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**Independent Academic-Style Discussion Draft**

# A Unified Clinical Framework for Optic Nerve–Initiated Cerebral Visual Impairment

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**Abstract**

Visual disability is commonly defined in terms of visual acuity and visual field. While appropriate for many conditions, this approach does not fully capture difficulties experienced by individuals whose primary impairment lies in the cerebral processing and functional use of visual information rather than in ocular structure alone.

This paper integrates neurodevelopmental theory, emerging neuroimaging insights, and service-level analysis to examine how disorders arising from congenital and early-life optic pathway disruption may, in some cases, be more appropriately understood as neurodevelopmental visual processing conditions. It argues that reliance on acuity-based certification, conventional cognitive assessment, and eye-centred clinical pathways may fail to identify this form of impairment, with consequences for access to educational support, disability recognition, and income protection.

Current definitions of cerebral visual impairment recognise that anterior visual pathway disorders may coexist with CVI where they do not fully explain the level or pattern of impairment. Building on this distinction, the paper argues that ONH and CVI should not be collapsed into a single diagnosis, but may need to be considered within a connected visual pathway. Literature on functional vision, neuroplasticity, and pathway-wide visual-system effects supports a cautious pathway-based model.

This perspective supports a more integrated care pathway in which early identification of optic pathway disruption is complemented by functional assessment and ongoing neurovisual support. It further suggests that models of visual disability centred primarily on acuity and ocular structure may not fully reflect the range of clinically relevant visual impairment, with implications for policy, assessment frameworks, and service provision.

## I. Why this Paper is Relevant

This paper argues that optic nerve hypoplasia and cerebral visual impairment should not always be understood as two disconnected conditions, one belonging to the eye and the other to the brain. In some children, their difficulties may be better understood through an integrated neuro-ocular processing framework. Recognising this functional unity may support earlier identification, more coordinated intervention, and improved long-term outcomes.

Vision begins with light entering the eye and being transmitted through the optic nerve, but it is the brain, through the visual cortex and associated pathways, that constructs the experienced world of space, motion, depth, and meaning. Vision is therefore not simply a sensory input. It is also a neurological and developmental process.

Current clinical and service frameworks often divide this process into separate domains. Ophthalmology addresses the eye and optic nerve. Neurology addresses the brain. Education and social care assess functional performance. Between these domains are individuals, particularly children, whose visual processing may be unstable, effortful, and cognitively demanding. They may fall between systems that are not designed to recognise this form of impairment.

This fragmentation may be particularly evident in some children with optic nerve hypoplasia, especially where their functional difficulties resemble or overlap with cerebral visual impairment. Optic nerve hypoplasia is often approached clinically through the eye and optic nerve, while cerebral visual impairment is usually understood as a disorder of brain-based visual processing. In practice, however, both may involve disruption to an integrated visual system. The result may be difficulty constructing a stable and meaningful perceptual world.

The consequences are significant. Children whose visual systems develop with degraded, reduced, or inconsistent input may be assessed mainly through acuity-based measures. They may be mischaracterised by cognitive tests that rely heavily on visual processing, or denied appropriate support on the basis that they "see too well" or are unlikely to benefit.

These are not necessarily failures of intent, but failures of classification.

This paper therefore proposes a unifying clinical and functional framework for understanding the overlap between optic nerve hypoplasia, abnormal visual development, and cerebral visual impairment. It recognises that where early optic pathway disruption affects neurodevelopment, the resulting impairment may be expressed at the level of cerebral visual processing. This may be so whether the initial pathology is located in the eye, optic nerve, visual pathways, or brain.

The distinction between "eye disease" and "brain disease" may remain clinically and administratively useful. However, it does not always capture the functional reality of visual impairment in these cases. If applied too rigidly, it may constrain assessment, delay support, and obscure the need for coordinated ophthalmological, neurological, educational, and social intervention.

## II. What Optic Nerve Hypoplasia Does to a Developing Brain

Optic nerve hypoplasia is not merely a structural abnormality of the optic nerve. Reviews describe ONH as being associated, in some cases, with developmental, endocrine, hypothalamic, and neuroanatomical abnormalities. Garcia-Filion and Borchert, for example, describe ONH as the unifying feature of a syndrome that may include developmental and neuro-anatomical abnormalities (Garcia-Filion and Borchert, 2013). This places ONH at the boundary between ophthalmology, neurodevelopment, neurology, endocrinology, and functional vision.

