

Subjective experience of visual field defects caused by cortical infarctions

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Introduction

Visual field defects caused, for example, by infarction of the occipital cortex, are often not subjectively detected by the patient, similar as is the case with the blind spot. This lack of awareness regarding blind portions of the visual field is probably due to mechanisms of filling in that will perceptually fill in the defect with patterns present close to its borders: within our blind spot, we usually perceive the type of pattern displayed at the borders of the blind spot. We developed dynamic visual stimuli that are difficult for the visual system to fill in and hence allow patients to subjectively experience their visual field defects.

Methods

In order to make patients aware of their defects we presented 12 different stimuli to the right eye of so far 25 patients suffering from recent cerebral infarction. Twenty-two patients had experienced an infarction in the occipital region, while 3 had suffered from infarction of the medial cerebral artery. Some examples of these stimuli are shown in figure 1, albeit as stationary samples of the originally dynamic patterns. The dynamic noise pattern symbolized in figure 1 pattern 1 in reality appears as a form of visual noise, similar to the impression on a TV-screen when the antenna is disconnected, or no programme broadcast. These stimuli, presented on a colour monitor, subtended over an area of the visual field with a diameter of more than 60 deg. Luminance, colour, orientation, position, or else stereoscopic depth of the stimulus elements varied rapidly in order to preferentially stimulate different submodalities of vision. Patients sat at an observation distance of 26 cm, fixated steadily on a central fixation spot, and indicated whether there were any regions in the stimulus where the pattern appeared to differ from the remainder of the visual field. The outlines of such field defects were then fed into a computer by means of a graphics tablet. Afterwards, Goldmann perimetry was performed on each eye.

Results

Most of our patients indeed experienced, under appropriate conditions, at least parts of their visual field defects (as defined by Goldmann perimetry) when looking at these stimuli. The spatial extend of the defects experienced subjectively was generally clearly smaller than the extend of the scotomata present in the Goldmann perimetry that was performed in all patients as a gold standard. Fifteen of the patients showed a good or even excellent correlation between the defects showing up in Goldmann perimetry on one side and in the different patterns of component perimetry on the other hand. Examples of such good correlations are presented in figure 2 a,b. Four other patients out of the 19 we tested so far with clear scotomata in Goldmann perimetry, however, were able to completely fill in their defects, and did not perceive any defect in component perimetry in spite of large visual field defects as shown in figure 2c. Table 1 summarises the results by indicating, for each individual patient, the cause for his or her visual field defect and the location as assessed by Goldmann perimetry as well as by component perimetry. There were no false positives. All six patients with intact Goldmann visual fields also experienced normal component perimetry. An interesting sideresult is that some patients differed systematically regarding the extend of subjectively experienced field defects depending on the pattern of component perimetry employed, for example with larger defects for patterns testing colour vision than for those testing motion perception, or vice versa.

Conclusions

Obviously, not for all types of stimuli, visual field defects can be filled in and some people are even able to perceive their blind spots when looking at our stimuli. The reason for the strong inter-individual differences between patients remains to be clarified. Possibly, the patients able to fill-in are also able to achieve "blindsight", while the others are not. This hypothesis is presently under investigation. Component perimetry seems to be a moderately successful screening method to detect visual field defects caused by cortical damage (earlier investigations have shown that it is an excellent method to detect defects caused by retinal damage). The majority of our patients with visual field defects caused by cortical lesions were able to subjectively experience these defects when confronted with patterns of component perimetry. The method seems also able to differentiate between lesions that affect ALL submodalities of vision, e.g. when the primary visual cortex is damaged, and those primarily involving, for example, colour vision (predominant involvement of V4) or motion perception (predominant involvement of V5). We presently develop methods to quantitatively investigate the defects in patients with defects in only some of the patterns of component perimetry while not in others to obtain a more objective correlate to the so far subjective measurements.

