Exertion headaches

Cefaleas por esfuerzo

Abstract

Introduction: Headaches related to exertion include headaches precipitated by coughing or other Valsalva maneuvers, headaches brought on by prolonged physical exercise, and sexual headaches.

Objective: To analyze and actualize the clinical characteristics of headaches related to exertion.

Methods: We have revised publications, both in PubMed and in the main textbooks, on the different headaches brought on by physical exercise.

Results: Headaches related to exertion include mainly three entities: cough headaches, exercise headaches, and sexual headaches. These three varieties of exertional headaches can be primary or secondary. The treatment of primary varieties (indomethacin for cough headaches, beta-blockers for exercise and sexual headaches) and etiologies for secondary varieties (mainly Chiari type I malformation for cough headaches, vascular malformations for exercise and sexual headaches) are different for cough versus exercise and sexual headaches.

Conclusions: In contrast to classical works, cough headaches are clearly a separate entity from headaches related to prolonged exercise and, in general, sexual headaches, and these two latter conditions share many aspects.

Keywords
Cough headache, exercise headache, sexual headache.
Resumen

Introducción: La cefaleas en relación con esfuerzo físico incluyen la cefalea desencadenada por la tos u otras maniobras de Valsalva, la cefalea desencadenada por ejercicio físico prolongado y la cefalea sexual.

Objetivo: Analizar y actualizar las características clínicas de las cefaleas en relación con esfuerzo físico.

Métodos: Revisión de la publicaciones, tanto en PubMed como en los principales libros de texto, referentes a las diferentes cefaleas desencadenadas por esfuerzo físico.

Resultados: Las cefaleas en relación con el ejercicio o cefaleas de esfuerzo comprenden fundamentalmente tres grandes entidades, la cefalea tusígena, la cefalea de esfuerzo prolongado (que en la nueva versión se llamará cefalea por ejercicio) y la cefalea orgásmica o sexual. Las tres variedades pueden a su vez ser primarias o sintomáticas. El tratamiento de las variantes primarias (indometacina para la cefalea tusígena y beta-bloqueantes para cefaleas por ejercicio y sexual) y las etiologías para las variantes secundarias (fundamentalmente malformación de Chiari tipo I para la cefalea tusígena y malformaciones vasculares para cefaleas por ejercicio y sexual) difieren para cefalea tusígena y cefaleas por ejercicio y sexual.

Conclusiones: En contra de la concepción de los trabajos clásicos, la cefalea tusígena es una entidad claramente separada clínicamente de la cefalea de esfuerzo prolongado y, en general, de la cefalea sexual, condiciones que comparten a su vez muchos aspectos comunes.

Palabras clave
Cefalea tusígena, cefalea de esfuerzo, cefalea sexual.

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Introduction

Headaches induced by coughing, prolonged physical exercise, and sexual activity may be secondary to an intracranial structural injury or an annoying but unimportant picture. Until relatively recently, these headaches have been poorly cataloged and defined in the literature. In 1932, Tinel was the first to publish about several patients with intermittent, paroxysmal headaches related to exercise or maneuvers that increased intrathoracic pressure. Subsequently, Sir Charles Symonds published about 27 patients with short-term headaches caused by Valsalva maneuvers: coughs, laughter, defecation, or abrupt postural changes. This author subdivided these patients into two groups. The first group consisted of six cases in which he could demonstrate intracranial structural damage. The second group contained the remaining 21 patients, in whom he could not find a space-occupying intracranial lesion. Symonds was, therefore, the first author to consider cough headaches “benign,” that is, not secondary to an intracranial lesion. Despite this, the first large series of exertion-triggered headaches published by Rooke in 1968 still encompassed all headaches produced by coughing, prolonged physical exercise, and sexual arousal within the umbrella of “exertion headaches.” Rooke’s influence remained until the 1990s. In 1991, Sands et al. grouped together 219 cases of headaches caused by coughing and physical exercise. In this study, they found one in five cases was secondary to intracranial structural pathology—primarily posterior fossa lesions.

Stemming from the initial descriptions, the “benign” sexual headache became a well-defined entity. The first classification of the International Headache Society has already described three types of headaches related to sexual activity. Type 1 mimics a tension headache, and type 3 is actually a headache of hypotension due to leaking of cerebrospinal fluid, secondary to a burst of the dura, related to the exertion of intercourse. The most common sexual headache, also known as type 2 or vascular sexual headache, occurs abruptly during orgasm and is probably related to hemodynamic changes. The relationship between these three headaches is uncertain. In Lance’s experience, for instance, the relationship between sexual headache and headache from prolonged physical exercise is non-existent. In contrast, Silbert et al. found that 40% of patients with sexual headaches also experienced headaches from prolonged physical exercise.

