

Contrast Adaptation in Human Retina and Cortex

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PURPOSE. Although cortical contrast adaptation has been extensively studied with both psychophysical and electrophysiological techniques, little is known about retinal contrast adaptation in humans.

METHODS. Retinal and cortical long-term contrast adaptation was assessed with simultaneous measurement of pattern electroretinogram (PERG) and cortical visual evoked potentials (VEPs). This study involved three approaches: sampling of the contrast transfer function from 2.7% to 98% with adaptation to high (98%) and low (7.3%) contrasts, linearity of adaptation effects, and transfer of contrast adaptation between parallel and orthogonal grating orientations.

RESULTS. Contrast adaptation affected retinal and cortical recordings quite differently. The VEP showed a sigmoid contrast transfer function, which was shifted toward higher contrasts (by a factor of 1.9), whereas amplitudes at higher test contrasts were enhanced to 127%. The PERG decreased in amplitude to approximately 90%, and the latency was significantly reduced by 4 to 6 msec ($P < 0.05$). All measured effects were linear with adaptation contrast. Orientation played no role in the PERG results, whereas the VEP was enhanced to 125% when tested parallel and to 150% when tested orthogonal to adaptation.

CONCLUSIONS. VEP results confirm and extend previous findings and fit well with single-cell recordings. The PERG findings suggest that retinal contrast adaptation occurs and mainly operates in the temporal domain, comparable to rapid gain-control findings in cats and primates. (*Invest Ophthalmol Vis Sci.* 2001;42:2721-2727)

Whereas the usable light intensity in our visual world varies by approximately eight orders of magnitude, the range over which we discriminate differences in light intensity varies only approximately 100-fold. Photochemical and neural mechanisms keep the limited range of discriminability in the range of the prevailing luminance, known as luminance adaptation. Similarly, although on a smaller scale, contrast adaptation shifts the steep part of the contrast transfer function to match the prevailing contrast condition.¹ On a single-cell basis, there is a distinction between contrast adaptation and contrast gain control. The latter represents rapid changes, taking approximately 100 msec, as found in retinal ganglion cells of cats² and M_x ganglion cells of macaques.³ Conte et al.⁴ also found rapid changes in humans by means of pattern electroretinogram (PERG) and visual evoked potentials (VEPs). Con-

trast adaptation refers to relatively long-term changes (seconds to minutes).^{5,6}

There are only two studies addressing retinal contrast adaptation in humans, using the PERG, and their findings are contradictory: Brigell et al.,⁷ based on recordings in three subjects, found no retinal contribution to contrast adaptation, using a contrast sweep technique with the PERG to observe changes in threshold contrast. Odom and Norcia⁸ recorded in one subject and reported a reduction in PERG amplitude after contrast adaptation.

Cortical contrast adaptation is amply documented in single-cell studies.⁹⁻¹¹ In VEP studies, Mecacci and Spinelli¹² and Suter et al.¹³ found a reduction in amplitude after prolonged exposure to high contrasts, whereas Bach et al.¹⁴ found that adaptation to high contrasts increased VEP amplitude at high test contrasts, and decreased it at low test contrasts (for a resolution of this seeming contradiction see the Discussion section). Nelson et al.¹⁵ and Brigell et al.⁷ used a contrast sweep paradigm (0%-20%-0% over 40 seconds) and found a hysteresis caused by adaptation to the stimulus contrast. This corresponds to a threshold elevation, possibly due to a rightward shift of the contrast transfer function.

We set out to assess the relative contribution of the retinal and cortical mechanisms in contrast adaptation and to reconcile these contradicting findings by covering an extended adaptation and test contrast range. Thus, we recorded simultaneously the PERG (which mirrors the function of the retinal processing chain from the photoreceptor up to and including the retinal ganglion cells) and the VEP (an index of cortical processing).

All measurements used rapidly varying stimuli to evoke steady state signals that are easily analyzed by Fourier transformation and allow quantitative estimates of signal-to-noise ratios and significance estimates. This is particularly important for the PERG, the amplitude of which declines linearly with reduced contrast,¹⁶⁻¹⁸ leading to extremely low signal-to-noise ratios.

