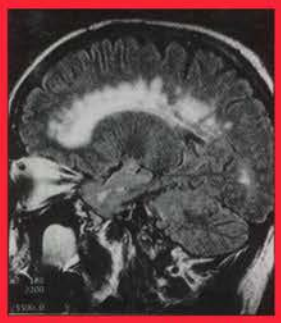
Volume 26 Number 4 November 1999

THE CANADIAN JOURNAL OF Neurological Sciences

LE JOURNAL CANADIEN DES Sciences Neurologiques

AN INTERNATIONAL JOURNAL / UN JOURNAL INTERNATIONAL

Dr. Charles G. Drake
1920 – 1998



Neuroimaging Highlight

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

NOW ON PROVINCIAL FORMULARIES
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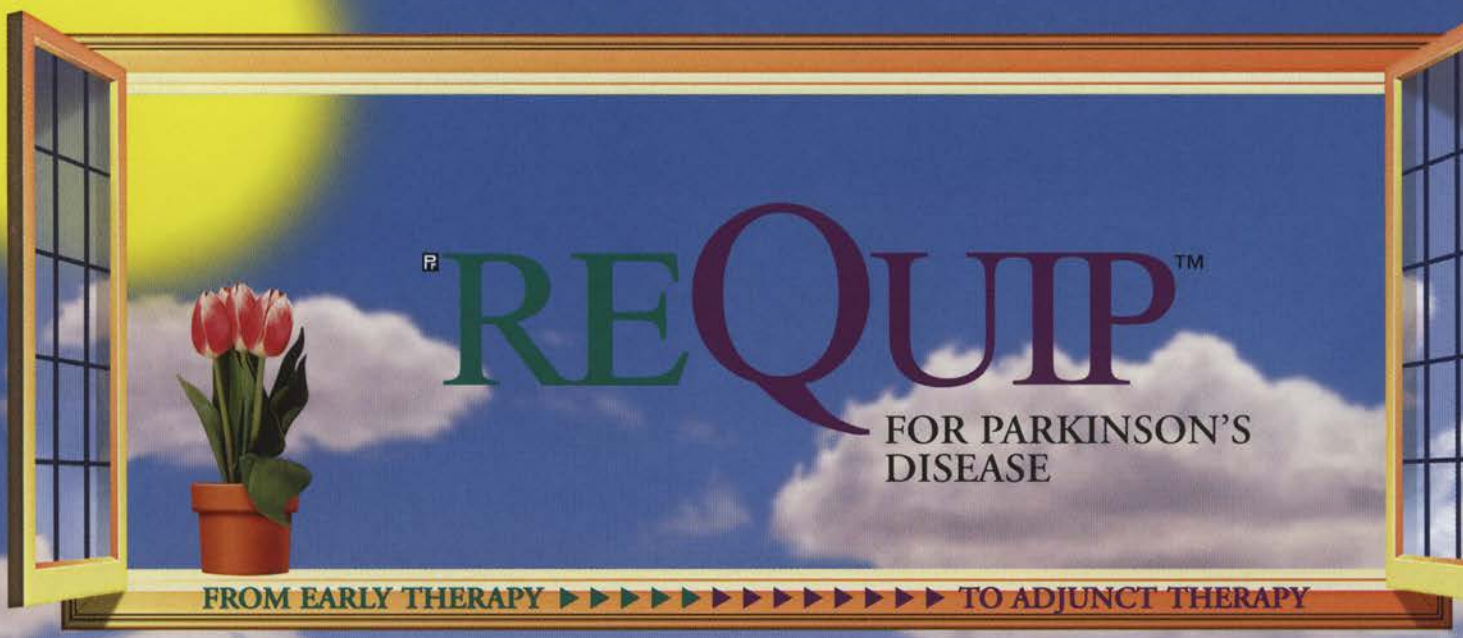
A DOPAMINE AGONIST YOU CAN START WITH AND STAY WITH.



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

PAAB MEMBER PMAC

ropinirole
℞REQUIP™
Early Therapy → Adjunct Therapy
RIGHT FROM THE START.

https://doi.org/10.1017/S0317167100050058 Published online by Cambridge University Press

For brief prescribing information see pages A-41, A-42

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La Société Canadienne de Neurologie

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La Société Canadienne de Neurochirurgie

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L'Association Canadienne de Neurologie Pédiatrique

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Have a Nice Triptan

Migraine relief with tolerability similar to placebo

As shown in controlled clinical trials, AMERGE is a highly tolerable triptan with an incidence of adverse events similar to placebo.[1-3†] AMERGE provided significant migraine relief maintained over 24 hours.[1,2‡] A recent study demonstrated that 93% of attacks per patient did not require a second dose for recurrences.[4§]

†In controlled clinical trials, the incidence of adverse events was similar to placebo (31% for AMERGE 2.5 mg vs. 32% for placebo[2]).
‡Headache relief=reduction of moderate or severe pain to mild or no pain. AMERGE 2.5 mg n=586: p<0.001 vs. placebo 60 min post-dose to 4 hrs; p<0.05 vs. placebo at 4, 8, 12, 24 hrs.
§The median 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.

AMERGE (naratriptan hydrochloride) is a selective 5-HT$_1$ receptor agonist indicated for the acute treatment of migraine attacks with or without aura. AMERGE 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 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. In addition, patients with other significant underlying cardiovascular diseases should not receive AMERGE. AMERGE is also contraindicated in patients with uncontrolled or severe hypertension.

℞ Amerge®
naratriptan hydrochloride

Making tolerability a part of migraine relief.

GlaxoWellcome PAAB CCPP Member R&D

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

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 increased incidence of serious dermatological reactions.

Product Monograph available to health care professionals upon request.

https://doi.org/10.1017/S0317167100050058 Published online by Cambridge University Press

Gastaut Syndrome

Lamotrigine

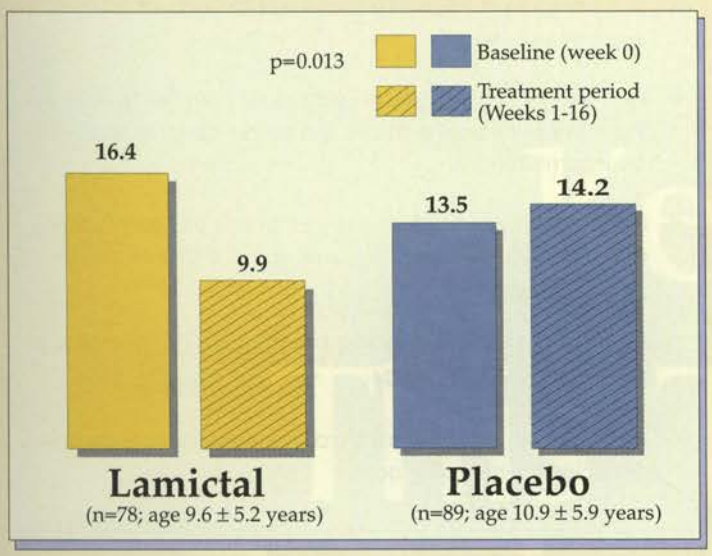
Lamictal®

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.

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Lamotrigine

Lamictal®

For a Brighter Future

PAAB CCPP

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For brief prescribing information see pages A-53, A-54, A-55, A-56

CALL FOR ABSTRACTS / DEMANDE DE RÉSUMÉS

35th Meeting of the
Canadian Congress of Neurological Sciences
Ottawa, June 13-17, 2000

34e Assemblée annuelle du
Congrès canadien des sciences neurologiques
13 - 17 juin 2000

CALL FOR ABSTRACTS
Scientific Papers and Posters

1. Abstracts for the scientific program must be submitted on the official 2000 Congress Abstract Form.
2. Work presented must conform with MRC guidelines for experimental procedures.
3. Scientific material presented at this meeting should not have been published or presented at other national or international meetings.
4. It is suggested that presentations emphasize the significance of the results and general principles involved rather than ordinary methods and procedures.
5. **ABSTRACTS MUST BE RECEIVED BY JANUARY 14, 2000.**
6. Abstracts accepted for presentation will be published in the Canadian Journal of Neurological Sciences.

DEMANDE DE RÉSUMÉS
Présentations orales et par affiches des résumés

1. Les résumés pour le programme scientifique doivent être soumis sur le formulaire officiel du congrès désign.
2. Le travail présent, dans les résumés doit être conforme aux principes du C.R.M. relatifs aux procédures expérimentales.
3. Le matériel scientifique présent, à ce congrès ne doit pas avoir été publié ou présent, à d'autres congrès nationaux ou internationaux.
4. Il est recommand, de mettre l'emphase sur les résultats et les principes généraux plutôt que d'élaborer sur la méthodologie.
5. **LES RÉSUMÉS DOIVENT ETRE RECUS AVANT LE 14 JANVIER 2000.**
6. Les résumés acceptés paraîtront dans le Journal canadien des sciences neurologiques.