Thus, in 1996 we published our retrospective experience with the 72 patients who had consulted in our Neurology Service due to triggered headaches. Irrespective of the different precipitants, we were able to clinically separate the three types of headaches. In addition, and even when sharing precipitants, we verified that the clinical characteristics of benign headaches for the three varieties were clearly different from those of secondary headaches, which made it possible to suspect an etiological diagnosis based on clinical parameters. Finally, our data coincided with that of Silbert’s findings, that headaches due to physical exercise and sexual headaches were conceptually similar entities. The emergence of our work made it possible for the International Headache Society in its new Classification of Headaches and Facial Neuralgias to clearly differentiate between a cough headache, a headache from prolonged exercise, and a headache attributed to sexual activity, and within the three consider the varieties as primary (idiopathic) or secondary (symptomatic) to structural intracranial injury. An important limiting factor of our work was that the data collection was retrospective and that many of the patients in the series had not been studied with modern magnetic resonance techniques, with all that the diagnostic elaboration in an entity such as a cough headache—in which detailed study of the posterior fossa is essential—entails.

Today we know, and this is reflected in the new headache version of the International Headache Society, that headaches in relation to exercise or exertion headaches basically comprise three major entities: cough headaches, prolonged effort headaches (in the new version will be called exercise headaches), and orgasmic or sexual headaches. All three can be either primary or symptomatic. Contrary to the conception of the classic works,
the cough headache is clearly separated from the headache of prolonged exercise and, in general, of the orgasmic headache, and the latter two share many common aspects. In this chapter, we will review the different varieties of exertion headaches, and provide recent data to clarify some of the statements we have just posed.\textsuperscript{11,12}

## Cough headaches

### Definition
A cough headache is considered to be induced, not aggravated, by coughing or other abrupt Valsalva maneuvers such as laughter, sneezing, defecation, sudden incorporation, weightlifting, etc.\textsuperscript{10}

### Epidemiology
Cough headaches are considered an infrequent entity. The published data put the prevalence of triggered headaches in between 0 and 2\% of the general population.\textsuperscript{13,14} They are, therefore, very uncommon headaches when compared to, for instance, migraines, whose prevalence in the general population is closer to 20\%.\textsuperscript{13} Our data confirm that these headaches are rare, accounting for about 1.5\% of headache consultations in a Neurology Service (93 patients out of a total of 6412 patients who had consulted for headache in 10 years).\textsuperscript{11} If we consider that headaches account for almost 25\% of neurological consultations in our country, then a simple calculation indicates that one out of 250-300 patients seeking a neurology consultation will do so due to a triggered headache. At least in terms of consultation, however, cough headaches are the most frequent, accounting for 75\% of triggered headaches.\textsuperscript{9,11} Indeed, in our series, 1\% of the patients who consulted for headache did so for cough headaches (64 patients with cough headaches out of the total 6,412 patients with headaches). Cough headaches may be benign/primary or secondary to intracranial structural pathology. Following the availability of modern neuroradiological techniques, fundamentally in this case MRI, it has been shown that at least half the cough headache cases are symptomatic of a posterior fossa lesion.\textsuperscript{9,11,15-18}

### Pathophysiology
The pathophysiology of a cough headache secondary to a Chiari malformation type I is simple to understand. The headache is secondary to the temporal impact of the cerebellar tonsils below the foramen magnum, which conditions a dissociation of craniospinal pressure evidenced during coughing or other Valsalva maneuvers.\textsuperscript{19-22} The fact that a decompressive suboccipital craniectomy cures cough headaches confirms this pathophysiological proposal.\textsuperscript{9} There is a significant relationship between cough headaches and the extent of tonsillar descent, and Cine MRI has shown this abnormal pulsation of the cerebellar tonsils specific to patients with Chiari type I and capable of interrupting the circulation of cerebrospinal fluid from the cranial cavity to the spinal subarachnoid space (Figure 1). The onset of cough headaches was correlated with the degree of collapse of the subarachnoid space.\textsuperscript{23,24}

