Delayed Visual Evoked Potentials in Adults After Monocular Visual Deprivation by a Dense Cataract

John J. Sloper* and Alan D. Collins†

Purpose. To study the effect of monocular visual deprivation caused by dense unilateral cataracts in adults.

Methods. Visual evoked potentials have been recorded in 11 patients after removal of a dense unilateral cataract acquired in adulthood. These were compared with those from 8 control patients after removal of a mild lens opacity.

Results. Visual evoked potentials recorded on the first day after removal of a longstanding, dense, unilateral cataract showed a marked delay to stimulation of the operated eye compared to the unoperated eye. No delay was found in the operated or unoperated eye of control patients. Delays in the visual evoked potential returned to normal within approximately 3 months after surgery, with the exception of the two patients with the most longstanding cataracts in whom the delays persisted much longer.

Conclusions. The adult central visual system is sensitive to visual deprivation caused by a longstanding, dense, unilateral cataract. The changes found may be important in understanding the causes of intractable diplopia, which sometimes occurs after the removal of such cataracts. Invest Ophthalmol Vis Sci. 1995; 36:2663-2671.

The effects of monocular visual deprivation on the central visual pathways have been studied extensively in children and in young animals. In particular, the amblyopia and associated changes resulting from monocular cataracts in children have been well described using both clinical and electrophysiological methods. However, monocular cataracts that develop in adulthood do not result in amblyopia, and it generally has been assumed that the adult central sensory visual pathways are no longer sensitive to visual deprivation. Previous studies that have examined visual recovery after cataract extraction in adults have been concerned mainly with the quality of visual function after surgery, particularly in relation to intraocular lens implantation and the effects of anisometropia. These studies have demonstrated that recovery of visual function, including stereopsis, is generally excellent when posterior chamber intraocular lenses are implanted.

The current study differs from this previous work in that it reports the results of visual evoked potentials (VEPs) recorded after cataract extraction in a particular subgroup of adult patients who have had a longstanding, dense, monocular cataract and have thus had a considerable period of monocular visual deprivation. These results are compared with those from a control group of patients who had surgery for mild lens opacity. This allows the effects of visual deprivation to be separated from those of cataract surgery and lens implantation. Prolonged monocular deprivation is known to lead, in some adults, to loss of fusion, with divergence of the nonseeing eye and the risk of a persistent squint with intractable diplopia after cataract extraction, but little attention has been paid to the possible role that changes in the sensory visual pathways might play in such patients.

SUBJECTS AND METHODS

Eleven patients were studied after removal of a longstanding, dense, unilateral cataract. These patients all
TABLE 1. Clinical Data for Patients With Dense Unilateral Cataract

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Duration of Cataract (years)</th>
<th>Anesthetic</th>
<th>Divergent</th>
<th>Snellen Acuity Before Surgery</th>
<th>Snellen Acuity After Surgery</th>
<th>Fellow Eye</th>
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<tr>
<td>A</td>
<td>41</td>
<td>18</td>
<td>GA</td>
<td>Yes</td>
<td>HM</td>
<td>6/5</td>
<td>6/5</td>
</tr>
<tr>
<td>B</td>
<td>67</td>
<td>&gt;1</td>
<td>LA</td>
<td>No</td>
<td>CF</td>
<td>6/9</td>
<td>6/6</td>
</tr>
<tr>
<td>C</td>
<td>59</td>
<td>~3</td>
<td>LA</td>
<td>Yes</td>
<td>HM</td>
<td>6/4</td>
<td>6/6</td>
</tr>
<tr>
<td>D</td>
<td>63</td>
<td>5</td>
<td>GA</td>
<td>Yes</td>
<td>6/60</td>
<td>6/7.5</td>
<td>6/6</td>
</tr>
<tr>
<td>E</td>
<td>38</td>
<td>&gt;2</td>
<td>LA</td>
<td>No</td>
<td>CF</td>
<td>6/6</td>
<td>6/4</td>
</tr>
<tr>
<td>F</td>
<td>59</td>
<td>5</td>
<td>LA</td>
<td>No</td>
<td>HM</td>
<td>6/6</td>
<td>6/5</td>
</tr>
<tr>
<td>G</td>
<td>73</td>
<td>?</td>
<td>GA</td>
<td>Yes</td>
<td>HM</td>
<td>6/9</td>
<td>6/6</td>
</tr>
<tr>
<td>H</td>
<td>42</td>
<td>&gt;1</td>
<td>GA</td>
<td>Yes</td>
<td>HM</td>
<td>6/5</td>
<td>6/4</td>
</tr>
<tr>
<td>I</td>
<td>70</td>
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<td>GA</td>
<td>Yes</td>
<td>CF</td>
<td>6/5</td>
<td>6/7.5</td>
</tr>
<tr>
<td>J</td>
<td>83</td>
<td>&gt;1</td>
<td>GA</td>
<td>Yes</td>
<td>PL</td>
<td>6/6</td>
<td>6/6</td>
</tr>
<tr>
<td>K</td>
<td>76</td>
<td>2</td>
<td>GA</td>
<td>No</td>
<td>CF</td>
<td>6/6</td>
<td>6/6</td>
</tr>
</tbody>
</table>