METHODS

Subjects

Seventeen visually normal subjects participated in one or several of the experiments, 9 in experiments 1 and 3 and 10 in experiment 2. Visual acuity was normal or corrected to normal (1.0). All subjects gave informed written consent to participate in the experiment and were naive as to the specific experimental question. The studies were performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.¹⁹

Stimuli

All stimuli were generated by computer (Power Macintosh G4; Apple Computer, Cupertino, CA) and presented on a monochrome monitor (GD 402; Phillips, Eindhoven, The Netherlands) with a framerate of 85 Hz at a viewing distance of 57 cm under a viewing angle of 29.1° × 38.0°. Luminance nonlinearity (γ)²⁰ was corrected. The actual pattern was displayed in a centered circular area with a diameter of 26.6°. Over the outer 2° rim of the circular stimulus, the contrast faded to zero, filling the rest of the screen with a luminance of 115 cd/m². A small cross in the center of the screen served as fixation target. The adapting stimulus reversed at 1.89 reversals per second to avoid local luminance

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adaptation. The test stimulus was a phase-reversing pattern with a reversal rate of 8.5 reversals per second, appropriate for steady state analysis.²¹

Experiment 1 sampled the contrast characteristic in two widely different adaptation states: After adaptation to either 98% or 7.3% we tested with five contrast levels in the following order: 17%, 98%, 17%, 2.8%, 40%, 7.3%, 2.8%, and 7.3%. Stimuli with low contrast were presented more often to compensate for the low amplitude. A checkerboard pattern with a check size of 0.97° ^{22,23} served as a pattern for both adaptation and test. A check size of 0.97° corresponds to a fundamental spatial frequency (at 45°) of 0.73 cyc/deg.

Experiment 2 compared four adaptational states. After adaptation to 0%, 7.3%, 29%, and 98%, a test contrast of 98% was applied. We used the same spatial pattern as in experiment 1.

Experiment 3 tested the transfer of adaptation between different orientations (in the retina, receptive fields are nonoriented²⁴ in contrast to most cortical cells). A horizontally or vertically oriented square-wave grating with a bar width of 0.67° (fundamental spatial frequency of 0.75 cyc/deg) served as a pattern for adaptation and test. Adaptation contrast was 7.3% or 98%, and the test contrast was 98%.

Procedure

The following structure was common to all experiments: after 10 minutes of initial adaptation to the particular contrast level, the sequence (941-msec test, 2020-msec readaptation) was repeated 100 to 200 times to achieve a reasonable signal-to-noise ratio. The various test and adaptation conditions were applied in a block design with intermittent breaks. One session lasted approximately 3 hours.

Electrophysiological Recording

PERGs were picked up in both eyes with DTL electrodes^{25,26} referenced to the ipsilateral canthi. VEP was recorded from Oz, referenced to averaged ears. A ground electrode was attached to the right wrist. Signals were amplified (Physiological Amplifier; Toennies, Höchberg, Germany) with band-pass settings of 2.5–70 Hz (PERG) and 0.3–70 Hz (VEP), digitized at 1 kHz, and streamed to disc.

Data Analysis

Signals were averaged off-line. As a first step, all trials exceeding $\pm 100 \mu\text{V}$ in any PERG channel were considered to be artifacts and excluded. All recordings in the same condition and the PERGs from the two eyes were averaged, always excluding the first response, to stay in a reasonable steady state. Further analysis took place in the frequency domain after discrete Fourier transformation (using IGOR Pro 4.0; WaveMetrics, Lake Oswego, OR). Based on the response magnitude at 8.5 Hz and the neighboring noise estimates at 7.3 and 9.7 Hz, we calculated a veridical amplitude estimate including phase, signal-to-noise ratio, and significance value.^{17,27} All amplitudes were normalized for each subject to the 98% test and 7.3% adaptation contrast condition (and additionally, in experiment 3 to the parallel condition). The phase convention was such that increasing phase also meant increasing latency (here used as a synonym for implicit time or peak time). In the phase analysis we excluded responses with $P > 0.05$.

Amplitude-contrast functions were fitted with the Naka-Rushton equation

$$r(c) = r_{\max} \cdot \frac{c^n}{c^n + c_{50}^n}$$

where c is contrast, $r(c)$ is the response estimate and (as free parameters) r_{\max} is the maximal response, n is the slope factor, and c_{50} is the contrast evoking 50% r_{\max} .

Statistical significance of experimental results was assessed with a repeated measures ANOVA on the non-normalized data (SPSS ver. 10.0; SPSS, Chicago, IL).

