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Nummular headache, trochleitis, supraorbital neuralgia, and other epicranial headaches and neuralgias: the epicranias

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Abstract Nummular headache is characterized by mild to moderate, pressure-like head pain exclusively in a small, rounded or oval area without underlying structural lesions. Either during symptomatic periods or interictally, the affected area shows a variable combination of hypoesthesia, dysesthesia, paresthesia, tenderness or discomfort. The particular topography and signs of sensory dysfunction suggest that nummular headache is an extracranial headache probably stemming from epicranial tissues such as the terminal branches of sensory nerves. Apart from nummular headache, other headaches and neuralgias such as idiopathic stabbing headache, trochleitis, supraorbital neuralgia, external compression headache, nasociliary neuralgia, occipital neuralgias, and auriculotemporal neuralgia have temporal or spatial features that suggest a peripheral (extracranial) origin, i.e. stemming from the bone, scalp, or pericranial nerves. Common to these disorders is a focal localization or a multidirectional sequence of paroxysms, paucity of

accompaniments, tenderness on the emergence or course of a pericranial nerve or on the tissues where pain originates, and possible presence of symptoms and signs (including effective treatment with locally injected anesthetics or corticosteroids) of nerve dysfunction. These observations led to the emergence of a conceptual model of head pain with an epicranial origin that we propose to group under the appellation of epicranias (headaches and pericranial neuralgias stemming from epicranial tissues). Nummular headache is the paradigm of epicranias. Epicranias essentially differ from other primary headaches with an intracranial origin and features of visceral pain, i.e. splanchnocranias that are characterized by a painful area wider than that of epicranias, no clear borders, presence of autonomic features, regional muscle tension, and driving of the process from the brain and brainstem.

Key words Nummular headache • Trochleitis • Supraorbital neuralgia • Epicranias • Splanchnocranias

Introduction

In the secondary headaches section of the 1988 classification of the International Headache Society (IHS), it was stated that

there are a few phenotypes a headache can express [1]. These are migraine, tension-type headache, cluster headache, increased and decreased intracranial pressure, local lesion type, vasodilator type and stabbing type. Theoretically, all such types of headache may have a primary and a symptomatic form.

Local lesion-type headache was described as continuous pain having a distinct maximum in a circumscribed area of 5 cm or less, but eventually spreading to the surroundings or referring to more distant areas. Pain from cranial bone metastasis was noted as the prototype of this symptomatic focal headache. A primary circumscribed headache has recently been described as nummular headache (NH) [2]. It is characterized by mild to moderate, pressure-like, rather continuous pain, exclusively felt in a rounded or elliptical area typically 2–6 cm in diameter, and neither attributed to local lesion nor systemic disorder [2].

Head pain is mostly conveyed by the trigeminal nerve, but the clinical manifestations may essentially differ depending on the level of trigeminal lesion or dysfunction. Intracranial activation of the trigeminovascular system is a final common pathway for many primary headaches [3]. However, the extracranial, sensitive branches of the trigeminal nerve could be at either the origin of the pain or the conveyance of local (extracranial) nociceptive head pain. In such cases, the painful area would be restricted to either the cutaneous territory of a dysfunctioning pericranial nerve or the involved tissues within a focal area. In addition, signs and symptoms of neuropathic pain, such as tenderness along the course of the nerve, hypoesthesia or dysesthesia should be found, indicating nerve dysfunction. Otherwise, there may also be focal discomfort or tenderness on the tissues of the affected area.

Herein, we review a number of headaches and neuralgias that we postulate share the common characteristics of being highly localized and supposedly stemming from extracranial structures. For the head pain with a presumed peripheral origin, we propose the general term epicranias which includes epicranial headaches and epicranial neuralgias.

Epicranial headaches

Nummular headache

This recently described headache [2] has an unusual, distinct feature: it is characterized by mild to moderate, pressure-like pain exclusively felt in a rounded or oval area typically 2–6 cm in diameter, without underlying structural lesions. The disorder tends to start from the fourth decade of life, and it prevails in women.

