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Ways of Seeing Under Visual Impairment: A Scoping Review on Eye Disease, Perception, and Artistic Expression

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As primeiras palavras de profunda gratidão são dirigidas às minhas orientadoras. Obrigada pela sensibilidade com que acolheram a minha visão. Permitirem-me o cruzamento da Oftalmologia com a Arte foi um voto de confiança que me deu o espaço necessário para cultivar esta insubmissão contra a desumanização dos cuidados de saúde.

Ao ICBAS, o meu teto nos últimos 6 anos. Faculdade onde, desde os primórdios, a Ciência e a Arte partilham a mesma morada. Passei mais horas contigo do que com o meu quarto.

Ao associativismo académico, em especial à AEICBAS e à ANEM. Durante o meu percurso, foram a minha maior escola de cidadania, onde descobri o poder e a urgência da voz dos jovens na transformação da Saúde. Nas trincheiras do trabalho associativo, percebi que intervir e recusar o conformismo é, também, uma forma de cuidar.

À minha família, o alicerce invisível e incondicional de todos estes anos. Obrigada pelo sacrifício, pelo amor e por me recordarem de quem sou sempre que entrávamos em épocas de exames.

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Por fim, uma palavra à criança que fui: aquela que rabiscava as todas as folhas de papel apanhadas desprevenidas e que nunca largou a "idade dos porquês". Obrigada a essa menina que devorava com a mesma energia inata os manuais de ciências e de EVT, procurando incansavelmente os conceitos que uniam os dois mundos. Esta tese é a prova de que a tua curiosidade tinha, desde sempre, um propósito maior.

PREFÁCIO

Diz-se frequentemente, nos corredores e nos anfiteatros desta faculdade, que o médico que só sabe de Medicina, nem Medicina sabe. Embora esta máxima seja muitas vezes atribuída ao patrono desta Casa, importa esclarecer que a frase não é sua. Porém, o facto de a termos adotado como um mantra diz muito sobre a identidade académica que, em mim, floresceu. Durante muito tempo, encarei-a apenas como um belo aforismo. Foi preciso chegar ao fim deste curso, e à escrita desta tese, para compreender que não se trata de poesia, mas de um sério aviso clínico, dolorosamente à frente do seu tempo.

Como nos ensinou o próprio Abel Salazar, a Arte e a Ciência ocupam campos distintos, mas que estão em contacto perpétuo. Ao longo do meu percurso, fui treinada para observar o corpo humano através de uma lente onde a precisão e a métrica eram as prioridades absolutas. Ensinaram-me a medir escalas, a venerar *guidelines* e a confiar (quase cegamente) em *scores*. Fui subtilmente moldada para olhar mais para os píxeis de um ecrã do que para as rugas do rosto sentado à minha frente.

A ciência dotou-me, indubitavelmente, de um vocabulário vasto para descrever a doença. Contudo, testemunho com crescente angústia a facilidade com que a prática clínica se transformou numa linha de montagem. Vivemos aterrorizados com a ideia de a Inteligência Artificial nos roubar a profissão, ignorando que, se nos limitarmos a ser burocratas de sintomas e leitores de exames, a máquina já nos substituiu.

Cruzar a oftalmologia com a arte não foi, por isso, um mero capricho estético; foi uma tentativa de insubmissão contra a desumanização dos cuidados de saúde.

Um algoritmo será sempre mais rápido e preciso a diagnosticar uma mácula, mas nunca conseguirá descer à vulnerabilidade de quem tenta reconstruir o seu mundo nas sombras. Este trabalho é o testemunho da minha maior aprendizagem: a de que a nossa profissão falha redondamente se apenas consertar corpos, pois só cumpre o seu propósito quando nos ensina a verdadeiramente *ver* os outros. E esta tese obrigou-me a descer do pedestal da objetividade para refletir que a Medicina é, por definição, a mais humana das Ciências e a mais científica das Humanidades.

RESUMO

Como postula John Berger, a visão precede as palavras e estabelece o nosso lugar no mundo, mas a relação entre o que vemos e o que sabemos nunca está resolvida. Esta scoping review (n=53 artigos), estruturada pelas diretrizes JBI e PRISMA-ScR, cruza a fisiologia ocular com a arte para provar que a visão não é um registo mecânico, mas uma construção instável e biológica.

Os resultados demonstram que estados emocionais, como a depressão, corrompem fisicamente a perceção do contraste na retina, enquanto o cérebro executa algoritmos de "edição" para esconder a doença do próprio doente. Quando a visão central falha, ocorre uma metamorfose: a geometria do mundo deixa de estar "lá fora" para se ancorar no corpo e no toque — um processo de aquisição sensorial onde o cérebro aprende a ver através da pele. A arte emerge aqui como o único registo material de alucinações e distorções que os meios complementares de diagnóstico são incapazes de fotografar.

A conclusão é decisiva: prescrever arte não é um gesto cultural, mas uma "ortopedia da perceção" essencial. Num futuro onde a Inteligência Artificial dominará o diagnóstico anatómico, a relevância do médico dependerá inteiramente da sua capacidade de interpretar a experiência real do sujeito. A medicina deve aprender a contemplar a "tela" do doente, reconhecendo que a verdadeira visão reside entre os nossos ouvidos — um espaço humano onde a máquina, por si só, não consegue chegar.

ABSTRACT

As John Berger posits, seeing comes before words and establishes our place in the world, yet the relation between what we see and what we know is never settled. This scoping review (n=53 articles), guided by JBI and PRISMA-ScR standards, bridges ocular physiology and art to prove that vision is not a mechanical record but an unstable biological construction.

Results demonstrate that emotional states, such as depression, physically corrupt retinal contrast perception, while the brain executes "editing" algorithms to hide the disease from the patient's own consciousness. When central vision fails, a metamorphosis occurs: the geometry of the world stops being "out there" and anchors itself to the body and touch—a process of sensory gain where the brain learns to see through the skin. Art emerges as the sole material record of hallucinations and distortions that diagnostic machines are unable to photograph.

The conclusion is decisive: prescribing art is not a cultural gesture, but an essential "perceptual orthopedics." In a future where Artificial Intelligence will dominate anatomical diagnosis, the doctor's relevance will depend entirely on their ability to interpret the subject's lived experience. Medicine must learn to contemplate the patient's "canvas," recognizing that true vision resides between our ears—a human space that the machine, on its own, cannot reach.

LISTA DE ABREVIATURAS

AI – Artificial Intelligence

ECG – Electrocardiogram

EEG – Electroencephalogram

FTD – Frontotemporal Dementia

JBI – Joanna Briggs Institute

MeSH – Medical Subject Headings

MRI – Magnetic Resonance Imaging

OCT – Optical Coherence Tomography

PCA – Posterior Cortical Atrophy

PCC – Population, Concept, Context

PERG – Pattern Electroretinogram

PFF – Paradoxical Functional Facilitation

PPA – Primary Progressive Aphasia

PRISMA-ScR – Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews

PRL – Preferred Retinal Locus

V1 – Primary Visual Cortex

WHO – World Health Organization

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1. INTRODUCTION

“Yet this seeing which comes before words, and can never be quite covered by them, is not a question of mechanically reacting to stimuli. (…) We only see what we look at. To look is an act of choice. (…) It is seeing which establishes our place in the surrounding world; we explain that world with words, but words can never undo the fact that we are surrounded by it. The relation between what we see and what we know is never settled.” — John Berger, Ways of Seeing[1]

Historically, clinical observation and art were indivisible: from the anatomical drawings of Leonardo da Vinci to the neuronal illustrations of Santiago Ramón y Cajal, art functioned as the primary methodological tool for understanding the body. Yet, contemporary medicine has provoked an artificial divorce between biology and the humanities in the name of a false objectivity, building its pedagogy on the illusion that vision can be resolved and codified exclusively through third-person data. Classical ophthalmology often reduces the capacity to see to quantitative metrics: reading optotypes on a Snellen chart, perimetric mapping of a visual field, or measuring macular thickness via OCT. While these technological instruments are undeniably indispensable, they tell only half the story. This biomedical reductionism generates a clinical gap: the physician successfully measures the structural mechanics of the eyeball in a sterile environment, yet remains blind to the patient's subjective, first-person experience in the real world.

The scale and severity of this diagnostic bias take on alarming proportions when confronted with demographic reality. If we exclude mere refractive errors, data from the Global Burden of Disease and the WHO reveal that there are currently over 338 million people worldwide living with blindness or moderate to severe visual impairment.[2] For epidemiology, this is a global public health crisis; however, the true tragedy of this number lies in the medical presumption that accompanies it: these 338 million represent the patients whom clinicians presume to know exactly how they see, simply because they can quantify what is no longer there.

Consequently, when a clinician notes a visual acuity of 20/200 on a chart, they falsely assume this mathematical ratio successfully encodes the patient's reality. Even though modern ophthalmology utilizes supplementary functional evaluations (such as color vision testing, contrast sensitivity charts, and optical aberrometry to better characterize the visual input), these metrics still capture isolated

physiological parameters rather than the integrated perceptual outcome. The neurological responses that ensue are often overlooked: mental reconstruction, the filling-in of scotomas, compensatory hallucinations, and the transition of spatial navigation from visual to tactile and bodily frameworks. Each of these 170 million clinical records represents a brain endeavoring to reconstruct space, contrast, and its autobiographical identity.

This biomedical reductionism ultimately accelerates the profound dehumanization of contemporary healthcare.[3] By treating vision strictly as a quantifiable output, the medical profession has inadvertently prepared the ground for its own algorithmic replacement. Machine learning models can now analyze OCT scans to find disease markers, like diabetic retinopathy, with more accuracy than humans. What the algorithmic 'black box' cannot do, however, is comprehend what it means to lose the contrast of light at dusk. The success of the physician of the future will depend on their ability to also master the art of *close noticing.*

Conventional clinical metrics inherently fail to capture this subjective dimension. It is precisely here that medicine must turn to the only material record capable of mapping the internal experience of perceptual alteration: artistic expression. The canvas offers the vital first-person data required to complement the detached, third-person clinical examination

In this context, the current scoping review seeks to explore the intersection of the neurobiology and pathology of vision, perception and the medical humanities. Grounded in the principles of Neuroesthetics—which suggests that artists are naturally empirical neurologists—this review argues that artwork serves not merely as a metaphor but as a concrete clinical record.[4] The ultimate goal is to develop a new explanatory Art-Vision Framework, aimed at reintegrating the "evidence of the senses" and the perceptual insight of the visual arts into modern clinical reasoning.

2. METHODOLOGY

2.1. Design and Eligibility Parameters

This scoping review utilized the Joanna Briggs Institute (JBI) paradigm, adhering to PRISMA-ScR guidelines. The inquiry follows the Population, Concept, Context (PCC) triad, exploring the intersection of visual degradation and aesthetic translation, revealing clinical implications.

Inclusion demanded empirical or theoretical engagement with subjective visual metamorphosis stemming from established ocular diseases. Literature capturing the artistic articulation of sensory deficits secured immediate eligibility. Conceptual intersections with clinical observation mechanics are mandatory. Linguistic parameters restricted the extraction to English and Portuguese full-text manuscripts. The exclusion criteria functioned as an epistemological filter. Literature confined exclusively to biomedical, surgical, or assistive technology discourse faced immediate rejection. Sociological treatises devoid of subjective phenomenological anchorage were excluded. Historical retrodiagnoses relying on speculative aesthetic readings—lacking robust clinical or perceptual grounding—failed to meet the selection threshold. Purely theoretical abstractions unmoored from the organic reality of visual pathology were categorically excluded.