RESULTS

Experiment 1

In this experiment, test contrast varied, whereas one of two levels of contrast adaptation was maintained. The PERG and VEP traces of one subject are depicted in Figure 1. There was an obvious increase in amplitude with test contrast for both PERG and VEP. At contrasts below 10% the PERG traces were too noisy to detect signals by visual inspection. Nevertheless, Fourier analysis extracted a significant response in this subject, even at 7.3% test contrast. The effect of contrast adaptation was subtle and can best be seen in the parametric plot of the grand mean over all subjects after Fourier analysis (Fig. 2). Figure 2A depicts PERG amplitude versus linear test contrast, and Figure 2B shows VEP amplitude versus log test contrast, fitted with the Naka-Rushton equation. The overall shape of the contrast response function remained unchanged during contrast adaptation. For the PERG, it showed a nearly linear shape (on a linear-linear plot) and for the VEP, a sigmoidal shape (on a semilogarithmic plot). Adaptation reduced the PERG amplitude at high test contrasts to 90%; at lower test contrasts the adaptation effect, if any, was within the error margins. The VEP contrast transfer function was shifted by adaptation to higher contrast values (c_{50} changed from 3.5% to 6.5%), with an enhancement at high test contrasts (r_{\max} rose from 1.0 to 1.2).

On an individual basis, changes in PERG amplitude were not significantly correlated with amplitude changes in the VEP (for high test contrasts, $P = 0.24$).

Compared with the amplitude, the phase (latency) showed a different picture (Fig. 2C). VEP latency decreased from the lowest to the intermediate test contrasts, whereas there was a moderate latency increase above approximately 30%. In the PERG, a highly significant effect on latency was seen: a decrease of 4 to 5 msec. Adaptation to high contrasts reduced the latency at all test contrasts at which a reliable PERG could be evoked. Possibly, adaptational effects in the VEP were buried in latency scatter, which was higher than in the PERG.

In a control experiment suggested by one of our reviewers, we tested whether psychophysical contrast thresholds were changed by adaptation to the two contrast levels. Adaptation and test timing was similar to the electrophysiological setup, and contrast threshold was measured with a custom acuity-contrast test.²⁸ In all six subjects tested, threshold rose by adaptation to high contrast levels. Interindividual variability was high, with the effect ranging from a factor of 1.4 to 7.8 (mean 3.4; $P < 0.05$).

Experiment 2

To assess the linearity of the adaptation effects, in this experiment we varied adaptation contrast in four steps from 0% to 98%, whereas retinal and cortical responses were obtained with 98% test contrast. Figure 3 presents amplitude (Fig. 3A) and latency (Fig. 3B) versus adaptation contrast for PERG and VEP. The amplitudes are normalized to the 7.3% adaptation contrast condition to facilitate comparisons with experiment 1. The results were similar to those from experiment 1 for the 98% adaptation contrast: a decrease of PERG amplitude to 89% and an increase of VEP amplitude to 133%. These effects appeared to be linearly related to adaptation contrast, in that a straight line connected all data points within the range of measurement error.

In the phase domain (latency) the findings of experiment 1 were also reproduced and, again, the effects depended linearly on adaptation contrast (Fig. 3B).

Experiment 3

This experiment was designed to test the interactions between adaptation orientation and test orientation. All combinations of