Although any region of the head may be affected, the parietal area, particularly its most convex part (tuber parietale) is the common localization of this circumscribed headache. The pain remains confined to the same symptomatic area without duplication or multiplication of the symptoms in other parts of the head. Moreover, the

painful area does not seem to change in either shape or size with time.

Either during symptomatic periods or interictally, the affected area may show a variable combination of hypoesthesia, dysesthesia, paresthesia, tenderness or discomfort. The pain is usually continuous with a chronic or remitting temporal pattern. Pseudoremissions may be observed when the pain reaches a very low grade or when only discomfort (not pain) in the affected area is reported. At times discomfort may prevail. Lancinating exacerbations lasting for several seconds or gradually increasing from 10 minutes to 2 hours may superimpose the baseline pain. Autonomic symptoms are typically absent.

Most patients worried that a serious underlying disease could be the cause of their focal cranial pain. Since the pain itself is, in most cases, not annoying, only reassurance is generally necessary.

Idiopathic stabbing headache

Idiopathic stabbing headache (ISH) is characterized by the extreme brevity of the paroxysms, generally lasting 1–2 seconds and rarely up to 10 seconds. The stabs can affect any area of the head, with erratic changes in the location of the pain between one paroxysm and the next, either in the same or the opposite hemicranium. Stabs may even synchronously occur on either side of the hemicranium. The paroxysms may appear always in the same region, usually the orbit [4–9].

The lack of topographic organization of ISH paroxysms makes their occurrence unpredictable both temporally and spatially. Such chaotic localization could be the expression of multiple presumed origins, most probably in the terminal sensory fibers of the pericranial nerves [9]. This is why we postulate that ISH may have a “peripheral” origin.

ISH is a benign entity, it does not associate with any structural lesion and it often coexists with other primary headaches, usually migraine, tension-type headache, and hemicrania continua. ISH either appears synchronously with the concurrent headache, or both headaches follow an independent course. Only in those cases in which the stabs are confined to the same area, it is highly recommended to exclude an underlying structural process.

The frequency is highly variable, going from one to several paroxysms a day. Rarely, it has been described a “stabbing headache status”. Usually the clinical course is either sporadic or recurrent, and only exceptionally can a chronic temporal pattern be recognized.

The paroxysms are of moderate intensity, but at the same time surprising and alarming to the patients, who

ask for a reasonable explanation. Therefore, the patients must be reassured and clearly informed of the benignity of the process. Only on rare occasions, when the paroxysms become extremely frequent, treatment with indomethacin is recommended. A dose of 75 mg/day has proved to be partially or totally effective in two-thirds of patients.

External compression headache

External compression headache results from continued stimulation of cutaneous nerves of the head by the application of pressure, for example by a band around the head, a tight hat, oxygen or anesthetic masks, or swimming goggles [1, 10]. It is an in crescendo, non-pulsating headache without accompaniments, with maximum pain in the points of more external compression. Involved nerves in such a type of headache seem to be mostly the supraorbital and the supratrochlear.

The headache is clearly dependent on the local compression and resolves once the external pressure has been removed. Interestingly, external compression may lead to a more severe migrainous headache if the stimulus is prolonged enough.

Trochleitis

Trochleitis is a local inflammatory process of the trochlea-oblique muscle complex [11, 12] with pain felt in the inner angle of the orbit, frequently extended to the ipsilateral forehead. In trochleitis, the pain is of moderate intensity, typically exacerbated upon situations that require vertical eye movements: reading, knitting, computer work, watching TV, etc. There are no oculofacial autonomic accompaniments. Pain in the trochlear region, exacerbation in supraduction, and hypersensitivity upon palpation of the trochlea strongly suggest the syndrome. Conversely, pain is relieved by injecting lidocaine or corticosteroids on the sore trochlea, but not by placebo injection.

