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# Update en migraña

abril 2009 – Época I

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## British Medical Journal (BMJ)

- No hay artículos sobre cefalea-migraña publicados este mes



## JAMA

- No hay artículos sobre cefalea-migraña publicados este mes



## New England Journal of Medicine

- No hay artículos sobre cefalea-migraña publicados este mes



## Lancet

- No hay artículos sobre cefalea-migraña publicados este mes

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## How Children and Parents Evaluate the Headache Centre's Intervention

Anna Ferrari, Francesca Pileri, Luca Spaccapelo, Maurizio Zappaterra, Ciro Ruggerini, Raffaella Tacchi, Alfio Bertolini

Headache: The Journal of Head and Face Pain Volume 49 Issue 2 (February 2009) Pages 194 – 201

### ABSTRACT

**Background.**—While adult headache patients' satisfaction with treatments has been widely investigated, less attention has been paid to children and adolescent headache patients' opinions and their parents' views.

**Objective.**—The aim of our follow-up survey was to analyze the outcomes of the Headache Centre's intervention and the evolution of headache according to patients until the age of 16 and their parents.

**Methods.**—We studied all outpatients suffering from episodic primary headache according to International Classification of Headache Disorders 2nd edition criteria, seen for the first time in 2005-2006 at the Headache Centre of the University Hospital of Modena (Italy), and at least one of their parents. The duration of the follow-up ranged from 1 to 3 years. For the purpose of the study, a specific questionnaire was created and administered by a telephone interview.

**Results.**—We enrolled 84 patients (38 females, 45%; 46 males, 55%; mean age  $\pm$  SD: 12.9  $\pm$  2.9 years) with primary headache: migraine without aura 66%, episodic tension-type headache 23%, migraine with aura 11%. At the follow-up, 70% of the patients reported that headache had improved; frequency had decreased significantly more than severity ( $P = .000$ , Fisher's exact test), both in those who had followed a prophylactic treatment and in those who had not. A high percentage of the children and parents could precisely indicate trigger factors for headache: especially excessive worrying and studying. The patients reporting an improvement attributed it to pharmacological prophylactic treatment, but also to other factors: first of all, better school results and more happiness than before. Seventy-seven percent of the parents thought that the Headache Centre's intervention had helped them to better understand and manage their children's headache.

**Conclusions.**—Children's and adolescents' headache has in most cases a favorable prognosis; the Headache Centre's intervention is considered effective by most parents. We must increase and focus therapeutic efforts addressed to the few patients with worsening headaches in spite of treatment, since these children's/adolescents' headache also is at risk to progress in the adult age

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## Migraine in Adolescents: Validation of a Screening Questionnaire

Luca Valentini, Francesca Valent, Marco Mucchiut, Fabio Barbone, Paolo Bergonzi, Giorgio Zanchin  
Headache: The Journal of Head and Face Pain Volume 49 Issue 2 (February 2009) Pages 202 – 211

### ABSTRACT

Background.—Few studies in adolescents deal with the level of agreement between questionnaire and interview information in relation to headache symptoms.

Objective.—To evaluate the validity of a self-administered questionnaire on headache for use in epidemiological studies of Italian high school students.

Methods.—The questionnaire incorporated all items required for diagnosing migraine according to the criteria from the 2004 International Classification of Headache Disorders. The migraine diagnoses obtained from questionnaires were validated against the gold standard diagnoses by a headache specialist.

Results.—Out of 104 students answering the questionnaires, 93 (89.4%) participated in extensive semi-structured interviews by a neurologist. The chance-corrected agreement rate (kappa) was 0.66, which is considered good. The questionnaire-based migraine diagnosis had a sensitivity of 67.3%, specificity of 100%, positive predictive value of 100%, and negative predictive value of 73.3%.

Conclusions.—The results indicate that our self-administered questionnaire may be an acceptable instrument in determining the prevalence of migraine sufferers in the northeast Italy adolescent population, useful in identifying subjects with "definite" migraine.

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## Electronic Medical Record System in a Headache Specialty Practice: A Patient Satisfaction Survey

Marshall C. Freeman, Anne P. Taylor, James U. Adelman

Headache: The Journal of Head and Face Pain Volume 49 Issue 2 (February 2009) Pages 212 – 215

### ABSTRACT

Background.—Since November 2002, Headache Wellness Center, a specialty headache practice in Greensboro, NC, has used exclusively an electronic medical record (EMR) system for all patient clinical contacts.

Objective.—To assess patient satisfaction and perceptions regarding this new office technology and to better understand the EMR-patient-physician relationship.

Methods.—An EMR satisfaction survey was administered from February to June 2006 and was completed by 394 patients. All patients were known to the practice and completed the survey at the time of a headache revisit evaluation.

Results.—The majority of respondents believed that EMR system use was not intrusive; did not draw attention away from patient contact; provided more efficient and accurate interactions; and preferred an EMR system in this particular headache setting.

Conclusion.—Patient satisfaction with an EMR system in a headache specialty practice was high.

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## Evaluation of Carisbamate for the Treatment of Migraine in a Randomized, Double-Blind Trial

Roger K. Cady, Ninan Mathew, Hans-Christoph Diener, Peter Hu, Magali Haas, Gerald P. Novak  
Headache: The Journal of Head and Face Pain Volume 49 Issue 2 (February 2009) Pages 216 – 226

### ABSTRACT

**Objective.**—This study explored the dose-response relationship of carisbamate administered at doses of 100 mg per day, 300 mg per day, or 600 mg per day, in the prevention of migraine.

**Background.**—Carisbamate ([S]-2-O-carbamoyl-1-o-chlorophenyl-ethanol; RWJ 333369) is a new chemical entity being studied for efficacy as adjunctive therapy in partial onset epilepsy. Because some antiepileptic drugs are also efficacious in migraine, for example, topiramate and valproate sodium, we tested carisbamate in migraine prophylaxis.

**Design/Methods.**—This was a double-blind, placebo-controlled trial, approximately 22-week duration. The primary efficacy variable was the percent reduction from baseline through the double-blind phase in average monthly migraine frequency using a 48-hour rule. Patients were randomized 1 : 1 : 1 : 1 to treatment with carisbamate 100, 300, or 600 mg per day, or placebo. Migraine attacks were counted during a prospective 4-week baseline period, which was followed by a 2-week titration period, a 12-week maintenance period, a 1-week medication reduction period, and a 3-week observation period. Patients had an established history of migraine, with or without aura, for at least 1 year and a 3-month history of 3-12 migraine attacks per month.

