Volume 26 Number 2 May 1999

THE CANADIAN JOURNAL OF Neurological Sciences

LE JOURNAL CANADIEN DES Sciences Neurologiques

AN INTERNATIONAL JOURNAL / UN JOURNAL INTERNATIONAL

34th CANADIAN CONGRESS OF NEUROLOGICAL SCIENCES
June 15 - 19, 1999
Edmonton, Alberta

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

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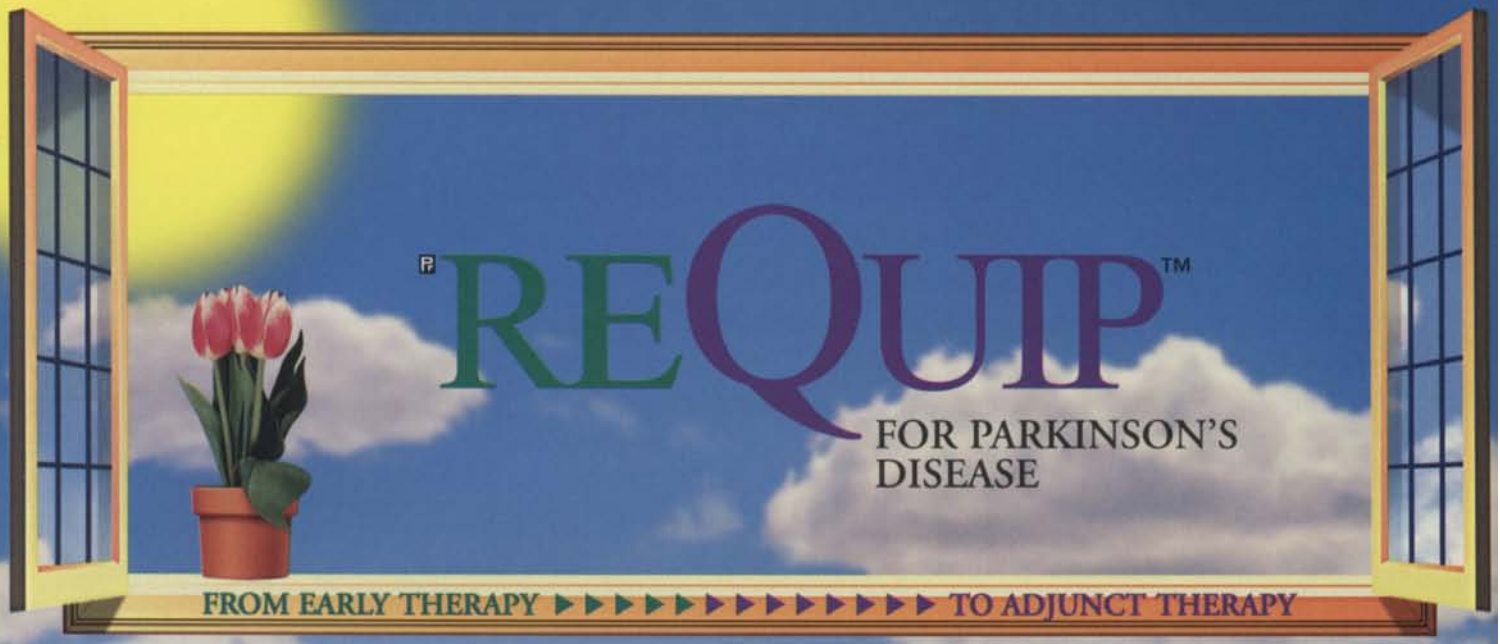
A DOPAMINE AGONIST YOU CAN START WITH AND STAY WITH.



REQUIP IS A NON-ERGOLINE DOPAMINE AGONIST THAT IS INDICATED FOR BOTH EARLY THERAPY WITHOUT LEVODOPA AND ADJUNCT THERAPY WITH LEVODOPA.

EQUALLY EFFECTIVE TO LEVODOPA IN EARLY DISEASE.[1]

After 6 months, ReQuip and levodopa showed no difference in Clinical Global Improvement in patients at Hoehn and Yahr stages I-II, although levodopa showed greater improvement in patients with more severe disease.[1*] As well, ReQuip monotherapy was shown to be significantly more effective than bromocriptine in early disease after 6 months.[2^]

CAN DELAY LEVODOPA FOR AT LEAST 3 YEARS.

Using ReQuip in early disease can sustain symptom control and has been shown to delay the need for levodopa in the majority of patients (61 of 102) who completed a full 3 year study.[3,4]

CONTINUED BENEFITS IN ADJUNCT THERAPY.

ReQuip allowed a 20% or greater reduction in levodopa dose and also increased patients' 'on' time by 20% or more after 6 months of combination therapy.[5*]

SPARING LEVODOPA CAN DELAY COMPLICATIONS.

Because ReQuip spares levodopa in both early and adjunct therapy, it can substantially reduce levodopa load for Parkinson's patients. As a result, ReQuip can delay and reduce long-term levodopa complications such as dyskinesias, 'on-off' effect and 'wearing off' effect.[1,6] So starting ReQuip today can give Parkinson's patients a brighter outlook for tomorrow.

*Mean dosage: 9.7 mg (SD 6.0) ReQuip (n=179), 464.0 mg (SD 266.0) l-dopa (n=89). 95% CI of 0.28, 2.26 Stage I or I.5; 0.43, 3.07 Stage II; 0.04, 0.35 Stage II.5 or III.
^Mean UPDRS improvement in the non-selegiline subgroup. Mean dosage: 9.0 mg (SD 5.2) ReQuip (n=109), 17.2 mg (SD 8.8) bromocriptine (n=101). 95% CI of 6.0%, 21.1%.
*Achieved by 28% of ropinirole (n=94) and 11% of placebo (n=54) treated patients. 95% CI of 1.533, 12.658.[5]
In early therapy[7], nausea (59.9%), dizziness (40.1%) and somnolence (40.1%) were the most common side effects of ReQuip. Postural hypotension occurred in 6.4% of patients.
In adjunct therapy with levodopa[7], dyskinesias (33.7%) and nausea (29.8%) were the most common side effects of ReQuip.

SmithKline Beecham Pharma

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℗REQUIP™
Early Therapy → Adjunct Therapy
RIGHT FROM THE START.

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For brief prescribing information see pages A-34, A-35

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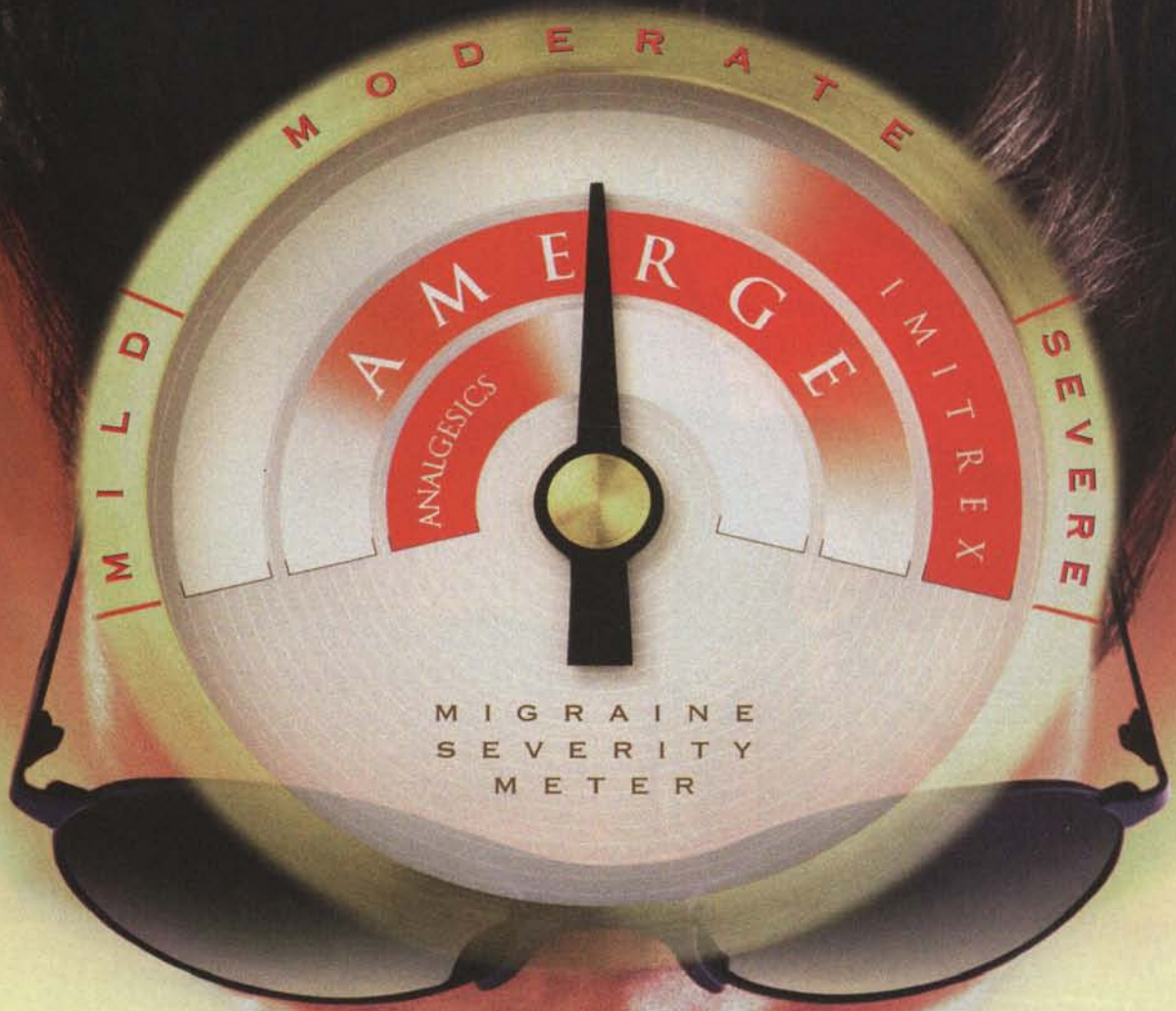
Because not all migraines are created equal...



Highly Tolerable

- Overall incidence of adverse events in controlled clinical trials similar to placebo[1-3†]
  (31% AMERGE 2.5 mg vs. 32% placebo)[2]
- Chest and neck sensations characteristic of the 5-HT_1 agonist class reported in *only* 1.2 – 2.1% of patients[1‡]

Long-lasting Migraine Relief

- Significant migraine relief was sustained over 24 hours[2§]
- 93% of attacks per patient did not require a second dose for recurrence[4||]

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naratriptan hydrochloride

Highly tolerable, long-lasting migraine relief
Available in 2.5mg & 1mg tablets

AMERGE is a 5-HT_1 agonist indicated for the acute treatment of migraine attacks with or without aura. AMERGE is contraindicated in patients with hemiplegic, basilar, or ophthalmoplegic migraine. Safety and efficacy have not been established for cluster headache which is present in an older, predominantly male population.

†AMERGE 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 AMERGE. AMERGE is also contraindicated in patients with uncontrolled or severe hypertension.

‡With 2.5 mg naratriptan. §Headache relief = reduction of moderate or severe pain to mild or no pain. ||Percentage does not represent recurrence rate. Headache recurrence equals a return of moderate or severe pain in 4 to 24 hours post-dose following initial relief.