In trochleitis, the typical induration and swelling of the inflamed trochlea is easily assessed by palpation. Eye movement restriction is an extremely rare feature of idiopathic trochleitis. In the eventual rare case of diplopia appearing, it will be of minimum intensity and intermittently appearing. This remark is important to distinguish idiopathic trochleitis from other trochlear syndromes that typically present with restriction and diplopia: Brown's syndrome (oblique superior syndrome) is a

congenital restrictive ophthalmopathy with shortening and fibrosis of the superior oblique muscle tendon. It causes strabismus but no pain. Acquired Brown's syndrome (symptomatic trochleitis) [13–17] is produced by local inflammatory processes, such as rheumatoid arthritis, hyperthyroidism, lupus, psoriasis or enteropathic arthropathy (inflammatory bowel disease: ulcerative colitis and Crohn's disease). Exceptionally, it can be caused by sinusitis, trauma or metastasis. In symptomatic trochleitis there is a severe restriction of vertical eye movements with diplopia. Blood tests (routine blood work, erythrocyte sedimentation rate, standard biochemical determinations, thyroid function, antinuclear antibodies, rheumatoid factor) and urine analysis are essential to exclude symptomatic trochleitis. Only exceptionally it is necessary to perform a biopsy. It is important to remark that when biopsies of cases of idiopathic trochleitis have been performed, the only findings were non-specific inflammation.

The efficient treatment of trochleitis consists of corticoid infiltration in the trochlear region. This procedure provides a rapid (24–48 hours) and substantial or absolute benefit. Only on rare occasions, reappearance of pain makes it necessary to repeat corticoid infiltration. The procedure is simple and safe. Exceptionally, traumatic hemorrhage may be a complication, but it generally disappears spontaneously.

Trochleitis may occur with migraine [12]. The striking association of migraine and trochlear pain seems to exceed any expected coexistence just by chance, bringing to mind the concept of a possible comorbidity. Interestingly, patients with concurrent migraine ipsilateral to the trochlear pain were substantially alleviated upon local trochlear treatment [12], suggesting that the trochlear painful process could have contributed to the worsening of the migrainous pain. Seemingly successful treatment of the trochlear pain may considerably decrease the nociceptive contingent to the caudalis trigeminal nuclei, thus relieving such neurons from the excessive input and decreasing their firing.

Epicranial neuralgias

The term "neuralgia" means pain in the territory supplied by a nerve or a nerve root [18]. The temporal pattern of a neuralgia does not have to be necessary abrupt, lancinating, and short-lived. In fact, central neuropathic pain may be brief and abrupt whereas peripheral neuropathic pain may show a continuous pattern. Precisely, pain arising from lesions or dysfunctions of pericranial nerves (Fig. 1) mostly attains a chronic-continuous pattern.

