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# The Discovery of Visual Field Representation in the Brain

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## Abstract

This article comprises a historical review on the discovery of the visual cortex in the human brain. Studies of wartime victims by Inouye and Holmes, animal and imaging studies are discussed. The phenomenon of macular and temporal crescent sparing and its anatomical basis are explained. A unique case from our neuro-ophthalmological practice is presented and interpreted in light of the key studies.

## Introduction

The study of cortical representation of visual function is a quest to understand the true function-anatomy relationship between visual field and its cortical representation. It started a century ago with observations by ophthalmologist Tatsuji Inouye[1] and a decade later by consultant ophthalmologist Gordon Holmes and Lieutnant-Colonel Lister[2]. Both studied wounded war survivors, the former of the Russo Japanese War in 1904-1905 and the later in the First World War (1914-1918). Holmes and Listers work yielded a manuscript published in the British Journal of Ophthalmology in 1918 later referred to as *the Holmes maps*. The work provides a prototypical model of our current understanding of visual cortical function based on the study of soldiers with traumatic brain injuries correlating anatomical location of an injury with the respective visual field defect. The work was refined by several authors over time, mainly through observations in animal studies. The advent of modern day cranial imaging led to a revision of the classic Holmes map: McAuley and Russel used computer tomography to correlate visual field defects in stroke patients with visual pathway lesions[3]. 12 years later in 1991 Horton and Hoyt provided a revision of the classic Holmes map in another landmark paper by correlating visual field defects with magnetic resonance scans[4].

In this present review we will outline the historical development of the study of visual cortical anatomy. Also, we will discuss a recent clinical case challenging our current concept of visual cortical function.

## Studies of Wartime Victims: Inouye and Holmes

Inouye was a Japanese Ophthalmologist trained in Tokyo before the outbreak of the Russo-Japanese War 1904-1905. He served as a physician in the army where he examined patients who were injured by bullets and subsequently experienced loss of vision. At that time the Russians introduced a new type of rifle, called the Mosin-Nagant Model 91, which shot bullets at a much higher velocity which resulted often in a complete wound penetration without causing

widespread damage to the surrounding tissue. Therefore, the wounded soldiers often survived their injuries and had more demarcated penetration wounds, which facilitated the conduction of Inouye's experiments: He studied and plotted the visual fields of 29 patients as well as the bullets trajectory and correlated the respective lesions by drawing the defects into an introduced coordinate system (Figure 1). Although the proportions were not correct by today's understanding, Inouye recognized that a large portion of the visual cortex is devoted to central vision[1]. A decade later during World War I (1914-1918) Lister and Holmes conducted research on the same principles as Inouye by working in military hospitals in France over 18 months. The result of this mission was "The Holmes maps", which is – like Inouye's work - an excellent example of a topographic map developed through empiric scientific work. The maps which confirm Inouye's work were made in the same principle but were presented in a more understandable and illustrated way. They demonstrate representation of the contralateral hemifield of vision in each cerebral hemisphere. As Figure 1 shows, Holmes realized that the vertical meridian of the visual field is represented along the perimeter of the visual cortex whereas the horizontal meridian runs along the calcarine fissure. The central retina is mapped on the posterior pole and the visual field periphery on the anterior part of the visual cortex. Horton and Hoyt later calculated that both Inouye and Holmes assigned 25% of the surface area of striate cortex to the central 15° of vision. Holmes and Lister also realized that the macula has a much larger representation in the visual cortex than peripheral retinal areas – a fact that he highlighted in his famous 1944 Ferrier Lecture[5] and that was confirmed later by Spalding[6] in 1952 who had collected the largest set of penetrating head injuries. However, Holmes and Lister did not accept an observation Inouye made in his studies: he found that even soldiers with large cortical defects often had a central remaining vision. He was not able to deduct a theoretical explanation of this "macular sparing phenomenon". This remained a scientific mystery for the coming decades.

## Animal Studies

Talbot and Marshall[7] presented 1941 in their publication called "physiological studies on neural mechanisms of visual localization and discrimination" an experimental arrangement on cat and Rhesus monkeys shown in Figure 2 to study cortical representation of the visual field. The animals head and gaze were fixed using a special head frame and a wire ring around the corneal limbus. Then visual stimuli were presented on different locations on a tangent of the animal's visual field. Electrodes in the cortex deflect action potentials generated in the neurons when excited by the light stimulus. The experiment produced a topical map and confirmed that there was a magnification factor between central visual field and cortical surface. This experiment also excluded a bilateral representation of the macula.

