Trends in Cognitive Sciences

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Opinion

When visual metacognition fails: widespread anosognosia for visual deficits

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Anosognosia for visual deficits – cases where significant visual deficits go unnoticed – challenges the view that our own conscious experiences are what we know best. We review these widespread and striking failures of awareness. Anosognosia can occur with total blindness, visual abnormalities induced by brain lesions, and eye diseases. We show that anosognosia for visual deficits is surprisingly widespread. Building on previous accounts, we introduce a framework showing how apparently disparate forms of anosognosia fit together. The central idea is that, to notice a deficit, individuals need to form expectations about normal vision, compare expectations and visual input, and judge any mismatch at the metacognitive level. A failure in any of these three steps may lead to unawareness of visual deficits.

Highlights

Anosognosia for visual deficits – cases where significant visual deficits go unnoticed – challenges the view that our own conscious experiences are what we know best.

Anosognosia for visual deficits is surprisingly prevalent. For some visual deficits, such as loss of vision in half the visual field (hemianopia), anosognosia is very common.

Understanding how anosognosia occurs for visual deficits is a key challenge in metacognition research. There are currently multiple disparate explanations.

We propose a framework that builds on previous work and explains how those disparate explanations of anosognosia for visual deficits can fit together.

The central idea of our framework is that, for visual metacognition, no news is good news. Anosognosia for visual deficits occurs when the metacognitive system does not take into account error signals, either because they fail to propagate or because of a metacognitive deficit.

Introspection and visual experience

Introspection seems to provide a direct window on our conscious experiences. Philosophers have sometimes held that it is infallible, or at least highly reliable [1,2]. According to this intuitive picture, a drastic change in the way one experiences the world should be immediately noticeable by introspection. We challenge this intuitive view by examining instances of **anosognosia for visual deficits** (see Glossary), where individuals remain unaware of a broad range of visual impairments, including total blindness, partial vision loss, color blindness, and severe **glaucoma** (Figure 1). Throughout, we emphasize the prevalence of these surprising gaps in visual self-awareness. After showing that **anosognosia** for visual deficits does not have a single cause, we develop a unified framework that can account for the wide range of instances of this striking form of anosognosia.

Forms of anosognosia for visual deficits

In this section we review several forms of anosognosia for visual deficits: **Anton syndrome**, anosognosia for partial visual deficits, and anosognosia for eye diseases. In particular, we emphasize that anosognosia for partial visual deficits and anosognosia for eye diseases is surprisingly common.

Anton syndrome

Anton syndrome (also called Anton–Babinski syndrome [3]) is a rare disorder where a person has complete blindness with unawareness of vision loss [4]. It was first described by Seneca who details the case of Harpaste: '[S]he suddenly became blind. The story sounds incredible, but I assure you that it is true: she does not know that she is blind. She keeps asking her attendant to change her quarters; she says that her apartments are too dark' [5]. Notable features include the sudden onset of symptoms (most modern cases described are due to stroke), the unawareness of complete loss of vision, and (at least in certain cases) **confabulation** in the face of the deficit [6,7].

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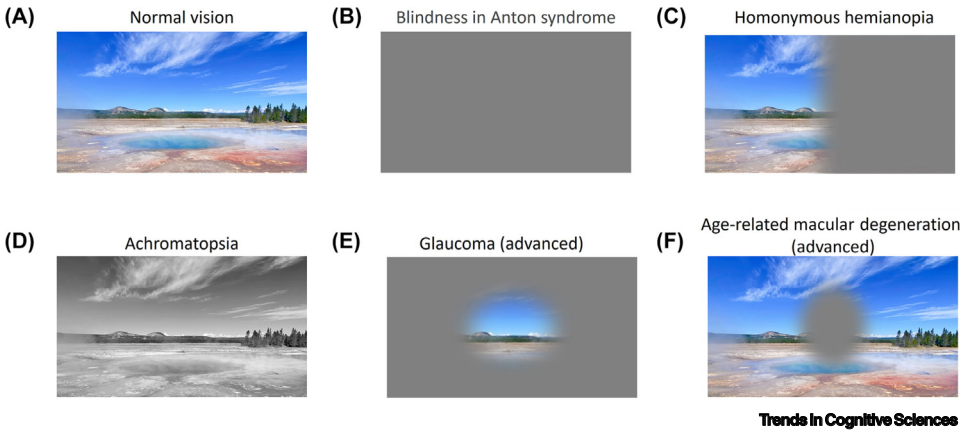