The pathophysiology of primary cough headaches remains unknown and must be different from that of secondary cough headaches, as there is no involvement of the posterior fossa and there is a selective response to indomethacin.\textsuperscript{9,11,25} Other factors can be involved, such as a transient hypersensitivity reaction to a previous viral infection in the foramen magnum area receptors. Recently, a somewhat reduced size of posterior fossa structures in these patients has been described in relation to controls, which could contribute to the development of this headache.\textsuperscript{26} A novel finding, of undoubted practical interest, has been the detection of a large number of patients with cough headaches whose triggers have been ACE inhibitor drugs which are capable of inducing cough as a side effect.\textsuperscript{11,17} These drugs undoubtedly revealed cough headaches in predisposed patients but were not causal factors. In any case, it is important to take this information into consideration when questioning and managing these patients, since it is usually sufficient to withdraw the cough-inducing drug in these cases.

### Diagnosis and differential diagnosis
The new diagnostic criteria for benign or primary
cough headaches are listed in Table 1. 40% of patients with cough headaches have primary cough headaches, i.e. without underlying structural pathology. The picture of a primary cough headache is very characteristic, which allows its differentiation from secondary cases from a clinical point of view on many occasions.

Primary cough headache is a typical entity of elderly men. The mean age at onset is 60 years and approximately 60-80% of the patients who consult about it are men, though in the most recent data the male predominance is no longer so clear. Primary cough headaches are an episodic disorder with a duration that usually ranges from two months to two years (the mean duration in our series was 11 months). The pain begins immediately after a coughing fit or any other Valsalva maneuver (and not of prolonged exercise) and is moderate-intense. As for its location, pain is predominantly hemicranial in half of the cases, bilateral in 40%, and occipital-suboccipital in the remaining 10%. The quality of the pain is variable. In our series, 22% of the cases described it as stabbing, 17% as explosive, 17% as oppressive, and 44% as a mixture of all of them. The typical duration of the pain is mere seconds for more than 75% of the cases, while one in 10 patients said it lasted over a minute. As for the precipitants, in our experience, 100% of the patients reported their headache was triggered by coughing, 56% by abrupt postural changes, 39% by weightlifting, 33% by laughter, and 22% by defecation. In most patients, and this is a relevant aspect, there are no other accompanying symptoms. About 10% report a non-specific feeling of dizziness. There are cases of headache in patients taking Captopril or derivatives, drugs whose side effects include coughing. The intake of these drugs is, therefore, a question that we have to ask every patient with cough headaches.

Thanks to magnetic resonance techniques we know that approximately 50-60% of the patients who consult for cough headaches have a demonstrable underlying intracranial structural pathology. These data clearly differ from previous series. Only 10% of Rooke’s patients and 22% of Symonds’ had intracranial structural lesions. In our first series, in which not all patients could be studied with resonance, the proportion of symptomatic cases was lower, demonstrating the importance of the MRI study in the cataloging of these patients and in their differential diagnosis. As for the etiology, more than 80% of cases of symptomatic
Exertion headaches

Cough headache have a type I Chiari malformation, while the rest have other space-occupying lesions in the posterior fossa and, more specifically, in the foramen magnum region.\textsuperscript{9,11,27} The mean age of onset of symptomatic cough headaches is 44 years (range 21-60). The predominant localization of pain in the cases in our series is 72% occipital-suboccipital, 14% hemicranial, and 14% bilateral. The quality of the pain is very variable: oppressive in 31% of cases, explosive in 21%, stabbing in 21%, and a mixture of all of them for 24%. The duration of the pain lasted mere seconds for fewer than 50% of the cases. In our series, the duration of the pain episode at the time of diagnosis averaged 5 years (maximum 30 years). As for the precipitants, 72% had it triggered by coughing fits, 41% by abrupt postural movements, 33% by laughter, and 17% by defecation. More than 80% of the patients have subjective or objective semiologic findings, in this order: dizziness, instability, paresthesia in the face or upper limbs, vertigo, syncope, and miscellaneous.\textsuperscript{9,11,12,15-18,27}