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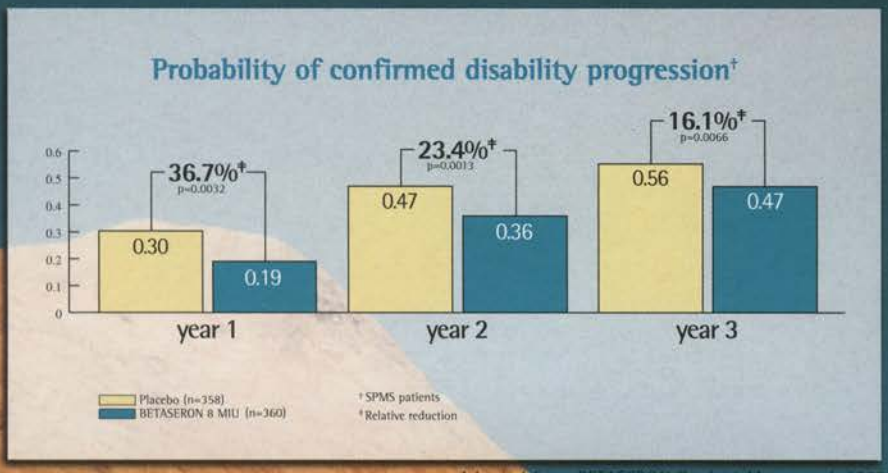
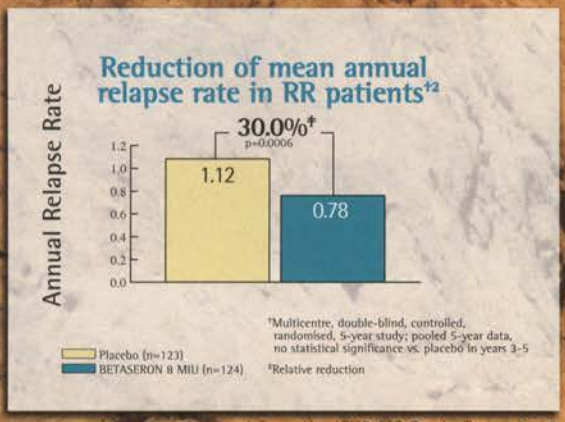
BETASERON delays disability progression*[1]

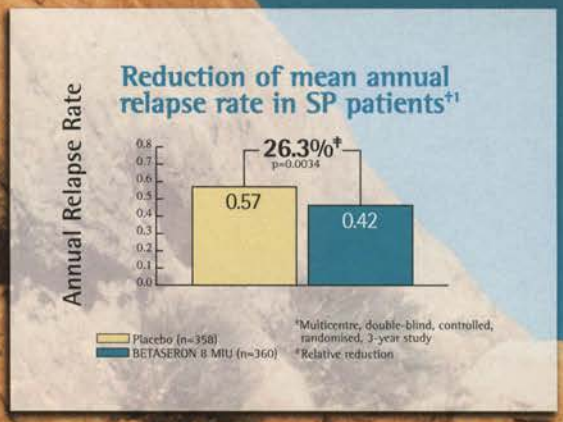


Adapted from BETASERON Product Monograph 1999

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



Adapted from the IFNB MS Study Group 1995



Adapted from BETASERON Product Monograph 1999

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.

MULTIPLE SCLEROSIS PATHWAYS FOR CANADA
1-800-977-2770 CODE 4000

Member R&D PAAB CCPP

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For brief prescribing information see pages A-43, A-44, A-45

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ONCE IT TOOK EXCEPTIONAL EFFORT OR EXTRAORDINARY TALENT FOR PEOPLE WITH EPILEPSY TO SUCCEED. LUCKILY, BOTH YOUR ADULT AND PEDIATRIC PATIENTS CAN NOW ENJOY LESS TAXING ALTERNATIVES.

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NOW INDICATED FOR CHILDREN

[Pr]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.[1]

Efficacy in Partial Onset Seizures:

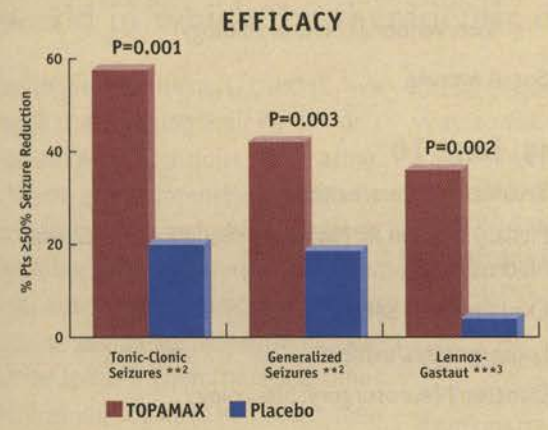
Dosage Individualized to Patient Response:[4,5]

	N	≥50% Seizure Reduction	Seizure Free
Adults[4,a]	450	59%	19%
Children[5,b]	41	73%	22%

Adapted from references 4 and 5
a Open label, 20 week trial in adults with partial onset seizures. TOPAMAX administered b.i.d. as adjunctive therapy, optimal dosage appeared to be 300-350 mg/day.
b Open label trial in children with partial-onset seizures following participation in a double-blind, placebo controlled trial. Reductions in seizure frequency were determined for children treated for at least 3 months. TOPAMAX administered b.i.d. as adjunctive therapy. Children received open label topiramate for a mean duration of 8 months at an average dose of 10 mg/kg/day (4-20 mg/kg/day).
For recommended dose refer to TOPAMAX* Prescribing Information.

Improved control over a wide range of seizure types:

- With additional data demonstrating efficacy as adjunctive therapy from randomized, double-blind, placebo-controlled trials in adults and a small number of children for:
- **Primary Generalized Tonic-Clonic Seizures**[1]
- **Seizures associated with Lennox-Gastaut syndrome**[1]



Adapted from references 2 and 3
**20 week double-blind treatment phase (8 week baseline and a 12 week treatment period) with either TOPAMAX (n=39, including 8 children ≤ 16 yrs) b.i.d. as adjunctive therapy or placebo (n=41). TOPAMAX was titrated to target doses of approximately 6 mg/kg/day.
***Drop attacks and tonic-clonic seizures: 11 week double blind treatment phase with either TOPAMAX (n=48) b.i.d. as adjunctive therapy or placebo (n=50); patient mean age 11.2 yrs. TOPAMAX was titrated to a target dose of approximately 6 mg/kg/day.

TABLETS NOW ON FORMULARY‡

‡ Limited use benefit–Ontario, Nova Scotia, New Brunswick, PEI.
Full Benefit–Quebec, Saskatchewan, British Columbia, Alberta, Manitoba.

An appropriate first choice adjunctive therapy for many of your patients:

Favourable Side-effect Profile:

- Like most antiepileptic drugs, the most common side effects are CNS related††[1,6]:

- Usually mild to moderate occurring early in therapy and transient[1,6]
- If encountered:

Consider reducing the TOPAMAX dosage, rate of titration, and/or the concomitant AED dosage.[8]

- In children, there were no discontinuations due to adverse events at 5 to 9 mg/kg/day in the controlled clinical trials.[1]

Safety Considerations:

- No evidence to date of a proven association of TOPAMAX usage and the following: life threatening rash, permanent visual field constriction or polycystic ovary disease.[1,c]
- Weight loss

Adults: Modest weight loss may be sustained ≤ 12 months with the greatest weight loss occurring between 3 and 6 months and peaking at 9 months.[7]
Pediatrics: Of those pediatric subjects treated in clinical trials for at least a year who experienced weight loss, 96% showed a resumption of weight gain within the period tested.[1†]

Convenient BID dosing[1]

Now available in a convenient 15 mg and 25 mg Sprinkle Capsule formulation[1]:

Swallow whole or sprinkle on food
Bioequivalent to TOPAMAX Tablets

† The long term effects of weight loss in pediatric patients is not known.
†† 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%).[1] In an audit of 1446 adults and 303 children there appeared to be a similar pattern of adverse events.[1]

Please refer to the TOPAMAX Prescribing Information for complete prescribing details.
c Data on file JANSSEN-ORTHO Inc May 1999
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 PAAB JATX991005A

TOPAMAX® topiramate

Helping patients with epilepsy make more of their lives

For brief prescribing information see pages A-68, A-69, A-70

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35th Meeting of the
Canadian Congress of Neurological Sciences

Ottawa, June 13-17, 2000

PRELIMINARY PROGRAM

As we move to the millenium, the CCNS will celebrate over 50 years of progress and promise.
Participate in the new developments leading to neurosciences for the future.
Mark your calendar for Ottawa in June!

Tuesday, June 13

- Neurobiology Review Course 2000
- ALS Symposium
- Clinical Epilepsy Video Session (evening)
- Vascular Dementia (evening)

Wednesday, June 14

- Meet the Expert Breakfast - Pediatric Neurology
- Courses
  1. Evidence-based Neurology (am)
  2. Management of Disorders of the Craniocervical Junction (full day)
  3. Current Educational Issues in the Clinical Neurosciences (am)
  4. Medical Legal Issues in Child Neurology (am)
  5. Molecular Mechanisms of Epileptic Syndromes (am)
  6. Medical Ethics in Neurology (pm)
  7. Molecular Mechanisms of Neuromuscular Disease (pm)
  8. Case Studies in Neurocritical Care (Neurocritical Care Group) (pm)
  9. Epilepsy (pm)
- Welcome Reception

For additional information contact:
The Canadian Congress of Neurological Sciences
PO Box 4220, Station C
Calgary, Alberta, Canada T2T 5N1
Tel: (403) 229-9544 Fax: (403) 229-1661
Email: brains@ccns.org

Thursday, June 15

- Meet the Expert Breakfast - Neurosurgery and Neurology
- Breakfast/posters/exhibits
- Plenary Session I:
  The Millenium and the Future of Clinical Neuroscience
- Oral Platform Sessions
- Lunch/posters/exhibits
- Plenary Session II:
  Cerebrovascular Disease
  Interventional Neuroradiology
- Social evening

Friday, June 16

- Breakfast/posters/exhibits
- Plenary Session III: Molecular Genetics and Clinical Neuroscience
- Oral Platform Sessions
- Lunch/posters/exhibits
- Debates: Neurosurgery; Neurology

Saturday, June 17

- Child Neurology Day: Neurobehavioural Disorders
- Courses
  1. Emergent Therapies in Acute Stroke (full day)
  2. Multiple Sclerosis (am)
  3. Migraine 2000: A New Era in Migraine Therapy (pm)
- Child Neurology Dinner