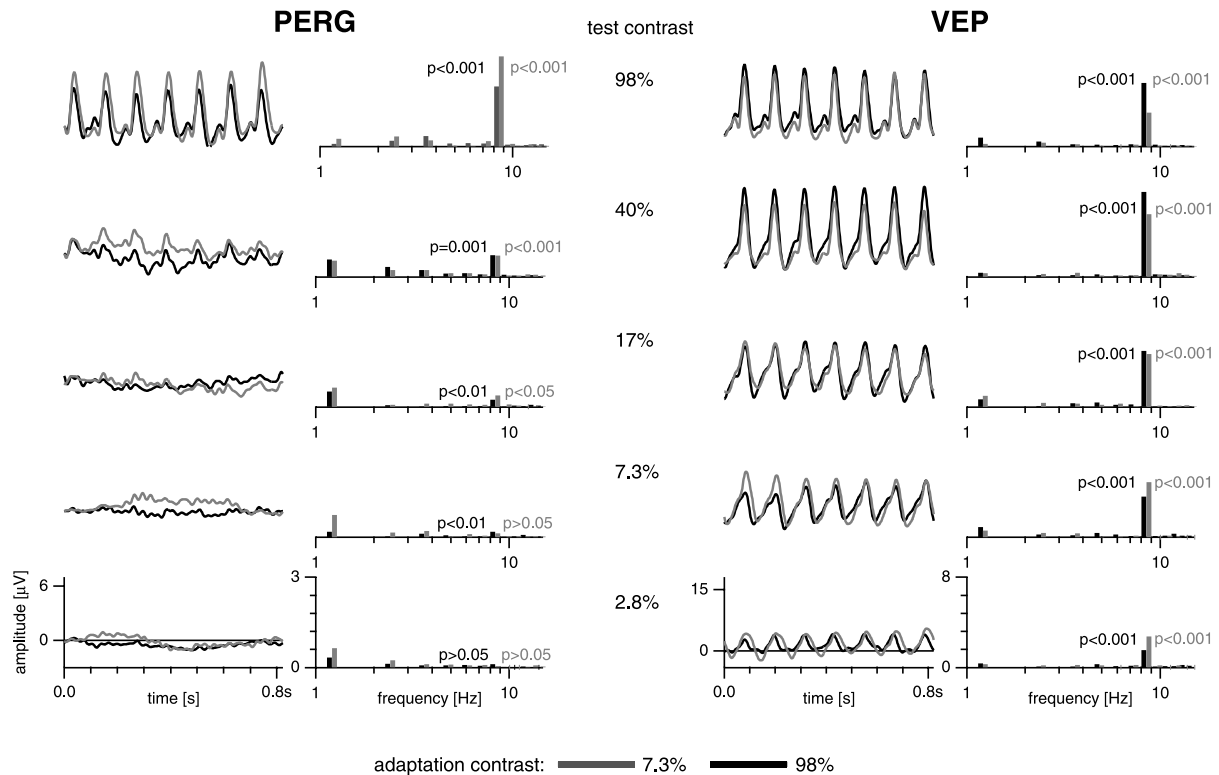


FIGURE 1. Raw traces and corresponding Fourier spectra of the PERG (*left*) and VEP (*right*) at five test contrasts (*top to bottom*) in a low- and high contrast-adaptation condition. Data are from one subject. In the spectrum, the response appears as a spike at the reversal rate of 8.5 reversals per second. Probabilities indicate the significance, based on the signal-to-noise ratio. Test contrast had a marked effect on amplitude, adaptation effects (*black versus gray* curves) are comparatively subtle.

vertical and horizontal adaptation and test gratings with low and high adaptation contrasts were applied and pooled into parallel or orthogonal stimulus orientations, depending on the differences between adaptation and test gratings.

A 2×2 ANOVA (adaptation contrast \times orientation) was performed for retinal and cortical responses. In the PERG, a significant effect was seen only for adaptation contrast ($P < 0.001$). In the VEP, a significant main effect ($P < 0.001$) of adaptation contrast and a significant interaction of orientation and adaptation contrast ($P < 0.05$) was observed. Post hoc analysis revealed that the effect of orientation occurred only with the high adaptation contrast, as expected.

Figure 4A displays these findings with mean retinal responses normalized to the parallel low adaptation-contrast condition. With low adaptation contrast (Fig. 4A, left), there was no difference when the test grating was parallel or orthogonal to the adaptation grating. After adaptation to a high-contrast grating, the PERG was significantly reduced to 87% ($P < 0.05$) in both the parallel and the orthogonal conditions, confirming the results of the two previous experiments.

Figure 4B shows the mean cortical responses, normalized to the parallel low adaptation-contrast condition. Although there was, again, no effect of stimulus orientation at low adaptation contrast (Fig. 4B, left), high adaptation contrast enhanced the VEP response in the parallel condition to 125%. The results match closely the findings of the two previous experiments. If the test grating was orthogonal to the adaptation grating, there was a more pronounced enhancement to 149% ($P < 0.05$ compared with the parallel condition).

Phase (latency) effects for the PERG were as in experiments 1 and 2—that is, a latency reduction by contrast adaptation ($P < 0.05$) of 6.0 msec in the parallel and 6.6 msec in the orthogonal conditions. In the VEP, the latency characteristic

was rather constant, and the scatter was higher than with the PERG.

DISCUSSION

We found pronounced effects of contrast adaptation in both retinal and cortical recordings. However, quantity and quality of the effects differed markedly at these two levels of visual processing. In the VEP, the effects were predominantly on amplitude: Adaptation shifted the contrast transfer function toward higher test contrasts; the amplitude to high test contrasts was enhanced by adaptation, and with oriented adaptation and test gratings, there was further enhancement in the case of orthogonal orientation. In the PERG, contrast adaptation had two major effects: The amplitude was reduced to 90%, and the latency was reduced by approximately 6 msec.