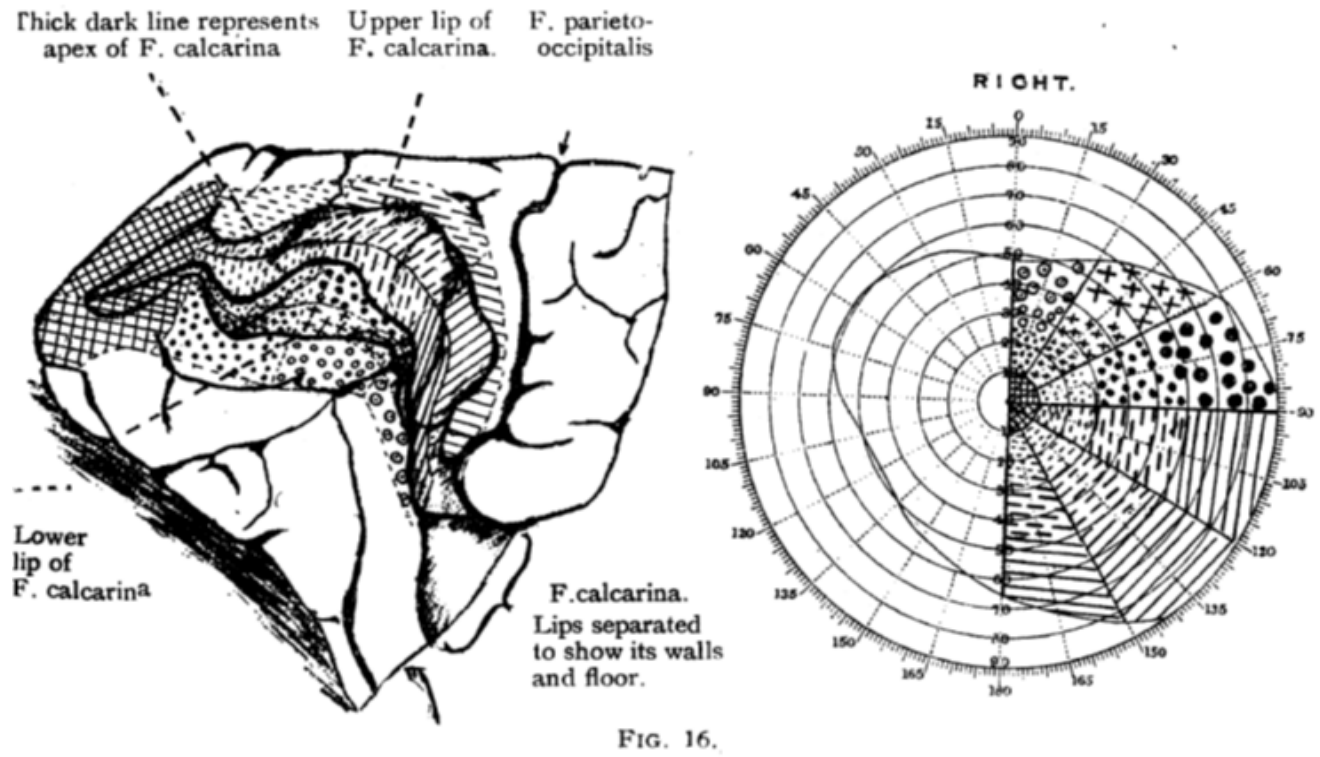


**Figure 1:** Correlation maps of anatomical region and visual field according to Holmes, 1918
Source: Holmes G. Disturbances of Vision by Cerebral Lesions. *Br J Ophthalmol*. 1918;2(7):353-384