Figure 1. Anosognosia for different visual deficits: a depiction of the visual deficits in several visual disorders. The gray areas in the figure represent absence of input rather than an actual perception of gray. (A) Normal perception of hot springs on a sunny day. (B) Anton syndrome patients are completely blind. However, patients are unaware of their blindness and sometimes confabulate non-existent objects or deny their blindness. (C) Hemianopic patients experience only half of their visual field. Patients with anosognosia for hemianopia fail to report anything different about their visual experiences after the deficit has occurred. (D) Achromatopsia patients cannot identify colors and describe colored objects as gray. Individuals with anosognosia for achromatopsia can lack awareness of their color vision impairment. (E) Patients with glaucoma can experience progressive loss of peripheral vision. Those with anosognosia for glaucoma are unable to recognize these deficits. (F) In the advanced stage of age-related macular degeneration (AMD), individuals experience dense scotoma in their central vision. Subjects with anosognosia for AMD are unaware of this visual impairment.

Confabulation in Anton syndrome varies. Patients sometimes offer complex descriptions of non-existent objects [8–12]. For example, when questioned about an object held in front of him (which was actually a comb), a patient described it as a small exercise book [10]. When asked 'could it be that the image of this exercise book comes from your memory, and that in reality there is no exercise book?', the patient answered: 'I do see that there is an exercise book' ([10] p.1379). Other patients simply deny the deficit. For instance, faced with his inability to assess how many fingers the doctor was holding up, a patient answered 'I can't attempt to tell how many you have' [13]. When the doctor replied, 'I thought you said you could see well?' the patient raised his voice and answered 'I can see well!!!' (see also [8]). Despite many case studies, the prevalence of Anton syndrome is difficult to estimate [14–30].

In most cases, blindness in Anton syndrome is caused by bilateral lesions of the occipital lobes. But lesions to the occipital cortex are not the only cause. In rare cases, the disorder may arise from damage to the pre-cortical visual pathways, such as frontal tumors compressing the optic tract [31]. Cases in which Anton syndrome occurs with blindness caused by lesions in pre-cortical visual pathways tend to be associated with concomitant prefrontal lesions, which can cause behavioral and cognitive deficits [31–34], making the cases more difficult to interpret. While cognitive deficits might sometimes contribute to lack of awareness in Anton syndrome (e.g., [13,31,33]), this may not always be the case (e.g., [35]).

The typical course of Anton syndrome is mostly with sudden onset of symptoms, often followed by slow recovery of awareness over days [36], weeks [37,38], or even years in some cases [7], without reverting back to unawareness of blindness. Somewhat counterintuitively, in several cases, awareness of blindness appeared at the same time as the partial recovery of vision (e.g., [7,39]). As will become clear, the fact that awareness of the deficit occurs with partial visual recovery is a common and theoretically significant finding in anosognosia for visual deficits.

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Anosognosia for partial visual deficits

While Anton syndrome is relatively rare, other types of anosognosia are surprisingly prevalent. One common type is anosognosia for **hemianopia** (Figure 1C): blindness in half the visual field following a lesion to the occipital cortex [40]. Anosognosia for hemianopia is often diagnosed if patients lose vision in a part of their visual field but fail to notice or report this deficit even when explicitly asked about it [41]. Unlike Anton syndrome, it rarely involves confabulation: patients often recognize the presence of a deficit once clinicians demonstrate it using objective methods such as perimetry tests, although anosognosia can sometimes persist for months despite demonstrations of the deficit [42,43].

Depending on the time between deficit onset and testing, the prevalence of anosognosia for hemianopia ranges from 19% [41] to 41% [44], 55% [45], 62% [42], and up to 85% [46] and 88% [47]. Timing of testing may have something to do with this broad range of reported prevalence: the study with the lowest estimated prevalence (19%) tested subjects within a week after stroke onset [41], whereas some studies that reported a higher prevalence (62%) tested them within 24 h [42]. Of note, the latter study also excluded patients with psychiatric disorders (such as schizophrenia and dementia) [42]. In other words, more than half of patients with hemianopia are initially unaware of it. Some suggest that strategies allowing patients to adapt following the visual deficit, such as using eye and head movements, might be contributing factors in cases of long-lasting anosognosia [48]. Mograbi and Morris [49] noted that such strategies might indicate an implicit awareness of the deficit (Box 1).