GA = general; LA = local; HM = hand movements; CF = count fingers; PL = perception of light.

had a best-corrected preoperative acuity of 6/60 or less in the affected eye and an acuity of 6/9 or better in the fellow eye. In all instances, the cataract was documented to have been present for at least 1 year before surgery, or else the eye was divergent and there was no history of strabismus (Table 1). Thus, these patients had a substantial period of markedly reduced acuity in the eye with the cataract, with normal acuity in the fellow eye. The eight control patients who underwent surgery for mild lens opacities had preoperative acuity of 6/18 or better in the affected eye and 6/9 or better in the fellow eye. None of the eyes of the control patients was divergent (Table 2). Most underwent surgery because of their need to drive or because of problems with glare. Because of the relatively minor nature of their visual disabilities, it often was difficult to determine an accurate time of onset of cataract in these patients, although the duration of symptoms was usually short.

TABLE 2. Clinical Data for Control Patients With Early Cataract

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Duration of Cataract (years)*</th>
<th>Anesthetic</th>
<th>Divergent</th>
<th>Snellen Acuity Before Surgery</th>
<th>Snellen Acuity After Surgery</th>
<th>Fellow Eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>L</td>
<td>24</td>
<td>?</td>
<td>GA</td>
<td>No</td>
<td>6/6</td>
<td>6/5</td>
<td>6/5</td>
</tr>
<tr>
<td>M</td>
<td>65</td>
<td>2nd eye</td>
<td>GA</td>
<td>No</td>
<td>6/18</td>
<td>6/5</td>
<td>6/5</td>
</tr>
<tr>
<td>N</td>
<td>66</td>
<td>?</td>
<td>LA</td>
<td>No</td>
<td>6/9</td>
<td>6/5</td>
<td>6/4</td>
</tr>
<tr>
<td>O</td>
<td>64</td>
<td>?</td>
<td>LA</td>
<td>No</td>
<td>6/12</td>
<td>6/4</td>
<td>6/5</td>
</tr>
<tr>
<td>Q</td>
<td>81</td>
<td>2nd eye</td>
<td>GA</td>
<td>No</td>
<td>6/12</td>
<td>6/9</td>
<td>6/9</td>
</tr>
<tr>
<td>R</td>
<td>68</td>
<td>1</td>
<td>LA</td>
<td>No</td>
<td>6/9</td>
<td>6/9</td>
<td>6/9</td>
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<tr>
<td>S</td>
<td>72</td>
<td>Approx. 10</td>
<td>LA</td>
<td>No</td>
<td>6/9</td>
<td>6/9</td>
<td>6/9</td>
</tr>
</tbody>
</table>

* Because of the relatively minor nature of visual symptoms, it often was difficult to determine with any accuracy time of onset for cataract in these patients, although the duration of symptoms was usually short.

GA = general; LA = local.
bulbar block. Patients undergoing surgery under ret-
robulbar local anesthetic block were excluded because
of possible effects of the local anesthetic on the optic
nerve. Patients L, M, N, and R underwent surgery by
phacoemulsification using a small incision technique.
There were no significant differences in results be-
tween these patients and control patients undergoing
extracapsular surgery. All patients received a standard
posterior chamber implant except patient A, in whom
an implant was not used initially because of the risk
of intractable diplopia (see Case Reports). Patients A,
B, C, D, E, I, O, P, and S were operated on by the
same surgeon (JS). Patients G, K, and R and patients
H and J were operated on by the same surgeons. Each
of the remaining patients was operated on by a differ-
tent surgeon.