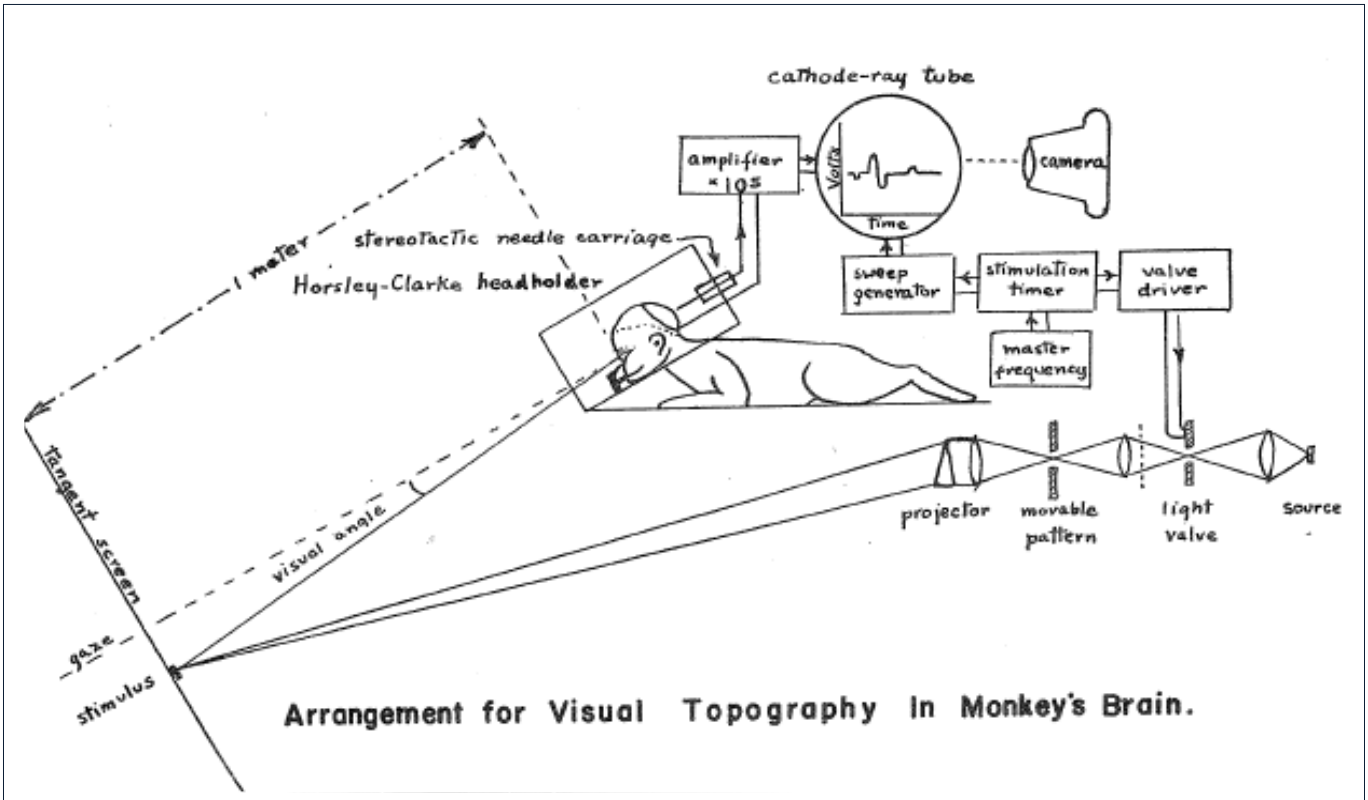


**Figure 2:** Experimental arrangement for visual topography from Talbot and Marshall: Physiological studies on neural mechanisms of visual localization and discrimination
Source: Talbot SA, Marshall WH. Physiological Studies on Neural Mechanisms of Visual Localization and Discrimination. Am J Ophthalmol. 1941;24(11):1255-1264.

Daniel and Whitteridge[8] published 1961 the first complete map of macaque striate cortex based on microelectrode recordings in macaques. Their contribution is noteworthy because they provided a more detailed mapping by taking account of the calcarine sulci by taking a mathematical approach to provide a geometrical model of the visual cortex. This approach was typical for a time where the mathematical field of topology gained in popularity. Based on this model, a magnification factor was elaborated, meaning the millimeters of cortex representing 1° of visual field at a given point. A ratio of more than 40:1 was found in linear magnification between the fovea and periphery. Van Essen[9], who conducted similar studies, reported in 1984 that between 55%-60% of the surface area of the striate cortex is occupied by the representation of the central 10° of vision. A more detailed understanding of visual cortical physiology based on animal studies was provided in the work of two neurophysiologists Hubel and Wiesel[10] from Harvard and Zeki[11] from University College London who showed great interest in cytoarchitecture.