Perhaps even more striking is anosognosia for **achromatopsia**: loss of color perception (Figure 1D). The first case was described by Verrey [50], whose patient could not see colors in the right visual field but was unaware of this deficit. Two similar cases were later reported [51]: patients with partial achromatopsia described objects as 'washed out' without recognizing any color vision loss. Anosognosia also occurs for complete achromatopsia. Here is a description of such a patient: 'When objects of various colors were presented, he maintained that all were greyish. […] Confronted with the color vision deficit, he was astonished and attributed it to poor lighting' ([52] p. 971). Importantly, the patient had preserved semantic memory for color: when

Box 1. Metacognitive systems and explicit metacognition

Our framework aims to explain how patients come to explicitly report their visual deficits. We hypothesize that metacognitive processing plays a crucial role in this. A rich literature associates metacognitive systems with prefrontal and cingulate structures (for reviews, see [104,105]). From this perspective, it is no surprise that anosognosia in other domains is often associated with prefrontal impairments. For example, frontotemporal dementia (FTD) is a straightforward case to consider, as it is characterized by atrophy of frontal and temporal lobes. Anosognosia is so common in FTD that it is one of its main clinical features: around 75% of patients with FTD exhibit a lack of awareness of their illness [114]. Meta-analyses have confirmed that the level of impaired awareness of illness in FTD correlates with frontal atrophy [114,115], and FTD is associated with impaired metacognitive capacities across a variety of tasks [116–118]. Anosognosia is similarly associated with prefrontal and metacognitive impairments in Alzheimer's disease [84,119,120], hemiplegia [65,121,122], and schizophrenia [123,124].

However, while the prefrontal cortex is necessary for explicitly representing the deficit, it might not be necessary for having implicit insight. For instance, patients might adapt their behavior to the deficit by moving their head more often to turn their intact visual field toward visual targets [49]. An explicit representation of the deficit is not necessary to adjust one's behavior to the deficit. As a result, this kind of implicit insight might be preserved even with prefrontal dysfunctions and failures of explicit metacognition. Critically, such compensating behavior can both forestall and facilitate explicit insight. Corrective eye and head movements expand patients' effective visual fields, in turn minimizing any mismatch between prior expectations and incoming sensory input. However, noticing that they are behaving in new ways might prompt patients to update their beliefs about their visual capacities. Observing one's own unsuccessful overt behavior might lead one to encode an explicit metacognitive representation of the deficit. This account fits well with the finding that, in many cases of anosognosia – including hemianopia and achromatopsia – patients discover their deficits only once they fail some behavioral tests that they expected to get right.

Glossary

Achromatopsia: a rare visual deficit characterized by total or partial absence of color vision.
Age-related macular degeneration (AMD): an eye condition, common among older adults, that gradually injures the central portion of the retina, often leading to blindness in the center of the visual field.
Anosognosia: a deficit of self-awareness, often resulting from various neurological or psychiatric conditions, where a person is unaware of their impairment.
Anosognosia for visual deficits: an unawareness of vision loss across different forms of vision loss and disorders, including complete cortical blindness, visual field loss such as hemianopia or quadrantanopia, and peripheral and central vision loss.
Anton syndrome: a rare syndrome where an individual becomes blind but is unaware of vision loss, often accompanied by confabulation. This has also been called 'visual anosognosia'.
Charles Bonnet syndrome: a condition in which visually impaired individuals experience complex visual hallucinations with maintained awareness.
Computation failure: the failure of a cognitive system due to computational suboptimalities, unrelated to the incoming signal (see also 'input failure').
Confabulation: the production of fabricated or distorted memories about oneself or the world, without the conscious intention to deceive.
Glaucoma: a group of eye conditions involving damage to the optic nerve that can lead to blindness, often caused by abnormally high pressure in the eye.
Hemianopia: the loss of vision in half the visual field, often resulting from stroke or brain injury.
Input failure: the failure of a cognitive system due to input signals being noisy, corrupted, or incomplete.
Metacognition: the capacity to monitor and form beliefs about one's own mental states.
Reality monitoring: the process of distinguishing internally generated from externally triggered sensory signals.
Visual neglect: a neurological disorder in which individuals fail to notice or respond to stimuli on one side of their visual field.

asked whether he had not found it difficult to eat colorless food, he replied: 'No, not at all! You just know what color your food is. Spinach, for example, is just green' ([52] p. 971). As with other cases, the patient became aware of the deficit as color vision slowly recovered over 2 months.

