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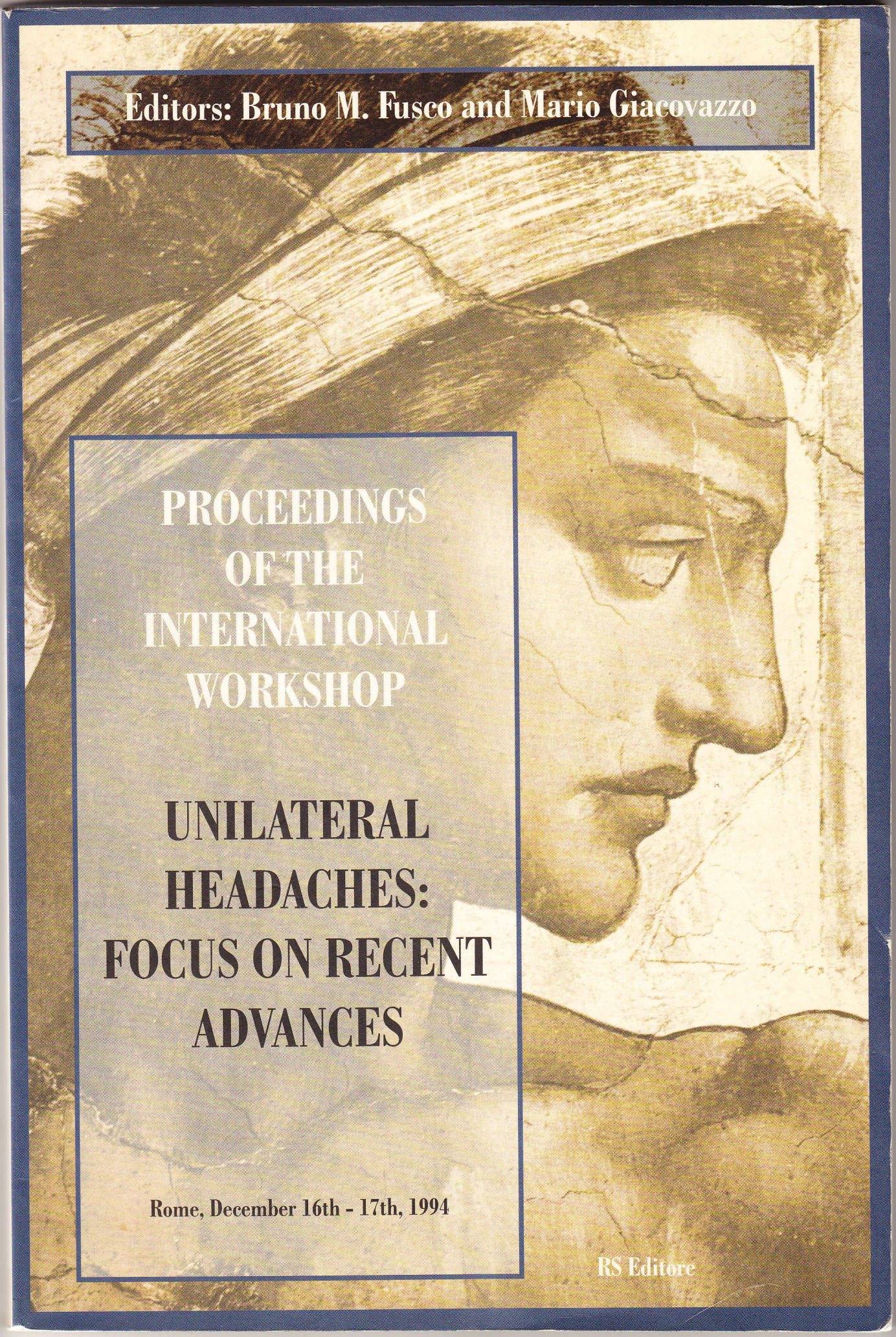

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PROPOSAL FOR A PATHOGENETIC CLASSIFICATION OF OPHTHALMOPLEGIC MIGRAINE