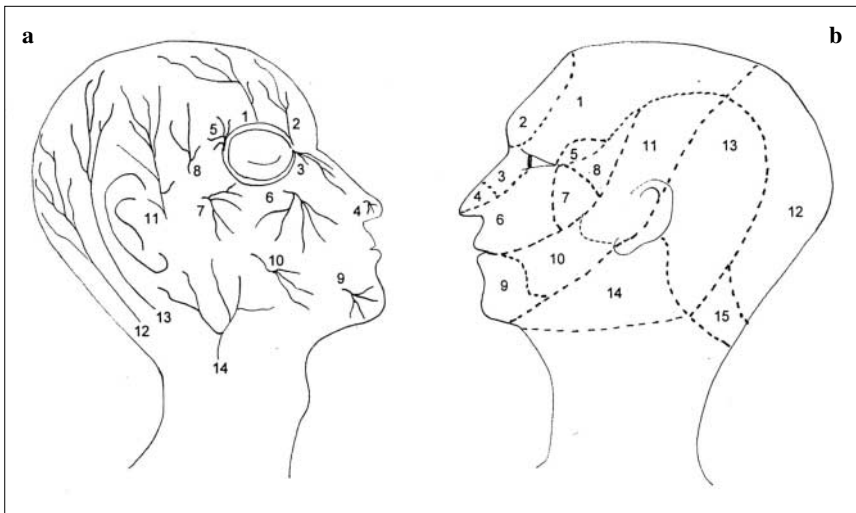


Fig. 1a,b Cranial and facial sensory nerves. **a** Emergence and course. **b** Territory. **Ophthalmic division of the trigeminal nerve (V-I):** 1, supraorbital nerve; 2, supratrochlear nerve; 3, infratrochlear nerve; 4, anterior ethmoidal nerve (external nasal branch); 5, lacrimal nerve. **Maxillary division of the trigeminal nerve (V-II):** 6, infraorbital nerve; 7, zygomaticofacialis nerve; 8, zygomaticotemporalis nerve. **Mandibular division of the trigeminal nerve (V-III):** 9, mentonian nerve; 10, buccalis nerve; 11, auriculotemporal nerve. **Second cervical root (C2):** 12, greater occipital nerve. **Second and third cervical roots (C2,C3):** 13, lesser occipital nerve; 14, greater auricular nerve; 15, third occipital nerve

Supraorbital neuralgia

Supraorbital neuralgia is well defined by the triad: pain in the territory (forehead) innervated by the supraorbital nerve (SON), tenderness on the emergence (supraorbital notch) or on the course of the nerve, and absolute relief upon anesthetic blockade of the nerve [19, 20].

The pain is usually chronic, continuous and, less frequently, remitting. Primary supraorbital neuralgia rarely associates typical features of neuropathic pain, such as hypoesthesia, paresthesia, allodynia or lightning exacerbations [20]. However, we have observed such signs in all patients suffering from post-traumatic supraorbital neuralgia. We postulate that traumatic supraorbital neuralgia produced by strong external forces (delivered suddenly once and for all) is clinically different from primary supraorbital neuralgia (probably caused by subtle chronic trauma to the orbit or supraorbital notch). In the orbit, the SON may run close to both the orbit wall and nearby arteries. This particular anatomic trajectory would permit a subtle intermittent nerve trauma during ocular movements, which would produce stretching, angulation, traction or friction of the SON. The nerve and a small artery can be close, and even share the adventitia [21]. Therefore, an adjacent artery might produce a microvascular compression of the nerve. Since the supraorbital artery accompanies the SON passing through the supraorbital notch, an impingement of the nerve by vascular engorgement should also be taken into account; this may explain the unexpected effect of triptans in relieving some cases of supraorbital neuralgia [19, 20]. Otherwise, SON may be subject to compression in the supraorbital notch by bony excrescences or tissue bands [19].

Treatment includes surgical exploration of the supraorbital notch and eventual liberation of the nerve from tissue

bands or bone excrescences. This procedure has been remarkably successful in the group of patients reported by Sjaastad et al. [19]. Identification of the causes of chronic compression inside the orbit is more complicated, although it should be looked for carefully, in order to clarify the mechanisms of such painful syndrome. This is part of an ongoing research effort in our institute.

Gabapentin proved to be of avail in some patients [20]. Capsaicin topically applied on the symptomatic forehead has also rendered substantial benefits (JA Pareja, unpublished observations).

Supratrochlear neuralgia

The term supraorbital neuralgia is preferable to neuralgia of the SON in view of the possible anatomic variations that make difficult determining the origin of the pain, in one (SON) or the other (supratrochlear nerve) nerves supplying the forehead [21]. Nevertheless, we have observed several patients with pain confined to the medial part of the forehead innervated by the supratrochlear nerve (STN). According to these observations, we believe that, at least in some patients, it may be possible to distinguish between SON and STN neuralgias. The possibility of trauma, or any other etiologic agent, affecting both nerves, has to be considered, since anatomic variations make both nerves equally susceptible to trauma.

Our experience is solely based on two patients - so with due reservations - we postulate that STN neuralgia is defined as the presence of pain in the medial region of the forehead, with absolute response to anesthetic blockade of the STN.

Nasal nerve neuralgia (Charlin's nasociliary neuralgia)

When Charlin described nasociliary neuralgia in 1931 [22], he did not know of cluster headache (described in 1939), chronic paroxysmal hemicrania (1974) or SUNCT (1989). Therefore, these circumstances were perfect to mix features of different primary orbital syndromes in his observations. This is probably the reason why the reported features of nasociliary neuralgia are so varied. The possible impurity of the original description could have made it easily forgettable, and in fact there have been few reports of nasociliary neuralgia after the impetuous emergence of cluster headache in 1939.