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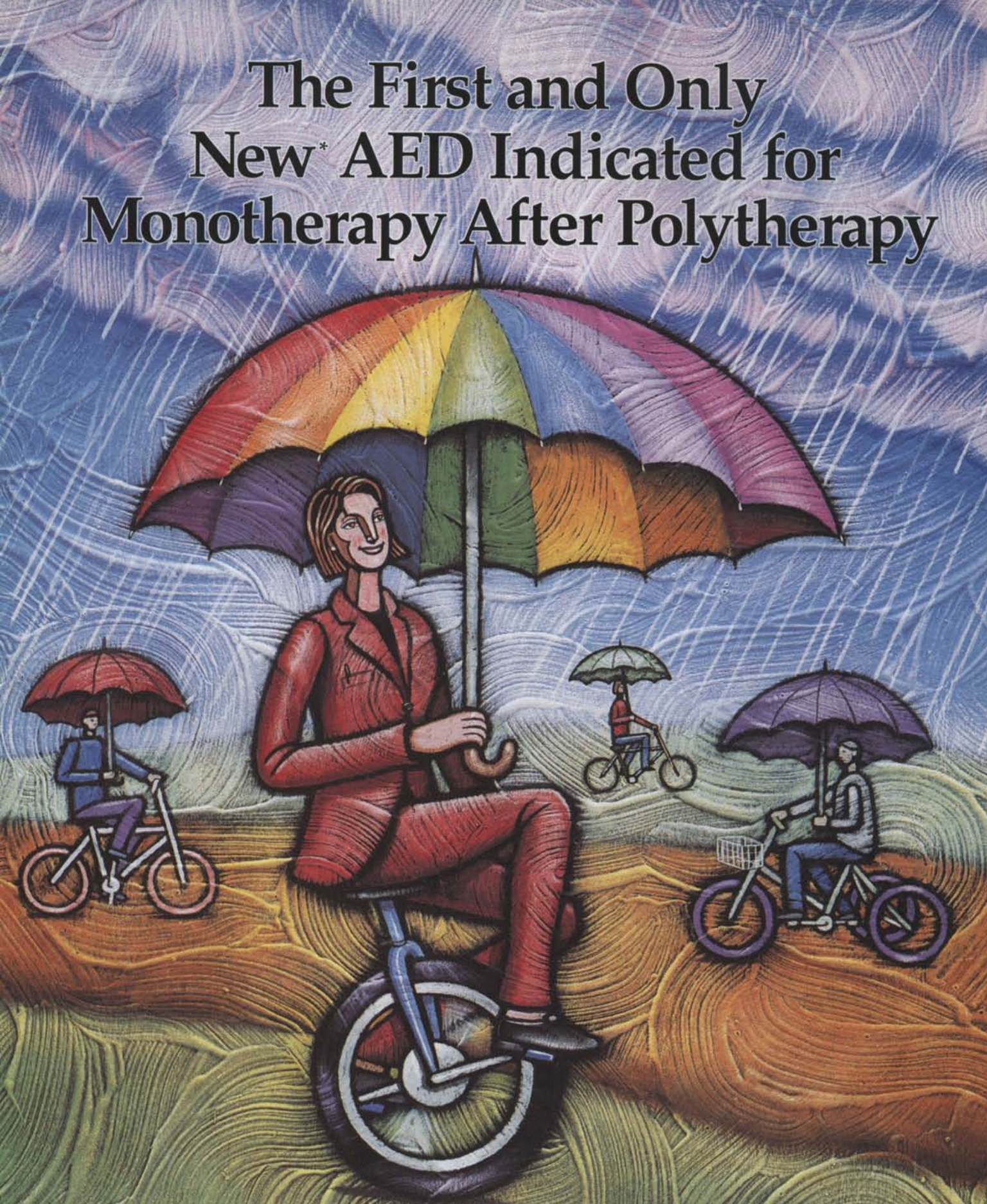
The First and Only New* AED Indicated for Monotherapy After Polytherapy



*Refers to lamotrigine, gabapentin, vigabatrin, and topiramate, to be distinguished from standard AEDs.
**A successful conversion to lamotrigine monotherapy was achieved in 50 of the 69 patients.
*** The three phases included add-on, withdrawal, and monotherapy. Should not be taken as an absolute measure of efficacy because patients with less satisfactory responses did not progress into all phases.
†The most common adverse experiences associated with discontinuation of LAMICTAL monotherapy were rash (6.1%), asthenia (1.1%), headache (1.1%), nausea (0.7%), and vomiting (0.7%).[3] See Product Monograph for further information.
††Please refer to Product Monograph for dose adjustment of LAMICTAL according to the concomitant AED withdrawn.

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For Control Over a Wide Range of Seizure Types, with a Low CNS Side-Effect Profile

Effective monotherapy has been largely accepted as the regimen of choice for achieving seizure control with minimal side effects in the management of patients with epilepsy.[1] Now, extending its proven success as adjunctive therapy,[2] LAMICTAL is indicated for monotherapy in adults following withdrawal of concomitant antiepileptic drugs (AEDs).[3]

HIGHLY EFFECTIVE MONOTHERAPY

In one add-on/withdrawal to monotherapy open-label trial, LAMICTAL monotherapy following withdrawal of concomitant AEDs kept 30% (n=50) of the successfully treated patients seizure-free.**[4] In a similarly designed trial, ≥ 40% of the patients were maintained with at least 50% reduction of seizure frequency across all phases of the trial.***[5]

GENERALLY BETTER TOLERATED†

Pooled data from three monotherapy trials show that withdrawals due to CNS-related side effects were 2.5% (n=443) with LAMICTAL monotherapy compared to phenytoin (7.4%; n=95) or carbamazepine (7.7%; n=246).[6] Incidence of somnolence, asthenia, and ataxia were reported less frequently with LAMICTAL compared to carbamazepine and phenytoin. There was no difference in the rate of withdrawal due to skin rash between LAMICTAL (6.1%) and phenytoin (5.3%) or carbamazepine (8.9%).[6] A higher incidence of skin rash has been associated with more rapid initial titration of LAMICTAL or use of concomitant valproic acid.[3]

CONTROL OVER A WIDE RANGE OF SEIZURE TYPES

LAMICTAL add-on polytherapy has been successfully used across a wide range of seizure types.[2] Now, opt to switch with confidence from LAMICTAL polytherapy to LAMICTAL monotherapy,†† particularly when you are concerned with CNS-related side-effects.

Lamotrigine

Lamictal®

FROM POLYTHERAPY TO MONOTHERAPY

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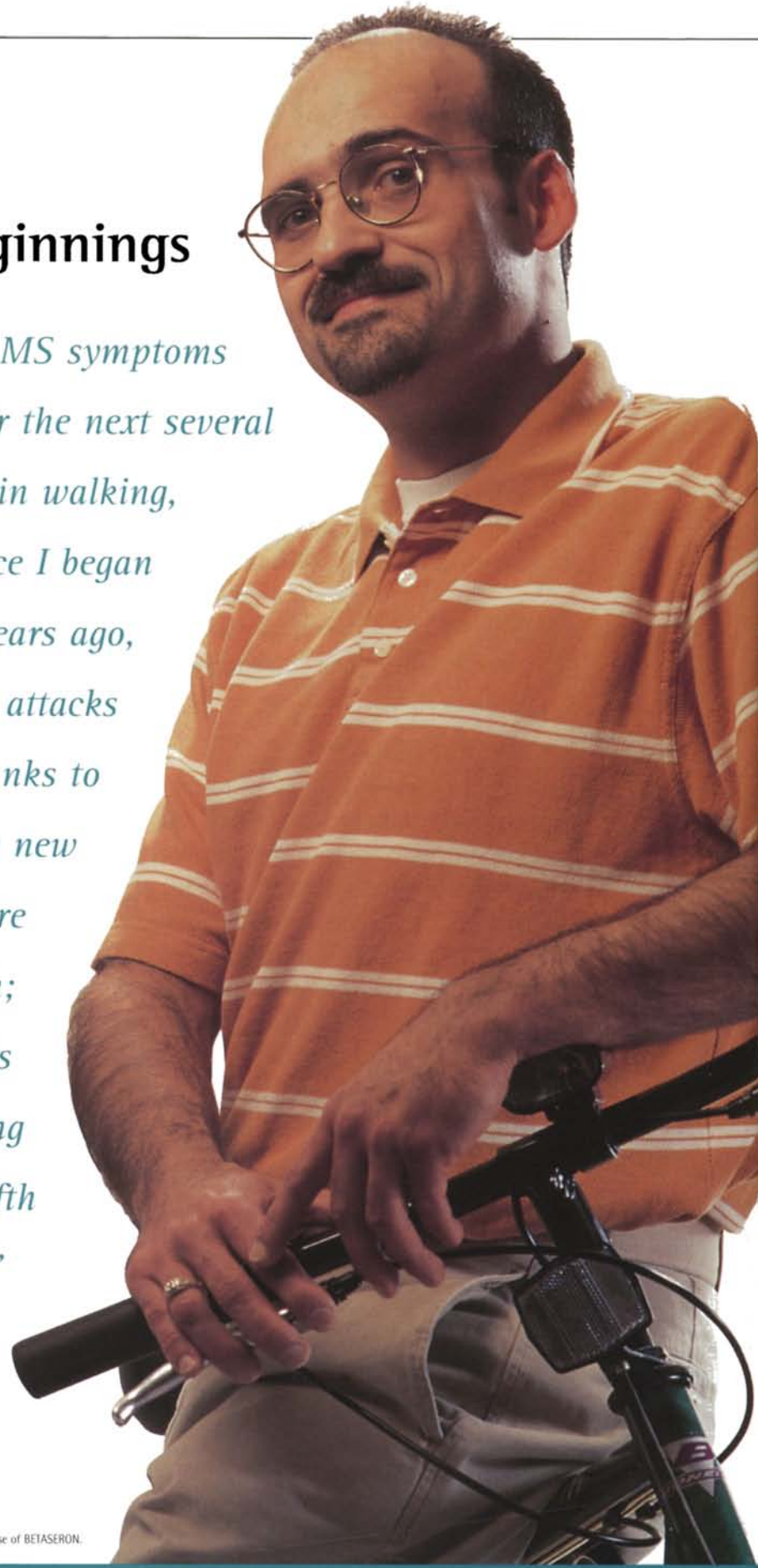
For brief prescribing information see pages A-36, A-37

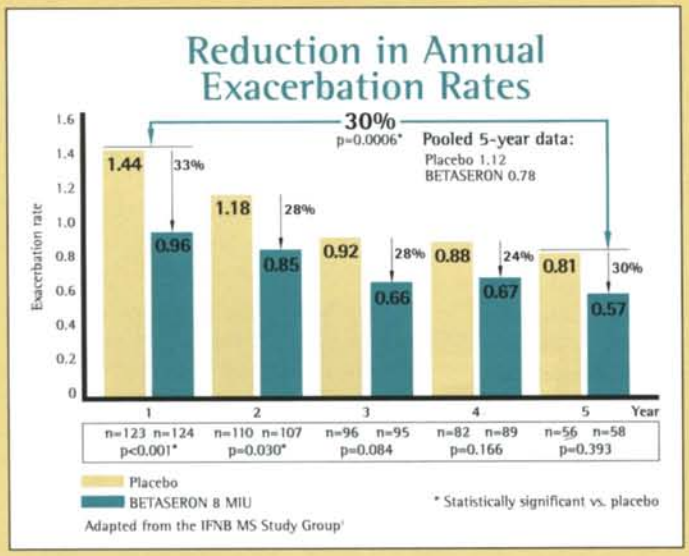
Fred on New Beginnings

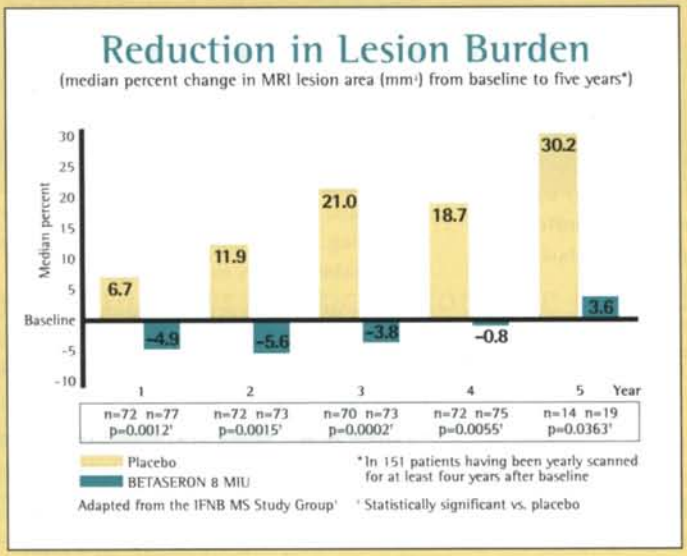
"I first experienced MS symptoms in 1985. Over the next several years, I had difficulties in walking, with speech ... But since I began BETASERON, *11 years ago, I have had no attacks whatsoever. Thanks to* BETASERON, *I now face new challenges. I can be there for my children; I'm preparing my master's degree; and I'm getting ready for my fifth MS bike-a-thon!"*



This real BETASERON patient testimonial may not be representative of all cases involving the use of BETASERON.