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Summary

The finding of postmigrainous defects of ocular motility, without neurogenic origin (to be more precise: involving the superior oblique muscle, due to jamming of the myofascial-tendinous complex at the level of the trochlea, with subsequent diplopia and in the presence of migrainous neurogenic oedema) questions the picture of Ophthalmoplegic Migraine offered by the International Headache Society. The writer examines here the pathogenesis of the motor defect in the districts of the III nerve, of the IV, of the VI and of the VII, pointing out the unreliability of the election of the cavernous sinus as the site of the pathogenetic mechanism of the damage. He furthermore suggests the epidemiological revaluation of the defect of the intrinsic motility in O.M..

Introduction

The International Headache Society (IHS) in its "Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain" (1988) proposes, with the "Ophthalmoplegic Migraine" (O.M.) definition, the following framing for the post-migrainous defects in the oculopalpebral motility: (1)

Ophthalmoplegic migraine

Description: Repeated attacks of headache associated with paresis of one or more ocular cranial nerves in the absence of demonstrable intracranial lesion.
Diagnostic criteria:
A. At least 2 attacks fulfilling B.
B. Headache overlapping with paresis of one or more of cranial nerves III, IV and I.
C. Parasellar lesion ruled out by appropriate investigations.
Comment: Whether ophthalmoplegic migraine in fact has anything to do with migraine is uncertain since the headache often lasts for a week or more. Association with other forms of migraine has often been noted, but a relationship to the Tolosa-Hunt syndrome has also been suggested. The condition is extremely rare.[1]

In the above classification, the IHS deals with the pathogenetic content, which can be shared or not, starting from the title itself.
"Ophthalmoplegic migraine": first of all it must be said that the IHS, in its exposition, decided to hold on to a strict objectiveness, thus refusing any previous thesis or terminolgy which was not supported by objective and consolidated data. The term "plegic", therefore, was used to refer to a neurogenic pathogenesis of the defects. Nobody would ever talk about plegia in the case of trigger finger or in a jaw blocked in dislocation.
On the other hand, there exist cases of postmigrainous defects of the ocular motility, actually involving the superior oblique or throclearis muscle, having a non neurogenic origin, because they concern

[1] Excerpt from "Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain" by the Headache Classification Committee of the International Headache Society in *An International Journal of Headache*, Volume 8, Supplement 7, 1988, Norwegian University Press, pag. 25.

jamming of the myofascial-tendinous complex at the level of the trochlea, with subsequent diplopia, in the presence of migrainous neurogenic oedema (Ezio Del Ponte, *Cefalea Primaria e Occhio*, Ed. Minerva Medica, Torino, 1993). The above mentioned case is not included in the term "plegic".
It must be pointed out that migraine can involve the pathogenetic premises necessary for the myogenic mechanism of the defect regarding the throclearis muscle: a sterile inflammation, with neurogenic vasodilation and oedema, which may also involve the myofascial complex; a relatively unextensible trochlea and the subsequent jamming in the tendon sliding.
Among the involved cranial nerves, the IHS lists the III, the IV and the VI. The VII and the correspondent defect of the eyelid orbicular muscle are not included. The defect of the eyelid muscles, if any, would be therefore limited to the levator palpebrae superioris, innervated by the III cranial nerve.
More than twenty years ago Boeri (2), in his well-documented treatise which included 13 cases of O.M. with III nerve paresis, reported 4 cases in which, right after the disappearance of the paresis of the III, an extension of the paresis, of peripheral type, to the VII nerve had appeared with subsequent defect of the eyelid orbicular. Neither this occurrence is contemplated by the IHS which leaves the VII nerve out of the list of involved nerves.
We will see how any relationship between O.M. and the Tolosa-Hunt syndrome, that would involve an election of the cavernous sinus as the site of the pathogenetic mechanism of O.M., is to be rejected. Some reservation should be made as well with regard to the final sentence which maintains the extreme rarity of O.M.. We shall see why.

