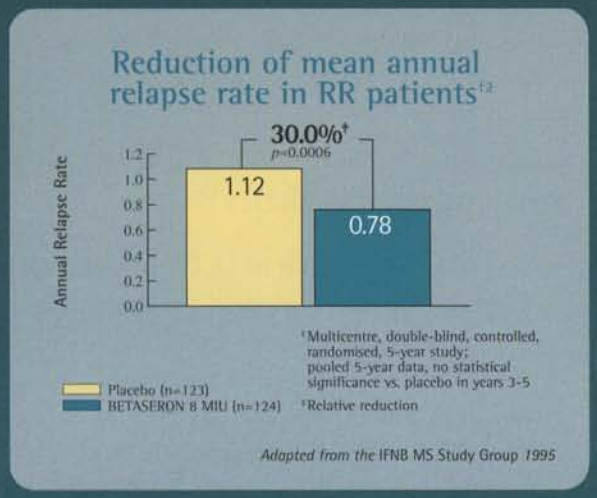
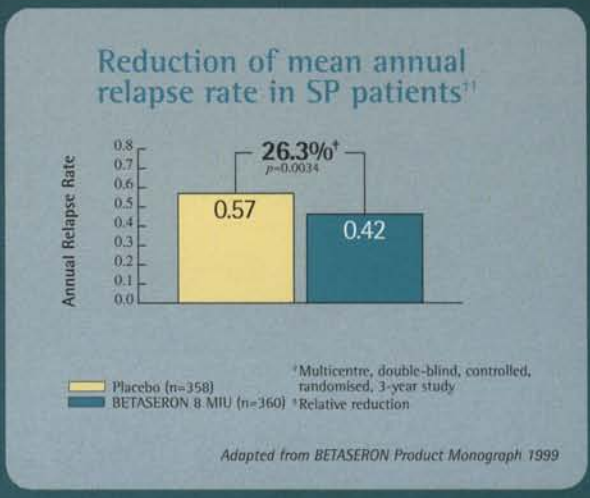
For brief prescribing information see pages A-52, A-53, A-54, A-55

BETASERON delays disability progression*[1]

BETASERON reduces relapse rate in both relapsing-remitting[2] and secondary progressive MS[1]





BETASERON has a manageable side-effect profile[1]

The most common side effects related to BETASERON in patients with SPMS are: flu-like syndrome (61%); fever (40%); chills (23%); injection-site inflammation (48%); injection-site reactions (46%); myalgia (23%); hypertonia (41%); rash (20%)[1]

Flu-like symptoms and injection-site reactions are manageable and lessen markedly with time[1]

*BETASERON has been demonstrated to delay the progression of disability in secondary progressive MS patients.[1] The safety and efficacy of BETASERON in primary progressive MS have not been evaluated. Efficacy of treatment for longer than 2 years has not been substantially demonstrated in relapsing-remitting MS. For secondary progressive MS, safety and efficacy data beyond 3 years are not available.

FOR COMPLETE WARNINGS AND PRECAUTIONS, PLEASE REFER TO THE PRODUCT MONOGRAPH. PRODUCT MONOGRAPH AVAILABLE TO HEALTH CARE PROFESSIONALS UPON REQUEST.

Delays Disability Progression*

Indicated for both RRMS and SPMS

In RRMS and SPMS

Pr **BETASERON®**

INTERFERON BETA-1b

From Onset Onwards

MULTIPLE SCLEROSIS PATHWAYS FOR CANADA

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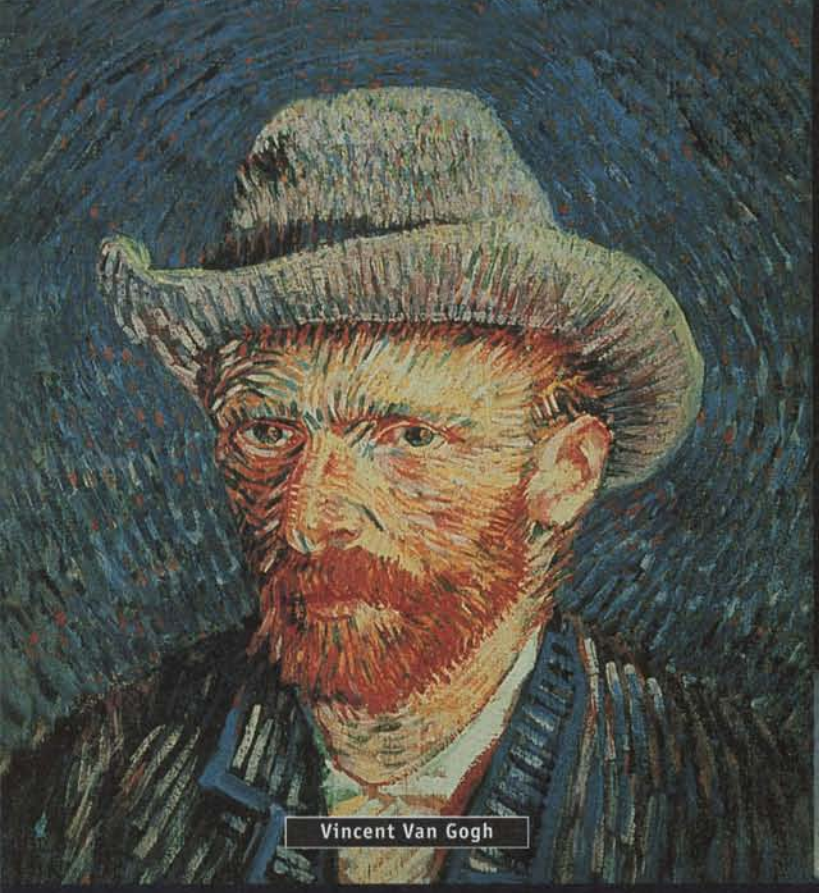
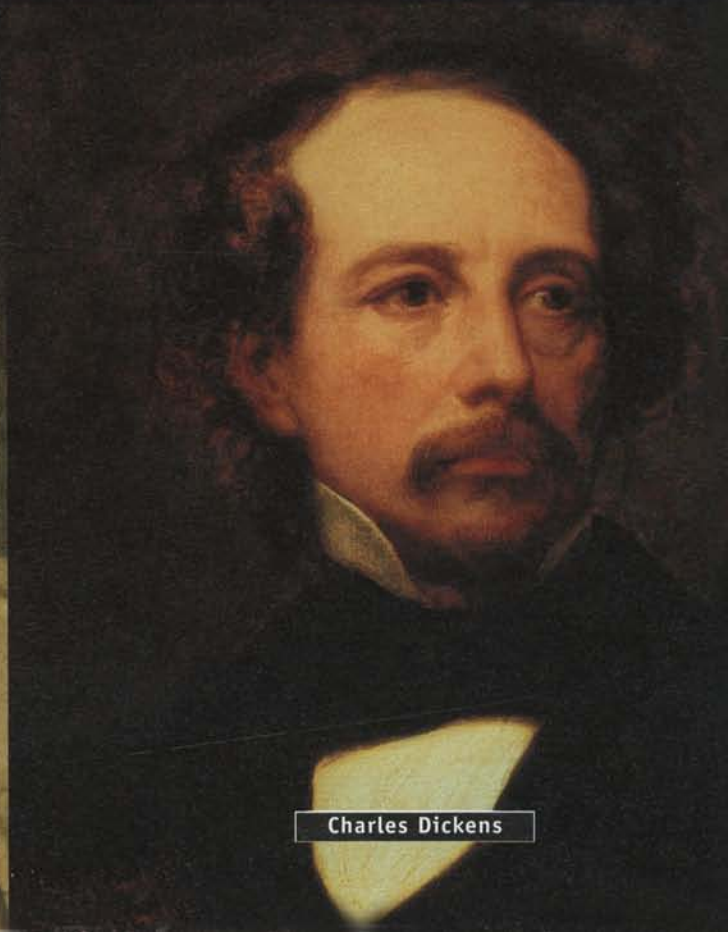
For brief prescribing information see pages A-56, A-57, A-58



YESTERDAY, PEOPLE WITH EPILEPSY HAD TO BE EXTRAORDINARY TO SUCCEED.