**Results.**—Patients (n = 323) were predominantly women (85%) and white (89%); mean age was 41 years. There were no statistically significant differences between any of the carisbamate groups and placebo (P ≥ .6) for the median (range) percentage reduction from baseline to end point in average monthly migraine frequency (P value vs placebo): 37% (–250%, 100%) for placebo; 33% (–210%, 100%; P = .7) CRS 100 mg/day; 27% (–100%, 100%; P = .8) CRS 300 mg/day; and 35% (–87%, 100%; P = .6) CRS 600 mg/day. Results for secondary efficacy measures (responder rate, percent reduction in average monthly migraine frequency using the 24-hour rule, and percent reduction in average monthly migraine days) were consistent (P ≥ .075). The proportion of patients discontinuing because of adverse events was similar for placebo and carisbamate-treated patients (13% each). The most common (occurring in ≥5% of patients) treatment-emergent adverse events in patients treated with carisbamate were fatigue (17%) and nasopharyngitis (13%). Fatigue appeared to be dose related.

**Conclusions.**—Carisbamate was not more efficacious in migraine prophylaxis than placebo in this well-controlled study that included a suitable population. However, carisbamate monotherapy was well tolerated at doses up to 600 mg per day.

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## Association Between Polymorphisms in the 2-adrenoceptor Gene and Migraine in Women

Markus Schürks, Tobias Kurth, Paul M Ridker, Julie E. Buring, Robert Y.L. Zee  
Headache: The Journal of Head and Face Pain Volume 49 Issue 2 (February 2009) Pages 235 – 244

### ABSTRACT

Objective.—To investigate the role of three common polymorphisms in the  $\beta$ 2-adrenoceptor gene in migraine.

Background.—Migraine has been associated with increased risk of cardiovascular disease and asthma in which  $\beta$ 2-adrenoceptors play an important role;  $\beta$ -adrenoceptor antagonists are used in migraine prevention. However, the role of variants in the  $\beta$ 2-adrenoceptor gene in migraine is unclear.

Methods.—Association study among 23,753 white women, participating in the Women's Health Study, for whom we had information on migraine at baseline and genotype status of the polymorphisms rs1042713 (Gly16Arg), rs1042714 (Gln27Glu), rs1800888 (Thr164Ile). Migraine was self-reported and we distinguished between any history of migraine, active migraine with and without aura, and prior migraine (history of migraine but not active migraine) in our analyses.

Results.—At baseline 4339 women reported any history of migraine. Of these, 3041 had active migraine (1221 migraine with aura, 1820 migraine without aura) and 1298 prior migraine. No migraine was reported by 19,414 women. Genotype- and haplotype-based analyses did not show an association of any of the gene variants tested with any history of migraine. The multivariable-adjusted odds ratios (ORs) (95% confidence intervals) for any history of migraine in the additive model were 1.0 (0.96-1.05) for rs1042713, 1.0 (0.95-1.05) for rs1042714, and 0.84 (0.68-1.05) for rs1800888. In the haplotype analysis the ORs ranged from 0.83 (0.67-1.03) to 1.01 (0.94-1.07) with Gly16-Glu27-Thr164 as the reference. We also did not find associations in the genotype- and haplotype-based analyses within migraine-specific subgroups.

Conclusions.—Our results do not support a role of 3 investigated polymorphisms in the  $\beta$ 2-adrenoceptor gene in migraine pathophysiology.

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## Serum Concentrations of s100b and NSE in Migraine

Michael Teepker, Karoline Munk, Veit Mylius, Anja Haag, Jens C. Möller, Wolfgang H. Oertel, Karsten Schepelmann

Headache: The Journal of Head and Face Pain Volume 49 Issue 2 (February 2009) Pages 245 – 252

### ABSTRACT

**Background.**—The protein s100b indicates astrocytal damage as well as dysfunction of the blood-brain barrier (BBB), and neuron-specific enolase (NSE) is regarded as a marker for neuronal cell loss. Recently, s100b was shown to be a potentially useful marker for migraine in children. In this study, we investigated the levels of s100b and NSE in adult migraineurs during and after migraine attacks in order to gain some more insight into migraine pathophysiology.

**Methods.**—Serum levels of s100b and NSE were measured in 21 migraineurs and compared with 21 healthy subjects matched by sex and age. In migraineurs, blood samples were taken during a migraine attack and following a pain-free period of 2-4 days.

**Results.**—During migraine attacks elevated s100b levels could be observed. Maximal concentrations were detected in the pain-free period after 2-4 days. Regarding NSE, serum levels were decreased slightly during and after migraine bouts.

**Conclusions.**—Our data suggest a prolonged disruption of BBB during and after migraine attacks. Other possible explanations concerning the detected serum levels of s100b and NSE will be discussed; however, neuronal cell death can be ruled out by the decreased serum concentrations of NSE. With regard to the results of the present study, further research is necessary to evaluate the role of s100b and NSE in migraine.

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## The Mode of Action of Migraine Triggers: A Hypothesis

Geoffrey A. Lambert, Alessandro S. Zagami

Headache: The Journal of Head and Face Pain Volume 49 Issue 2 (February 2009) Pages 253 – 275

### ABSTRACT

Objectives.—To review conjectured modes of action of migraine triggers and to present a new hypothesis about them.

Background.—Migraine attacks are initiated in many migraineurs by a variety of "triggers," although in some patients no external trigger can be identified. Many triggers provoke attacks with such a short latency that only some kind of neural mechanism can explain the triggering.

Results.—We present here a hypothesis that the pain of migraine has its ultimate origin in the cortex, but that the immediate generator is in the brainstem. Our hypothesis is that most migraines have triggers that produce excitation of cortical neurons and that this directly causes withdrawal of descending sensory inhibition originating in the brainstem. A wide range of evidence from the literature that cortical activation induced by a number of different mechanisms often produces headache is presented to support this notion. Several nuclei in the brainstem appear to participate in the selective control of trigeminovascular sensation through descending inhibitory mechanisms that arise in the cortex. In this review we focus on 2 of them, the periaqueductal gray matter and nucleus raphe magnus. Our own past results and those of others show that this inhibition is specific for craniovascular sensation and involves the neurotransmitter 5-hydroxytryptamine. Finally, we summarize our own recent experiments, which show that cortical activation by migraine triggers (including cortical spreading depression) inhibits neuronal discharge in the brainstem and facilitates trigeminovascular sensation.