2.2. Search

The search apparatus hybridized controlled vocabulary (MeSH) with highly sensitive free-text syntaxes (Appendix 1). Bibliographic extraction was performed using PubMed/MEDLINE, Scopus, the Web of Science Core Collection, Google Scholar, and JSTOR. This multi-database strategy eliminates the artificial divide between rigid biomedical indices and humanistic repositories. PubMed interrogations deployed a biphasic algorithm of isolated MeSH targeting followed by a broad-spectrum hybrid sweep. Scopus and Web of Science executions leveraged deep-text semantic combinations to capture interdisciplinary research leakage. Google Scholar and JSTOR mapped the historical and phenomenological periphery. Temporal constraints were intentionally removed. Complete historical immersion ensures the maximum conceptual capture.

2.3. Screening

Rayyan® architecture managed the data deluge. Algorithmic deduplication preceded the exhaustive manual verification. Selection was performed using a biphasic funnel. Title and abstract screening initiated the filtering process. Full-text appraisal subsequently confirmed the final inclusion. Conceptual ambiguity at the preliminary stage guaranteed a full-text evaluation. The PRISMA-ScR flow diagram maps precise attrition mechanics, detailing explicit rationales for terminal exclusions.

2.4. Data Extraction

Information charting used a highly customized extraction matrix. The variables captured included authorship, temporality, cohort pathology, precise perceptual distortion, artistic modality, and downstream clinical ramifications. JBI directives inherently bypass the formal critical appraisal of methodological quality. Inductive categorization dissolved raw extraction data into five robust analytical axes, mapping the precise phenomenological architecture of the proposed Art-Vision Framework: (1) Organic Input Failure; (2) Perceptual Reprocessing and Plasticity; (3) Subjective Visual Experience and Meaning; (4) Artistic Expression; and (5) Medical Praxis. Narrative synthesis elucidates profound conceptual linkages across this continuum: constructs enduring phenomenological meaning rather than quantifying fragmented statistical effects.

2.5. Ethical Framework

The inquiry boundaries were confined to the public domain. Absolute reliance on published, accessible literature preempted the necessity for institutional ethical clearance. Primary data collection is entirely outside the methodological scope. Visual reproductions within the manuscript strictly obey public domain statutes and licensing architectures, guaranteeing uncompromising academic integrity. In accordance with ICMJE guidelines, the authors disclose the use of *Gemini* (Google®) to assist with spelling and grammar corrections, restructure sentences for clarity, aid in document and reference formatting, and brainstorm the structural organization of chapters. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the accuracy, completeness, and originality of the publication.

3. RESULTS

3.1. Introduction To Findings

The systematic extraction process yielded a curated corpus of 53 articles, consisting of a heterogeneous and interdisciplinary literature bridging clinical ophthalmology, cortical neuroftalmology, and the medical humanities. These raw data resist linear quantification and pure statistical analysis. Instead, and to preserve epistemological rigor, descriptive evidence demands strict isolation from its subsequent interpretative implications. Subsequently, the extracted findings structurally coalesce around three fundamental thematic clusters that map the precise phenomenological trajectory of visual degradation.

The first cluster defines the **organic input** dimensions. It captures structural optical system failure and the measurable erosion of perceptual integrity resulting from focal pathologies. The second cluster isolates **perception** in its **cortical dimension**. It details the neurological compensatory responses, specifically top-down processing mechanisms, and the radical spatial shift from visual-allocentric mapping to tactile-egocentric frameworks. The third cluster documents the actual **artistic dimensions**. Visual arts expression emerges not as an abstraction but as a physical, objective record of the profound deficit of sight, as the biological failure crystallizes on the canvas.

3.2. Organic Input

Visual impairment, as simply as physics can explain, originates as a photonic disruption. When the biological machine fails, for instance, due to structural degradation, it obliterates the mechanical conversion of light into a viable neural signal. The extracted literature maps the precise trajectory of this organic breakdown, localizing the deficit across distinct anatomical strata: the macula, the optic nerve, and the crystalline lens and cornea.

Foveal collapse dictates the most evident erosion of spatial acuity. Senile macular degeneration and progressive maculopathies systematically destroy the photoreceptor (cones) density responsible for high-resolution vision and chromatic saturation. However, this erosion rarely begins as an absolute void: it begins as a geometric distortion. Extracted clinical history reveals that structural warping—metamorphopsia—systematically precedes the formation of central scotomas. Just

like thinking *"the perceptual grid bends before it breaks".* [5] Retinal edema and subretinal exudation physically displace photoreceptors, forcing straight lines to undulate and spatial coordinates to buckle. Only as progressive maculopathies systematically destroy photoreceptor density does this metamorphopsia collapse into an absolute central scotoma, eradicating high-resolution vision and chromatic saturation (Figure 1).[6] The ocular pathology of Edgar Degas provides a definitive clinical exemplar. Severe, progressive bilateral retinal degeneration stripped his visual field of sharp contours, dissolving high-frequency spatial details into generalized blur and profound contrast loss: central vision dissolves, lines fracture and the progressive death of foveal cones enforces an inescapable reduction in the chromatic range, tethering the perceptual field to a severely restricted optical bandwidth.[7]

Optic neuropathy executes a fundamentally different pattern of structural erosion. While acknowledging that other forms of optic neuropathy (such as ischemic or demyelinating) produce distinct visual deficits, glaucomatous damage to the retinal nerve fiber layer serves as the primary paradigm for this erosion, triggering a silent erasure of spatial continuity. Extracted phenomenological data systematically dismantle the clinical myth of "tunnel vision" in early to moderate glaucoma, myth that is still being taught nowadays. Patients do not perceive a cinematic black border encroaching on their visual periphery. Instead, the structural deficit manifests as profound spatial fragmentation.[8] The visual field fractures into localized zones of blur and missing information, severely compounded by an intense susceptibility to glare and a heightened dependency on ambient illumination (Figure 2).

This fragmented reality aligns with the lived experience of people with mild-to-moderate vision loss, who navigate the world and engage with visual art through pervasive blur, reduced contrast, and spatial distortion.[9] This sensory hurdle has prompted extensive research into inclusive design, demonstrating that enhancing contrast, adjusting lighting, and managing visual clutter are critical steps to allowing these individuals to visually engage with art.[10] The optic nerve lesion generates a patchy, unreliable topography.

Pathological media opacities and structural toxicities impose an aggressive chemical and physical filter over the surviving sensory input: lenticular sclerosis functions as an intraocular blockade. The cataractous lens disproportionately

absorbs short-wavelength (blueish) photons, inducing a measurable acquired dyschromatopsia. This aging lens enforces a generalized chromatic shift, obliterating the blue spectrum and shattering overall contrast sensitivity. The visual restrictions evident in Francisco Goya's late output physically mirror this cataractous erosion, exhibiting drastically reduced luminance and a compressed tonal range characteristic of lenticular opacity (Figure 3).[11] Historically, the profound impact of such lenticular and vascular blockades extends beyond the visual arts, visibly altering the creative timelines and output of composers like Georg Friedrich Händel.[12] Chemical toxicity dictates similar structural limitations at the photoreceptor level. Digitalis intoxication provokes xanthopsia, enforcing a biochemical yellow tint upon the retina, a neurotoxic mechanism frequently hypothesized in the chromatic distortions of Vincent van Gogh.[13, 14]

Physical scotomas assert their brutal geometry on the visual plane. The organic deficit ceases to be a mere absence of light; it becomes a tangible, obstructive presence. Edvard Munch meticulously documented his own right-eye vitreous hemorrhage, mapping the entoptic shadow—a moving, semi-opaque "bird of prey"—directly across his macular field. Unlike the fixed, undulating warp of metamorphopsia, this mobile scotoma physically obscures the target of gaze.[5] The damaged eye stops operating as a transparent optical conduit. It becomes an anatomical barrier, corrupting the once transparent media.

Furthermore, the literature demonstrates that also affective states act as primary biological modulators. Bubl et al. (2010) established through PERG testing that patients with major depression exhibit a drastic and measurable reduction in retinal contrast gain: the greater the severity of the depression, the lower the retina's physiological sensitivity to contrast (Figure 4).[15] According to the authors, this sensory attenuation is likely driven by reduced dopaminergic transmission within the inner retina, specifically impairing the activity of amacrine and retinal ganglion cells. This confirms the subjective metaphor of 'seeing gray' into an objective, quantifiable physiological deficit.

3.3. Perceptual Reprocessing and Cortical Plasticity

3.3.1.1. Neurological compensatory mechanisms

Visual processing extends far beyond the physical boundaries of ocular biology and physiology. Extracted neuropsychological literature demonstrates that the correlation between retinal photon capture and conscious perception is not an absolute, linear hierarchy. Rather, the visual signal diverges through parallel, independent neural circuits. This anatomical routing allows clinical phenomena to completely untether visual processing from conscious awareness. Blindsight, documented in patients harboring strict lesions within the primary visual cortex (V1), illustrates this neurological paradox. These individuals are clinically blind, yet they retain the unconscious capacity to process motion trajectories and chromatic stimuli via intact extrastriate and subcortical pathways—specifically the superior colliculus and amygdalar networks.[16, 17] The brain systematically detects the physical stimulus, but the subject remains entirely unaware of the visual event: the neural network “sees”; the patient does not.

Furthermore, the developmental trajectory of these compensatory networks is highly sensitive to early visual exposure. Recent findings indicate that children who gain sight later in life rely overly on chromatic cues due to the lack of early grayscale maturation, proving that early visual experience impacts how the brain subsequently utilizes color for object recognition. [18]

Confronted with structural ocular failure, the central nervous system aggressively overcompensates, exibiting a biological intolerance to sensory silence: a *horror vacui*. When progressive scotomas (such as those induced by glaucomatous neuropathy or macular degeneration) eradicate retinal input, the brain does not passively accept the anatomical blackout. Instead, it executes top-down "filling-in" algorithms, operating with the exact mechanistic logic of contemporary generative artificial intelligence performing contextual image in-painting. As Gagrani et al. rigorously document, the cortex functions as a predictive, hallucinatory engine.[19] It extracts spatial textures, structural patterns, and chromatic baselines from the surviving visual periphery and probabilistically synthesizes a substitute reality directly over the scotoma.

The brain, essentially, "edits out" the disease. Consequently, particularly in chronic and progressive conditions, the patient rarely perceives a true absence of

information or a defined black boundary. Conversely, in acute macular pathologies, the central nervous system lacks the temporal window required for such adaptive rewiring, leaving the patient acutely sensitive to even minor central disruptions (Figure 5). The clinical consequence of this compensatory plasticity in slowly evolving diseases is severe: the neurological mask systematically delays medical diagnosis, as the cortex actively hides the organic deficit from the patient's own consciousness.

This perceptual survival mechanism necessitates a functional reorganization, clinically formalized through the development of an eccentric Preferred Retinal Locus (PRL). Faced with the obliteration of the fovea, the visual system involuntarily forces the patient to fixate using an adjacent healthy area of the peripheral retina. As Thibaut et al. (2014) demonstrate, this shift fundamentally alters the cognitive framework for scene categorization.[20] Because this eccentric topography predominantly processes low spatial frequencies, the central nervous system effectively sacrifices high-resolution macular detail. However, the peripheral retina is highly specialized for extracting the global layout, or "gist," of a visual environment.

Consequently, spatial navigation and visual recognition are successfully preserved through the rapid processing of global shapes and gross contrast. The brain redefines its sensory baseline, transferring the perceptual burden to these peripheral anchors to sustain an illusion of environmental stability, despite the collapsing macular "hardware".