EFFICACY ACROSS A BROAD RANGE OF SEIZURES.

- TOPAMAX demonstrates efficacy in Partial Onset, Primary Generalized Tonic-Clonic, and Lennox-Gastaut Seizures[1]
- Desirable seizure-free results were shown in both Adults (19%)† and Children (22%)‡ with Partial Onset Seizures[2,3]

NO EVIDENCE OF LIFE-THREATENING SIDE EFFECTS.

- Like most antiepileptics, the most common side effects are CNS related, usually mild to moderate and transient§[1]

ADULT PATIENTS MAY EXPERIENCE WEIGHT LOSS.

- 73% of patients (n=52) showed a mean weight decrease of 5.97 lb (Interim analysis. Average duration 60 days)[4]
- 96% of children in clinical trials (≥ one year) who lost weight showed resumption of weight gain in test period**[1]

TODAY, THERE'S TOPAMAX.

B.I.D. DOSING WITH THE PATIENT IN MIND.

- TOPAMAX is initiated and titrated to clinical response regardless of existing anticonvulsant therapy
- Tablets available on formulary††

NOW AVAILABLE IN SPRINKLE CAPSULES

TOPAMAX* topiramate

NOW INDICATED FOR CHILDREN

HELPING PATIENTS MAKE MORE OF THEIR LIVES.

**TOPAMAX* topiramate Tablets and Sprinkle Capsules: indicated as adjunctive therapy for the management of patients (adults and children two years and older) with epilepsy who are not satisfactorily controlled with conventional therapy. There is limited information on the use of topiramate in monotherapy at this time.*

† 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.
** 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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For brief prescribing information see pages A-34, A-35

INFORMATION FOR AUTHORS

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• For detailed instructions regarding style and layout refer to *"Uniform requirements for manuscripts submitted to biomedical journals"*. Copies of this document may be obtained by writing to the Journal office, but the main points are summarized here. Articles should be submitted under conventional headings of *introduction, methods and materials, results, discussion,* but other headings will be considered if more suitable. Clinical trials must be reported in Consort format (JAMA 1996; 276: 637-639). Pages of text should be numbered consecutively.

• ***A title page*** should identify the title of the article which should be no more than 80 characters including spaces; name of institution(s) from which the work originated; and the name, address, telephone, and fax number of the corresponding author.

• ***Abstract*** Original Articles should be accompanied by an abstract of 250 words or less on a separate page, preferably in English and French, although the Journal will provide translation if required. Abstracts of original articles should consist of four paragraphs headed: *Background (or objective), Methods, Results and Conclusions*. Review articles should be accompanied by an abstract of 150 words or less.

• ***Acknowledgements*** including recognition of financial support should be typed on a separate page at the end of the text.

• The SI system (système international d'unités) should be used in reporting all laboratory data, even if originally reported in another system. Temperatures are reported in degrees celsius. English language text may use either British or American spelling, but should be consistent throughout.

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Journals

Yang JF, Fung M, Edamura R, et al. H-Reflex modulation during walking in spastic paretic subjects. Can J Neurol Sci 1991; 18: 443-452.

Chapter in a book

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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For brief prescribing information see pages A-46, A-47, A-51

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AS IN MS, SOME THINGS ARE NOT ALWAYS OBVIOUS.

Danger can lurk behind the face of an apparently healthy MS patient.

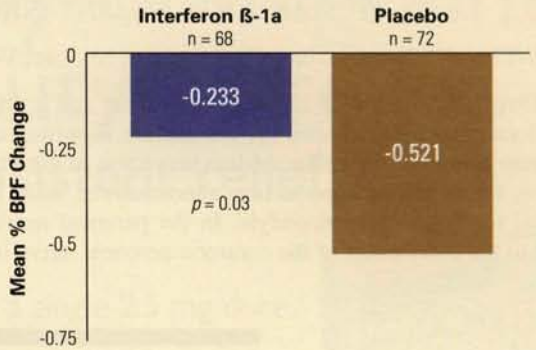
Progressive brain atrophy begins early in the course of MS and is likely irreversible.[1] Cognitive disturbances begin early in the MS process, but are often subtle and easily overlooked by patient and clinician alike.[2-4]

AVONEX® has shown a 55% reduction in brain atrophy progression.[5]

The use of AVONEX® can help patients with relapsing forms of MS maintain both physical AND mental function longer. In a clinical trial, patients treated with AVONEX® showed a 55% reduction in brain atrophy progression versus placebo, during the second year of treatment.*[5] AVONEX® is proven to slow the progression of physical disability - patients treated with AVONEX® showed a 37% reduction in the risk of disability progression and a 32% reduction in annual exacerbation rate over two years.†[6] AVONEX® also demonstrated a significant MRI effect showing an 89% reduction in gadolinium-enhanced lesions in patients with enhancement at baseline.◊[7]

Change in Brain Parenchymal Fraction[5]

(Adapted from Rudick et al.)



Change in brain parenchymal fraction (BPF) according to treatment arm in the interferon ß-1a clinical trial. Significantly less brain atrophy in interferon ß-1a patients during the second year.

Once-a-Week AVONEX® is generally well tolerated.[6]

The once-a-week intramuscular dosing regimen with AVONEX®, means few opportunities for injection-related side effects to disrupt patient's lifestyle.[6] The most common side effects associated with treatment are flu-like symptoms and usually resolve within 24 hours after injection.[6,8] Incidence of side effects decrease over time with continued treatment for most people.[8] Please see product monograph for important patient selection and monitoring information.

ONCE-A-WEEK AVONEX® (Interferon beta-1a) IM Injection

Helping people with relapsing forms of MS get on with their lives.

* It remains to be determined whether brain atrophy during the relapsing-remitting stage of MS will predict long-term disability progression better than clinical features in the majority of patients. Additional prospective studies are needed to determine the biologic factors associated with atrophy progression, the clinical significance of BPF change during the relapsing-remitting disease stage, and the impact and time course of therapeutic intervention.

† Kaplan-Meier estimate of percentage progressing at two years for placebo patients: 34.9% (n=143); AVONEX®-treated patients: 21.9% (n=158);(p=0.02). Placebo annual exacerbation rate: 0.90 (n=87); AVONEX® annual exacerbation rate: 0.61(n=85);(p=0.002).

◊ The exact relationship between MRI findings and clinical status is unknown (n=44).

AVONEX® is indicated for the treatment of relapsing forms of MS.

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For brief prescribing information see pages A-44, A-45, A-55

25 Years Ago in the Canadian Journal of Neurological Sciences

Direct Leukocyte Migration Inhibition in Multiple Sclerosis – A Possible Assessment of Activity

M.P. Day, J.H. Day and P.L. Mann

SUMMARY: Twenty-four patients with multiple sclerosis were evaluated and classified according to their clinical state. Specific migration inhibition studies were carried out on blood samples from each using myelin basic protein, an acid soluble protein fraction isolated from normal human CNS white matter, and multiple sclerosis myelin basic protein from patients who had the disease, as the antigenic material. This test system employed selected media. Results were compared with those of normal controls and patients with other neurological disease states. Antigen concentration of 500μg/ml in serum free medium combined to produce the greatest inhibiting effect on leukocytes in patients with apparent multiple sclerosis and differentiated these patients from those in other groups tested.

Leukocytes in patients who had probable multiple sclerosis with partial impairment, signifying possible current activity of the disease, were especially inhibited as compared to the leukocytes from other groups tested against myelin antigen. Cerebrospinal fluid from affected patients when used as a media enhanced the test.