Postoperative Clinical Assessment
On postoperative day 1, all patients eyes were exam-
ined at the slit lamp and were then refracted. Visual
acuity was measured using a standard Snellen chart
with the appropriate spectacle correction. Reading
acuity was measured both monocularly and binocu-
larly using an appropriate reading addition. Color vi-
sion was tested using Ishihara plates with the reading
correction; it was normal in all patients except patients
B and Q, who were deficient in both eyes. No patient
had an afferent pupillary defect. Ocular motility was
examined clinically, and stereacuity was measured
with the Titmus stereo test and the TNO test for ste-
reoscopic vision using the reading correction. There
were no postoperative complications in either group
of patients.

Electrophysiological Methods
The initial VEP recordings were made on postope-
tative day 1 in all patients except patient A, who was
initially recorded on postoperative day 5. Spectacle
corrections as determined above were used. All pa-
tients had an acuity of at least 6/9 in each eye (Tables
1 and 2), and all patients were asked to confirm that
they could see the checkerboard stimulus and the
small central fixation target clearly with each eye at
the time of recording. No patient had difficulty main-
taining fixation monocularly with either eye during
recording; this was confirmed by observation of the
subjects. Pattern-reversal VEPs were recorded to black-
and-white checks subtending 20° and 40° of visual
angle, displayed on a television monitor subtending
14° horizontal and 11° vertical of visual angle at 1.6
m from the subject. The checkerboard pattern-rever-
sal rate was two per second, contrast ratio 98%, and
mean luminance 44 cd/m². Silver–silver chloride elec-
trode placements were ground forehead, reference
Cz, and active Oz. Amplifier filter settings were low-
frequency 0.8 Hz (−3 dB point, attenuation 20 dB/
decade) and high-frequency 100 Hz (−3 dB point,
attenuation 40 dB/decade). One hundred ninety-two
250-msec sweeps subdivided into three subaverages
of 64 sweeps were recorded for each stimulus condition.
Recordings were rejected if the three subaverages
were not consistent. Binocular recordings were made
using both stimulus sizes, and then monocular re-
cordings were made from each eye. Amplitude and
latency of the peak of the P100 response were mea-
sured. Delay was calculated as the P100 latency from
the operated eye minus the P100 latency from the
fellow eye. Statistical comparisons between dense cata-
ract and control groups were made using Student’s t-
test for independent groups. Comparisons between
operated and fellow eyes within groups were made
using paired t-tests. The normal P100 latency ranges
for pattern-reversal VEPs recorded under these condi-
tions in our laboratory are 90 to 114 msec for both
20' and 40' checks, with a standard deviation for the interocular P100 latency difference of 2.66 msec.

For patient A, pattern electroretinograms were recorded to the same 40' check stimulus used for the pattern VEP except that the reversal rate was 6 per second. Recording conventions and an interrupted stimulus technique were as described by Holder. Mean normal laboratory values for P50 were: latency, 50.4 ± 1.74 msec; amplitude, 1.8 ± 0.44 μV. For N95 they were: latency, 95.5 ± 5.11 msec; amplitude, 3.8 ± 0.65 μV.

RESULTS

All patients in both groups achieved a best-corrected acuity on postoperative day 1 of 6/9 or better, with no complications from surgery (Tables 1 and 2). Reading acuity of all patients was N6 or better with each eye separately and binocularly, except for the two patients with diplopia and one control patient with planned anisometropia.

Initial Visual Evoked Potentials

The pattern-reversal VEPs to 20' checks recorded from patient E on postoperative day 1 are shown in the top trace of Figure 1. The latency of the P100 response from the operated eye is longer by 17 msec than that from its fellow eye and falls well outside the normal range; in addition, the P100 waveform is widened. The response to 40' checks similarly was delayed by 14 msec. This patient had an uncorrected acuity of 6/5 in both eyes at the time of recording, with normal color vision and pupil responses.