The nasal (or nasociliary) nerve is a terminal branch of the first division of the trigeminal nerve (V-1); it gives ciliary branches, and divides into infratrochlear nerve and ethmoidal (posterior and anterior) nerves. The infratrochlear nerve supplies the inner part of the eyelids, lacrimal ducts and surrounding skin, including the upper part of the nose. The posterior ethmoidal nerve supplies the sphenoidal and ethmoidal sinuses. The anterior ethmoidal nerve gives four nasal branches that supply the internal and external (inferior) parts of the nose.

According to Charlin [22, 23], nasociliary neuralgia is characterized by paroxysms of periocular pain with maximum intensity in the region between the inner angle of the orbit and the base of the nose, as well as in the nasal fossae. The pain may last from several minutes to hours. Charlin even admitted the existence of some moderate interictal pain. The attacks were accompanied by ipsilateral rhinorrhea or nasal congestion that Charlin believed were due to hyperemia and edema of the nasal mucosa. The paroxysms could also be accompanied by conjunctival injection, lacrimation, conjunctival hemorrhage, palpebral edema, and blepharospasm. Apparently, the symptoms appeared in the form of attacks during symptomatic periods, that lasted from 3 to 12 weeks [23, 24].

Charlin pointed out the presence of hypersensitivity in the emergence of the infratrochlear nerve and external nasal branch of the anterior ethmoidal nerve, as well as the characteristic quick alleviation of the pain (considered to be pathognomonic by Charlin) after intranasal instillation of a 5% cocaine and adrenaline solution.

Charlin attributed the symptoms to nasal nerve neuritis. He admitted the existence of variants or frustes forms [23], with lack of either the ocular components (ocular pain, conjunctival injection, lacrimation) or the nasal component (rhinorrhea). Although questioning some signs and symptoms, he postulated the invariable characteristics of nasal nerve neuralgia: hypersensitivity in the area of emergence of the nasal nerve, triggering of lancinating pain in the medial frontal region upon touching the lateral aspect of the symptomatic nostril, and the prodigious relief after endonasal instillation of a cocaine-adrenaline solution.

It is important to remark that Charlin considered several ophthalmologic lesions of great importance in this syndrome (minor cheratitis, small corneal ulcers, conjunctivitis, iritis, cyclitis, hypopyon). As he was an ophthalmologist, it is understandable that he was impressed by such ocular lesions. In any case, this syndrome was forgotten shortly after it was described, although we believe now it is the time for nasal nerve neuralgia to be revisited. Hopefully, a thorough study of these patients, excluding those with SUNCT, cluster headache, CPH, etc., will finally rehabilitate this interesting syndrome.

Occipital neuralgias

Occipital neuralgias should be located in the distribution of the greater and lesser occipital nerves, and third occipital nerve [25, 26]. Some features of occipital neuralgia may be of diagnostic help: hypoesthesia or dysesthesia in the affected area, and superimposed very short-lasting, severe paroxysms, although continuous pain may remain in between. The affected nerve may be tender to palpation. Anesthetic block of the nerves usually provides transient or relatively long-lasting relief.

Auriculotemporal neuralgia

This rare neuralgia seems to be more prevalent in middle-aged female patients. The symptoms consist in strictly unilateral attacks of lancinating pain mostly in the temple. The pain may also be felt in the mandibular joint, parotid region, and auricle but spreads upwards into the temple. The symptoms may be relieved by anesthetic block of the auriculotemporal nerve [27].

Conclusions

We propose the term "epicrania" to group all head pain syndromes apparently stemming from the epicranial tissues (Table 1). The objective considering epicranias is to heighten awareness on the role of the cranium and epicranium in generating headaches and neuralgias. As in any head pain, distinction between primary and symptomatic epicranias is mandatory, so local structural lesions involving any layer of the epicranium should be ruled out.