Conclusion.—If the hypothesis can be proven and the neurotransmitters involved in the hypothetical trigger pathway can be identified, it may be possible to develop novel migraine preventative therapies.

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## Intravenous Lidocaine in the Treatment of Refractory Headache: A Retrospective Case Series

Michael Marmura, Noah Rosen, Muhammad Abbas, Stephen Silberstein  
Headache: The Journal of Head and Face Pain Volume 49 Issue 2 (February 2009) Pages 286 – 291

### ABSTRACT

**Background.**—New treatments are needed to treat chronic daily headache (CDH) and chronic cluster headache (CCH). New treatments are needed to treat this population and intravenous (IV) lidocaine is a novel treatment for CDH.

**Objective.**—The aim of this study was to examine the use of IV lidocaine for refractory CDH patients in an inpatient setting.

**Methods.**—This was an open-label, retrospective, uncontrolled study of IV lidocaine for 68 intractable headache patients in an inpatient setting. We reviewed the medical records of patients receiving IV lidocaine between February 6, 2003 and June 29, 2005.

**Results.**—Pretreatment headache scores averaged 7.9 on an 11-point scale and posttreatment scores averaged 3.9 representing an average change of 4. Average length of treatment was 8.5 days. Lidocaine infusion was generally well tolerated with a low incidence of adverse events leading to discontinuation of treatment.

**Conclusions.**—This study suggests benefit of lidocaine treatment and the need for further prospective analyses. The mechanism of lidocaine in treating headache is unknown.

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## Polymorphisms in the Renin-Angiotensin System and Migraine in Women

Markus Schürks, Robert Y. L. Zee, Julie E. Buring, Tobias Kurth

Headache: The Journal of Head and Face Pain Volume 49 Issue 2 (February 2009) Pages 292 – 299

### ABSTRACT

Background.—Recent findings suggest an association between the renin-angiotensin system and migraine. However, genetic studies are scarce and controversial.

Objective.—To investigate the association between the AGTR1 1166A > C and AGT Met235Thr polymorphisms with migraine and migraine aura status.

Methods.—We performed an association study among 25,000 Caucasian US women, participating in the Women's Health Study, with information on the AGTR1 1166A > C and AGT Met235Thr polymorphisms. Migraine and migraine aura status were self-reported. We distinguished between any history of migraine, active migraine with aura, active migraine without aura, and prior migraine (history of migraine, but not in the year prior to baseline). We used logistic regression to investigate the genotype–migraine association.

Results.—At baseline, 4577 (18.3%) women reported any history of migraine; 39.5% of the 3226 women with active migraine indicated aura. The polymorphisms were not associated with migraine or migraine-specific subgroups. We also did not find a significant interaction between the polymorphisms.

Conclusions.—Data from this large cohort of Caucasian women do not suggest an association of polymorphisms in the renin-angiotensin system with migraine or aura status. Future studies should focus on haplotype analyses and additional gene–gene as well as gene–environment interactions.

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## Prevalence of primary headaches and cranial neuralgias in men and women aged 55–94 years (Bruneck Study)

J Schwaiger, S Kiechl, K Seppi, M Sawires, H Stockner, T Erlacher, ML Mairhofer, H Niederkofler, G Rungger, A Gasperi, W Poewe, J Willeit  
Cephalalgia Volume 29 Issue 2 ,(February 2009) Pages 179 – 187

### ABSTRACT

The aim of the current study was to estimate the prevalence of all primary headaches and cranial neuralgias in the general community. As part of the population-based Bruneck Study, 574 men and women aged 55–94 years underwent extensive neurological and laboratory examinations involving a standardized headache interview. In the Bruneck Study population the lifetime prevalence of all primary headaches combined and of cranial neuralgias was 51.7 and 1.6%, respectively. Tension-type headache (40.9%) and migraine (19.3%) emerged as the most common types of headache. In men and women aged 55–94 years the 1-year prevalence of primary headaches was high at 40.5%. In this age range headaches caused significant impairment of health-related quality of life. The Bruneck Study has confirmed the high lifetime prevalence of primary headaches and cranial neuralgias in the general population and provided first valid prevalence data for all primary headaches based on International Classification of Headache Disorders, 2nd edition criteria.

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## Headache classification by history has only limited predictive value for headache episodes treated in controlled trials with OTC analgesics

H-C Diener, V Pfaffenrath, L Pageler, H Peil, B Aicher, RB Lipton  
Cephalalgia Volume 29 Issue 2 ,(February 2009) Pages 188 – 193

### ABSTRACT

We investigated the consistency between the headache diagnosis based on medical history and three treated headache episodes diagnosed based on a diary. In a randomized double-blind study including individuals with either migraine or tension-type headache (TTH) we showed significant superiority of the fixed combination of acetylsalicylic acid + paracetamol + caffeine over the combination without caffeine, the single preparations, and placebo in the treatment of headache. A neurologist performed a classification of the usual headache episodes and each of the three treated ones in a blinded fashion based on a structured questionnaire. This was done for the 1734 patients included in the efficacy analysis who usually treated their episodic TTH or migraine attacks with non-prescription analgesics. The overall percentage of patients with migraine and TTH remained relatively stable. The treated headache episodes were between 75 and 77% migraine, 18–20% were TTH and 5–7% could not be classified. We observed some shift in headache type within patients from prior history and in treated attacks. In 60% of patients all three treated episodes were of the type initially diagnosed by the neurologist by history (56% migraine and 4% episodic TTH). Of those with an initial diagnosis of migraine, 24% had at least one attack meeting criteria for TTH. Of patients with an initial diagnosis of TTH, 54% had at least one attack meeting the diagnostic criteria for migraine. Our results demonstrate that an initial headache diagnosis does not accurately predict the headache type treated in a randomized trial. Symptom features of treated headaches should be captured to ensure that the attack is of the type targeted by the clinical trial. The International Headache Society Guidelines for controlled clinical trials should be updated accordingly.