THE FIRST TREATMENT ALTERING MS NATURAL HISTORY





- **Proven long-term efficacy as first-line treatment in relapsing-remitting MS**
  - Over five years, exacerbation rate reduced by 30% (p=0.0006)[1]
  - At two years, moderate and severe exacerbations reduced by 49% (p=0.002);[2] significant annual reduction maintained over five years[1]
  - Median time to first exacerbation twice as long as in placebo patients (p=0.015)[2]
- **The only treatment studied for five years, both clinically and with MRI[1]**
  - MRI measured *lesion burden* significantly reduced over five years[1]
  - MRI measured *lesion activity* decreased (a median of 80% fewer active scans compared to placebo; p=0.0062; measured for two years)[3]
- **Trend toward slower disability progression demonstrated over a five year period[1]**
- **Low incidence of serious side effects:[1] injection-site reactions and flu-like symptoms are manageable and lessen markedly with time[4]**

More than 60,000 patients treated to date worldwide[5]

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WITH LONG-TERM BENEFICIAL EFFECTS FOR MS PATIENTS

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• Submit five high quality copies of the manuscript and original illustrations. Papers will be accepted in English or French. Manuscripts must be double spaced throughout including references, tables and legends for illustrations. Margins of at least 25mm should be left on all sides.

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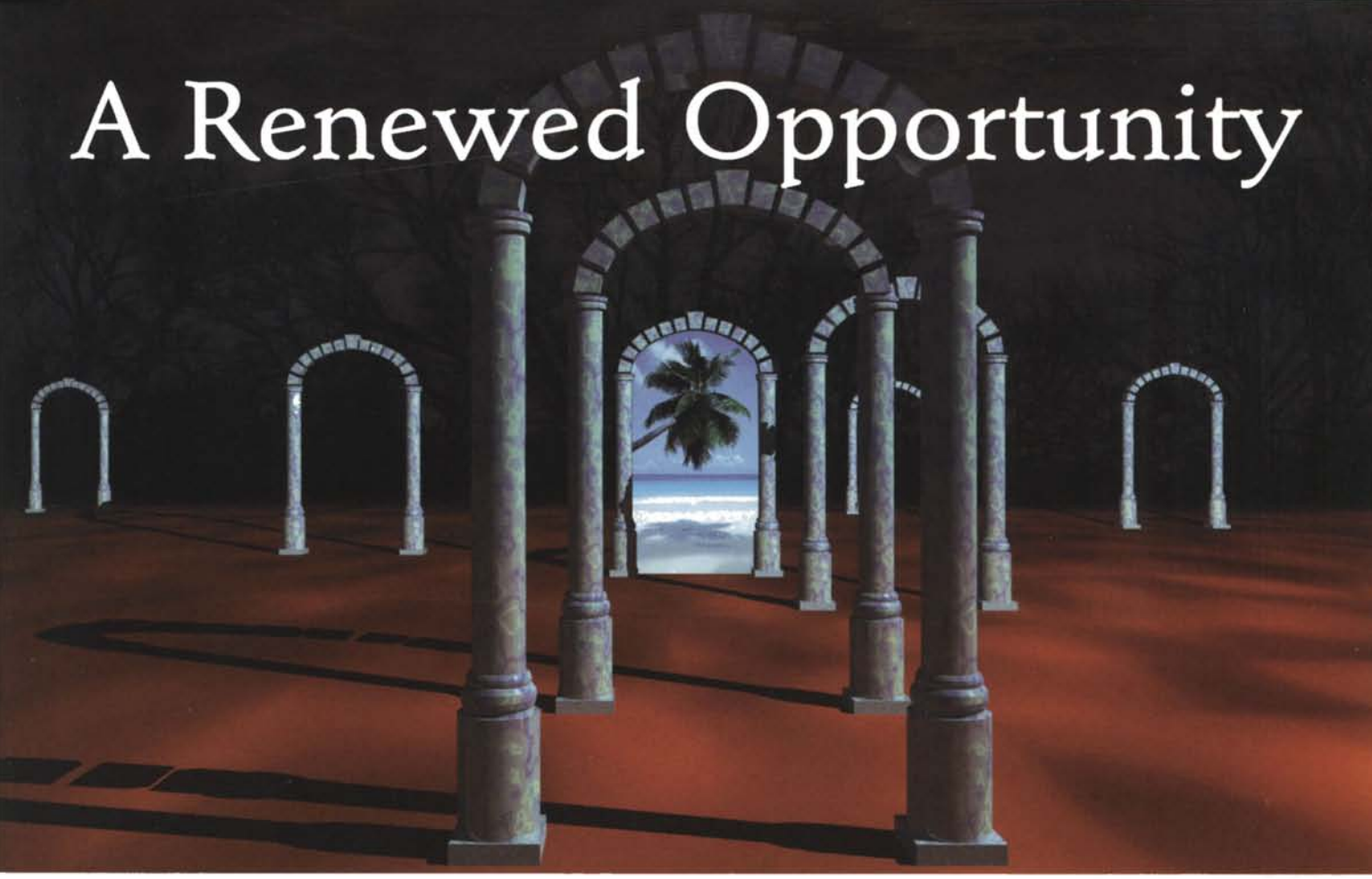

PARKINSON'S DISEASE

A world in which the therapeutic options are limited[1]

For those who have it, treat it, live with it; managing their Parkinson's disease can be quite frustrating. Yet there are moments that can be most rewarding. Motor function improves, the number of "off" hours decreases, rigidity subsides, gait improves. Their levodopa seems to be working... at least for today! Then there are times when nothing seems to help. Even their medication seems to be causing problems. It seems hopeless...

Today, however, there is another way to renew their hope. Even after its discovery more than fifteen years ago, Permax (pergolide mesylate) is still considered the most potent dopamine agonist available for the treatment of Parkinson's disease.[1-3] With its unique mode of action, i.e. stimulating both D_1 and D_2 dopamine receptors, Permax has demonstrated (n=376) statistically significant improvement in virtually all those numerous parameters of parkinsonian function, including bradykinesia, rigidity, gait, dexterity, etc. Equally important, these benefits were achieved with significantly less levodopa... 24.7% ($p < .001$), and by starting Permax at low doses "Adverse reactions were, for the most part, mild, reversible, and not of major clinical significance."[3]*

Successful treatment with Permax can last for up to 3-5 years[4,5] and renewed improvement has been demonstrated when Permax was given to patients (n=10) in whom the beneficial effect of bromocriptine had waned,[4] whereas the reverse was not true in a separate, non-comparable study (n=11) when bromocriptine was given to Parkinson's patients in whom Permax had waned.[6]

So, when given an opportunity to manage Parkinson's disease, there may be a way of renewing hope.

PERMAX®
pergolide mesylate

DRAXIS

Draxis Health Inc.
Mississauga, Ontario

PAAB

* Rapid escalation of pergolide dosage may cause severe adverse reactions. Therefore a slow increase combined with a concomitant gradual and limited reduction of levodopa is recommended. See ADVERSE REACTIONS section in Prescribing Information

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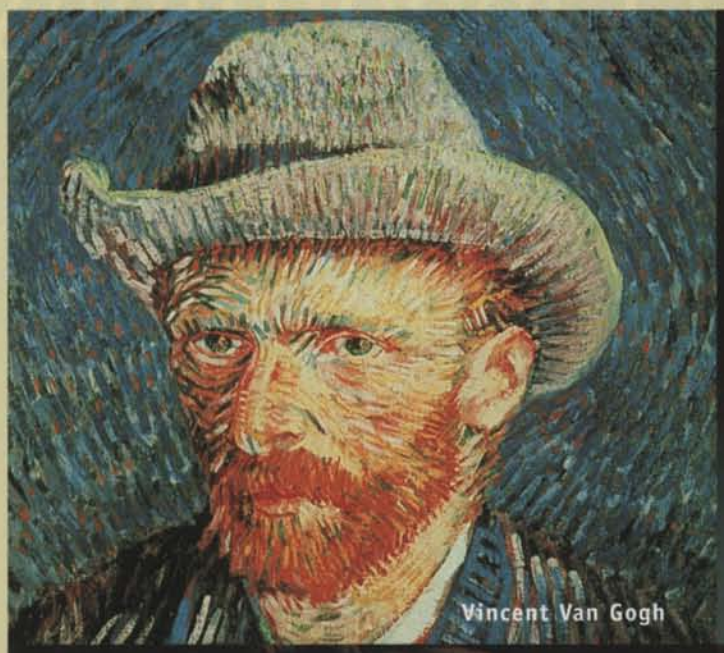
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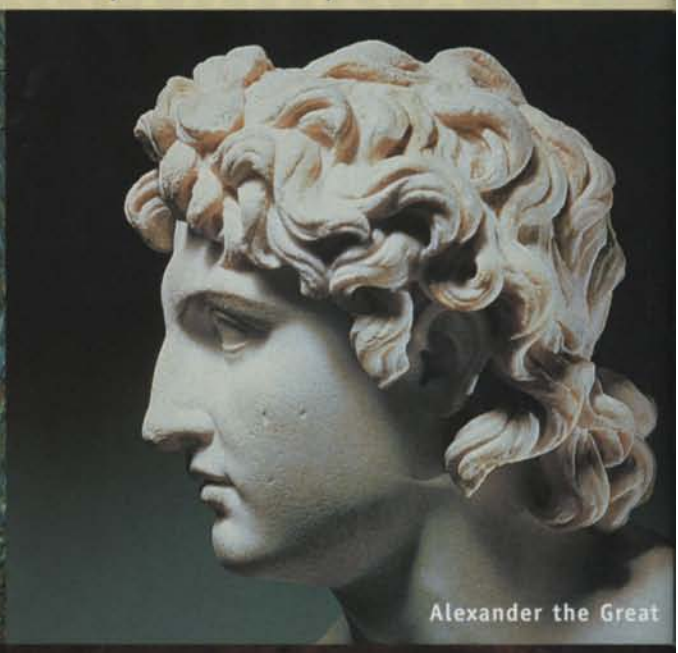
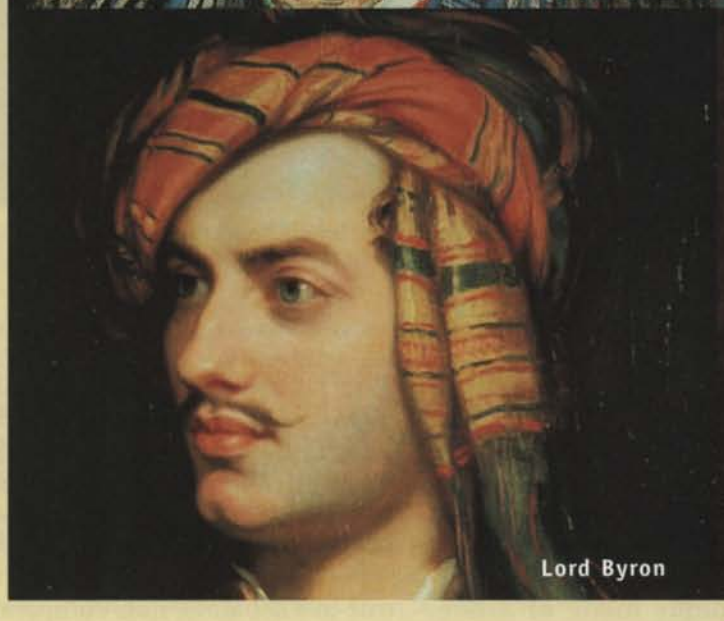
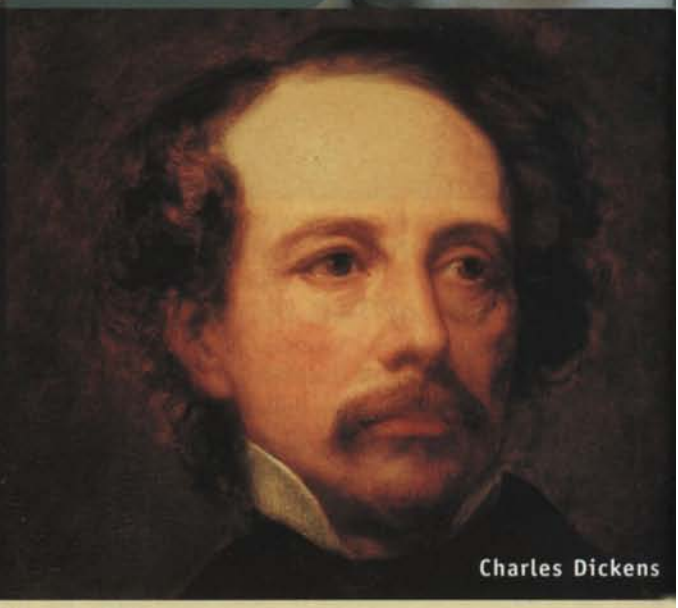
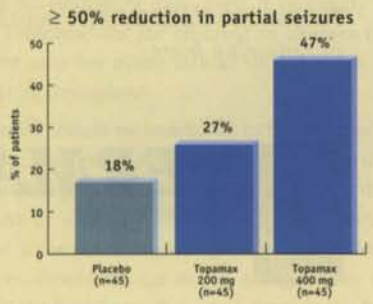