Clinical data allow in most cases to differentiate between primary and secondary headaches. In fact, unlike the primary variety, secondary cough headaches are predominantly posterior, have a more irregular and prolonged duration (more than 50% of patients have episodes lasting at least one minute) and are not precipitated by coughing but by other Valsalva maneuvers in almost a third of the cases. Secondary cough headaches begin an average of 16 years earlier than primary headaches, so we have to suspect a primary cough headache if it’s an elderly patient and the secondary variant if we are treating a young patient. Gender does not seem to be clearly clinically useful in the etiological diagnosis. There are other clinical data that help us to differentiate between a primary cough headache and a secondary cough headache due to posterior fossa lesions. Patients with secondary headaches report a long history of headache (the mean is five years), compared to primary cases (mean is 11 months). In addition, while patients with cough headaches did not present other accompanying symptoms/signs, 85% of cases of secondary cough headache had semiology of posterior fossa. Another diagnostic possibility that we have to consider with a cough headache patient is a reversible tonsillar descent secondary to a cerebrospinal fluid hypopressive syndrome, spontaneous or secondary to dural puncture (Figure 2). These patients worsen with standing (“orthostatic headache”) and improve with bed rest.\textsuperscript{28}

### Treatment

Primary cough headaches respond well to indomethacin, administered prophylactically at doses of 25 to 150 mg daily with the corresponding gastric protection.\textsuperscript{9,11,24,29,30} The mechanism of action of indomethacin is unknown, although it is thought that it may decrease intracranial pressure, which may explain why some patients also improve with lumbar punctures\textsuperscript{31} or acetazolamide.\textsuperscript{32,33} For some patients, withdrawal of cough-inducing drugs may be sufficient to control the clinical picture.

Patients with symptomatic cough headaches do not respond to indomethacin and require specific surgical treatment. Indications for surgery would be incapacitating headache or the occurrence of posterior fossa semiology or the development of syringomyelia (Figure 3). In our experience, approximately one-third of patients with secondary cough headaches require surgical treatment. It has been consistently demonstrated that decompressive suboccipital craniectomy with a successful reconstruction of the posterior fossa eliminates cough headaches.\textsuperscript{9,11,27}

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**Table 1. ICHD-III diagnostic criteria for primary cough headache.\textsuperscript{10}**

| A. | At least two episodes that meet the B-D criteria |
| B. | Precipitated and occurring only in association with coughing or other Valsalva maneuvers |
| C. | Sudden onset |
| D. | Duration between 1 second and 2 hours |
| E. | Not attributable to another ICHD-III diagnosis |
Headaches due to prolonged exercise or exertion headaches

Definition
Exertion headache or exercise headache is a headache triggered by prolonged physical exercise, such as running, cycling, etc., but not in relation to sudden Valsalva maneuvers, such as coughing. Like cough headaches, exercise headaches may be benign/primary or symptomatic/secondary.\textsuperscript{8,9,11,34,35}

Epidemiology
The prevalence of headaches due to prolonged physical exercise is unknown, although it has been estimated at approximately 1% of the general population.\textsuperscript{12} In terms of medical consultation, headaches due to prolonged physical exercise are clearly less frequent than cough headaches, with approximately one patient consulting for this headache for every six patients consulting for cough headaches.\textsuperscript{8,11} As we will see later, most cases are primary and occur in middle-aged males.

Pathophysiology
The pathophysiology of primary exercise headache is speculative. It has been linked to the development of cerebral vasodilation. Patients with primary exercise headache have a higher frequency than the expected migraine and orgasmic headache, so it is logical to assume that they share pathophysiological

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Figure 2. A: Tonsillar descent in a patient with cough headaches and orthostatic headache after a lumbar puncture. B: Disappearance of the tonsillar descent after an epidural patch of autologous blood.

Figure 3. A: MRI in which typical tonsillar descending of Chiari type I malformation (arrow) is observed in a 34-year-old patient who consulted for cough headaches. B: MRI of the same patient, already asymptomatic, after reconstruction of the posterior fossa. Note the appearance of the cisterna magna after surgery.
mechanisms. The fact that patients with primary exercise headaches tend to suffer crises on hot days and after prolonged exercise suggests a vasodilation component in their pathophysiology, although we do not have objective evidence to confirm this point. The pathophysiology of the symptomatic exercise headache is specific to the causative process of the headache.\textsuperscript{17,18}

**Diagnosis and differential diagnosis**

The new diagnostic criteria for primary exercise headaches are listed in Table 2.\textsuperscript{10} Unlike primary cough headaches, primary stress headaches affect younger people, with a mean age at onset of 40 years (with a range of 10 to 48 years in our series). Primary exercise headaches are more common in males (90%) than in females. A good number of cases have a personal and family history of migraines. Heat, humidity, barometric changes, altitude, caffeine, hypoglycemia, and alcohol have been described as predisposing factors in these patients. This headache can be triggered by any type of prolonged physical exercise, although the exercise needs to double the basal heart rate for at least 10 seconds (and usually minutes or hours). The headache appears in the acme of exercise and usually disappears if physical activity ceases, although it may persist for one to four days (median duration four hours). It’s usually bilateral and accompanied by migraine characteristics such as pulsatile quality, nausea, vomiting, photophobia, and sonophobia.\textsuperscript{8,11,34-36}