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## Ictal headache and visual sensitivity

M Piccioli, P Parisi, P Tisei, MP Villa, C Buttinelli, DGA Kasteleijn-Nolst Trenité  
Cephalalgia Volume 29 Issue 2 ,(February 2009) Pages 194 – 203

### ABSTRACT

Migrainous headache is reported by patients with photosensitive epilepsy, whereas their relatives complain more often about headache than the relatives of patients with other types of epilepsy. We therefore investigated whether headache itself could be an epileptic symptom related to photosensitivity. Four probands with headache and photosensitive epilepsy were selected. Their first-degree family members were studied using video-EEG with extensive intermittent photic stimulation and pattern stimulation. Nine of the 12 subjects (10 female and two male, mean age 30 years, range 14–46 years) proved to be photosensitive with either focal (n = 5) or generalized (n = 4) epileptiform discharges. In two subjects an ictal recording of headache occurred after visual stimulation. We found evidence that, in specific patients, headache could be an ictal sign of epilepsy. Photic stimulation during EEG recording can contribute to correct diagnosis and lead to the best care and management of the patient.

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## Correlation between abnormal brain excitability and emotional symptomatology in paediatric migraine

M Valeriani, F Galli, S Tarantino, D Graceffa, E Pignata, R Miliucci, G Biondi, A Tozzi, F Vigeveno, V Guidetti  
Cephalalgia Volume 29 Issue 2, (February 2009) Pages 204 – 213

### ABSTRACT

We investigated a possible correlation between brain excitability in children with migraine and tension-type headache (TTH) and their behavioural symptomatology, assessed by using the Child Behaviour Checklist (CBCL). The mismatch negativity (MMN) and P300 response were recorded in three successive blocks to test the amplitude reduction of each response from the first to the third block (habituation). MMN and P300 habituation was significantly lower in migraineurs and TTH children than in control subjects (two-way anova:  $P < 0.05$ ). In migraineurs, but not in TTH patients, significant positive correlations between the P300 habituation deficit and the CBCL scores were found ( $P < 0.05$ ), meaning that the migraineurs with the most reduced habituation showed also the worst behavioural symptomatology. To the best of our knowledge, this is the first study showing a correlation between neurophysiological abnormality and emotional symptomatology in migraine, suggesting a role of the latter in producing the migrainous phenotype.

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## Medication overuse headache and chronic migraine in a specialized headache centre: field-testing proposed new appendix criteria

P Zeeberg, J Olesen, R Jensen  
Cephalalgia Volume 29 Issue 2 ,(February 2009) Pages 214 – 220

### ABSTRACT

The classification subcommittee of the International Headache Society (IHS) has recently suggested revised criteria for medication overuse headache (MOH) and chronic migraine (CM). We field tested these revised criteria by applying them to the headache population at the Danish Headache Centre and compared the results with those using the current criteria. For CM we also tested two alternative criteria, one requiring  $\geq 4$  migraine days/month and  $\geq 15$  headache days/month, the second requiring  $\geq 15$  headache days/month and  $\geq 50\%$  migraine days. We included 969 patients with migraine or tension-type headache (TTH) among 1326 patients treated and dismissed in a 2-year period. Two hundred and eighty-five patients (30%) had TTH, 265 (27%) had migraine and 419 (43%) had mixed migraine and TTH. The current criteria for MOH classified 86 patients (9%) as MOH, 98 (10%) as probable MOH and 785 (81%) as not having MOH after a 2-month drug-free period. Using the appendix criteria, 284 patients (29%) were now classified as MOH, no patients as probable MOH and 685 (71%) as not having MOH. For CM only 16 patients (3%) fulfilled the current diagnostic criteria. This increased to 42 patients (7%) when we applied the appendix criteria. Using the less restrictive criteria of  $\geq 4$  migraine days and  $\geq 15$  headache days, 88 patients (14%) had CM, whereas the more restrictive criteria of  $\geq 15$  headache days and  $\geq 50\%$  migraine days resulted in 24 patients (4%) with CM. Our data suggest that the IHS has succeeded in choosing new criteria for CM which are neither too strict, nor too loose. For MOH, a shift to the appendix criteria will increase the number of MOH patients, but take into account the possibility of permanent changes in pain perception due to medication overuse and the possibility of a renewed effect of prophylactic drugs due to medication withdrawal. We therefore recommend the implementation of the appendix criteria for both MOH and CM into the main body of the International Classification of Headache Disorders.

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# Update en migraña

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## Management of medication overuse headache: 1-year randomized multicentre open-label trial

K Hagen, C Albrechtsen, ST Vilming, R Salvesen, M Grønning, G Helde, G Gravidahl, J-A Zwart, LJ Stovner  
Cephalalgia Volume 29 Issue 2, (February 2009) Pages 221 – 232

### ABSTRACT

It is a general belief that patients with medication overuse headache (MOH) need withdrawal of acute headache medication before they respond to prophylactic medication. In this 1-year open-labelled, multicentre study intention-to-treat analyses were performed on 56 patients with MOH. These were randomly assigned to receive prophylactic treatment from the start without detoxification, undergo a standard out-patient detoxification programme without prophylactic treatment from the start, or no specific treatment (5-month follow-up). The primary outcome measure, change in headache days per month, did not differ significantly between groups. However, the prophylaxis group had the greatest decrease in headache days compared with baseline, and also a significantly more pronounced reduction in total headache index (headache days/month  $\times$  headache intensity  $\times$  headache hours) at months 3 ( $P = 0.003$ ) and 12 ( $P = 0.017$ ) compared with the withdrawal group. At month 12, 53% of patients in the prophylaxis group had  $\geq 50\%$  reduction in monthly headache days compared with 25% in the withdrawal group ( $P = 0.081$ ). Early introduction of preventive treatment without a previous detoxification programme reduced total headache suffering more effectively compared with abrupt withdrawal. (ClinicalTrials.gov number, NCT00159588).

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## Are cortical spreading depression and headache in migraine causally linked?