Anosognosia for vision loss due to eye disorders and diseases

Anosognosia for visual deficits of ocular origins may be even more prevalent. For instance, according to the CDC[I] only half of the people with open-angle glaucoma (where increased intraocular pressure damages the optic nerve) are aware of their condition. Glaucoma develops slowly over stages, spanning 10–15 years [53]. Initially, peripheral vision becomes blurred (Figure 1E), eventually leading to vision loss. Anosognosia for these visual deficits (in combination with the slow progression) may contribute to the failure of patients to seek care or adhere to treatment recommendations [54]. One patient remarked: 'If I had symptoms, I might be worried'. Some even claim to have improved vision compared with before developing glaucoma [54].

Another eye disorder associated with anosognosia is **age-related macular degeneration (AMD)**, an eye condition that primarily damages the macula (the central portion of the retina) (Figure 1F). A study involving 153 individuals with AMD reported that 56% were unaware of having binocular scotomas, despite dense scotomas as large as 30° in diameter [55]. To put this into perspective, these patients had a blind spot roughly the size of their hand held at arm's length, yet were unaware of it. Other studies reported even higher prevalence of unawareness, ranging from 84% [56] to 98% [57].

Given the slowly progressive nature of these conditions [58,59], patients might gradually adapt their daily activities to compensate for the visual impairment, making it difficult to recognize the extent of the vision loss [60,61], as in other forms of anosognosia involving implicit awareness of the deficit [49] (Box 1).

Possible accounts

Despite being described in modern neurology for over 100 years [3,4], explanations for the striking unawareness of vision loss in Anton syndrome and other visual deficits are sparse. Early theories proposed a disconnection between visual and language systems [62]. While this might address the confabulatory aspect in Anton syndrome, it does not explain the patient's behavior and inability to express their deficits through nonverbal means. Motivational accounts were also proposed early on and suggested that denial of the deficit is a coping mechanism [63]. But this view cannot explain cases such as anosognosia for hemianopia and eye diseases, since patients acknowledge the deficit once given objective proof.

Anosognosia in other domains – such as hemiplegia (unilateral paralysis) and cognitive deficits (e.g., Alzheimer's and related dementias) – has received far more attention [64]. However, the relevant models in these domains often focus on motor processing (e.g., [65]), memory systems (e.g., [66]), or concomitant cognitive deficits resulting in persisting delusions (e.g., [67]), and do not directly address the unique features of visual anosognosia. In this section we show that there is no 'catch-all' explanation of anosognosia for visual deficits. In the following section we integrate those different perspectives within a unifying framework.

Mental imagery and filling-in

One explanation for anosognosia for visual deficits is that patients do not report visual deficits because they still have internally generated visual experiences [10]. Similarly, some have hypothesized that Anton syndrome results from a disconnection of primary from visual association cortices where higher-order visual representations emerge [22,68]. But hallucinations alone do not

explain the denial of blindness. While it is true that hallucinations often occur without insight, such as in some cases of schizophrenia and Parkinson's disease, patients with **Charles Bonnet syndrome** who are blind and experience hallucinations are aware of their deficit [69]. The key difference may lie in **reality monitoring** deficits: the capacity to distinguish internally generated from externally triggered sensory signals. In the presence of such a deficit, hallucinations might explain some cases of Anton syndrome, where patients describe complex visual scenes [8–12,22]. Supporting this idea, studies show that visual imagery can be preserved despite early visual cortex lesions (for reviews see [70,71]). This explanation is also in line with accounts of anosognosia for hemiplegia that emphasize deficits in reality monitoring [65,72].

However, hallucinations and filling-in do not explain all cases. First, many anosognosia patients do not describe non-existent objects in the affected visual field [8,13], which would be expected if anosognosia always emerged from mental imagery and filling-in. Second, this account makes recovery mysterious; it suggests that hallucinations stop during recovery, or that patients realize that their experiences were just imagery, something not specifically reported by the patients. Finally, despite reports of filling-in across the vertical median in hemianopia [45,73,74], there is no evidence of filling-in for an entire visual field.