## Imaging Studies

Introduction of computed tomography (CT) in the 1970s allowed to correlate anatomy obtained through images with functional information obtained from visual fields[3,12–14].The CT studies allowed for good correlation with the Holmes maps although there were limitations to those studies due to the poor image quality of the CT devices of the time. One of the larger studies correlating lesions of the posterior visual pathways with visual fields was conducted by Kattah et al[12] in 1981. He studied 39 patients with thrombotic and embolic strokes, neoplasms and hematomas. McAuley and Russell[3] correlated CT scan and visual field defects in vascular lesions of the posterior visual pathway using fewer cases. They were interested in the phenomenon of macular sparing and analyzed two of their cases hoping to demonstrate tissue survival in the occipital pole by CT imaging. However, the results were inconsistent and did not allow for clear conclusions.

Magnetic Resonance Imaging (MRI), which was invented at around the same time as the CT, was a more promising modality to study brain anatomy. In a landmark paper titled "A revision of the Classic Holmes Maps", Horton and Hoyt showed three clinical cases in which the Holmes map misrepresented the anatomy both in location and size. Another finding was that central vision occupies a greater proportion of the human striate cortex than Holmes portrayed in his retinotopic maps[4].

Horton and Hoyt provided a revision to the Holmes map

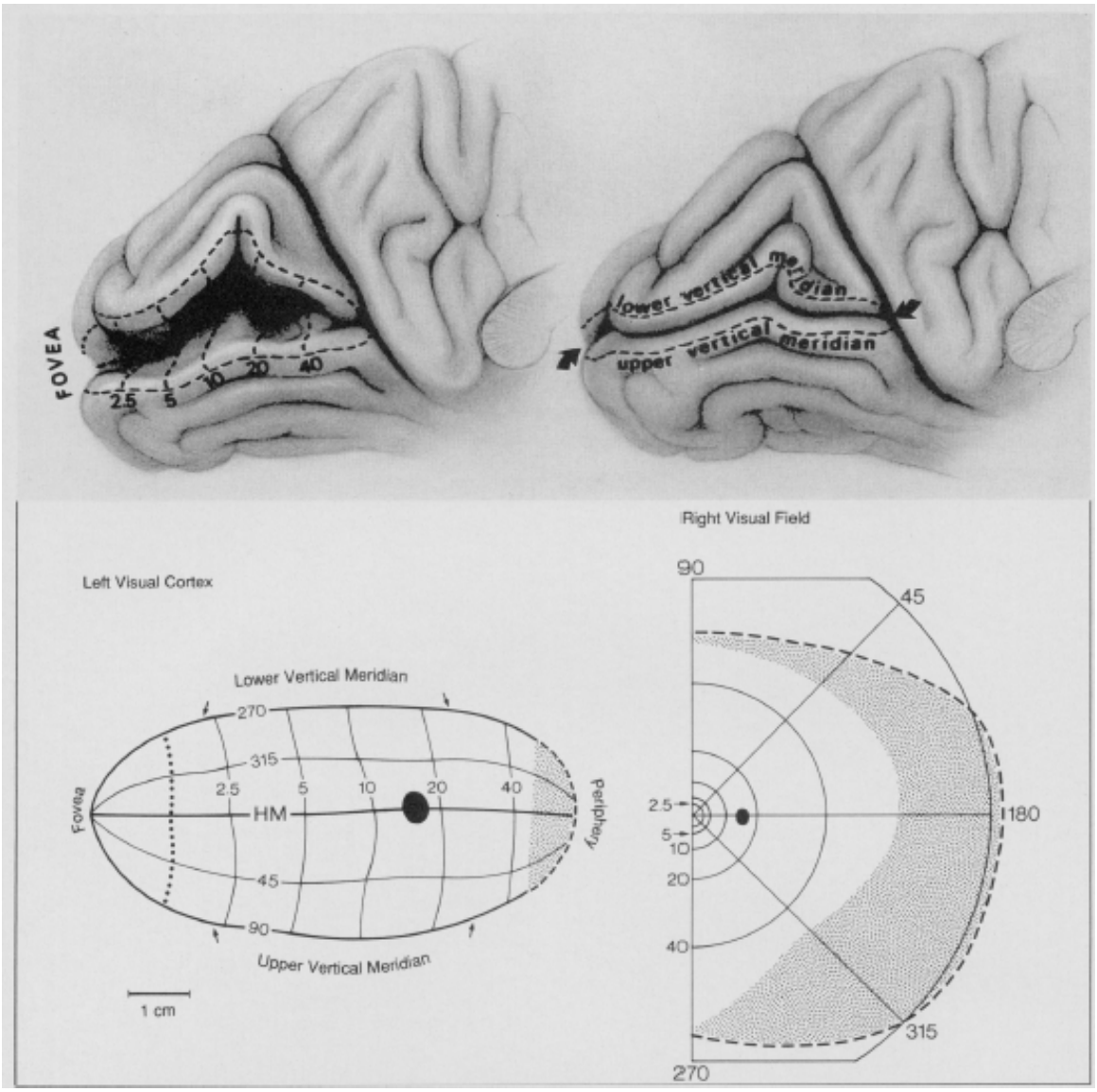


*Figure 3:* Revised Holmes map according to Horton and Hoyt
Source: Horton JC, Hoyt WF. The Representation of the Visual Field in Human Striate Cortex: A Revision of the Classic Holmes Map. *Arch Ophthalmol*. 1991;109(6):816-824.

based on the findings of the Macaques[8,9] and MRI studies. They purported that most of the striate cortex is buried in the depth of the calcarine fissure. This makes it difficult to map retinal locations directly on the cortex. Instead, they schematically unfolded the visual cortex and created a revised planar map. Figure 3 shows that the striate cortex contains a topographic but highly distorted representation of the contralateral hemifield of vision. It depicts how a cortical tissue defect can be correlated with a visual field defect. Horton and Hoyt used their map to predict visual field defects based on MRI detected lesions and proved their map as highly accurate. Their findings were later verified in a larger case series by Robert McFadzean[15] on 26 patients using CT and/or MRI. He was able to confirm that the central 10 degrees occupies at least 50-60% of the striate cortex. Horton and Hoyt calculated that testing the central 30 degree of vision provides information on 83% of the cortex. This fact has implications for visual field testing as someone could argument to focus only on central vision field testing. However, as Landau et al. pointed out in a case series on missing temporal crescents. Testing of just the central visual field can result in overlooking the temporal crescent potentially resulting in mass lesions being missed[16].