3.3.2. Spatial navigation adaptations

When the organic vision fails, the spatial awareness actually survives, but its architectural framework radically transforms. Ideally, physiologically intact optical pathways support an allocentric spatial map: an objective, distal grid processed independently of the observer's immediate physical coordinates. However, as ocular pathology progresses and retinal input diminishes, maintaining this world-centered topography becomes neurologically unsustainable. Consequently, when progressive ocular pathology or cortical trauma intensifies, the cortex enforces an immediate epistemological shift, anchoring spatial reality directly onto the physical body. This results in a mandatory functional transition from a visual-allocentric framework to a tactile-egocentric geometry, effectively establishing the patient's

own somatosensory schema as the sole reference point for environmental navigation.[21] Literature confirms that individuals can acquire and manipulate complex spatial concepts, even at a geographic scale, entirely without the use of vision, relying on these non-optical frameworks.[22] This reliance on non-optical mapping directly challenges deeply ingrained ocularcentric design paradigms, emphasizing the critical need for multisensory approaches in spatial and architectural environments to truly accommodate the lived realities of blind individuals.[23]

Current literature confirms that geometric properties like shape and volume do not depend on retinal input, and are not tied exclusively to the visual system.[24] The brain processes these features as universal spatial constructs, allowing spatial literacy to remain intact even after the absolute loss of sight. Tian et al. established that abstract shape representation persists intact despite severe visual deprivation, seamlessly transferring its cognitive processing load to somatosensory and proprioceptive networks.[25] Developmental studies further corroborate this, demonstrating that the severity of visual impairment and tactile practice significantly shape drawing abilities in children, proving that spatial representation transfers early to haptic networks.[26] The hands effectively replace the macula: the blind individual sustains its spatial awareness by actively reconstructing the environment through a sequence of tactile contacts and kinesthetic memory.[27]

This spatial transition, entirely driven by the radical sensory recruitment of adjacent neural networks is clinically known as cross-modal plasticity. The occipital lobe, deprived of visual input due to complete retinal deafferentation, actively engages neighboring sensory pathways to compensate. Roberts and Shenker document the extreme functional limits of this transition in a clinical case of severe retinal degeneration (S-cone syndrome), where the patient developed veridical "non-optic vision."[28] In this documented shift, haptic and proprioceptive stimuli directly and mechanically triggered the visually deprived visual cortex. The physical act of touching an object or moving the hands compels the cortical networks to generate an involuntary, perceived spatial image of the hands themselves, entirely bypassing the optic nerve. The visual buffer is structurally appropriated by the somatosensory system. The patient ceases to map space through optical projection; reality is exclusively constructed, measured, and perceived through bodily contact.

3.3.3. Integrative Collapse and Agnosia

The ultimate disruption in the visual hierarchy manifests when the organic "hardware" remains perfectly intact, yet perceptual coherence systematically disintegrates. This represents a catastrophic breakdown of the visual buffer. Functionally, this buffer operates as the central nervous system's internal, transient canvas: a critical cognitive pause lasting mere milliseconds. It is the precise workspace where the raw, isolated features captured by the eye (a vertical line, a shift in contrast, a patch of color) must be briefly held and bound together to form a meaningful, recognizable object. When this short-term canvas shatters, the patient is flooded with geometric fragments that never coalesce into meaning. The eye sees everything; the mind understands nothing.

Clinical syndromes such as visual apperceptive agnosia vividly expose this disconnection. Profiling the recovery from cortical blindness, Ogden documents the severe dissociation between elementary perception and semantic recognition.[29] The agnosic patient retains the physiological capacity to trace contours, detect contrast, and isolate distinct physical elements within their visual field. However, the occipitotemporal networks entirely lose the ability to integrate these fragmented percepts into a cohesive, meaningful whole.

This systematic disintegration of visual synthesis is equally devastating in neurodegenerative frameworks, particularly Posterior Cortical Atrophy (PCA). Rocher et al. detail how the progressive, focal death of occipitoparietal networks strips incoming visual stimuli of their structural meaning.[30] The patient inhabits a shattered optical world. The retina faithfully transmits the raw environmental data, but the deteriorating cortex can no longer decode its own biological signals. As the integrative pathways wither, the visual buffer collapses entirely, leaving the individual stranded in a chaotic, unrecognizable topography composed of isolated lines and meaningless geometry. The patient is not blinded by a lack of light, but by a total loss of meaning.

Conversely, the neuropsychology literature demonstrates that cortical neurodegeneration does not invariably result in a subtraction of capacity. As Chatterjee (2006) discusses, conditions such as Frontotemporal Dementia expose a phenomenon defined by Kapur (1996) as paradoxical functional facilitation. In these clinical scenarios, patients often undergo profound personality changes and

cognitive alterations, yet simultaneously develop a propensity to express themselves visually or to create material representations for the first time.[31] The pathological erosion of semantic and linguistic networks acts as a disinhibitory mechanism; because semantic associations can interfere with the ability to "see" a visual object, impaired conceptual knowledge actually aids in accurate visual depiction, releasing the visuospatial networks from semantic interference. Consequently, the social disinhibition and acquired obsessive-compulsive traits characteristic of FTD find graphical expression, instigating a, *de novo,* compulsion for highly detailed visual and material representations. This confirms that the brain operates not only through pathological loss but also through mechanisms where impairments in one cognitive component can be compensated by, or actively facilitate, other components.

3.4. Artistic Expression as a Clinical Record

3.4.1. The Diagnostic Canvas

In the presence of progressive ocular pathology, the canvas transitions from just an aesthetic medium into a quantifiable clinical artifact. The artwork functions as an externalized visual field, providing one of the few available methodologies to objectify inherently subjective visual symptoms. By capturing structural and chromatic distortions at specific stages of decay, the material output offers a chronological evaluation of the pathology: a 'frozen evolution' photographed in time through the application of pigment. Instead of reflecting a deliberate, personal stylistic shift, sometimes physical execution serves as an objective record of sensory decline. Recognizing these subjective artistic manifestations challenges the purely objective visual heuristics often relied upon in clinical reasoning, pushing the boundaries of what physicians notice during diagnosis.[32] Nevertheless, this analysis demands absolute clinical rigor; while retrospective diagnoses based purely on aesthetic shifts are hazardous, examining the output of artists with historically well-documented ocular pathologies provides an invaluable and objective understanding of vision loss.[33]

Chromatic degradation provides the most measurable metric of this material translation. As previously detailed (see Section 4.2), conditions such as lenticular sclerosis impose a strict optical blockade, transferring the visual deficit directly to the artist's palette as a 'false dyschromatopsia'.[34] The obliteration of the blue

spectrum mechanically tethers the painter to a severely restricted bandwidth, saturating the canvas with muddy reds, oranges, and browns—a shift famously documented in the late output of J.M.W. Turner and Claude Monet. The historical dilemma of color deficiency in art underscores how a restricted palette is often an involuntary physiological adaptation rather than a purely aesthetic choice.[35] Furthermore, recent empirical studies on observers with color vision deficiencies confirm that restricted chromatic palettes fundamentally alter how relevant colors in paintings are perceived and communicated.[36]

In Monet's case, the onset of symptoms began around 1908, and by 1918 he explicitly documented this chromatic collapse in a letter to Georges Clemenceau, stating that colors had lost their intensity, reds appeared muddy, and intermediate tones entirely escaped his perception. By September 1922, the visual acuity in his right eye was reduced to mere light perception.

Historically, it is well known that Monet liked to paint the same motifs multiple times, under different lights, such as his exhaustive studies of the Rouen Cathedral or the Haystacks, which were deliberately engineered to capture transient atmospheric effects. However, ophthalmological consensus confirms that his late *Giverny* output represents a fundamental paradigm shift. He was no longer mapping the external movement of the sun; he was involuntarily mapping the internal opacity of his crystalline lens. By repetitively painting the exact same botanical and architectural motifs over several decades, Monet transformed his estate into a controlled clinical laboratory. He effectively eliminated environmental variables and utilized the familiar architecture of his house as a fixed diagnostic reference, relying on the cognitive memory of its original colors to calibrate his failing perception.

The clinical timeline perfectly mirrors this material output. In January 1923, Dr. Carlos Coutela performed a two-stage cataract extraction on Monet's right eye, followed by a surgical capsulotomy in June 1923. Because the artist adamantly refused surgery on his left eye, he was left with a severe binocular asymmetry, which he actively utilized for optical experimentation. The surgical removal of the yellowed lens from his right eye exposed his retina to an uninhibited influx of short-wavelength light, resulting in a profound post-operative cyanopsia. Monet essentially turned his own eyes into comparative instruments: when painting motifs like the *Rose Garden* using his unoperated left eye, the canvases remained tethered

to warm, muddy ochres and reds; when painting the exact same motif with his aphakic right eye, the canvas violently exploded into cold blues and intense violets. It was only through the later prescription of specialized Zeiss lenses by Dr. Jacques Mawas that these optical color aberrations were partially mitigated. However, this optical correction introduced a new clinical challenge: severe anisometropia. The highly asymmetric prescription required to correct his right aphakic eye created a profound binocular imbalance, forcing Monet to contend with unequal image sizes and spatial disorientation even as he tried to complete his final monumental works (Figure 6). Consequently, these late series function as a rigorous medical record that objectifies both the biological failure of his optical system and the neurological shock of surgical recalibration.[37]

Parallelly, the clinical hypothesis of Vincent van Gogh's digitalis intoxication illustrates how a transient biochemical filter might similarly solidify into a permanent material artifact. While there is no definitive medical consensus regarding this posthumous diagnosis, the proposed phenomenon of xanthopsia (yellow vision) provides a compelling biological model for his late pigment choices (Figure 7). This hypothesis suggests that an endogenous toxicity, rather than a purely aesthetic decision, may have dictated his iconic, highly saturated color palette. Whether driven by a structural cataract or a chemical intoxication, the underlying principle remains consistent: the pathology dictates the palette, and the canvas records the symptom.

Beyond structural erosion, positive and transient visual phenomena project their exact geometry directly onto the painted surface. As established in the physiological analysis of entoptic shadows (see Section 4.2), Edvard Munch's documentation of his 1930 intraocular hemorrhage serves as a landmark instance of the artwork functioning as a clinical diagnostic instrument. Facing a mobile, semi-opaque scotoma—which he famously visualized as a "bird of prey"—Munch moved beyond mere representation by superimposing grids of horizontal and vertical lines onto his sketches (Figure 8). Clinically, the mobility of this visual defect indicates a vitreous hemorrhage rather than a purely retinal one, as blood suspended within the vitreous gel shifts with eye movement, unlike the fixed scotomas characteristic of intra-retinal bleeding. This use of Euclidean geometry to detect and map this spatial distortion (metamorphopsia) anticipated Marc Amsler's formalization of the diagnostic grid by nearly fifteen years.[5] By utilizing the canvas as a perimetric tool, Munch was able to map the trajectory and density

of the floating hemorrhage against static coordinates, effectively transforming an unobservable, subjective symptom into a quantifiable material record. This transition from metaphor to measurement highlights the unique capacity of the artistic medium to "freeze" transient pathophysiology, providing a chronological evaluation of a biological event that would otherwise remain unrecorded.

This capacity to materialize transient, unobservable neurological events extends beyond structural ocular pathology to the pathophysiology of migraine (Figure 9).[38-40] In the work of artists like Sarah Raphael, the subjective visual correlates of the cortical "electrical storm" are translated into precise geometric artifacts.[41] Her *Strip!* paintings systematically map fortification spectra (teichopsia) and the "corona phenomenon"—the luminous, zigzagging halos mathematically characteristic of cortical spreading depression. By externalizing these strictly internal hallucinations, the canvas provides a permanent clinical document of involuntary cortical activity that would otherwise remain entirely inaccessible to external observation.