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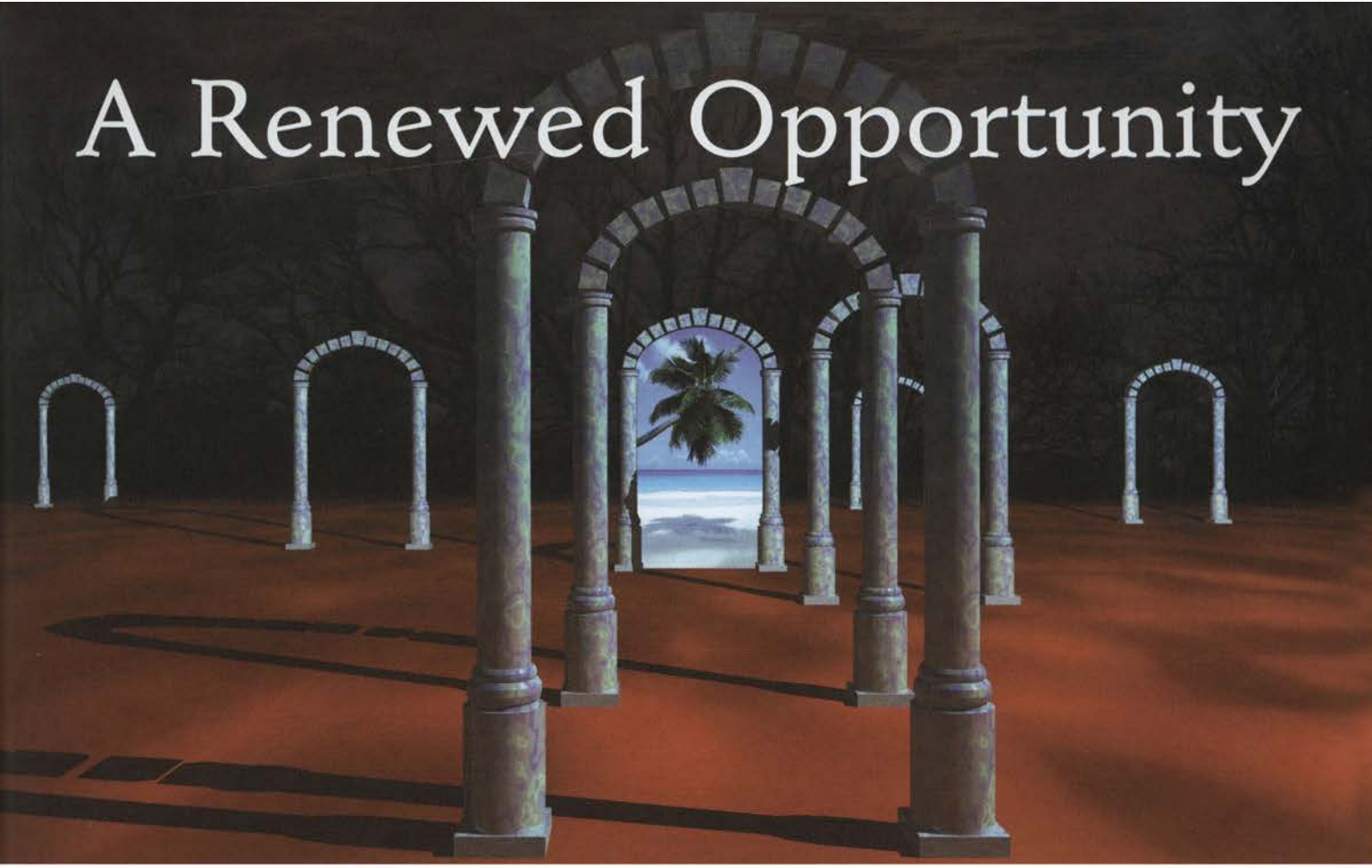

PARKINSON'S DISEASE

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.

Pr 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

For brief prescribing information see pages A-59

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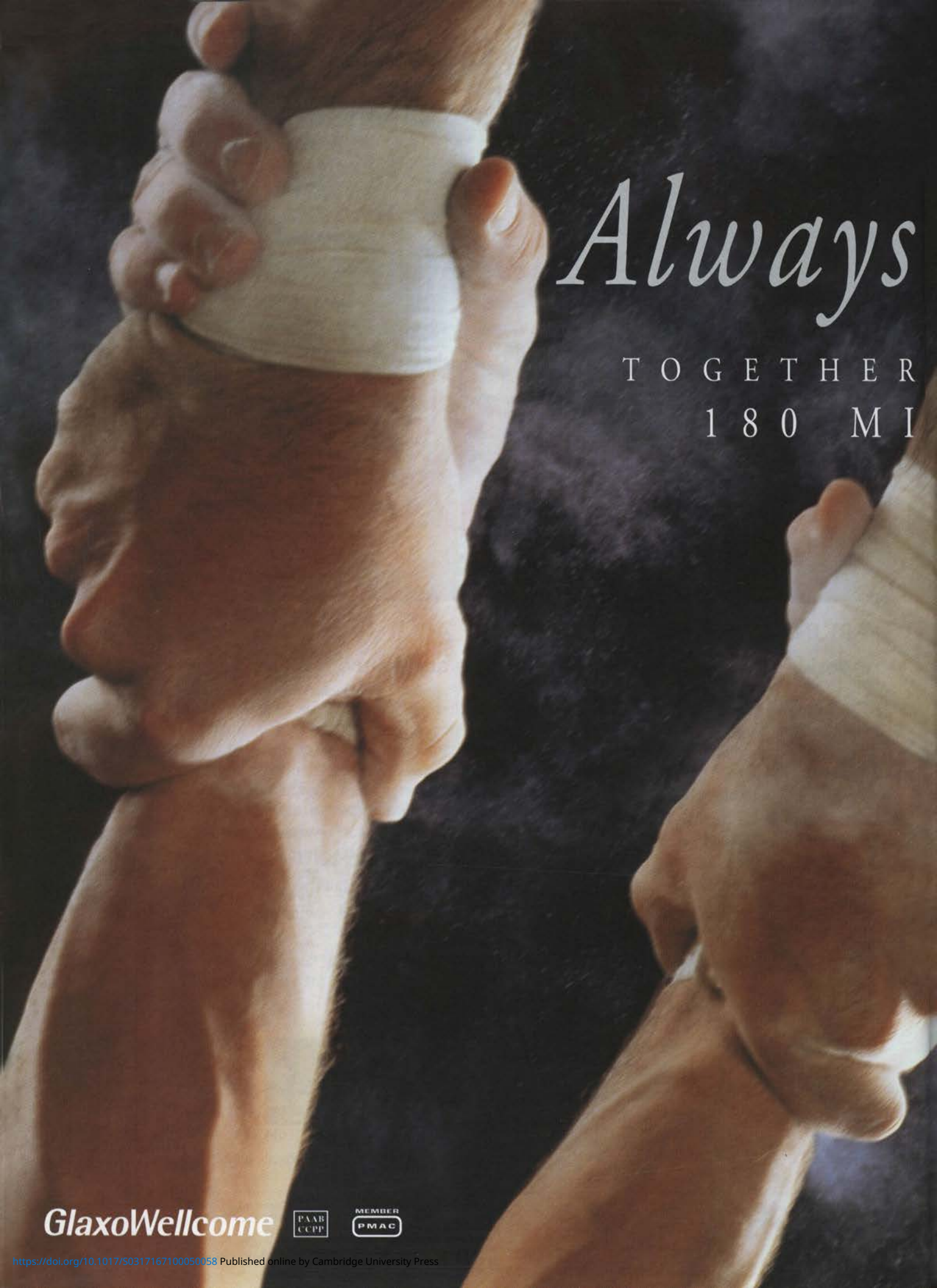

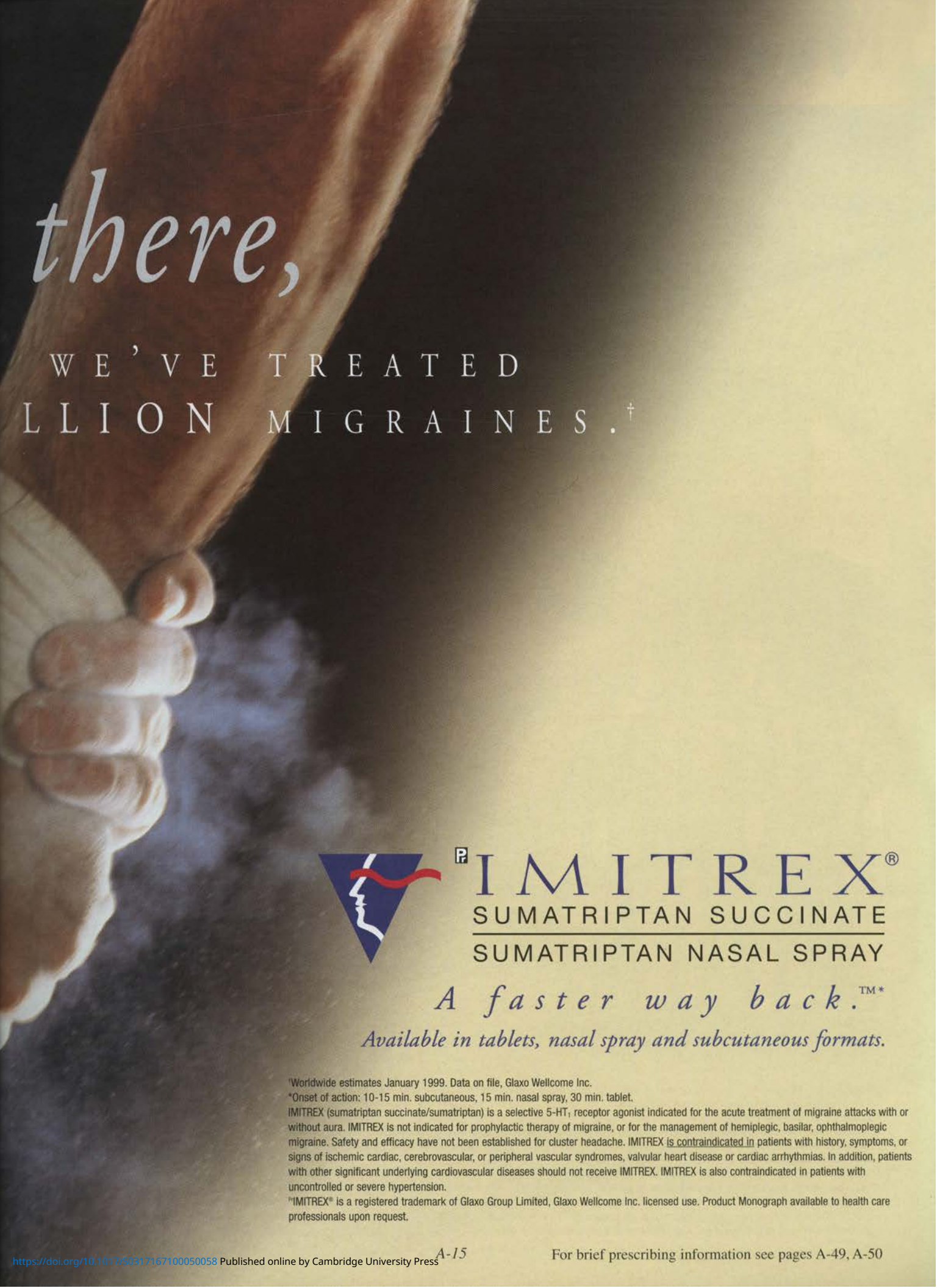

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IMITREX (sumatriptan succinate/sumatriptan) is a selective 5-HT_1 receptor agonist indicated for the acute treatment of migraine attacks with or without aura. IMITREX is not indicated for prophylactic therapy of migraine, or for the management of hemiplegic, 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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For brief prescribing information see pages A-49, A-50