J Wolthausen, S Sternberg, C Gerloff, A May  
Cephalalgia Volume 29 Issue 2, (February 2009) Pages 244-249

### ABSTRACT

During the past few decades, much controversy has surrounded the pathophysiology of migraine. Cortical spreading depression (CSD) is widely accepted as the neuronal process underlying visual auras. It has been proposed that CSD can also cause the headaches, at least in migraine with aura. We describe three patients, each fulfilling the International Headache Society criteria for migraine with aura, who suffered from headaches 6–10 days per month. Two patients were treated with flunarizine and the third patient with topiramate for the duration of 4 months. All patients reported that aura symptoms resolved completely, whereas the migraine headache attacks persisted or even increased. These observations question the theory that CSD (silent or not) is a prerequisite for migraine headaches.

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## Prevalence of neck pain and headaches: impact of computer use and other associative factors

L Smith, Q Louw, L Crous, K Grimmer-Somers  
Cephalalgia Volume 29 Issue 2, (February 2009) Pages 250 – 257

### ABSTRACT

Headaches and neck pain are reported to be among the most prevalent musculoskeletal complaints in the general population. A significant body of research has reported a high prevalence of headaches and neck pain among adolescents. Sitting for lengthy periods in fixed postures such as at computer terminals may result in adolescent neck pain and headaches. The aim of this paper was to report the association between computer use (exposure) and headaches and neck pain (outcome) among adolescent school students in a developing country. A cross-sectional study was conducted and comprehensive description of the data collection instrument was used to collect the data from 1073 high-school students. Headaches were associated with high psychosocial scores and were more common among girls. We found a concerning association between neck pain and high hours of computing for school students, and have confirmed the need to educate new computer users (school students) about appropriate ergonomics and postural health.

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## The cholinomimetic agent carbachol induces headache in healthy subjects

HW Schytz, T Wienecke, PS Oturai, J Olesen, M Ashina  
Cephalalgia Volume 29 Issue 2, (February 2009) Pages 258-268

### ABSTRACT

The parasympathetic nervous system is likely to be involved in migraine pathogenesis. We hypothesized that the cholinomimetic agonist carbachol would induce headache and vasodilation of cephalic and radial arteries. Carbachol (3 µg/kg) or placebo was randomly infused into 12 healthy subjects in a double-blind crossover study. Headache was scored on a verbal rating scale from 0–10. Velocity in the middle cerebral artery (VMCA) and diameter of the superficial temporal artery (STA) and radial artery (RA) were recorded. Nine participants developed headache after carbachol compared with three after placebo. The area under the curve for headache was increased after carbachol compared with placebo both during infusion (0–30 min) ( $P = 0.042$ ) and in the postinfusion period (30–90 min) ( $P = 0.027$ ). Carbachol infusion caused a drop in VMCA ( $P = 0.003$ ) and an increase in STA diameter ( $P = 0.006$ ), but no increase in the RA diameter ( $P = 0.200$ ). In conclusion, the study demonstrated that carbachol caused headache and dilation of cephalic arteries in healthy subjects.

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## Body mass index, migraine, migraine frequency and migraine features in women

AC Winter, K Berger, JE Buring, T Kurth  
Cephalalgia Volume 29 Issue 2, (February 2009) Pages 269-278

### ABSTRACT

We evaluated the association of body mass index (BMI) with migraine and migraine specifics in a cross-sectional study of 63 467 women aged  $\geq 45$  years, of whom 12 613 (19.9%) reported any history of migraine and 9195 had active migraine. Compared with women without migraine and a BMI  $< 23$  kg/m<sup>2</sup>, women with a BMI  $\geq 35$  kg/m<sup>2</sup> had adjusted odds ratios (ORs) (95% confidence intervals) of 1.03 (0.95, 1.12) for any history of migraine. Findings were similar for active migraineurs. Women with a BMI of  $\geq 35$  kg/m<sup>2</sup> had increased risk for low and high migraine frequency, with the highest estimate for women who reported daily migraine. Compared with women with the lowest associated risk (migraine frequency  $< 6$  times/year; BMI between 27.0 and 29.9 kg/m<sup>2</sup>), women with a BMI  $\geq 35$  kg/m<sup>2</sup> had an OR of daily migraine of 3.11 (1.12, 8.67). Among the women with active migraine, a BMI  $\geq 35$  kg/m<sup>2</sup> was associated with increased risk of phonophobia and photophobia and decreased risk of a unilateral pain characteristic and migraine aura. Our data confirm previous findings that the association between BMI with migraine is limited to migraine frequency and specific migraine features.

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NEUROLOGY

## ACE D/I polymorphism, migraine, and cardiovascular disease in women

M. Schürks, R.Y.L. Zee, J. E. Buring, and T. Kurth  
Neurology February 17 2009, Volume 72, Issue 7: Pages: 650-656.

Background: Interrelationships among the ACE deletion/insertion (D/I) polymorphism (rs1799752), migraine, and cardiovascular disease (CVD) are biologically plausible but remain controversial.

Methods: Association study among 25,000 white US women, participating in the Women's Health Study, with information on the ACE D/I polymorphism. Migraine and migraine aura status were self-reported. Incident CVD events were confirmed after medical record review. We used logistic regression to investigate the genotype-migraine association and proportional hazards models to evaluate the interrelationship among genotype, migraine, and incident CVD.

Results: At baseline, 4,577 (18.3%) women reported history of migraine; 39.5% of the 3,226 women with active migraine indicated aura. During 11.9 years of follow-up, 625 CVD events occurred. We did not find an association of the ACE D/I polymorphism with migraine or migraine aura status. There was a lack of association between the ACE D/I polymorphism and incident major CVD, ischemic stroke, and myocardial infarction. Migraine with aura doubled the risk for CVD, but only for carriers of the DD (multivariable-adjusted relative risk [RR] = 2.10; 95% CI = 1.22–3.59;  $p = 0.007$ ) and DI genotype (multivariable-adjusted RR = 2.31; 95% CI = 1.52–3.51;  $p < 0.0001$ ). The risk was not significant among carriers of the II genotype, a pattern we observed for myocardial infarction and ischemic stroke.

Conclusions: Data from this large cohort of women do not suggest an association of the ACE deletion/insertion (D/I) polymorphism with migraine, migraine aura status, or cardiovascular disease (CVD). The increased risk for CVD among migraineurs with aura was only apparent for carriers of the DD/DI genotype. Due to limited number of outcome events, however, future studies are warranted to further investigate this association.