The marked differences between the VEP and PERG warrant separate discussion, first of the VEP, because this field is more developed in the literature. The VEP-amplitude findings confirm and extend previous reports. Mecacci and Spinelli¹² used a test contrast of 8% and observed a decrease of the VEP amplitude to 25% after contrast adaptation in their single subject. In the present study, we found a reduction only to 75% (Fig. 2B, 7.3% contrast). Nelson et al.¹⁵ used a contrast sweep technique, in which the test contrast rose and fell over the range from 0% to 20% during 20 seconds. If contrast adaptation were rapid enough, a hysteresis between the ascending and descending parts of the contrast sweeps would be expected. They found that contrast adaptation elevated the VEP threshold,¹⁵ which corresponded to a decrease in amplitude at low test contrasts. Brigell et al.,⁷ using the same recording procedure, reported similar findings for the VEP. Their simultaneous

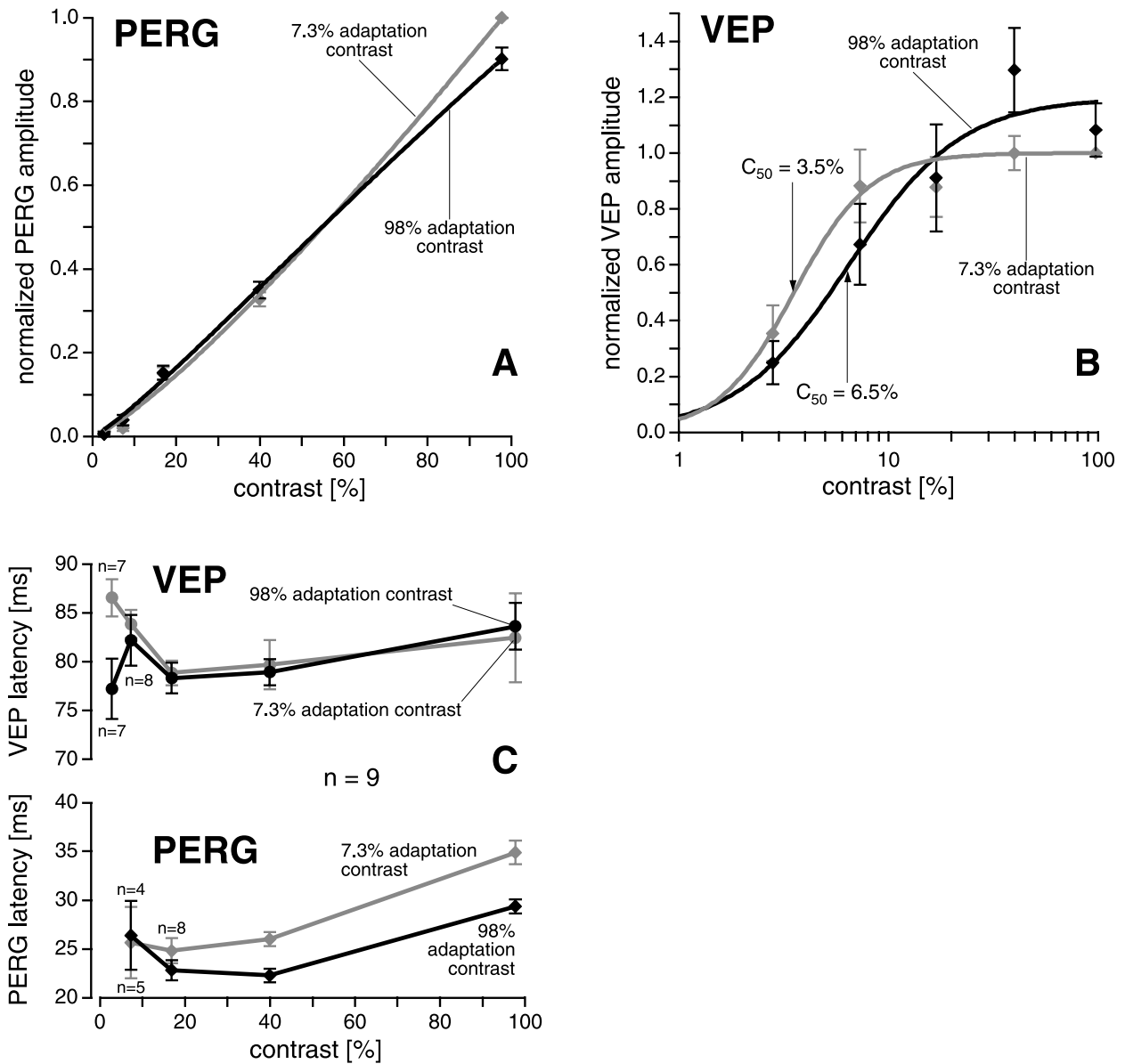


FIGURE 2. Effect of contrast adaptation on the contrast transfer function in PERG amplitude (A), VEP amplitude (B), and PERG/VEP phase (C). Data points represent the averages from nine subjects, error bars indicate SEM, *gray lines* show adaptation to 7.3%, and *black lines* adaptation to 98% contrast. Note the different scales of the x-axis. PERG amplitudes depend linearly on test contrast. Adaptation reduced amplitude at high test contrasts. VEP amplitudes displayed a sigmoidal function over logarithmic contrast. The function was shifted to higher test contrast and increased in r_{\max} with adaptation to high contrasts. PERG phase was significantly reduced at 40 and 98% test contrast after adaptation to high contrast. Adaptation in the VEP was more apparent for the amplitude, in the PERG more for the temporal domain.