Classification

The following is our proposal for a pathogenetic classification. (Tab 1)
A change in the title is, therefore, needed: instead of Ophthalmoplegic Migraine, "Postmigrainous defects of the oculopalpebral motility", without entering upon the subject of the neurogenic or myogenic origin of the defect. Then a distinction between neurogenic defects and myogenic defects.
Furthermore the inclusion of the possible defect in the province of the facial, following up Boeri's observations.
Finally, in the place of the cavernous sinus, a different site for the compression mechanism on the nerve, downstream interrupting the axonal flow and the nervous conduction and bringing about the plegic symptomatology.
It must be pointed out that the

Tab.1

PATHOGENETIC CLASSIFICATION OF THE SO-CALLED OPHTHALMOPLEGIC MIGRAINE RECTIUS

PATHOGENETIC CLASSIFICATION OF POSTMIGRAINOUS DEFECTS OF OCULOPALPEBRAL MOTILITY

NEUROGENIC DEFECTS

THE OCULOMOTOR - III CRANIAL NERVE - (ACCORDING TO SOME AUTHORS, IN 90% OF THE CASES OF O.M.). MUSCLES INVOLVED: SUPERIOR, MEDIAL AND INFERIOR RECTUS, LEVATOR PALPEBRAE SUPERIORIS, INFERIOR OBLIQUE (EXTRINSIC MUSCLES); CILIARY AND SPHINCTER PUPILLAE (INTRINSIC MUSCLES).
MECHANISM: INTERRUPTION OF AXONAL FLOW AND NERVOUS CONDUCTION DUE TO A COMPRESSION BY THE POSTERIOR CEREBRAL AND ANTERIOR CEREBELLAR ARTERIES ON THE NERVOUS ROOTS OR ON THE NERVE AT THEIR EMERGENCE FROM THE BRAINSTEM TO THE INTERPEDUNCULAR CISTERN, IN THE PRESENCE OF VASODILATION AND MIGRAINOUS OEDEMA.
THE ABDUCENS - VI CRANIAL NERVE - (RARELY INVOLVED). MUSCLE INVOLVED: RECTUS LATERALIS.
MECHANISM: INTERRUPTION OF AXONAL FLOW AND NERVOUS CONDUCTION DUE TO A COMPRESSION ON THE NERVE BY SOME OF THE BRANCHES OF THE BASILAR ARTERY AT THE EMERGENCE FROM THE BRAINSTEM INTO THE CEREBELLOPONTINE ANGLE CISTERN, IN THE PRESENCE OF VASODILATION AND MIGRAINOUS OEDEMA.
THE FACIAL - VII CRANIAL NERVE - MUSCLE INVOLVED: EYELID ORBICULAR.
MECHANISM: INTERRUPTION OF AXONAL FLOW AND NERVOUS CONDUCTION DUE TO A COMPRESSION ON THE NERVE FOLLOWING THE HYDRODYNAMIC ACTION OF THE MIGRAINOUS OEDEMA IN THE FACIAL CANAL, DOWNSTREAM THE GENICULATE GANGLION.

MYOGENIC DEFECTS

MUSCLE INVOLVED: SUPERIOR OBLIQUE OR TROCHLEARIS (RATHER FREQUENT DEFECT).
MECHANISM: JAMMING OF THE MYOFASCIAL COMPLEX IN THE TROCHLEA, IN THE PRESENCE OF MIGRAINOUS OEDEMA.

exclusion of the V cranial nerve and of the sympathetic nerve (in line with the IHS text) is meaningful, as we will see, in interpreting the pathogenetic mechanism.

Comment

The cavernous sinus

Why the exclusion of the cavernous sinus, or should we say the cavernous cavity[2], as the site of the damaging mechanism?
According to the numerous upholders of the cavernous thesis, the damage at that level should be provoked by a dilation of the internal carotid artery, which is congested during some migrainous phase: the compression of the artery walls on the nerves in the cavity and the formation of exudates should be responsible of the interruption on the nervous conduction. This is maintained by analogy to what happens in the organic forms of the cavity of aneurysmatic, arteritic and periarteritic kind (see Tolosa-Hunt syndrome) (3, 4), thromboflebitic, arteriovenous fistulae, invasive forms of the cavity, where ophthalmoplegia is part of the symptoms.
This thesis is not convincing for the following reasons:

