Optic Nerve Elongation: Does It Exist?

Abstract—The length of the optic nerves is a reflection of normal postnatal cranio-orbital development. Unilateral elongation of an optic nerve has been observed in two patients with orbital and skull base neoplasms. In the first case, as compared to the patient's opposite, normal optic nerve, an elongated length of the involved optic nerve of 45 mm was present. The involved optic nerve in the second patient was 10 mm longer than the normal, opposite optic nerve. The visual and extraocular function was preserved in the second patient. The first patient had only light perception in the affected eye. In this paper, the embryology, anatomy, and physiology of the optic nerve and its mechanisms of stretch and repair are discussed. (*Skull Base Surgery*, 6(3):171–180, 1996)

The optic nerve is an anatomic structure that is often encountered in skull base and cranio-orbital surgery. Knowledge of the normal as well as abnormal embryology, anatomy, and physiology of this cranial nerve is crucial for safe surgery. We present two cases of optic nerve length abnormality. Both cases illustrate optic nerve elongation beyond its normal anatomic range. Experimental and clinical data are reviewed to explain this observation.

Case One

A nine-year-old female with a history of neurofibromatosis type I was referred from Central America for a progressively enlarging right eye growth. The patient was born following normal gestation with no complications. At birth she had a "red mark" around her right eye which, by 3 months, was protruding, including the eye itself. She had cafe-au-lait spots on her back, abdomen, and thighs. By the age of 2 years she was referred to another institution in the United States, where a lid biopsy was performed and confirmed the diagnosis of neurofibroma. Computed tomography (CT) scan revealed a mass replacing the orbit and greater wing of the sphenoid

with extension to the orbit and displacement of the eye. On examination, the patient had marked proptosis, but also had light perception in the right eye with the ability to count fingers. Chromosome analysis revealed a normal female.

By the age of 9 years, the mass was substantially larger (Figs. 1, 2). The patient was still able to count fingers but could not read an eye chart. Gross extraocular motor function was minimally present. Pulsation of the mass, synchronous with the patient's pulse was evident. CT scan (Fig. 3) revealed extension of the mass posterolaterally through the lateral orbital wall. There was a meningoencephalocele which protruded into the orbit, in addition to the enhancing mass. The optic nerve appeared elongated compared with the normal eye, but was not infiltrated with tumor. Because the tumor encased the globe and optic nerve, the patient underwent orbital exenteration, right frontotemporal craniotomy, repair of meningoencephalocele, and reconstruction with titanium mesh. Postoperatively, an orbital prosthesis was placed into the defect.

Gross pathologic examination revealed a 58 × 64 × 45 mm mass which totally surrounded the globe and optic nerve but was minimally adherent to its dura. The subdural and subarachnoid spaces were normal, and the mass

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Figure 1. Inferior view demonstrating dramatic right eye proptosis.

Figure 2. Superior view demonstrating right eye proptosis.

did not invade the nerve. The intraorbital optic nerve with its meninges was 4.5 mm in diameter and 45 mm in length (Figs. 4, 5). Microscopic examination of the globe revealed minimal decrease in the retinal ganglion cells and no atrophy or papilledema of the optic disc. The optic nerve was myelinated, with no atrophy and with no astrogliosis (Fig. 6).

The patient is being followed in Central America. Two-year follow-up examination was reported to us as revealing no recurrence. The patient has been wearing an eye prosthesis.

Case Two

A 75-year-old white male was referred for evaluation of significant proptosis due to a left orbital mass. The patient had undergone an excision of a "sebaceous cyst" from the left upper eyelid at the age of 8 years. The proptosis developed in his early teens and gradually worsened over the ensuing years. He was examined periodically throughout the years. When first seen at the age of 50, his visual acuity was OD-20/20 and OS-20/50 (corrected). Color vision was normal. The patient later developed a slight nuclear cataract in his right eye and a moderate nuclear cataract in his left eye. Dilated fundus examination showed a myopic conus in the left eye and a normal fundus in the right eye. He had a vertical muscle imbalance of 4 prism diopters which was corrected with prisms in his spectacles.