Alexander the Great


Lord Byron


Charles Dickens

ONCE IT TOOK EXCEPTIONAL EFFORT OR EXTRAORDINARY LUCKILY, YOUR PATIENTS CAN NOW



Adapted from reference 1. Double-blind trial of placebo vs. TOPAMAX b.i.d. as adjunctive therapy in 181 patients with refractory partial onset epilepsy receiving one or two other AEDs. *p=0.013.

Improved control over a wide range of seizure types

- TOPAMAX is indicated as adjunctive therapy for the management of patients with epilepsy who are not satisfactorily controlled with conventional therapy. There is limited information on the use of TOPAMAX in monotherapy at this time.
- High responder rate: 27%(200mg/day, n=45) and 47% (400mg/day, n=45) of patients experienced ≥ 50% reduction in partial seizures(16 week study)[1]
- Effective control for patients with secondarily generalized tonic-clonic seizures: 36% of patients experienced a 100% reduction (200-600 mg, n=42, 16 week study)[1]
- Unique three-way mechanism of action(Na+ channel blockade, GABA potentiation, glutamate antagonism)[2]

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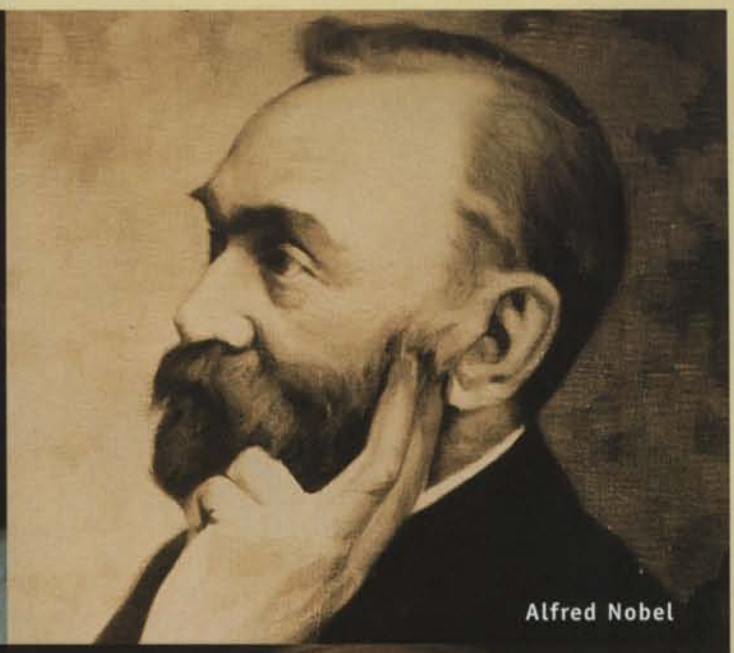
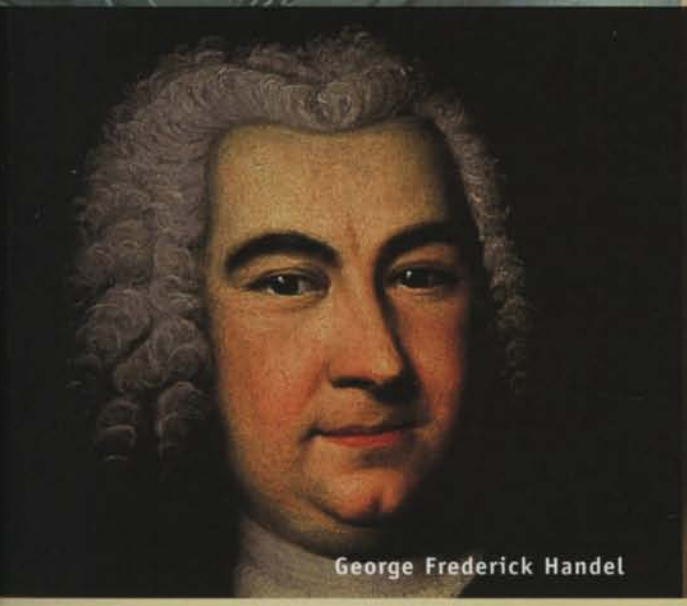
NOVA SCOTIA & QUEBEC FORMULARIES.


Joan of Arc


Alfred Nobel


George Frederick Handel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- Generally well tolerated: Discontinuations due to adverse events were 10.6% at 200-400 mg/day compared to 5.8% in the placebo group (this appeared to increase at dosages above 400 mg/day)[2]
- No evidence of serious rash or aplastic anemia[2]
- Dosage adjustments to primary therapy are generally not required; patients on phenytoin showing clinical signs or symptoms of toxicity should have phenytoin levels monitored†[2]
- **Convenient BID dosing**

Favourable side effect profile
(the most common are CNS related)

	TOPAMAX 200-400 mg (n=113)	PLACEBO (n=216)
Somnolence	30.1	9.7
Dizziness	28.3	15.3
Ataxia	21.2	6.9
Psychomotor slowing	16.8	2.3
Speech disorders	16.8	2.3
Nervousness	15.9	7.4
Nystagmus	15.0	9.3
Paresthesia	15.0	4.6

†As with other AEDs, please see prescribing information for complete information on drug interactions. A 1.5%(n=1715) incidence of kidney stones has been reported.[1] In one study(n=1200), 83%(15 of 18) of patients elected to continue therapy.[4] Ensure adequate hydration and avoid concomitant use with other carbonic anhydrase inhibitors.[1] *Trademark © Janssen-Ortho Inc. 1997

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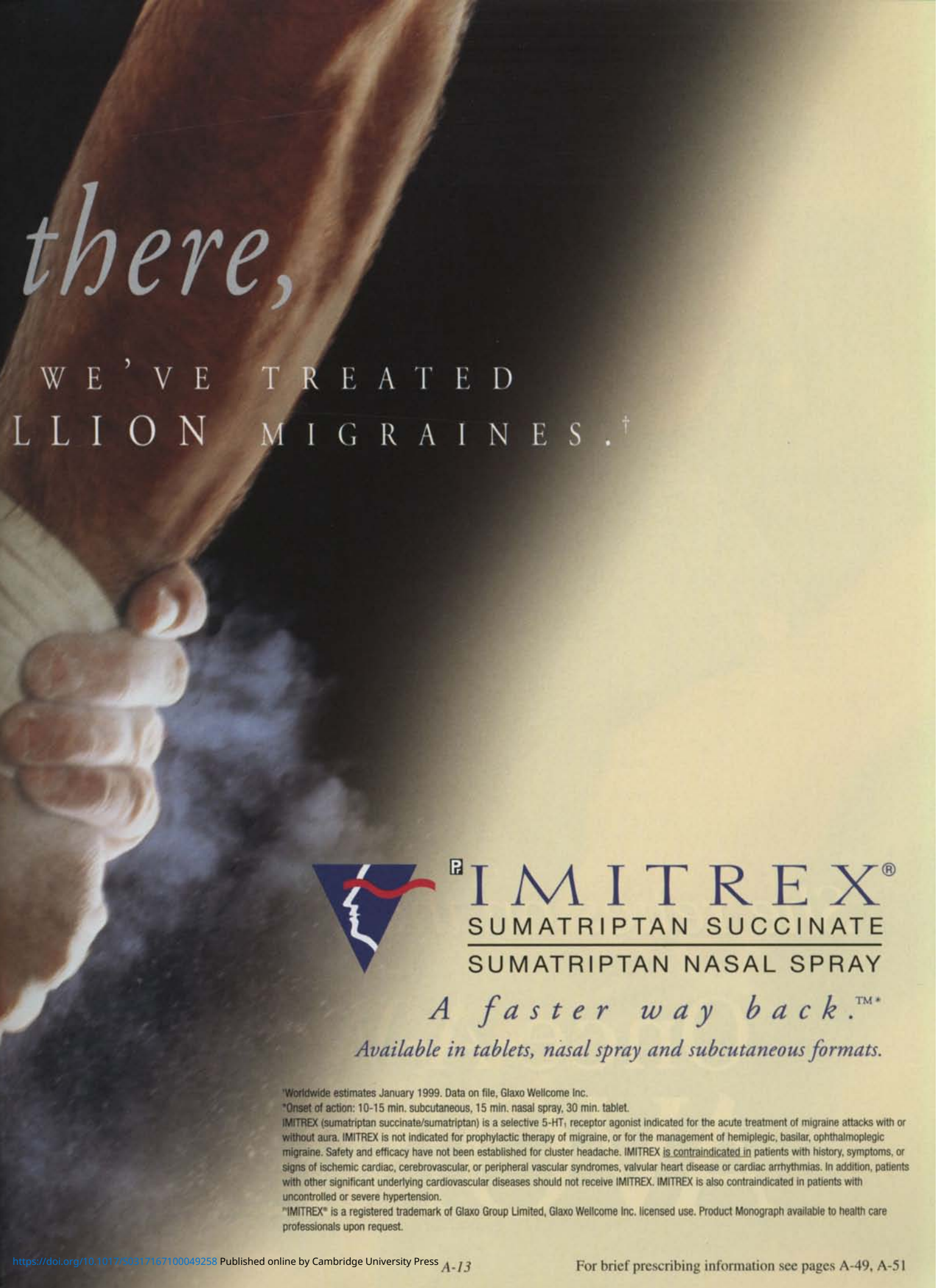

†Worldwide estimates January 1999. Data on file, Glaxo Wellcome Inc.

*Onset of action: 10-15 min. subcutaneous, 15 min. nasal spray, 30 min. tablet.