ONH may also be understood as a developmental disruption affecting the quality and quantity of visual input available to the brain. From birth, and in many cases before birth, the developing visual system may therefore be organised around reduced, degraded, or incomplete visual information. The issue is not only that less information reaches the brain, but that the information available for visual development may be inconsistent or unreliable.

During early development, the visual brain is shaped by the pattern and reliability of incoming sensory input. The literature on critical-period visual development and amblyopia supports the broader principle that reduced, abnormal, or imbalanced visual input may alter visual cortical organisation and neural processing, making the proposed pathway biologically plausible (Hooks and Chen, 2007; Tailor et al., 2016). Where this input is compromised from the outset, visual development may follow a different trajectory.

This has important functional consequences. Instead of developing under conditions of clear and consistent visual input, the brain may have to

organise spatial localisation, motion perception, depth awareness, object recognition, and visual attention under conditions of reduced signal and increased uncertainty. Neuroplastic adaptation may support useful function, but it may also reflect the limits imposed by the original input.

The result may be a visual system that is less stable, less efficient, and more cognitively demanding to use. Functionally, this may present as difficulty stabilising visual scenes, distinguishing figure from background, constructing consistent spatial representations, tracking movement, or extracting meaning from complex visual environments.

This distinction is important because a child may be able to detect visual stimuli without being able to use vision reliably in everyday life. They may appear to "see" in a simple clinical or testing environment, but experience disorientation, fatigue, or overload in visually busy, fast-moving, or unfamiliar settings.

In this sense, the functional difficulty is not explained by acuity alone. It may arise from the interaction between reduced optic nerve input, altered visual development, and the cognitive effort required to construct a stable perceptual world. This is why ONH-related visual impairment should be considered not only in terms of eye structure or visual acuity, but also in terms of neurodevelopmental visual processing and real-world functional vision.

By school age, these patterns may already be established, reflecting a visual system that has developed under conditions of degraded or inconsistent input. In this sense, ONH may provide a developmental pathway through which early optic pathway disruption can contribute to higher-order visual processing difficulties consistent with those described in cerebral visual impairment.

This perspective highlights a functional continuum between optic nerve pathology and cerebral visual processing impairment, rather than a strict separation between "eye" and "brain" disorders.

## III. What Cerebral Visual Impairment Really Is (and What It Is Not)

### Current Definitions of CVI, Functional Vision, and the Relationship with ONH

Current definitions of cerebral visual impairment recognise CVI as a neurodevelopmental disorder affecting visual function and functional vision, caused by neurological damage to visual pathways and processing areas in the brain. The NIH CVI Working Group definition is important because it also recognises that anterior visual pathway disorders may coexist with CVI, provided that they do not fully explain the level or pattern of visual impairment (Chang, Merabet and the CVI Working Group, 2024). This distinction matters. Optic nerve hypoplasia is primarily an anterior visual pathway abnormality, whereas CVI is usually framed as a disorder of cerebral visual processing.

This paper does not seek to collapse that distinction. Rather, it accepts the clinical importance of distinguishing ONH from CVI, while questioning whether the developmental relationship between optic-nerve-originated degraded visual input and downstream visual processing has been fully integrated into current service thinking.

The significance of CVI literature is that it has already moved clinical understanding beyond the eye chart. The Royal College of Ophthalmologists describes CVI as an umbrella term for brain-related visual problems that may include abnormalities of acuity, contrast sensitivity, colour, ocular motility, visual field and the processing of visual input. Its practice point also identifies functional difficulties including crowding, impaired recognition, route finding and orientation, divided visual attention, figure-ground difficulty, movement perception and visually guided movement (Royal College of Ophthalmologists, 2023).

CVI is often best understood in network-level terms, rather than only as a single-lesion disorder. Although the dorsal and ventral streams may be affected in different ways, the broader functional difficulty lies in the brain's attempt to integrate, prioritise, and stabilise compromised visual input into a coherent and usable perceptual world.

For this reason, the functional approach developed within CVI can also be relevant to ONH-related impairment. Where acuity, nystagmus, or structural

findings do not fully explain everyday visual difficulty, a broader assessment of functional vision may be needed. The argument is not that ONH and CVI are identical, but that they may interact within a single developing visual pathway. ONH may begin as an ocular or anterior pathway condition, but its developmental consequences may extend into the way visual information is processed, interpreted, and used.