Neglect

Another possible explanation of anosognosia for visual deficits could be an attentional deficit (an inability to attend to the deficit) or **visual neglect** [75]. Anosognosia might occur if patients fail to attend to the deficit, a plausible hypothesis given that anosognosia is common in neglect itself [76], though anosognosia for attentional disorders such as neglect, extinction, and Balint syndrome is outside the scope of this opinion article. Neglect and anosognosia often co-occur in other domains, such as anosognosia for hemiplegia, mostly following right hemisphere lesions [77]. Recent research supports the idea that connectivity to attention networks may contribute to Anton syndrome [78]. Network modeling showed that lesions found in Anton syndrome connect to parietal and prefrontal areas, including regions in the dorsal attention network. This was interpreted as suggesting that attention deficits contribute to the severity of anosognosia in Anton syndrome patients [78].

While neglect and anosognosia are difficult to disentangle, especially in anosognosia for hemianopia [79], they should be distinguished, as they have been historically [76]. They dissociate even in disorders like anosognosia for hemiplegia [47,77,80,81], and studies on anosognosia for hemianopia show dissociations from neglect of the relevant side [42,47]. The dissociation holds in anosognosia for hemiachromatopsia [82]. An account of anosognosia for hemianopia in terms of neglect also conflicts with the patients' use of implicit compensatory strategies to attend to the relevant side of space (Box 1). Finally, neglect does not explain cases of anosognosia in which patients have a visual deficit for a specific feature, such as anosognosia for achromatopsia, as well as cases in which the visual deficit does not correspond to a particular side of space, such as in eye diseases like AMD. While anosognosia is common in many cases of neglect, neglect does not account for all, and probably not even most, cases of anosognosia for visual deficits.

Memory deficits

Memory deficits could contribute to anosognosia, as hypothesized by the cognitive awareness model (CAM) (Box 2): a model originally developed to explain anosognosia for cognitive deficits in Alzheimer's disease [66,83]. One possibility is that patients forget what normal vision was like before the deficit. Another possibility is that they fail to recall their own poor performance on visual tasks. These hypotheses are especially plausible in some cases of Anton syndrome (e.g., [33,63]). A recent network-based analysis of lesions associated with visual and motor

Box 2. The cognitive awareness model (CAM)

The CAM, originally developed to explain anosognosia for cognitive deficits in Alzheimer's disease, has been extended to visual deficits [6,66,83]. In the CAM, a module called the cognitive comparator mechanism identifies task failure by comparing task performance with performance stored in a 'personal database'. When this mechanism detects a failure, it updates the database and sends a signal to a 'metacognitive awareness system' which provides awareness of the deficit. According to the CAM, anosognosia can occur at three levels: primary anosognosia corresponds to a failure of the metacognitive awareness system, executive anosognosia is a failure of the comparator mechanism, and mnemonic anosognosia is a failure to update the personal database. To extend their model to all cases of anosognosia – including visual deficits – Morris and Mograbi added 'local perceptual comparators specifically providing input about expected and perceived activity within domains of cognitive and motor functioning' ([66] p.1560). They hypothesize that anosognosia for visual deficits could be explained by a failure of the local visual comparator, while a failure of the domain general comparator mechanism would result in lack of awareness across abilities. While the CAM successfully accounts for key findings, the model appears incomplete in several ways in the case of anosognosia for visual deficits. More specifically, the local visual comparator may fail to produce a mismatch signal due to qualitatively different process failures in different cases: habituation to the 'new normal' in the case of gradually progressing conditions such as glaucoma and AMD, a combination of filling in and a reality monitoring failure in some cases of Anton syndrome, a failure to form visual expectations due to lesions to the visual pathway, or a failure to form a prediction error based on the absence of a signal. Further, the CAM does not include a role for hallucinations and filling-in in anosognosia for visual deficits. Our framework extends the CAM by unpacking these different modes of failure, building on insights from the visual metacognition literature.

anosognosia showed that hippocampal memory-associated structures and cingulate metacognitive-associated structures may be related to the emergence of Anton syndrome [6]. However, most patients with Anton syndrome slowly learn with time that they have a visual deficit, unlike anosognosia for cognitive deficits in Alzheimer's disease which often persist and worsen. As a general rule, patients with Anton syndrome can update their memory with time without clearly reverting back to a lack of insight (e.g., [7,36–38]), which would not be explained by persistent memory deficits. In addition, memory deficits are not present in all cases of visual anosognosia, including anosognosia for hemianopia and visual deficits caused by eye diseases (e.g., [42]). In fact, the story might be more complex even in the case of anosognosia for cognitive deficits: anosognosia in Alzheimer's disease is not always associated with more severe memory impairment [84], and might instead be associated with reality monitoring impairments. So, while memory deficits might sometimes contribute, they are not the full story.