Nouveau dans le syndro

[‡]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[2].**
[¶]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.

me de Lennox-Gastaut

lamotrigine

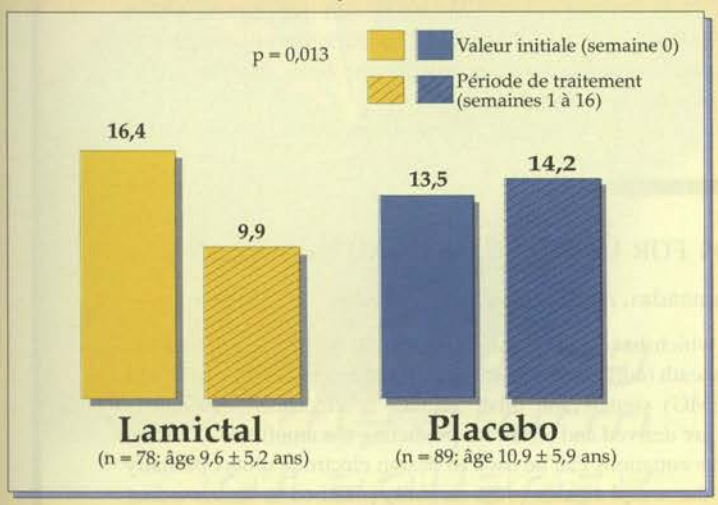
Lamictal®

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].

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lamotrigine

Lamictal®

L'avenir en tête

ADIM
CCPP PAAB

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Pour documentation voir pages A-53, A-54, A-55, A-56

25 Years Ago in the Canadian Journal of Neurological Sciences

Mechanisms in Motor Control
A Symposium on Clinical and Basic Research in Neuroscience
June 20-21, 1975 London, Ontario

Clinical Problems of Motor System Disease

J.C. Richardson

Summary: This prologue to a symposium of research studies on motor mechanisms is a general commentary by a clinical neurologist. The vast extent and intricacy of modern basic neurological scientific knowledge presents a rather bewildering challenge to reasonable clinical application. In some degree this is being handled by complex and diverse neurological subspecialization. It is recalled that many past advances in the knowledge of neurological disease were achieved by a series of alternating and supporting bedside and laboratory observations and studies. The varied disorders of movement and muscle tone which signal disordered motor mechanisms will continue to demand explanation and will keep the human model in a leading research position. Clinical and laboratory research leading to part discoveries of mechanisms of disease is sometimes productive of dramatic new means of therapy. The story of Wilson's disease is briefly reviewed in that context. Some recent studies on hypoxic myoclonus are described with the evidence of a serotonin defect and useful related therapy.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):223

Central Mechanisms of Tremor in Some Feline and Primate Models

Y. Lamarre, A.J. Joffroy, M. Dumont, C. de Montigny, F. Grou, J.P. Lund

Summary: For several years our interest has been in a postural Parkinson-like tremor at 4-6/sec. which can be produced in the monkey by lesions of the central nervous system. We have also studied the effects of harmaline, a drug which evokes or intensifies the Parkinson-like tremor in lesioned animals and which also induces a fine, generalized tremor at 7-12/sec. in normal animals. The results obtained so far indicate that these two types of tremor are generated by two independent central mechanisms which do not require the integrity of peripheral feedback loops. The experimental Parkinson-like tremor is generated by a thalamo-cortical mechanism while the olivo-cerebellar system is responsible for the faster 'physiological' tremor. Similar tremor mechanisms may be involved in some movement disorders in man.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):227

Principles Underlying New Methods for Chronic Neural Recording

R.B. Stein, D. Charles, L. Davis, J. Jhamandas, A. Mannard, T.R. Nichols

Summary: Chronic recording is possible from nerve fibers which have grown through holes in an insulating medium (regeneration electrodes) or which are enclosed by an insulating sheath (cuff electrodes). Use of three electrodes in a balanced configuration permits good rejection of electromyographic (EMG) signals and other sources of electrical interference (fluorescent lights, 60Hz signals form the mains, etc.). Equations are derived and tested for predicting the amplitude and form of the signals expected for a given cuff length and diameter. These equations can be used to design electrode units optimally for a given application. Finally, the use of transformers permits the neural signals to be carefully matched to the recording apparatus and further optimizes the neural signal-to-noise and signal-to-EMG ratios. Use of these methods in several physiological and clinical applications, as well as potential abuses, are discussed.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):235

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PRIX DES SOCIÉTÉS

Prix de la Société, canadienne de neurologie
PRIX COMMÉMORATIF FRANCIS McNAUGHTON et le prix COMMÉMORATIF ANDRÉ BARBEAU 2000

La Société, canadienne de neurologie accepte des soumissions de communications pour ses deux prix: le prix commémoratif Francis McNaughton pour la recherche clinique et le prix commémoratif André Barbeau pour la recherche fondamentale.

Les prix commémoratifs Francis McNaughton et André Barbeau ont pour but d'encourager les résidents en neurologie à entreprendre des projets de recherche. Ils sont décernés aux meilleures communications pour un travail exécuté au cours d'un programme de formation en résidence ou post-résidence en neurologie. Il n'est pas nécessaire que les candidats soient les seuls auteurs, mais ils doivent être les principaux responsables de la communication présentée.

Les communications gagnantes dans chaque catégorie seront présentées à l'assemblée annuelle du Congrès canadien des sciences neurologiques. La date limite pour la soumission des communications est fixée au 31 décembre 1999.

Société canadienne de neurophysiologie clinique
LE PRIX HERBERT JASPER

Le prix Herbert Jasper est décerné chaque année au résident ou au chercheur qui a soumis la meilleure communication dans le domaine de la neurologie fondamentale ou clinique. Ceux qui doivent obtenir leur doctorat ou leur bourse de recherche d'ici trois ans sont aussi admissibles.

Le prix comprend une somme de 250 $, l'hébergement à l'hôtel, les frais d'inscription, et un billet d'avion en classe économique pour assister au Congrés canadien des sciences neurologiques où sera présentée la communication gagnante.

La date limite des soumissions est fixée au 31 décembre 1999.

Le Collège royal des médecins et chirurgiens du Canada en collaboration avec la Société canadienne de neurochirurgie présente
LES PRIX COMMÉMORATIFS K. G. McKENZIE 2000

Le prix McKenzie pour la recherche fondamentale en sciences neurologiques et le prix McKenzie pour la recherche clinique en sciences neurologiques.

Dans chacune des catégories de la recherche fondamentale et de la recherche clinique en sciences neurologiques, on attribuera une mention et un prix de 1 000 $ au (-a la) résident(e) en neurochirurgie qui est le principal auteur du meilleur manuscrit soumis au comit, du prix McKenzie de la Société canadienne de neurochirurgie. Les prix comprendront également les dépenses des lauréats, y compris le transport aérien, l'hébergement à l'hôtel et les frais d'inscription. Un deuxième prix de 500 $ pourrait également être remis dans chaque catégorie.

La date limite des soumissions pour les prix de 2000 est fixée au 31 décembre 1999 et cette date est ferme.

L'Association canadienne de neurologie pédiatrique
LE PRIX DU PRÉSIDENT

Le prix du président est décern, chaque année au résident ou au chercheur qui a soumis la meilleure communication dans le domaine de la neuroscience chez l'enfant. Le prix comprend une somme de 500 $ et un parchemin commémoratif, et l'inscription à l'assemblée annuelle. La communication gagnante sera présentée à l'assemblée du Congrès canadien des sciences neurologiques.

La date limite de la réception des soumissions est fixée au 31 décembre 1999.