1) muscle defects in the district served by the oculomotor nerve are by far the most frequent occurrences (observed in 90% of patients with O.M., according to De Bruy) (2), yet there are no anatomical relations between this nerve and the cavernous cavity or its contents such to justify this primacy. Indeed, unlike the other nerves, which run the whole length of the cavity, the oculomotor nerve enters the cavity passing through the superior wall of its middle third, and runs along the cavity only partly, from the rear forwards into the upper extremity, being encompassed by the two dural leaflets of the lateral wall and separated from the carotid by venous vessels of the sinus. All in all, therefore, it is little exposed to aggression by the carotid, compared with other nerves, the VI for example, which runs the whole cavity free from the bone for a long section, and just in contact with the wall of all the horizontal segments of the carotid, which it crosses in its first ascending segment: VI nerve paresis has an extremely low percentage incidence in O.M.;

2) trigeminal involvement, of irritative or deficient type, usually of the first branch (opthalmic) which runs the full length of the cavity, is virtually a constant feeling of cavernous sinus syndromes, both those of the posterior part, where the gasserian ganglion and the nerve's three divisions are located, and the middle and anterior parts, (according to the Jefferson-Bonnet classification) (5, 6). In O.M., on the other hand, symptoms related with mechanical involvement of the trigeminal are absent and they are not even contemplated by the IHS (this reasoning, of course, is unrelated to the suggestion that migraine attacks are of trigemino-vascular origin); ophthalmoplegia usually appears at the exit of the pain crisis, 8 to 18 hours after the disappearance of pain, and hence no longer in relation to any possible sign of trigeminal involvement, whereas these are contemporaneous with paralysis in organic forms. It must be noted that persistence of pain concomitant with ophthalmoplegia imposes a differential diagnosis between the migrainous crisis persisting or repeating and symptomatic headache. A significant factor in this respect is represented by signs of trigeminal defect, such as corneal anaesthesia and corneal arreflexia, not to mention neuroparalytic keratitis;

3) the sympathetic internal carotid plexus surrounds the artery with a thin network in contact with its wall. Well before any involvement of the oculomotor nerve, which is located on the periphery of the cavernous cavity, dilation and oedema of the carotid must necessarily have a much more incisive effect on this network: signs of sympathetic lesion are neither early nor constant in O.M.. This is particularly true with respect to Horner's syndrome. The aetiology, structural lesions and progression of Raeder's (7) paratrigeminal syndrome show that it belongs to a different clinical situation;

4) the venous sinus of the

[2] The definition of "cavernous sinus" should be reserved for the venous complex, one of the structures contained in the cavity. It has, however, long been extended to denote the cavity as a whole. This formulation can cause misunderstanding. The present treatise will, therefore, differentiate the two terms.

cavernous cavity is composed of a tangled mass of veins with "very thin and transparent" walls (Vassura) (8). A channel of communication is established through the superior orbital fessure between the orbit and the cavernous cavity; this channel enables the sinus to receive the outflow of the opthalmic vein (or of superior and inferior ophthalmic veins) carrying blood from most of the orbit structures, including the central vein of the retina (which sometimes empties into the sinus directly). Compression in the cavity leading to damage to the III, and even the other oculomotor nerves and the trigeminus, could well bring about collapse of the sinus veins and, therefore, upstream blood stagnation. Retinal, uveal, conjunctival and palpebral venous congestion, oedema of the optic disk and even exophthalmos, caused by stagnation and oedema within the orbit are, indeed, the usual features of syndromes affecting the middle and anterior parts of the cavernous cavity, i.e. those crossed by the oculomotor nerve (as we have seen, it is absent in the posterior part). If the compressing action on the oculomotor nerve were to exercise itself on the inside of the cavernous cavity by the internal carotid, the phenomena of venous congestion and upstream stasis would not be lacking.

Why at the emergence of the brainstem the site of the damage involving the III and VI cranial nerves?