A unifying framework

An intuitive account of introspection is that it provides us with direct knowledge of our own experiences: we do not have to make inferences to know our own conscious states [2,85–88]. Following this model, introspection is a matter of directly translating conscious experience into metacognitive belief: a belief about one's conscious experience. A difference in experience – for instance, with respect to the presence of colors – should thus lead to a difference in metacognitive belief. However, anosognosia shows that metacognitive beliefs can fail to reflect drastic changes in experience. As previously emphasized by Levine [13], changes in experience brought by visual deficits are not immediately obvious but have to be discovered through a process of inference. The empirical literature on **metacognition** – the capacity by which we monitor and form beliefs about our own mental states – is also in tension with this intuitive view, as it has revealed that metacognitive beliefs often fail to adequately track perceptual states [89]. We now propose a unifying framework showing how the apparently disparate sources of anosognosia reviewed herein fit together, hoping that doing so might further shed light on some of the principles that govern visual metacognition.

Similar to the CAM (Box 2) (see also [13,72]), we suggest that subjects identify visual deficits by detecting a mismatch between expected and received visual signals. A mismatch triggers error signals leading to an update of one's metacognitive beliefs about one's visual capacities (Figure 2). Various factors might prevent those error signals from occurring or impacting

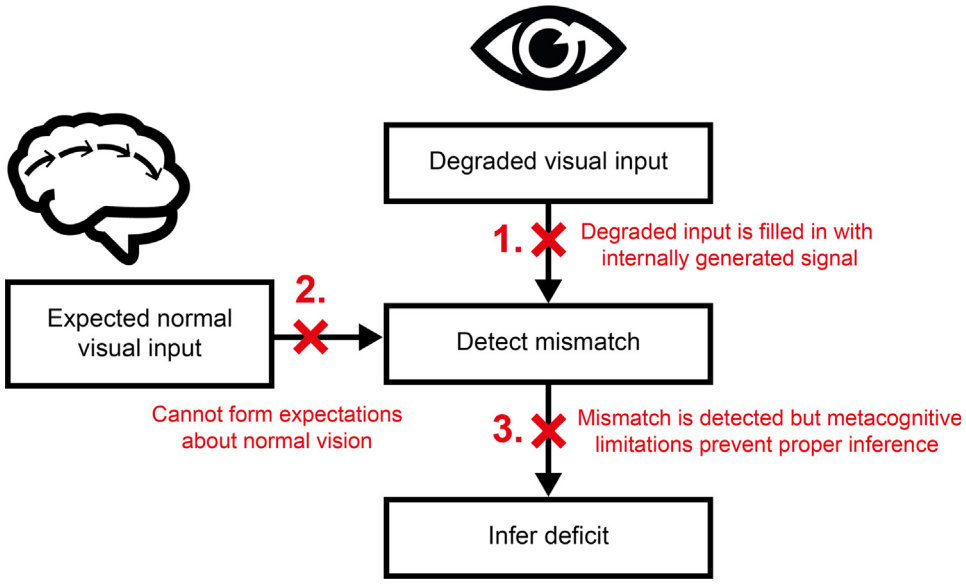

Figure 2. A unified framework for failing to notice visual deficits. According to our proposed framework, awareness of visual deficits requires several steps. To begin with, due to a failure of the visual system, visual input becomes degraded or absent. When compared against expectations, a mismatch signal is generated, which is then integrated with prior beliefs about one's own vision at the metacognitive level, resulting in an explicit insight into one's visual deficit. This healthy flow of information that alerts us about visual deficits can fail at three levels. (1). Filling-in and reality monitoring failures: degraded or missing input can be filled in by internally generated signals, unrecognized as having been internally generated by the subject. This includes cases of Anton syndrome in which subjects provide complex descriptions of objects. (2). Failures to form expectations: the same lesions that compromise vision may disturb patients' ability to form visual expectations about normal vision, for example, in hemianopia and achromatopsia. In cases of gradual degeneration, such as glaucoma or age-related macular degeneration (AMD), visual priors may gradually update to reflect unhealthy vision. (3). Metacognitive limitations: general impairments of metacognitive systems might render subjects unable to update their metacognitive beliefs.

metacognitive beliefs. Without those error signals, subjects continue to believe that their vision is fine, thereby leading to anosognosia. In a slogan: anosognosia occurs because, for visual metacognition, no news is good news.