IMITREX (sumatriptan succinate/sumatriptan) is a selective 5-HT_1 receptor agonist indicated for the acute treatment of migraine attacks with or without aura. IMITREX is not indicated for prophylactic therapy of migraine, or for the management of hemiplegic, basilar, ophthalmoplegic migraine. Safety and efficacy have not been established for cluster headache. IMITREX is contraindicated in patients with history, symptoms, or signs of ischemic cardiac, cerebrovascular, or peripheral vascular syndromes, valvular heart disease or cardiac arrhythmias. In addition, patients with other significant underlying cardiovascular diseases should not receive IMITREX. IMITREX is also contraindicated in patients with uncontrolled or severe hypertension.

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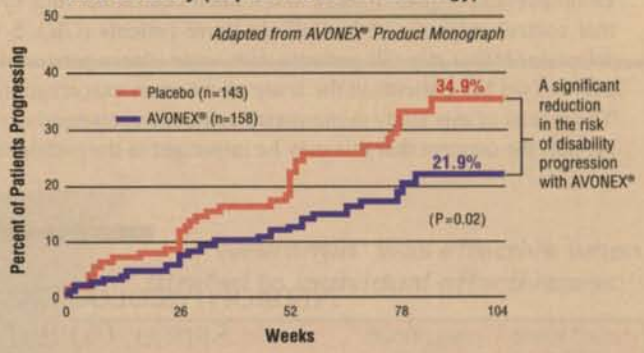
Proven to slow the progression of disability in relapsing forms of MS.[1]

- Patients treated with AVONEX® showed a significant reduction in risk of disability progression and a 32% reduction in annual exacerbation rate over two years.[2]
- AVONEX® also demonstrated a significant MRI effect, showing an 89% reduction in gadolinium-enhanced lesions in patients with enhancement at baseline.[3]
- Prescribed for more than 50,000 patients worldwide, now available in Canada.[4]

Onset of sustained disability progression by time on study (Kaplan-Meier Methodology)[1]



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

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PAAB

Please see product monograph for important patient selection and monitoring information.

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

25 Years Ago in the Canadian Journal of Neurological Sciences

Progress in Understanding Huntington's Chorea

André Barbeau

Summary: This report is based on presentations and discussions during three recent meetings on Huntington's Chorea: a meeting of the Research Group on Huntington's Chorea of the World Federation of Neurology (Leuven, Belgium, 8-12 September, 1974); a Symposium on Huntington's Chorea of the Huntington Society of Canada (Toronto, Canada, 8-9 November, 1974); and a Workshop of the Huntington's Disease Foundation (Los Angeles, U.S.A., January 10-12, 1975). To the lecturers and discussors of these meetings belong many of the new ideas expressed herein, even if they are not acknowledged by name.

Can. J. Neurol. Sci. 1975;2:81

Cellular Hypersensitivity to Basic Myelin (P2) Protein in the Guillain-Barré Syndrome

William Sheremata, Susan Colby, Y. Karkhanis, and Edwin H. Eylar

Summary: Lymphocytes of the 29 subjects were assayed for MIF production in response to P2 peripheral nerve protein, crude human peripheral nerve and human central nervous system A1 basic myelin protein. Seven were performed in normal control subjects, 12 in Guillain-Barré patients (GB), 5 with other poly neuropathies and 5 in patients with multiple sclerosis (MS). Only GB patients with acute illness produced MIF in response to neuritogenic P2 protein and crude human nerve. Two MS patients in the acute phase of an exacerbation and one GB patient produced MIF in response to A1 protein. The results of this study demonstrate cellular hypersensitivity to a neuritogenic constituent in peripheral nervous tissue and support the concept that this may be important in the pathogenesis of GB.

Can. J. Neurol. Sci. 1975;2:87

Neurophysiological Changes Following Spinal Cord Lesions in Man

P. Ashby and M. Verrier

Summary: A study has been made of the neurophysiological changes that follow spinal cord lesions in man. The Achilles tendon reflex (ATR) is used to estimate transmission in the Ia monosynaptic pathway, and the tonic vibration reflex (TVR) to estimate transmission to the Ia polysynaptic pathway to motoneurons. The inhibition of the H reflex by vibration is used as an estimate of presynaptic inhibition of the Ia monosynaptic pathway. Immediately following a complete lesion of the spinal cord, presynaptic inhibition of the Ia monosynaptic pathway appears to be greatly increased. This enhanced inhibition may last several months but it eventually declines and in some instances becomes less than normal. Transmission in the Ia polysynaptic pathway is permanently abolished by a complete spinal lesion. An hypothesis is developed from these findings to explain the evolution of some of the clinical features that follow complete spinal lesions in man. Distinct differences are observed when the spinal lesion is incomplete. Transmission in the Ia polysynaptic pathway may be preserved and there may be no increase in presynaptic inhibition. These differences may depend upon the integrity of certain spinal long tracts which cannot be tested clinically.

Can. J. Neurol. Sci. 1975;2:91

25 Years Ago in the Canadian Journal of Neurological Sciences

Meningo-encephalomyelitis due to the Saprophagous Nematode, Micronema Deletrix

Jan Hoogstraten and W. Gerard Young

Summary: A five-year-old boy succumbed 24 days following an unusual farm accident in which considerable manure was deposited in multiple lacerations. Death was due to an extensive meningo-encephalomyelitis caused by a nematode that is ordinarily saprophagous.

Can. J. Neurol. Sci. 1975;2:121

Isolated Spinal Cord Arteritis

Thomas E. Feasby, Gary G. Ferguson and J.C.E. Kaufman

Summary: This patient presented as a subacute progressive cervical myelopathy and the differential diagnosis included cervical spondylotic myelopathy and intramedullary mass. Microscopically, vascular lesions plus a patchy myelomalacia indicated a vasculitis. However, there was no suggestion of a generalized vasculitis at autopsy and the only supporting laboratory study was a raised erythrocyte sedimentation rate. It would seem that a vasculitis similar to polyarteritis nodosa or other collagen disease may be confined to the spinal cord.

Can. J. Neurol. Sci. 1975;2:143

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25 Years Ago in the Canadian Journal of Neurological Sciences

Relative Prognostic Significance of Vasospasm Following Subarachnoid Hemorrhage

Bryce Weir, Charles Rothberg, Michael Grace and Faye Davis

Summary: A retrospective analysis of 274 patients with intracranial aneurisms, diagnosed either angiographically or at autopsy between 1968 and 1973 at the University of Alberta, was carried out. One hundred and forty-six patients had intracranial clipping of the aneurism. Clinical and radiologic data were abstracted from the chart and the angiographic studies. Probability of survival curves were constructed. Association between various clinical factors and survival at two months were demonstrated. The most important prognostic factors are the clinical grade at angiography or surgery, followed by the presence of pre-operative spasm, hematoma or focal edema, elevated blood pressure on admission, time interval from hemorrhage to surgery and age. The data lends some support to the policy of operating on patients in good neurological condition, even if the pre-operative angiogram shows spasm.

Can. J. Neurol. Sci. 1975;2:109

The Chiari Malformation in Adults

F.B. Maroun, J.C. Jacob, M. Mangan

Summary: The clinical features of the Chiari Malformation in seven adult patients are presented. It is suggested that the clinical syndromes associated with this malformation, in adults, can be classified as (a) compression of structures at the level of the foramen magnum (with or without radiologically demonstrable associated bony anomaly at the cranio-vertebral junction) (b) increased intracranial pressure of obstructive hydrocephalus and (c) intramedullary cervical cord syndrome. The usefulness of tomography, and demonstration of the vertebro-basilar circulation in the neuro-radiologic investigation of these patients is emphasized. The surgical procedures performed in the management of these patients are outlined.

Can. J. Neurol. Sci. 1975;2:115

Giant Intracranial Dermoid Cyst: Case Report and Review of the Literature on Intracranial Dermoids and Epidermoids

Neil R. Miller and Melvin H. Epstein

Summary: A 45-year old man was referred to the Johns Hopkins hospital with a seven-year history of repeated episodes of light-headedness, increasing irritability, and forgetfulness. examinations revealed a right superior, incongruous quadrantanopsia. EEG showed an abnormality in the left temporal lobe, and a cerebral angiogram outlined an avascular mass in the left cerebral hemisphere. At operation the patient was found to have a giant dermoid cyst involving the left frontal, temporal and parietal lobes. Over the last 30 years we have encountered only 6 cases of intracranial epidermoids and 3 cases of intracranial dermoids. These cases are cited and a discussion of the embryology, histology, clinical characteristics, and treatment of these lesions with a review to the literature is undertaken.

Can. J. Neurol. Sci. 1975;2:127

25 Years Ago in the Canadian Journal of Neurological Sciences

Compression of the Cauda Equina due to a Necrobiotic Granuloma of Ligamentum Flavum

Juan M. Bilbao, William Horsey, Charles Gonsalves and Ara Chalvardjian

Summary: A 56-year old woman developed symptoms of lumbar nerve root compression caused by a granuloma arising in the ligamentum flavum. The histological features of the lesion are discussed and the clinical and radiological findings of the patient are described.

Can. J. Neurol. Sci. 1975;2:135

Fetal Cerebellar Tissue Associated with a Primitive Neuro-epithelial Tumor in an Ovarian Teratoma

Paul J. Boor and William C. Schoene

Summary: This is a singe case report of an ovarian teratoma. It is a unique case of a primitive neuroepithelial tumor with many similarities to a medullo-blastoma arising in an ovarian teratoma, and the second report of fetal cerebellum occurring in a teratoma of the ovary.

Can. J. Neurol. Sci. 1975;2:139

For brief prescribing information see page A-26

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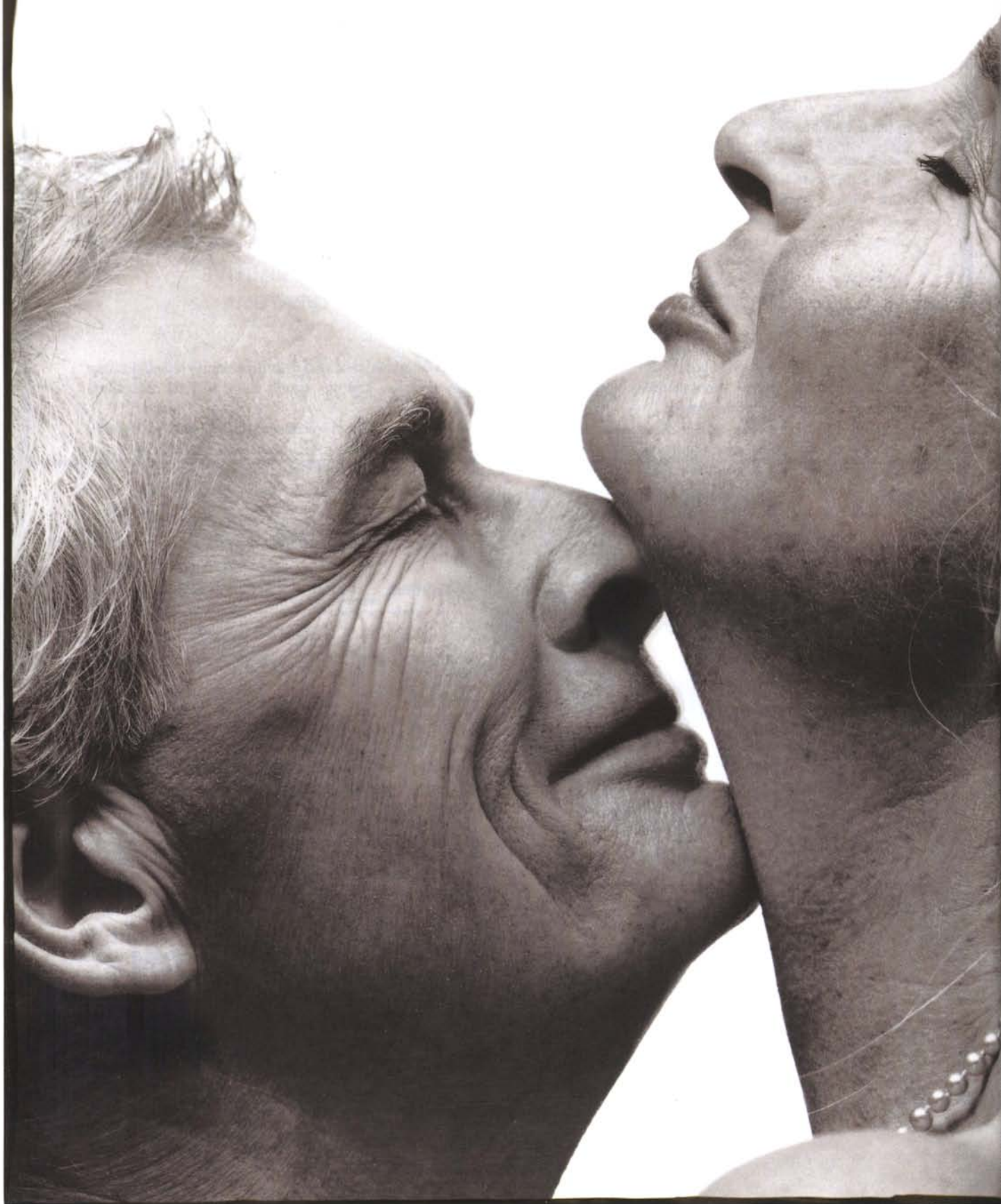
If the closeness of their relatio



nship becomes a distant memory, we've got an answer.