This study suggests that migration inhibition of peripheral leukocytes using myelin protein may be useful in the diagnosis of patients with multiple sclerosis. There is additional evidence that the degree of leukocyte migration inhibition may reflect activity of the disease with its consequent implications on treatment and prognosis.

Can. J. Neurol. Sci. 1976;3:99

The Location of Conduction Abnormalities in Human Entrapment Neuropathies

William F. Brown, Gary G. Ferguson, Michael W. Jones and Stephen K. Yates

SUMMARY: Direct stimulation of 23 median, 13 ulnar and 2 peroneal nerves at the time of surgical exploration has been used to locate, and characterize the conduction abnormalities in the nerves. The most frequent location of the major conduction abnormalities in the median nerve was in the first 1-2 cm distal to the origin of the carpal tunnel. In the ulnar nerve, the important conduction abnormalities were located most frequently in the segments 1 cm proximal and distal to the medial epicondyle. In the peroneal nerve, the major conduction abnormalities occurred proximal or distal to the entry point of the common peroneal nerve into the peroneus longus muscle.

Can. J. Neurol. Sci. 1976;3:111

Interpretation of the Computerized Tomographic Scan

M. Banna

SUMMARY: This paper is based on the experience gained from over three thousand examinations by computerized tomography (CT). The appearances of various intracranial lesions are presented. The reliability and limitations of this new technique are also described and some pitfalls in scan interpretation are mentioned.

Can. J. Neurol. Sci. 1976;3:123

Continued on page 20

Turn the agony of migraine into the beauty of relief.

Zomig® provides consistent relief.

- Rapid relief within one hour.[1]
- Significant headache response* after a single 2.5 mg dose.[1]
- Consistent efficacy across multiple attacks.[2-4]
- Effective in a wide variety of migraine subtypes.[1†]
- Effective when taken at any time during a migraine attack.[2]
- Treats associated symptoms of photophobia, phonophobia and nausea.[1]
- Proven safety profile in over 5,500 patients treating more than 89,000 attacks.[5,6††]

*Improvement from severe or moderate headache to mild or no pain at two hours.

† Zomig® is indicated for the acute treatment of migraine with or without aura.

Zomig® is not intended for use prophylactically or in hemiplegic, basilar, or ophthalmoplegic migraine. Safety and efficacy have not been established for cluster headache, which is present in an older, predominantly male population.

†† The most common side effects reported with Zomig® compared to placebo were nausea (9% vs. 3.7%), head/face sensations (8.6% vs. 1.7%), dizziness (8.4% vs. 4%) and neck/throat/jaw sensations (7% vs. 3%).

Zomig® is contraindicated in patients with history, symptoms, or signs of ischemic cardiac, cerebrovascular or peripheral vascular syndromes, valvular heart disease or cardiac arrhythmias (especially tachycardias). In addition, patients with other significant underlying cardiovascular disease should not receive Zomig®. Zomig® is also contraindicated in patients with uncontrolled or severe hypertension.

Please see Product Monograph.

For more information about Zomig® please contact AstraZeneca Customer Relations by phone at 1-800-668-6000 or fax at (905) 896-4745.

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Zomig® (zolmitriptan) is a registered trademark of the AstraZeneca group of companies.

Zomig®
zolmitriptan tablets 2.5 mg

Consistent migraine relief.

AstraZeneca PMAB

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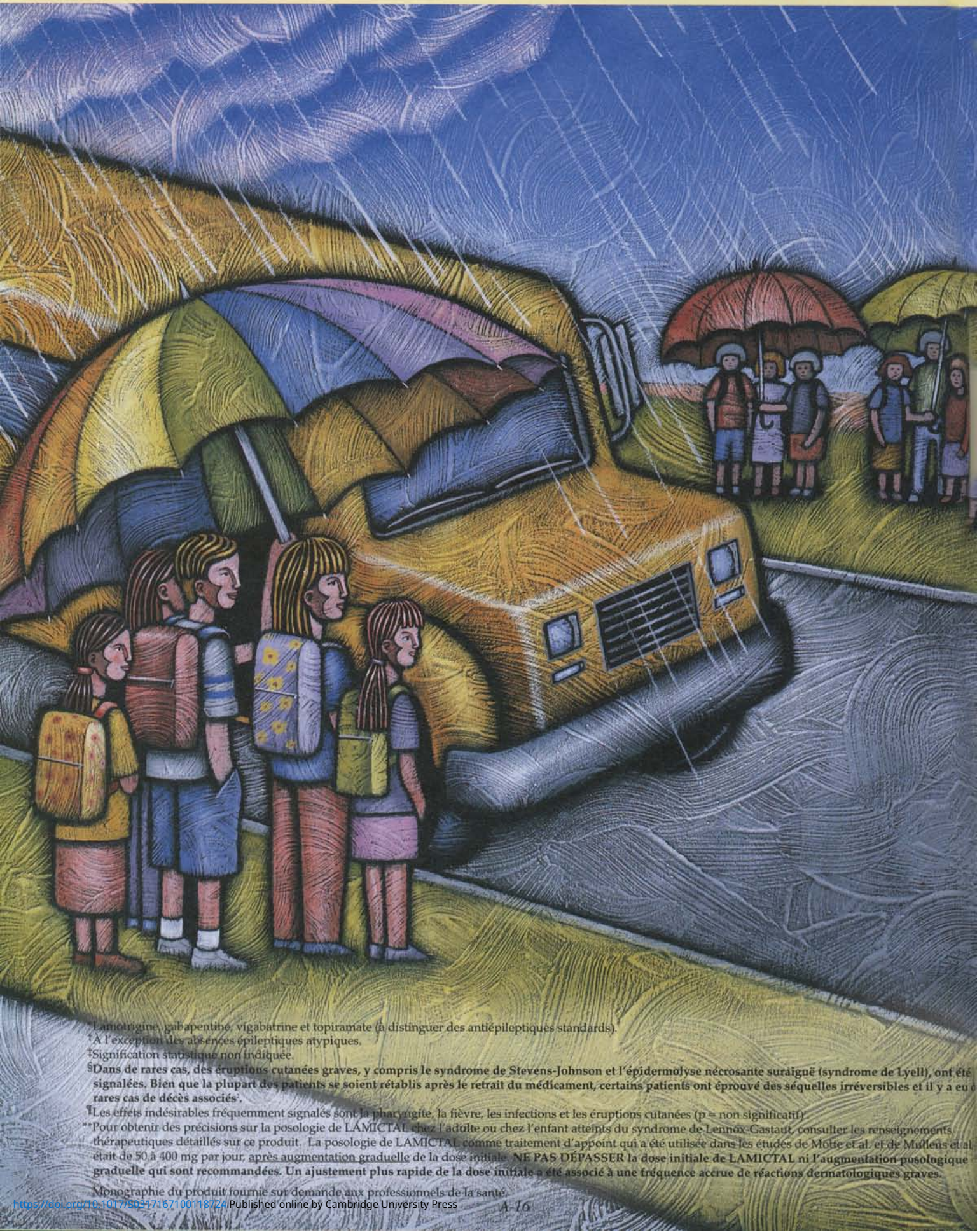
For brief prescribing information see pages A-32, A-33

Nouveau dans le syndr



*Lamotrigine, gabapentine, vigabatrine et topiramate (à distinguer des antiépileptiques standards).
†À l'exception des absences épileptiques atypiques.
‡Signification statistique non indiquée.
§**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 plupart des patients se soient rétablis après le retrait du médicament, certains patients ont éprouvé des séquelles irréversibles et il y a eu de rares cas de décès associés.**
¶Les effets indésirables fréquemment signalés sont la pharyngite, la fièvre, les infections et les éruptions cutanées (p = non significatif).
Pour obtenir des précisions sur la posologie de LAMICTAL chez l'adulte ou chez l'enfant atteints du syndrome de Lennox-Gastaut, consulter les renseignements thérapeutiques détaillés sur ce produit. La posologie de LAMICTAL comme traitement d'appoint qui a été utilisée dans les études de Motte et al. et de Mullens et al. était de 50 à 400 mg par jour, après augmentation graduelle de la dose initiale. **NE PAS DÉPASSER la dose initiale de LAMICTAL ni l'augmentation posologique graduelle qui sont recommandées. Un ajustement plus rapide de la dose initiale a été associé à une fréquence accrue de réactions dermatologiques graves.