Diagnosis of primary exertion headache requires thorough investigation, although, unlike cough headaches, less than 20% of patients will have the secondary variant. In typical patients (middle-aged/young men and normal examination) it is mandatory to exclude any type of intracranial space-occupying lesion and sentinel hemorrhage from a vascular malformation (Figure 4). Rarely, exercise headaches may be secondary to cranial arterial dissection or pheochromocytoma.\textsuperscript{37} In patients who report a “thunderclap” component, it is necessary to exclude reversible cerebral angiopathy syndrome.\textsuperscript{38} Today, therefore, MRI and MR angiography are indicated. In doubtful cases, lumbar puncture is necessary (Figure 5).

There is also a variant of exercise headache in relation to coronary ischemia (“cardiac cephalgia”). Their new diagnostic criteria are listed in Table 3.\textsuperscript{10} In those patients with exertion headaches and history of ischemic heart disease or atherosclerosis, it is necessary to request an electrocardiogram and determine cardiac enzymes. The diagnosis of this condition is not trivial since the anti-migraine drugs (ergot, triptan) are formally contraindicated in these patients.\textsuperscript{39-41}

Some of these patients with cardiac cephalgia also have episodes of headache in relation to angina at rest. There are three proposed pathophysiological explanations for cardiac cephalgia: 1) that it is a pain referred at a distance, similar to right shoulder pain in subphrenic lesions; 2) that it results from the release of algogenic peptides, such as the peptide in relation to the calcitonin gene or the vasoactive intestinal peptide, by the ischemic zone; and 3) that it is secondary to transient stasis due to momentary heart failure in relation to coronary ischemia. Be that as it may, we should suspect the diagnosis of cardiac cephalgia in those episodic headaches of recent onset in elderly people with a history of ischemic heart disease, and especially if the pain subsides after administration of nitrites.\textsuperscript{39-41}

**Table 2. ICHD-III diagnostic criteria for primary exercise headache.\textsuperscript{10}**

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<tr>
<td>A.</td>
<td>At least two episodes meeting criteria B and C</td>
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<td>B.</td>
<td>Precipitated by, and occurring only during or after intense physical exercise</td>
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<tr>
<td>C.</td>
<td>Duration less than 48 hours</td>
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<td>D.</td>
<td>Not attributable to another ICHD-III diagnosis</td>
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**Treatment**

For those patients with non-disabling primary exertion headache or with a low frequency of...
physical exercise, temporary abstinence or moderation may suffice. In these cases, treatment with NSAIDs or triptans immediately prior to physical exercise may also be tested. If necessary, beta-blockers used in migraine prevention such as propranolol or nadolol, for example, are usually effective, although they may reduce exercise tolerance (Figure 5).\(^{42}\) For those patients who do not tolerate beta-blockers or in whom they are contraindicated, indomethacin at a dose of 25 to 150 mg/day has been shown to be effective.\(^{43,44}\) There is no consensus regarding the duration of background treatment in these patients. Primary exertion headache is a transient condition that usually lasts less than three months and rarely more than six months; therefore, common sense indicates that preventive treatment should be withdrawn three to six months after initiation to see if it continues to be necessary.\(^{18}\)

Treatment of secondary exertion headache is specific to its underlying process.

**Sexual headaches**

**Definition**

Classically, three types of headaches have been described in relation to sexual activity. The first of the varieties is the sexual headache by muscle contraction and it would actually be a tension headache triggered by the sexual act. This headache is similar to tension headache. The second subtype or “postural” headache is actually a headache due to cerebrospinal fluid hypotension, which would be secondary to a small burst of the dura as a result of the effort in the sexual act.\(^{9}\) These two headaches do not occur in the acme of sexual activity, but as a consequence of it, and are clearly separated from the actual sexual headache, now also known as orgasmic headache. The true orgasmic headache occurs in relation to the orgasm and is the most common type of headache in relation to sexual activity.\(^{9,42-45}\)

**Pathophysiology**

The pathophysiology of primary orgasmic headache is superimposable to that of primary exertion headache. In fact, sexual activity is still a “prolonged” physical exercise, so everything said for exercise headaches is valid for orgasmic headaches. Many patients suffer exertion and orgasmic headaches together, whereas there is a minority of patients with orgasmic headaches and cough headaches. Undoubtedly, this is a consequence of the Valsalva that also takes place in the acme of the sexual act and explains that the orgasmic headache is a headache that we could determine as intermediate between cough headache and exercise headache.