Physical examination revealed an 11 mm proptosis in the left eye with 3 mm and 5 mm displacement inferiorly and medially, respectively (Fig. 7). There was moderate lagophthalmos with no corneal exposure signs. The extra-

Figure 3. Axial CT scan showing mass effect of the neurofibroma with meningoencephalocele causing right eye proptosis.

ocular movements were grossly intact. No afferent pupillary defect was noted OS. Vision (corrected) was 20/20 and 20/100 in the right and left eyes, respectively, as measured on a Snellen reading chart. The pupils were round, regular, and reactive. CT scan demonstrated a low density mass at the posterosuperior aspect of the left orbit (Fig. 8). There was remodeling of the orbit. The left optic nerve was stretched, measuring 10 mm longer than the right optic nerve. There was no tenting of the posterior globe. Due to the absence of afferent pupillary defect, the presence of normal color vision, and the presence of the cataract in the left eye, it was thought that his relative decreased vision in that eye was due to the cataract and not optic nerve stretching. Cranio-orbital surgery revealed the mass to be extraconal and connected to the upper eyelid cyst. The bilobed mass was excised, and the superior fronto-orbital region was reconstructed with titanium mesh. Pathologic examination revealed a dermoid cyst.

Figure 4. Intraoperative view demonstrating the diameter of the optic nerve.

Figure 5. Intraoperative view demonstrating the length of the optic nerve.

Figure 6. Histologic micrograph showing a normal appearing optic nerve. (Hematoxylin & eosin, mag. $\times 400$).

Figure 7. Inferior view showing left eye proptosis.

DISCUSSION

Embryology

Eye formation is first evident at approximately 22 days of gestation. The optic nerve develops in the substance of paired structures called the optic stalks. They represent an evagination of the forebrain. Therefore, the optic nerve is actually an extension of the central nervous system. At the end of the stalk is a hollow diverticula called the optic vesicle. This vesicle invaginates to form an optic cup which eventually becomes the globe itself (Fig. 9). This invagination is analogous to the otic vesicle which forms the otic pit in inner ear development.

The retinal ganglion cells develop from the walls of

the optic cup. The axons of the ganglion cells enter the previously hollow optic stalk and proceed toward the brain and gradually convert it into the optic nerve.

By the fifth month, dura is distinguishable as a separate layer, and by the sixth and seventh months, the arachnoid sheath differentiates from the dura. During this period, the optic nerve increases in both diameter and length. The most rapid increase occurs during the fifth month when the dura becomes distinguishable. At this time, the nerve length increases from 3 mm to 7 to 8 mm (Table 1). By the eighth month, the optic nerve is well developed but is not yet of adult size. After the visual pathway has developed, myelination of nerve fibers occurs from the brain towards the optic nerve. As a consequence, myelination of the optic nerve is usually incomplete at birth and is completed shortly thereafter. This is manifested clinically by the less developed visual acuity noted in newborns. Myelination is not necessary for vision, however. It only speeds the transmission of nerve impulses and sharpens visual acuity.

The process of myelination affects the dimensions of the optic nerve. This must be considered when assessing optic nerve dimensions pathologically and radiographically. At birth, the diameter of the nerve is approximately 2 mm and its length is 24 mm. Myelination increases the thickness of the nerve to 2.4 mm during the third month after birth. Around the sixth month after birth, the nerve lengthens considerably to accommodate the growth of the orbit and brain. At puberty, it has a diameter of 3 to 4 mm, and its length averages 40 mm.¹ In the patient discussed in case one, the resected intraorbital optic nerve was 4.5 mm and 45 mm in diameter and length, respectively. These

Figure 8. Axial CT scan showing elongated right optic nerve.

Figure 9. The formation of the optic vesicle and cup in a 7.5 mm human embryo. (Miller N: Embryology of the afferent visual system. In Walsh and Hoyt's Clinico Neuro-ophthalmology. Baltimore: Williams and Wilkins, 1982, p. 5. Reprinted with permission.)

dimensions are adult size and are therefore abnormal for this child.

The well-established developmental concept of a "critical" period of visual development can explain this patient's relatively poor vision despite a histologically benign appearing optic nerve. It has been demonstrated experimentally that early visual deprivation during a "critical" period shortly after birth can irreversibly affect the development of the visual cortex, which can lead to decreased vision.² Clinically, this condition is termed amblyopia. This has led to the common practice of correcting pediatric visual problems early in life. Patient one had impaired vision for most of her life in the affected eye, presumably due to tumor distortion of the globe with gross proptosis. This probably led to underdevelopment of the visual cortex with resultant amblyopia. Her decreased visual acuity, therefore, may have been due to impaired central processing rather than impairment of the optic nerve itself.

Table 1. The Dimensions of the Optic Nerve

Age	Optic Nerve Length	Optic Nerve Diameter
5 months (gestation)	7–8 mm	approx. 1 mm
Birth	24 mm	2 mm
Puberty	40 mm	3–4 mm
Adult	35–55 mm	4 mm

Adapted from: Duke-Elder SS, Cook C: Normal and abnormal development. In Duke-Elder SS (ed): System of Ophthalmology. St. Louis: C.V. Mosby, 1963.