A further reason for thinking in pathway terms is that structural and functional changes within the visual system may not always be confined to one anatomical point. The literature on trans-synaptic degeneration suggests that damage or abnormality in one part of a connected visual pathway may be associated with changes elsewhere in that pathway. This does not prove the proposed ONH-to-functional-vision pathway, but it supports the broader principle that the eye, optic nerve, visual pathways, and brain should not always be considered as wholly separate compartments (Bosch et al., 2014).

The Royal College of Ophthalmologists' practice point is also useful because it stresses the need for flexible, individualised assessment and wider professional involvement. It draws on discussion with orthoptists, optometrists, ophthalmologists, parents, teachers of the visually impaired and third-sector organisations, and notes that referral to professionals such as neurodisability teams, occupational therapists, psychologists and QTVIs may contribute to a broader understanding of a child's visual dysfunction (Royal College of Ophthalmologists, 2023). This supports the argument that visual impairment should be understood not only through structural findings but also through how vision is used in ordinary environments.

## IV. A Developmental Model Linking Optic Nerve Hypoplasia and Cerebral Visual Impairment

**Neuroimaging evidence:**

Neuroimaging studies of congenital and acquired disorders affecting the eye, optic nerve, and visual pathways suggest that early disruption of visual input can be associated with changes in the organisation of the visual cortex and its networks. Where patterned visual signals are absent, reduced, or degraded from early life, the brain does not simply receive a weaker image. It develops under altered input conditions. Retinotopic organisation, interhemispheric connectivity, cortical thickness, and higher-order visual pathways may develop or function atypically, with lasting implications for visual processing. Importantly, such cortical changes may occur even when the primary site of disruption lies outside the visual cortex itself, including within the eye, optic nerve, or visual pathways.

Optic nerve hypoplasia does not only reduce the quantity of visual information reaching the brain. It may also affect the reliability, consistency, and structure of that input during sensitive periods of neurodevelopment. The signal transmitted through the optic nerve may be reduced, unstable, or incomplete.

For the developing visual system, these conditions are significant. Neural circuits are shaped through experience-dependent exposure to patterned sensory input. When that input is degraded or variable, the resulting organisation of visual networks may reflect those constraints. Neuroplastic adaptation may support functional use of vision, but it may also produce a system that is less stable, less efficient, and more sensitive to noise.

This process can be understood as a possible developmental cascade. Early optic pathway disruption may alter visual input during sensitive periods of development. The brain may then adapt to this input through neuroplastic reorganisation. The resulting visual system may show limitations in higher-order processing, including spatial mapping, motion processing, object recognition, and visual attention. Functionally, this may produce a profile consistent with difficulties described in cerebral visual impairment.

From a systems perspective, ONH may introduce chronic reduction, variability, or degradation into the visual signal, requiring higher-order networks to compensate. This may increase cognitive demand, perceptual instability, and variability in performance across environments.

This model helps to explain why ONH and CVI overlap in some individuals. ONH describes an early site of disruption within the visual pathway, while CVI describes functional difficulties at the

level of visual processing. The relationship between them may therefore be understood, in some cases, as developmental and continuous rather than categorically distinct.

## V. Why Acuity, Cognitive Testing, and Educational Systems Misclassify Neuro-Visual Impairment

Visual disability arising from higher-order processing differences is not always well captured by conventional assessment frameworks. Because visual acuity may be relatively preserved, and because affected children may demonstrate intact or high cognitive ability, they may be judged as "not visually impaired enough" to qualify for support.

This risks becoming a category error.

Acuity measures the resolving power of the eye. It does not assess how the brain separates figure from background, tracks motion, constructs spatial representations, stabilises visual scenes, or manages visual attention under conditions of complexity. As a result, a child may perform adequately on standard vision tests while experiencing significant functional difficulty in real-world environments.

A similar limitation applies to cognitive and educational assessment, including IQ tests. Many commonly used assessments include visually mediated tasks, such as drawing, block design, pattern recognition, visual memory, visual-motor copying, and symbol manipulation. Where visual input is unstable, degraded, or effortful to process, performance on such tasks may underestimate underlying cognitive ability.