Monographie du produit fournie sur demande aux professionnels de la santé.

ne de Lennox-Gastaut

lamotrigine

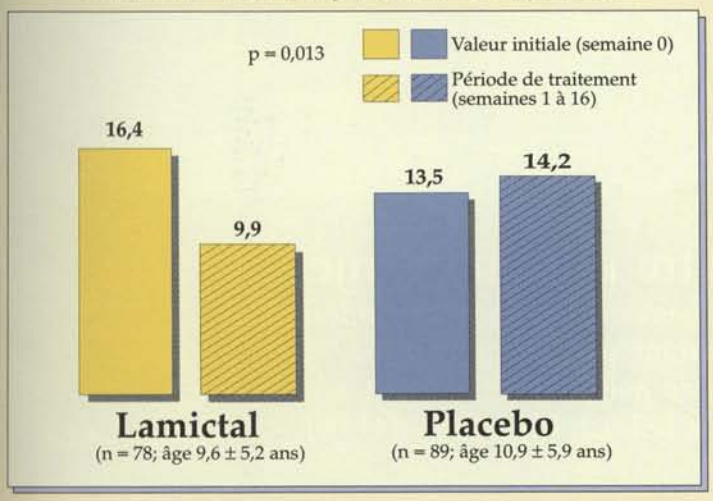
Lamictal®

LAMICTAL est le premier et le seul parmi les nouveaux antiépileptiques* qui soit indiqué comme traitement d'appoint chez les enfants et les adultes atteints du syndrome de Lennox-Gastaut (SLG)[1]. LAMICTAL est également le premier et le seul parmi les antiépileptiques récents* qui soit indiqué comme monothérapie après polythérapie chez l'adulte.

Une supériorité significative pour *maîtriser les divers types de crises* liées au syndrome de Lennox-Gastaut†

- L'adjonction de LAMICTAL réduit, de façon significative, le nombre de crises majeures, les effondrements épileptiques et les crises tonico-cloniques chez les patients atteints de SLG[1].

NOMBRE MÉDIAN
DES CRISES MAJEURES/SEMAINE



Essai à double insu, à répartition aléatoire et à contrôle placebo chez des patients de 3 à 25 ans

Maintien d'un *faible profil d'effets indésirables touchant le SNC* chez les patients de 3 à 25 ans atteints du syndrome de Lennox-Gastaut

- Faible taux d'abandons comparativement au placebo‡[1,2] : 3,8 % pour le groupe LAMICTAL (principalement reliés aux éruptions cutanées§) contre 7,8 % pour le groupe placebo (principalement reliés à une détérioration de la maîtrise des crises).
- Aucune différence significative dans la fréquence des effets indésirables entre LAMICTAL et le placebo, sauf pour le rhume ou des maladies virales (LAMICTAL, 5 % contre placebo, 0 %; p = 0,05)¶[1].

Amélioration de la fonction neurologique et des facultés cognitives[2,3]

- Une plus forte proportion de patients (de 3 à 25 ans) atteints de SLG, traités à l'aide de LAMICTAL comme traitement d'appoint (n = 79) c. un placebo d'appoint (n = 90), ont connu une **amélioration cliniquement significative des symptômes neurologiques** durant la période de traitement de 16 semaines : comportement (30,4 % c. 14,4 %), parole (11,4 % c. 2,2 %) et communication non verbale (11,4 % c. 7,8 %)‡[3].

LAMICTAL offre une plus grand maîtrise des divers types de crises liées au SLG, avec faible profil d'effets indésirables touchant le SNC. Vous pouvez aussi améliorer la fonction neurologique et les facultés cognitives de vos patients[2,3]. Ajoutez LAMICTAL** dès que l'on soupçonne un SLG[4].

GlaxoWellcome
Glaxo Wellcome Inc.

lamotrigine

Lamictal®

L'avenir en tête

Pour documentation voir pages A-52, A-53, A-54, A-55

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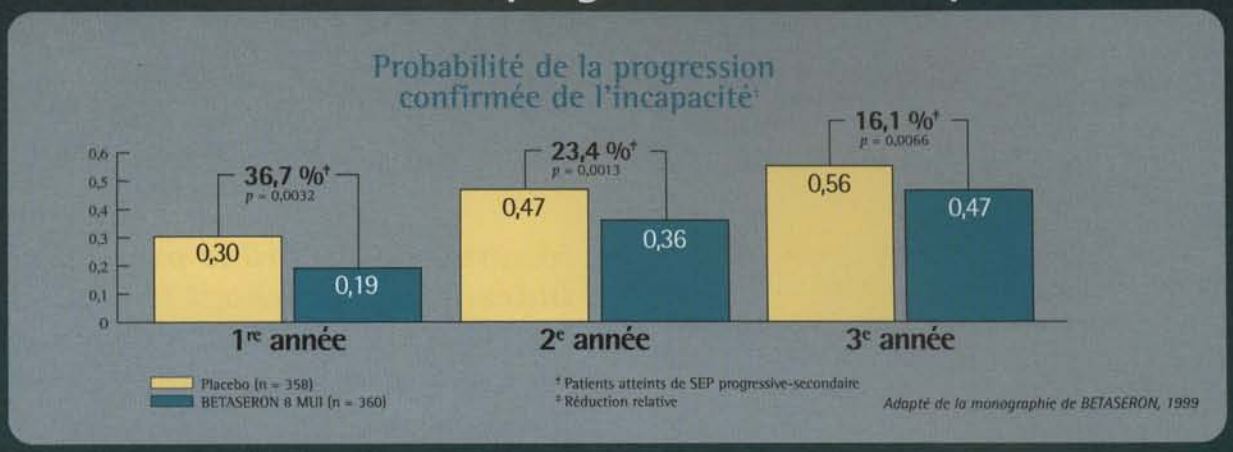
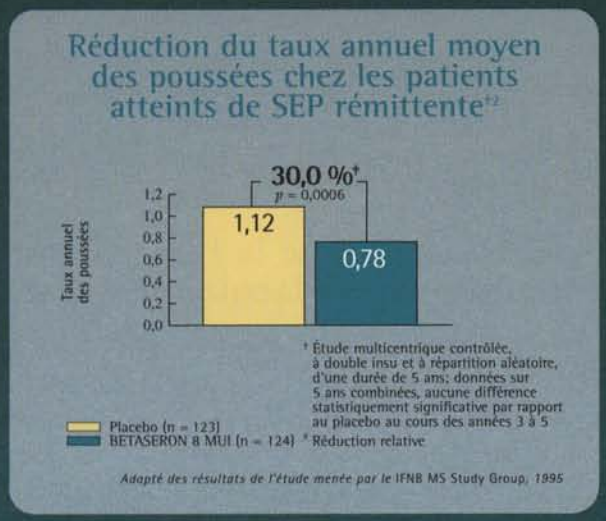
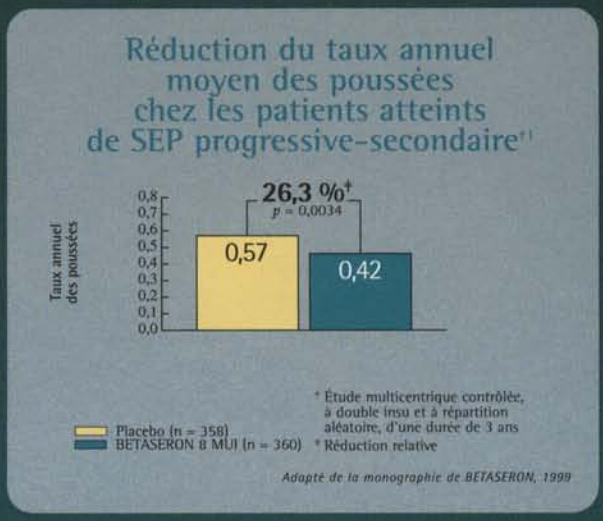
BETASERON retarde la progression de l'incapacité*[1]



