New insights into the subjective perception of visual field defects

Glaucomatous defects commonly start insidiously at the nasal side of the visual field and their progression is very slow. Still, this hardly explains the nearly unlimited lack of awareness of the glaucoma patient. New insights into the subjective perception of visual field defects can better explain this. The defects are probably filled-in by the brain by use of information received from the surrounding retina, by analogy with the filling-in of the blind spot. Thus, visual stimuli are perceived as arising from the blind area(s). As a result, the glaucoma patient is unable to notice his visual problem until a late stage of the disease, where the brain no longer receives sufficient visual input to allow it to compose an image.

Brain remapping

It was long held, that the brain is only capable of filling-in:

- congenital field defects, namely the blind spot[1] and the angioscotomas caused by the retinal vessels[2].
- field defects acquired during a so-called critical period, early in life, up to the age of seven[3].

It has however become evident, that the adult brain is also able to adapt to acquired defects[4]. This is thanks to the so-called plasticity of the adult primary visual cortex, i.e. the region where the visual input is first received. This plasticity involves activation of long-range synapses between cortical cells, which in normal individuals have only subtreshold effects[5]. So that normal cells, in the cortex surrounding the lesion, take control of the deprived ones. Thus, contrary to long-held beliefs, cortical maps in the adult brain are not predetermined. This not only holds true for the visual cortex, but also for other sensory areas of adult cortex[6-9]. Cortical maps can for instance also reorganize after loss of sensory afferent nerves from a limb [8,9]. Cortical plasticity not only allows the brain to adapt to damage of the nervous system, it also underlies learning processes[10].

Remapping of the visual cortex takes place after all kinds of damage to the retina. The filing-in process is probably responsible for the delay in diagnosing primary open-angle glaucoma and pigmentary retinopathy[11,12]. Further, it is helpful in the treatment of various retinal disorders such as diabetic retinopathy, as it allows us to perform extensive retinal photocoagulation without the patient noticing the defects inflicted by the laser. The filling-in process also occurs during migrainous attacks, and after occipital lesions or other postchiasmal disorders of the visual pathways[12].

Ever since Hubel and Wiesel[3] first began to explore the primary visual cortex with electrodes, our view of how the brain works, has been dominated by studies of the visual system. Lessons from the visual

system are also enlightening our understanding of other cortical regions. Thus, researchers outside the field of ophthalmology have performed the most substantial part of the research into the plasticity of the visual cortex. This is illustrated by the fact that only 5 references to ophthalmic journals are listed in this paper. This probably explains the wide gap between basic research and clinical ophthalmic practice. The first key paper on the plasticity of the visual cortex has already been published 13 years ago[4] and a lot of papers on the subject were published in the top journal Nature. Yet the impact of the effects of cortical plasticity on daily clinical practice still remains largely unrecognized by ophthalmologists.

Characteristics of the plasticity of the adult visual cortex

Reports

Because acquired lesions of the retina or visual pathways vary markedly between patients, it is practically impossible to systematically investigate the characteristics of cortical plasticity in selected eye disorders. Individual reports of the filling-in process are however available, amongst others even a historical one. King Charles II (°1630, England) whose own father was beheaded, used to "behead" his courtiers in a harmless manner, probably by gazing at them in such a way that their heads were positioned in his visual field defect, the gap being filled-in by the surround. More recently a researcher, he himself suffering from a migrainous attack, reported that while he was talking to a friend, he looked just to the right of his friend's face whereon his head disappeared[13]. His friend's shoulders were still visible, but the wallpaper behind him seemed to extend right down to his necktie. Prompt visual field testing revealed a large area covering about 30 degrees, of total blindness just off the macula.

Research

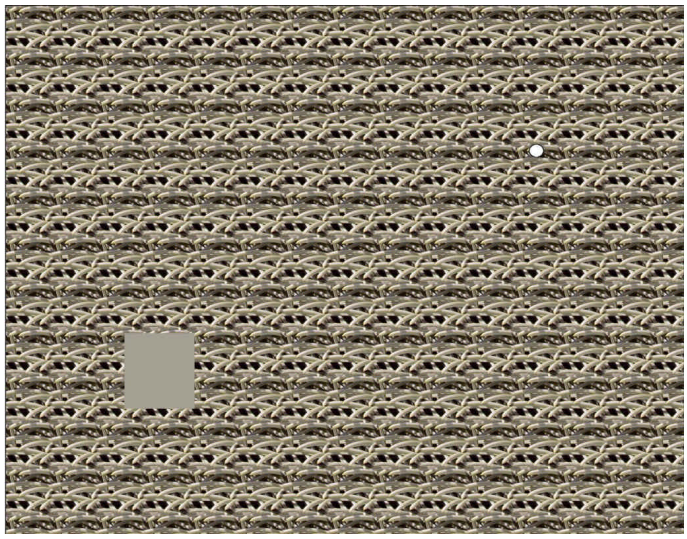
Neurophysiological tests

Neurophysiological tests measure electrical activity of cortical cells in experimental animals, such as the cat or monkey, among other things after destruction of selected areas of the retina with a laser. This research tells us that cortical reorganization depends on the potentiation of existing but normally ineffective (subtreshold) synapses, in a highly organized system of long-range horizontal connections in a large zone of the visual cortex[5]. When retinal lesions remove a competing source of activation, the subtreshold signals gradually become effective in activating neurons to suprathreshold levels. This process may take some weeks to months.

