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# Bilateral Optic Neuropathy: What Facts in a Valsalva's Maneuver Context? A Case Report

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## ABSTRACT

**Introduction: Repeated Valsalva's maneuvers can result in severe optic neuropathy. We describe here, for the first time in the literature, a case of blindness related to this mechanism. Observation: This was a young autistic man engaged several times a day in Valsalva's maneuvers and who presented with a bilateral and rapidly progressive decrease in visual acuity. The anamnesis, the clinical examination and the radiological explorations made it possible to make the diagnosis of bilateral optic neuropathy secondary to these Valsalva's maneuvers. Discussion: The optic neuropathy occurring during the Valsalva's maneuver would be the result of three associated mechanisms: abnormalities of the orbital vascularization, ischemia and chronic compression of the optic nerve responsible for an almost total destruction of the optic nerve and source of blindness.**

**Keywords:** Optic neuropathy, Valsalva, bilateral, vascular involvement.

## INTRODUCTION

Bilateral optic neuropathies are most often secondary to a hereditary, toxic or deficiency mechanism. The impact of Valsalva's maneuvers in the genesis of bilateral optic nerve involvement is exceptional, hypothetical and no publication has reported such a case to date. This maneuver is readily associated with certain physical exercises or professions such as weightlifting or in active trumpeters; but also, in case of cough, constipation, urinary obstructive syndrome or even asthma [1].

We report a case of bilateral optic neuropathy that occurred in the context of Valsalva's maneuvers performed several times a day by a young autistic man with no hereditary history of decrease in visual acuity or genetic component in favour of Gilles de la Tourette's syndrome (GTS).

## OBSERVATION

This is a 36-year-old patient, a known autistic young man living in a nursing home and seen in consultation for a decrease in bilateral visual acuity, predominantly on the right. He had no motor tics or vocal tics (coprolalia or repeated abuses). This painless decrease in visual acuity is described as quite sudden, observed a month earlier and accompanied by treated purulent conjunctivitis. The patient has been performing Valsalva maneuvers daily for several years. He has an ENT follow-up for bilateral chronic otitis media. The corrected visual acuity measured 2 years before the initial consultation was 8/10$^{th}$ in the right eye and 10/10$^{th}$ in the left eye. The ophthalmological examination carried out in our service found a corrected visual acuity reduced to the movements of the hands on the right and 1/10$^{th}$ on the left. The anterior segments are normal. There is neither proptosis nor oculomotor disorder but there is a relative pupillary deafferentation on the right. The intraocular pressure is 13 mmHg in the forced air on the right and 11 mmHg on the left. Fundus examination shows papillary pallor with small retinal vessels on the right and a normal-coloured optic disc with a 4-5/10 cup/disc on the left. On Goldmann's visual field, there is a concentric narrowing of the isopters on the left (figure 1), that of the right eye being impossible.