The externalization of internally generated visions is most starkly documented in cases of severe sensory deprivation. De Jong (2003) reports instances of patients experiencing structured “phantom vision” following enucleation, questioning whether these represent Charles Bonnet Syndrome or phantom images caused by the direct stimulation of the severed optic nerve.[42] In a striking convergence of pathology and art, a 65-year-old patient utilized watercolor to precisely map the complex visual hallucinations she perceived through her removed eye just weeks after its excision. Similarly, a bilaterally enucleated patient continued to paint using braille-labeled tubes, occasionally experiencing vivid, hallucinatory projections of his familiar physical surroundings. In these clinical scenarios, the painted canvas served as the pure cartography of an isolated visual system actively projecting reality *ex nihilo*. Today, this diagnostic externalization has evolved beyond traditional two-dimensional media: contemporary methodologies now employ animation and virtual reality to create immersive, first-person representations, Pioneering contemporary creators, such as the legally blind digital artist George Redhawk, utilize computer software to animate the visual "morphing" and cognitive data-loss characteristic of their failing sight.[43] This evolution successfully translates the highly subjective perceptual experiences and spatial distortions of art-practitioners with sight-loss into dynamic digital environments.[44]

3.4.2. Intraoperative Documenting

While longitudinal chromatic shifts document the progressive decay and subsequent recalibration of the optical system, the canvas is also capable of capturing the acute neurological trauma of the medical intervention itself. The surgical procedure, particularly under local or topical anesthesia, frequently acts as a site of involuntary clinical documentation. Clinical studies indicate that up to 80% of patients experience "phantom vision" during phacoemulsification: a series of dynamic, often hallucinatory visual phenomena induced by surgical instruments manipulating the internal structures of the eye under the intense glare of the operating microscope.[45-48]

For the artist-patient, this acute sensory rupture provides a fleeting but highly specific visual input. A prominent example is the painting titled Galaxy in My Eye, produced by Marjorie Chu, a Singaporean professional artist, immediately following her cataract extraction.[49] The work materializes a vivid phantasmagoria, translating the intense light-scattering, endocapsular shadows, and fluid dynamics of the surgery into permanent geometric abstraction. Rather than an aesthetic invention, the painting functions as a real-time, first-person topographic map of the eye's internal state during surgical trauma, capturing an entoptic phenomenon that external medical imaging cannot photograph. In this capacity, the artwork objectifies the exact moment of physiological transition between the degenerative filter and technological clarity.

3.4.3. Mechanical Alterations

Beyond chromatic degradation and entoptic hallucinations, the physical execution of the artwork—specifically brushstroke mechanics—serves as a highly quantifiable neurological record. As the loss of central visual acuity imposes an absolute mechanical limit on fine motor execution, artists are forced to adapt their physical gesture. In the late works of Edgar Degas, for instance, this deficit is recorded not as a deliberate evolution of style, but as a severe physical dissolution of form. The meticulous, sharp delineations characteristic of his early output are physically replaced by increasingly broad, coarse, and physically aggressive strokes of pastel.[7] On the canvas, individual forms lose their distinct boundaries and fuse with the background. This physical enlargement of the brushstroke serves as a direct topographic map of his expanding central scotoma. A definitive clinical record of

this progression is found in *Scene from the Steeplechase: The Fallen Jockey*, a painting reworked by Degas across three distinct decades (1866, 1880, 1897). As demonstrated in Marmor's comparative study, which provides striking computer simulations of the artist's declining view at each stage of the painting's revision, the successive alterations to the brushstroke mechanics function as a clinical eye chart, objectively documenting the progressive blurring of the artist's visual field directly into the physical layers of pigment.

3.4.4. Spatial Asymmetry: Hemineglect

While ocular disease alters the mechanics of the brushstroke, structural cortical pathology dictates the fundamental architecture of the canvas. The most profound materialization of this brain pathology manifests as spatial asymmetry, classically observed in cases of hemispatial neglect following a right-hemisphere stroke. In this condition, the artist does not suffer from optical blindness, but from a profound attentional deficit that completely obliterates the left side of their perceived reality.[50]

The self-portraits of the German painter Anton Raederscheidt, produced after a severe right parietal stroke, provide a definitive clinical timeline of this phenomenon (Figure 10).[51] In his immediate post-stroke canvases, the left side of his face and the left spatial field are entirely omitted, leaving half the canvas physically void. As his brain gradually reorganized through neuroplasticity over several months, the left side of his portraits incrementally repopulated with detail. In these instances, the canvas functions analogously to standard clinical diagnostic tools—such as the clock-drawing or line-bisection tests—but operates on a macro-aesthetic scale. The artwork provides a literal, topographical map of cortical stroke and subsequent neurological recovery, proving that the spatial composition of the canvas is strictly dictated by the functional geography of the artist's brain.

3.4.5. The Blindness Gain

As established previously (see Section 4.3.2), the absolute obliteration of retinal input forces a radical cortical migration from an allocentric visual framework to a tactile-egocentric geometry. In the context of artistic production, this profound neurological adaptation is frequently misinterpreted by classical medicine merely as a quantitative deficit. However, contemporary neuroaesthetics and Critical Disability Studies propose a radical epistemological inversion through the concept

of "Blindness Gain".[52] This paradigm postulates that visual deprivation is not simply an aesthetic "blackout," but a powerful catalyst for a hyper-refined sensory reconfiguration. As extensively explored by Kleege (2018), blindness does not diminish artistic capacity but rather brings a unique, multi-sensory dimension to art that sighted individuals often overlook. This suggests that the unique haptic engagement of blind individuals can profoundly teach sighted viewers new, multi-sensory ways to experience art.[53] The artist does not cease to perceive or create; instead, the creative drive is entirely outsourced to the newly recruited somatosensory networks. Qualitative investigations into the practices of blind and low-vision visual artists further confirm that they actively deconstruct their visual experiences, utilizing specialized sensory strategies to perceive details and engage creatively with their environment.[54]

This biological reliance on cross-modal plasticity fundamentally alters the genesis of the artwork. For the visually impaired creator, the two-dimensional canvas loses its validity as an optical mirror of a distal world, transforming instead into a proximate space of physical contact. The late career of the British painter Sargy Mann provides the definitive clinical-aesthetic benchmark for this transition. Completely blind by the age of 68, Mann did not abandon painting; rather, he developed a complex system of tactile measurement, utilizing pieces of *blu-tack* and measuring sticks to physically map spatial coordinates and anatomical proportions directly onto the canvas. This rigorous application of tactile information and physical measurement allowed him to vividly depict the visual world, proving that the eyes are not the sole seat of vision.[55] His late works ceased to be optical captures of light, becoming instead rigorous haptic and kinesthetic records. The canvas was literally constructed through the very "non-optic vision" generated by the exploratory movements of his hands. Today, this adaptive creativity extends into digital realms, as blind creators engage with AI image generators to navigate the intersection between tactile media and abstract visual art.[56]

Ultimately, this organic necessity for tactile anchoring explains a material migration observed in many creators with severe visual impairment: the inevitable shift from two-dimensional painting to three-dimensional sculpture and clay media.[57, 58] While the brain sustains spatial literacy and shape representation through touch, the flat canvas often lacks the physical resistance and feedback needed by an egocentric sensory schema. Clay's three-dimensionality provides necessary haptic validation.

Thus, a blind artist's sculpture is not an imperfect visual replication but an exact exteriorization of their cortical architecture. The artwork functions as a topographic cast of a brain that sees through touch. This cross-modal engagement validates the expanding scope of arts therapies, which increasingly utilize diverse modalities—such as visual art, music, and dance—to support the emotional and spatial rehabilitation of individuals with visual impairments.[59]

4. DISCUSSION

4.1. The Diagnostic Gap

As established in the Results (Section 4.3), when the fovea fails, the brain's mandatory shift to the peripheral retina—the Preferred Retinal Locus (PRL)—fundamentally redefines the sensory baseline. Specialized for low-resolution "gist" extraction (Thibaut et al., 2014), this adaptation allows patients to reconstruct the world through a sequence of global shapes and contrasts. From a purely mechanistic standpoint, as observed in the late career of Edgar Degas, the artist "failed" the criteria for optical precision. Yet, his work demonstrates that spatial literacy and environmental categorization survived through this newly forged sensory baseline.

This biological reality dismantles the deepest premises of traditional medical semiology. The systematic dissociation between clinical perception, objective recognition, and subjective consciousness proves that classical ophthalmology has historically confined the concept of "vision" to a set of quantitative and static metrics, primarily the reading of optotypes on a Snellen chart. However, as argued by De Jong (2003), this mechanistic reductionism is clinically "myopic," as it measures the eyeball in a sterile, high-contrast environment that fails to reflect the complexity of the patient's lived experience.

Consequently, a profound disparity is revealed in clinical practice: a patient may retain a numerically acceptable visual acuity while simultaneously suffering from a catastrophic loss of functional navigability. The clinical definition of "deficit" must, therefore, be confronted by the emergence of "blindness gain". This paradigm shift requires a methodological leap analogous to cardiovascular medicine. Just as a static, in-office electrocardiogram (ECG) frequently fails to capture the dynamic reality of an arrhythmia, ophthalmology must recognize the Snellen chart as a mere visual "resting ECG". Rehabilitation must focus on evaluating and enhancing the patient's real-world navigability within their egocentric space, demanding the conceptual equivalent of a "visual Holter": an evaluation of dynamic, functional survival outside the sterile confines of the clinic.

4.2. Affective Modulators

The definitive proof of the failure of a purely fragmented clinical model lies in the impact of affective states on visual perception (Section 4.2). As demonstrated by the data from Bubl et al. (2010), dysphoria acts as a primary biological modulator. Major depression induces a measurable loss of contrast gain in the pattern electroretinogram (PERG)—secondary to impaired dopaminergic transmission within the inner retina. The emotional state physically corrupts the visual input at its very anatomical origin.

This core analytical finding necessitates an ontological redefinition of the act of seeing within a clinical framework. Traditional ophthalmology often adheres to a rigid Cartesian dualism, treating the eye as a standalone optical device (the hardware), while the mind and emotions are consigned to psychiatry. However, our data extraction challenges this clinical divide. Normal vision is not merely a passive, sterile recording of the external world; rather, it is a stable illusion, actively constructed and highly susceptible to the individual's neurochemical state. The act of "seeing the world in gray" definitively transitions from the realm of poetic metaphor or purely psychosomatic symptom to become an objective, quantifiable neurophysiological deficit.

For daily medical practice, this demands a paradigm shift in triage and prognosis. When a patient presents at the clinic reporting a deterioration in visual quality—claiming "everything looks dimmer" or "washed out"—and the structural imaging remains completely stable, the clinician cannot simply dismiss the complaint or attribute it to systemic fatigue. Recognizing depression as a retinal pathogenic agent means that treating the patient's affective state is, in itself, a direct ophthalmological intervention. Dopaminergic rebalancing or psychotherapeutic support cease to be mere palliative care; they become the causal therapeutics necessary to functionally restore contrast sensitivity. The physician must acknowledge that they are treating a perceptual system where emotion and photonics are biologically inseparable.