PERG recording are discussed later. Bach et al.¹⁴ observed that contrast adaptation reduced the VEP amplitude below approximately 4% contrast and increased it at higher test contrasts. With a slight shift on the contrast axis, which may be due to different stimuli (sinusoids versus checkerboards), this fits well with the current data. The enhancement at high test contrasts was confirmed in the present study. This initially contrainuitive effect of an amplitude increase in high-contrast adaptation has, however, also been found in single-cell recordings from macaque cortex by Sclar et al.¹⁰ They modeled their cells' contrast transfer function with the Naka-Rushton equation and found that adaptation to high contrast clearly increased c_{50} in most of their 24 cells, moderately increased in the slope factor n , and increased r_{\max} in approximately half of their cells. Their Figure 4 (see Ref. 10, Fig. 4, top right) shows that pre- and postadaptational spike rates are roughly evenly distributed on

a log-log plot. In other words, a 200% r_{\max} -increase cell was balanced by a 50% r_{\max} -decrease cell. When VEP amplitude is interpreted as an arithmetic population average of the spike rate, amplitude consequently increases in adaptation.

In general, all VEP findings fit very well into the general context of contrast adaptation mechanism. The contrast transfer function is shifted to higher contrasts, also known from single-cell studies.¹⁰ The c_{50} increases to keep optimal discriminability (the steepest part of the transfer function) in the middle of the current range, and r_{\max} increases moderately. The c_{50} -shift also entails an increase in the psychophysical threshold.²⁹

Phase (latency) effects in the VEP, depending on spatial frequency, have been described previously. For instance, Strasburger et al.³⁰ analyzed the VEP at various test contrasts and temporal and spatial frequencies. At conditions similar to ours