Envoyer votre application au Congrès canadien des sciences neurologiques
810, 906-12th Avenue SW, Calgary AB Canada T2R 1K7
Téléphone : (403) 229-9544 Télécopieur : (403) 229-1661
Courrier électronique : brains@ccns.org

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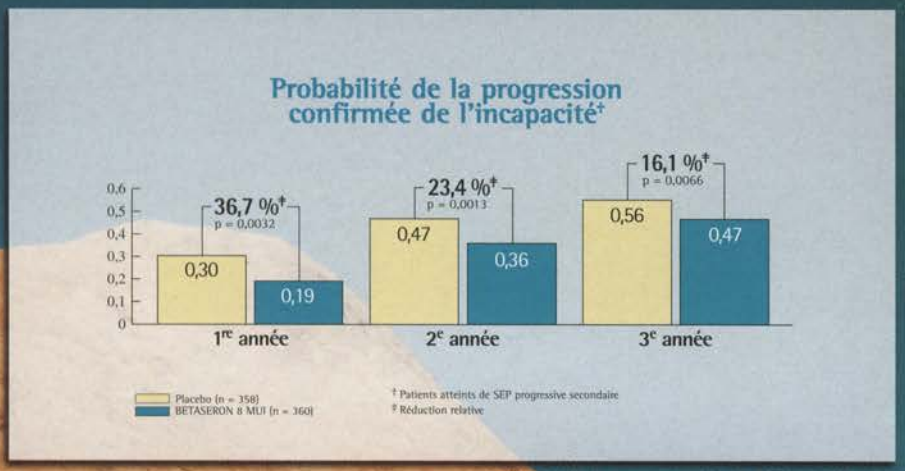
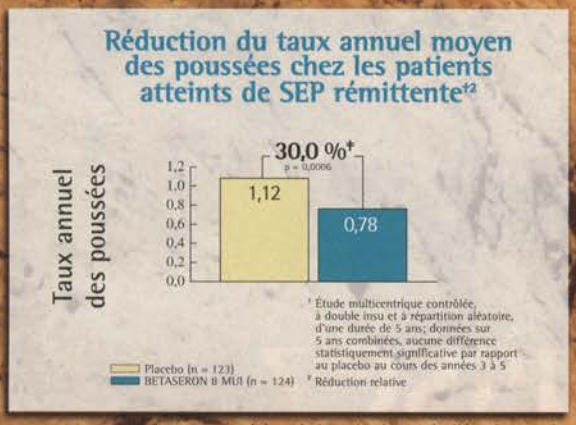
BETASERON retarde la progression de l'incapacité*[1]

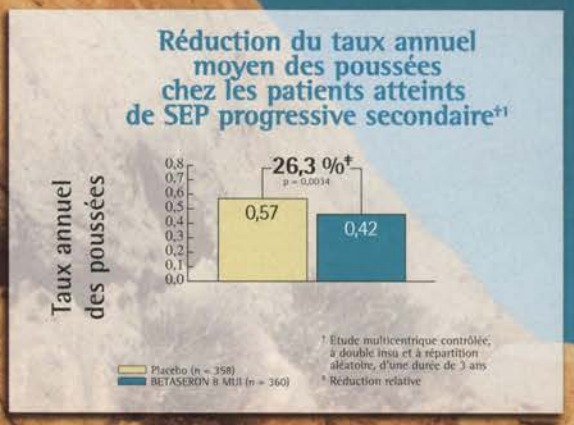


Adapté de la monographie de BETASERON, 1999

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



Adapté des résultats de l'étude menée par le IFNB MS Study Group, 1995



Adapté de la monographie de BETASERON, 1999

Effets indésirables pouvant être pris en charge[1]

Chez les patients atteints de la 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].

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* 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.

Membre R&D CCPP PAAB

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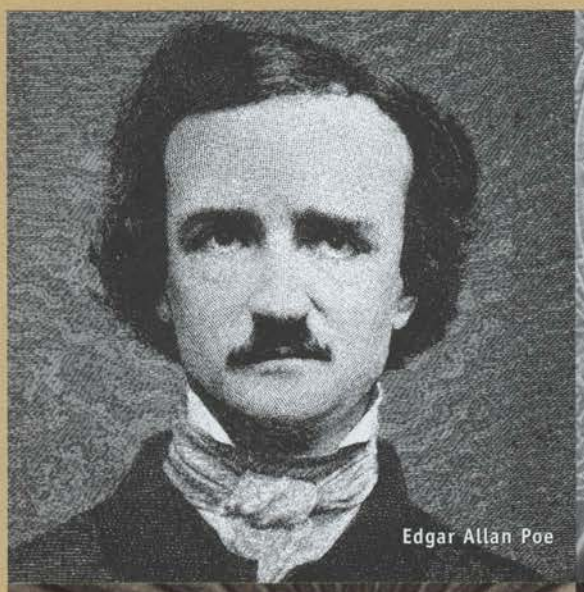
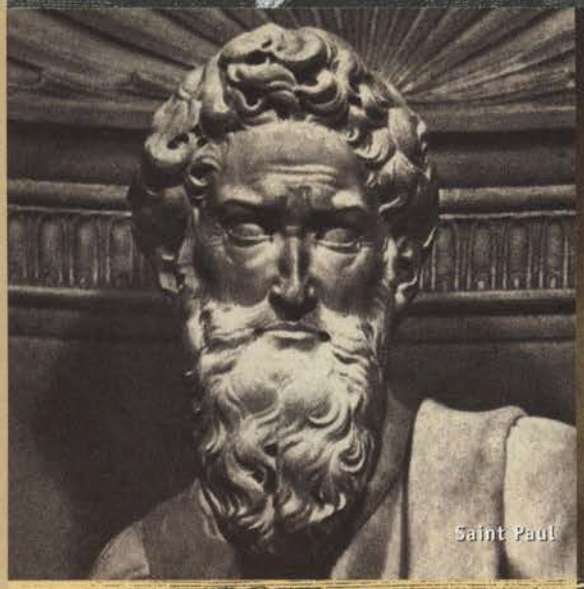
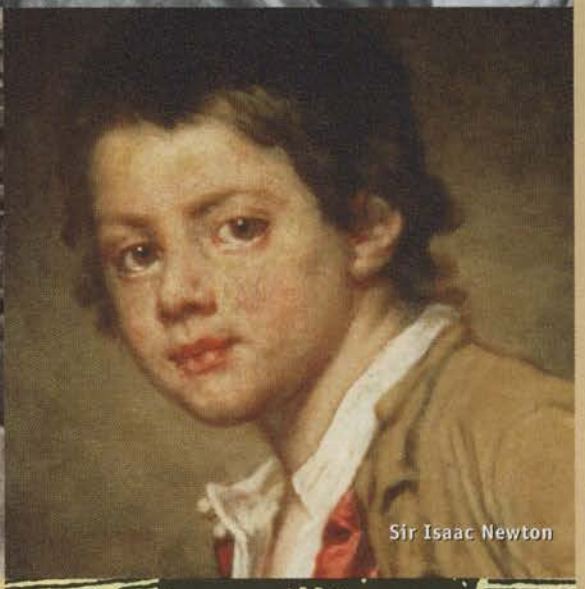
Pour documentation voir pages A-43, A-44, A-45

IL FUT UN TEMPS OÙ LES PERSONNES ÉPILEPTIQUES DEVAIENT DÉPLOYER DES EFFORTS CONSIDÉRABLES OU FAIRE PREUVE DE TALENTS EXTRAORDINAIRES POUR RÉUSSIR DANS LA VIE. HEUREUSEMENT, LES ENFANTS ET LES ADULTES ÉPILEPTIQUES QUE VOUS TRAITEZ PEUVENT MAINTENANT BÉNÉFICIER D'OPTIONS MOINS ÉPROUVANTES QUE PAR LE PASSÉ.

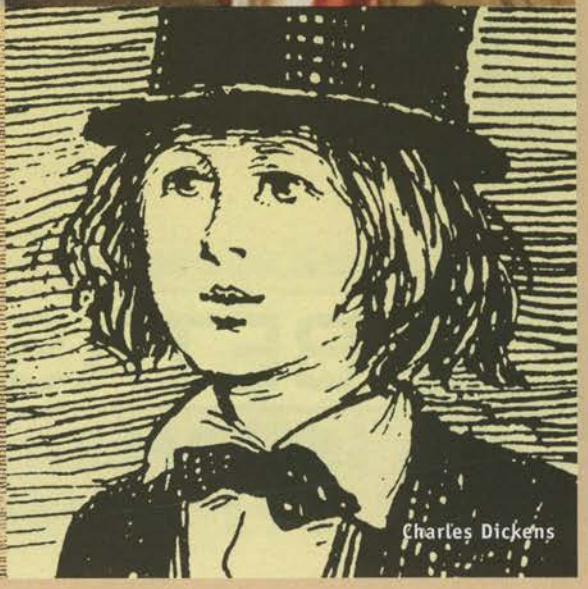

Edgar Allan Poe


Jeanne d'Arc


Saint Paul


Sir Isaac Newton


Pythagore


Charles Dickens

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MAINTENANT INDIQUÉ CHEZ L'ENFANT

Comprimés et capsules à saupoudrer PrTOPAMAX* (topiramate) : indiqués en tant que traitement d'appoint dans la prise en charge des patients (adultes et enfants de deux ans ou plus) épileptiques dont l'état n'est pas maîtrisé de façon satisfaisante par le traitement traditionnel. Les renseignements sur l'emploi du topiramate en monothérapie sont encore limités[1].

Efficacité en cas de crises partielles initiales :

Posologie ajustée en fonction de la réponse de chaque patient[4,5] :

	N	Réduction ≥ 50 % du nombre de crises	Absence de crises
Adultes[a,c]	450	59 %	19 %
Enfants[b,c]	41	73 %	22 %

D'après les références 4 et 5

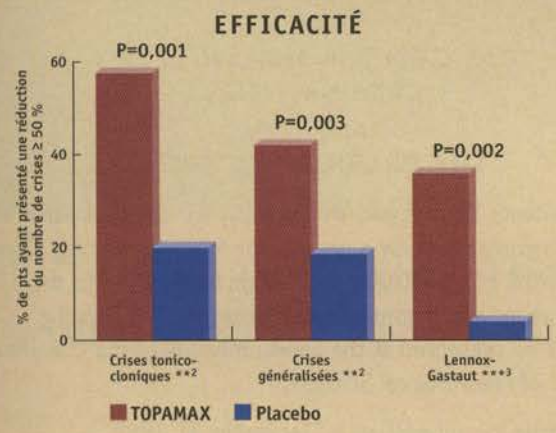
[a] Étude ouverte d'une durée de 20 semaines portant sur des adultes atteints de crises partielles initiales. Administration biquotidienne de TOPAMAX en tant que traitement d'appoint. La posologie optimale semblait comprise entre 300 et 350 mg/jour.