BETASERON réduit le taux de poussées dans la SEP rémittente[2] et dans la SEP progressive-secondaire[1]





Effets indésirables pouvant être pris en charge[1]

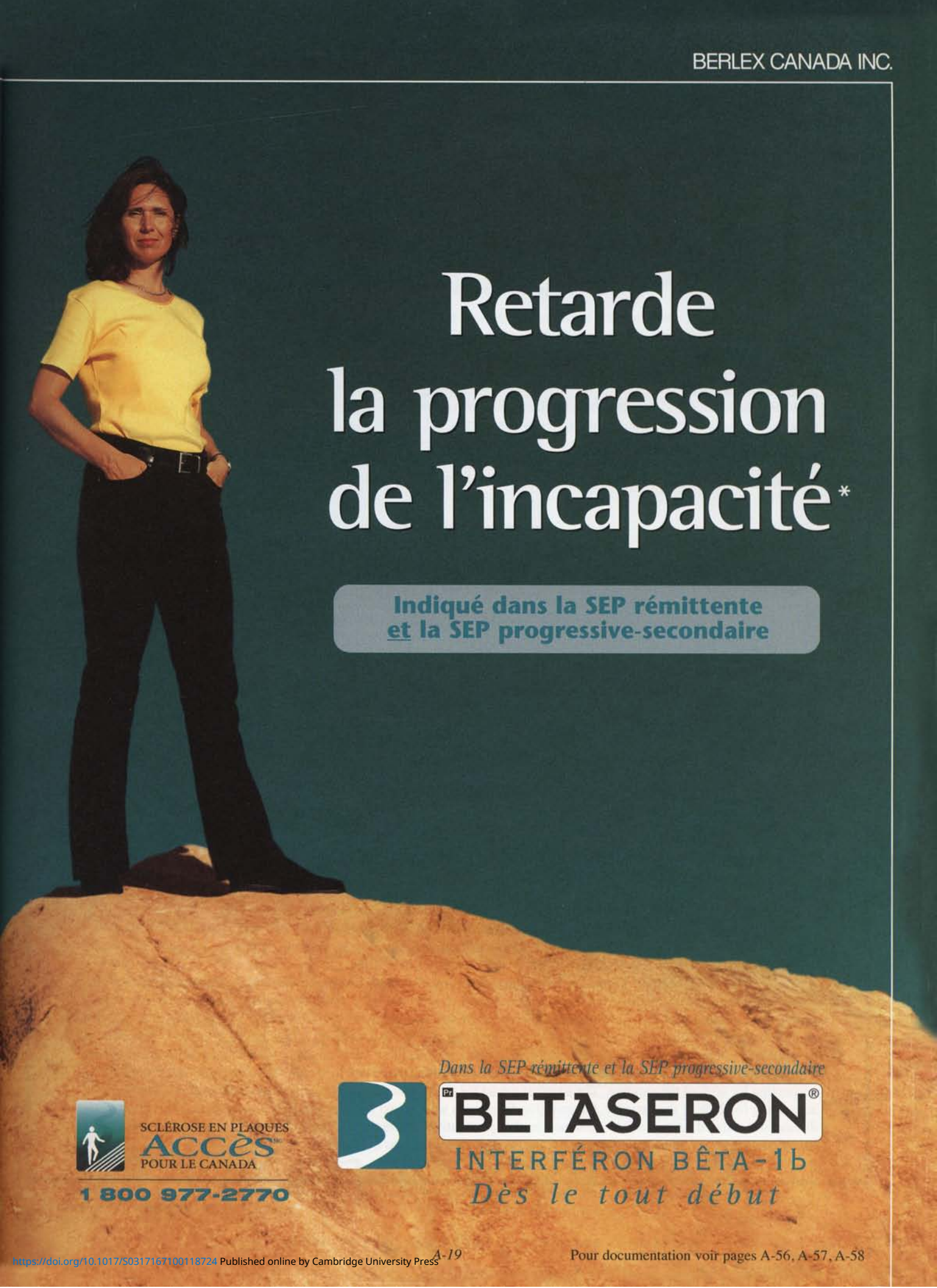
Chez les patients atteints de SEP progressive-secondaire, les effets indésirables les plus fréquents de BETASERON sont : syndrome pseudo-grippal (61 %), fièvre (40 %), frissons (23 %), inflammation au point d'injection (48 %), réactions au point d'injection (46 %), myalgie (23 %), hypertonie (41 %) et éruption cutanée (20 %)[1].

Les symptômes pseudo-grippaux et les réactions au point d'injection peuvent être pris en charge et diminuent de façon marquée avec le temps[1].

*Il a été démontré que BETASERON retarde la progression de l'incapacité chez les patients atteints de SEP progressive-secondaire[1].
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.

VEUILLEZ CONSULTER LA MONOGRAPHIE DE PRODUIT POUR OBTENIR LA LISTE COMPLÈTE DES MISES EN GARDE ET DES PRÉCAUTIONS.
MONOGRAPHIE DE PRODUIT OFFERTE SUR DEMANDE AUX PROFESSIONNELS DE LA SANTÉ.

Modèle R&D CCPP



Pour documentation voir pages A-56, A-57, A-58

https://doi.org/10.1017/S0317167100118724 Published online by Cambridge University Press

25 Years Ago in the Canadian Journal of Neurological Sciences

Shy-Drager Syndrome. Neuropathological correlation and Response to Levodopa Therapy

Jacques de Lean and John H.N. Deck

SUMMARY: The postmortem examination of the nervous system of a patient with Shy-Drager syndrome successfully treated with levodopa (Sharpe et al, 1972) revealed features of striato-nigral degeneration and amyotrophic lateral sclerosis, a cerebellar system degeneration and a loss of approximately 75% of sympathetic preganglionic neurons. Lewy bodies were not present and no detectable changes were observed in the sympathetic prevertebral ganglia.

While the limited and transient beneficial effect of levodopa on the bradykinesia in our case is possibly due to the progressive loss of striatal dopaminergic receptors seen in striatonigral degeneration, we propose that in Shy-Drager syndrome, levodopa therapy benefits orthostatic hypotension because of a suppression of the central depressor action of this drug. This suppression is attributable to functional disconnection of sympathetic ganglia secondary to the loss of preganglionic neurons or to degeneration of central autonomic catecholaminergic systems.

Can. J. Neurol. Sci. 1976;3:167

Myophosphorylase Deficiency (McArdle's Disease): Report of a family

M. Zafar Mahmud, R. Rodney Howell, Roger E. Stevenson and John Gilroy

SUMMARY: The clinical and biochemical findings are presented of two brothers suffering from McArdle's Disease (Myophosphorylase Deficiency). Tissue enzyme estimations and lactate levels were done in affected and nonaffected members of the family. Affected members showed absence of phosphorylase enzyme by histochemical and quantitative estimation. No quantitative abnormalities were found in other enzyme systems of glycolytic pathways in the family investigated. Various other aspects of clinical features, biochemical abnormalities and inheritance are discussed.