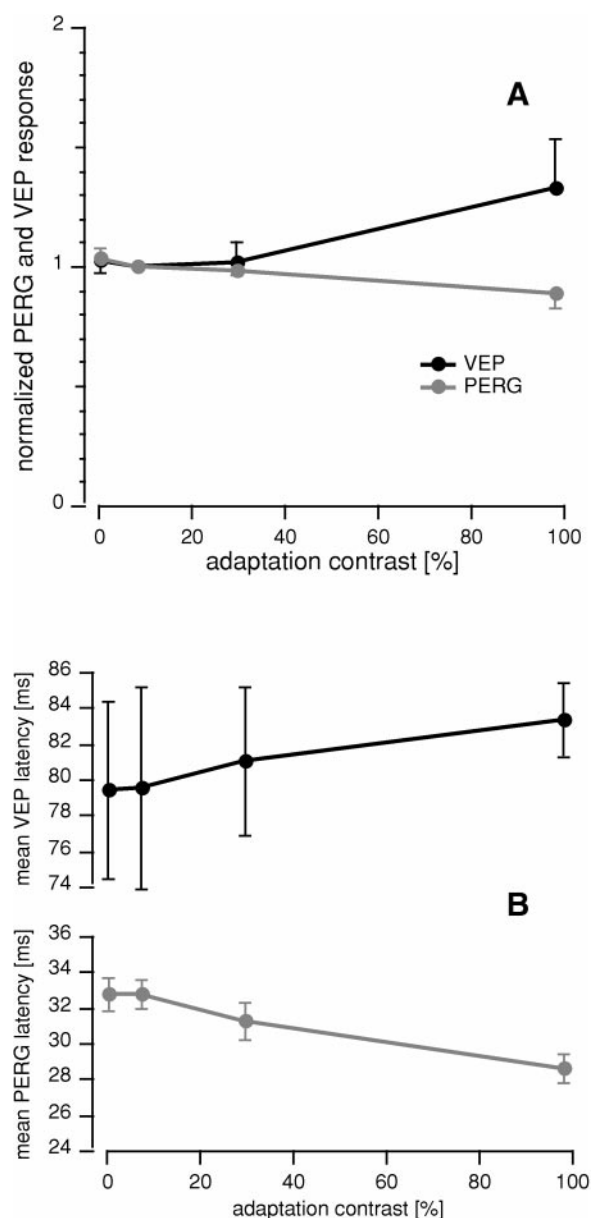


FIGURE 3. Strength of adaptation effects as a function of adaptation depth at high test contrast (98%). Data are averages of 10 subjects. The amplitudes are normalized to the low adaptation contrast condition (7.3%). Within the error margin, all changes in amplitude and phase appear to be linearly dependent on adaptation contrast.

(0.5 and 1 cyc/deg), they report that latency declines with increasing test contrast up to 20% and then stays roughly constant, where phase at lower test contrast is much more variable. This is also seen in the present study (Fig. 1C). Adaptation effects on VEP phase have not been reported so far. All effects, if any, appeared to be submerged in latency variability in the present study (but see the following description of PERG findings).

In experiment 3 we used oriented gratings and compared the responses for the conditions of parallel or orthogonal orientation between adaptation and test. Keeping in mind that the VEP is a mass response, presumably reflecting the maximally stimulated cells at any stimulus orientation,^{31,32} the same results would be expected as with checkerboards (above) for the parallel condition. Indeed, we found an amplitude enhancement to 125% (compare Fig. 4B with Fig. 2B).

For the orthogonal condition, the VEP displayed an even more marked amplitude enhancement to approximately 150% (Fig. 4B). We interpret this as follows: Let us assume that there is orientation inhibition from cells with an orthogonally preferred direction, as found in cat visual cortex.⁹ In the low-contrast adaptation condition, the orthogonal inhibitors have some, but little, effect, because they are not stimulated at their preferred orientations. When they have been adapted, they inhibit even less, because their transfer characteristic has been shifted to higher contrast values (we here assume that suboptimal orientation has comparable effects as low contrast on cellular activity). Because of this reduced inhibition, the cells tuned optimally to the test orientation are more strongly activated, further adding to the VEP enhancement that was described.

In view of the discussion on r_{\max} , we conclude that there is satisfactory agreement between single-cell and VEP findings.

Our PERG findings differed markedly from the VEP findings. The results of the two previous PERG studies were controversial. Odom and Norcia⁸ adapted one subject to 0% and 50% contrast for 2 minutes. With a test contrast of 30% they found an amplitude decrease of 30%. This strong finding is surprising, given the low PERG amplitude at 30% contrast, and unfortunately no error margins are reported. Brigell et al.⁷ recorded both PERG and VEP in three subjects with a sweep technique, where the test contrast rose and fell over the range from 0% to 20% during 20 seconds. If contrast adaptation were rapid enough, a hysteresis between the ascending and descending parts of the contrast sweeps would be expected. This was indeed observed in the VEP, but not in the PERG.

We hypothesize that the most parsimonious explanation for the effect of contrast adaptation on PERG amplitude is a small relative amplitude reduction to approximately 90%, which is only significant at high test contrasts (Fig. 2A). Although contrast adaptation may have little effect on PERG amplitude, it leads to a highly significant PERG latency reduction of approximately 6 msec (Figs. 2C, 3B), which has not yet been described in previous studies. Given that the VEP reflects activity from sites more central along the visual pathway than the PERG, this latency effect would be expected to be visible also in the VEP. However, this trend, if existent, is submerged in the scatter of VEP latency which is much more than in PERG latency. This can be expected, given that several synaptic processes interpose between the PERG and VEP generation sites.