4.3. The Neurobiology of Creation: Between Control and Release

As detailed in the extracted literature, the neurobiology of artistic creation operates under a delicate tension between two opposing mechanisms: top-down reconstruction and paradoxical disinhibition. On one hand, in patients with

peripheral visual deprivation (such as glaucomatous neuropathy), there is a pathological hypertrophy of top-down processing, where the brain acts as a generative architect, "editing" reality to sustain its coherence. On the other hand, neurodegenerative frameworks such as Frontotemporal Dementia (FTD) reveal Paradoxical Functional Facilitation (PFF), where the erosion of semantic meaning liberates the visuospatial networks from the "tyranny of concepts" (Chatterjee, 2006).

These contrasting dynamics support an explanatory axiom: human perception exists as a biological balance between the intellect that invents vision to compensate for sensory loss and the visual instinct that is released when the intellect itself collapses. The collapse of descending control does not generate a perceptual void; instead, it triggers the emancipation of a raw, unfiltered perception. Human vision is a hierarchical equilibrium whose organic disruption inevitably flows into the urgency of material and artistic expression.

This neurobiological understanding must transform the clinical perception of neurodegeneration. The emergence of a sudden impulse for visual expression in patients with Primary Progressive Aphasia (PPA) or FTD must no longer be dismissed by neurologists as a mere "bizarre symptom" or a side effect of disinhibition. The primary clinical outcome is the recognition of graphic and plastic expression as a critical therapeutic window. When linguistic and semantic networks fail, the "visual brain" provides an alternative, non-verbal channel for communication. Prescribing targeted visual-expression therapies is not a cultural accessory, but a clinical intervention to harness the remaining visuospatial capacity, turning a pathological "release" into a functional tool for social and emotional connectivity.

4.4. Art as Semiology

As established in the Results through extreme cases of sensory deprivation (De Jong, 2003), the absolute absence of biological input does not silence the visual cortex. Instead, it triggers phenomena such as Charles Bonnet Syndrome or "phantom vision," where the brain actively projects reality *ex nihilo*. When an enucleated 65-year-old patient utilized watercolor to paint the complex visual hallucinations generated by her severed optic nerve, she was not engaging in an

abstract aesthetic exercise. The painted canvas captured the isolated hyperactivity of her occipital lobe—a neural electrical storm translated into pigment.

This phenomenon exposes the epistemological limit of contemporary medical imaging and its structural confinement. An MRI or an OCT scan can flawlessly photograph the biological container, but it remains entirely blind to the perceptual content. Imagiology maps the anatomy of a lesion, but it cannot photograph what the lesion actually *sees*.

To bypass this technological blockade, modern medical practice must formally integrate graphic expression into diagnostic protocols. The patient's graphic externalization executes the first step of the Art-Vision Framework: Visual Production as Clinical Semiology. The clinical outcome of this realization is the validation of the resulting material record as the only existing phenomenological biomarker. Just as a neurologist uses an electroencephalogram (EEG) to map seizure activity, the ophthalmologist must utilize drawing to map subjective visual distortions. This diagnostic imagery ceases to be a passive product of pathology and becomes a rigorous medical examination, granting the clinician unprecedented access to the intricate visual chaos locked inside the patient's mind.

4.5. The Education of the Gaze

In contemporary clinical environments, diagnostic proficiency heavily relies on heuristics—mental shortcuts designed for rapid pattern recognition (Bleakley et al., 2003). This is brilliantly materialized in the work of De Jong (2003), who digitally manipulated Rembrandt's masterpiece, *The Anatomy Lesson of Dr. Nicolaes Tulp*, to simulate the phenomenology of scotomas. In the 17th century, Rembrandt's canvas celebrated the physical dissection of a corpse; today, the manipulation of that exact same painting teaches modern physicians that dissecting the ocular anatomy does not encapsulate the truth of lived vision.

As Bleakley et al. warn, these visual heuristics are inherently "Janus-faced". They frequently trap clinicians in a cycle where the conceptual textbook image overrides perceptual reality, leading them to "see" only what they expect to see, thereby completely neglecting the singular phenomenology of the patient's subjective deficit.

To counteract this automatism, the culmination of the Art-Vision Framework demands a radical transformation in medical pedagogy. Medicine must reclaim its intrinsically aesthetic dimension through the active development of "clinical connoisseurship." The analysis of visual arts is stripped of its status as a mere "cultural luxury"; it emerges as a rigorous, mandatory educational exercise designed to "educate attention." By training the aesthetic gaze through the practice of *close noticing*, physicians acquire the interpretative tools necessary to transition from a superficial gaze (*looking*) to deep, critical observation (*seeing*). Medicine must learn to contemplate the patient's "canvas," acknowledging the ultimate clinical axiom: all vision resides between our ears.

4.6. Art as Clinical Synergy

As documented throughout the artistic findings, visual impairment forces a migration toward haptic expression. The transition from vision to touch does not merely represent a change in sensory modalities; it constitutes a radical shift in the metrics of existence and temporality. While vision operates as an allocentric and instantaneous recording—a "snapshot" of the distal world—touch requires an egocentric and sequential space, painstakingly constructed piece by piece through a chronology of physical contacts and kinesthetic memory.

Ignoring this fundamental difference means ignoring the cognitive fatigue and massive reconstructive effort inherent in the daily lives of these patients. The Art-Vision Framework (Figure 11) culminates in Stage 2: "Art as a Solution." This phase proposes the use of material expression as a "2-in-1" therapeutic synergy. Just as pharmacology seeks combined formulations, art must act simultaneously as a diagnostic tool (mapping distortion) and a rehabilitative tool (facilitating the transition to a tactile-egocentric model).

The resistance of conventional medicine to accepting targeted graphical expression as a science-based intervention is, therefore, paradoxical. In pediatrics and child psychiatry, drawing is routinely utilized as the gold standard for assessing neurodevelopment and cortical maturity. If medical praxis trusts that a child's stroke reveals the health of their brain, it is imperative that adult ophthalmology adopts the same logic. Structured visual and haptic expression (such as clay modeling) must be recognized as a form of perceptual orthopedics. It allows the patient to mitigate spatial exclusion and emotional isolation, offering structured

support for the transition between the lost visual world and the newly conquered tactile world.

5. CONCLUSION

The present scoping review demonstrates that visual impairment radically transcends the mechanical failure of the ocular globe. It instigates a profound, hierarchical reorganization of the individual's sensory and cognitive architecture. Vision is not a finished product of the retina, but an unstable and actively negotiated biological construct, constantly subjected to cortical "filling-in" algorithms, affective modulations, and paradoxical plasticities.

In this context, the visual arts cease to be a mere cultural accessory or a passive symptom of pathology. The artwork emerges as the most rigorous material translation of this organic metamorphosis. Through the Art-Vision Framework, we establish that the canvas and the sculpture function as vital **phenomenological biomarkers**, granting the clinician access to subjective, first-person distortions that remain completely invisible to conventional imaging.

5.1. Limitations

Despite the robustness of this conceptual framework, certain methodological and practical limitations must be acknowledged. Methodologically, the nature of a scoping review implies a broad synthesis of heterogeneous literature across ophthalmology, neurology, and medical humanities. This interdisciplinarity, while epistemologically necessary, prevents the execution of strict quantitative meta-analyses. The findings are primarily descriptive and phenomenological, relying heavily on historical case studies (e.g., Monet, Degas, Munch) and highly specific clinical subsets (e.g., enucleated patients).

Clinically, the pragmatic implementation of the proposed "visual Holter"—an assessment of dynamic, real-world navigability in the egocentric space—faces significant logistical hurdles. Standardized metrics for evaluating the efficiency of a Preferred Retinal Locus (PRL) or the efficacy of art therapy in a busy, time-constrained clinical setting remain underdeveloped, requiring further validation before widespread integration into routine practice.

5.2. Future Directions

Looking forward, clinical ophthalmology and neurology face a profound diagnostic dichotomy. We must acknowledge that Artificial Intelligence (AI) and machine

learning models will inevitably dominate structural imaging. The algorithmic parsing of OCT scans to identify micro-lesions will be executed with a mathematical exactitude that comfortably eclipses human capability. The purely mechanical gaze has been irreversibly automated.

Consequently, the survival and clinical relevance of the human physician will depend entirely on mastering a domain inherently inaccessible to algorithmic computation: the phenomenological diagnosis. The physician of the future must specialize in the subjective experience of the deficit. Integrating the rigorous aesthetic training of close noticing (clinical connoisseurship) and prescribing targeted graphical expression as "perceptual orthopedics" will become essential survival tools for the medical profession. Only by embracing the "evidence of the senses" and utilizing visual expression as a bridge will it be possible to close the abyssal fracture between the eye that the machine measures and the world that the patient effectively reconstructs. In the end, this thesis reaffirms the ultimate neurobiological and clinical axiom: the anatomy of the eye does not contain the whole truth of sight. Medicine must transcend the mere dissection of the optical organ and learn to contemplate the canvas of the human subject, because, as both art and neurology testify, **all vision resides between our ears**.

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LIST OF FIGURES

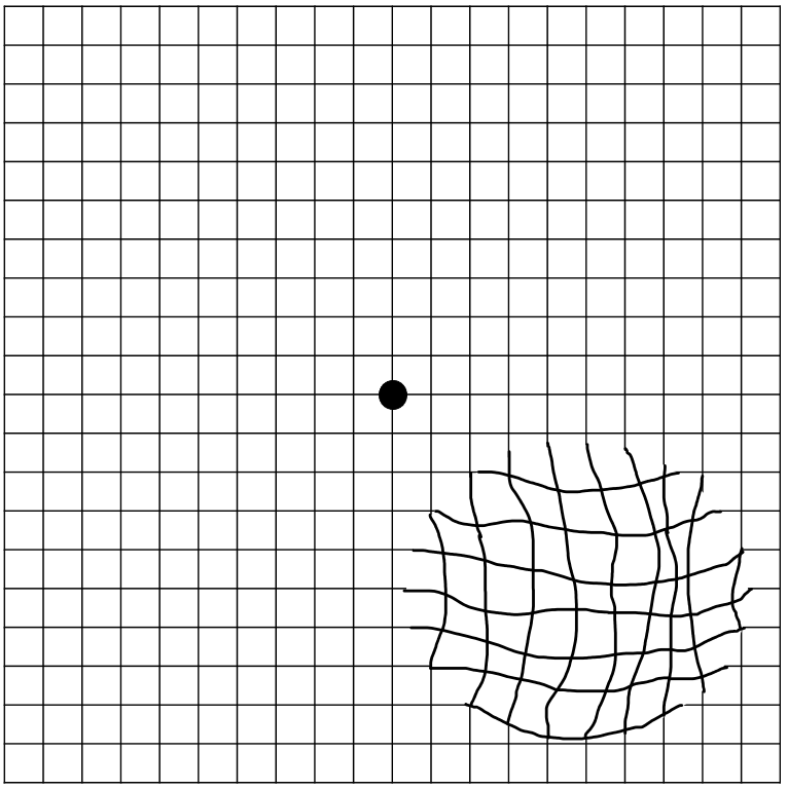


*Figure 1 - **Clinical representation of metamorphopsia via Amsler grid testing.** The image illustrates a localized geometric distortion—structural warping—demonstrating how the perceptual grid bends before it breaks. In progressive maculopathies, phenomena such as retinal edema and subretinal exudation physically displace macular photoreceptors. This mechanical disruption forces straight lines to undulate and spatial coordinates to buckle, a subjective visual symptom that systematically precedes the collapse into an absolute central scotoma. The grid effectively functions as a quantitative canvas, capturing the exact spatial coordinates of the early organic input failure. (Image source: Wikimedia Commons)*

a.

b.

c.