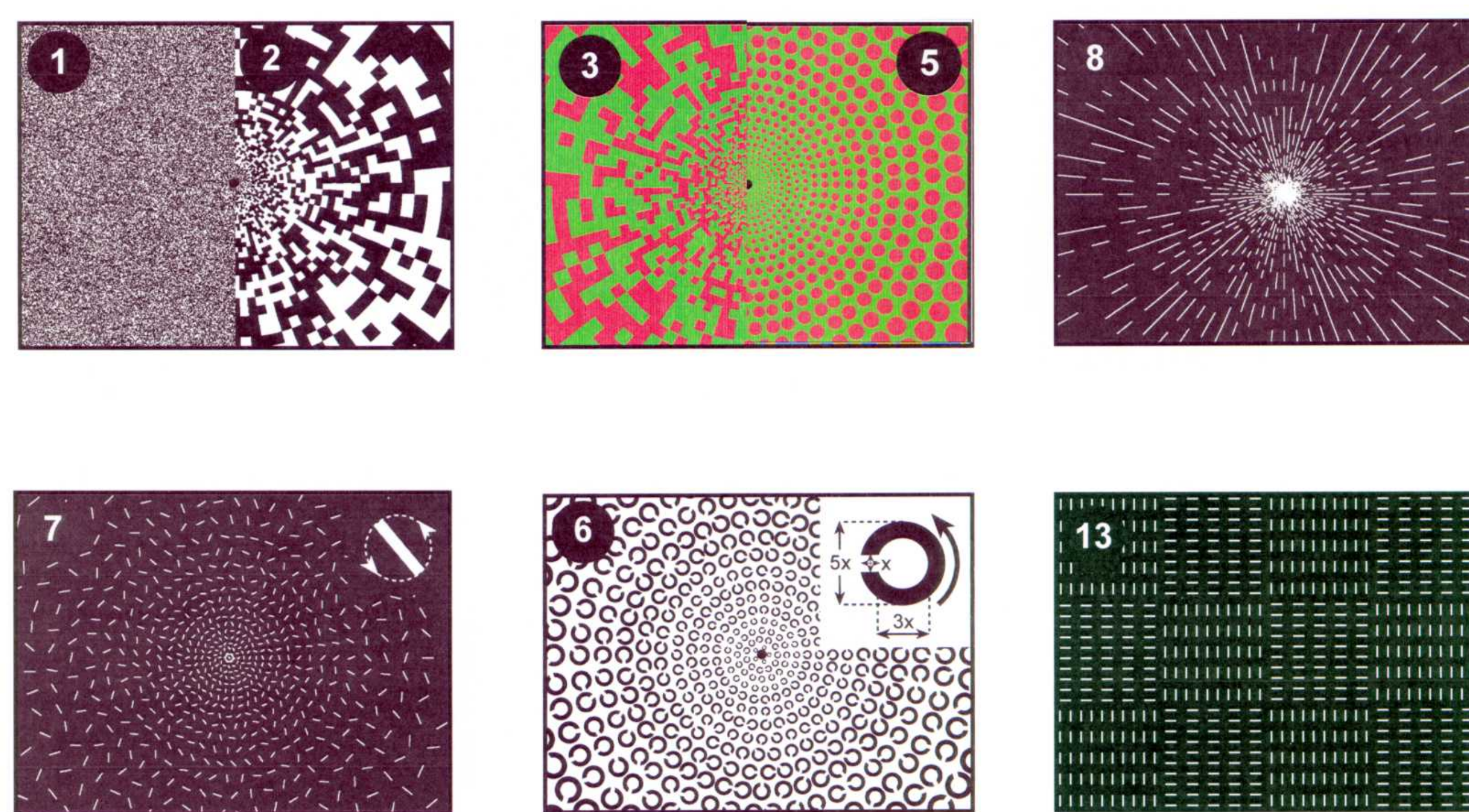


Figure 1: Examples of stimuli

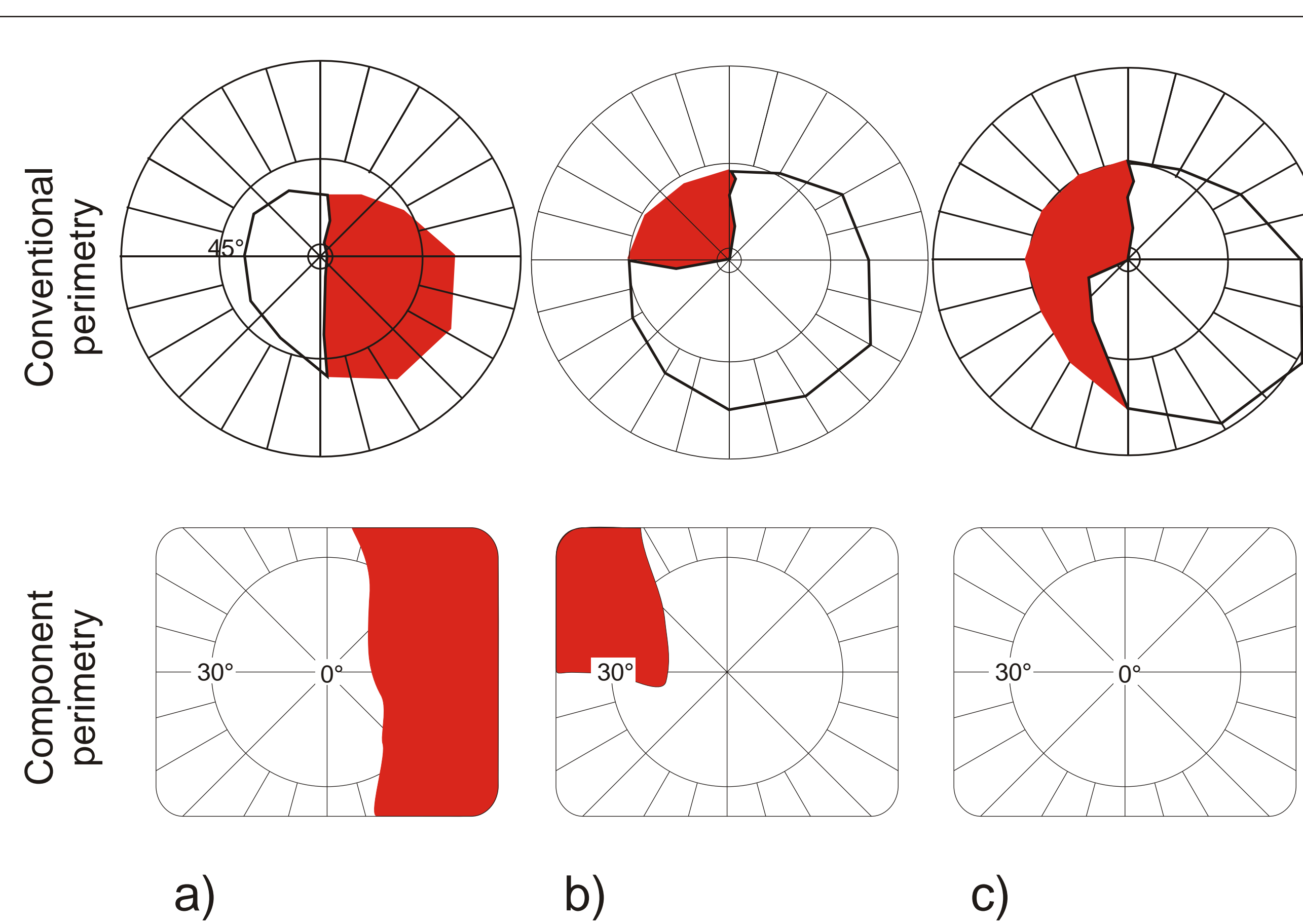


Figure 2: Conventional and component perimetry

Patient	Region of infarction	Date of birth	Goldmann	Goldmann Perimetry	Component Perimetry
1	bilateral posterior	4/20/42	pos.	left incompl. hemianopia + right lower quadrantanopia	pos.
2	right posterior	11/6/60	pos.	left hemianopia	pos.
3	left posterior	12/22/33	pos.	right hemianopia	pos.
4	left posterior	3/29/30	pos.	right upper quadrantanopia	pos.
5	right posterior	5/10/15	pos.	left hemianopia	pos.
6	left posterior	9/27/38	pos.	right hemianopia	pos.
7	left posterior	7/31/52	pos.	right upper quadrantanopia	pos.
8	right posterior	1/19/29	pos.	left upper quadrantanopia	pos.
9	right posterior	3/23/47	pos.	incompl. left lower quadrantanopia	pos.
10	left posterior	6/18/38	pos.	right hemianopia	pos.
11	left thalamus	5/5/54	pos.	incompl. right hemianopia	pos.
12	left posterior	1/23/21	pos.	incompl. right lower quadrantanopia	pos.
13	left posterior	4/8/23	pos.	incompl. right hemianopia	pos.
14	left posterior (injury)	2/26/23	pos.	right hemianopia	pos.
15	right posterior	5/30/37	pos.	left hemianopia	pos.
16	right medial & posterior	9/17/74	pos.	left lower quadrantanopia	neg.
17	left posterior	6/18/46	pos.	incompl. right lower quadrantanopia	neg.
18	right posterior	5/14/46	pos.	incompl. left hemianopia	neg.
19	right temporal & posterior	9/14/27	pos.	left hemianopia	neg.
20	right posterior	9/27/29	neg.		neg.
21	right posterior	6/25/38	neg.		neg.
22	right posterior	10/25/28	neg.		neg.
23	right internal capsule	3/30/67	neg.	control	neg.
24	multiple small infarctions	8/30/46	neg.	control	neg.
25	left medial	3/13/20	neg.	control	neg.

Table 1: Results for each patient