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## Macular pigment and fixation after macular translocation surgery

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4                                    **Macular pigment and fixation**  
5                                    **after macular translocation surgery**  
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31

32 **Abstract**

33

34

35 **Background:** After full macular translocation (MT) surgery with 360 degrees retinotomy, the  
36 fovea is rarely identifiable. Our aim was to verify the position of the fovea, to determine how  
37 patients fixate after MT and to examine distribution and optical density of macular pigment  
38 (MP).

39 **Methods:** 9 patients after MT were investigated. The Utrecht Macular Pigment Reflectometer  
40 was used to quantify the MP optical density. A Scanning Laser Ophthalmoscope (SLO) was  
41 used to identify the fovea as the centre of MP distribution and determine the retinal locus of  
42 fixation.

43 **Results:** In all patients, we identified the fovea as the centre of MP distribution. The retinal  
44 areas used for fixation were displayed by SLO fixation analysis. Comparing their spatial  
45 relationship with the fovea, five patients fixated centrally and four eccentrically up to 7.5  
46 degrees. In those patients, microperimetry showed that the atrophy caused by choroidal  
47 neovascularization (CNV) extraction prevented central fixation.

48 **Conclusion:** The combination of MP distribution and fixation analysis permits quantifying  
49 fixation behaviour even if the fovea morphologically cannot be localized. Our results suggest  
50 that the scotoma caused by spreading chorioretinal atrophy is the main cause for reduced  
51 visual acuity after MT and therefore the MT rotation angle is crucially important.

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53

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*(200 words)*

55

## 56 **Introduction**

57

58 Macular translocation (MT) with 360-degree retinotomy has been introduced in 1993 by  
59 Machemer and Steinhorst.[1] During the vitrectomy, the retina is completely detached from  
60 the retinal pigment epithelium (RPE) and the choroidal neovascularization is removed. The  
61 retina is turned around the optic nerve head in an angle of 30 to 40 degrees in order to place  
62 the fovea on a healthy-appearing RPE region. Following the translocation surgery, the eye is  
63 filled with silicone oil for several months and is counter-rotated by extraocular muscle  
64 surgery (Figure 1). Usually, the translocation surgery is preferred in the last eye if the fellow  
65 eye has a central disciform scar secondary to age-related macular degeneration (AMD) or in  
66 monocularly viewing patients. Although the indications for MT have been reduced since anti-  
67 VEGF therapy is available, it is still an important therapy option in cases of extensive  
68 subretinal hemorrhage, rupture of the RPE or if the anti-VEGF therapy fails.[2]

69

70 Since the MT technique was introduced, it was unclear whether patients used their original  
71 fovea or an eccentric preferred retinal locus (PRL) for fixation. In the majority of patients  
72 undergoing MT, it is not possible to identify and localize the original fovea after translocation  
73 because the morphology of the central retina has changed as due to macular disease, the  
74 vitrectomy and a spreading chorioretinal atrophy resulting from the surgical extraction of the  
75 chorioretinal neovascularisation. Therefore, it is not obvious whether patients fixate foveally  
76 or not after MT surgery.

77

78 We have recently introduced a new fixation quality index that allows precise quantification of  
79 the retinal locus of fixation and its stability in patients with macular diseases.[3] In this  
80 method, the position of the original fovea is estimated by using the optic nerve head position,

81 which is the best available measure in patients with severe morphologic changes in the  
82 macular region. However, in patients who have undergone MT surgery, this method is not  
83 suitable because the spatial relation between optic nerve head and original fovea can not be  
84 predicted anymore after surgical rotation of the retina and counter-rotation of the eye.

85

86 Therefore, we used the macular pigment, located in the retinal Henle fibre layer, as a  
87 physiological retinal marker for the original fovea. (The macular pigment should not be  
88 confused with the RPE and is, as an inner-retinal pigment, rotated together with the whole  
89 retina during MT surgery.) We measured the MP distribution and overlaid it with the retinal  
90 area that was used for fixation in patients having undergone MT surgery. Our main questions  
91 were whether the post-MT-surgery patients fixate with their original fovea. We further  
92 analyzed the influence of the fixation stability and the location of the PRL on visual acuity.