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NEUROLOGÍA

## Pruebas complementarias y cefaleas

L.C. Lopes, F. Dach, J.G. Speciali

REV NEUROL 2009; 48: 183-187 - Fecha de publicación: 16/02/2009

**Introducción.** En vista de la elevada prevalencia de cefaleas entre la población general, es muy conveniente disponer de criterios bien definidos que orienten al médico a la hora de solicitar pruebas complementarias. **Objetivo.** Analizar las peticiones de pruebas complementarias durante el estudio de las cefaleas. **Pacientes y métodos.** Los datos se obtuvieron al revisar las historias clínicas de todos los pacientes que estaba previsto que fueran visitados en una consulta externa de cefaleas terciaria en 2004. **Resultados.** La prueba solicitada con más frecuencia fue la tomografía computarizada (TC) craneal, y las exploraciones que más contribuyeron a un cambio del diagnóstico clínico o la conducta médica fueron la TC de los senos paranasales, la radiografía simple de los senos paranasales y la resonancia magnética (RM) del cerebro. Las pruebas que no contribuyeron a un cambio del diagnóstico clínico ni de la conducta médica fueron la TC y la radiografía simple de la columna cervical. Tal como era de esperar, las exploraciones más caras para la institución fueron la TC y la RM. **Conclusión.** La importancia de las pruebas complementarias en el estudio de las cefaleas es indiscutible en muchos casos. Sin embargo, es necesario disponer de más estudios que evalúen la petición de pruebas complementarias para los pacientes con cefalea.

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 Ficha Técnica FORVEY

## DENOMINACIÓN DEL MEDICAMENTO

FORVEY 2,5 mg Comprimidos recubiertos con película

## COMPOSICIÓN CUALITATIVA Y CUANTITATIVA

Cada comprimido con cubierta pelicular contiene 2,5 mg de frovatriptán (en forma de succinato monohidratado)

Excipientes:

*Núcleo del comprimido*

Lactosa anhidra  
Celulosa microcristalina  
Sílice coloidal anhidra  
Carboximetilalmidón de sodio (Tipo A)  
Estearato de magnesio

*Cubierta pelicular*

Opadry blanco:  
Hipromelosa (E464)  
Dióxido de titanio (E171)  
Lactosa anhidra  
Macrogol 3000  
Triacetina

## FORMA FARMACÉUTICA

Comprimido con cubierta pelicular.

Comprimido redondo biconvexo con cubierta pelicular de color blanco, con una "m" grabada en una cara y "2.5" en la otra.

## DATOS CLÍNICOS

### Indicaciones terapéuticas

Tratamiento agudo de la fase de cefalea de los ataques de migraña con o sin aura.

### Posología y forma de administración

### Generalidades

Frovatriptán debe administrarse tan pronto como sea posible después del comienzo de un ataque de migraña, pero también es efectivo si se toma más tarde. Frovatriptán no debe usarse profilácticamente. Los comprimidos deben tragarse enteros y con agua.



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Si el paciente no responde a la primera dosis de frovatriptán, no debe tomar una segunda dosis para la misma crisis, dado que no se ha demostrado ningún beneficio.

Frovatriptán puede usarse en posteriores ataques de migraña.

## Adultos (entre 18 y 65 años de edad)

La dosis recomendada de frovatriptán es de 2,5 mg.

Si vuelve a parecer la migraña después de un alivio inicial, se puede administrar una segunda dosis, siempre que se deje un intervalo mínimo de 2 horas entre ambas dosis.

La dosis diaria total no debe ser superior a 5 mg al día.

## Niños y adolescentes (menores de 18 años)

No hay datos sobre el empleo del frovatriptán en niños y adolescentes. En consecuencia, no se recomienda su uso en este grupo de edad.

## Personas de edad avanzada (de más de 65 años)

Los datos de frovatriptán en pacientes de más de 65 años son limitados, por lo que no se recomienda su uso para esta categoría de pacientes.

## Insuficiencia renal

No se requiere un ajuste de dosis en pacientes con insuficiencia renal.

## Insuficiencia hepática

No se requiere un ajuste de dosis en pacientes con insuficiencia hepática leve o moderada. Frovatriptán está contraindicado en pacientes con insuficiencia hepática severa (véase Contraindicaciones).

## Contraindicaciones

Frovatriptán está contraindicado en los siguientes casos:

- hipersensibilidad a frovatriptán o a cualquiera de los excipientes.
- pacientes con historia de infarto de miocardio, cardiopatía isquémica, vasoespasmio coronario (p. ej. angina de Prinzmetal), enfermedad vascular periférica, pacientes que presentan síntomas o signos compatibles con cardiopatía isquémica.
- hipertensión severa o moderada, hipertensión leve no controlada.
- accidente cerebrovascular previo (AVC) o accidente isquémico transitorio (TIA).
- insuficiencia hepática severa (Child-Pugh C).
- está contraindicada la administración concomitante de frovatriptán con ergotamina o derivados de la ergotamina (incluida la metisergida) u otros agonistas del receptor 5-hidroxitriptamina (5-HT<sub>1</sub>).

## Advertencias y precauciones especiales de empleo

Frovatriptán sólo debe usarse cuando se haya establecido claramente el diagnóstico de migraña.

Frovatriptán no está indicado para el tratamiento de la migraña hemipléjica, basilar u oftalmopléjica.



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Al igual que en otros tratamientos de las crisis de migraña, es necesario excluir otras enfermedades neurológicas potencialmente graves antes de tratar la cefalea de pacientes sin diagnóstico previo de migraña, o pacientes migrañosos que presentan síntomas atípicos. Debe tenerse en cuenta que las personas que padecen de migraña presentan un mayor riesgo de determinados episodios cerebrovasculares (p. ej. AVC o TIA).

No se ha establecido la seguridad y eficacia del frovatriptán administrado durante la fase de aura, antes del inicio de la fase de cefalea de la migraña.

Al igual que otros agonistas del receptor 5-HT<sub>1</sub>, frovatriptán no debe administrarse a pacientes con riesgo de cardiopatía isquémica (CAD), incluidos los pacientes con hábito tabáquico o pacientes (ver sección Contraindicaciones). Debe prestarse especial atención a las mujeres posmenopáusicas y a los varones de más de 40 años con esos factores de riesgo.