Figure 2 - ***Deconstructing the representation of glaucomatous visual field loss.***

a. *Normative visual processing, establishing the baseline spatial, structural, and tonal complexity of the environment.*

b. *A visual manifestation of the traditional "tunnel vision" paradigm. This representation conceptualizes retinal nerve fiber layer (RNFL) damage as an encroaching cinematic border, achieved digitally through the isolated application of a uniform peripheral dark vignette and mild central glare. This model reduces the complex neurodegeneration of the optic nerve to a simplistic structural occlusion.*

c. *A pathophysiologically grounded simulation of early-to-moderate glaucomatous erosion, translating the structural deficit into profound spatial fragmentation and a patchy, unreliable topography. The simulation was constructed through a multi-layered digital approach:* ***Pervasive Blur and Reduced Contrast:*** *A foundational sensory veil was established via dynamic range compression, mimicking global contrast degradation.* ***Asymmetric Spatial Fragmentation:*** *Rather than applying dark borders, localized zones of missing information were simulated using heterogeneous, asymmetric blurring and cortical filling-in distortions (extrapolating adjacent textures). This illustrates the silent erasure of spatial continuity in the mid-periphery.* ***Intense Glare:*** *The heightened susceptibility to ambient illumination was modeled by expanding the highlights of the sky (utilizing a Gaussian blur coupled with an additive blending mode), forcing the luminous peaks to bleed opressively over adjacent shadows and midtones.* ***Central Preservation:*** *The foveal focal axis remains completely untouched by the blurring and distortion filters, accurately reflecting the biomechanical resilience of the papillomacular bundle and the maintenance of central visual acuity amidst peripheral spatial collapse.*

(Composite image organized by the authors. Individual artworks in the public domain, sourced via Wikimedia Commons).

Figure 3 - ***Material documentation of lenticular sclerosis and acquired dyschromatopsia in the works of Francisco Goya.***

a. and b. *Early works depicting normative visual processing prior to significant optical media opacities. The palette demonstrates an uninhibited influx of light, capturing high luminance and a broad chromatic range.*

c. and d. *Late works reflecting severe cataractous erosion. Lenticular sclerosis functions as an intraocular blockade, where the cataractous lens disproportionately absorbs short-wavelength (blueish) photons, inducing a measurable acquired dyschromatopsia. This aging lens enforces a generalized chromatic shift, obliterating the blue spectrum and shattering overall contrast sensitivity. The resulting canvases serve as an objective clinical record of this structural toxicity, exhibiting the drastically reduced luminance and compressed tonal range characteristic of advanced lenticular opacity.*

(Composite image organized by the authors. Individual artworks in the public domain, sourced via Wikimedia Commons).

a.

b.

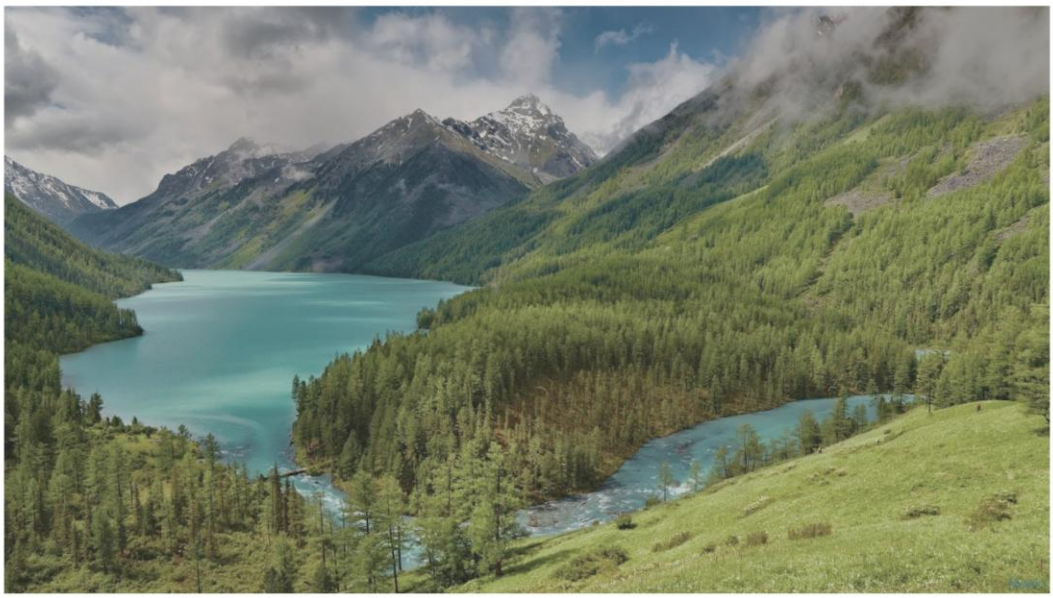


Figure 4 - ***Visual modeling of the contrast processing deficit in major depressive disorder. a.*** *Normative visual processing of a stimulus with high spatial and tonal complexity.* ***b.*** *Simulation of visual impairment in depression, anchored in PERG-documented reduced retinal contrast gain. Retinal ganglion cell hyporeactivity is modeled via dynamic range compression, a 70% contrast reduction, and a 20% saturation attenuation. This rigorously flattens atmospheric depth and blurs high spatial frequencies, objectively translating the subjective experience of "seeing gray" into a tangible manifestation of an altered primary visual pathway. (Composite image organized by the authors. Individual artworks in the public domain, sourced via Wikimedia Commons).*

a.

b.

c.

Figure 5 - ***Deconstructing the phenomenological representation of central vision loss in macular pathology.***

a. Normative Visual Processing: *Baseline spatial and structural complexity. High-resolution macular processing allows for the extraction of fine spatial frequencies, enabling precise facial recognition and unimpeded focal engagement with the central subject.*

b. The Traditional Textbook Representation: *A visual manifestation of the classic "black hole" scotoma frequently taught in medical curricula. Digitally, this was achieved by first applying a subtle structural distortion (metamorphopsia via the Liquify tool)—a nod to more advanced textbook models that recognize the initial anatomical misalignment of the retinal grid. However, it ultimately conceptualizes the death of macular photoreceptors as a direct, positive black occlusion over the visual field, achieved by applying a dense, dark central vignette over the foveal target. While this representation maps the gross anatomical deficit, it entirely ignores the central nervous system's active compensatory mechanisms, presenting a clinically inaccurate perceptual reality of a dark void.*

c. Proposed Model: *A pathophysiologically grounded simulation of central visual field erosion, illustrating the brain's biological intolerance to sensory silence (horror vacui). The simulation was constructed through a multi-layered digital approach to accurately reflect cortical adaptation:* ***Metamorphopsia:*** *To represent the anatomical misalignment of the retinal grid preceding complete spatial erasure, the structural geometry of the face was actively warped using distortion tools (Liquify/Push Forward).* ***Cortical "Filling-in":*** *Dismantling the myth of the black border, the central scotoma was modeled as an active hallucinatory overcompensation. Surrounding peripheral textures (background colors and garments) were probabilistically synthesized and cloned over the central defect, mimicking the brain's top-down generative inpainting algorithms.* ***Loss of High Spatial Frequencies:*** *A localized, severe Gaussian blur was applied to the central amalgamation, illustrating the sacrifice of macular detail and the resulting inability to extract fine facial features.* ***Eccentric Fixation (Preferred Retinal Locus - PRL):*** *To simulate the involuntary functional reorganization of the visual axis, absolute sharpness was intentionally displaced from the central face to the hands at the bottom of the canvas. This forces the observer to rely on peripheral anchors to sustain an illusion of environmental stability, preserving the global "gist" of the artwork despite the complete collapse of the macular hardware.*

(Composite image organized by the authors. Individual artworks in the public domain, sourced via Wikimedia Commons).

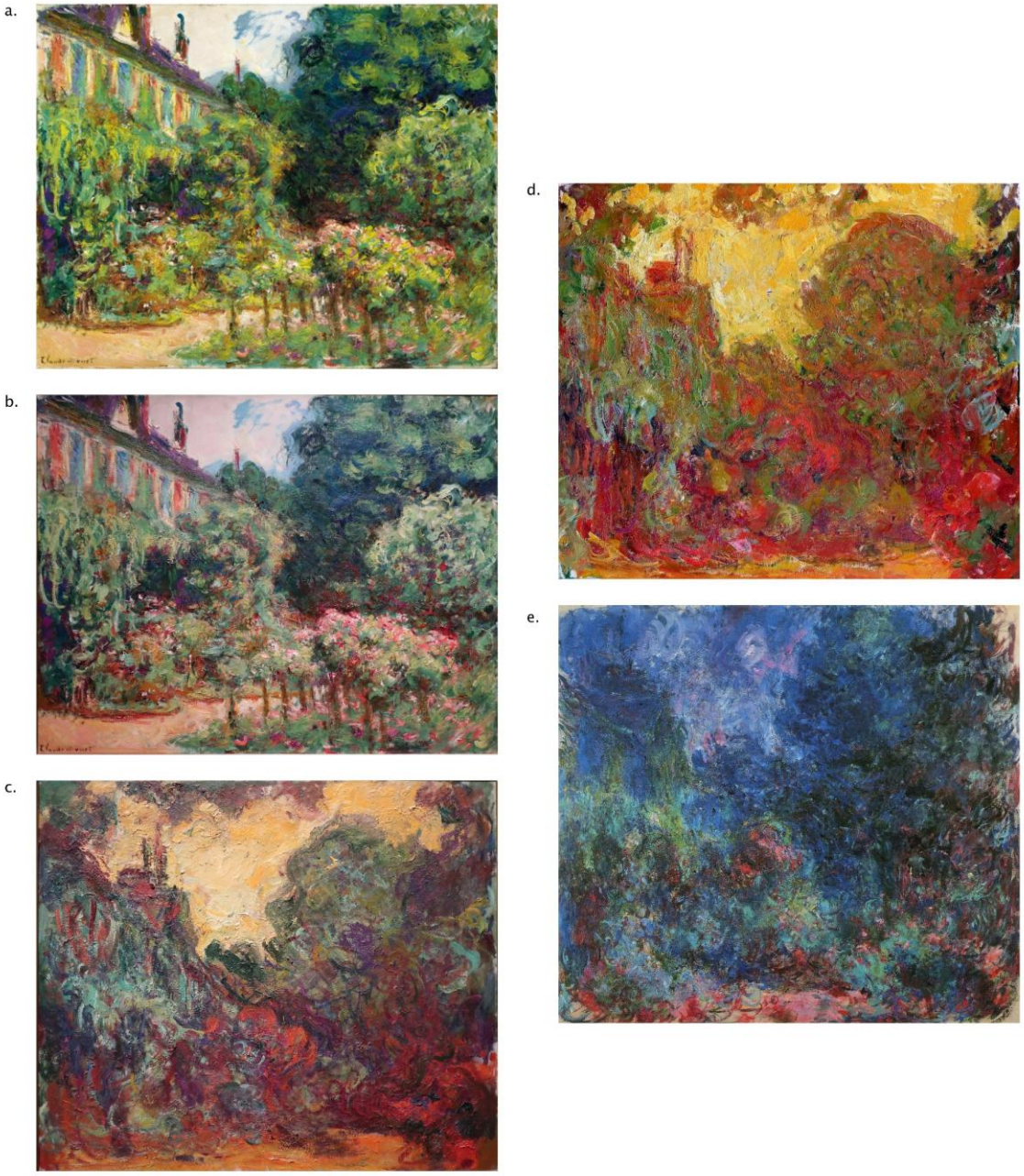
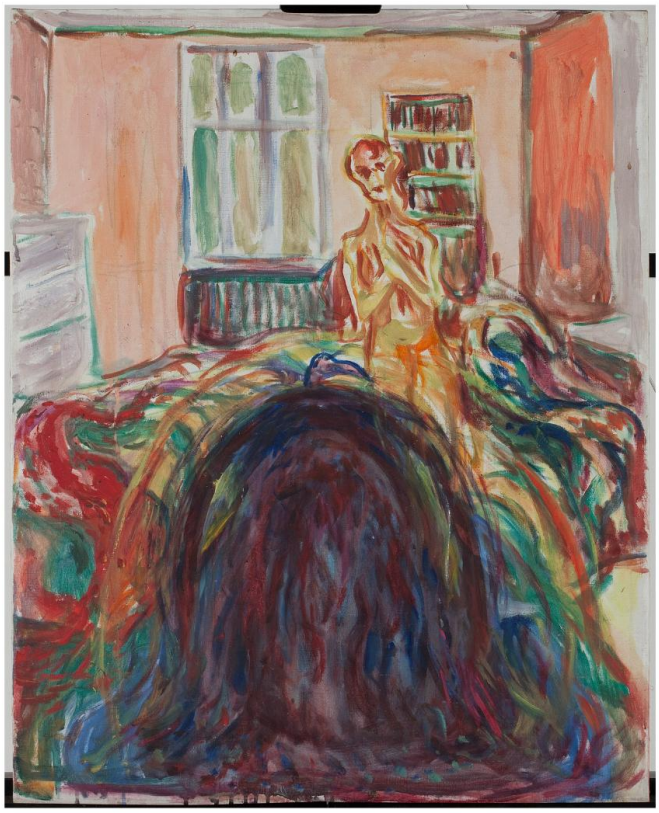


