One of the most pressing questions in vision research is why we see the world around us uniformly sharp, when we should not. Visual acuity is highest in the fovea and falls off rapidly toward the near periphery. Therefore, we should see the world as depicted by Medardo Rosso (1858–1928) with the central area of the visual field in focus and the peripheral area progressively blurred. Yet, when we look straight ahead, we do not normally notice any blur outside the foveal area. Why is that?

A plausible assumption is that we compose our field of view from individual glimpses taken when we move our eyes. It is rare that we keep our gaze fixed at one given point as we scan the world before us; instead, we may retain a sequence of sharp images of it, which we assemble into a coherent whole.

There are many unsolved questions attached to this assumption: (i) Where in the brain is the short-term buffer that enables us to store those glimpses? (ii) How do we stitch the glimpses together, so that they yield a continuous representation of the world? (iii) Which mechanism provides the correct location of those glimpses and puts them in register as the world jumps across the retina with every eye movement? (iv) Why are there no borders separating individual glimpses and empty areas in between? (v) Why do we see the world sharply and coherently, even before we have swept it with our eyes (eg, when we enter a classroom)? It all boils down to the overarching question: Why do we not have more blind spots?

Bruno Breitmeyer in his fascinating book describes a large number of phenomena in normal and defective vision showing that we indeed have many blind spots, most of which we are hardly aware of. I will list a few.

**Retro-blindness.** The world behind us, which is outside of our visual field, is always present in our experience, although we do not actually see it. Yet, we have blind faith in it. The implicit assumption, of course, is that the world has not changed since we last saw it. If it had, we might be in trouble. Just think of a conductor stepping backward into the orchestra pit. Hares, rabbits and other animals of prey can see what is behind them without turning, because they have lateral eyes (allowing them to tell how close they are to being eaten by a predator).

**Blind spots.** Few people know that they have two physiological blind spots, one in each eye. These are the result of the anatomy of the optic disk, an area of the retina from where the optic nerve exits from the eye. No signals reach the brain from this area since there are no photoreceptors in it. Consequently, a small light spot falling onto the optic disk will not be perceived, unless it casts stray light onto the adjacent retina. Yet, we do not see the blind spot, even if we close one eye. Why is this? The answer is, because of fading and filling-in. Uniform perception of brightness, colour, texture, even motion occurs across the blind spot and thereby provides us with a coherent picture of the world in an area where we should see a hole of 6 by 8 deg visual angle, large enough to hide a car. Filling-in may thus be conceived of as an attempt of the brain to perceptually bridge gaps by interpolating surround features and thereby restore a retinal image that is incomplete due to a lesion or occlusion. At the same time, fading and filling-in precludes us from seeing stimuli that are clearly before our eyes due to local adaptation. An object that does not move will disappear from view and become embedded in the background during prolonged fixation. This is a striking example of interpolation by the brain that has attracted much research in recent times.

**Foveal blue scotoma.** There are no short-wave cones present in the innermost part of the fovea (an area less than 0.5 deg in diameter). Therefore, we cannot see a small spot of 470 nm projected to the centre of vision. However, when we look at the blue sky, we do not normally see a hole or even a dark spot, as we would expect. The reason again is: filling-in. No rods are present in the foveola either. Therefore, if we want to see a faint star in the night sky, we aim our gaze slightly to the side of the star, without being aware of the central blind spot.
Transient blindness in dark and light adaptation. In normal vision, we are always optimally adapted to the prevailing luminance. However, when we enter a dark cave because a wild dog is chasing us, we do not see much for many seconds, and similarly when we leave the cave and re-enter daylight, the bright light temporarily blinds us. The same is true in a poorly illuminated tunnel. These are examples illustrating that under some conditions the sensitivity of the eye does no catch up fast enough with changes in the environment, leaving us with a large blind spot. People who, for example, adhere to a diet of mostly rice and eat neither carrots nor fish are likely to develop (a reversible) night blindness due to a lack of vitamin A.