Our framework hypothesizes three main sources of anosognosia (Figure 2). First, degraded inputs might be filled in with internally generated signals, preventing the comparison of actual inputs (or lack thereof) with visual expectations. Second, the conditions that compromise vision might prevent patients from forming the kind of visual expectations necessary to trigger error signals when matched against visual inputs. Third, general impairments of metacognitive systems – rather than anomalies in the inputs those systems receive – might prevent updating metacognitive beliefs. We discuss each of these sources of anosognosia in turn.

Filling-in and reality monitoring failures

Some failures of awareness of perceptual deficits might be due to self-generated signals that match one's expectations about the way the world should appear. Anosognosia might occur when this is combined with a failure of reality monitoring [90]. Hallucinations and filling-in could contribute to anosognosia by replacing missing visual inputs with internally generated content, similarly to how the metacognitive system overlooks the physiological blind spot due to low-level filling-in.

Our review showed that some cases of Anton syndrome feature vivid descriptions of visual objects, possibly generated by imagery [8–11]. For instance, the patient mentioned earlier

confidently answered 'I do see that there is an exercise book' (even though no such book was present), and described visual objects consistent with contextual cues [10]. Vivid visual imagery combined with a reality monitoring failure could hide the absence of visual input, leading to a failure to update one's metacognitive beliefs. Recent network-based analyses of lesion-induced Anton syndrome [6,78] reviewed earlier could support this hypothesis. Both studies show that lesions that cause Anton syndrome are connected to regions involved in metacognitive processing and reality monitoring, including the cingulate [6,41,91] and the prefrontal cortex [78] which are implicated in these processes [90,92]. The involvement of the precuneus in Anton syndrome [6,41] supports a similar interpretation given that this area is involved in metacognition for memory [93,94].

A speculative hypothesis is that some Anton syndrome patients might mischaracterize memory imagery as perception and believe that they perceive visual contents. As previously noted [95]: 'By confusing memory images with perceptions the patient has sufficient material for his pretended vision. When he hears the sound of lighting a match or feels its warmness he believes to see it' ([95], p. 950, translated and cited in [10]). The finding that the hippocampus might be involved in Anton syndrome aligns with this hypothesis [6], as the hippocampus is implicated in hallucinations in disorders such as schizophrenia and Lewy body dementia, possibly due to its role in binding or in the retrieval of visual priors [96–98].

Failures to form expectations

According to our framework, an error signal is triggered by comparing incoming evidence with top-down expectations. If the lesion causing the visual deficit also disrupts the formation or transmission of these descending signals, no error signal forms, leaving the deficit unnoticed. This hypothesis could explain certain cases of anosognosia for hemianopia and achromatopsia, as well as cases of Anton syndrome where mental imagery is absent.

When asked whether something is wrong with their vision, patients may attempt to mentally simulate what their vision was like before the incident. Since the lesioned areas might be involved in imagining or remembering what their visual experiences used to be like [99,100], the simulation might match the patient's current experiences, preventing them from identifying the deficit. Our framework gives a central role to memory: anosognosia occurs because memory recruits sensory systems in order to compare the state of one's vision before and after the lesions. In line with the CAM (Box 2), and as a complementary mechanism, some cases of Anton syndrome might be explained by memory deficits making it difficult to retrieve expectations about what one's visual capacities should be like [6].

A counterintuitive prediction of our account is that patients are more likely to detect partial rather than complete deficits. Detecting deficits is easier if enough preserved machinery and visual context exist to form and send top-down expectations, creating a mismatch with degraded signals. This prediction is counterintuitive because one might normally expect complete deficits to be more noticeable than partial deficits. Yet this prediction seems validated in several cases of anosognosia in which patients notice their deficits once their vision starts recovering [7,39,42,52].

Anosognosia for visual deficits could also result from a failure to form expectations about normal vision. Gradual changes in visual experience, as in glaucoma or AMD [58,59], might cause expectations to adapt to this 'new normal'. The gradual change in experience might not attract attention, in a phenomenon akin to cases of slow change blindness: the failure to see large changes slowly introduced in visual scenes [101,102]. Patients might thus believe that their vision is normal because this is the way their metacognitive system has progressively come to expect their vision

to be. When this happens, predictions and incoming signals match, thereby failing to trigger error signals.