93

94

## 95 **Materials and Methods**

96

### 97 **Patients**

98 We investigated 9 patients (9 eyes) after MT with 360-degree-retinotomy. All had a subfoveal  
99 CNV; in 8 eyes the CNV as due to AMD and in the remaining 1 eye due to pathologic myopia  
100 (patient no. P1-02). 5 were males and 4 females, the age of the AMD patients was  $74.8 \pm 5.8$   
101 years (mean $\pm$ SD); the myopic patient was 39 years old. The mean time interval between the  
102 examination and the MT was  $2.1 \pm 0.9$  years. Best corrected visual acuity before translocation  
103 ranged from 20/666 (0.03) to 20/80 (0.25) and after translocation from 20/666 (0.03) to 20/50  
104 (0.4). All patient data are shown in Table 1. None of the subjects had received eccentric  
105 viewing training before. Patients gave their informed consent before recruitment. The study  
106 was approved by the ethics committee of the Tübingen University Hospital, and it conformed

107 to the tenets of the Declaration of Helsinki. The surgical technique was described  
108 elsewhere.[2]

109

### 110 **Fixation monitoring**

111 We used a Rodenstock SLO 101 (Rodenstock, Ottobrunn, Germany) to image the fundus and  
112 to measure the fixation behaviour and the distribution of the macular pigment. For fixation  
113 assessment, we projected a bright red fixation cross of 36 arcmin diameter and  $3.6 \times 10^4$   
114 trolands on a dark background (Michelson contrast 0.986) onto the patient's retina for 10  
115 seconds. We asked the patient to look at the cross the way he/she sees it best. The SLO video  
116 recordings were digitized and processed by image analysis software [3] that tracks the retinal  
117 movements automatically and draws the fixation plot, onto the SLO image of the retina  
118 showing those retinal locations used to fixate the cross (Figure 3C). Temporal resolution was  
119 50 per second by analyzing every video field (half frame). The position of the PRL was  
120 defined as the medians of the horizontal and vertical distributions of all retinal coordinates.

121

### 122 **Microperimetry**

123 For the determination of spatial retinal function, we used custom developed SLO  
124 microperimetry software using gaze-contingent stimulus placement.[4] This method  
125 represents a further development of Rodenstock microperimetry and allows retinal function  
126 testing with automatic real-time correction for eye movements. The software detects retinal  
127 movements by tracking a vessel branching 25 times per second in every video frame and  
128 shifts the stimulus automatically if an eye movement occurred. If the eye moved during a  
129 stimulus presentation (120 ms), the patients' answer was automatically discarded. We used  
130 this method to detect the border of the absolute scotoma resulting from CNV extraction using  
131 a stimulus size corresponding to Goldmann III stimulus (0.43 degrees diameter).

132

133

134 **Spatial distribution of the macular pigment and localization of the fovea**

135 Fundus reflectance maps were recorded at 488 and 514 nm Argon laser wavelengths with the  
136 SLO that is equipped with an Argon laser (Figure 2). If the fundus was imaged with blue  
137 488 nm Argon light, the pigment absorbed the light because of its specific absorbance  
138 spectrum and the macular region appeared black. The green Argon light (514 nm) causes only  
139 low absorption by the pigment. Peripheral retinal areas have approximately the same  
140 reflection characteristics for both wavelengths, apart from a small difference in the lens  
141 optical density. Since the lens and the macular pigment are the only absorbers in this  
142 wavelength region, digital subtraction at two wavelengths of log reflectance maps provided  
143 density maps of the sum of both absorbers, i.e. the spatial distribution of the pigment. Note  
144 that the reflectance images were first aligned, using anatomical landmarks such as retinal  
145 blood vessels. We fitted the observed density distribution with a Gaussian distribution on a  
146 constant background.[5,6] The retinal position with the highest MPOD was considered as the  
147 position of the fovea.