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The ease of oral treatment to help restore natural erectile function, that works only with sexual stimulation.

Erectile dysfunction is a serious medical condition estimated to affect 2-3 million couples.[1,2†] Yet less than 10% of men seek help.[3‡] VIAGRA may help to change all this. Clinical studies have demonstrated a 78% improvement in erections versus 20% for placebo.[4§] Adverse events were generally transient and mild-to-moderate in nature.[5¶] Finally, a new oral treatment to help couples rediscover their sexual intimacy.

VIAGRA has been shown to potentiate the hypotensive effects of nitrates and is therefore contraindicated in patients who are taking any type of nitrate drug therapy, either regularly and/or intermittently, in any form (e.g., oral, sublingual, transdermal, by inhalation).[5] Treatment for erectile dysfunction should not generally be used in men for whom sexual activity is inadvisable.[5**]

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† Derived from Canadian census data (ages 40-69) and published U.S. prevalence rate.[1,2]

‡ Approximation based on number of males reporting to physicians for impotence.

§ $p < 0.0001$. Results from 12-week, double-blind, placebo-controlled, flexible-dose (25-100 mg) studies in ED patients. VIAGRA: n = 278; placebo: n = 262.[4] Response varies depending upon etiology of disorder.

¶ Most frequently reported adverse events in controlled clinical trials were headache (15.8%), flushing (10.5%), dyspepsia (6.5%) and nasal congestion (4.2%).[5] Abnormal vision (2.7%) was mild and transient, predominantly impairment of colour discrimination (blue/green), but also increased perception to light or blurred vision.[5]

** Please consult prescribing information for complete patient selection criteria.

VIAGRA is indicated in the treatment of erectile dysfunction.[5]

In most patients, the recommended starting dose is 50 mg, taken as needed, approximately one hour before sexual activity — no more than one dose/day. A starting dose of 25 mg should be considered in patients with: ≥ age 65 years, hepatic impairment, severe renal impairment and concomitant use of potent cytochrome P-450 3A4 inhibitors (e.g., erythromycin, ketoconazole, itraconazole).

Pfizer

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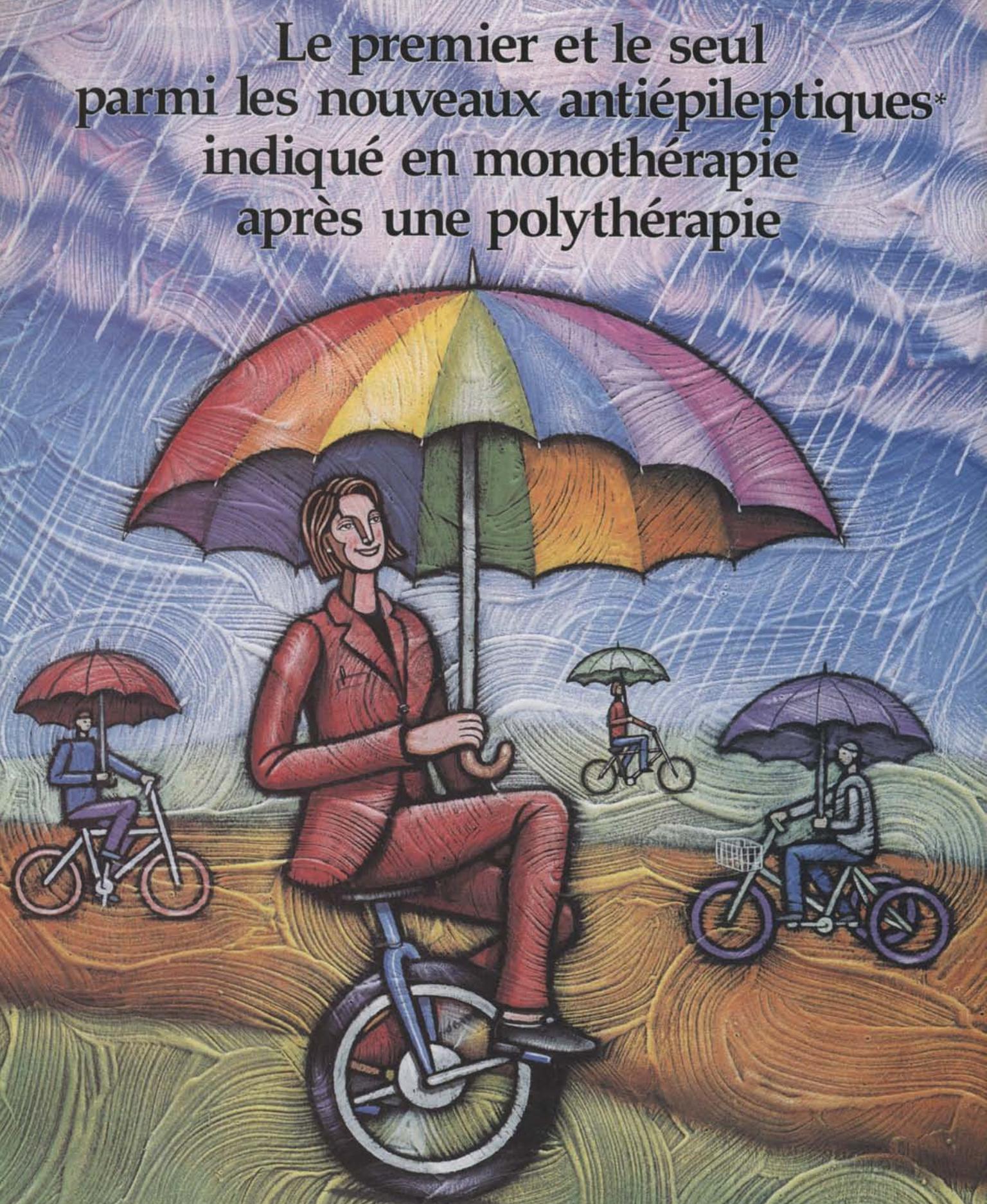
For brief prescribing information see pages A-50, A-51

Le premier et le seul parmi les nouveaux antiépileptiques* indiqué en monothérapie après une polythérapie



*C'est-à-dire la lamotrigine, la gabapentine, la vigabatrine et le topiramate, qui se distinguent des antiépileptiques traditionnels.
**Un passage réussi à la lamotrigine en monothérapie a été obtenu chez 50 patients sur 69.
***L'essai comprenait trois phases : traitement d'appoint, retrait des autres antiépileptiques et monothérapie. Ne doit pas être considéré comme une mesure absolue de l'efficacité parce que les patients n'ont pas terminé toutes les phases de l'essai lorsque leur réponse n'était pas satisfaisante.
†Les effets indésirables le plus fréquemment associés à un arrêt de la monothérapie à LAMICTAL ont été les éruptions cutanées (6,1 %), l'asthénie (1,1 %), la céphalée (1,1 %), la nausée (0,7 %) et les vomissements (0,7 %)[3]. Pour de plus amples renseignements, consulter la monographie de LAMICTAL.
††Veuillez consulter la monographie pour ce qui est de l'ajustement posologique de LAMICTAL lors du retrait des antiépileptiques administrés en concomitance.

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Pour la maîtrise d'un vaste éventail de crises, associée à un profil discret d'effets indésirables liés au SNC

D'une manière générale, une monothérapie efficace a été reconnue comme le traitement de choix pour obtenir la maîtrise des crises avec le minimum d'effets indésirables chez les patients souffrant d'épilepsie[1]. Maintenant, renforçant son succès éprouvé comme traitement d'appoint[2], LAMICTAL est indiqué comme monothérapie chez l'adulte après le retrait d'antiépileptiques administrés en concomitance[3].

MONOTHÉRAPIE HAUTEMENT EFFICACE

Dans le cadre d'un essai ouvert sur le passage d'un traitement d'appoint à la monothérapie incluant le retrait des antiépileptiques administrés en concomitance, la monothérapie à LAMICTAL a permis à 30 % (n = 50) des patients traités avec succès de rester exempts de crises[**4]. Dans un autre essai du même type, ≥ 40 % des patients ont obtenu une réduction de la fréquence de leurs crises d'au moins 50 % pendant toutes les étapes successives de l'essai[***5].

GÉNÉRALEMENT MIEUX TOLÉRÉ[†]

Selon les données regroupées de trois essais sur la monothérapie, la fréquence des retraits dus aux effets indésirables sur le SNC était de 2,5 % (n = 443) avec la monothérapie à LAMICTAL, par rapport à 7,4 % pour la phénytoïne (n = 95) ou à 7,7 % pour la carbamazépine (n = 246)[6]. La fréquence de somnolence, d'asthénie et d'ataxie a été moins élevée pour LAMICTAL que pour la carbamazépine et la phénytoïne. On n'a noté aucune différence quant à la fréquence des retraits dus aux éruptions cutanées entre LAMICTAL (6,1 %) et la phénytoïne (5,3 %) ou la carbamazépine (8,9 %)[6]. Une fréquence plus élevée d'éruptions cutanées a été associée à une augmentation posologique plus rapide de la dose initiale de LAMICTAL ou à l'utilisation concomitante d'acide valproïque[3].

MAÎTRISE SUR UN VASTE ÉVENTAIL DE CRISES

LAMICTAL a été utilisé avec succès pour un vaste éventail de crises comme traitement d'appoint dans une polythérapie[2]. Vous pouvez passer avec confiance de LAMICTAL comme traitement d'appoint en polythérapie à LAMICTAL en monothérapie[††], en particulier lorsque les effets indésirables liés au SNC sont une considération importante.

lamotrigine

PrLamictal®

DE LA POLYTHÉRAPIE À LA

MONOTHÉRAPIE

GlaxoWellcome

Glaxo Wellcome Inc.
Bureau d'affaires du Québec

MEMBRE ACIM

PAAB CCPP

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

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Pour documentation voir pages A-36, A-37

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

NEURONTIN

(gabapentin capsules)

(Gabapentin)100 mg, 300 mg, 400 mg Capsules
(Antiepileptic Agent)

INDICATIONS AND CLINICAL USE

Neurontin (gabapentin) is indicated as adjunctive therapy for the management of patients with epilepsy who are not satisfactorily controlled by conventional therapy.

