anterior ischemic optic neuropathy, disc oedema is pale non-hyperaemic and accompanied by a decrease in visual acuity in the form of stroke.

Other entities to be excluded are infiltrative processes (leukaemia, lymphoma) where pupil impairment is predominant. Compressive optic neuropathy, which may be caused by a meningioma of the optic nerve sheath, has the optociliary shunt vessel as pathognomonic sign. Papillitis is usually unilateral, with decreased visual acuity and pupillary alterations and is usually associated with pain upon ocular movements.[2,7,8] Foster Kennedy syndrome, secondary to an olfactory groove meningioma, occurs with papillary oedema in one eye and optic atrophy in the other.[2,9]

In relation to table 1, which sets out the ophthalmoscopic differences, we propose adding that the papilla is not hyperaemic in pseudopapilloedema—unlike in papilloedema. In recent years, we have worked with optical coherence tomography (OCT), and although the authors believe that this study "has not proved effective in differentiating an incipient papilloedema from a pseudopapilloedema, since in both cases there is an increased thickness of the nerve fibre layer of the retina",[1] we believe along with other researchers[3,10] that, although these measurements in a first consultation have not been useful in establishing differences between both entities, the evolutionary repetitions of the protocol used indeed manage to observe differences.

Finally, we celebrate the quality of the photographs that illustrate the text and we reiterate our gratitude to the authors; such reviews, which clarify knowledge about controversial issues, are very necessary for the proper development of neuroophthalmology and neuroscience in general.

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Reply to: Papillary oedema: True or false?

Respuesta a: Papiledema: ¿verdadero o falso?

Dear Editor:

We thank Drs Fuentes-Pelier and Hodellín-Tallada for their interest in the review "Papilloedema: true or false?". We want to clarify that the fundamental purpose of our work[1] is to describe the diagnostic approach for a clinical suspicion of papilloedema and the role of new technologies in this context. That is why we consider it appropriate to present two distinct clinical scenarios like those appearing under the headings "Papilloedema versus pseudopapilloedema" and "Oedema versus papilloedema".

In the first section, we discuss the changes of the optic disc that may pose reasonable diagnostic doubt, especially at the stage of incipient papilloedema, such as buried drusen, full disc in hyperopia, nasal elevation of the myopic disc and myelin fibre presence. Papillary tumours[2] such as astrocytoma and melanocytoma present differential characteristics (very dark pigmentation in the optic disc that partially or completely obscures the papillary margins, or a round lesion that may indicate a blackberry-like spot superimposed on the disc with intralesional calcifications, respectively) which, according to our experience, we do not need to include in the differential diagnosis of pseudopapilloedema (figs. 1 and 2). Furthermore, we do not think it appropriate to consider abnormalities in disc development[3] (papillary coloboma, morning glory anomaly and peripapillary staphyloma) in the differential diagnosis for the same reason.

We agree that papillary oedema can be produced by multiple causes and that the differential diagnosis should be carried out with optic neuropathies that eventually appear with papillary oedema at some point in their evolution (ischemic, infectious, infiltrative, tumour or compressive). Therefore, we propose the second clinical scenario oedema versus papilloedema; however, the appearance of papillary oedema in itself may be non-

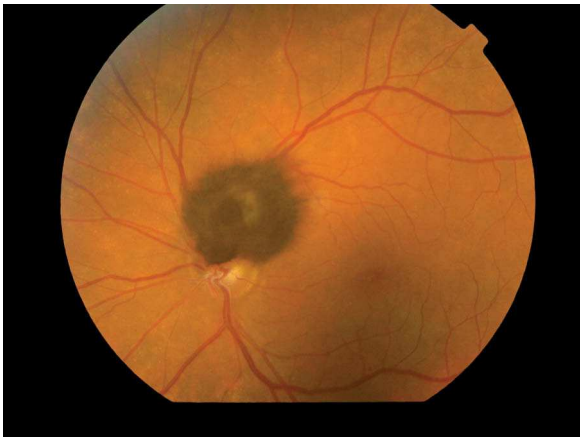

Figure 1 Papillary melanocytoma (Dr. Lorenzo's case).