148

149 **Retinal fixation locus and stability**

150 The centre of the fixation plot, i.e. its x- and y-median, defines the preferred retinal locus used  
151 for fixation (PRL), as described previously.[3] We compared its position with the position of  
152 the fovea. A spatial distance between PRL and the fovea less than 1 degree was considered as  
153 a central fixation. To quantify the fixation, we used the fixation stability index [3] (FSI). In  
154 brief, this index does not depend on a normal distribution of the fixation data and quantifies  
155 the number of all 'new' locations on the retina that are used for fixation even if multiple PRL  
156 are present. Theoretically, 'perfect' fixation uses only one location and, thus, has an FSI of  
157 100 %. Additionally, we calculated the conventional BCEA values of the fixation stability in  
158 order to allow easier comparison with previous work of other groups.

159

160 **Optical density of the macular pigment**

161 The recently developed Macular Pigment Reflectometer (MPR) has been used to quantify the  
162 MPOD at the retinal locus of fixation. The MPR has been described in detail by Van de  
163 Kraats et al.[7] In short, the clearly visible illumination beam was set in the subject's pupil a  
164 little above its centre, allowing room for the invisible detection beam. After calibration of the  
165 reflectance and focusing of the light beam on the retina, patients were asked to fixate a 1-  
166 degree white light spot (retinal illuminance  $1.04 \times 10^7$  Troland, and retinal irradiance  $4.6$   
167  $\text{mW}/\text{cm}^2$ ). At the pupil position with highest reflectance, 5 measurements were performed.  
168 For the safety of the patients, a UV-cutoff filter was used (GG395, Schott, Mainz, Germany).  
169 Calculated maximum safe exposure times are 26 min. for healthy eyes, and 20 min. for  
170 aphakics (Health Council of the Netherlands, committee on optical radiation. Health based  
171 exposure limits for electromagnetic radiation in the wavelength region from 100 nanometer to  
172  $1 \text{ mm}^{\circ}$ , Health Council of the Netherlands, The Hague, 1993). With a separation of 0.8 mm,  
173 the reflected light is caught by a detection pupil located underneath the illumination pupil.  
174 Finally, the reflected light is spectrally analyzed by a fibre spectrometer (Ocean Optics  
175 SD2000, Ocean Optics Inc, Dunedin, Florida). A wavelength range of 400–800 nm was used  
176 for MPOD analysis by using custom optical modelling software. Briefly, the model contains  
177 three reflectors, the inner-limiting membrane (ILM), the cone-photoreceptor discs in the outer  
178 segments, and the choroidal space. Anterior to the receptor layer, absorption takes place in the  
179 lens and in the macular pigment. Posterior to the receptor layer, absorption takes place in  
180 melanin and blood. The Levenberg-Marquardt routine [8] was used by the software to fit the  
181 measured data with the optical model by minimizing chi-square values.

182

183 **Blood samples**

184 Relations between lutein blood concentration and optical density have been described in the  
185 literature.[9–11] Plasma concentration of lutein was measured in order to correlate it with the  
186 optical density of the pigment. Fasting blood samples were drawn into Monovettes (Sarstedt,  
187 Germany) containing EDTA. Plasma was collected after centrifugation at 3000 g for 10 min  
188 under light protection. The samples were stored at  $-80^{\circ}\text{C}$  until analysis. The analysis of  
189 lutein was performed at BioTeSys GmbH, (Esslingen, Germany) using an isocratic reversed  
190 phase HPLC method consisting of a pumpsystem (1525 Waters, Eschborn, Germany). In  
191 brief, lutein was extracted from plasma, proteins were precipitated and supernatant was used  
192 for analysis. Separation was performed with a C18-column and the peak detection occurred  
193 with UV-Vis detector at 445 nm.