This is not only a theoretical concern. In practice, children with neuro-visual processing difficulties may be judged as less academically able because the assessment method relies on the very visual functions that are impaired. A child may therefore be denied support not because functional need is absent, but because the test has mistaken a visual-processing constraint for a cognitive limitation.

Consequently, children with neuro-visual processing difficulties may be misclassified as having reduced ability, attention difficulties, behavioural challenges, or low engagement, when the primary constraint lies in visual processing. A child who cannot reliably stabilise a visual scene may appear confused. One who is overwhelmed by visual complexity may appear inattentive. One who experiences visual fatigue may appear disengaged.

A similar pattern may be observed in motor and physical development. Difficulties in coordination are often attributed to motor impairment, but may also reflect impaired visual-spatial processing, including reduced ability to judge distance, track moving objects, or integrate visual information with body position. These functions are associated with dorsal-stream processing and may be affected even where primary motor function is relatively intact.

The cumulative effect is significant. Children with neuro-visual processing impairment can fall outside conventional criteria for visual impairment based on acuity, appear cognitively able, and still not fit established categories of need. Consequently, access to SEND support and appropriate accommodations may be reduced, even where functional need is substantial. This pattern of difficulty may be understood within a neuro-ocular processing framework, in which visual disability arises from impaired processing rather than reduced acuity alone, and where existing assessment systems may not fully capture functional need.

## VI. Prevalence, Scale, and Public Health Implications

Neuro-visual processing difficulties arising from early visual pathway disruption are unlikely to be confined to rare or isolated conditions. They may emerge from two major contributors to childhood visual impairment: optic nerve hypoplasia and cerebral visual impairment.

ONH is a leading cause of congenital visual impairment, while CVI is widely recognised as the most common cause of visual disability in children in developed countries. These observations may be related, although they should not be treated as identical. ONH disrupts visual input from early development, while CVI describes the functional processing profile that may arise when the visual system develops under conditions of degraded or inconsistent input.

When considered within a developmental framework, this relationship suggests that the population affected by higher-order visual processing difficulties may be broader than is currently recognised. It may include children

described as having mild visual impairment, unexplained learning difficulties, coordination problems, or reduced educational attainment despite apparent ability.

Because classification systems tend to prioritise ocular structure and visual acuity over functional visual processing, many of these children may not be formally identified within existing categories of visual impairment.

This has important public health implications. Children with unrecognised neuro-visual processing differences are likely to require educational support, encounter barriers to academic progress, and face longer-term risks relating to social participation, employment, and mental health. Without an integrative framework linking early visual pathway disruption with later functional outcomes, these patterns may remain unrecognised at a systems level.

The prominence of both ONH and CVI within childhood visual impairment is unlikely to be coincidental. Although they are usually classified differently, they may be developmentally connected in some children: ONH describes an early disruption to visual input, while CVI describes functional difficulty in the brain's processing and use of visual information.

Recognising this relationship is therefore not solely a matter of diagnostic clarification. It has implications for population-level identification, service design, and resource allocation. Given the significance of ONH and CVI within childhood visual disability, the number of children affected by related visual processing difficulties may be larger than is currently captured by existing assessment and support systems.

## VII. The Institutional Gap

Current service structures reveal a gap in how visual impairment is conceptualised and managed across clinical and support systems. This gap does not arise from any single discipline, but from the way responsibilities are distributed across them.

The visual system is often addressed as if it comprised separate domains: an ocular problem for ophthalmology, and a brain-based or functional problem for neurology, education, and disability services. In practice, however, vision depends on a single integrated neuro-sensory pathway. When disruption occurs during development, its effects extend beyond the eye to influence how visual information is processed and used. This division is difficult to sustain where visual impairment arises within a connected developmental pathway. As discussed above, changes in one part of the visual pathway may be associated with changes elsewhere in that pathway. A service model that separates "eye", "brain" and "function" too rigidly may therefore fail to recognise impairments that emerge across those connected levels.

Ophthalmological assessment appropriately focuses on structural and measurable aspects of visual function, including acuity, retinal integrity, optic nerve structure, and eye movements. In cases such as optic nerve hypoplasia (ONH), this leads to accurate identification of ocular pathology. However, where no further medical or surgical intervention is indicated, ongoing functional consequences may fall outside the scope of this model.