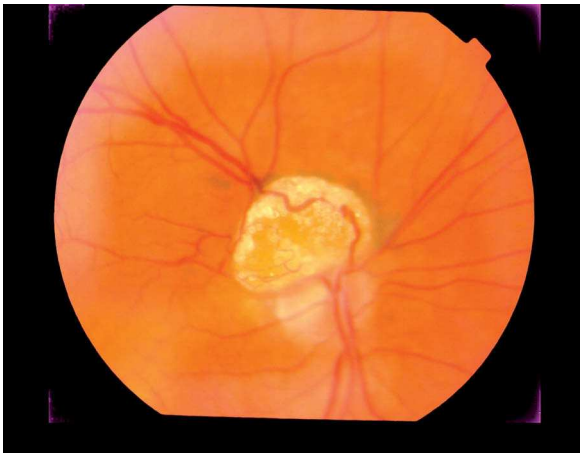

Figure 2 Papillary astrocytoma or papillary hamartoma (Dr. Alcubierre's case).

specific. Other anomalies present in the fundus may indicate a different aetiology of intracranial hypertension, so we emphasise the importance of a fundus examination under mydriasis. Still, the main indicator that the papillary oedema under study is being caused by an optic neuropathy is the visual function impairment from the early stages. The history of acute or chronic visual loss, colour vision impairment, afferent pupillary defect or atypical findings in the fundus all count for neuropathy and against papilloedema. We believe that the description of neuropathies and their differential diagnosis are beyond the scope of this publication and refer readers to the text of Arruga et al.[4] We have attached our proposed diagnostic approach to papillary oedema suspected of papilloedema in adults and children (figs. 3 and 4).

Finally, with reference to the usefulness of optical coherence tomography, its main limitation in the differential diagnosis of papilloedema is that the current resolution

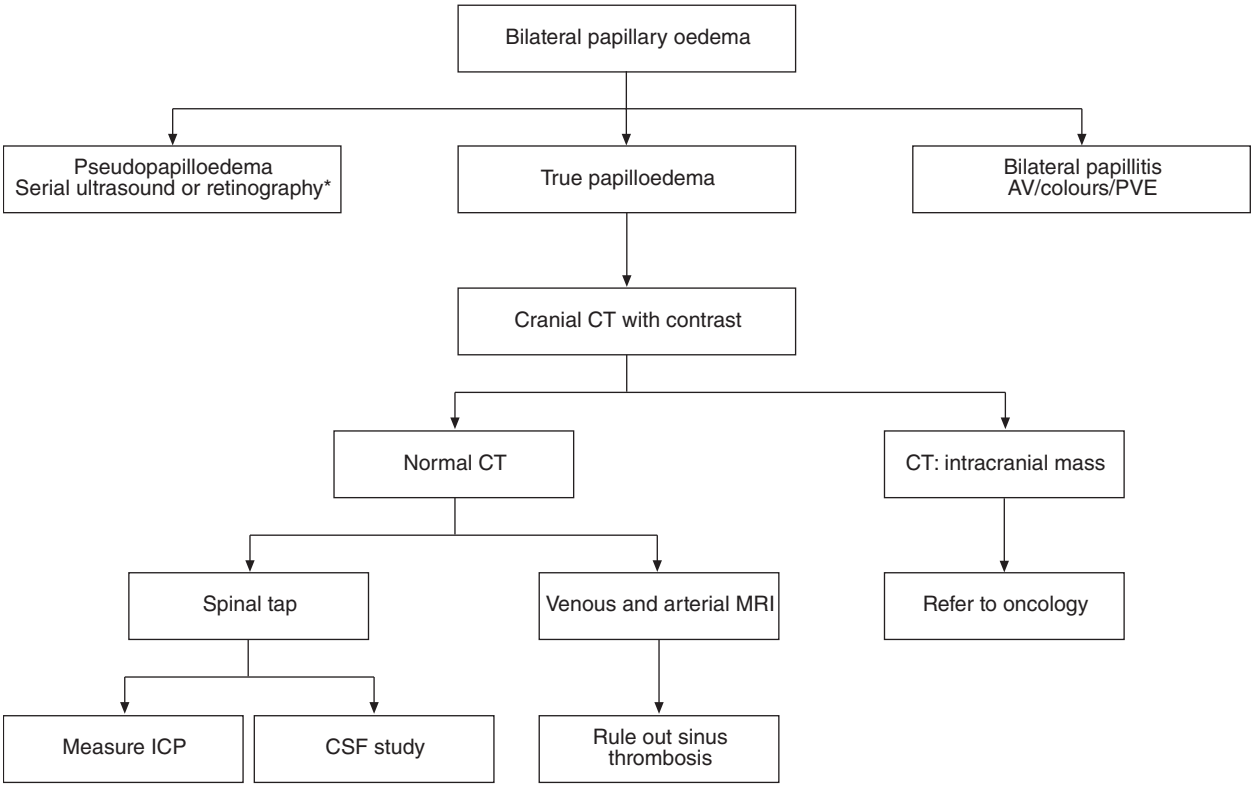

Figure 3 Papilloedema in childhood. Proposed approach guide. *Serial retinography based on patient cooperation. CSF: cerebrospinal fluid; CT scan: computed tomography scan; ICP: intracranial pressure; MRI: magnetic resonance imaging.