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196

197 **Results**

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199 **Preferred retinal locus, fixation stability and microperimetry**

200 In all 9 patients, the fovea position was clearly identified by calculating the centre of the  
201 pigment density distribution. Comparing its location with the position of the PRL, we found  
202 that 5 patients fixated centrally, i.e. their PRL was less than 1 degree apart from the fovea (i.e.  
203 the maximum of the MP density). By this definition, 4 patients fixated eccentrically. The  
204 latter group had a PRL that was 3.4 to 7.5 degrees away from the fovea. In two of the  
205 eccentrically fixating patients, the PRL was shifted nasally (towards the optic disc), in one  
206 patient upwards, and in one patient temporally. In none of these patients, the PRL was shifted  
207 downwards (towards the atrophic lesion). All fixation graphs and the pigment distribution are  
208 shown in Figure 4.

209 Fixation stability ranged from 59.6% to 89.8% (mean in the centrally fixating patients 85.8%  
210 and in the eccentrically fixating 71.4%, which was not found as a statistical significant  
211 difference). Furthermore, the stability showed no significant correlation with the eccentricity  
212 of the PRL, with visual acuity or with time since macular translocation. The clinical data of  
213 all patients are shown in Table 1.

214 All patients who fixated eccentrically used a PRL at the border of the absolute scotoma in the  
215 region of the CNV extraction zone.[12]

216

### 217 **Visual acuity**

218 The patients' visual acuity before and after MT is graphically shown in Figure 5. Visual  
219 acuity after translocation was significantly correlated with the eccentricity of the PRL  
220 ( $r^2=0.52$ ;  $p=0.04$ ), but not significantly with the fixation stability. Visual acuity after MT  
221 (logMAR) was significantly better in the centrally fixating patients than in the eccentrically  
222 fixating ones.

223

### 224 **Macular pigment density and lutein serum concentration**

225 The optical density of the pigment varied between 0.11 and 0.59 (mean: 0.23). The lutein  
226 plasma concentrations varied between 0.1 and 0.56  $\mu\text{mol/l}$  (mean: 0.29  $\mu\text{mol/l}$ ). Both  
227 parameters were significantly correlated ( $r^2=0.64$ ,  $p<0.01$ ).

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233 **Discussion**

234

235 Extensive chorioretinal and pigment epithelium atrophy following MT has been described  
236 before. In a long-term study of patients after MT [13], it was seen in 44 of 90 patients. Cahill  
237 et al.[14] compared the occurrence of RPE atrophy between neovascular AMD patients and  
238 geographic atrophy patients after MT and found a significantly higher prevalence in the latter.  
239 The reason may be that, in both studies, the fovea was not accurately localized and the extent  
240 of the atrophic zone was estimated from fundus photographs and/or fluorescein angiographic  
241 recordings. In an SLO study [15] on MT patients, microperimetry was performed and the  
242 scotoma resulting from the RPE atrophy was determined. It was not reported whether the  
243 extent of the scotoma and the morphological atrophy were corresponding.

244

245 In eyes after MT with 360 degree retinotomy, the whole retina is turned around the optic  
246 nerve head and is tilted in an irregularly way. Thus, it becomes impossible to estimate the  
247 foveal position by its spatial relationship to the optic nerve head. By ophthalmoscopy it is  
248 difficult to detect the exact position of the fovea in these patients. From the  
249 pathophysiological point of view, macular translocation creates an interesting situation:  
250 patients with severe submacular changes (in their better eye) are treated in order to restore  
251 foveal vision by connecting the central retina with an intact RPE and choroidal substrate.

252

253 Therefore, it is interesting whether the patients indeed use their former fovea for fixation. By  
254 detecting the fovea through the macular pigment distribution and overlaying it with the  
255 fixation curve, the spatial relationship between the morphological fovea and the functional  
256 area of fixation can be measured.

257

258 We found that more than the half of the patients after translocation fixates centrally. These  
259 patients had a significant better visual acuity than the patients who fixate eccentrically. In a  
260 recent study [16] about quality of life and reading performance after MT, we showed that  
261 reading after MT is still possible. Those patients who fixated eccentrically, showed  
262 chorioretinal and pigment epithelium atrophy, which caused an absolute scotoma that  
263 involved the foveal region. In the current study, we have shown that the extent of the scotoma  
264 was bigger than the morphologic atrophy (Figure 3). The fixation stability of our patients was  
265 comparable to those of the MT patients in a recent study on MT versus patch graft in  
266 neovascular AMD. [17]

267

268 It can be concluded that the expansion of the subretinal RPE atrophy is one crucial point for  
269 the postoperative outcome of visual acuity. The bigger the angle of rotation around the optic  
270 nerve head and the smaller the extent of the subretinal CNV are, the higher is the probability  
271 that the subretinal scar does not involve the fovea.

