

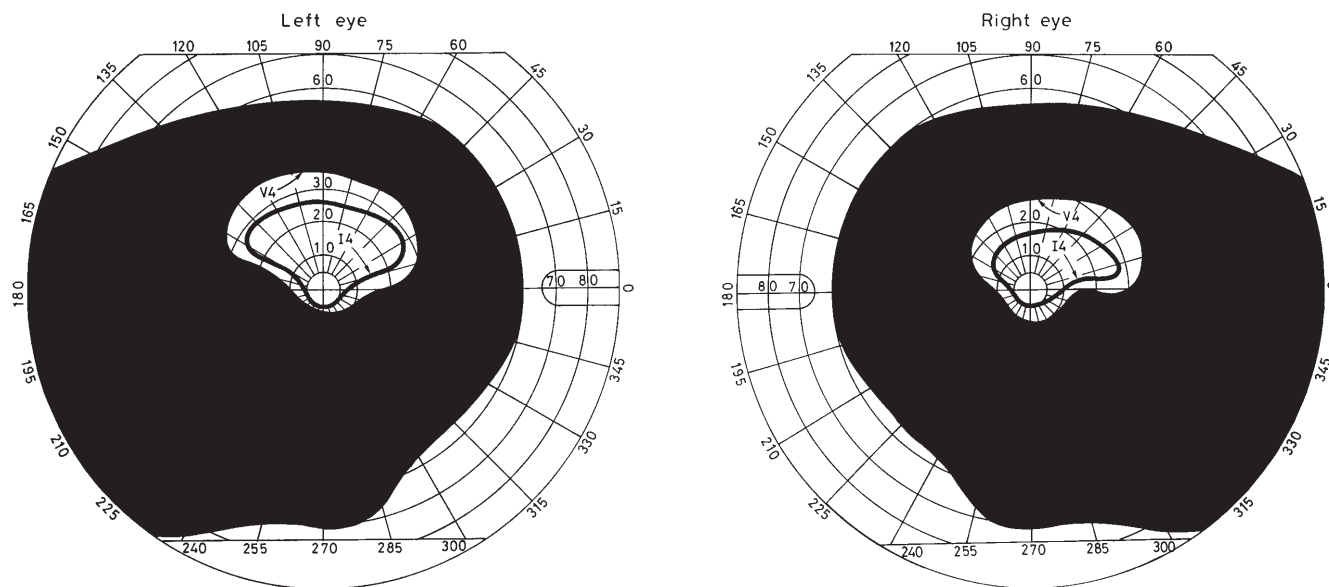
Vincenzo Silani  
Stefano Zago  
Mario Farabola  
Guglielmo Scarlato

### Macular preservation with bilateral visual field defect caused by embolic occlusion of the posterior cerebral arteries

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Sirs: The interpretation of macular sparing in patients with uni- or bilateral homonymous hemianopia is still under discussion [12]. Four different hypothesis have been formulated to explain the origins of the phenomenon, namely unstable or eccentric fixation during perimetry [3]; double macular representation in both hemispheres [9]; enlarged representation of the macula at the cortical level (retino-cortical magnification factor)