Some authors have suggested that the sexual headache is due to poor brain self-regulation, as dysregulation of cerebral vasodilation has been demonstrated in these patients compared to patients with migraines or healthy subjects.\(^{46,47}\) Hypertension as such does not seem to play a definitive role since the percentage of hypertensive patients with sexual headaches does not exceed that expected for the general population, although it has been shown that these patients experience a
**Table 3. ICHD-III diagnostic criteria for cardiac cephalalgia.**

| A. Any headache that meets criterion C |
| B. Acute myocardial ischemia demonstrated |
| C. Evidence of causation demonstrated by at least two of the following: |
| 1. Headache has developed in temporal relation with the onset of acute myocardial ischemia |
| 2. One or both of the following: |
|   a. The headache has worsened significantly in parallel with the worsening of the myocardial ischemia |
|   b. The headache has improved or has been resolved in parallel with the resolution of the myocardial ischemia |
| 3. The headache has at least two of the following four characteristics: |
|   a. Moderate-severe intensity |
|   b. Accompanied by nausea |
|   c. Not accompanied by photophobia or sonophobia |
|   d. Aggravated by exertion |
| D. Not attributable to another ICHD-III diagnosis |
greater increase in blood pressure under conditions of physical stress.44

**Diagnosis and differential diagnosis**

The diagnostic criteria for orgasmic headaches are listed in Table 4. Similar to headaches of prolonged physical effort, primary orgasmic headaches are more frequent in males (80%). The mean age of onset is 40 years (limits 19-58 years) and coincides with the time of greater sexual activity of individuals. Type 2 or true orgasmic headache occurs at the time of, or just before, orgasm (either as a result of coitus or through masturbation). Its location is bilateral "at the temples" in three out of four patients and hemicranial in the remaining patient. The duration of sexual headaches ranges from one minute to six days (median, in our experience, is 10 minutes). It may be accompanied by migraine-like symptoms such as nausea or sonophobia/photophobia and is described as an intense and usually pulsatile pain. The mean duration of the symptomatic period is three months (minimum two days and maximum six years) and the number of headaches in the symptomatic period is very variable since it depends on sexual activity, generally ranging between a minimum of one and a maximum of 50.

**Treatment**

The management of true orgasmic headache is schematized in Figure 5 and is similar to that developed for prolonged exercise headache.9,11,41,44,47 There are no controlled studies in the management of this entity, so the recommendations are based on clinical series. It is important to explain to the patient the benignity of the process and its excellent prognosis. Since the risk of recurrence of this headache is high the days immediately following one of the attacks of pain, it seems reasonable to advise sexual abstinence if possible for several weeks. In the case of intercourse, it is advisable to play a role as passive as possible. Non-steroidal anti-inflammatory drugs (see symptomatic treatment of migraines) or triptans can be used immediately before the sexual act although we do not know their real effectiveness.

If changes in sexual habits are not sufficient or are not possible and the patient is symptomatic, preventive treatment is advisable. Short guidelines have been tested with non-steroidal anti-inflammatories, triptans, and even benzodiazepines, with poor results. The preventive treatment of choice for sexual headaches are the beta-blocking drugs we have already mentioned for headaches due to prolonged physical exercise, which are effective in
about 80% of the cases. For resistant cases or with contraindications to treatment with beta-blockers we can use indomethacin at doses of 50-150 mg/day. Given the usual duration of the symptomatic period, it is advisable to try to withdraw the preventive treatment after three months of initiation.

**Table 4. ICHD-III diagnostic criteria for primary sexual headache.**

| A. At least two episodes of head and/or neck pain that meet the B-D criteria |
| B. Precipitated by and occurring only after sexual activity |
| C. One or two of the following: |
| 1. Increased intensity by increasing sexual arousal |
| 2. Abrupt, explosive intensity just before or during orgasm |
| D. Not attributable to another ICHD-III diagnosis |

**Conclusions**

Faltan conclusiones

**Conflict of interest statement**
The authors declare there are no relevant conflicts of interest in this study.

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