[b] Étude ouverte portant sur des enfants atteints de crises partielles initiales ayant participé à un essai à double insu contrôlé par placebo. Les réductions de la fréquence des crises ont été déterminées chez les enfants qui avaient été traités pendant au moins 3 mois. Administration biquotidienne de TOPAMAX en tant que traitement d'appoint. Les sujets ont reçu un traitement par topiramate pendant une période moyenne de 8 mois, selon une posologie moyenne de 10 mg/kg/jour (4-20 mg/kg/jour).

Pour connaître les posologies recommandées, reportez-vous aux Renseignements thérapeutiques concernant TOPAMAX*.

Meilleure maîtrise d'un grand nombre de types de crises :

- Des données complémentaires recueillies dans le cadre d'études randomisées, à double insu et contrôlées par placebo portant sur des adultes et un nombre restreint d'enfants ont en outre montré que ce médicament était efficace en tant que traitement d'appoint en cas de :
- **crise tonico-clonique primaire généralisée[1]**
- **crise associée au syndrome de Lennox-Gastaut[1]**



D'après les références 2 et 3

**Phase de traitement à double insu d'une durée de 20 semaines (données de départ recueillies pendant une période initiale de 8 semaines, et période de traitement de 12 semaines) consistant en l'administration de TOPAMAX (n = 39, y compris 8 enfants ≤ 16 ans) en tant que traitement d'appoint à raison de 2x/j, ou d'un placebo (n = 41). La posologie de TOPAMAX était ajustée jusqu'à ce qu'une dose cible d'environ 6 mg/kg/jour soit atteinte.

***Chutes brusques par dérobement des jambes et crises tonico-cloniques : phase de traitement à double insu d'une durée de 11 semaines consistant en l'administration de TOPAMAX (n = 48) à raison de 2x/j en tant que traitement d'appoint, ou d'un placebo (n = 50); âge moyen des patients : 11,2 ans. La posologie de TOPAMAX était ajustée jusqu'à ce qu'une dose cible d'environ 6 mg/kg/jour soit atteinte.

COMPRIMÉS DÉSORMAIS INSCRITS AU FORMULAIRE‡

‡Indemnité partielle – Ontario, Nouvelle-Écosse, Nouveau-Brunswick, Î.-P.-É.
Indemnité intégrale – Québec, Saskatchewan, Colombie-Britannique, Alberta, Manitoba

Un traitement d'appoint approprié en première intention pour nombre de vos patients :

Profil d'effets secondaires favorable :

- Comme pour la plupart des antiépileptiques, les effets secondaires le plus fréquemment signalés relèvent du SNC[†,†1,6] :

- Généralement légers à modérés, ils surviennent à un stade précoce du traitement et sont passagers[1,6]
- En cas de survenue d'effets secondaires :

Envisagez de réduire la posologie de TOPAMAX, le taux d'augmentation de la posologie, et/ou la posologie de l'antiépileptique administré de façon concomitante[8].

- Chez les enfants traités dans le cadre des essais contrôlés, on n'a signalé aucun abandon du traitement attribuable à des manifestations indésirables lorsque la posologie était de 5 à 9 mg/kg/jour[1].

Profil d'innocuité :

- Aucune donnée n'a montré, jusqu'à présent, qu'il existait un lien entre l'emploi de TOPAMAX et les affections suivantes : éruption cutanée potentiellement mortelle, rétrécissement permanent du champ visuel ou syndrome des ovaires polykystiques[1,c].
- Perte de poids

Adultes : une perte de poids modérée peut se produire au cours des 12 premiers mois, les pertes pondérales les plus importantes survenant entre le 3e et le 6e mois, avec un pic au 9e mois[7].

Enfants : 96 % des enfants traités dans le cadre des essais cliniques pendant au moins un an et ayant subi une perte pondérale ont repris du poids au cours de la période d'exécution des essais[11].

Posologie BID commode[1]

Maintenant offert sous forme de capsules à saupoudrer à 15 et 25 mg, une présentation encore plus commode[1]:

La capsule peut être avalée entière ou on peut en saupoudrer le contenu sur de la nourriture
Les capsules sont bioéquivalentes aux comprimés TOPAMAX

† Les effets à long terme d'une perte pondérale n'ont pas été établis chez l'enfant.

†† Manifestations indésirables associées au SNC : Somnolence (30,1 %), étourdissements (28,3 %), ataxie (21,2 %), troubles de la parole (16,8 %), ralentissement psychomoteur (16,8 %), nystagmus (15,0 %), paresthésie (15,0 %), nervosité (15,9 %), problèmes de concentration/d'attention (8,0 %), confusion (9,7 %), dépression (8,0 %), anorexie (5,3 %), troubles du langage (6,2 %) et troubles de l'humeur (3,5 %)[1]. Une analyse portant sur 1 446 adultes et 303 enfants indique que ces deux groupes semblent présenter des profils de manifestations indésirables similaires[1].

Pour obtenir des renseignements complets sur les modalités de prescription de TOPAMAX, veuillez vous reporter aux Renseignements thérapeutiques concernant ce produit.

c Données internes. JANSSEN-ORTHO Inc. Mai 1999

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 JATX991005FA

TOPAMAX topiramate

Pour aider les patients épileptiques à mieux profiter de la vie

Pour documentation voir pages A-68, A-69, A-70

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SOCIETY PRIZES

Canadian Neurological Society announces the 2000 FRANCIS McNAUGHTON MEMORIAL PRIZE and the ANDRÉ BARBEAU MEMORIAL PRIZE

The Canadian Neurological Society invites submissions for its two prizes: the Francis McNaughton Memorial Prize for clinical research and the André Barbeau Memorial Prize for basic research.

The Francis McNaughton and André Barbeau Memorial Prizes, designed to encourage Neurology Trainees to undertake research projects, are awarded for the best submitted papers based on work done during the Neurology residency or in post-residency training. The contestants need not be the sole authors but should have been primarily responsible for the work to be presented.

The winning papers in each category will be presented at the annual meeting of the Canadian Congress of Neurological Sciences. The deadline for submission is December 31, 1999.

Canadian Society of Clinical Neurophysiologists announces THE HERBERT JASPER PRIZE

The Herbert Jasper Prize is awarded annually for the best submitted paper in clinical or basic neurophysiology by a resident or fellow in training. Others who are within three years of receiving a doctorate or fellowship are also eligible.

The prize consists of an honorarium of $250.00, economy air transportation and 2 nights hotel accommodation to attend the annual meeting of the Canadian Congress of Neurological Sciences where the winning paper will be presented.

The deadline for submission is December 31, 1999.

The Royal College of Physicians and Surgeons of Canada with the co-operation of the Canadian Neurosurgical Society announces the 2000 K.G. McKENZIE MEMORIAL PRIZE AND AWARD

The McKenzie Prize for Basic Neurosciences Research and the McKenzie Prize for Clinical Neuroscience Research

There will be one citation and prize of $1,000.00 in each of the Basic Neuroscience and Clinical Neuroscience categories for the best manuscripts submitted to the McKenzie Award Committee of the Canadian Neurosurgical Society, by a neurosurgical resident, in which he or she is the principal author. The recipients will have their expenses, including air fare, 2 nights hotel accommodation and registration fees, paid for as part of the Prizes. A second prize of $500.00 may also be awarded in each category.

The deadline for the Prize for 2000 is December 31, 1999 and it is firm.

The Canadian Association for Child Neurology announces THE PRESIDENT'S PRIZE

The President's Prize is awarded annually for the best paper in pediatric neuroscience by a resident or fellow in training. The prize consists of a $500.00 honorarium, registration to the annual meeting and a commemorative scroll. The winning paper will be presented at the annual meeting of the Canadian Congress of Neurological Sciences.

The deadline for submission is December 31, 1999.

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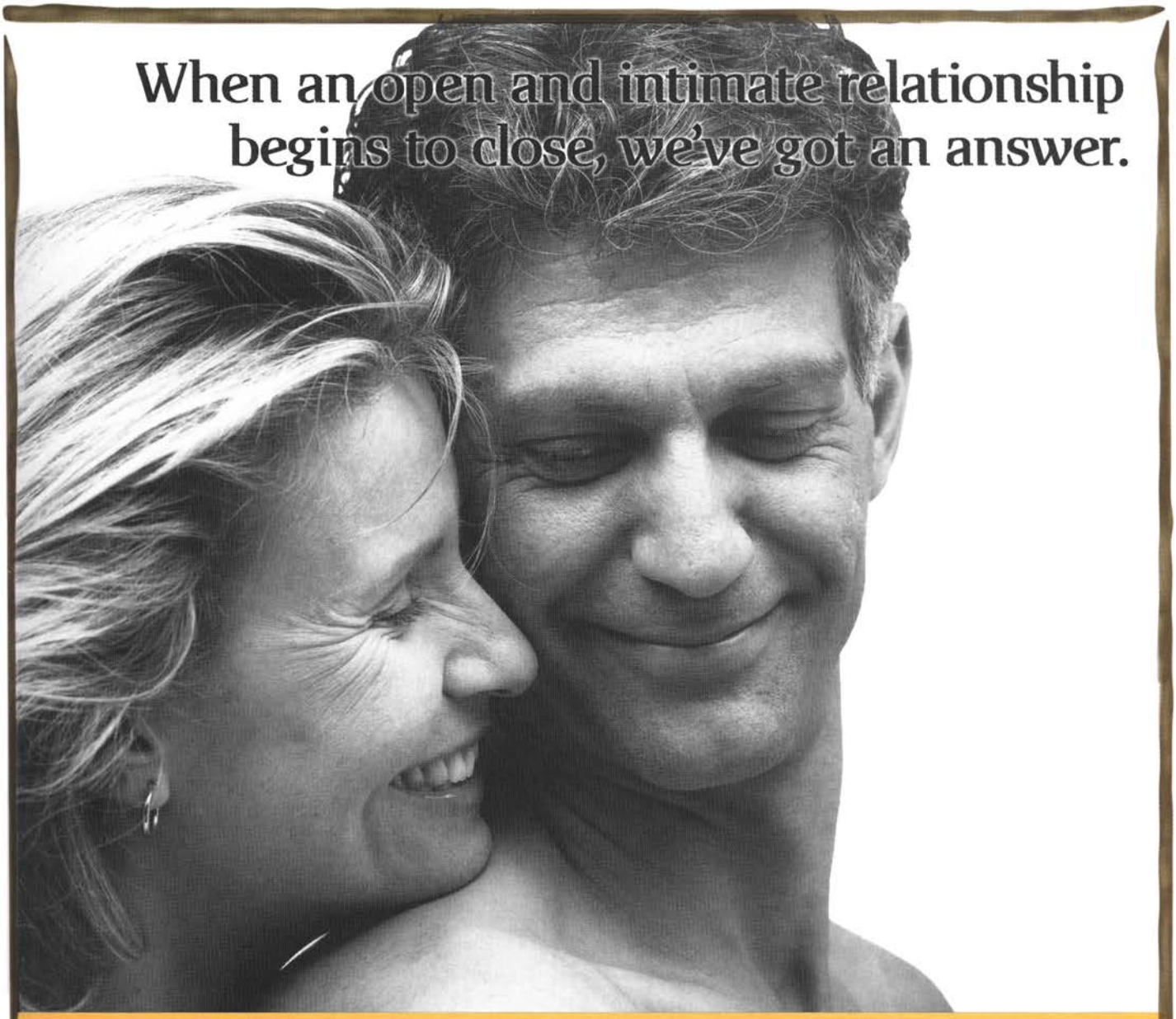

Introducing VIAGRA®. The ease of oral treatment to help restore natural erectile function, that works only with sexual stimulation.

