Examples of Blind Spots

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One of the most pressing questions in vision research is why we see the world around us equally sharp, although we should not. Visual acuity is highest in the fovea and falls off sharply to the near periphery. Yet, when we look straight ahead, we do not notice any blur outside the foveal area. How come?

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

There are many unsolved questions attached to this: (i) Where in the brain is the short-term buffer that enables us to store those glimpses? (ii) How do we stitch them together so that they yield a continuous representation of the world? (iii) Which mechanism provides the correct location of those glimpses within the visual field as the world slides across the retina with every eye movement? (iv) Why are there no visible borders separating the individual glimpses or empty spots?

There are also counterarguments to the above hypothesis: Why do we see the world sharply and coherently, even before we have swept it with our eyes (e.g., when we enter a classroom)? Why do we see it equally well regardless of the size of our eye movements (e.g., traversing the scene with one large saccade as opposed to many small saccades)?

Finally, is it true that we see the world before us equally detailed all across? Can we really tell what is on the far left or far right without looking there; even only 10 deg away from fixation? Many years ago I was interested in face recognition in peripheral vision. I placed 5 people in front of me and looked at the nose of the person in the center. I not only KNEW who was sitting on the left and right, I also SAW their faces. But not for long. After some time, those faces in the periphery became increasingly vague, until I could no longer say, whether these were the same faces that I had seen before. Of course, nobody had actually moved. However, if I now asked the two outermost persons to exchange their seats with two new people, I could not tell, not even guess, who these people were. I had to look in their direction to find out. Thereafter, my visual world was again clear and sharp, although not for long. Importantly, blinking (closure of the lids) did not bring the percept back. A foveal refresh was needed.

These observations tell us that eye movements are necessary for our awareness of the world and how long a percept of given detail and eccentricity can be retained.

The sequence of sustained vision, loss of detail and reappearance is reminiscent of what we find in Troxler-type fading under conditions of prolonged fixation.

It would be lovely if somebody would consider doing such an experiment, even casually. I don't think it has been done, although the literature needs to be checked as much experimentation on faces has been going on. Bob Wurtz at this year's VSS nicely summarized the neurophysiology on saccades, and Patrick Cavanagh at ECVP discussed the phenomenology and psychophysics.

Bruno Breitmeyer in his delightful book on "Blind Spots", to be published by Oxford University Press in October, describes a large number of phenomena in normal and defective vision, showing that we have many blind spots of which we are hardly, if ever, aware. I will list a few.

1. Retro-blindness. The world behind us that is outside of our visual fields 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 has, we might be in trouble. Just think of a conductor stepping backward into the orchestra pit. As I have told you before, hares, rabbits, horses and other animals of prey can see behind, because they have lateral eyes telling them how close they are to being caught by their predators. (Ivan Schwab at UCDMC is writing a book on this topic.)

2. Blind spots. Many people do not know that we have two physiological Blind spots where the optic nerve leaves the eye. A small light spot falling onto these areas will not be perceived, unless it casts stray light onto the adjacent areas. Normally, the Blind spot of one eye is "covered" by the functioning retina in the corresponding location of the other eye. Yet, we do not see the Blind spot, even if we close one eye. Why is this? The answer is, because of filling-in of brightness, color, and texture, even motion, from the surround. Filling-in provides us with a coherent picture of the world in the area where we should see a dark hole of 6 by 8 degrees. This is a striking example of interpolation by the brain. We have found that only a small rim next to the boundary of the Blind spot is needed to see it become uniformly filled-in. Kellman and Shipley have shown that only such stimuli are completed whose parts are "relatable" according to the Gestalt factor of good continuation. Gaps in the stimulus will also be filled-in because the brain does not know that there is a gap in the actual stimulus.

3. Foveal blue scotoma. There are no short-wave receptors in the most inner part of the fovea, the so-called foveola. Therefore, we cannot see a small (0.5 deg) spot of

470 nm projected to the center of vision. However, when we look at a uniform surface, we do not see a hole or even a dark spot, as we would expect, if there were a foveal blue scotoma. The reason again is: filling-in. The innermost fovea not only lacks short-wave cones, but also rods. Therefore we do not see a faint star in the night sky when looking directly at it. To be able to see it, we must aim our gaze slightly to the side of the star, so that it projects onto the more sensitive parafoveal rods.

The military, police and surveillance authorities use infrared-converters (heat cameras) and residual light intensifiers to see at night. However, this improvement is due to a technical trick that occurs before the eye, not in the eye itself.

4. Transient blindness in dark and light adaptation. In normal vision, we are always optimally adapted to the prevailing luminance, even when the sun rises or sets. However, when we enter a dark cave because of a wild dog chasing us, we do not see for many seconds, and similarly when we leave the cave and reenter into daylight, we are temporarily blinded by the bright light. The same is true in a poorly illuminated tunnel. These are examples showing that we are sometimes confronted with conditions where the sensitivity of the eye does not catch up fast enough. The neural adaptation that regulates fast sensitivity changes has a span of only 2 log units (a factor of 100), which is large enough for almost all luminance contrasts encountered in our environment. However, the photochemical adaptation that is based on photopigment regeneration is slow, taking many minutes after a full bleach. On the other hand, it has a range of up to 7 log units (a factor of 10 million), covering the full luminance variation from bright sunlight to the darkness at night. People who eat mostly rice and neither carrots nor fish are likely to develop (a reversible) night blindness due to a lack of vitamin A. This is called dietary blindness.

5. 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. This is another example, where vision is absent in the presence of a stimulus shown to the eyes. Sometimes, the two shapes break up, and one sees a combination of them. Presenting two competing monocular stimuli produces a conflict that the brain tries to resolve by switching back and forth.