The initial VEP results for all patients after removal of a dense unilateral cataract are shown in Table 3. These show that the latencies of the P100 responses recorded from the operated eye were significantly longer than those from the fellow eye for both 20' and 40' checks (paired t-tests; 20' checks: t = 3.67, df = 10, P < 0.01; 40' checks: t = 4.47, df = 9, P < 0.002). The mean delay was 9.8 msec for the 20' checks and 8.4 msec for the 40' checks. No consistent latency difference between operated and fellow eyes was found in the control group of patients for responses to either check size (paired t-tests; 20' checks: t = -0.40, df = 7, P = 0.70; 40' checks: t = -0.74, df = 7, P = 0.48) (Table 4). The interocular latency differences for the dense unilateral cataract group are significantly larger than those for the control group for both check sizes (unpaired t-tests; 20' checks: t = 3.04, df = 17, P < 0.01; 40' checks: t = 3.83, df = 16, P < 0.001). The mean latencies for the operated eye in the unilateral cataract group lie well outside the normal range for both 20' and 40' checks, whereas those for the operated eyes of the control group and for the fellow eyes of both groups are within the normal range. No consistent difference was found in the amplitudes of VEPs recorded from operated and fellow eyes in either group, although there were differences between eyes for individual patients in both groups.

The VEPs recorded on postoperative day 1 for control patients P and S are shown in Figure 2. The record for patient S includes responses for the operated eye recorded with and without the required spectacle correction of -0.50 DS/-2.00 DC at 175°. Similar comparisons in one other control and two unilateral cataract patients confirmed that small refractive errors had no significant effect on the evoked potentials.

Relation of Visual Evoked Potential Delay to Age

The delay in the evoked potential from the operated eye is plotted in relation to age for both 20' and 40'

### Table 3. P100 Latencies to 20' and 40' Checks Shortly After Removal of Dense Unilateral Cataract

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>P100 Latency to 20' Checks (msec)</th>
<th>Delay (msec)</th>
<th>P100 Latency to 40' Checks (msec)</th>
<th>Delay (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Operated Eye</td>
<td>Fellow Eye</td>
<td>Binocular</td>
<td>Operated Eye</td>
<td>Fellow Eye</td>
</tr>
<tr>
<td>A</td>
<td>41</td>
<td>123</td>
<td>107</td>
<td>114</td>
<td>16.0</td>
</tr>
<tr>
<td>B</td>
<td>67</td>
<td>115</td>
<td>106</td>
<td>105</td>
<td>9.0</td>
</tr>
<tr>
<td>C</td>
<td>59</td>
<td>120</td>
<td>112</td>
<td>113</td>
<td>8.0</td>
</tr>
<tr>
<td>D</td>
<td>63</td>
<td>134</td>
<td>105</td>
<td>107</td>
<td>29.0</td>
</tr>
<tr>
<td>E</td>
<td>58</td>
<td>119</td>
<td>102</td>
<td>108</td>
<td>17.0</td>
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<tr>
<td>F</td>
<td>59</td>
<td>112</td>
<td>109</td>
<td>109</td>
<td>3.0</td>
</tr>
<tr>
<td>G</td>
<td>73</td>
<td>113</td>
<td>102</td>
<td>103</td>
<td>11.0</td>
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<td>H</td>
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<td>K</td>
<td>76</td>
<td>126</td>
<td>122</td>
<td>121</td>
<td>4.0</td>
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</tbody>
</table>

Mean ± SD

|                | 120.5 ± 7.43 | 110.7 ± 9.74 | 112.3 ± 9.02 | 9.8 ± 8.87* | 113.8 ± 6.53 | 105.4 ± 7.68 | 107.7 ± 8.99 | 8.4 ± 5.94* |

* P < 0.01 versus controls.

SD = standard deviation.
TABLE 4. P100 Latencies to 20' and 40' Checks Shortly After Removal of Control Cataract

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Operated Eye</th>
<th>Fellow Eye</th>
<th>Binocular</th>
<th>Delay (msec) Operated Eye</th>
<th>Fellow Eye</th>
<th>Binocular</th>
<th>Delay (msec)</th>
<th>Delay (msec)</th>
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<tbody>
<tr>
<td>L</td>
<td>24</td>
<td>114</td>
<td>122</td>
<td>117</td>
<td>-8.0</td>
<td>125</td>
<td>121</td>
<td>114</td>
<td>4.0</td>
</tr>
<tr>
<td>M</td>
<td>65</td>
<td>107</td>
<td>111</td>
<td>101</td>
<td>-4.0</td>
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<td>105</td>
<td>91</td>
<td>-7.0</td>
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<tr>
<td>N</td>
<td>66</td>
<td>113</td>
<td>109</td>
<td>110</td>
<td>4.0</td>
<td>108</td>
<td>108</td>
<td>106</td>
<td>0.0</td>
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<td>112</td>
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<td>110</td>
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<td>115</td>
<td>113</td>
<td>-1.0</td>
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<tr>
<td>S</td>
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<td>119</td>
<td>117</td>
<td>-3.0</td>
<td>110</td>
<td>114</td>
<td>112</td>
<td>-4.0</td>
</tr>
</tbody>
</table>