Sin embargo, las exploraciones cardíacas pueden no identificar a todos los pacientes con afectación cardíaca. En muy raros casos, se han producido acontecimientos cardíacos graves en pacientes sin enfermedad cardiovascular subyacente cuando se han administrado agonistas de los receptores 5-HT<sub>1</sub>.

La administración de frovatriptán, puede asociarse a la aparición de síntomas pasajeros incluyendo dolor y opresión torácicos que pueden llegar a ser intensos e irradiarse hacia la garganta. (Véase Reacciones adversas).

Si se considera que estos síntomas pueden ser indicativos de cardiopatía isquémica, no deben tomarse nuevas dosis de frovatriptán y deberán realizarse exploraciones adicionales.

Se aconseja esperar 24 horas después del uso de frovatriptán antes de administrar un medicamento tipo ergotamina. Debe esperarse un intervalo mínimo de 24 horas después de la administración de un preparado conteniendo ergotamina, antes de administrar frovatriptán (véase Contraindicaciones y Interacción con otros medicamentos y otras formas de interacción).

En caso de uso demasiado frecuente (administración repetida durante varios días seguidos por un mal empleo del producto), el principio activo puede acumularse dando lugar a un incremento en las reacciones adversas. El uso prolongado de cualquier tipo de analgésico para cefaleas, puede empeorar el cuadro. Si ocurre esta situación o se sospecha, el tratamiento debería ser suspendido. Se debería considerar la posibilidad de cefalea por abuso de medicación en pacientes que padecen cefaleas con frecuencia o a diario a pesar de (o debido a) el uso regular de medicación para la cefalea.

No exceder la dosis recomendada de frovatriptán.

Este medicamento contiene lactosa, por tanto no deben tomarlo pacientes con problemas hereditarios raros de intolerancia a la galactosa, deficiencia de lactasa o malabsorción de glucosa-galactosa.

Las reacciones adversas pueden ser más frecuentes durante el uso concomitante de triptanes (agonistas 5HT) y las preparaciones que contienen hierba de San Juan (*Hypericum perforatum*).

### Interacción con otros medicamentos y otras formas de interacción

USO CONCOMITANTE CONTRAINDICADO:

**Ergotamina y derivados de la ergotamina (incluida la metisergida) y otros agonistas del 5 HT<sub>1</sub>**



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Riesgos de hipertensión y vasoespasmo de arterias coronarias debido a la adición de efectos vasoconstrictores cuando se usan de forma concomitante para la misma crisis de migraña (Véase Contraindicaciones).

Los efectos pueden ser aditivos. Se recomienda esperar al menos 24 horas después de la administración de medicación tipo ergotamina antes de administrar frovatriptán. A la inversa, se recomienda esperar 24 horas después de la administración de frovatriptán antes de administrar una medicación tipo ergotamina (Véase Advertencias y precauciones especiales de empleo).

## USO CONCOMITANTE NO RECOMENDADO:

Inhibidores de la monoaminoxidasa

Frovatriptán no es un sustrato de la MAO-A. No puede ser excluido un riesgo potencial de síndrome serotoninérgico o hipertensión.

## USO CONCOMITANTE CON PRECAUCIÓN:

**Inhibidores selectivos de la recaptación de serotonina** (citalopram, fluoxetina, fluvoxamina, paroxetina, sertralina)

Posible riesgo de hipertensión, vasoconstricción coronaria o síndrome serotoninérgico.

Un factor esencial para evitar este síndrome es respetar estrictamente la dosis recomendada.

### ***Metilergometrina***

Riesgos de hipertensión y vasoespasmo coronario.

### **Fluvoxamina**

La fluvoxamina es un potente inhibidor del citocromo CYP1A2 y se ha demostrado que incrementa los niveles en sangre de frovatriptán en un 27-49%.

### **Anticonceptivos orales**

En mujeres que toman anticonceptivos orales, la concentración de frovatriptán es un 30% superior a la de mujeres que no toman anticonceptivos orales. No se ha registrado una mayor incidencia en el perfil de reacciones adversas.

### **Hypericum perforatum (hierba de San Juan) (vía oral)**

Como sucede con otros triptanes, puede aumentar el riesgo de aparición de un síndrome serotoninérgico.

### **Embarazo y lactancia**

### **Embarazo**

La seguridad de frovatriptán en mujeres embarazadas no ha sido establecida.



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Estudios en animales han demostrado toxicidad en la reproducción. El riesgo potencial en humanos se desconoce. Frovatriptán no debe administrarse durante el embarazo a menos que ello sea claramente necesario.

## Lactancia

Frovatriptán y/o sus metabolitos se excretan en la leche de ratas lactantes, con una concentración máxima en leche cuatro veces superior a los niveles máximos en sangre. Aunque se desconoce si frovatriptán o sus metabolitos se excretan en la leche materna, no está recomendada la administración de frovatriptán a mujeres durante la lactancia, a menos que ello sea estrictamente necesario. En este caso, debe respetarse un intervalo de 24 horas.

## **Efectos sobre la capacidad para conducir y utilizar máquinas**

No se han realizado estudios para evaluar el efecto de frovatriptán sobre la capacidad para conducir y utilizar máquinas.

La migraña o el tratamiento con frovatriptán pueden producir somnolencia. Se debe recomendar a los pacientes que evalúen su habilidad para realizar tareas complejas tales como conducir durante las crisis de migraña y tras la administración de frovatriptán.

## **Reacciones adversas**

Frovatriptán ha sido administrado a más de 2700 pacientes a la dosis recomendada de 2,5 mg y los efectos secundarios más frecuentes (<10%) incluyeron mareo, fatiga, parestesias, cefalea y rubefacción vascular. Las reacciones adversas registradas en los estudios clínicos con frovatriptán han sido transitorias, generalmente entre leves y moderadas, y se han resuelto de forma espontánea. Algunos de los síntomas comunicados como reacciones adversas pueden ser síntomas asociados a la migraña.

La tabla siguiente muestra todas las reacciones adversas relacionadas con el tratamiento con 2,5 mg de frovatriptán, las cuales han presentado una mayor incidencia respecto a la registrada para el placebo en los 4 estudios clínicos controlados con placebo. Las reacciones adversas se muestran clasificadas por órganos y sistemas según un orden decreciente de incidencia.