CONTRAINDICATIONS

Neurontin (gabapentin) is contraindicated in patients who have demonstrated hypersensitivity to the drug or to any of the components of the formulation.

PRECAUTIONS

General

Neurontin (gabapentin) is not considered effective in the treatment of absence seizures and should therefore be used with caution in patients who have mixed seizure disorders that include absence seizures.

Tumorigenic Potential

Gabapentin produced an increased incidence of acinar cell adenomas and carcinomas in the pancreas of male rats, but not female rats or in mice, in oncogenic studies with doses of 2000 mg/kg which resulted in plasma concentrations 14 times higher than those occurring in humans at the maximum recommended dose of 2400 mg/day. The relevance of these pancreatic acinar cell tumours in male rats to humans is unknown, particularly since tumours of ductal rather than acinar cell origin are the predominant form of human pancreatic cancer.

Drug Discontinuation

As with other anticonvulsant agents, abrupt withdrawal is not recommended because of the possibility of increased seizure frequency. When in the judgement of the clinician there is a need for dose reduction, discontinuation or substitution with alternative medication, this should be done gradually over a minimum of one week.

Occupational Hazards

Patients with uncontrolled epilepsy should not drive or handle potentially dangerous machinery. During clinical trials, the most common adverse reactions observed were somnolence, ataxia, fatigue and nystagmus. Patients should be *advised to refrain from activities requiring mental alertness or physical coordination until they are sure that Neurontin* does not affect them adversely.

Drug Interactions

Antiepileptic Agents:

There is no interaction between Neurontin and phenytoin, valproic acid, carbamazepine, or phenobarbital. Consequently, Neurontin may be used in combination with other commonly used antiepileptic drugs without concern for alteration of the plasma concentrations of gabapentin or the other antiepileptic drugs.

Gabapentin steady-state pharmacokinetics are similar for healthy subjects and patients with epilepsy receiving antiepileptic agents.

Oral Contraceptives:

Coadministration of Neurontin with the oral contraceptive Norlestrin does not influence the steady-state pharmacokinetics of norethindrone or ethinyl estradiol.

Antacids:

Coadministration of Neurontin with an aluminum and magnesium-based antacid reduces gabapentin bioavailability by up to 24%. Although the clinical significance of this decrease is not known, coadministration of similar antacids and gabapentin is not recommended.

Probenecid:

Renal excretion of gabapentin is unaltered by probenecid.

Cimetidine:

A slight decrease in renal excretion of gabapentin observed when it is coadministered with cimetidine is not expected to be of clinical importance.

Use in Pregnancy

No evidence of impaired fertility or harm to the fetus due to gabapentin administration was revealed in reproduction studies in mice at doses up to 62 times, and in rats and rabbits at doses up to 31 times the human dose of 2400 mg/day. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should only be used during pregnancy if the potential benefit to the mother justifies the potential risk to the fetus.

Use in Lactation

It is not known if gabapentin is excreted in human milk, and the effect on the nursing infant is unknown. However, because many drugs are excreted in human milk, and because of the potential for serious adverse reactions in nursing *infants from gabapentin, breast-feeding is only recommended if the potential benefit outweighs the potential risks.*

Use in Children

Systematic studies to establish safety and efficacy in children have not been performed. Data in 39 patients between the ages of 12 and 18 years included in the double-blind, placebo-controlled trials showed that gabapentin was superior to placebo in reducing seizure frequency. Safety data showed that the incidence of adverse events in this group of patients were similar to those observed in older individuals.

Use in the Elderly

Systematic studies in geriatric patients have not been conducted. Adverse clinical events reported among 59 patients over the age of 65 years treated with Neurontin did not differ from those reported for younger individuals. The small number of individuals evaluated and the limited duration of exposure limits the strength of any conclusions reached about the influence of age, if any, on the kind and incidence of adverse events associated with the use of Neurontin. As Neurontin is eliminated primarily by renal excretion, dosage adjustment may be required in elderly patients because of declining renal function (See Dosage and Administration).

Use in Renal Impairment

Gabapentin clearance is markedly reduced in this patient population and dosage reduction is necessary (See Table 3 in Dosage and Administration).

Laboratory Tests

Clinical trials data do not indicate that routine monitoring of clinical laboratory parameters is necessary for the safe use of Neurontin. Neurontin may be used in combination with other commonly used antiepileptic drugs without concern for alteration of the blood concentrations of gabapentin or other antiepileptic drugs. For urinary protein determination the sulfosalicylic acid precipitation procedure is recommended, as false positive readings were reported with the Ames *N-Multistix SG® dipstick test, when gabapentin or placebo was added to other anticonvulsant drugs.*

ADVERSE REACTIONS

Adverse Events in Controlled Trials

The most commonly observed adverse events associated with the use of Neurontin in combination with other antiepileptic drugs, not seen at an equivalent frequency in placebo-treated patients, were somnolence, dizziness, ataxia, fatigue, nystagmus and tremor. Among the treatment-emergent adverse events occurring in Neurontin-treated patients, somnolence and ataxia appeared to exhibit a positive dose-response relationship. Patients treated with 1800 mg/day (n=54, from one controlled study) experienced approximately a two-fold increase, as compared to patients on lower doses of 600 to 1200 mg/day (n=489, from several controlled studies), in the incidence of nystagmus (20.4%), tremor (14.8%), rhinitis (13%), peripheral edema (7.4%), abnormal coordination, depression and myalgia (all at 5.6%). Adverse events were usually mild to moderate in intensity, with a median time to resolution of 2 weeks. Since Neurontin was administered most often in combination with other antiepileptic agents, it was not possible to determine which agent(s) was associated with adverse events. Data from long-term, open, uncontrolled studies shows that Neurontin treatment does not result in any new or unusual adverse events.

Withdrawal From Treatment Due to Adverse Events

Approximately 6.4% of the 543 patients who received Neurontin in the placebo-controlled studies withdrew due to adverse events. In comparison, approximately 4.5% of the 378 placebo-controlled participants withdrew due to adverse events during these studies. The adverse events most commonly associated with withdrawal were somnolence (1.2%), ataxia (0.8%), fatigue, nausea and/or vomiting and dizziness (all at 0.6%).

Other Adverse Events Observed in All Clinical Trials

Adverse events that occured in at least 1% of the 2074 individuals who participated in all clinical trials are described below, except those already listed in the previous section:

Body As a Whole	: aesthenia, malaise, facial edema
Cardiovascular System	: hypertension
Digestive System	: *anorexia, flatulance, gingivitis*
Hematologic/Lymphatic System	: purpura, most often described as bruises resulting from physical trauma
Musculoskeletal System	: arthalagia
Nervous System	: vertigo, hyperkinesia, parasthesia, anxiety, hostility, decreased or absent reflexes
Respiratory System	: pneumonia
Special Senses	: abnormal vision

SYMPTOMS AND TREATMENT OF OVERDOSAGE

Acute, life-threatening toxicity has not been observed with Neurontin (gabapentin) overdoses of up to 49 grams ingested at one time. In these cases, double vision, slurred speech, drowsiness, lethargy and diarrhea were observed. All patients recovered with supportive care.

Gabapentin can be removed by hemodialysis. Although hemodialysis has not been performed in the few overdose cases reported, it may be indicated by the patient's clinical state or in patients with significant renal impairment.

Reduced absorption of gabapentin at higher doses may limit drug absorption at the time of overdosing and, hence, reduce toxicity from overdoses.

An oral lethal dose of gabapentin was not identified in mice and rats given doses as high as 8000 mg/kg. Signs of acute toxicity in animals included ataxia, laboured breathing, ptosis, hypoactivity, or excitation.

DOSAGE AND ADMINISTRATION

Adults

The usual effective maintenance dose is 900 to 1200 mg/day. Treatment should be initiated with 300 to 400 mg/day. Titration to an effective dose, in increments of 300 mg or 400 mg/day, can progress rapidly and can be accomplished over three days (see Table 1). Neurontin is given orally with or without food.

Table 1. Titration Schedule

DOSE	Day 1	Day 2	Day 3
900 mg/day	300 mg OD	300 mg BID	300 mg TID
1200 mg/day	400 mg OD	400 mg BID	400 mg TID

Data from clinical trials suggest that doses higher than 1200 mg/day may have increased efficacy in some patients; however, higher doses may also increase the incidence of adverse events (See Adverse Reactions).

Daily maintenance doses should be given in three equally divided doses (See Table 2), and the maximum time *between doses in a three times daily schedule should not exceed 12 hours. It is not necessary to monitor gabapentin* plasma concentrations in order to optimize Neurontin therapy. Further, as there are no drug interactions with commonly used antiepileptic drugs, Neurontin may be used in combination with these drugs without concern for alteration of plasma concentrations of either gabapentin or other antiepileptic drugs.

Table 2. Maintenance Dosage Schedule

Total Daily Dose (mg/day)	Schedule
900	300 mg TID
1200	400 mg TID
1800	2 x 300 mg TID
2400	2 x 400 mg TID

Dosage adjustment in elderly patients due to declining renal function and in patients with renal mpairment or undergoing hemodialysis is recommended as follows:

Table 3. Maintenance Dosage of Neurontin in Adults With Reduced Renal Function

Renal Function Creatinine Clearance (mL/min)	Total Daily Dose (mg/day)	Dose Regimen (mg)
>60	1200	400 Three Times a Day
30-60	600	300 Twice a Day
15-30	300	300 Once a Day
<15	150	300 Once Daily Every Other Day
Hemodialysis[a]	--	200-300[b]

[a] Loading dose of 300 to 400 mg

[b] Maintenance dose of 200 to 300 mg Neurontin following each 4 hours of hemodialysis

Children Over 12 Years of Age

The dosage used in a limited number of patients in this age group was 900-1200 mg/day. Doses above 1200 mg/day have not been investigated.

AVAILABILITY OF DOSAGE FORMS

Neurontin (gabapentin) capsules are supplied as follows:

100-mg capsules;

Hard gelatin capsules with white opaque body and cap printed with "PD" on one side and "Neurontin/100 mg" on the other.
-bottles of 100 capsules

300-mg capsules;

Hard gelatin capsules with yellow opaque body and cap printed with "PD" on one side and "Neurontin/300 mg" on the other.
-bottles of 100 capsules

400-mg capsules;

Hard gelatin capsules with orange opaque body and cap printed with "PD" on one side and "Neurontin/400 mg" on the other.
-bottles of 100 capsules

Full Prescribing Information Available On Request

Parke-Davis Division
Warner-Lambert Canada Inc.
Scarborough, Ontario M1L 2N3

References:
1. The Neurontin STEPS Study Team. Study of Neurontin: Titration to Effect, Profile of Safety. In: Program and Abstracts of the I.L.A.E., Dublin, Ireland July 1997. 2. Data on file: Bruni, J.: "Outcome Evaluation of Gabapentin as Add-on Therapy for Partial Seizures". Canadian Journal of Neurological Science. 1998: vol 25: 134-140. 3. Neurontin Product Monograph