Figure 6 - ***The chronological and clinical evolution in the series The Artist's House from the Rose Garden (Claude Monet, c. 1922–1925).*** *This composite sequence, arranged chronologically, materializes the progressive degradation of the painter's optical system and the subsequent post-operative chromatic shock.* ***a., b., c.****The advancement of lenticular sclerosis acts as an increasingly dense yellow-brown filter. There is a gradual loss of visual acuity, a dissolution of architectural contours, and a progressive absorption of short-wavelength light.* ***d. (1922-1924):*** *The pathological peak. Optical blockade of the blue spectrum is nearly absolute. The canvas becomes saturated with a muddy palette of deep reds, oranges, and ochres, mechanically reflecting the "false dyschromatopsia" imposed by the mature cataract.* ***e. (1925):*** *The material record of post-operative cyanopsia. Following cataract extraction, the aphakic right eye is suddenly flooded with short-wavelength light. The palette violently shifts into highly saturated cobalt blues and intense violets, documenting the visual cortex's struggle to calibrate the sudden absence of the lenticular filter. (Composite image organized by the authors. Individual artworks in the public domain, sourced via Wikimedia Commons).*

Figure 7- ***Xanthopsia and the materialization of transient biochemical toxicity in Vincent van Gogh's Sunflowers.*** *The clinical hypothesis of digitalis intoxication illustrates how a transient biochemical filter might solidify into a permanent material artifact. While there is no definitive medical consensus regarding this posthumous diagnosis, the proposed phenomenon of xanthopsia (yellow vision) provides a compelling biological model for his late pigment choices. This hypothesis suggests that an endogenous toxicity, rather than a purely aesthetic decision, may have dictated his iconic, highly saturated color palette. Ultimately, the underlying principle remains consistent: the pathology dictates the palette, and the canvas records the symptom. (Image source: Wikimedia Commons)*

a.



b.

Figure 8 - ***Figure 8: The artwork as a clinical diagnostic instrument in Edvard Munch's documentation of an intraocular hemorrhage. a. and b.*** *The projection of positive and transient visual phenomena directly onto the painted surface, capturing the exact geometry of an entoptic shadow. These works document the artist's 1930 intraocular hemorrhage, materializing a mobile, semi-opaque scotoma that he famously visualized as a "bird of prey". Clinically, the mobility of this visual defect indicates a vitreous hemorrhage, as blood suspended within the vitreous gel shifts with eye movement, unlike the fixed scotomas characteristic of intra-retinal bleeding. By utilizing the canvas as a perimetric tool, Munch successfully transformed an unobservable, subjective symptom into a quantifiable material record. This transition from metaphor to measurement highlights the unique capacity of the artistic medium to "freeze" transient pathophysiology, providing a chronological evaluation of a biological event that would otherwise remain unrecorded. (Composite image organized by the authors. Individual artworks in the public domain, sourced via Wikimedia Commons).*

a.

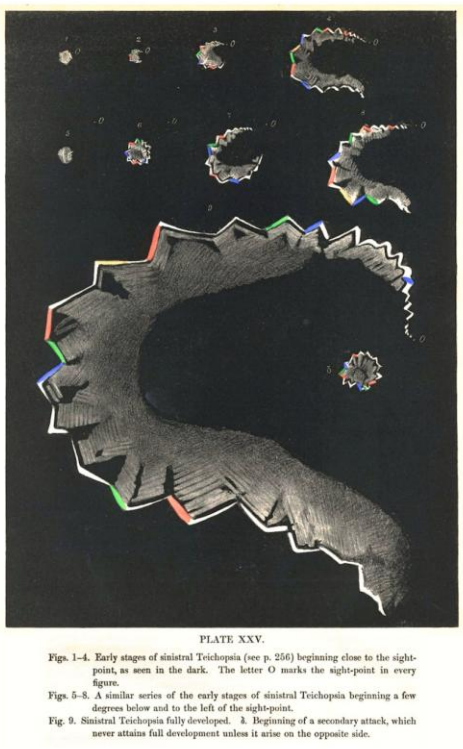


PLATE XXV.

Figs. 1–4. Early stages of sinistral Teichopsia (see p. 256) beginning close to the sight-point, as seen in the dark. The letter O marks the sight-point in every figure.

Figs. 5–8. A similar series of the early stages of sinistral Teichopsia beginning a few degrees below and to the left of the sight-point.

Fig. 9. Sinistral Teichopsia fully developed. *h*. Beginning of a secondary attack, which never attains full development unless it arise on the opposite side.

b.

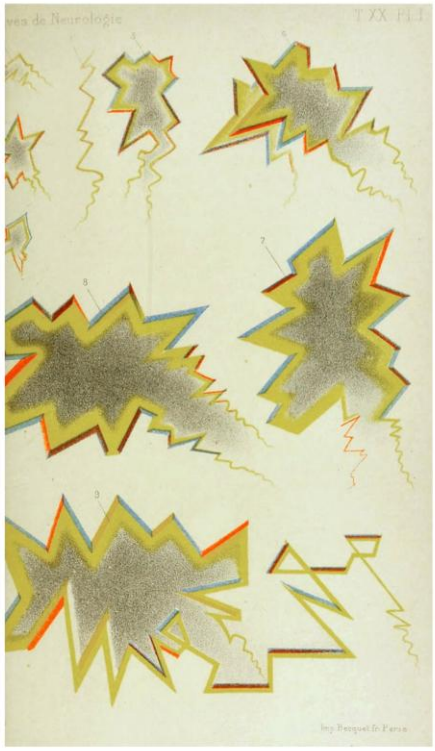


Figure 9 - ***Geometric externalization of cortical spreading depression in migraine aura. a. and b.*** *Illustrations demonstrating the capacity to materialize transient, unobservable neurological events associated with the pathophysiology of migraine. Panel (a.) features the seminal 1870 drawings by English physician Hubert Airy, representing one of the first historical depictions of a migraine aura (subsequently published in Jelliffe and White's 1915 textbook, Diseases of the Nervous System). These clinical records systematically map fortification spectra (teichopsia) and the "corona phenomenon"—the luminous, zigzagging halos that are mathematically characteristic of cortical spreading depression. By translating the subjective visual correlates of a cortical "electrical storm" into precise geometric artifacts, the visual medium provides a permanent clinical document of involuntary cortical activity. This effectively externalizes strictly internal hallucinations that would otherwise remain entirely inaccessible to external observation. (Composite image organized by the authors. Individual artworks in the public domain, sourced via Wikimedia Commons).*

Figure 10 - ***Topographical mapping of hemispatial neglect in the post-stroke self-portrait of Anton Raederscheidt.*** *The artwork provides a definitive clinical timeline of spatial asymmetry following a severe right parietal stroke. In this immediate post-stroke canvas, the left side of the artist's face and the left spatial field are entirely omitted, leaving half the canvas physically void. This material record proves that the spatial composition of the canvas is strictly dictated by the functional geography of the artist's brain, serving as a literal, topographical map of the cortical stroke prior to subsequent neuroplastic recovery. (Artistic Skills Recovery and Compensation in Visual Artists after Stroke - Scientific Figure on ResearchGate. Available from: https://www.researchgate.net/figure/Anton-Raederscheidt-self-portrait-1-post-stroke_fig3_303030415 [accessed 2 Jun 2026])*

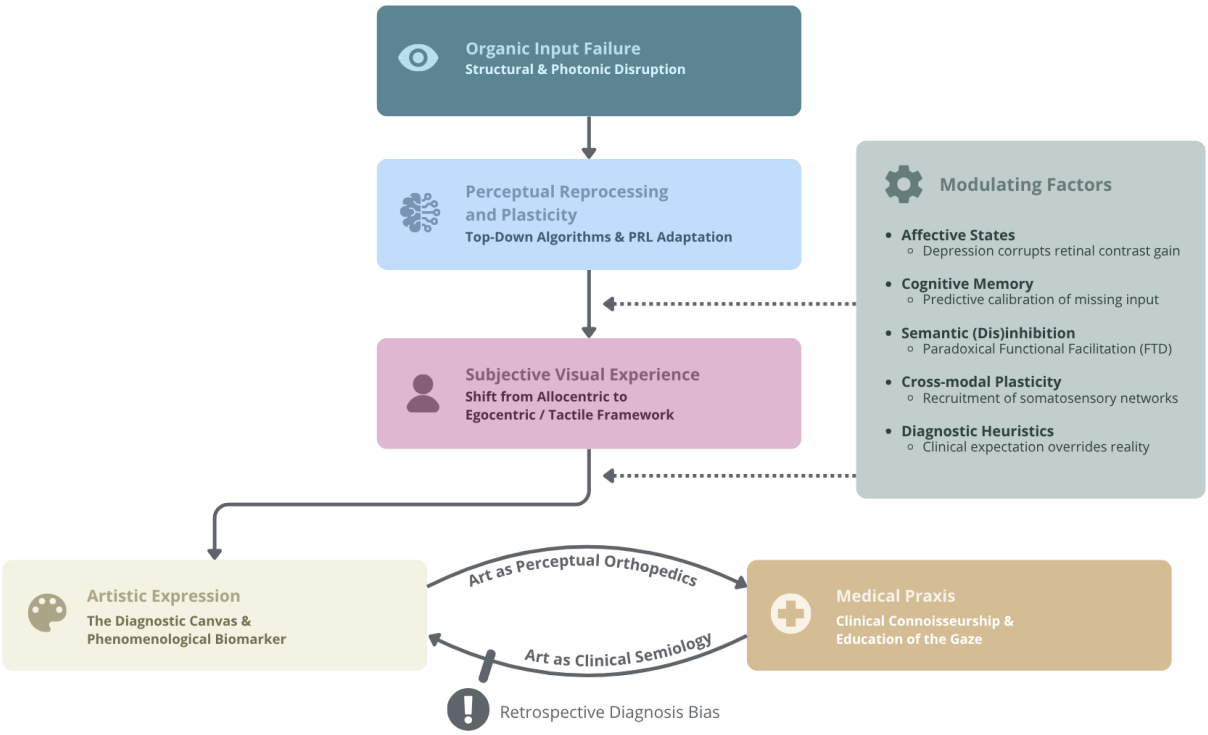


Figure 11 - ***The Art-Vision Framework: A dynamic model integrating phenomenological biomarkers into clinical practice. The Perceptual Cascade:*** *The framework maps the trajectory of visual degradation, initiating at organic input failure (photonic disruption) and advancing through cortical perceptual reprocessing, which includes top-down algorithms and Preferred Retinal Locus (PRL) adaptation. This results in a transformed subjective visual experience, driving the patient's shift from an allocentric optical geometry to a tactile-egocentric framework.* ***Modulating Factors:*** *This biological cascade is continuously mediated by internal and external modulators, ranging from affective states (such as depression corrupting retinal contrast gain) and cross-modal plasticity to the interference of the physician's own rigid diagnostic heuristics.* ***Clinical Synergy:*** *The culmination of the model proposes a bidirectional therapeutic loop. Artistic expression functions as a diagnostic canvas and phenomenological biomarker, elevating visual production to the status of rigorous clinical semiology. Concurrently, medical praxis utilizes art as "perceptual orthopedics" for rehabilitation, fostering the education of the physician's gaze while maintaining a strict epistemological filter against retrospective diagnosis bias (Original figure developed by the authors).*

APPENDIX 1- DETAILED SEARCH STRATEGY AND PRISMA FLOW DOCUMENTATION

1. Conceptual Framework and Descriptor Mapping (PCC)

The search strategy was structured according to the PCC framework, utilizing a balance of controlled vocabulary (MeSH) and free-text keywords to ensure optimal sensitivity and interdisciplinary capture. Subcomponents of perception were explored during thematic synthesis rather than operationalized as search filters to maximize conceptual breadth.