Fox et all[17] used a newly developed technique to map the visual cortex using functional positron emission tomography (PET) images in 1986. PET was previously considered unsuitable to differentiate cortical functional areas due to poor spatial resolution. The introduction of an newly developed image-analysis strategy which allowed to

isolate brain areas selectively activated by behavioral tasks by subtracting a paired control-state image from the task-state image, allowed to remove areas not recruited by the task and thereby produced a PET map with a higher spatial resolution. The authors mapped the retinal projection topography of the human striate cortex using this technique dividing it in functional zones of less than 3mm. Using an oxygen-15 tracer and a circular checkerboard pattern as a stimulus, they located the representation of the macula posterior and inferior, the periphery anterior and superior and perimacular regions in between. The experiment produced a map which was claimed to be in agreement with previously performed animal studies by van Essen and Talbot. However, the studies were criticized later by McFadzean et al[15] pointing out morphological misinterpretations as the PET scanning was carried out in the anterior/posterior commissural line and thereby not accounting for anatomical misinterpretations.

## Macula and Temporal Crescent Sparing

Inouye has already discovered the phenomenon of macular sparing in one of his cases. He concluded that this is the result of a dual representation of the macula. This concept was disproven by animal studies[7,18,19]. Horton and Hoyt have published the first case of a magnet resonance image of the posterior cortex of a patient with macular sparing. They explained the phenomenon with the theory of the extremely high cortical magnification factor of the central retina and the fact that in a minority of patients the posterior pole of the calcarine cortex have a dual supply of the middle and posterior cerebral artery. In these patients, the posterior pole remains intact after a posterior cerebral artery infarction due to perfusion by the middle cerebral artery. Because the representation of the central field is extremely magnified in the striate cortex, the likelihood that cortical area representing the macula is spared, is very high. As there is a reduction factor from visual cortex to central visual field, large variations in the amount of surviving cortex from patient to patient will manifest as modest differences in the precise number of degrees of macular sparing.