PARKE-DAVIS

For brief prescribing information see pages A-17, A-19, A-27

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34th Meeting of Canadian Congress of Neurological Sciences
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- ✔ Neurobiology Review Course
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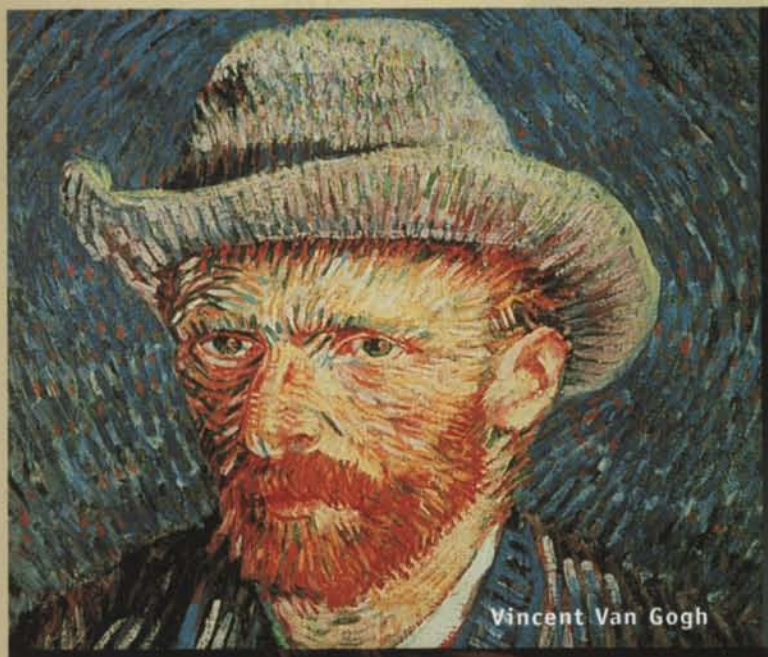
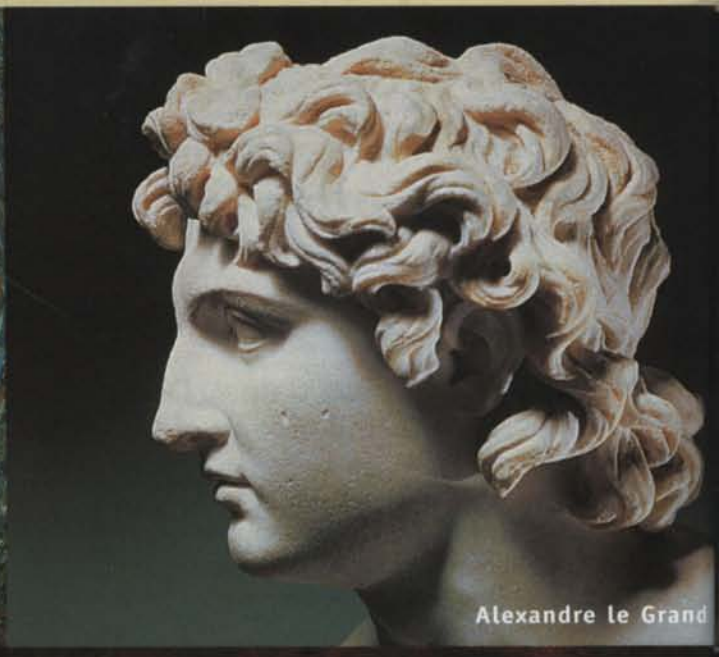
- ✔ Spinal Instrumentation
- ✔ Controversies in Neurocritical Care
- ✔ Roles of Intraoperative Brain Imaging
- ✔ The Triptans in Migraine Therapy
- ✔ Management Issues in Epilepsy
- ✔ Pediatric Epilepsy
- ✔ Behavioural Neurology
- ✔ Symptom Management and Current Therapies in Multiple Sclerosis
- ✔ Dementia, Treatment and ethics
- ✔ Stroke

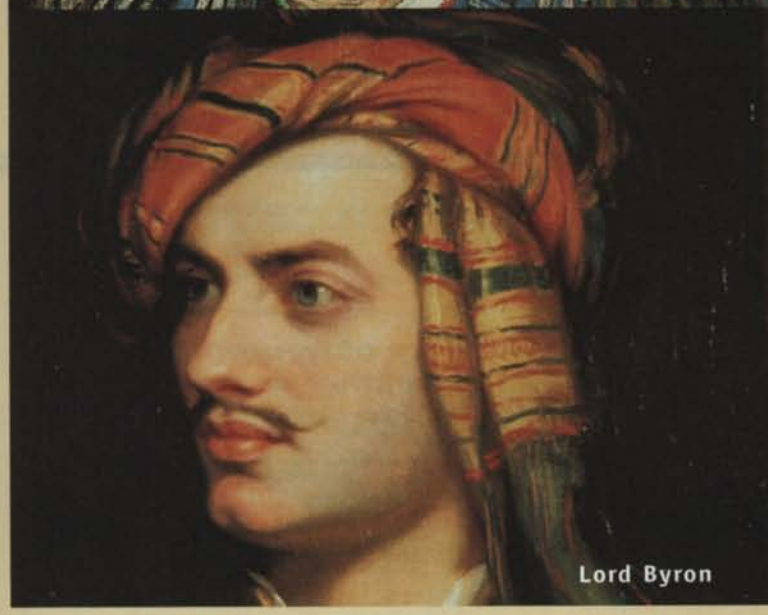
For brief prescribing information see page A-26

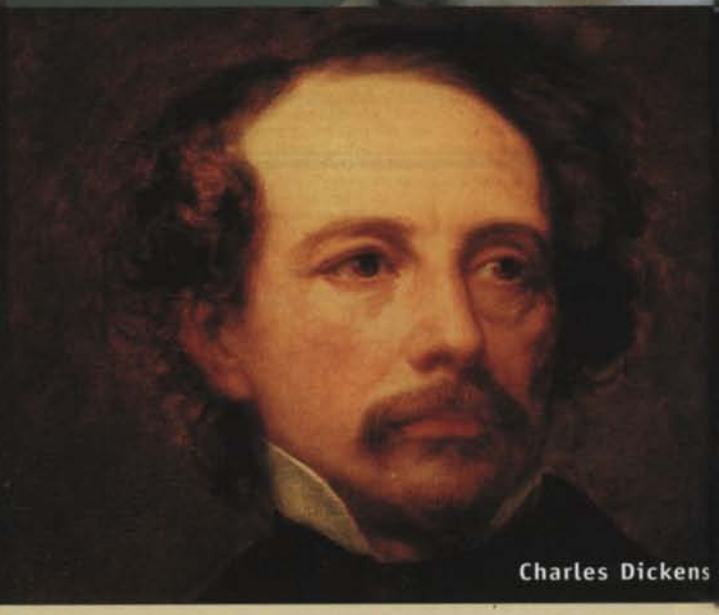
https://doi.org/10.1017/S0317167100049258 Published online by Cambridge University Press

DU NOUVEAU EN ÉPILEPSIE. MAINTENANT REMBOURSÉ PAR LES FORMULAIRES

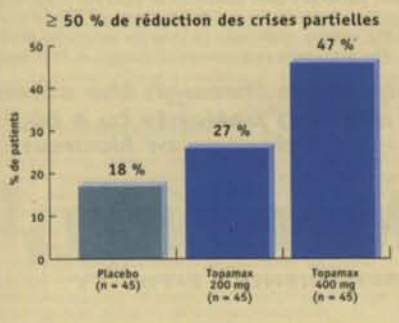








NAGUÈRE ENCORE, LA RÉUSSITE EXIGEAIT D'UN ÉPILEPTIQUE HEUREUSEMENT POUR VOS PATIENTS, IL EXISTE



Extrait de référence N° 1. Étude en double aveugle avec placebo contre TOPAMAX b.i.d. comme traitement d'appoint, portant sur 181 patients atteints d'épilepsie partielle réfractaire et recevant une ou deux autres médications antiépileptiques. *p = 0,013.

Contrôle amélioré d'une plus grande variété de types de crises.

- TOPAMAX est indiqué comme traitement d'appoint pour toutes les épilepsies réfractaires aux traitements conventionnels. À l'heure actuelle, les données sur l'utilisation de TOPAMAX comme traitement unique demeurent limitées.
- Taux élevé de répondants : 27 % (200 mg/jour, n = 45) et 47 % (400 mg/jour, n = 45) des patients ont manifesté une réduction des crises d'épilepsie partielle ≥ 50 % (étude d'une durée de 16 semaines)[1]
- Contrôle efficace pour les patients souffrant de cirses toniques-cloniques secondaires généralisées : 36 % des patients ont manifesté une réduction de 100 % (200-600 mg, n = 42, étude portant sur 16 semaines)[1]
- Triple mécanisme d'action unique : blocage des canaux sodiques, activation de l'acide gamma-aminobutyrique, antagonisme du glutamate)[2]

JANSSEN-ORTHO Inc. 19 Green Belt Drive, North York, Ontario M3C 1L9 *Marque déposée ©Janssen-Ortho Inc. 1997

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DE LA C.-B., L'ALBERTA, LA SASKATCHEWAN, LA NOUVELLE-ÉCOSSE ET DU QUÉBEC.

DES EFFORTS EXCEPTIONNELS OU UN TALENT EXTRAORDINAIRE. MAINTENANT DES SOLUTIONS PLUS ACCESSIBLES.

- Généralement bien toléré : les interruptions entraînées par des réactions adverses étaient de 10,6 % pour les doses journalières de 200 à 400 mg, comparé à 5,8 % pour le groupe placebo (cela semblerait augmenter pour les doses journalières supérieures à 400 mg)[2]
- Aucune preuve d'éruption cutanée sérieuse ni d'anémie aplasique[2]
- Il n'est généralement pas nécessaire de changer le dosage des médications principales; les patients prenant de la phénytoïne et manifestant des signes ou symptômes de toxicité devraient faire contrôler leurs niveaux de phénytoïne[†2]
- **Dosage commode BID**

Profil favorable des effets secondaires (les plus courants affectent le SNC)

	TOPAMAX 200-400 mg (n = 113)	PLACEBO (n = 216)
Somnolence	30,1	9,7
Étourdissements	28,3	15,3
Ataxie	21,2	6,9
Ralentissement psychomoteur	16,8	2,3
Troubles de la parole	16,8	2,3
Nervosité	15,9	7,4
Nystagmus	15,0	9,3
Paresthésie	15,0	4,6

†Comme pour les autres traitements antiépileptiques, veuillez vous reporter aux renseignements thérapeutiques pour plus de détails concernant les interactions médicamenteuses. On a rapporté l'occurence de 1,5 % (n = 1715) de calculs rénaux[2]. Dans une étude (n = 1200), 83 % des patients (15 sur 18) ont choisi de continuer le traitement[4]. Assurer un taux d'hydratation adéquat et éviter l'utilisation parallèle d'autres inhibiteurs de l'anhydrase carbonique[2].

TOPAMAX®
topiramate

Aide vos patients à mieux tirer parti de leur vie

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Pour documentation voir pages A-46, A-47, A-48

IN THE TREATMENT OF ALZHEIMER'S DISEASE

Once-a-day Aricept* improves patient function:

For a more *active* day, a *brighter* tomorrow.

The loss of function that comes with Alzheimer's disease has a devastating effect on everyone involved: patient, caregiver and family.[1] Once-a-day Aricept* **enhances cognition and improves patient function.**[2†] Once-a-day Aricept* (10 mg o.d.) has been shown to significantly improve complex Activities of Daily Living (ADL).[3] A recent Canadian economic evaluation predicts that improvement in patient outcome will result in an overall healthcare cost saving.[4] And once-a-day Aricept* has proven efficacy, dosing simplicity[5§] and tolerability[‡] in over 54 million patient days of therapy worldwide.[5]

Once-a-day Aricept*. To help your Alzheimer's patients enjoy more *active* days, and look forward to a *brighter* tomorrow.

Once-a-day Aricept*

donepezil HCl 5 & 10 mg tablets

Hope for a brighter tomorrow

Aricept* is indicated for the symptomatic treatment of patients with mild to moderate Alzheimer's disease. Aricept* has not been studied in controlled clinical trials for longer than 6 months.
† Cognition measured by ADAS-cog and MMSE; Function measured by CIBIC plus.
‡ The most common side effects observed with Aricept* include diarrhea, muscle cramps, nausea and insomnia; these effects are usually mild and transient, resolving with continued use.
§ For patients not responding after 4-6 weeks of therapy at 5 mg/d, a 10 mg/d dose may be considered.
Please see enclosed Prescribing Information before prescribing.

Pfizer
We're part of the cure

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

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