Anatomy

The optic nerve consists of approximately one million axons. The adult optic nerve is approximately 35 mm to 55 mm in length from the globe to the chiasm. It is divided into four segments: intraocular, intraorbital, intracanalicular, and intracranial (Fig. 10). The dimensions of these segments are illustrated in Table 2. The intraorbital nerve portion is 25 mm to 30 mm in length. This includes the short intraocular distance. The intraorbital length from the posterior globe to the orbital apex, measures 20 mm to 25 mm. This length is 6 mm to 8 mm longer than the straight line distance from the back of the optic nerve to the optic foramen. This redundancy causes a sinuous course of the nerve through the orbit, permitting the nerve to move freely during eye movements. It allows the nerve to be pulled at least 6 mm to 8 mm before it gets tethered (Fig. 11). Tethering of the nerve would affect normal extraocular gaze movements. Significant proptosis with intact ocular gaze function and without tenting of the posterior globe surface, would suggest optic nerve elongation, as dramatically illustrated in the patient discussed in case two.

Assessment of optic nerve elongation and proptosis requires radiographic knowledge of the orbit and optic nerve. A thorough description of the pertinent CT scan studies of this area is provided by Peyster et al.³ Approximately 40% to 50% of the globe lies behind the interzygomatic line, drawn by connecting the anterior margins of the zygomatic processes on a CT section which includes the lenses. The lens appears as a dense biconvex

Figure 10. Sagittal view of the intraorbital and intracranial optic nerve. (Spoor TC: The optic nerve— anatomy and physiology. In Atlas of Optic Nerve Disorders. New York, Raven Press, 1992, p. 3. Reprinted with permission.)

Table 2. The Anatomical Segments of the Adult Optic Nerve

Segment	Optic Nerve Diameter	Optic Nerve Length
Intraocular	1.5 × 1.0 mm	1.0 mm
Intraorbital	3–4 mm	25–30 mm
Intracanalicular	3–4 mm	10 mm
Intracranial	4 mm	10 mm

structure in the anterior portion of the globe and divides it into an anterior portion filled with aqueous humor, and a larger, posterior portion filled with vitreous humor. The orbital septum, a continuation of the periorbita, extends from the orbital rim to insert onto the tarsal plates of the eyelids.

Three factors that influence the appearance of the optic nerve on CT scan are the plane of section, sinuosity of the intraorbital optic nerve, and direction of gaze. No single plane of section is ideal for visualizing the full course of the optic nerve. Any single plane of section will

Figure 11. Dramatic proptosis with posterior globe tethering or "tenting" and optic nerve straightening. (Dolman PJ, Glazer LC, Harris GJ, Beatty RL, Massaro BM: Mechanisms of visual loss in severe proptosis. Ophthalmic Plastic and Reconstructive Surgery, 7:259, 1991. Reprinted with permission.)

Figure 12. Axial planes of section and external landmarks. 1: -20° to orbitomeatal baseline. 2: -10° to orbitomeatal baseline. 3: orbitomeatal baseline. (Unsold R, DeGroot J, Newton TH: Images of the Optic Nerve: Anatomic—CT Correlation. American Journal of Roentgenology 135: 768, 1980. Reprinted with permission.)

always extend through the nerve obliquely and will not be entirely parallel to the nerve.⁴ Previous studies have shown that a CT section plane close to the Frankfort-Virchow plane (infraorbitomeatal line) is preferred for adequate visualization of the optic nerve (Fig. 12).^{5,6} But modifications of this section plane are often needed due to anatomic variability among patients.

Salvolini et al⁷ suggest that for optimal imaging of the optic nerve, the CT plane should pass through three elements: lens, papilla (posterior globe), and the optic nerve up to the optic canal. This plane is called the neurocular plane (NOP). In this plane, the globe is cut at or near its equatorial diameter.

However, gaze shifts change the course of the optic nerve (Fig. 13). In certain gaze shifts, the optic nerve is better demonstrated in a plane other than the NOP. Linear hypodensities on CT may be mistaken for lesions near the nerve unless one takes into account the position of gaze

Figure 13. Gaze changes and redundancy of the intra-orbital optic nerve results in its sinuous course. (Spoor TC: The optic nerve—atomy and physiology. In Atlas of Optic Nerve Disorders. New York, Raven Press, 1992, p. 4. Reprinted with permission.)