Generally speaking, superficial pain tends to be ultrashort, i.e. jabbing, or localized in a small area, whereas deeper pain is less likely localized and may be accompanied by autonomic features. Topographically, epicranias are characterized by either a small symptomatic area, even with sharp borders

Table 1 Epicranias

Epicranial headaches
Nummular headache
Idiopathic stabbing headache
External compression headache
Trochleitis
Epicranial neuralgias
Supraorbital neuralgia
Supratrochlear neuralgia
Nasal nerve neuralgia (Charlin's nasociliary neuralgia)
Greater occipital nerve neuralgia
Lesser occipital nerve neuralgia
Auriculotemporal neuralgia

(nummular headache) or according to a pericranial sensory nerve territory (epicranial neuralgias). A rather chaotic localization indicating a multifocal origin of the pain (ISH) also fits with the concept, as such a lack of topographic organization may also suggest a "peripheral" origin of the paroxysms, most likely in the cutaneous nerves of the scalp (Table 1).

Epicranial pain is usually chronic but fluctuating. Autonomic accompaniments such as nausea, vomiting, photophobia, audiophobia, lacrimation, conjunctival injection, rhinorrhea or nasal stuffiness are not typical of the epicranias. Conversely, there may be local symptoms and signs of sensory dysfunction in the symptomatic area. Many times it is possible to find tenderness on the symptomatic area or over the emergence or course of a sensory nerve. This sign is constant in the case of epicranial neuralgias. Interventions on the apparent origin of the pain by anesthetic blockades or corticosteroid injection usually provide complete relief, either transitory or relatively long-lasting.

Epicranias vs. splanchnocranias

It is widely acknowledged that primary headaches such as migraine are visceral (i.g. splanchnic). Splanchnocranias originate intracranially and are characterized by a painful

Table 2 Major categories of splanchnocranias

Splanchnocranial headaches
Migraine
Tension-type headache
Hemicrania continua
Cluster headache
Chronic paroxysmal hemicrania
SUNCT
Splanchnocranial neuralgias
Trigeminal neuralgia
Glossopharyngeal neuralgia
Intermediate nerve neuralgia (Hunt's neuralgia)

area wider than that of epicranias, with no clear borders. The pain is frequently accompanied by a variable combination of oculofacial or general autonomic symptoms and signs, or regional (pericranial and cervical) muscle tenderness. Apart from migraine, we may also consider tension-type headache, cluster headache, CPH, SUNCT, and hemicrania continua to be splanchnocranias (Table 2). Neuralgias with an intracranial origin, i.e. generated in the root or sensory ganglion of a cranial nerve, can also be considered to be splanchnocranial neuralgias and clinically can be differentiated from the extracranial (epicranial) neuralgias. For unknown reasons, "upstream" and "downstream" trigeminal system pain is clinically different. For instance, first division trigeminal neuralgia behaves clinically different from terminal branch supraorbital nerve neuralgia.

A good example of the usefulness of topographic differentiation of epicranias and splanchnocranias is the innervation of the nuchal area. For anatomical reasons nuchal pain with intracranial origin (e.g. migraine) is conveyed by branches of the ophthalmic nerve (V-1) whereas extracranially originated nuchal pain is conveyed by occipital nerves stemming from C2 roots.

Grouping primary headaches under epicranias (and splanchnocranias) is not intended to be an alternative classification but a proposal to take into account the possible anatomic source of the pain. This may provide us with interesting clues for research purposes.