Can. J. Neurol. Sci. 1976;3:175

Intellectual Performance in Multi-Infarct Dementia and Alzheimer's Disease: A Replication Study

Francisco I. Perez, David A. Stump. Joe R.A. Gay and Vicki R. Hart

SUMMARY: A consistent feature in dementia is an overall intellectual degeneration. The present study investigated the intellectual performance of patients with Alzheimer's Disease (AD) and multi-infarct dementia (MID) using the Wechsler Adult Intelligence Scale (WAIS). For reliability and generality purposes two independent samples of patients were collected. Significant differences in Education Level (EDU) and Performance IQ (PIQ) were obtained for the first sample, with the AD group having a significantly higher EDU level. The MID group obtained a higher PIQ. No significant differences were found in the second sample, but EDU level approached significance with the AD group again having a higher EDU level. A discriminant function analysis classified 81% of the patients in the first sample and 100% diagnostic accuracy was obtained for the second sample using the 11 predictor variables. A maximum R^2 stepwise regression was performed in order to detect the "best" model of variables discriminating between the diagnostic groups. For the first sample the "best" model was the two variable model, including EDU and Full Scale IQ, accounting for 40% of the variance. The simplest model for the second sample was the one variable model including EDU, accounting for 20% of the variance. Quantitative differences were found between the AD samples. time since onset of the disease was offered as a possible influence in the quantitative differences in the AD samples. Sampling biases in the behavioural study of dementia are discussed. Successful replication was obtained.

Can. J. Neurol. Sci. 1976;3:181

Continued on page 26

Introducing Zomig Rapimelt™

A new twist in migraine relief

- Can be taken anytime, anywhere.[1,2*]
- Excellent choice for patients experiencing nausea and unable to drink water.
- Dissolves on the tongue.
- Effective migraine relief† demonstrated as early as in 30 minutes.[1]
- **Orange** flavoured, orally dispersible tablets.
- Side effect profile consistent with Zomig® 2.5 mg tablet.[2††]

Available in packages of 2 or 6 orally dispersible tablets 2.5 mg

* Zomig® is indicated for the acute treatment of migraine with or without aura. Zomig® is not intended for use prophylactically and is contraindicated in hemiplegic, basilar, or ophthalmoplegic migraine. Safety and efficacy have not been established for cluster headache, which is present in an older, predominantly male population.

†† The most common side effects reported with Zomig® compared to placebo were nausea (9% vs. 3.7%), head/face sensations (8.6% vs. 1.7%), dizziness (8.4% vs. 4%) and neck/throat/jaw sensations (7% vs. 3%).[2]

† Improvement in headache pain (a 1 point drop in headache intensity, 22% vs. 15%, p=0.0385). Double-blind, placebo-controlled, parallel group. Adapted from Purdy A et al.[1] n=470

Zomig® is contraindicated in patients with history, symptoms or signs of ischemic, cardiac, cerebrovascular or peripheral vascular syndromes, valvular heart disease or cardiac arrhythmias (especially tachycardias). In addition, patients with other significant underlying cardiovascular disease, cerebrovascular syndromes or peripheral vascular disease should not receive Zomig®. Zomig® is also contraindicated in patients with uncontrolled or severe hypertension.

Zomig® should not be used within 24 hours of treatment with another 5-HT_1 agonist, or an ergotamine-containing or ergot-type medication like dihydroergotamine or methysergide.

Concurrent administration of MAO inhibitors or use of zolmitriptan within 2 weeks of discontinuation of MAO inhibitor therapy is contraindicated.

AstraZeneca

Zomig Rapimelt™

zolmitriptan orally dispersible tablets 2.5 mg

Convenient migraine relief

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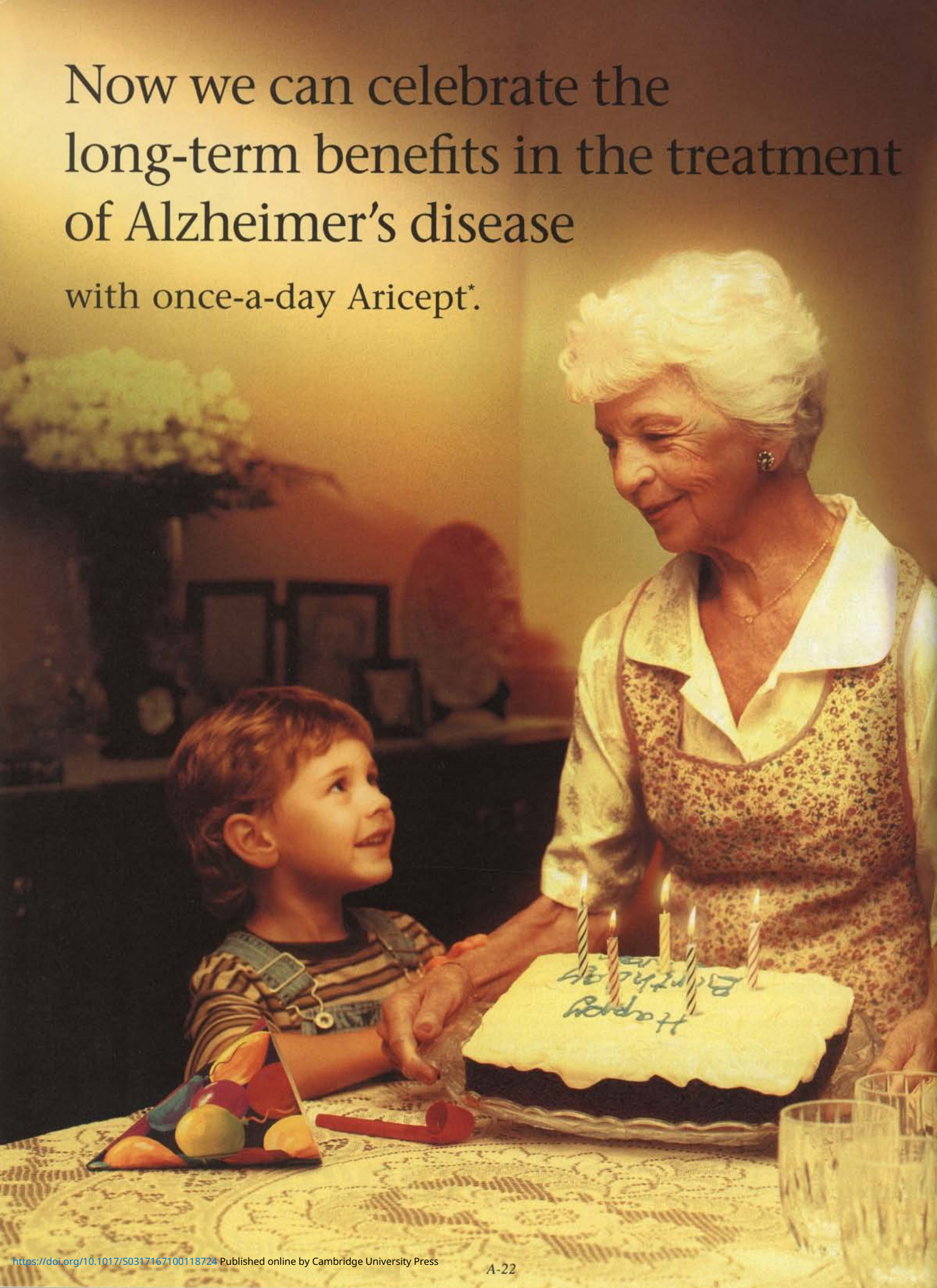

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Celebrate another birthday, another holiday, another family gathering. Because Aricept* has been shown to result in improvement or stabilization in 80% of Alzheimer's disease patients over six months of treatment[1‡] and our new long-term data is even more cause for making Aricept* your standard of care.[2]

After one year, placebo-controlled studies demonstrated that Aricept*-treated patients showed significantly less decline in their cognition, global functioning and Activities of Daily Living.[3,4§¶]

After almost 2 years, Aricept*-treated patients showed significantly less decline in their cognition and global functioning in comparison to data expected from untreated patients.[5††]

After 3 years, Aricept*-treated patients continued to show treatment benefits on cognition and global functioning compared to data expected from untreated patients.[6‡‡]

Aricept* has demonstrated long-term safety and tolerability profiles.[3-6] With appropriate dose escalation, 10 mg/day dose, 5 mg/day dose and placebo were shown to have comparable adverse events.[1†]

With Aricept*, patients may now be able to maintain their autonomy—for a longer time. Now that's cause for celebration.