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.

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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]
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For brief prescribing information see pages A-63, A-64

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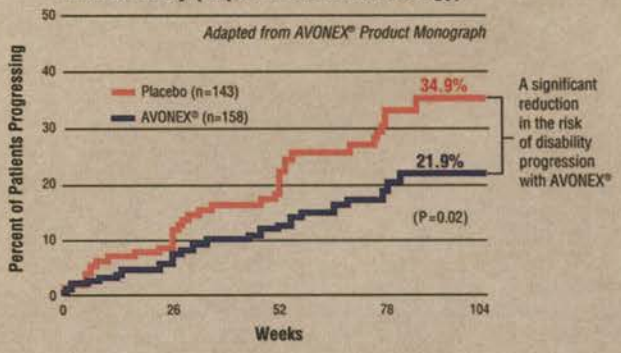

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Please see product monograph for important patient selection and monitoring information.

For brief prescribing information see pages A-65, A-66, A-67

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For brief prescribing information see pages A-61, A-62

25 Years Ago in the Canadian Journal of Neurological Sciences

The Effects of Motor Cortical Stimulation on the Excitability of Spinal Motoneurons in Man

S.H. Milner-Brown, J.P. Girvin, W.F. Brown

Summary: The pyramidal tract and particularly the direct cortico-motoneuronal components (DCM) have become increasingly important in the higher primates. Minimal single pulse precentral stimulation in man evokes EMG discharges from the contralateral hand muscles with a latency of 18-21 milliseconds. The excitability changes produced by such cortical stimulation on the upper limb H-reflex has been observed to include a short duration early facilitation probably corresponding to the DCM input and a later, longer lasting facilitation mediated by the same and probably other corticofugal projections. Potentiation of the H-reflex in the upper limbs by means of postcentral excitation required much higher single pulse stimulus intensities and the changes in excitability produced on the spinal motoneurons could have been explained by physical extension of the stimulus current to the precentral region. Isometric contraction potentiated the H-reflex produced by combinations of precentral cortical and peripheral nerve stimulation but no direct evidence was found to support a possible transcortical basis for the V_2 stretch reflex.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):245

Physiopathology of Experimental Parkinsonism in the Monkey

Louis J. Poirier, Michel Filion, Louis LaRochelle, Jean-Claude Péchadre

Summary: Postural or Parkinson-like tremor, which results from the impairment of mechanisms which are predominantly lateralized in the brain, is most likely related to the combined impairment of the dopaminergic nigrostriatal pathway and the corresponding rubro-olivo-cerebello-rubral loop (without excluding the possibility that other nervous mechanisms interconnected with these structures may represent an alternative disturbance). The integrity of the internal division of the pallidum and the ventrolateral area of the thalamus and their efferent fibers as well as the motor cortex and certain of its cortico-subcortico-spinal pathways is apparently an essential feature for the elaboration of the rhythmic burst associated with the appearance of postural tremor. The integrity of the spinal sensory roots and the rubro-tegmentospinal tract is not a prerequisite for the expression of postural tremor, a condition which seems essential for the production of rigidity. The latter facts suggest that the disturbances which subserve these two types of motor impairment, often concomitantly present in Parkinsonism, partially involve the impairment of different mechanisms although the loss of the DA fibers originating in the substantia nigra and ending in the neostriatum appears to represent a disturbance common to both types of disorders.

Bradykinesia which may be associated with an impairment of catecholamine metabolism (and more especially the neostriatal DA mechanisms) on both sides of the brain may also result from bilateral lesions of the pallidum or of its outflow corresponding, in the main, to the pallidothalamic fibers ending in the ventrolateral thalamus. The latter types of lesions most likely exclude the influence of the monoaminergic, cholinergic and gabaminergic activities normally originating in the striopallidal system and influencing the activity transmitted to other CNS mechanisms. Severe akinesia, however, apparently depends on more profound and generalized disturbances of brain monoamine metabolism with or without the involvement of other ill-defined mechanisms. At any rate the impairment of the brain DA mechanisms (and especially those of the neostriatum) seems to represent a major feature in the production of the Parkinsonian type of akinesia. Further work is needed to establish the relative importance of the loss of catecholaminergic mechanisms other than those of the neostriatum in the production of akinesia.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):255

Roles of Cerebellum and Basal Ganglia in Initiation and Control of Movements

V.B. Brooks

Summary: Theories of function of the cerebellum and basal ganglia are examined in the light of recent experimental findings obtained with the local cooling method, and both are matched against clinical observations. Evidence is summarized for a programming and initiating role in monkeys' elbow movements of the lateral, and to a lesser degree, intermediate, cerebellum. Cooling either nuclei affected movements, but neither seemed to be important for precentral cortical unit discharge accompanying compensation for suddenly applied load pulses. The globus pallidus seemed to be importantly involved in movement guidance in the absence of vision.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):265

Physiological Basis of Cerebella Dysmetria

John T. Murphy, Hon. C. Kwan, William A. MacKay, Yiu C. Wong

Summary: A primary control system for the arm position is formulated. The hypothesis that the cerebellum is a part of the system controller is checked by studying the nerve cells responses in the cerebellum, and motor cortex, to natural activation of muscular receptors. The results show that the cerebellum receives feedback information related to the speed of these receptors. The discussion concentrates on how the interruption of this feedback may result in excessive oscillations to instability. These observations are the base for evaluating how the cerebral lesion produce dismeasurements.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):279

For brief prescribing information see pages A-46

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25 Years Ago in the Canadian Journal of Neurological Sciences

Motor Responses to Sudden Limb Displacement in Primates with Specific CNS Lesions and in Human Patients with Motor System Disorders

R.G. Lee, W.G. Tatton

Summary: Central feedback pathways for motor control were studied by recording EMG responses to sudden upper limb displacements in humans and monkeys using a precision torque motor to generate step load changes. Normal human subjects showed three short-latency EMG responses (M1, M2 and M3) which appear to correspond to those recorded from trained monkeys. The M2 and M3 components, thought to represent feedback in supraspinal pathways, were significantly increased when the subjects were instructed to actively compensate for the load changes.

Parkinsonian patients with rigidity showed evidence of markedly increased feedback over the interval of the M2 and M3 responses and appeared to have lost the ability to modulate feedback according to the motor task being performed. The results are discussed with reference to recent research on motor control mechanisms in primates and a tentative model for the basis of Parkinsonian rigidity is proposed.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):285

Why Transcortical Reflexes?

Mario Wiesendanger, Dieter G. Rüegg, Gregory E. Lucier

Summary: Experiments in humans and in monkeys have indicated that load perturbations, occurring during voluntary movements and postural activity, may be automatically compensated for. Overall muscle stiffness opposing load changes is determined by the visco-elastic properties of the muscle, by segmental reflex actions and finally by long-loop reflexes. Under certain circumstances, for instance when the subject or the experimental monkey is "prepared" to counteract perturbations which are unpredictable in time, the long-loop "reflexes" appear to be responsible for most of the corrective muscle tension. Experiments in anaesthetized monkeys revealed that signals from stretch afferents reach neurons of the motor cortex, possibly via a relay in the cortical area 3a. The latencies of these responses to well controlled muscle stretches were in the same range as motor cortical cell discharges recorded in alert monkeys subjected to load perturbation. Furthermore, these responses of cells in the motor cortex also had the appropriate timing to indicate a causal relationship with the long-latency electromyographic responses to load changes referred to above. These experimental results therefore strongly support the hypothesis, first proposed by Phillips (1969), of a transcortical servo-loop adjusting motor cortical output according to the load conditions in which movements are performed.

The major advantage of transcortical regulations as opposed to segmental regulations, seems to be a powerful gain control acting at the cortical level; it was repeatedly shown that the long-loop reflexes are strongly modifiable and under voluntary control. It is suggested that an adaptive gain control at the cortical level is a prerequisite to preserve the complex capabilities of the motor cortex as the chief "executive" for skilled, preprogrammed movements. A loss of this adaptive gain control may be, at least partly, the cause of motor disorders such as rigidity in Parkinsonian patients, as reported by Tatton and Lee (1975). It is suggested that further investigations of the control of transcortical reflexes may aid in the understanding of the pathophysiology of motor disabilities.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):295

25 Years Ago in the Canadian Journal of Neurological Sciences

Decorticate Spasticity: A Re-examination using Quantitative Assessment in the Primate

R.R. Tasker, F. Gentili, K. Sogabe, M. Shanlin, P. Hawrylyshyn

Summary: Decorticate spasticity in the squirrel monkey was chosen as a convenient laboratory model of spasticity capable of quantitative assessment upon which to evaluate various currently popular clinical spasmolytic measures. The effects of a wide variety of cortical lesions were studied involving primary and supplementary motor, premotor and parietal cortex unilaterally, measuring muscle tone with the evoked integrated EMG technique. Measurable spasticity resulted only if primary motor cortex was ablated bilaterally usually but not always preferentially involving biceps brachii and quadriceps. Resulting postures were variable offering no justification for the term "decorticate posture". The integrated evoked EMG was proportional to rate of stretch and chiefly phasic in type as in hemiplegic man. Stereotactic dentatectomy resulted in this spasticity, but was without effect in intercollicular or anemic decerebrate cats. The mechanism of the spasticity and of the cerebellar effects are discussed.