Mean ± SD 113.4 ± 4.98 114.0 ± 6.68 112.4 ± 6.86 0.6 ± 4.47 109.5 ± 7.76 110.6 ± 5.73 106.0 ± 7.77 -1.1 ± 4.13

SD = standard deviation.

checks in Figure 3. For both check sizes, regression analysis showed a negative relationship between VEP delay and age at surgery, although this only reached statistical significance for the 40' checks. (For 20' checks, gradient -0.311, y-intercept 28.8; \( t = -1.87; df = 9; P = 0.09 \). For 40' checks, gradient -0.261, y-intercept 24.3; \( t = -2.75; df = 8; P < 0.05 \).)

Long-term Follow-up of Visual Evoked Potential Changes

The VEPs recorded to 20' checks from patient E at postoperative days 15 and 126 are shown in the middle and bottom traces of Figure 1. By 15 days after surgery, the difference between P100 latencies from operated and nonoperated eyes was reduced to 8 msec, and by 126 days after surgery, the interocular latency difference was well within the normal range. All seven patients with the largest delays were followed up. Except for patients A and D (see Case Reports below), the delay in all patients returned to normal or near normal by 3 months after surgery (Fig. 4).

CASE REPORTS

The two patients, A and D, who had the longest periods of visual deprivation are of particular interest because recovery from the VEP delay was either prolonged or did not occur. Patient D was first seen 10 years before surgery because of a watery eye, when acuities of 6/6 were recorded for each eye. He was subsequently seen 5 years before surgery when he had a moderate cataract in the left eye, reducing his vision to 6/18. This was possibly related to an episode of blunt trauma 3 years earlier, although vision was unaffected at the time. Two years before surgery, his left acuity was 6/60, and the eye was noted to be divergent. Left intraocular pressure was found to be 26 mm Hg, and he was started on timolol maleate 0.5% twice a day to the left eye, although the optic disc was normal. The acuity of the right eye was 6/6 with normal pressure. Immediately after left cataract extraction and lens implantation, his eye remained divergent with diplopia, although the acuity corrected to 6/7.5. A VEP recorded at this time showed a delay of 29 msec to 20' checks. By 17 days after surgery, his left eye was straight at times and had occasional diplopia; his VEP delay was now reduced to 19 msec. Over the next few weeks, the eye straightened spontaneously, the diplopia resolved, and he regained some binocular function. Automated perimetry showed minimal superior arcuate field loss in both eyes, and his treatment was increased to timolol maleate 0.5% twice a day to...
both eyes with dipivefrin 0.1% twice a day to the left eye. Slightly more than 1 year after surgery, he was asymptomatic, although his VEP still showed a delay of 7 msec. At 20 months after surgery, his corrected vision was 6/6 in the operated eye and 6/5 in the fellow eye, and the diplopia had completely resolved. He had no manifest strabismus but dissociated readily to a left divergent squint. Stereoacuity at this time was measured at 140 seconds of arc by the Titmus stereo test and 120 seconds of arc by the TNO test for stereoscopic vision. By this time, his P100 latency difference was reduced to 3 msec, well within the normal range (Fig. 4B).

Patient A, who had a documented dense cataract for 18 years before surgery, had a much longer period of monocular visual deprivation than any of the other patients, and his results differ from the rest of the group in several respects. He reported poor vision in his left eye since a blow to the eye in February 1973. He was first seen in July 1974, when his visual acuities were recorded as 6/5 in the right eye and 1/60 in the left eye. His left pupil was semidilated, and he was noted to have a mild ptosis and to have anterior and posterior subcapsular lens opacities. Fundal examination showed no gross abnormality in the left eye. Similar findings were recorded on two further occasions in 1974, after which he failed to attend.

