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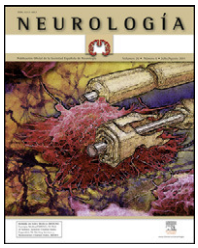

LETTERS TO THE EDITOR

## III cranial nerve palsy and brainstem disfunction following retrobulbar anaesthesia☆

## Paresia de III par craneal y afectación troncoencefálica por difusión intradural de anestesia retrobulbar

*Sir,*

There are currently various techniques of locoregional anaesthesia used in cataract surgery. One such technique, described by Knapp in 1884,[1] is retrobulbar anaesthesia, which is currently applied in certain cases of outpatient eye surgery. At present, the most commonly used technique in the treatment of cataract is phacoemulsification with laser. This is very useful in cases of soft cataract, which responds well to laser treatment. It requires a small incision and local anaesthesia techniques with a good safety profile, such as sub-Tenon's or peribulbar anaesthesia. However, the laser response may not be entirely satisfactory and accurate in very advanced and hardened cases of cataract. Such cases require, on the one hand, complete akinesia of the eye to correctly perform extracapsular extraction and, on the other hand, deeper anaesthesia because they require a larger incision and process that is more cumbersome and uncomfortable for the patient. Despite the improved safety profile of sub-Tenon's anaesthesia,[2] the retrobulbar technique makes faster and deeper anaesthesia possible in these cases, in addition to obtaining akinesia or fixing of the eyeball (which is not the case with sub-Tenon's or peribulbar application techniques), which is critical to the success of the procedure. For the same reasons, its drawback is an increased risk of complications. There have been reports of neurological complications associated with this technique, such as decreased level of consciousness, seizures, abnormal heart rate and breathing, arterial hypotension and dysfunction of cranial nerves. A very rare complication is paralysis of the contralateral extrinsic ocular musculature. We present a case with brainstem involvement and complete paresis of the third cranial nerve in the contralateral eye after retrobulbar anaesthesia, exposing the possible mechanisms involved.

We present the case of a 71-year-old patient with a history of diabetes mellitus who was admitted to our hospital for a scheduled intervention for a very advanced (hardened) cataract in the right eye. Given the morphological characteristics of the cataract, the technical choice of extracapsular extraction and the need for rapid anaesthesia and complete akinesia of the eyeball, we decided to use retrobulbar anaesthesia during the procedure. According to protocol, we preoperatively injected 5 ml of a 0.5% bupivacaine and 2% lidocaine mixture through a retrobulbar puncture using a 22 G Whitacre needle. We avoided exceeding a maximum eye pressure of 50 mmHg and there were no incidences during the application. We extracted the right eye lens by placing an optical lens on the posterior capsule, without any complications. However, a few minutes after the procedure, the patient presented a transient episode of disorientation, agitation, cyanosis, sweating, arterial oxygen desaturation, elevated blood pressure levels and lack of response to stimuli. After stabilising the patient, we requested an assessment from the Neurology Service. The initial scan showed unresponsive mydriasis, ptosis and limitation of left medial and superior rectus muscles, all compatible with complete paralysis of the third cranial nerve in the left eye and with no other focal neurological data. After 10 min, unresponsive mydriasis persisted along with minimal palpebral ptosis and mild limitation of the medial rectus muscle of the eye. Intraocular pressure was normal. A fundoscopy of the eye revealed no abnormalities. Minutes later, the symptoms had subsided. We carried out an urgent CT scan, which showed no acute changes and the patient was admitted to the Neurology Service, where a vascular study was conducted. The analyses included a vasculitis study (with a normal result) and a cerebral angioresonance, which ruled out any vascular alterations that justified the symptoms. The patient remained haemodynamically stable during admission and was discharged after a normal neurological exploration, which showed no ophthalmoparesis.

It has been estimated that the prevalence of neurological complications associated with retrobulbar anaesthesia is less than 1% of cases.[3] Nicoll et al.[4] studied 6000 patients undergoing retrobulbar anaesthesia, of which only 16 patients developed neurological complications. The onset time after injection was variable, with an

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average of 8 min (range from 2 to 40 min), and the possibility of life-threatening neurological complications was rare but unpredictable. In general, a transient and self-limited course has been observed, presumably coinciding with metabolism of the anaesthetic agent. There have been reports of loss of consciousness, apnoea and eventually cardiac arrest,[5] as well as episodes of hypotension and bradycardia.[6] Hypertension and tachycardia are due to a phenomenon of vagolysis or to blockage of the carotid sinus reflex. Hearing loss due to involvement of the eighth cranial nerve, vertigo, aphasia, dysarthria and hemiparesis are other symptoms described. There have also been reports of amaurosis and oculomotor paresis.[7,8] Antoszyk and Buckley[9] presented 3 cases of paralysis of the second and third contralateral cranial nerves.[9] A mechanism of anaesthetic diffusion into the subarachnoid space by an inadvertent puncture of the meningeal sheath surrounding the optic nerve has been postulated. Excessive local pain or elevated resistance during anaesthetic injection is due to ''prodromal'' symptoms or signs that enable this circumstance to be suspected. Communication, and thus a potential for diffusion, has been demonstrated between the optic nerve sheath and the subarachnoid space.[10] Orbitography showed the passage of contrast from the optic nerve sheath to the subdural space, covering the optic nerve to the medial fossa, in 5 patients undergoing retrobulbar anaesthesia.[11] This route has also been demonstrated in cadavers.[12] The case under discussion presented a transient episode of decreased consciousness level, hypertension and contralateral ophthalmoparesis, which receded after a few minutes. The spontaneous resolution of the symptoms, clinical course and normal results in the tests performed could be explained by a transient effect of the anaesthetic agent at the level of the central nervous system and point to its dissemination into the subarachnoid space as the most plausible cause. Other possible mechanisms have been described in the literature, such as an inadvertent injection of anaesthetic into the ophthalmic artery, with retrograde reflux into the carotid artery territory. Although the ophthalmic artery crosses the orbit above the optic nerve, it runs below in 15% of patients, very near the region where retrobulbar injection is applied. This mechanism has been proposed in those cases where epileptic crises occur during or immediately after the application of anaesthesia.[13] Finally, a myotoxic effect has been demonstrated in cases of strabismus after retrobulbar or peribulbar anaesthesia. In these cases, progressive fibrosis develops after the initial diplopia, with the inferior rectus muscle being the most commonly affected.[14]

We conclude that retrobulbar anaesthesia may infrequently be associated to the onset of neurological symptoms, mainly due to a diffusion mechanism of the local anaesthetic into the subarachnoid space. The possibility of such complications makes the presence of personnel trained in applying strict monitoring measures and immediate stabilisation of patients if they do appear into an essential requirement.

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