(Fig. 14). A light source is often used in CT scanners to help keep the patient in a fixed gaze position. The optimal technique for the CT evaluation of the full course of the optic nerve as related to position of gaze does not seem to be in the primary position in the NOP, as this allows the sinuous course of the nerve.⁸ The lateral and oblique positions of the nerve are inappropriate for scanning because the resulting asymmetry would make comparisons of both nerves difficult. The optic nerve may therefore be stretched either by having the patient look straight ahead and up while scanning in an appropriate, negative angulation or scanning during a downward gaze with a positive

Figure 14. Linear hypodensities crossing CT images of optic nerve may be related to partial volume effect due to optic nerve redundancy or gaze shifts. (Salvolini U, Cabanis EA, Rodallec A, Menichelli F, Pasquini U, Iba-Zizen MT: Computed tomography of the optic nerve: Part 1. Normal results. Journal of Computer Assisted Tomography 4:147, 1978. Reprinted with permission.)

Figure 15. Schematic drawing of the optic nerve in primary gaze (top) and in upgaze (bottom). In upgaze, the optic nerve is stretched, lying almost in one plane throughout its intraorbital course. (Unsold R, Newton TH, Hoyt WF: CT examination technique of the optic nerve. *Journal of Computer Assisted Tomography* 4:562, 1980. Reprinted with permission.)

angulation. An angulation of -20° to the orbitomeatal baseline is recommended. This allows the optic nerve to be shifted downward and stretched, lying almost along its entire orbital course in one plane (Fig. 15). Reliable judgments regarding the caliber of the optic nerve can then be made.

Taking the above information into account, comments on the CT evaluation of the case two patient can be made. The CT in this patient was taken in primary gaze, -20° to the orbitomeatal line. Although precise evaluation of the full course of the optic nerve is theoretically not possible, simultaneous comparison of both nerves showed that the left optic nerve was elongated, which was due to the intraorbital mass. Ideal CT scanning in patients with intraorbital masses causing elongation or straightening of the nerve is done in the primary gaze.

Normally, on CT the optic nerve lies in the central axis of the orbit. All three meningeal layers surround the nerve. Therefore, the intraorbital optic nerve as seen on CT is the composite of the nerve and its meningeal investments and is often referred to as the optic nerve/sheath. Enlargement of the optic nerve/sheath on CT may be due to processes involving the sheath and not the nerve itself. In the adult, the optic nerve diameter is 3.5 mm to 4 mm in the axial plane and 4 mm to 6 mm in the coronal plane. The course of the optic nerve is oblique. From the globe, it passes posteriorly, superiorly, and medially to exit the orbit through the optic canal. Because of this oblique course, the nerve, as seen on axial plane, may describe a gentle curve rather than a straight line (Fig. 16). In the coronal plane the nerve may appear elliptical.

Physiology

The case one patient presented with an orbital tumor which displaced the globe and elongated the optic nerve.

Figure 16. Left: Visualization of the optic nerve superiorly reveals that the most proximal part of the nerve has relatively straight course and becomes more sinuous distally. Middle: Axial plane of the optic nerve in primary position. Right: Sagittal plane of optic nerve in primary position of gaze in a plane parallel to the orbitomeatal line. (Unsold R, DeGroot J, Newton TH: Images of the optic nerve: anatomic—CT correlation. *American Journal of Roentgenology* 135:769, 770, 772, 1980. Reprinted with permission.)

The patient nevertheless had light perception present in that eye. Although we cannot be certain that her vision impairment was not from nerve elongation, the possibility of amblyopia and the presence of a histologically normal nerve suggest that nerve dysfunction may not have been due to nerve elongation. In the case two patient, the absence of an afferent pupillary defect and intact color vision suggest that his decreased visual acuity may not have been due to chronic nerve elongation and nerve dysfunction but, more likely, to cataract formation. The preservation of some or most visual function with such gross distortion and elongation of the optic nerve present in both patients suggests the adaptability and plasticity of the visual system when subjected to gradual expansion and elongation. This phenomenon has been seen clinically in other situations. For example, patients with slowly enlarging optic gliomas and neurofibromatosis have been known to have stable visual acuity.^{9,10} Imes et al¹¹ described a patient with neurofibromatosis with stable vision who had optic nerve expansion secondary to a glioma. The nerve was elongated "1½ times normal" and was thought to be secondary to axial growth of the tumor. No histology of the nerve was reported. In 1915, Cushing et al¹² analyzed 81 cases of optic chiasma compression and showed that restoration of vision was possible following surgical decompression. Kayan et al¹³ examined 11 patients with compressive lesions of the intracranial optic nerve and chiasm. Following surgical decompression, the majority of these patients made substantial visual recovery within 15 days. In fact in most cases in which adequate decompression was obtained, the majority of the patients recovered within 10 to 14 days after surgery.¹⁴