Binocular rivalry. If two different shapes overlapping each other are presented to the two eyes in a stereoscope, only one of the shapes will be perceived, while the other one will be suppressed. However, a few moments later, the suppressed shape will emerge, whereas the initially perceived shape will become invisible. Thereafter, the two shapes will alternate periodically, due to a mechanism that is not yet fully understood. Sometimes, the two shapes break up, and one sees a patchy combination of both. Apparently, the brain when confronted with competing monocular inputs tries to resolve the conflict by switching back and forth. In children suffering from untreated squint or strabismus, a similar kind of suppression affects the cortical processing of the input from one eye and leads to permanent stereo-blindness (among other deficits). There are more examples where blind spots are created despite the presence of stimuli falling onto the eyes.

Inattentional blindness. Paying attention to one kind of event may render you blind to another. This was beautifully demonstrated when two teams of players at Harvard tossed a ball back and forth among their team mates, while spectators were asked to count the number of passes for one team. While this was going on, a person disguised as a gorilla walked into the scene, stopped briefly in the middle and thumped his chest, before marching out. Surprisingly, hardly any of the spectators noticed the gorilla, although he was quite large and conspicuous. This suggests that we can be ‘blind’ for events that we do not attend to.

Change blindness. When a scene is briefly presented and shortly thereafter we see the same scene again with an essential part missing or significantly altered, observers will be unable in many cases to tell whether or not there was a change. In this way, you can make a person and, indeed, an entire house disappear, owing to the fact that our span of visual awareness is quite limited. The failure to notice even substantial changes casts doubt on peoples’ ability to testify as witnesses in court. Obviously, we are not as good observers as we think. Try to draw a familiar scene from memory—for example, your home or your laboratory. Although you have seen both many times, you will be astonished by how many such blind spots you have.

While the aforementioned blind spots occur in normal vision, there are many examples where vision is missing due to ocular or neural trauma or inherited defect. In the following, I mention a few from Breitmeyer’s book.

Retinal scotoma. When you injure your retina, for example as a result of a blow to the eye or by accidentally looking into an intense light, such as a laser, you are likely to end up with scotoma. This is an area of the retina within which you do not see. In the past, welders have damaged their eyes by not wearing protective goggles, and the famous British psychologist Kenneth Craik blinded himself in one eye by looking at the sun. (Not to be repeated!) A scotoma is comparable to the physiological blind spot, and in most cases it is permanent (irreversible). Eye doctors produce small retinal scars by laser coagulation in an effort to reattach a detached retina. However, despite the presence of such scars, patients do not report any scotomata when looking at a white wall. This is because of filling-in. It would be of interest to find out whether the same holds true for patients in the initial stage of age-dependent macular degeneration.

Cortical scotoma. When injury occurs to the visual pathway or brain, due to a stroke, tumor, or gunshot wound, the resulting scotoma will vary not only in size and location, but also in shape. A defect sometimes as large as half the visual field (hemianopia) can result. Such cortical scotomata are rather stable and change little over time, although there have been attempts at rehabilitation by frequent presentation of stimuli at the edge of the scotoma. However, not infrequently and despite their large size, such blind spots are first noticed only when patients start bumping into the door frame or hitting the entrance to their garage.
Neglect. A syndrome similar to hemianopia is unilateral neglect. Such patients have an intact visual field, but appear to be unaware of an entire hemifield, not unlike some hemianopes. A telling case is the painter Anton Raederscheidt, who following a stroke produced a self-portrait with only half his face present, but recovered some of his visual field in the course of one year. When the neck muscles of such patients are stimulated electrically, the neglect border can be shifted, thereby enlarging the visual field. The neglect border is body-centred, whereas the hemianopic border is eye-centred. To find out whether a patient suffers from one or the other, he need only be asked to turn his head and report the border of the hemifield. In the case of neglect, the border’s location in space remains the same.