He resumed treatment in 1992 because of poor vision in his left eye, which now threatened his employment. At this time, his right visual acuity was 6/5, and his left visual acuity was hand movements with accurate projection of light. There was no afferent pupillary defect. The left eye showed 20 prism diopters of divergence with intermittent down drifted of the eye. Slit lamp examination showed sphincter tears, with a hypermature cataract in a slightly subluxed lens. Ultrasound examination showed no abnormality of his left fundus. Intraocular pressure was normal in both eyes.

Because of the risk of postoperative diplopia, he underwent a left extracapsular cataract extraction without intraocular lens implantation.

On the postoperative day 1, his left visual acuity corrected to 6/6, and fundal examination showed no abnormality. Color vision was normal in both eyes. However, his left eye remained divergent, and he reported diplopia. Five days after surgery, his left refraction was +9.00 DS/+1.00 DC at 60°, giving him an acuity of 6/5 in the left eye and N5 for reading with a +3.00 addition. His right visual acuity was 6/4 unaided. His VEP to 20' checks recorded that day with spectacle correction showed a delay of 16 msec with a delay of 20 msec to 40' checks (Fig. 5, top; Table 3).

The operated eye remained divergent with diplopia. A VEP recorded with aphakic correction at 20 days after surgery showed delays of 11 msec and 18...
Visual Deprivation in Adults

FIGURE 5. Pattern-reversal visual evoked potentials to 20' checks recorded on days 1 and 111 after removal of a dense unilateral cataract from patient A and then 6 months after insertion of a secondary posterior chamber intraocular lens and the restoration of binocular single vision. The delay noted on stimulation of the operated eye on day 1 was still present more than 2 1/2 years after cataract removal in this patient, who had the longest period of monocular visual deprivation (see Case Reports).

msec to 20' and 40' checks, respectively. At 111 days after cataract extraction, he had a left acuity of 6/6 with a contact lens. His VEP recorded at this time using the contact lens showed delays of 17 msec and 15 msec to 20' and 40' checks, respectively (Fig. 5, middle). With aphakic correction, he had 6/6 acuity, and the VEP recorded using this was almost identical to that using the contact lens (Fig. 5, middle). After 3 weeks of contact lens wear, the delays were still 18 msec and 15 msec to 20' and 40' checks, and he still experienced constant diplopia. After this, he underwent an injection of botulinum toxin to his left lateral rectus muscle. This corrected his divergent squint and allowed him to regain binocular single vision, which he has since retained without further injection. Six months later, his VEP was still delayed to both 20' and 40' checks (20 msec and 24 msec, respectively). He subsequently underwent a secondary posterior chamber lens implant to his left eye, giving him an acuity of 6/5 with a small spectacle correction. Six months after this and almost 2 years after his original surgery, his VEP was still delayed to both check sizes (15 msec to 20' checks and 18 msec to 40' checks; Fig. 5, bottom).

These delays have persisted for three further visits over the next year (Fig. 4B). He continues to have binocular single vision with latent divergence and slow recovery. He gets diplopia, mainly torsional, when he is tired; if he closes his right eye, his visual world becomes unstable, and after a few seconds he feels as if he is tilting. The Titmus stereo test revealed stereoaucity of 400 seconds of arc. Normal pattern electroretinograms were recorded from both eyes at this time (Fig. 6).

DISCUSSION

This study shows that in adults, visual deprivation caused by a longstanding, dense, unilateral cataract results in substantial delay in the P100 component of the VEP recorded to stimulation of the deprived eye, together with changes in the waveform of the evoked response. This delay was found in the presence of clinically normal monocular visual function, although there was disruption of binocular function in two patients. The effect of deprivation on the VEP is age dependent and is more pronounced in younger adults. The delay was most marked immediately after removal of the cataract and was reversible over a period of weeks, at least with the degree of deprivation seen in the majority of the patients in this study. However, the findings on patients A and D suggest that the effect eventually may become irreversible. Although visual deprivation in adults does not produce the dramatic functional deficits found in children, the central

PATTERN ELECTRO-RETINOGRAMS

FIGURE 6. Normal pattern electroretinograms recorded from patient A.
visual pathways remain sensitive to monocular visual deprivation into adulthood.