Because this is the only point where compression on the nerve can bring about the plegic symptomatology of O.M. type, i.e. without causing orbital stasis, without involving also the trigeminal and the sympathetic nerves.

First of all we are going to examine the situation of

The oculomotor nerve

As a matter of fact, at the emergence of the brainstem the relations between the III cranial nerve and the vessels are such that the increase in the calibre of the posterior cerebral and of the anterior cerebellar arteries, congested by the migrainous effect, can cause a real strangling of the nervous roots or the nerve, which is enough to lead to damage. The contact with the posterior cerebral artery even in conditions of normality, is sometimes proved by the "vessel groove" on the cranial surface of the nerve, described by Fischer Brugge (9) in 1951.

Is any alternative possible?

The author reviews, tract after tract, the path and the relations of the III nerve both downstream the cavernous cavity and upstream, thus demonstrating that the hypothesis of a pathogenetic mechanism in the O.M. damage, alternative to the one here proposed, is unreliable. The author then dwells upon the functional anatomy of the III nerve nuclear complex and of the supranuclear afferences, axial and supra-axial. The same conclusions are reached.

The paralysis of the intrinsic component of the III deserves further attention as to different aspects, let alone the epidemiological one mentioned in the introduction. In the writer's experience, the statistical incidence of the intrinsic paralysis of the III within O.M. is largely undervalued.
In fact, the most frequent symptom of O.M. is not diplopia but anisocoria, associated with disturbances in the accommodation. Our stand is, therefore, in contrast with the common belief which looks for diplopia due to extrinsic defect or lid drop when assessing O.M..
A whole of semeiological conjunctures, both subjective and objective, renders the parasympathetic symptomatology (sphincter muscle of the iris and ciliary muscle) evasive during the diagnostic observation. Anisocoria, except in manifest cases, is not understood by the patient and often it is not even looked for by the general practitioner. When a physician finds it, he often has a puzzled reaction, other times he waits, and this is enough that at the following visit the symptomatology has attenuated or even solved itself, without the need to refer the patient to specialized structures. The waiting list to be seen by a specialist is often very long, even too long to grasp a temporary symptomatology. In the cases where the patient is seen by an oculist in time, the interpretation does not always address a migrainous pathogenesis of the symptoms. The fact that the intrinsic defect appears 8 to 18 hours after the migraine crisis is the reason why the two events are not thought to be connected and the patients are treated from the ophthalmologic point of view, without being