Colour blindness. Ten percent of the people in a normal population cannot see and discriminate some of the colours in the visible spectrum because they are lacking a type of photoreceptor, and a few cannot see any colours at all. These people are called colour anomalous or colour blind. The most frequent form of colour blindness, red–green blindness, is inherited due to a sex-linked, recessive gene. This condition may preclude the affected persons from assuming certain jobs where normal colour vision is required—for example, airplane piloting or firefighting—and may also be an impediment to obtaining a bus driver’s license. In the dark-adapted stage we are also colour blind because the cones are not functioning in scotopic vision. Yet, we rarely realise it, as we remember the colours of most objects from memory. An extreme case of complete colour blindness occurs in rod monochromacy, when there are no cones in the retina whatsoever. Knut Nordby, the world’s best-researched rod monochromat, writes in a memorable recollection of his childhood that he did not know he was lacking colour vision until told by his teacher in school.

Cortical colour and motion blindness. When brain damage occurs to visual area V4, colour vision will likely be affected. In this case one speaks of cortical colour blindness or achromatopsia. Alternatively, when area V5 (MT) is damaged, motion blindness, or cortical akinetopsia, will result. A woman who suffered from cortical motion blindness reported that she was unable to see an oncoming car progressively advancing toward her. Instead, she saw the car at increasingly closer locations until it had passed by. Similarly, when she poured coffee into her cup, she could not see the level of coffee gradually rising, but only saw a progression of stepped levels until the coffee spilled over.

Face blindness. Face blindness or prosopagnosia typically occurs after a stroke causing bilateral damage to the fusiform face area in the temporal lobes. Afflicted patients can no longer recognise their spouses, family members, and close acquaintances because of a failure to process configural or component facial stimuli. This syndrome is frequently associated with cerebral achromatopsia and suggests a cortical area of high functional specialisation (module). Face blindness is an important field of research that has blossomed during the last decade. There is also a congenital form of face blindness found in about 2% of school children.

Blindsight. This is a condition in which cortically blind people (V1 partially missing) behave as though they have some residual vision left. For example, if asked whether they see a flash of light in the defective hemifield, they say no. However, if they are asked to point in the direction of the presumed light spot, they will point correctly better than chance. Such patients have also been reported to be able to discriminate (to some extent) between colours and navigate around obstacles placed in their path. Evidently, this information reaches them via the so-called second visual system, which connects the retina via the superior colliculi to the motion area MT. This system is older and faster than the primary retino-geniculate system, but has poor spatial resolution. Yet, in all instances, patients are unaware of their blindsight.

Breitmeyer’s amply illustrated book is unique as it covers the phenomenology, psychophysics, neurophysiology, and neuropsychology of blind spots, many of which are topics of recent research. It is also fun to read, as the author conveys the breadth and depth of his insights in a readily understandable, entertaining manner, enriched by a wry sense of humor. Shrewd references to his own life reflect his joy in writing this book, and in many ways Breitmeyer is like Oliver Sacks, but writing at a higher academic level. A few terminological inaccuracies and stylistic informalities fall under the agnosias, which the author discusses (in Chapter 6). But there is never a moment when the reader is tempted to turn a blind eye. Occasional anecdotes are strung in and add to the scientific discourse, prompting one to finish this book in a single sitting. Toward the end there are even excursions into how failures to see occur in visual art and cognition (cognitive blind spots),
paving the way for future research. The book ends, appropriately, with an epilogue on love, bringing us back to the epigraph from “Krishna speaking” at the beginning.

Readers in psychology, the neurosciences, as well as the humanities will find *Blindspots* an insightful treatment of the many unexpected and often glaring instances of how humans can be unaware of what they are missing, by an author renowned for his scholarly research on meta-contrast and masking, who has poured his fascination and enthusiasm into a book that is as scientifically rewarding as it is enlightening. I highly recommend this remarkable book to anyone interested in the study of conscious perception, attention, and, indeed, the philosophy of how we see and conceive of the world around us.

Lothar Spillmann, Neurozentrum, Universitätsklinikum Freiburg
e-mail: lothar.spillmann@zfn-brain.uni-freiburg.de
Currently at: China Medical University, Taichung, Taiwan

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