Previous studies have attributed vision loss secondary to chronic compressive lesions to blockage of axonal transport and demyelination insufficient to cause cell death¹⁴ and not to vascular insufficiency, as previously thought.¹⁵ Remyelination of demyelinated fibers of a cat optic nerve after chronic compression has been demonstrated.¹⁶ This capacity to form myelin following demyelination occurs as long as the axon itself is intact.¹⁷ It is the acute lesions in the orbit that often produce vascular insufficiency with subsequent sudden vision loss.¹⁸

When visual axons are severed, but not compressed, they undergo Wallerian degeneration and chromatolysis. The retinal ganglion cells and optic nerve undergo atrophy and death. Clinically this is seen as pallor of the optic disc as opposed to the normal optic disc often seen in chronic compressive lesions. The regeneration of severed optic nerve axons has been demonstrated in animal models but not in humans. The cause of this failure to regenerate is obscure. Several hypotheses include the inhibitory influence of myelin breakdown products on regeneration, astrocytic scarring as a physical impediment to axonal growth, and formation of free radicals.¹⁹ Clearly, separate mechanisms of nerve injury and repair are involved in gradual compression or expansion versus transection/crush injury.

The response of the optic nerve to acute stretch injury and elongation has been demonstrated experimentally. Maxwell et al^{20,21} produced rapid stretch injury using a tension sling around the optic nerve of the guinea pig. The early observed response (within 15 minutes after injury) was disruption of axonal transport and damage to the nodes of Ranvier. Fourteen days after injury, reparative and recovery responses within the cell body were seen as were regenerative terminal axonal sprouts. Neurotrophic factors may be involved in this process.¹⁹ After stretch injury to the optic nerve, the guinea pig retina was found to show some loss of the retinal ganglion cells (RGC) by 14 days, but the majority of the RGC did not respond to the lesion.²¹ This process is in contradistinction to the mainly degenerative responses in the RGC and optic nerve in transection or crush injury of the optic nerve.

These studies provide evidence for optic nerve adaptation and regeneration after stretch injury and suggest a possible mechanism for the stable visual acuity in the cases presented. One must note, however, that these studies examined primarily rapid stretch injury and do not fully mimic the chronic elongation process in the present cases. To our knowledge, experimental studies on chronic elongation of the optic nerve have not been done. Our cases may be more analogous to gradual expansion and elongation to the peripheral nerves reported experimentally. Hall et al²² performed chronic expansion and elongation of the rat sciatic nerve, using a tissue expander, over the course of 2 weeks. They demonstrated that slowly expanded nerves showed no significant reduction in conduction velocity as compared with normal nerves. Nerves that were rapidly expanded exhibited large reductions in conduction velocity. Demyelination was seen early, but recovery of these histologic changes was not evaluated in this study. These data are supported by other studies.^{23,24}

We have observed a compensatory elongation and adaptability of the optic nerve to gradual globe displacement by tumor. Although there have been no studies examining the physiology and morphology of the optic nerve secondary to chronic elongation and stretch (eg, from tumor growth), the above studies provide inferential evidence that chronic elongation and expansion of nerves may not jeopardize their function. Although the optic nerve is not a peripheral nerve, but is indeed an extension of the central nervous system, extrapolation of these data sheds further light on the mechanisms of adaptability and plasticity of the optic nerve.

CONCLUSION

The optic nerve is often encountered by the skull base surgeon. After a review of the normal embryology, anatomy, and physiology of the optic nerve, we believe that we have provided clinical evidence of optic nerve

elongation with preservation of some or most vision. In patient one, the nerve was of adult size and did not cause tenting of the posterior globe. Similarly, there was no tenting of the posterior globe in patient two, and there was maintenance of normal extraocular gaze movements. Although we cannot definitively attribute the decrease in vision in patient one to amblyopia and not to chronic nerve elongation, amblyopia is a possibility since the child had poor vision in early childhood. Histologically, a relatively normal optic nerve does not support nerve elongation. In patient two, the absence of an afferent pupillary defect and the presence of color vision suggest that the optic nerve was not affected by its elongation. Still, optic nerve elongation and adaptation seems to exist clinically. Further research is needed to evaluate this phenomenon.

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