**Fig. 1** Concentrically contracted visual fields mapped on a Goldmann perimeter



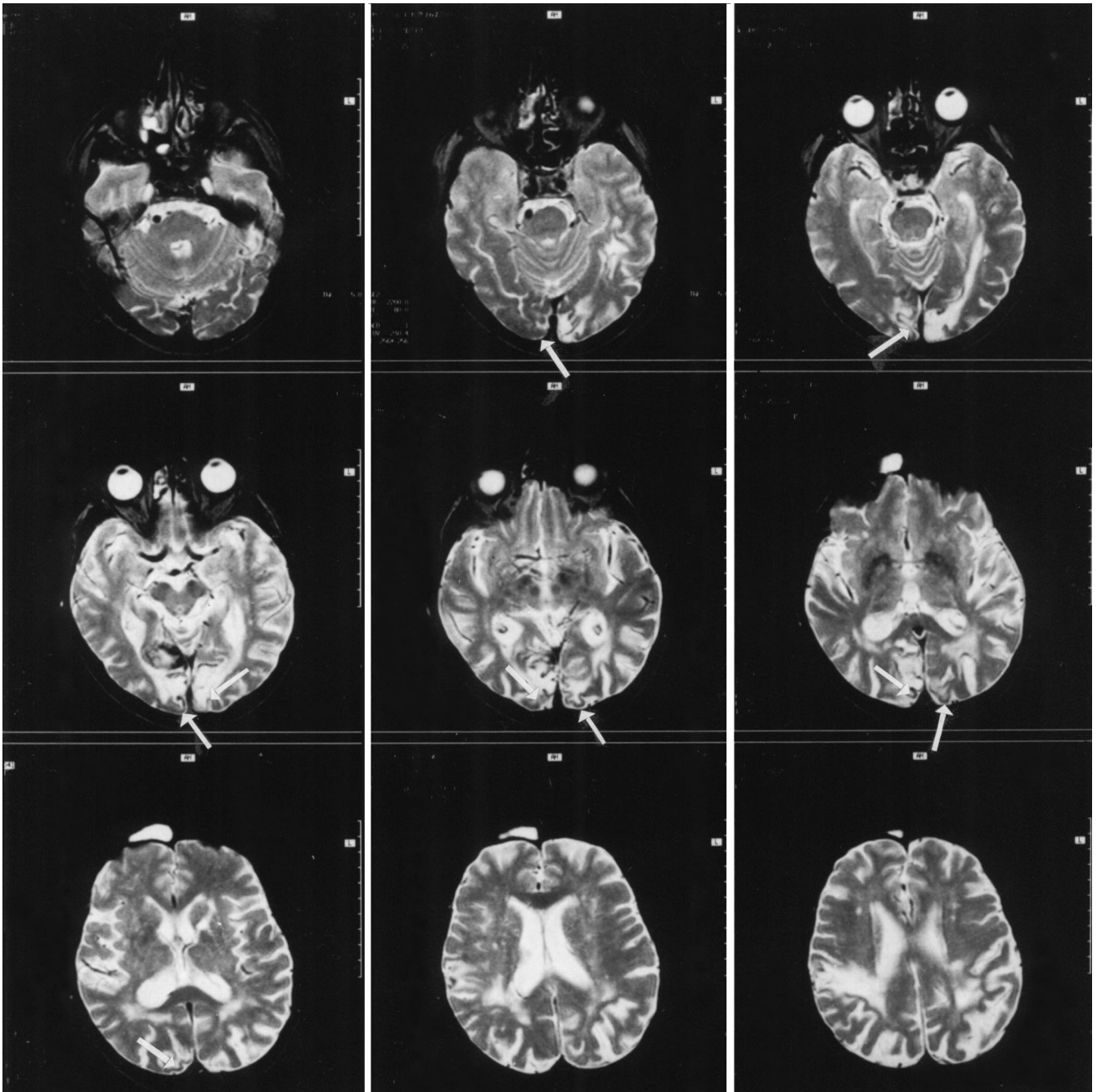
[7]; favourable vascular supply of the cortical macula [10]. However, it would appear that the first two hypotheses (unstable or eccentric fixation and double macular representation in both hemispheres) can at best only account for limited preservation in the foveolar/foveal representations (~ 1–5°). When the sparing occurs in the further limits of the macular representation (even up to 25° [6]), the most plausible interpretation is that the caudal portions of the striate cortex, in which the central visus is magnified, survives because of compensatory vascular supply.

We have observed a double homonymous hemianopia with sparing of the macula in a 68-year-old diabetic man with a history of myocardial infarction, pulmonary oedema, and multiple brain infarcts to cardioembolic origin. In May 1995, he suffered a sudden stroke, which resulted in a selective loss of the periphery of the visual field. Goldmann perimetric maps showed concentric contraction of fields, sparing approximately 10° of macular vision in the lower quadrants and of the macular and perimacular regions in the upper quadrants (Fig. 1). Ocular examination showed bilaterally

normal pupillary reflex, fundus appearance and visual acuity. Gaze and convergence were also intact. Magnetic resonance imaging (MRI) scan revealed ischaemic lesions involving bilaterally the striate cortex and optic radiations. Small, isolated remnants of the posterior parts of the calcarine fissure, present to an extent that hypothetically would be sufficient to guarantee some degree of central vision, were identifiable (Fig. 2).