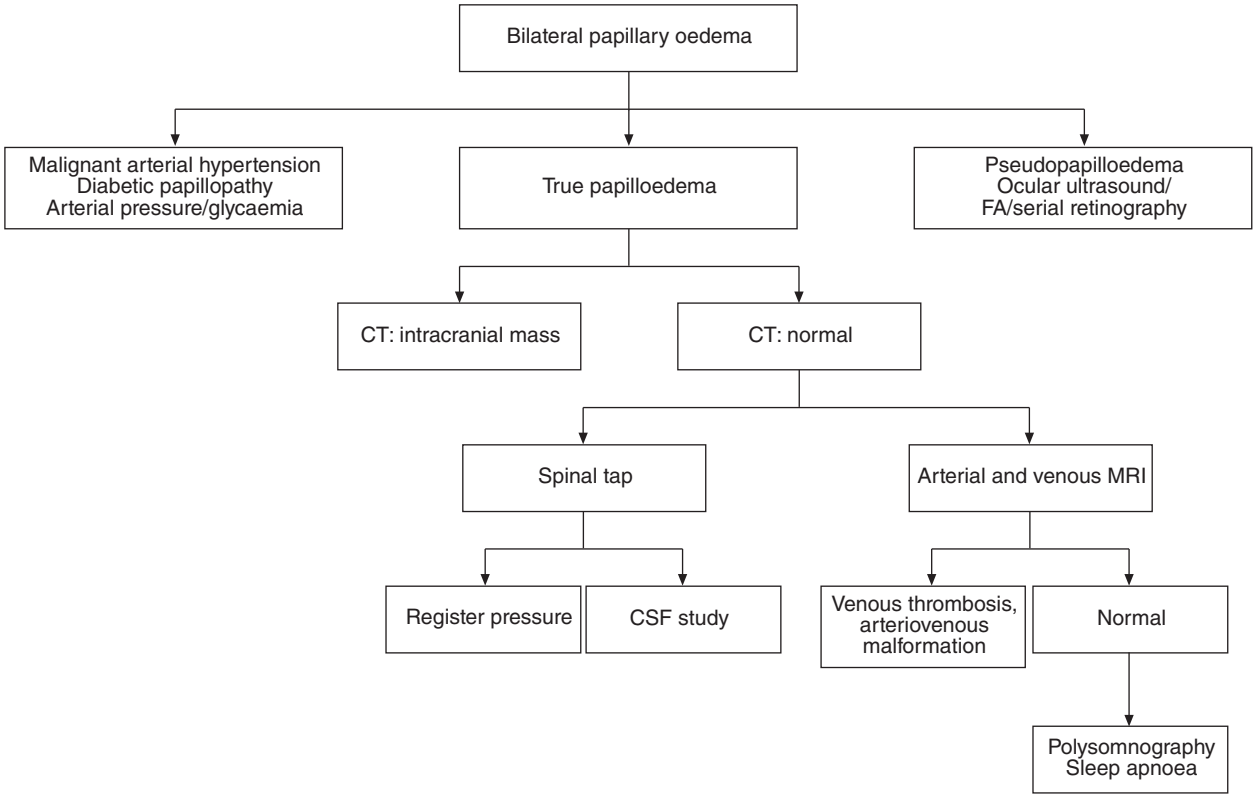

Figure 4. Papilloedema in adulthood. Proposed approach guide. CSF: cerebrospinal fluid; CT scan: computed tomography scan; FA: fluorescein angiography; MRI: magnetic resonance imaging.

does not differentiate between a thickening of the retinal nerve fibre layer from an increase in their number (pseudopapilloedema by full papilla), or from an intracellular oedema (in papilloedema and other neuropathies). Even in papilloedema monitoring, it should be interpreted with caution for the same reason, given that thinning or normalisation of the fibre layer thickness can occur either by resolution or by axonal loss.[5]

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