McFadzean[15] explained the phenomenon of macular sparing similar to Horton and Hoyt, with the blood supply of the posterior cortex. Although there is a considerable variation in the course and distribution of the arteries supplying the striate cortex[20], in 50% of normal brains the calcarine branch of the posterior cerebral artery supplies the entire striate cortex. In the remainder, the occipital pole and operculum are supplied by the posterior temporal or parieto-occipital branch of the posterior cerebral artery or an occipital branch of the middle cerebral artery. In the former situation a calcarine artery infarct would result in a macular splitting homonymous hemianopia but in the latter a similar infarct would allow a macular sparing homonymous hemianopia owing to a collateral circulation from the posterior temporal or parieto-occipital branch of the posterior cerebral artery or an occipital branch of the middle cerebral artery.

Temporal crescent sparing lesions also represent poorly understood phenomena. The temporal crescent is represented at the rostral end of the striate visual cortex. It constitutes less than 10% of the surface area of the striate cortex and receives input only from the contralateral eye. This part of the visual cortex lacks ocular dominance columns [21]. As the cortical area representing the temporal crescent is very small, it is very unlikely that a lesion is located only on the cortical area representing the crescent, therefore those lesions which would lead to a loss of a temporal crescent are very rare.

## Case Presentation

We discuss a case of our own clinical practice which shows macular and temporal crescent sparing at the same time. A 48-year-old man complained of tunnel vision after he experienced a cardiac arrest with out-of-hospital resuscitation. Best-corrected visual acuities were 20/15 on the right and left eye. Pupil responses were unremarkable. Goldmann perimetry showed bilateral homonymous hemianopia with central sparing and presence of temporal crescents (Figure 4). Funduscopic examination was normal on both eyes. MRI demonstrated bilateral posterior watershed infarctions (Figure 5).

This particular visual field pattern with sparing of two visual field regions located on opposite anatomic poles in the visual cortex is rare and presumably results from vascular redundancy and sparing of the anterior and posterior most aspects of the calcarine cortex.

In 1994 Landau et al. published a small case series on the missing temporal crescent. This is the inverse of our case describing a loss of the temporal crescent while the remainder of the visual field is normal. A case of a vascular cranial lesion and a pineal cyst was described in the case series, both resulting in a temporal crescent. It is noteworthy that both temporal crescents showed an asymmetry along the horizontal meridian which corresponds to the cortical location of the lesion. This asymmetry is also present in the spared temporal crescent in our case.

## Conclusion

The understanding of how the visual field is represented in the human visual cortex has developed over one century and has been refined using scientific methods which were pertinent for the time period in which the research was conducted. The temporal crescent and macular sparing phenomenon was observed in several studies using different methodologies and evidence shows that central vision is not bilaterally represented in the human visual cortex.