Table A1 – PCC Framework

PCC PARADIGM	CONTROLLED VOCABULARY (MESH)	FREE-TEXT KEYWORDS
POPULATION	Persons with Visual Disabilities; Vision Disorders ; blindness.	visual impairment ; low vision ; vision loss ; visual loss ; ocular disease ; eye disease ; retinal disease ; macular degeneration ; glaucoma ; cataract.
CONCEPT	Visual Perception.	visual experience ; altered perception ; perceptual experience ; perceptual change ; sensory perception ; visual experience of disease ; seeing ; ways of seeing.
CONTEXT	Visual Arts; Art; Art Therapy.	medical humanities ; humanities in medicine ; art and medicine ; medicine and art ; clinical observation ; seeing in medicine ; medical gaze ; creativity ; artistic expression ; artistic adaptation.

2. Database-Specific Search Architecture

Searches were conducted between 7 February 2026 and 9 February 2026, with syntax adapted for each respective database platform.

- **PubMed / MEDLINE:** Interrogations deployed a biphasic algorithm.
    - *Core MeSH Strategy:* ("Vision Disorders" [MeSH] OR "Persons with Visual Disabilities" [MeSH]) AND ("Visual Perception" [MeSH]) AND ("Visual Arts"[MeSH] OR "Art"[MeSH] OR "Art Therapy" [MeSH]).
    - *Hybrid Strategy:* Combined MeSH descriptors and free-text terms within Title/Abstract fields to bypass syntax-related automatic term mapping constraints.
- **Scopus:** Hybrid strategies were applied within the TITLE-ABS-KEY search fields to intentionally broaden interdisciplinary capture.
- **Web of Science (Core Collection):** Searches were executed using the Topic (TS) semantic field.
- **JSTOR & Google Scholar:** Emphasis was placed on experiential and perceptual terminology ("visual experience" OR "ways of seeing" OR seeing). Terms explicitly tied to "art" were excluded in JSTOR to mitigate excessive thematic heterogeneity unmoored from perceptual reality.

3. Prisma Flow Diagram

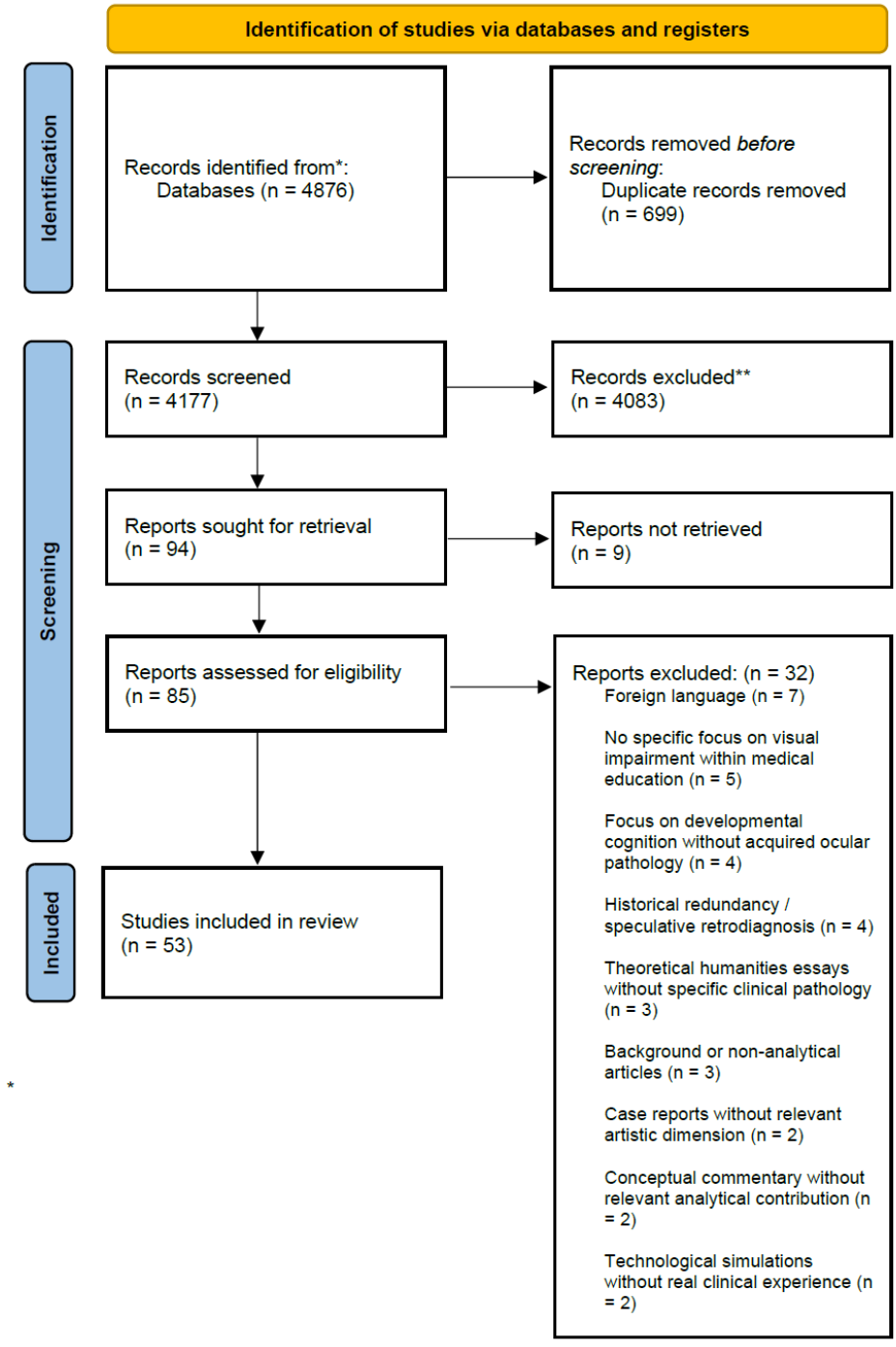


Figure A1: PRISMA flow diagram for new systematic reviews which included searches of databases and registers only. *The diagram maps the precise attrition mechanics and study selection process for the scoping review. Initial database searches identified 4,876 records. Following algorithmic deduplication (n = 699), 4,177 records underwent primary title and abstract screening, resulting in 4,083 exclusions. Full-text appraisal was sought for 94 reports, with 85 successfully retrieved and formally assessed against the study's eligibility parameters. Applying the established epistemological filters, 32 reports faced terminal exclusion based on specific rationales, such as historical redundancy/speculative retrodiagnosis, purely theoretical humanities essays, or lacking a specific focus on acquired visual impairment. The systematic extraction process ultimately yielded a curated corpus of 53 included articles.*

APPENDIX 2 - PREFERRED REPORTING ITEMS FOR SYSTEMATIC REVIEWS AND META-ANALYSES EXTENSION FOR SCOPING REVIEWS (PRISMA-SCR) CHECKLIST

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
TITLE			
Title	1	Identify the report as a scoping review.	I
ABSTRACT			
Structured summary	2	Provide a structured summary that includes (as applicable): background, objectives, eligibility criteria, sources of evidence, charting methods, results, and conclusions that relate to the review questions and objectives.	VII - VIII
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known. Explain why the review questions/objectives lend themselves to a scoping review approach.	10 - 11
Objectives	4	Provide an explicit statement of the questions and objectives being addressed with reference to their key elements (e.g., population or participants, concepts, and context) or other relevant key elements used to conceptualize the review questions and/or objectives.	11
METHODS			
Protocol and registration	5	Indicate whether a review protocol exists; state if and where it can be accessed (e.g., a Web address); and if available, provide registration information, including the registration number.	12
Eligibility criteria	6	Specify characteristics of the sources of evidence used as eligibility criteria (e.g., years considered, language, and publication status), and provide a rationale.	12
Information sources*	7	Describe all information sources in the search (e.g., databases with dates of coverage and contact with authors to identify additional sources), as well as the date the most recent search was executed.	12, 54
Search	8	Present the full electronic search strategy for at least 1 database, including any limits used, such that it could be repeated.	54, 55
Selection of sources of evidence†	9	State the process for selecting sources of evidence (i.e., screening and eligibility) included in the scoping review.	13
Data charting process‡	10	Describe the methods of charting data from the included sources of evidence (e.g., calibrated forms or forms that have been tested by the team before their use, and whether data charting was done independently or in duplicate) and any processes for obtaining and confirming data from investigators.	13
Data items	11	List and define all variables for which data were sought and any assumptions and simplifications made.	13
Critical appraisal of individual sources of evidence§	12	If done, provide a rationale for conducting a critical appraisal of included sources of evidence; describe the methods used and how this	13

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
		information was used in any data synthesis (if appropriate).	
Synthesis of results	13	Describe the methods of handling and summarizing the data that were charted.	13
RESULTS			
Selection of sources of evidence	14	Give numbers of sources of evidence screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally using a flow diagram.	13 - 14
Characteristics of sources of evidence	15	For each source of evidence, present characteristics for which data were charted and provide the citations.	14-28
Critical appraisal within sources of evidence	16	If done, present data on critical appraisal of included sources of evidence (see item 12).	N/A
Results of individual sources of evidence	17	For each included source of evidence, present the relevant data that were charted that relate to the review questions and objectives.	14 - 28
Synthesis of results	18	Summarize and/or present the charting results as they relate to the review questions and objectives.	14 - 28
DISCUSSION			
Summary of evidence	19	Summarize the main results (including an overview of concepts, themes, and types of evidence available), link to the review questions and objectives, and consider the relevance to key groups.	29 - 34
Limitations	20	Discuss the limitations of the scoping review process.	35
Conclusions	21	Provide a general interpretation of the results with respect to the review questions and objectives, as well as potential implications and/or next steps.	35 - 36
FUNDING			
Funding	22	Describe sources of funding for the included sources of evidence, as well as sources of funding for the scoping review. Describe the role of the funders of the scoping review.	N/A

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