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.

† 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 52-week, multicentre, double-blind, placebo-controlled study, 286 mild-to-moderate AD patients were randomized to receive Aricept* 5 mg/day for 28 days, followed by 10 mg/day, as per clinician's judgement, or placebo. At study endpoint, significant treatment differences were observed in MMSE in Aricept*-treated patients with mild AD (1.50; $p=0.049$) and moderate AD (2.11; $p=0.002$).

¶ In a 54-week, multicentre, double-blind, placebo-controlled study, 431 mild-to-moderate AD patients were randomized to receive Aricept* 5 mg/day for 28 days, followed by 10 mg/day, or placebo. At study endpoint, significant differences were observed in favour of Aricept* in IADL and ADL ($p=0.001$ and 0.007), and MMSE (1.21; $p=0.0005$). CDR-SB was also improved.

†† In an interim analysis (at 98 weeks of treatment) of a 192-week, multicentre, non-randomized, open-label extension study in which 133 mild-to-moderate AD patients continued to receive Aricept* (up to 10 mg/day) after a 14-week, double-blind, placebo-controlled study. Differences with placebo were observed in cognitive and global functioning as measured by the ADAS-cog and CDR-SB.

‡‡ In a 162-week, multicentre, open-label extension study, 579 patients who had previously completed a randomized, double-blind, placebo-controlled study (n=818) 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.

Product Monograph available upon request.

Now on several provincial formularies.[§§]

Once-a-day

℞ ***Aricept****

donepezil HCl 5 & 10 mg tablets

Hope for a brighter tomorrow

* TM Eisai Co. Ltd., Tokyo, Japan
Pfizer Canada Inc., licensee

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Life is our life's work

§§ In Saskatchewan, Quebec, Alberta, Manitoba and Ontario. Please see individual formularies for special-, exceptional-, and limited-use drug status. For more information on coverage criteria, please call 1-800-510-6141.

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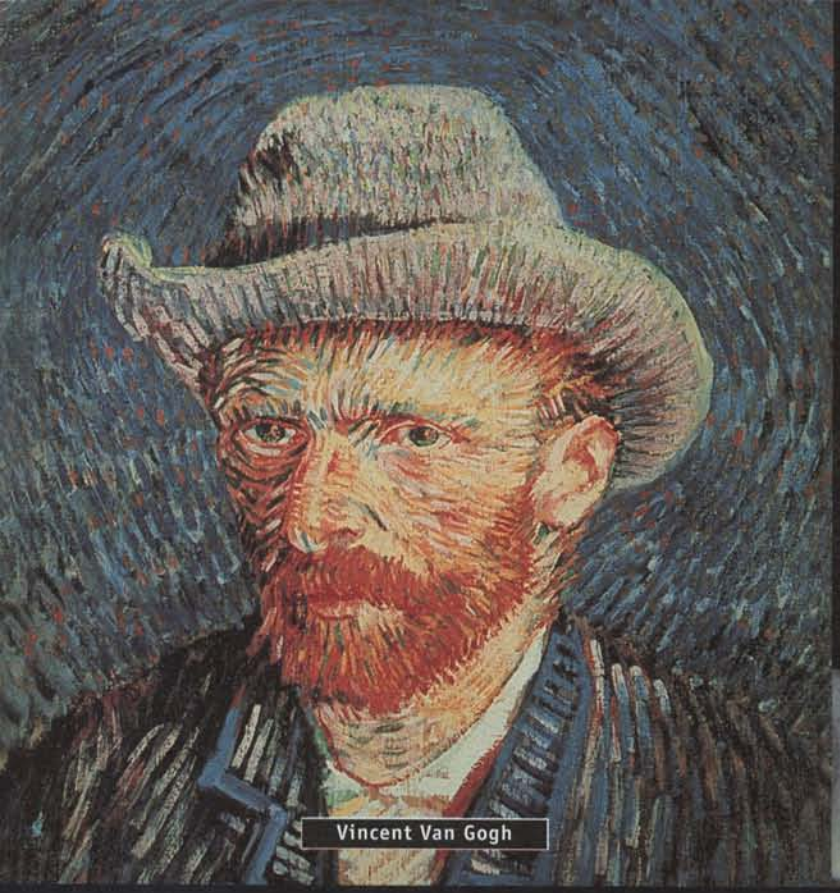
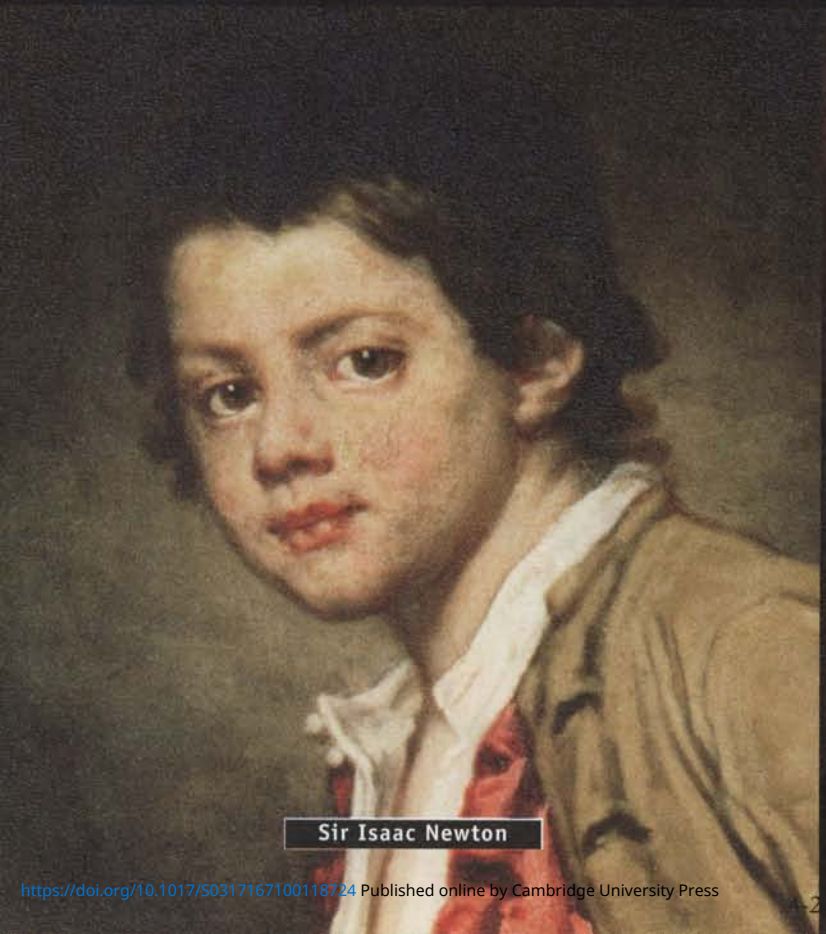


AUPARAVANT, LES PERSONNES ÉPILEPTIQUES DEVAIEN

SE MONTRER EXCEPTIONNELLES POUR RÉUSSIR.