referred to IHS-oriented centres. But the fact remains that, according to our personal observations, the most adopted aetiopathogenetic interpretation when dealing with anisocoria having uncertain origin is the viral or vascular one, even if convincing immunological or electrophysiological supports are lacking.
We could say that in the long lasting debate between the followers of Charcot (19) (the father of O.M., 1890) and those of Moebius (20), who supported the thesis of the independence between ophthalmoplegia and migraine (he defined the first one as "periodical ocular paralyses"), the oculist still sides with Moebius.
As far as the paralysis of accommodation is concerned, we must take into account that visual fogging takes place only if the subject accommodates (i.e. when the ciliary muscle is stimulated). In the case of emmetropia, this happens when he looks at objects placed at a distance less than 6 metres (light-rays coming from a longer distance are considered parallel and accommodation does not take place). This leaves several activities out of the disorder. In the case of a myope, who accommodates very little or does not accommodate at all for short distances, the disorder could even be unnoticed. In the elderly, presbyopia causes a gradual reduction of the accommodation width, to the point that at sixty years of age it naturally becomes nothing, so much so that the accommodation disorder cannot be found. Then we have "dominance" which renders the accommodation disorder unnoticeable when it involves the not-dominant eye. Consider, for instance, how many youngsters suffering from amblyopia, with a visus reduced to 1-2/tenth in one eye, discover their disorder only when they enlist or when they are employed.
There are also other contingent situations, so to speak. For example, the ophthalmologic literature refers cases of paralysis of the III due to "pyrazolones" (21), which lead to think they are ophthalmoplegia caused in fact by migraine crises followed by taking of pyrazolone drugs.
The whole of the above-mentioned clinical and semeiological coincidences has certainly conditioned the inclusion of the cases of O.M. due to intrinsic paralysis in the oculistic case sheets (examined by the IHS), thus leaving them out of epidemiological data. In such a situation, we can deduce why, when talking about O.M., the IHS has defined it as "extremely rare". This is how the situation looks like, even though it is not the real one. Going back over the years, when the headache disease used not to be a current matter, we remember having examined some patients who suffered from temporary anisocoria which lasted for some days and used to resolve spontaneously during the observation period and before any deeper investigation could be carried out. How many of us used to connect these episodes with migraine crises happened some days before? Maybe the new cephalalgic age will bring about epidemiological discoveries in this field. This was confirmed by anamnestic surveys carried out with some colleagues.
The anatomical support fully justifies the higher involvement of the intrinsic component of the III to the compression damages by the posterior cerebral artery when this is turgid. On one hand we have the arrangement of the nervous fibres, on the other we have the relations between the vessel and the nerve.
At the emergence of the brainstem into the interpeduncular fossa, just above the pons, the fibres of the oculomotor nerve run, for a short tract, in separate fasciculi (or roots), which correspond to the various neuron assemblages that form the nuclear complex of the III, joined into two groups - 4/5 roots the internal group emerging from one of its own groove, 8/15 the external group -. A thin artery stemming from the posterior cerebral sometimes squeezes itself into the two groups (Whitnall) (22). After joining the extrinsic motility fibres into one trunk, the parasympathetic fibres run into the peripheral part of the nerve in craniomedial position in the first tract (23). The rather cranial position of the nucleus of Edinger-Westphall in the nuclear complex of the III and the craniomedial location of the correspondent parasympathetic fibres in the first tract of the nerve, make one think that also at the emergence of the brainstem such fibres run into the more cranial roots.
Therefore, both in the case that compression takes place on the roots, and in the case that this takes place on the nerve, the fibres of the internal motility will be the first ones to be involved in the calibre's increase of the posterior cerebral artery (as we have already pointed out, the

contact is testified by a "vasal groove"); at the same time, the cerebellar superior (or anterior) artery, turgid as well, prevents the nerve from moving towards the bottom so that the nerve is strangled. A rather small increase in calibre of the posterior cerebral will be therefore enough to functionally impair the parasympathetic fibres, while a stronger calibre's increase will cause a plegia of the whole nervous complex. Also the fibres going to the musculus levator palpebrae superioris have been acknowledged to be separate and located rather cranially: hence the possibility of an isolated defect of this muscle exists (24).

Bringing them to the clinical field, the presented considerations concerning the pathogenesis of ophthalmoplegia in the extrinsic and intrinsic components of the III, make it possible to point out some semeiological indications on O.M.:
a) higher probability of internal monolateral ophthalmoplegia (i.e. aniscoria and monolateral defect of accommodation) compared to external ophthalmoplegia (strabism-diplopia);
b) improbability of the opposite situation, i.e. external ophthalmoplegia without internal ophthalmoplegia (with the possible exception of an isolated defect of the levator palpebrae superioris);
c) progression of the symptomatology which can be foreseen according to the following pattern: 1st phase = aniscoria and monolateral defect of accommodation, 2nd phase = ptosis of the omolateral palpebra superior, 3rd phase = strabism-diplopia.

This is to sensibilize the physician in acknoweleging migrainous pathogenesis of aniscorias having uncertain nature, if any, in order to assess the exact epidemiological picture of opthalmoplegic migraine, whose frequency, in our opinion, is higher than what is assented in the IHS classification.

The vasomotor component in the pathogenesis of O.M. has been pointed out many times. It is now well ascertained that at a certain point in the migrainous development an increase in the calibre of the intracranial arteries takes place. To this picture well outlined in a century of investigations on the pathogenesis of migraine Olesen et al. (16, 17) gave the final touch when they demonstrated, using the SPECT method, how even in migraine with aura, characterized at the beginning of visual symptoms by regional oligaemia in the occipital cortex, there appears, some hours after resolution of aura, in the same areas, hyperaemia that does not regress with the end of pain and persists for hours.
The damage on the nerve is, in fact, related with the increase in the calibre of the neighbouring arteries, according to what has already been described and to what will be illustrated in detail later on.