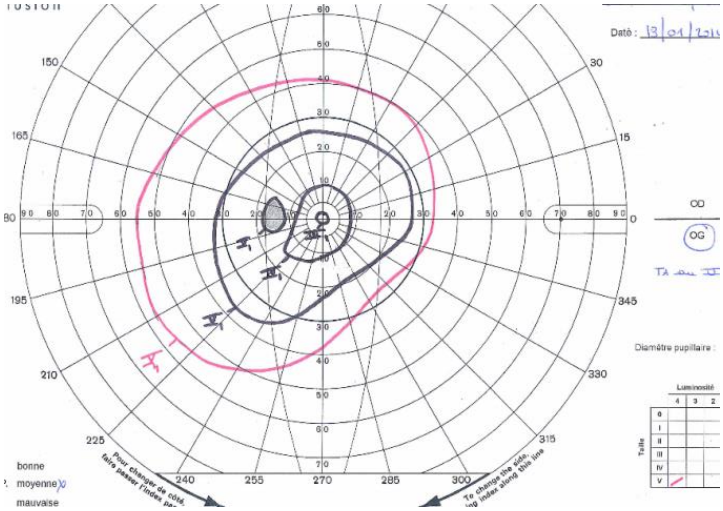



**Figure 1: Goldmann's visual field showing global constriction of all isopters in the left eye.**

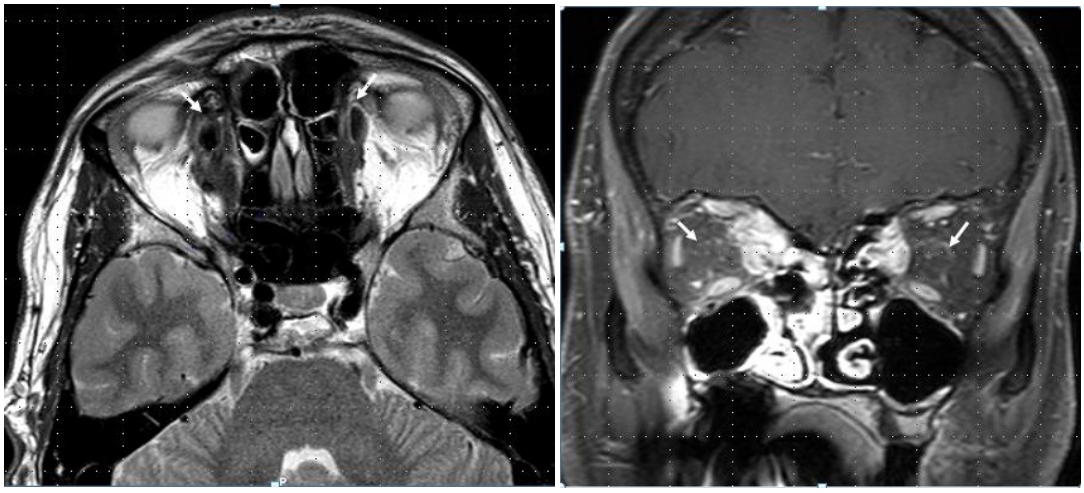
RNFL optical coherence tomography (OCT) shows a temporal and superior thinning more marked on the right; macular OCT is normal in both eyes. Exploration of colour vision reveals a red-green axis dyschromatopsia; the biological assessment (CBC, BSR and CRP) is normal. We did not find any genetic mutation or mitochondrial DNA mutations.

A cerebral MRI and an electroencephalogram performed could not show any abnormalities related to Gilles de la Tourette syndrome.

However, the radiological assessment (orbito-cerebral CT and MRI) found a bilateral bone defect of the internal wall of the orbit associated with a small tissue intra-orbital and ethmoidal formation, containing air and taking little the contrast in contact with the bony defect of the wall of the orbit without apparent compression of the optic nerve (figure 2).



**A and B**



**C and D**

**Figure 2: 2A Pneumorbit in the form of hypodense vacuoles (white arrows) on CT at the level of the internal walls with bone defect at the level of the 2 internal walls (red arrows)**

2B giant vacuoles during Valsalva's maneuver. 2C intra-tissular formation with pneumorbits within it on MRI; 2D coronal section showing the optic nerves.

Orbital surgical exploration found a mucosal appearance on the right without tumor and the anatomopathological examination concluded to a squamous cell mucosa associated with fibrosis. The mechanism of blindness is therefore bilateral optic neuropathy probably secondary to repeated Valsalva's maneuvers. Stopping the Valsalva's maneuvers and a long-term corticosteroid therapy temporarily stabilized visual acuity at $1/10^{th}$ on the right and $4/10^{th}$ on the left. In the long-term follow-up, the patient resumed the said maneuvers which led to a further decrease in visual acuity resulting in an absence of light perception on the right, a positive light perception on the left and bilateral papillary pallor (figure 3) causing irreversible bilateral blindness.