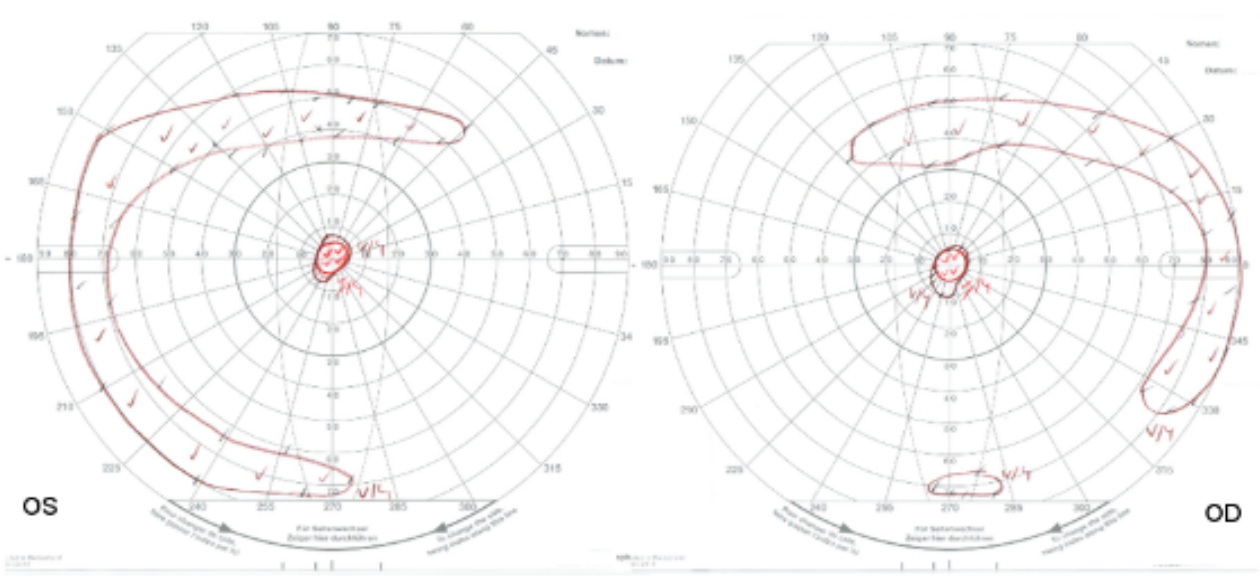


**Figure 4:** Goldmann kinetic perimetry 6 months after cardiac arrest shows bilateral central and temporal crescent sparing homonymous hemianopias
Source: Neuro-ophthalmology department, Eye Clinic of the Hospital of Canton of Lucerne, Lucerne, Switzerland

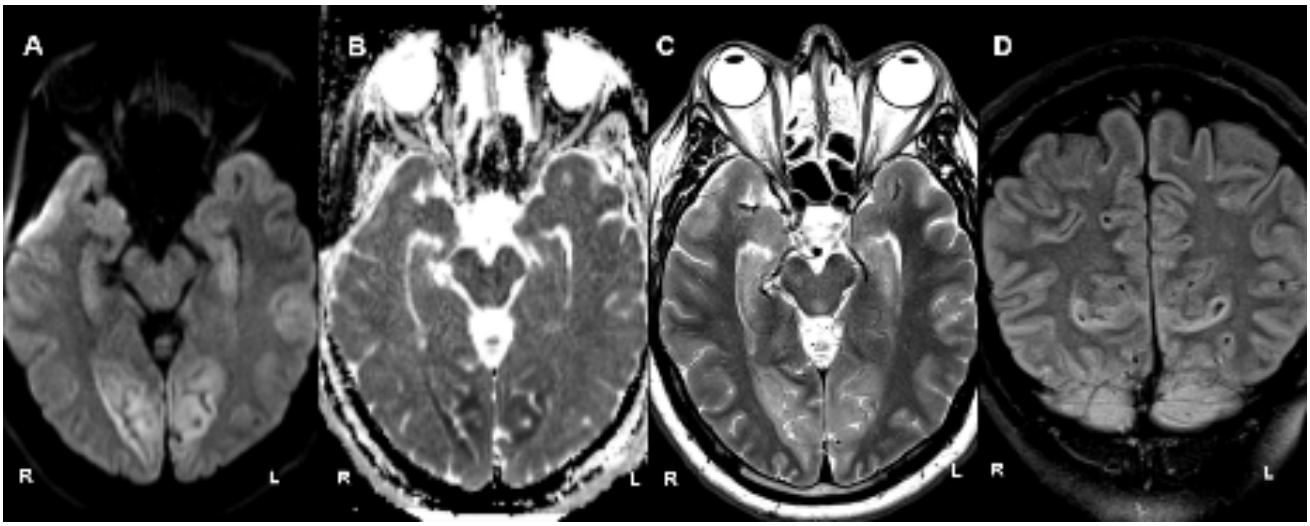


**Figure 5:** Axial diffusion-weighted imaging (a), apparent diffusion coefficient (b), T2-weighted (c), and coronal fluid-attenuated inversion recovery (d) MRI obtained 3 days after cardiac arrest demonstrate bilateral calcarine cortex signal abnormalities
Source: Neuro-ophthalmology department, Eye Clinic of the Hospital of Canton of Lucerne, Lucerne, Switzerland

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