272

273 The measurements of the optical pigment density in our study show that the macular pigment  
274 is not destroyed by the MT procedure. After MT, its optical density (mean: 0.23) is reduced  
275 compared to normal persons ( $0.53 \pm 0.13$ ) [18], but it is still present and the centre of its  
276 distribution can still be determined.

277

278

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294 **Competing interests**

295 There are no competing interests.

296

297

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356

357

## 358 **Legends**

359

### 360 **Figure 1:**

361 Principle of the macular translocation surgery with 360 degree retinotomy. Usually, this  
362 procedure is done in extensive submacular haemorrhage (A). The retina is rotated around the  
363 optic nerve with an angle of 30 to 40 degrees, i.e. the fovea is shifted upwards (B). The CNV  
364 is extracted; this leaves an atrophic choroidal and RPE lesion (white dot). The eye is filled  
365 with silicone oil. After a certain period (i.e. half a year), an extraocular muscle surgery is  
366 performed and the eye globe is rotated the other way around (C). The atrophic lesion is now  
367 located below the fovea. Our principle question was whether the patients use their original  
368 fovea for fixating (D).

369

### 370 **Figure 2:**

371 SLO visualization of the macular pigment. The relative optical density of the human macular  
372 pigment is shown versus the wavelength (white curve in A). Using the infrared diode laser for  
373 fundus imaging, the pigment does not absorb IR (B). The green wavelength of the SLO's  
374 Argon laser is absorbed moderately by the pigment; using this wavelength for fundus  
375 imaging, the fovea appears dark (C). The blue Argon wavelength is absorbed more, the fovea  
376 appears very dark (D). Digital subtraction of log reflectance maps of C and D provided  
377 density maps of the sum of both absorbers, i.e. the spatial distribution of the pigments.

378

### 379 **Figure 3:**

380 A: Fundus photograph of patient P1-15 (2.5 years after macular translocation). The position  
381 of the fovea is not recognizable. Around the optic disc, the retinal torsion is visible. B: SLO  
382 fundus image of the same patient. C: With our tracking software, the areas on the fundus that

383 'touch' the fixation cross within 10 seconds of fixation recording can be visualized and  
384 displayed on the fundus. The small yellow point represents the medians of the distribution, i.e.  
385 the preferred retinal locus (PRL). Thus, it is not clear if the patient fixates centrally or not. D:  
386 Digital overlay of fixation curve and macular pigment distribution – only now the fovea  
387 becomes visible as the centre of the pigment distribution and it is obvious that the patients  
388 fixates eccentrically (the PRL is located 3.6 degrees above the fovea). E: The results of  
389 microperimetry show that the patient has an absolute scotoma (black dots) with larger extent  
390 than the morphologic atrophic zone caused by the CNV extraction. The patient fixates at the  
391 scotoma border as near as possible.

392

393 **Figure 4:**

394 Synopsis of all patients including the fixation curve (red) and the macular pigment  
395 distribution (yellow). Left column: Patients who fixated centrally; right column: patients who  
396 fixated eccentrically.

397

398 **Figure 5:**

399 A: Visual acuity of the patients before and after macular translocation (logMAR). Black dots  
400 represent the centrally fixating patients, black rectangles the eccentrically fixating ones. The  
401 majority of the patients had a better acuity after translocation. B: Visual acuity (logMAR)  
402 and eccentricity of the PRL, which were statistically correlated ( $r^2=0.52$ ;  $p=0.04$ ), whereas  
403 acuity and fixation stability were not statistically correlated (C).