It should however be remembered that considerable reorganization can also be expressed subcortically[9,14], as has been shown after loss of the sensory afferents from the forelimb in monkeys. Thus, subcortical regions possibly contribute to the visual filling-in process. Further, the development of new connections might also play a role in the plasticity of the visual cortex[15], by analogy with other brain regions and the spinal cord[9,16,17]. The growth of new connections, previously viewed as unlikely, may in fact be very important in large-scale reactivation[14,16-18].

Psychophysical tests

Psychophysical tests involve normal human subjects, who are asked to look at a display, and report what they observe. Field defects are imitated by small homogeneous areas that subtend 1.5° to 2.8° and have the same luminance as the background stimulus. The subject is asked to fixate a spot, located about 6 degrees from this area, while various visual stimuli are presented.



This display allows you to check your own cortical plasticity. With strict fixation at the white fixation point, the homogenous gray square vanishes within 10 seconds and gets replaced by the pattern from the surround.

The average time for fading of the homogenous area and filling-in is 5 seconds[4]. The visual information that is filled-in, comprises:

- color
- patterns or texture
- motion

Filling-in, can occur separately for texture and color, suggesting separate mechanisms[4]. Color fills in first. And, although one would, intuitively, expect motion to be an enormous challenge to the brain, nothing could be farther from the truth. Motion actually increases the brain's capacity of filling-in. This has been shown, by at least two different experiments. First, when a line is presented across a masked area, its completion is accelerated, when the line is moving[19]. Alternatively, if the background presented to the test person has a dynamic random noise, for example when it is a flicker on a TV screen, the filling-in of a masked area is also facilitated[20].

Further, motion perception is not suppressed like other visual information[4]. So that, for instance, when a person is driving a car, the part of the image that is being filled-in will move just like the surround.

Interestingly, different mechanisms can compete for filling-in the blind area[12]. For example, when a black and a white line, perpendicular to each other, are shown crossing a blind area, only the one which attention is being paid to, looks continuous. Or if two perpendicular lines have different lengths, only the longest one looks continuous[21]. This competition shows that the filling-in process can be affected by brain activity arising in, or visual information received from, areas distant from the blind area.

Implications for visual field testing

Cortical plasticity greatly affects the results of the Amsler grid test. Nearly half of the scotomas resulting from macular disorders are not detected[22], probably because they are being filled-in[12]. The majority of field defects smaller than, or equal to, 6° in diameter are filled-in completely when tested with the Amsler grid. Larger field defects may fill-in only partly, the process starting from the surround towards the center of the blind area, so that the defect perceived by the patient during Amsler grid testing is smaller than the actual one.

Implications for the perception of glaucomatous field defects

The plasticity of the visual cortex of glaucoma patients specifically, has not been systematically analyzed. The abovementioned psychophysical tests however, give us some idea of what the glaucoma patient experiences.

In an early stage, the brain is probably able to compose a very plausible image, by filling-in the field defect with the colors, forms and textures of the surround. Yet, objects in the foreground, which are positioned completely in the blind area, are probably not perceived by the patient with the affected eye. This is because the brain cannot be expected to form an image of an object of which it receives no information at all. Apart from the problems this may cause in daily life, this might also lead to hazardous situations. It is for instance conceivable that when a patient is driving a car he might not notice, for instance, children suddenly crossing the street, if their image happens to be positioned completely in the field defect whilst it is not seen with the other eye. This is all the more dangerous, as the patient is unaware of any visual problem.

As the glaucomatous disease progresses, gradually less objects are seen in the periphery because increasingly more objects are completely located in the expanding blind areas. Further, objects located at the border of the field defects probably adopt a rather peculiar aspect. Lines are stretched, and colors spill over, into the field defects. Yet, the patient is probably still not alarmed as he is unable to scrutinize these distortions in the periphery of his visual field, and the defects are still concealed in the background colors and forms of the surround. Thus, glaucoma most often continues to progress unnoticed, until a late stage of the disease where visual input has decreased to such an extent that the brain is not able anymore to compose an image.

Patients who have paracentral field defects however, often report that they sometimes miss parts of the texts they are reading. The filling-in of the field defect with the background color of the sheet of paper and some text-like forms, can explain why they do not perceive black or gray spots. But as the brain probably does not succeed in producing a sensible text, these patients do notice that they have a visual problem.

Two matters hamper our understanding of the perception of glaucomatous field defects at this time. First, the asymmetry of the field defects in both eyes, might affect the filling-in process. The possibility cannot be excluded that the filling-in of a blind area is refined, if information on this area is received from the other eye. Second, as motion increases the brain's capacity of filling-in, the brain might be able to produce some image of an object that is completely located in a field defect for a short period of time only, when the object or the patient is moving.

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