Isolated third nerve palsies (P3N) can follow a variety of etiologies such as peripheral ischemia, inflammation, traumatism, cerebral vascular accidents and compression processes, including tumors and aneurisms. Of all these, the most urgent one for the patient’s life is the compression of the common ocular motor nerve caused by an aneurism of the posterior communicating artery (PCA). Along its path across the subarachnoid space, the third nerve runs very close to said artery which joins the flow of the posterior brain artery (a branch of the basilar artery) with that of the internal carotid artery. Precisely in the proximity of the union of the latter with the PCA is where most of the aneurisms causing a P3N become lodged (fig. 1). Pupil motor fibers, intended for the pupil sphincter, are located in the superior medial area of the common ocular motor, and are the most vulnerable fibers in nerve compressions.

Acute paralysis of the third nerve are almost always due to peripheral ischemia or PCA compression. In differential diagnostics, special relevance has been given to the size of the pupil. Accordingly, a P3N with normal pupil would indicate a microvascular disorder. P3N with partial pupil involvement and anisochoria above 0.5 mm are frequently produced by compression lesions other than aneurisms, at the brain level, of the cavernous sinus or of the posterior fossa (1,2). Likewise, if the pupil is not dilated in a case of total paralysis of the extrinsic muscles depending from the common ocular motor, we can consider pupil preservation. However, this is not so certain in partial extrinsic motor involvement. P3N consecutive to PCA are usually accompanied by ipsilateral orbital or frontal pain due to the involvement of the trigeminal fibers of the third nerve. Even so, the P3N caused by...
ischemia can also involve pain, whereas the absence of pain does not exclude aneurism. Therefore the importance of pain is relative when considering either etiology. As regards the age of the patient, aneurisms are rare in children under 10 while in advanced ages there are associated vascular risk factors (diabetes, high arterial pressure, dyslipemia, smoking).

From the neurological and radiological viewpoint, arteriography with catheter (AG) is the «Gold Standard» for detecting intracranial aneurisms (fig. 2), although it is associated to a 1-2% risk of permanent complications such as brain vascular accidents. For this reason, catheter arteriography is being gradually replaced by non-invasive neuroimaging techniques such as nuclear magnetic resonance angiography (MRA) (3) and computerized tomography angiography (CTA) (4,5), which have a lower prevalence of events. MRA does not employ ionizing radiation or requires the use of contrast and therefore it can be performed easily in patients with severe atheromatosis or kidney insufficiency. In turn, CTA is a quick and inexpensive procedure involving the IV injection of contrast without having to use the femoral artery for this, in contrast with GA. In addition, Nuclear Magnetic Resonance (NMR) shows thrombosed aneurisms which sometime go undetected even with GA. The size of a PCA is very important because the smaller ones (under 3-4 mm) rarely produce clinic symptoms, but those that are smaller than 4-5 mm are difficult to detect with non-invasive neuroimaging techniques. Therefore, the critical size of an aneurism causing a P3N is around 4 mm (1,3). Within this tight range PCA can exhibits symptoms, with good or poor display with RMA and CTA. Comparing both techniques, studies have shown a greater sensitivity of CTA than RMA for detecting aneurisms (4) (fig. 3).

In daily clinical practice, diagnostic problems should be resolved in the short term because any delay in treatment could involve serious and vital consequences for the patient. Before the appearance of non-invasive neuro-radiological techniques, an AG was prescribed for P3N with partial pupillary involvement or extrinsic paralysis without pupil involvement which refused to improve. In recent years, protocols have varied considerably, keeping AG for selected cases. In our intervention algorithm against acute isolated P3N (table I), we differentiate patients in age groups so those under 10 are prescribed only a NMR. As of this age, in the case of P3N with complete midriasis with also perform a CTA and if the result is normal an AG, taking into account the risk of PCA. For patients above 75 we only perform an AG when we consider endovascular treatment of the aneurism. When midriasis is not complete the extrinsic paresia is not complete either, we perform a NMR and a CTA due to the remote possibility of PCA. In turn, in a P3N with partial pupil and total extrinsic involvement, we only perform NMR which allows us to discard possible tumoral causes. Finally, if the full extrinsic paralysis includes a normal pupil, we only indicate said test in patients under age 50 and propose sim-
In any case, in a P3N with partial or null pupil involvement, the patient must always be observed closely for 10 days to discard the development of complete midriasis. Furthermore, in the cases in which a conservative approach is adopted due to a suspected microvascular ischemic involvement, it will be confirmed only if ocular motility returns to normal within 4 months.

REFERENCES