The abducens (VI cranial nerve)

The pathogenesis of the postmigrainous defect of the lateral rectus muscle repeats by intervention of other actors, the mechanism taking place in the district of the III. In both cases we are dealing with vessel/nerve relations.
It is meaningful, for assessing the similarity between the pathogenetic mechanism of migrainous plegias of the III and that of the VI (according to the thesis maintained in the present work) the fact that a "vessel groove" was found also on the abducens, produced by the branches of the basilar artery on the nerve (Cushing) (25), similar to that engraved on the III by the posterior cerebral, described by Fischer Brugge (9).

The path of the VI nerve, the nucleus and the supranuclear afferences are examined, highligting the peculiar features that characterize the abducens, compared to the other nerves.

To sum up: as far as the district of the III nerve is concerned, and - even though with a lower frequency - of the VI (due to the exposed likely reasons), there is no defect in opthalmoplegic migraine which cannot be intrepreted through the compressive mechanism, caused by an increase of the calibre of the basilar artery followed by oedema and performed on the respective nerves right after the emergence from the brainstem, at the entry into the subarachnoidal parapontine cisterns. All this is mediated by individual anatomical characteristics, congenital or acquired, in the relations between nerve trunks and vascular network.

The trochlear (IV cranial nerve)

The IV nerve is in a particular situation. It is the only cranial

nerve emerging from the dorsal surface of the brainstem, just below the lamina quadrigemina, after complete decussation of its fibres (someone admits the existence of not cruciate fibres); it passes round the cerebral peduncle, reaches, it too, the interpeduncular subarachnoidal cistern where it penetrates along the upper edge of the middle cerebellar peduncle and directs itself to the cavernous cavity along the cranial base.
This nerve certainly comes into contact with satellite arteries: posteriorly the quadrigeminal artery, the superior cerebellar beside the peduncle and forwards, at its entry into the cistern. Yet due to its immersion in CSF for the whole of its length it will be shifted rather than compressed by any increase in vessel calibre: in a situation, therefore, well different with respect to the III which could be strangled in the vice by dilation of the posterior cerebral and superior cerebellar arteries. Mechanical trochlear nerve lesions at para-peduncular level are thus a virtual impossibility in migraine. The reservations as to the hypothesis of a selective mechanical lesion on the IV in the subarachnoidal tract and in the cavernous cavity are those expressed for the III nerve.
As far as the intra-axial tract of the roots of the IV is concerned, the absence of symptoms involving other neighbouring mesencephalic structures (the medial longitudinal fasciculus, the aqueduct, the mesencephalic nucleus of the trigeminus, the inferior quadrigeminal bodies etc.) proves, again, the untruthfulness of a damaging mechanism at this level.
With regard to the nucleus of the IV, a small roundish mass (1x1.5 mm), located in the periaqueductal grey substance just below or close to the lateral nucleus of the III, cases of isolated damage are almost inexistent. It follows the same path of the nuclear complex of the III. When the pathogenic involvement of the two nuclei is unilateral (this is not the most frequent occurrence), the different anatomical location of their fibres and the almost complete decussation of those of the throclear, give rise to a rather typical pattern: paralysis in the district of the III on one side of the head and paralysis of the superior oblique muscle on the other side: the pattern is very different, as it can be easily understood, from that of O.M.. These are, of course, organic forms.
As far as the supranuclear afferences to the nucleus of the IV are concerned, we can say that, mutatis mutandis, the same arguments pointed out for the oculomotor nerve are valid: an absolute paralysis (voluntary and automatic-reflected) of the IV, just like it occurs with O.M., would involve a polyfocal symptomatology.
The neurophysiological laws of ocular motility applicable for the III nerve and pertinent deductions obviously regard also the supra-axial pathways of the IV.
To sum up, it would be difficult to find in O.M., a tract of the nervous pathways which could be assigned as the site of the pathogenetic mechanism of the defect of the superior oblique muscle. For this reason and, most of all, for the clinical development of symptoms, the defect in the four cases we have observed were declared having myogenic origin.