ORGANO/SISTEMA	Frecuentes (1-10%)	Poco frecuentes (0,1-1%)	Raras (0,01-0,1%)
Sistema nervioso central y periférico	Mareo, parestesia, cefalea, somnolencia,	Tembor, hiperestesia, vértigo, contracciones involuntarias de la musculatura	Hipertonía, hipotonía, lentitud de reflejos, parálisis lingual
Trastornos del sistema gastrointestinal	disestesia, hipoestesia Náuseas, sequedad de boca, dispepsia, dolor abdominal	Diarrea, disfagia, flatulencia, estreñimiento	Queilitis, eructación, trastornos gastrointestinales no especificados, reflujo gastroesofágico, hipo, espasmo esofágico, úlcera péptica, dolor en glándulas salivares, estomatitis, dolor dental
Trastornos generales del organismo	Fatiga, distermia, dolor torácico	Dolor, astenia, fiebre	Dolor en extremidades inferiores



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Alteraciones psiquiátricas		Ansiedad, insomnio, confusión, nerviosismo, agitación, dificultad en la concentración, euforia, depresión, pensamiento anormal, despersonalización	Amnesia, agravamiento de la depresión, sueños anormales, trastornos de la personalidad
Vascular (extracardíaco)	Rubor		
Respiratorio	Opresión de garganta	Rinitis, faringitis, sinusitis, laringitis	Hiperventilación
Músculo-esquelético	Dolor esquelético	Dolor de espalda, artralgia, artrosis, debilidad muscular	
Trastornos en la visión	Visión anormal		
Piel y anejos	Aumento en la sudoración	Prurito	Urticaria
Frecuencia y ritmo cardíaco	Palpitaciones	Taquicardia	Bradicardia
Oído y desórdenes vestibulares		Tinnitus, otalgia, desórdenes óticos no especificados	Hyperacusia
Alteraciones sensoriales		Disgeusia	
Trastornos nutricionales y metabólicos		Sed, deshidratación	Hipocalcemia, hipoglucemia
Trastornos en el sistema urinario		Polaquiritia, poliuria	Nicturia, dolor renal, coluria
Trastornos cardiovasculares generales		Hipertensión	
Trastornos en plaquetas, el sangrado y la coagulación			Epistaxis, púrpura
Sistema nervioso autónomo			Síncope
Trastornos en el sistema hepático y biliar			Bilirrubinemia
Efectos secundarios			Daño provocado
Linfocitos y sistema reticuloendotelial			Linfadenopatías

Los efectos observados en dos estudios clínicos a largo plazo abiertos no han sido diferentes de los arriba expuestos.

## Sobredosis

Existe información limitada acerca de sobredosis por frovatriptán en comprimidos. La máxima dosis única por vía oral de frovatriptán administrada a pacientes varones y mujeres con migraña fue de 40 mg (16 veces la dosis clínica recomendada de 2,5 mg) y la máxima dosis única administrada a sujetos varones sanos fue de 100 mg (40 veces la dosis clínica recomendada). Ninguno de los casos fue asociado a otros efectos secundarios que los mencionados en la sección Reacciones adversas. Aún así, se ha descrito un caso grave de vasoespasmo coronario post-comercialización, tras tomar una dosis de frovatriptán cuatro veces superior a la recomendada 3 días consecutivos, en un paciente que tomaba tratamiento profiláctico para la migraña junto con antidepresivos tricíclicos. El paciente se recuperó.



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No existe antídoto específico para el frovatriptán. La semivida de eliminación del frovatriptán es aproximadamente 26 horas.

No se conocen los efectos de la hemodiálisis o diálisis peritoneal sobre las concentraciones séricas de frovatriptán.

## Tratamiento

En caso de sobredosis con frovatriptán, debe controlarse cuidadosamente al paciente durante 48 horas, como mínimo, y aplicarle una terapia de soporte en caso necesario.

## DATOS FARMACÉUTICOS

### Incompatibilidades

No procede

### Precauciones especiales de conservación

No conservar a temperatura superior a 30°C.

Blister: Conservar en el estuche original.

Frasco: Mantener el frasco bien cerrado.

### Instrucciones de uso, manipulación y eliminación

Ninguna especial.

## TITULAR DE LA AUTORIZACIÓN DE COMERCIALIZACIÓN

Menarini International Operations Luxembourg, S.A.

1, Avenue de la Gare

L-1611 Luxembourg

## FECHA DE LA PRIMERA AUTORIZACIÓN/RENOVACIÓN DE LA AUTORIZACIÓN

23 de septiembre de 2002

## FECHA DE LA REVISIÓN DEL TEXTO

Fecha de la primera autorización: 23 de septiembre de 2002

Fecha de la última renovación de la autorización: 16 de julio de 2007

## FECHA DE LA REVISIÓN DEL TEXTO

Julio 2007

## PRESENTACIONES Y PVP

Envase de 4 comprimidos con cubierta pelicular

FORVEY 2,5 mg: PVPiva 21,40 euros

## CONDICIONES DE DISPENSACIÓN

Con receta médica. Financiado por el Sistema Nacional de Salud con aportación normal.



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De conformidad con la Ley Orgánica de Protección de Datos de 15/1999 de 13 de diciembre, le comunicamos que los datos que Vd. nos facilitó en el momento de la suscripción al presente boletín forman parte de un fichero propiedad de **LABORATORIOS MENARINI, S.A.**, cuyo fin es el de mantenerle informado de las actividades que desarrolla nuestra compañía así como de nuestros productos.

Le informamos además que sus datos podrán ser cedidos a las empresas de nuestro grupo en España que realizan actividades relacionadas con la fabricación, promoción y comercialización de especialidades farmacéuticas y actividades relacionadas con la salud, con el fin de mantenerle informado sobre las actividades y productos de las mismas. Le informamos que Vd. puede en cualquier momento ejercer su derecho de acceso, rectificación, cancelación y oposición en relación con sus datos personales realizando un escrito a tal efecto y enviándolo a [info@menarini.es](mailto:info@menarini.es) o bien a la dirección postal de nuestra compañía (Att. "Menarini on-line" Laboratorios Menarini, S.A. C/Alfons XII, 587 08912 Badalona)

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