At the same time, definitions of cerebral visual impairment (CVI) often emphasise impairment that cannot be explained by ocular or optic nerve pathology. This may create a classification tension: where optic nerve abnormalities are present, individuals may not be included within CVI frameworks, even when their functional profile is consistent with higher-order visual processing difficulties.

The result is a gap in classification and service responsibility. Individuals may present with significant functional visual impairment in educational and everyday contexts, yet not be fully captured within existing diagnostic or support pathways. This gap reflects differences in how visual impairment is defined across systems, rather than an absence of need.

This highlights the importance of approaches that recognise the continuity between early visual pathway disruption and later functional outcomes. A unified clinical framework for ONH-related functional visual impairment may therefore help avoid treating the condition as belonging exclusively to either the eye or the brain.

## VIII. Conclusion

This paper has argued that visual impairment arising from optic nerve hypoplasia (ONH) should not be understood solely in terms of ocular structure. When visual input is reduced, degraded, or unreliable during early development, the brain constructs vision under constraint. Literature on neurodevelopment, functional vision, neuroplasticity, and pathway-wide visual-system effects supports the cautious view that early optic pathway disruption may have consequences beyond acuity and optic nerve appearance alone.

The resulting functional profile may share key characteristics with cerebral visual impairment (CVI), including difficulty stabilising visual scenes, managing visual complexity, constructing spatial relationships, and using vision efficiently in real-world environments. This does not mean that ONH and CVI should be collapsed into a single diagnosis. Rather, it suggests that, in some cases, they may need to be considered within a connected developmental visual pathway.

Current systems, however, continue to prioritise visual acuity and visual field as primary measures of impairment. These measures are important, but they may not fully reflect the nature of the difficulty. Individuals may perform adequately on standard vision tests while experiencing significant functional limitations in everyday contexts, leading to under-recognition of need. This misalignment can persist across the life course, reflecting limitations in how visual impairment is defined and classified rather than deficiencies in individual services alone.

Within this context, a neurodevelopmental framework linking early optic pathway disruption with later visual processing differences provides a more coherent account of these patterns. It emphasises that visual disability may arise not only from reduced visual input, but also from how that input is processed, organised, and used by the brain. Visual impairment should therefore be understood as a distributed neurodevelopmental process rather than as a condition confined exclusively to either the eye or the brain.

Addressing the visual system as a unified neuro-sensory pathway has implications across clinical, educational, and social domains. It may support more accurate identification of need, more appropriate functional assessment, better-targeted educational support, and improved long-term outcomes.

Within this framework, reliance on visual acuity and visual field within ophthalmology-led certification systems may not fully capture functional impairment in individuals with complex visual disability. In the absence of integrated neurological and functional assessment, some neurovisual disorders are likely to remain under-recognised within current certification and support frameworks, with implications for access to appropriate assistance and disability recognition.

### Limitations

This manuscript proposes a conceptual and clinical framework rather than presenting an empirical study. It does not establish prevalence, causation, or diagnostic criteria for a new condition. The argument is based on a synthesis of lived experience, published literature, and service-level analysis, and should therefore be read as exploratory and hypothesis-generating.

The manuscript does not claim that optic nerve hypoplasia (ONH) and cerebral visual impairment (CVI) are identical conditions, nor that all individuals with ONH have CVI. Rather, it argues that, in a clinically significant group of cases, early optic pathway disruption may need to be considered in relation to later functional visual processing difficulties. Further clinical, neurodevelopmental, and neuroimaging research would be required to test this pathway model more directly.

A further limitation is that the proposed framework draws on literature from related areas, including critical-period visual development, amblyopia, functional vision, neuroplasticity, and pathway-wide effects in the visual system. The literature supports biological plausibility, but does not by itself prove the proposed ONH-to-functional-vision pathway.

The value of the manuscript lies not in statistical generalisability, but in its capacity to identify a possible gap in classification, assessment, and service response. The conclusions should therefore be read as conceptual and policy-relevant: they identify a pattern of risk and a need for more integrated ophthalmological, neurological, educational, and functional assessment.

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**Declarations**

**Ethics approval and consent to participate**

Not applicable. This manuscript is a conceptual, literature-informed policy and systems analysis. It does not involve the collection of new research data, clinical intervention, or research involving human participants.

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**Data availability**

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