The functional anatomy of the superior oblique muscle is described.

The trochlea, in fact, is the origin of the muscle in kinetic terms. Its prime function is to direct the anterior pole of the eyeball downwards and outwards: it is thus a depressor and abductor, as well as an incycloductor.
The action of the superior oblique is thus dependent on its functional efficiency and the ability of the tendon to slide back and forth in the trochlea. Jamming of this movement will hinder the action, resulting in limitation in the muscle's range of motion, both during the active phase when lowering and abducting the eyeball, and when the eye returns to its primary position. If the jamming at the throchlear level is total, this will prevent relaxation of the muscle, making the raising of the eye impossible (at least beyond the horizontal plane during adduction). The cinematics could then correspond to that of an apparent paralysis of the inferior oblique muscle, hence the description "pseudoparalysis of the inferior oblique muscle" found in the literature (Giardini) (26).
Hypotropia is possible and can lead to diplopia in the primary position (Miglior) (27).
Diagnosis is likely to be difficult in incomplete or intermittent cases, due to the intervention of instantaneous variations in the eye's motility, linked to jamming or to its sudden interruption, especially in relation to voluntary stimulations whose intensity is higher than the power of the friction.
An always present semeiological feature is pain at the correspondence of the

trochlea, which is exacerbated by pressure and sometimes spontaneous.
After some days of total block (5-10 days), reaching its climax during about 12 hours after the migraine crisis, the functional recovery begins. An almost pathognomonic symptom in this phase is the appearance of rather coarse, jerky movements of the eyeball within the range of the muscle's action, whereby the eye can reach the position desired by the patient and fusion is achieved. The defect then becomes intermittent and in some days time motility is back to normal, and diplopia disappears.
This behaviour rules out the neurogen defect and recalls a particular syndrome described by Brown (28) in the 1950's. Originally referred by him to congenital shortening of the tendon sheath of the superior oblique, it has since been described as a consequence of traumatic haematoma of the sheath itself, or of swellings of various kinds of the sheath or tendon causing friction in the trochlea (Maione-Mariani) (29). It is reminiscent of orthopaedic trigger finger (Lang) (30).
In our opinion, the oculomotor defect was the outcome of oedema imbibition of the muscle fascia and tendon at the trochlear level, within the vessel disorders and defective permeability associated, as we have seen, with a migraine crisis.

The following is, therefore, the pathognomonic triad of this syndrome:
1. jamming phenomena of the superior oblique muscle in the trochlea;
2. pain, sometimes spontaneous, but always present when pressing at the correspondence of the trochlea;
3. migraine crisis during the previous 6-18 hours.

The facial (VII cranial nerve)

According to Boeri (2), the postmigrainous paresis of the VII is peripheral. This means that nuclear and supranuclear hypotheses do not need to be taken into consideration.
The intracranial path and the relations of the VII nerve are described. The lack of signs of involvement of the VIII in the paresis of the VII and the lack of vegetative (lacrimal) participation show that the origin of the damage (compression on the nerve by the facial nerve artery and by neurogenic migrainous oedema) is peripheral and arises in the facial canal, downstream from the geniculate ganglion.

Conclusion

After more than a century since the interpretation suggested by Charcot, with his "Ophthalmoplegic Migraine" definition, the postmigrainous defects of the oculopalpebral motility, have not found a final location yet.
In this paper we have pointed out some new aspects, such as the postmigrainous myogenic defect of the superior oblique muscle, and underestimated aspects were revaluated, such as those regarding the defects of the intrinsic motility.
Furthermore, the possible participation of the VII nerve was mentioned, following a previous indication by another Author. The pathogenetic mechanism of the oculomotor defect in the districts of the III nerve, of the IV, of the VI and of the VII, was described, underlining the unreliability of the election of the cavernous sinus as the site of the pathogenetic mechanism of the damage.
In the author's opinion, these topics deserve to be examined.

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