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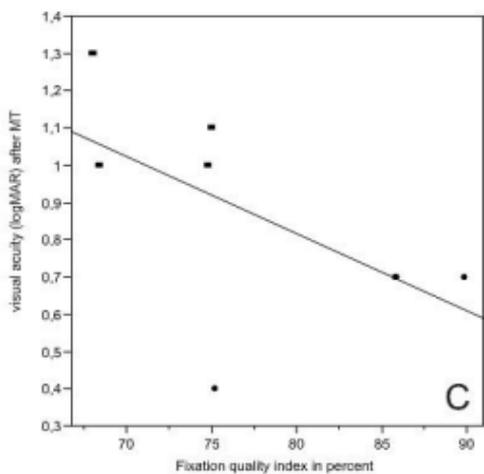
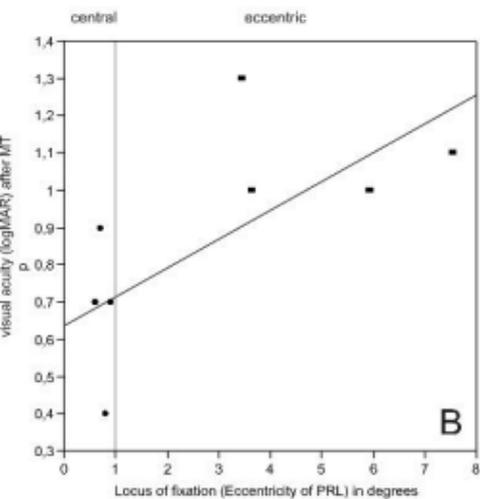
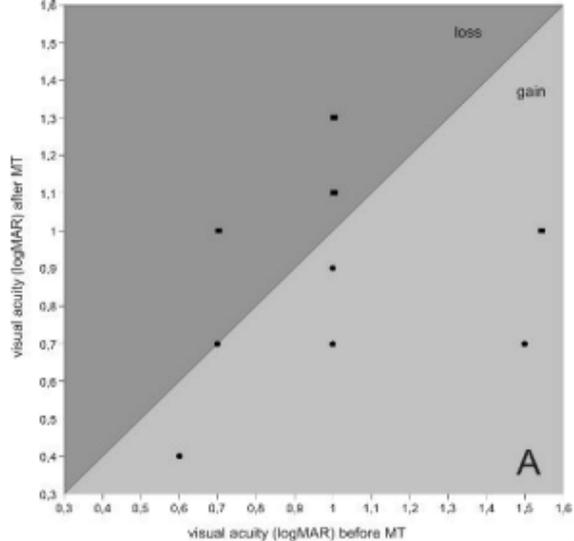
**Table 1**

Patient ID	age	eye	fixation	Eccentricity of PRL (degrees)	FSI (%)	BCEA (arcmin <sup>2</sup> )	visual acuity (logMAR) before MT	visual acuity (logMAR) after MT	duration since MT (years)	Optical density of macular pigment	Lutein plasma concentration (µmol/l)
P1-02	39	right	central	0.6	89.8	222	0.7	0.7	0.6	0.59	0.56
P1-04	73	right	central	0.6	85.8	267	1.0	0.7	2.7	0.27	0.17
P1-05	69	right	central	0.8	75.2	884	0.6	0.4	2.8	0.11	0.10
P1-08	74	left	eccentric	3.4	67.9	1991	1.0	1.3	0.7	0.31	0.52
P1-10	75	right	central	0.9	85.9	374	1.5	0.7	2.2	0.16	0.10
P1-14	86	right	central	0.7	59.6	3235	1.0	0.9	3.1	0.23	0.36
P1-15	76	right	eccentric	3.6	74.6	1230	1.5	1.0	2.5	0.11	0.20
P1-17	78	left	eccentric	5.9	68.2	8777	0.7	1.0	2.2	0.14	0.21
P1-18	67	right	eccentric	7.5	74.9	1387	1.0	1.1	2.1	0.21	0.35

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Table 1: Clinical data of all the patients after macular translocation (MT).

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Figure 5