Previously, visual deprivation has not been shown to affect visual evoked responses in adults. One study has described delays in the visual evoked response several weeks after cataract extraction; however, this study did not attempt to demonstrate that the delay was due to visual deprivation rather than any other aspect of the cataract or surgery. It did not relate the VEP delay to the duration or severity of the cataract and did not use a control group to differentiate the effects of deprivation from those of the surgery itself or of the implantation of an intraocular lens. In the current study, a control group of patients undergoing similar surgery for a mild cataract was studied to allow the effects of deprivation to be distinguished from those of cataract surgery and intraocular lens implantation per se. No delay was found in the control patients, showing that it was visual deprivation and not surgery itself that caused the delay in the patients after removal of the longstanding cataracts.

Several other possible causes of VEP delay were considered in the design of the study. Persistent effects of local anesthetic on the optic nerve can be excluded because the delay was also present in patients who underwent surgery under general anesthesia; delays were present several weeks after surgery in a number of patients and, in one patient, for more than 2 years. Patients undergoing surgery with retrobulbar block were excluded from the study. A defocused image also can cause delay in the VEP, so care was taken to ensure that all patients had a corrected acuity of 6/9 or better and a clear image of the stimulus during recording. In addition, the effects of small refractive errors were studied and shown not to have a significant effect on the VEP in these patients. Similar delays were seen for 20' and 40' checks.

A common cause of unilateral cataract in young patients is ocular trauma; therefore, the possibility of associated ocular nerve damage causing VEP delay must be considered. Only three of the patients had a history of ocular trauma that could be considered a possible cause of cataract, and substantial delays were seen in patients with no history of trauma. All patients had good postoperative acuity, no patient had an afferent pupillary defect, and only one elderly patient in each group had abnormal color vision. With the exception of patient A, the delayed VEP recovered to normal in all patients. Patient A had a corrected acuity of 6/5, full visual fields, normal color vision, normal afferent pupillary responses, and a normal pattern electroretinogram in the eye with the delayed response. Significant optic nerve trauma, therefore, can be excluded as a cause of the delay.

The current results provide no direct evidence concerning where in the visual pathways of each patient the delay arose or the mechanisms underlying it. However, patient A was shown to have a normal pattern electroretinogram, so the anterior visual pathway can be excluded as a cause of his delay. It has been shown in adult monkeys that experimental monocular deprivation causes changes in the levels of several neurotransmitters and of cytochrome oxidase, a metabolic enzyme, in deprived ocular dominance columns in the primary visual cortex. These changes were more marked in younger adults than in older adults, as are the current VEP changes. It is at least a strong possibility that the VEP delays recorded in these patients arise at the cortical level and that the visual input is slower to activate the visual cortex because of long-term changes in neurotransmitter levels.

The current findings are of interest in relation to recent experimental work in adult animals demonstrating changes in receptive fields of neurons in the visual cortex after retinal lesions or masking. However, there are important differences from these studies; in particular, it should be noted that in the current work, the changes in the visual evoked responses occurred in the absence of any structural lesions and that recovery occurred in the presence of a normal visual input from the fellow eye. The changes in the current study also occurred over a considerably longer time than most of the changes found in the animal work.

Comparison of the electrophysiological changes found here with those found in children after removal of a congenital cataract is difficult. In infants, the VEP is clearly much more sensitive to deprivation than in adults, and it may be undetectable after early removal of a congenital cataract. However, further comparisons are difficult because most children are treated by patching of the fellow eye, which produces recovery of the VEP in the operated eye. This recovered VEP shows an increased P100 latency comparable to the main change found in adults. However, it is not clear whether the changes seen in adults are a mild form of the changes seen in children or are fundamentally different.

The functional significance of VEP changes caused by deprivation in adults remains to be determined. Monocular visual function appears unaffected, but it will be interesting to study binocular function more closely in such patients. Although the delay was reversible in most of the patients studied, it appears to have the potential to become irreversible in some patients. The main risk factors for this are probably length of deprivation and younger age at onset of the cataract. Such changes may well play a part in the breakdown of binocular function and intractable diplopia seen in some adults after prolonged monocular visual deprivation.
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commonly with traumatic cataracts, it may well be the relatively young age of many of these patients rather than the traumatic nature of the cataract that makes them susceptible. Electrophysiological changes also may provide the basis for a test to recognize patients at risk for intractable diplopia before they undergo cataract extraction.

Key Words

cataract, diplopia, visual evoked potential (VEP), visual deprivation

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References