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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-Gastaut[1]
- Des résultats souhaitables avec absence totale de crises chez 19 % des adultes† et 22 % des enfants‡ 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[§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[**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

TOPAMAX*
topiramate

MAINTENANT INDIQUÉ CHEZ L'ENFANT

POUR AIDER LES PATIENTS À MIEUX PROFITER DE LA VIE

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 limités[1].*

†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, Î.-P.-É. Remboursement intégral : Québec, Saskatchewan, Colombie-Britannique, Alberta, Manitoba.
Veuillez 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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Pour documentation voir pages A-34, A-35

25 Years Ago in the Canadian Journal of Neurological Sciences

The Placing Reaction in Adult Neurology

M.I. Botez

SUMMARY: The first description of the placing reaction (PR) in 12 human adult cases is given. The optimum position for eliciting the PR is the dangling leg posture, i.e. the same as for the forward groping of the foot. There are three forms of PR quite similar to those noticed in animal physiology, i.e. the visual-PR, the dorsum-PR and the sole-PR. The term contact placing is preferred for the last two forms. The PR is encountered only in patients displaying a forward and medial groping of the foot as well as a groping of the hand on the same side. The PR is usually ipsilateral to the main cerebral lesion but there was no single case with a well-limited unilateral lesion. An involvement of retrorolandic areas seems to be necessary for the occurrence of PR. It is concluded that both groping phenomena and the PR are highly coordinated reflexes subserving self-preservation and belonging to antigravity mechanisms, i.e. the standing posture.

Can. J. Neurol. Sci. 1976;3:189

Intracranial Dysgerminomas

G. LeBlanc, J. Francouer, M. Copty, C. Contreras and F. Gagne

SUMMARY: We have reported three cases of intracranial dysgerminoma. The origin of these tumors has been controversial for a long time. It is now accepted that they develop from germ cells. The diagnosis is often difficult to confirm because of the variety of signs. If their origin is now accepted, the best way to deal with these lesions is still controversial. They can be macroscopically removed if they are in the pineal region. Such treatment is not possible for suprasellar tumors. Since these tumors can metastasize, radiation should be administered as soon as a pathological diagnosis is obtained. It is our belief that it should be administered to the whole spinal axis.

Can. J. Neurol. Sci. 1976;3:199

Congenital Intraspinal Extradural Cysts (Intraspinal Meningocele)

Dwight Parkinson, A. Chaudhuri and I. Shwartz

SUMMARY: A case of congenital, intraspinal, extradural cyst is reported with pain as the only presenting feature. In the past, the lack of pain has been considered one of the characteristics of these rare lesions. In other reported cases, when pain has been present, it was minimal and never the presenting feature. The clinical and radiographic features and the surgical treatment are described. The pathogenesis is discussed and the literature reviewed. An addition to the surgical technique is given whereby one of the postoperative complications may be obviated.

Can. J. Neurol. Sci. 1976;3:205

Computer Tomography in Disseminated Sclerosis

K.G. Warren, M.J. Ball, D.W. Paty and M. Banna

SUMMARY: A case is reported where the appearance of acute, diffuse, disseminated sclerosis on computer tomography (CT scan) is described and the literature is reviewed. This disease may give rise to multiple, small areas of diminished X-ray absorption which may decrease in size during the course of the disease. The histological features of one of the lesions was correlated with the radiological findings. It is suggested that, perhaps, only during the active stage of demyelination can the lesions be detected on the CT scan.

Can. J. Neurol. Sci. 1976;3:211

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* 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 ß-1a Subcutaneously in Multiple Sclerosis), 1998. Randomised double-blind placebo-controlled study of Interferon ß-1a in relapsing/remitting multiple sclerosis. *Lancet*, 352:1498-1504

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Pour documentation voir pages A-38, A-39

36th Meeting of the

Canadian Congress of Neurological Sciences

Halifax, June 12-16, 2001

Tuesday June 12, 2001

Neurobiology Review Course – full day

ALS Strategies for Quality Life/Quality Care – full day

Movement Disorders Videos – evening

Interesting Headache Case Presentations – evening

Wednesday June 13, 2001

Common Movement Disorders: From the Laboratory to the Clinic – full day

Spinal Course – full Day

Recent Concepts in Electroencephalography – a.m.

Dementia – The Diagnosis, Investigation and Treatment of Lewy Body Disease – a.m.

Assessment and Management of Neuromuscular Disease – p.m.

Controversies in Neurocritical Care – p.m.

Thursday June 14, 2001

Plenary Session I: Transplantation

Parallel Platform Sessions

Poster Sessions

Plenary Session II: Regeneration/Plasticity

Friday June 15, 2001

Plenary Session III: Neuroprotection

Parallel Platform Session

Poster Sessions

Debates (Neurology/Neurosurgery)

Saturday June 16, 2001

Neuropathic pain (half day)
Spinal Cord Injuries (half day)
(with the Canadian Association of Physical Medicine and Rehabilitation)

Child Neurology Day – full day (Oncology)

Stroke Symposium – full day

MS Course – full day

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We are indebted to the expert referees who have reviewed submission to the Canadian Journal of Neurological Sciences in 2000 (names in bold reviewed five or more papers). Their thoughtfulness and expertise has served our journal well. A special thank you to Dr Larry Becker for the years of dedicated service as an associate editor. We welcome Dr. William Fletcher as the new associate editor.

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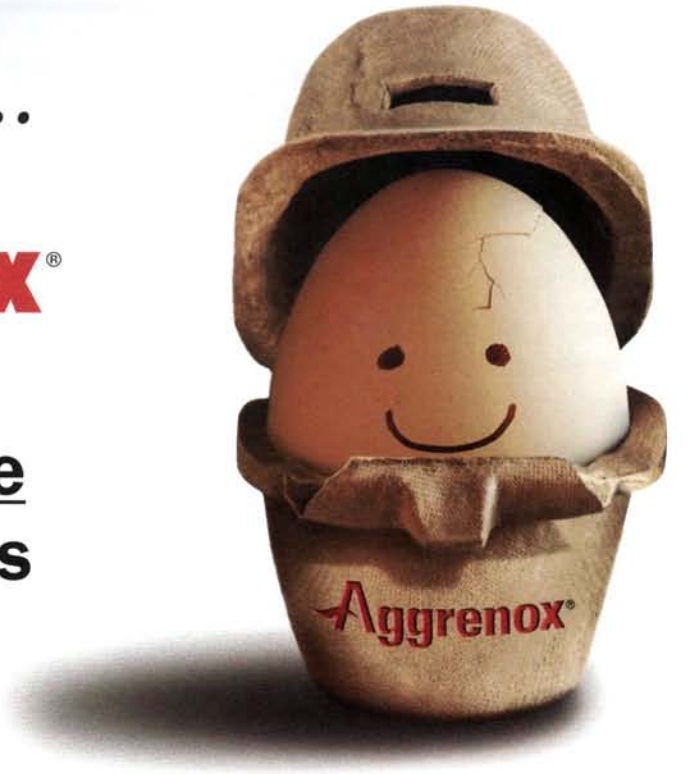

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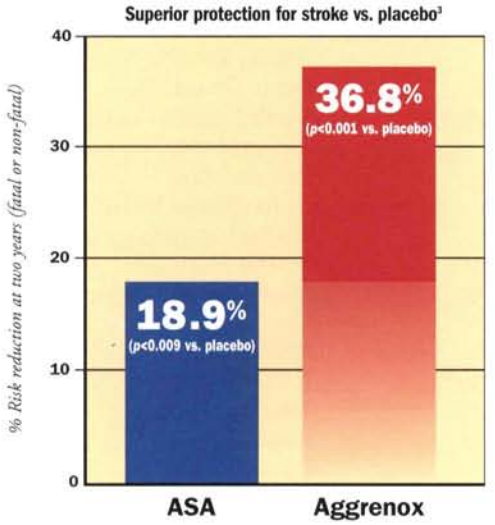

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† percentage of patients experiencing a stroke within two years: Aggrenox 9.5%, ASA 12.5%, placebo 15.2%

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

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