Metacognitive limitations

In the cases discussed so far, anosognosia results from problems with the inputs received by metacognitive systems. Following a taxonomy recently developed [89], these cases correspond to metacognitive **input failures**. But some cases of anosognosia could also reflect **computation failures**, namely, cases in which inaccurate metacognitive inferences result from suboptimal metacognitive computations. This could happen either because the metacognitive system is functioning abnormally, such as following a lesion, or because it naturally performs suboptimal computations [103]. We discuss these two possibilities in turn.

Metacognitive systems are generally associated with prefrontal and cingulate cortices (for reviews see [104,105]). Accordingly, anosognosia in various domains is often linked to prefrontal impairments (Box 1). In light of this, one might understand why the few known cases of Anton syndrome caused by lesions in pre-cortical pathways also involve prefrontal cortex lesions [31–34], although those cases are admittedly very rare and difficult to interpret. In those instances, Anton syndrome could stem from an inability of the metacognitive system to perform the computations required to update one's metacognitive model. In addition, patients with anosognosia for hemianopia tend to be overly confident in their responses when solving riddles, compared with patients without anosognosia [44]. As such, suboptimal metacognition might be a preexisting trait that predisposes individuals to develop anosognosia following a lesion, or might result from additional neurocognitive deficits after the stroke.

Concluding remarks

Initial unawareness of visual deficits is surprisingly common, especially for hemianopia and eye diseases. Visual deficits are not always immediately obvious; they often have to be discovered by inference [13]. This should prompt a broader reconsideration of the limits of introspection across domains. Evidence from the laboratory and the clinic indicates that these superficially similar failures of awareness occur for a variety of different reasons. We have presented a cohesive framework in which each source of anosognosia for visual deficits is understood as impacting a specific stage leading to the production and propagation of error signals, and computations over those signals. Whether this framework applies beyond visual deficits remains an open question, depending partly on whether metacognition is a domain-general system using the same computations across different domains, a topic of ongoing debate [106,107] (see Outstanding questions). Understanding the specific disruptions in the cascade of error signal processing illuminates not only the peculiarities of visual anosognosia but also potentially provides a roadmap for deciphering metacognitive functions at large.

Let us finish with some of the philosophical implications. A common, intuitive view is that self-knowledge of our conscious experiences is very reliable [1,2,85,86], such that drastic changes in the way one experiences the world should be obvious. Following skeptical accounts of introspection [108], we have argued that those changes do not have to be manifest. Instead, in line with inferential theories of self-knowledge [109], individuals with anosognosia often come to know about their visual deficits not via direct introspection, but by finding out what they can or cannot do. Indeed, according to our framework, noticing a change to our experience requires not only access to our current perceptual states, but also an ability to form expectations based on memory of past experiences, to compare actual and expected experiences, and to make metacognitive inferences that generalize from concrete events to general abilities and deficits.

Outstanding questions

Are the reasons for anosognosia the same across different perceptual and cognitive domains?

Are failures of metacognition specific to noticing subtractive changes to the visual system? Are there opposite cases where people fail to notice an additive change (e.g., following a successful treatment)?

Under what conditions do anosognosia patients attribute a failure of the visual system to external conditions ('the apartment is too dark' [4]), and under what conditions do they deny the failure altogether ('I can see well!!!' [9])? What explains confabulations?

Are some people more susceptible to anosognosia? Is this related to their metacognitive sensitivity before the occurrence of the deficit?

Do certain cases of anosognosia result from the failure of multiple processes? How could we parcel out these different causes?

Beyond behavioral adjustments, how does the brain adapt its coding strategies in response to different types of visual anosognosia?

What visual aspects are commonly affected by anosognosia or poor metacognition, and conversely, what aspects of vision are typically not impacted, retaining good metacognitive awareness?

Can phenomena similar to visual anosognosia be experimentally induced in normal observers under controlled conditions?

Our framework also opens up intriguing avenues of research on failures of awareness outside of pathological cases. For instance, some have argued that consciousness is regularly interrupted by moments where one does not experience anything [110], or that it is constituted by discrete episodes interspersed with moments of unconsciousness [111]. Extending our framework to the temporal domain, our impression of consciousness as a continuous stream may reflect a more general failure of introspection to register experiential absences [112,113], similar to that observed in anosognosia. The scientific method could therefore have an important role in revealing the surprising ways in which the structure of conscious experience is different – perhaps more fragmented – than it seems by introspection.

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Declaration of interests

No interests are declared.

Resources

[i]www.cdc.gov/vision-health/about-eye-disorders/glaucoma.html#:~:text=Open%2Dangle%20glaucoma%2C%20the%20most,t%20know%20they%20have%20it

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