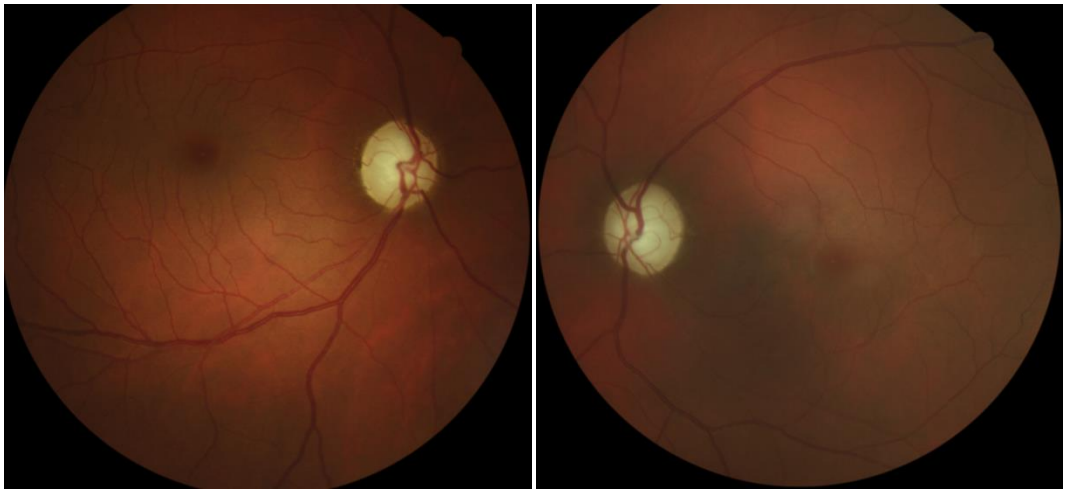


**Figure 3: Bilateral optic atrophy with a "waxy" appearance.**

In addition, there was the protrusion of a right orbital mass sitting at the level of the right internal canthus and increasing in volume during the Valsalva's maneuver associated with bilateral semi-mydriasis and right exotropia in the primary position (figure 4) whose surgical management was postponed due to rhino-sinus superinfection.



**A**



**B**

**Figure 4: Aspect of the right orbital mass before (4A) and after the Valsalva maneuver (4B).**

## DISCUSSION

The Valsalva's maneuver consists of blowing violently with full lungs, forcing with the chest and abdominal muscles, nose and mouth closed, which has the effect of increasing chest pressure and abdominal pressure; it is performed in particular in weightlifting, in trumpet players, during vomiting, coughing and abdominal contraction during certain physical exercises [2]. This maneuver is associated with an increase in blood pressure depending on the duration and the force of exhalation. According to some authors [3, 4, 5], the Valsalva's maneuver results in an increase in peripheral vascular resistance, acute venous congestion due to an obstacle to venous return and a decrease in the protective effects of cerebral vessels. The combination of the increase in blood pressure and the failure of cerebral blood flow autoregulation systems would be the cause of an increase in the trans-parietal pressure gradient likely to cause damage to the vascular tissue or even aneurysm rupture in exposed patients [6]. Thus, according to Farrell et al [7], the transient increase in cerebral perfusion pressure during physical stress such as that observed during Valsalva's maneuvers would lead to the destruction of the lamina responsible for dissection at the level of vessels, resulting in ischemia with lack of perfusion of structures below, including the optic nerves.

At the ocular level, the choroid, dynamic vascular tissue ensures the vascularization of the outer layers of the retina, the supply of nutrients, the maintenance of thermoregulation and the regulation of ocular pressure by acting on uveoscleral drainage [8]. During the Valsalva's maneuver, the rise in intrathoracic and intra-abdominal pressure rapidly affects the jugular, orbital and vortex venous vascularization, resulting in an increase in pressure within the choroidal veins [9], which would be at the origin of an increase in the choroidal volume and by extension of the uveal volume. This cascade of physiopathological events has the immediate result of an increase in intraocular pressure (with a direct mechanical action on the optical fibers at the level of the riddled plate, an area of weakness known par excellence of the optic nerve) which will depend on the duration and the exhalation force created.

Apart from the above-mentioned mechanisms, compression of the optic nerve is undoubtedly the main mechanism of this optic neuropathy. The bone window images in this patient show breaches in the internal wall of both orbits as well as a pneumorbit. These hiatuses result from the sudden nasal hyper-pressure that occurs during each Valsalva's maneuver and ends up

weakening the bony septum [10]. The solution of continuity between the nasal cavity and the orbit caused suffering of the optic nerve secondary to compression by the pneumorbit (tissue mass associated with one or more vacuoles) whose increasing volume due to the repetition of the maneuvers created a "vicious circle" of compression – relaxation compression. Moreover, the pallor of the neuro-retinal ring is an argument in favour of a compressive neuropathy and not pure pressure, this pallor being in 94% of cases specific to a non-glaucoma excavation [11,12]. Greenfield [13], Uber-Zak [14] and Orssaud [15] studied the papillae of patients followed for normal pressure glaucoma (NPN) and those of patients with compressive optic neuropathy and concluded that the pallor of the neuro-retinal ring is specific to non-glaucomatous optic neuropathy in 70% of cases.

Our patient's functional prognosis was at stake with irreversible blindness due to the difficulties of management, but especially the persistence of repeated Valsalva's maneuvers.

## CONCLUSION

Valsalva's maneuver commonly used in the learning to dive and in the learning of certain sports activities and in certain professions could be the cause of damage to the optic nerve with a sometimes-severe prognosis. The observation reported above should make it possible to draw attention to practices accompanied by repeated Valsalva's maneuvers in order to prevent possible visual accidents and to be able to develop a physio-pathogenic approach to the probable occurrence of impairment of optic nerves.

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