6. Inattentional blindness. Paying attention to one kind of event may render you blind to another. This was beautifully demonstrated when two teams of players tossed

a ball back and forth among their team mates, while spectators were asked to count the number of ball exchanges for each team. While this was going on, a person disguised as a gorilla walked into the scene, stopped briefly in the middle, waved his hands towards the audience, and marched out. Surprisingly, hardly any of the spectators noticed the gorilla although he was quite conspicuous. This suggests that we can be "blind" for events that we do not attend to.

7. Change blindness. When a scene is briefly presented and shortly thereafter the same scene again with an essential part missing, observers will be unable in many cases to tell whether or not there was a change. In this way, you can make houses, ships and even people disappear, showing that your span of visual awareness is quite limited. The failure to notice even substantial changes casts an important light on peoples' ability to testify as witnesses in court. Obviously, we are not as good observers as we think. Try to draw familiar scenes from memory, for example, your home or your laboratory. Although you have seen them many times, you will be astonished how much you miss.

Whereas the aforementioned examples occur in normal vision and can be seen by all of us, there are examples, where vision is missing due to ocular or neural trauma or an inherited defect. In the following, I mention some examples.

8. Retinal scotoma. When you injure your retina, e.g., by a blow to the eye or by accidentally looking into a laser light or even the sun (DON'T EVER DO IT!), you are likely to end up having a scotoma. This is an area of the retina within which you do not see. It is comparable to the Blind spot, and in most cases is permanent (irreversible). Eye doctors use weak lasers to attach a detached retina, producing scars to hold it in place. In diabetics they also coagulate small retinal arteries to prevent them from bursting and bleeding into the vitreous. Sometimes, as many as a thousand laser burns are needed to fix the retina. However, patients do not report any dark spots (e.g., a sieve with many small holes) when looking at a white wall, presumably because of filling-in. Thus, one might interpret the underlying mechanisms as a repair mechanism that nature gave us to perceptually complete incomplete stimulus or scene in the outer world. Neurophysiological recordings have shown that there are single cells in area V2 of the monkey that respond as though they are mediating continuous contours although the contours are really incomplete. Such cells must receive input from "beyond the classical receptive field." Esther Peterhans and Ruediger von der Heydt have done the pioneering experiments in this field.

9. 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 shape, but also in size. A defect of one half of the visual field in both eyes (i.e., homonymous hemianopia) points towards an injury above the optic chiasm. In comparison, when two different halves are affected (heteronymous hemianopia), the injury is likely lower, at or near the chiasm. The size of a cortical scotoma varies with eccentricity. This is because the retina is mapped differently on the cerebral surface. The fovea and inner 10 deg of the visual field take up about half of the visual cortex, whereas the entire periphery takes up the rest. In other words, the fovea is overrepresented, whereas the periphery is underrepresented. This is called cortical magnification and accounts for the high visual acuity in central vision and the superior sensitivity of the peripheral retina for motion. Thus, injury to the occipital pole will produce a relatively small foveal scotoma, with loss of foveal vision. In comparison, an equally large injury to the more lateral parts of the visual brain will produce a disproportionately large scotoma affecting peripheral vision. Cortical scotomata are rather stable and change little over time although there have been attempts of rehabilitation by repeated presentation of stimuli at the edge of the scotoma. The success of such efforts depends on whether the edge is shallow or steep. In the latter case the gains are negligible.

10. Color blindness. There are a small percentage of people in a normal population who cannot see and discriminate some of the colors in the visible spectrum or who cannot see colors at all. These people are called color anomalous or color blind. Most of the color blindnesses, e.g. red blind or protanop, green blind or deuteranop, are inherited. (Do you know, whether they are autosomal, sex-linked, recessive, or dominant?) These conditions may preclude them from certain jobs where normal color vision is required and may also be in the way of a driver's license. In the dark-adapted stage, we are also colorblind because the cones are not functioning in scotopic vision. A German proverb says: At night all cats are grey.

11. Cortical color and motion blindness. When brain damage occurs to visual area V4, color vision will likely be affected. In this case one speaks of cortical achromatopsia. A retired professor of genetics who also painted portraits, landscapes and stills, no longer saw the colors in his paintings. They were all dull. He also was unable to recognize the people depicted in his portraits because of face blindness or prosopagnosia, which frequently occurs together with cortical color blindness. 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 advancing towards her. Instead, she saw the car in increasingly closer locations until it had passed by. Similarly, when she poured coffee into her cup, she could no longer see the level of coffee gradually rising, but only saw a progression of levels until the coffee spilled over.

12. Blindsight. This is a condition in which cortically blind people (V1 partially missing) behave as though they had some residual vision left. For example, if asked whether they had seen a flash of light in the defective hemifield, they say no. However, if they are asked to point in the direction of the light spot, they will point correctly better than chance. Such patients have also been reported to be able to discriminate between colors and navigate around obstacles placed in their path. Apparently, this information reaches them via the so-called second visual system, which connects the geniculate nucleus (LGN) via the superior colliculi to the motion area MT. It is older and faster than the primary visual system via LGN and area V1. In all instances, patients are unaware of their achievements. (Petra Stoerig has done much research in this field, see her review in TINS).

13. Neglect. A syndrome similar to hemianopsia is unilateral neglect. Such patients have an intact visual field, but appear to be unaware of an entire half of it. As a result they run into the door frame and other obstacles and smash the fender of their car because the garage door "has become too small." A telling case is the painter Anton Raederscheidt who following a stroke portrayed his own face only half, but recovered to some extent in the course of one year. Jim Lackner stimulated the neck muscles of such patients electrically and observed a shift of the neglect border, thereby enlarging their visual field. The neglect border is body centered, whereas the hemianopic border is eye centered. To find out whether a patient suffers from one or the other, one only need ask him to turn his head and report the border of the hemi-deficit.