Can. J. Neurol. Sci. 1975;2:(Suppl 1):303

For brief prescribing information see pages A-46

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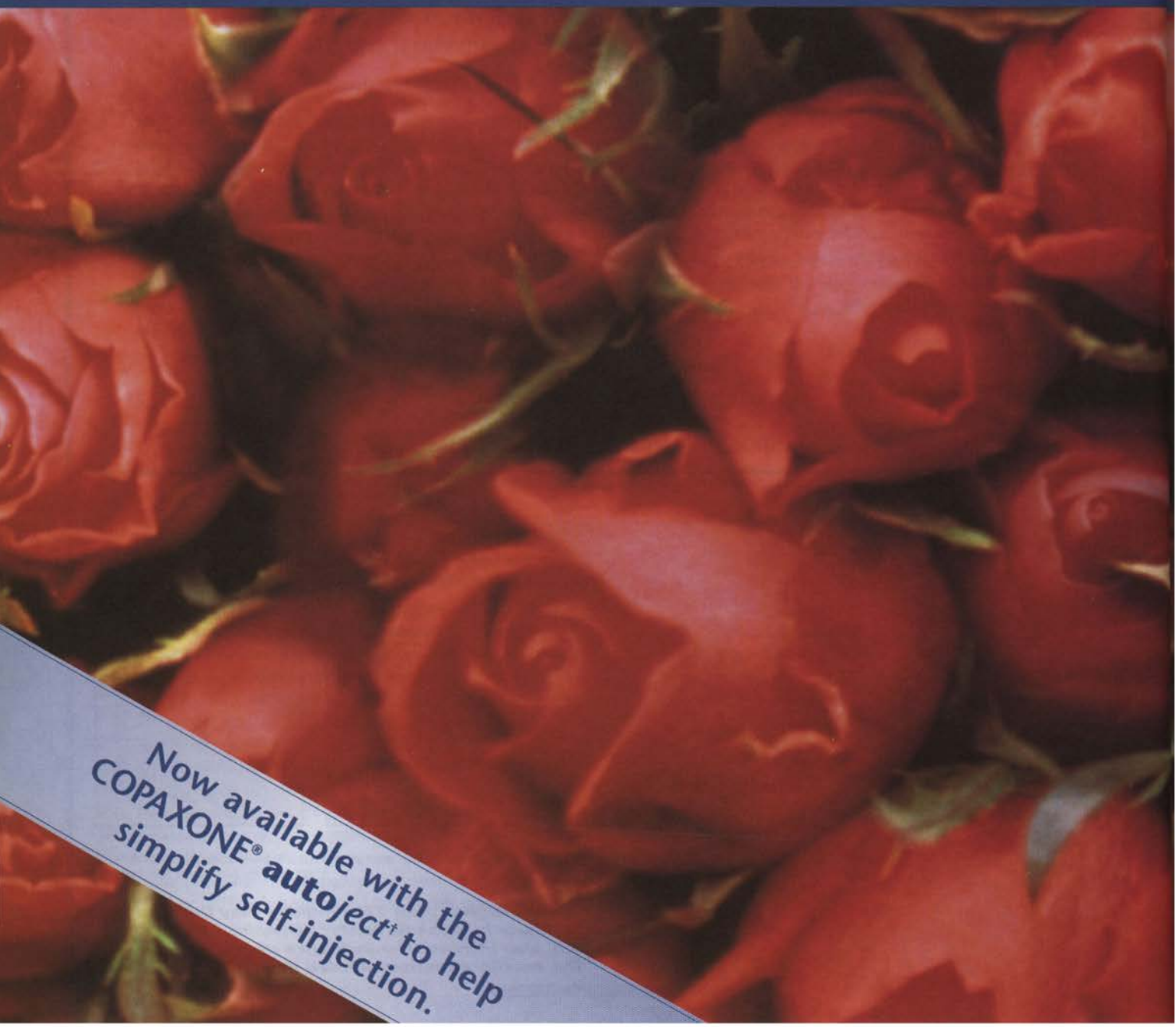

Safety and efficacy of COPAXONE® beyond 2 years have not been adequately studied in placebo-controlled trials. A correlation between reduction in attack frequency alone and decreased risk of future disability remains to be established.

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† **auto***ject* is manufactured by Owen Mumford, Ltd.

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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[§] and tolerability[‡] in over 129 million patient days of therapy worldwide.[5]

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† 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.
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For brief prescribing information see pages A-51, 52

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Message from the Editor

It is with great pleasure and delight that I have assumed the role as Editor of the Canadian Journal of Neurological Sciences. The Journal has had strong leadership through its founding Editor Dr. R. Ross followed by the guidance of Dr. Robert G. Lee and more recently Dr. James Sharpe. We are all indebted to these editors and the editorial staff at the Journal for creating a self sufficient, strong and internationally recognized Journal. Unlike a number of its contemporaries, The Canadian Journal of Neurological Sciences is unique in its combination of clinical neurology, neurosurgery, child neurology and clinical neurophysiology, the four components of our Canadian Congress of Neurological Sciences.

We have a number of challenges ahead of us that include maintaining our leadership over newer journals of limited scope and making a smooth transition into the era of electronic publishing. For this we have important local expertise and you will be hearing more about our electronic initiatives in the near future.

In the last issue and in this issue, we honor several outstanding Canadian neuroscientists. This issue honors Dr. Charles Drake a Canadian neurosurgeon who revolutionized the treatment of vertebral-basilar territory cerebral aneurysms. We include an introductory "In Memoriam" by his colleagues, Gary Ferguson and Vladimir Hachinski; an address prepared by Dr. Drake just before his death, on his early training years in Toronto; a reproduction of one of his famous "Track Sheets" used to record details of his aneurysm surgery; and a reproduction of one of Dr. Drake's articles originally published in the Journal of Neurosurgery. It is my hope that these articles in our Journal will help to highlight the outstanding contributions made by Dr. Drake to neurosurgery internationally.

In this issue we introduce a new educational article "Neuroimaging Highlight" edited by Drs. Mark Hudon and William Hu. Each subsequent issue of the Journal will include a Neuroimaging Highlight and I hope it will serve as an important feature for our readers.

This editor would like to acknowledge the superb editorial staff that will help me to guide the Journal, including Sally Gregg, Sue Impey and Margaret Peterson. I will continue to call on Canadian and international neuroscientists to help in our well established review process. In advance, I would like to thank those of you who can continue to contribute to our peer review.

Our Journal has international contributors and readership and the strength of its citations will depend on the quality of the work we can attract. We encourage Canadian and international authors to send us their best work.

Douglas Zochodne

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INFORMATION FOR AUTHORS

The Canadian Journal of Neurological Sciences publishes original articles in neurology, neurosurgery and basic neurosciences. Manuscripts are considered for publication with the understanding that they, or the essence of their content, have not been published elsewhere except in abstract form and are not under simultaneous consideration by another journal. Articles undergo peer review. Manuscripts should be submitted to: James A. Sharpe, M.D., Editor. Canadian Journal of Neurological Sciences, P.O. Box 4220, Station C, Calgary, AB Canada T2T 5N1

Manuscript Preparation

• 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.

• After a paper has been reviewed, the author will be requested to submit four copies of the revised manuscript, including illustrations. Supply a computer diskette (3 1/2" size) containing the article *saved in an RTF format*. Identify clearly first author's name, file name, word processing program and version, and system (i.e. PC or Mac). Clearly indicate the order and importance of headings.

• 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.

• ***References*** should be numbered in the order of their citation in the text. Those cited only in tables and legends for illustrations are numbered according to the sequence established by the first identification in the text of a particular table or illustration. Titles of journals should be abbreviated according to the style used in Index Medicus. References should list the names of up to five authors; if there are more, cite the first three, then *et al*. Provide the full title, year of publication, volume number and inclusive pagination for journal articles. For any reference cited as "in press", five copies of the article must accompany the author's manuscript. Do not reference unpublished or "submitted" papers; these can be mentioned in the body of the text and authors must provide five copies of "submitted" manuscripts. Avoid "personal communications" and, if necessary, include them in the body of the text, not among the references. Reference citations should not include unpublished presentations or other non-accessible material. Books or chapter references should also include the place of publication and the name of the publisher. Examples of correct forms of reference follow:

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.

• ***Illustrations*** Submit five original sets of illustrations. We will not return illustrations; therefore, authors should keep negatives for all photographs. Submit high quality glossy black and white photographs preferable 127 x 173 mm (5" x 7"). This includes graphs and diagrams. Do NOT send photocopies of illustrations. Original artwork and radiographs should not be submitted. The additional cost of coloured illustrations must be borne by the author; quotations are available upon request from the Journal office. Identify each figure with a label at the back indicating top, figure number and first author. Letters and arrows applied to the figures to identify particular findings should be professional appliques suitable for publication. Photomicrographs should include a calibration bar with a scale indicated on the figure or in the legend. Legends for illustrations should be typed on a separate page from the illustrations.

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• ***Review articles*** on selected topics are also published. They are usually invited, but unsolicited reviews will be considered. It is recommended that authors intending to submit review articles contact the Editor in advance.

• ***Letters to the Editor*** concerning matters arising in recent articles are welcome. Letters should be limited to two double-spaced pages and may include one illustration and a maximum of four references.

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