In anatomical terms, the central sparing present in our patient can be attributed to a certain degree of preservation of the posterior extremity of the striate cortex (and corresponding fibres of optic radiations) responsible for macular representation.

Potentially, two factors would appear to determine the lower level of vulnerability of the central compared with the peripheral fields: (a) the enlarged representation of the macula at cortical and optic radiation levels and (b) the more intense arterial circulation of the cortical region representing the macula. In their re-examination of Holmes' map, Horton and Hoyt [7] further amplified the percentage of striate cortex assigned to represent central vision (see also



**Fig. 2** Horizontal slices on T2-weighted MRI arranged with the most inferior section in the *upper left corner* and the most superior in the *lower right corner*. There are bilateral ischaemic lesions involving the striate cortex and optic radiations. Note the small isolated islands of posterior striate cortex (*arrows*)

[11]). Expanded central representation increases the probability that macular portions of a damaged striate cortex will survive a random lesion. In order for macular sparing to occur, however, high cortical magnification must combine with a certain vascular supply capable of saving the spared striate cortex from tissue ischaemia. It is known that when occlu-

sion occurs in the posterior cerebral artery territories, collateral vessels (compensatory or supplemental) open up, improving circulation at the occipital pole. Beevor [2] studied vascular distribution in 87 brains by injecting, under equal pressure, liquid gelatine containing different colour agents into the anterior, middle and posterior arteries. He found

that when the posterior cerebral artery was occluded (by ligature), its territory very often could be filled in by the adjacent field, vascularized by the middle cerebral artery through the nearby anastomotic zones. Beauvieux and Ristich-Goelmino [1] injected Prussian blue and minium into the posterior cerebral artery of 12 brains, after which they submitted the specimens to a radiographic examination. The occipital pole was found to be vascularized not only by the calcarine artery, but also both by the posterior temporo-occipital and cuneus arteries. Moreover, the macular area was protected from possible occlusion of the posterior artery by means of a collateral circulation, which was sustained by the sylvian artery and its anastomoses with the temporo-occipital artery. Smith and Richardson [14] examined 32 brains and observed that in three-quarters of these, two or more arteries (posterior temporal, parieto-occipital, middle and calcarine) converge on the posterior end of the calcarine fissure.

In addition to the above experimental studies, pathological evidence has demonstrated the integrity of the posterior extremities of the calcarine fissure in patients with uni- or double homonymous hemianopia and macular sparing [e.g. 6, 8, 10, 13].

Because of the marked differences in the extent of striate cortex dedicated to central vision [15] and in collateral circulation [1, 2, 14], varying from individual to individual, the amount of spared cortex and degree of macular sparing varies considerably from patient to patient. Our case seems to reflect a favourable combination of these two factors. A remarkable finding in our patient is the symmetrical size and shape of the contracted fields, in spite of a noncorresponding pattern of infar-

tion in the occipital poles. This can be explained by the effect of the magnification factor capable of sustaining in some cases a quasi-symmetrical narrowing of the halves of each field.

From the observations in our patient and similar cases, we conclude that the high magnification factor as well as the collateral irrigation of the cortical macula may be the fundamental causes of macular sparing. This is certainly the case in patients with a level of preservation in excess of 5°, but may also be true for patients with a lower level of sparing. Other findings argue against the attribution of macular sparing to double macular hemispheric representation [4–5], and a shift in fixation can account only for sparing in unilateral hemianopias.

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V. Silani (✉) · S. Zago · G. Scarlato  
Institute of Neurology  
IRCSS – Ospedale Maggiore  
University of Milan  
via Francesco Sforza 35  
I-20122 Milan, Italy  
e-mail: vincenzo.silani@venere.inet.it  
Tel.: +39-2-55033855  
Fax: +39-2-55190392

M. Farabola  
Institute of Neuroradiology,  
IRCSS – Ospedale Maggiore  
via Francesco Sforza 35  
I-20122 Milan, Italy