References

1. – (1988) Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. Headache Classification Committee of the International Headache Society. *Cephalalgia* 8[Suppl 7]:1–96
2. Pareja JA, Caminero AB, Serra J, Barriga FJ, Dobato JL, Barón M, Vela L, Sánchez del Río M (2002) Numular headache: a coin-shaped cephalgia. *Neurology* 58:1678–1679
3. Moskowitz MA (1984) The neurobiology of vascular head pain. *Ann Neurol* 16:157–168
4. Lansche RK (1964) Ophthmodynia periodica. *Headache* 69:247–249
5. Sjaastad O, Egge K, Horven I, Kayed K, Lund-Roland L, Russel D, Slordahl Conradi I (1979) Chronic paroxysmal hemicrania. Mechanical precipitation of attacks. *Headache* 19:31–36

6. Sjaastad O, Apfelbaum R, Cokey W et al (1980) Chronic paroxysmal hemi-crania (CPH): the clinical manifestations. A review. *Ups J Med Sci* 31:27–35
7. Raskin NH, Schwartz RK (1980) Ice pick-like pain. *Neurology* 3:203–205
8. Pareja JA, Ruiz J, de Isla C, Al-Sabbah H, Espejo J (1996) Idiopathic stabbing headache (jabs and jolts syndrome). *Cephalalgia* 16:93–96
9. Pareja JA, Kuszewski P, Caminero AB (1999) SUNCT syndrome vs. idiopathic stabbing headache (jabs and jolts syndrome). *Cephalalgia* 19[Suppl 25]:46–48
10. Pestronk A, Pestronk S (1983) Goggle migraine. *N Engl J Med* 308:226
11. Tychsen L, Tse DT, Ossoinig K, Anderson RL (1984) Trochleitis with superior oblique myositis. *Ophthalmology* 91:1075–1079
12. Yangüela J, Pareja JA, Gili P, Sánchez del Río M (2002) Trochleitis and migraine headache. *Neurology* 58:802–805
13. Goldhammer Y, Lawton Smith J (1974) Acquired intermittent Brown's syndrome. *Neurology* 24:666–668
14. Olivares JP, Schiano A, Bardot A, Santini R (1988) Acquired Brown syndrome. An unusual complication of rheumatoid polyarthritis. *Rev Rhum Mal Osteoartic* 55:1035
15. Thorne JE, Volpe NJ, Liu GT (1999) Magnetic resonance imaging of acquired Brown syndrome in a patient with psoriasis. *Am J Ophthalmol* 127:233–235
16. Bradshaw DJ, Bray VJ, Enzenauer RW, Enzenauer RJ, Truwit CL, Damiano TR (1994) Acquired Brown syndrome associated with enteropathic arthropathy: a case report. *J Pediatr Ophthalmol Strabismus* 31:118–119
17. Hadjadj E, Conrath J, Ridings B, Denis D (1998) Brown syndrome: current status. *J Fr Ophthalmol* 21:276–282
18. Merskey H, Bogduk N (1994) Classification of chronic pain: description of chronic pain syndromes and definition of pain terms. IASP, Seattle
19. Sjaastad O, Stolt-Nielsen A, Pareja JA, Vincent M (1999) Supraorbital neuralgia. The clinical manifestations and a possible therapeutic approach. *Headache* 39:204–212
20. Caminero AB, Pareja JA (2001) Supraorbital neuralgia. A clinical study. *Cephalalgia* 21:216–223
21. Andersen NB, Bovim G, Sjaastad O (2001) The frontotemporal nerves of the head. Topographic variations of the supraorbital, supratrochlear, and auriculotemporal nerves. Possible clinical significance. *Surg Radiol Anat* 23:97–104
22. Charlin C (1931) Le syndrome du nerf nasal. *Ann Ocul* 168:86–102
23. Charlin C (1931) Le syndrome fruste du nerf nasal. *Ann Ocul* 168:808–815
24. Bruyn GW (1986) Charlin's neuralgia. In: Rose FC (ed) *Headache*. Elsevier, Amsterdam, pp 483–486 (*Handbook of clinical neurology*, vol. 4)
25. Bogduk N (1985) Greater occipital neuralgia. In: Long DM (ed) *Current therapy in neurological surgery*. BC Decker, Toronto Philadelphia, pp 175–180
26. Hammond SR, Danta A (1978) Occipital neuralgia. *Clin Exp Neurol* 15:258–270
27. Damarjian E (1970) Auriculo-temporal neuralgia – an original diagnostic and therapeutic approach. *R I Med J* 53:100–101