In primates, contrast adaptation has not been investigated on the single-cell level. Rapid gain-control mechanisms over a few hundred milliseconds were studied by Benardete et al.³ They found contrast gain control for a subset of P_{α} -ganglion cells, the M_x . They used a range of 2% to 12% adaptation contrast and plotted their findings in terms of gain (see Fig. 1 in Ref. 3) and temporal filter parameters (see Fig. 2 in Ref. 3). In terms of amplitude, these findings translate to an amplitude reduction due to adaptation in the range of 30% and to a latency reduction of approximately 10 msec. M_y and P_{β} cells were not found to be markedly affected by adaptation. In the present study, which was designed to address long-term contrast adaptation, we tested for 900 msec at any given test contrast and then readapted. Consequently, rapid gain control should have been completed early during any of the test procedures. Although the PERG is an unknown mix of magno- and parvocellular activity (M ganglion cells are less frequent³³ but have larger cell bodies,^{33,34} possibly resulting in a larger contribution to the PERG), we note that the direction and the order of magnitude of the present findings are similar with those found by Benardete et al.³ This suggests that both contrast gain control and contrast adaptation lead to similar effects, although on a different time scale.

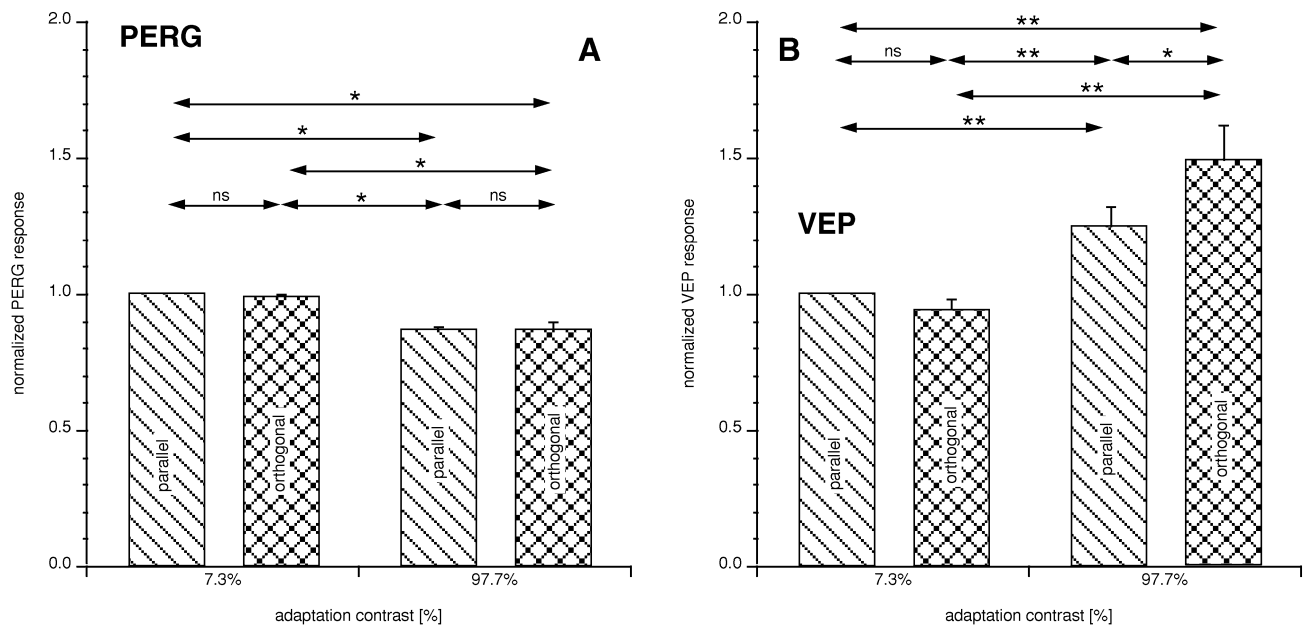


FIGURE 4. Effects of orientation: adaptation with parallel or orthogonal test grating. Contrast adaptation of 7.3% and 98% with horizontal or vertical gratings. Normalized PERG (A) and VEP (B) amplitudes \pm SEM. Data are averages of nine subjects. Significance values represent ANOVA post hoc comparisons. At low adaptation contrasts, there was no significant difference between the parallel and the orthogonal orientations. The PERG amplitude showed the same contrast adaptation characteristic as in Figure 2, with no effect of stimulus orientation. The VEP, for the parallel orientation, showed the same contrast adaptation characteristic as in Figure 2. With the orthogonal orientation, there was an even more pronounced enhancement after adaptation to high contrast. The significance levels are indicated as follows: ns (not significant) or $P > 0.05$; $*P \leq 0.05$; and $**P \leq 0.01$.

A rapid gain control mechanism in magnocellular ganglion cells appears reasonable, because motion detection, which mainly relies on the magnocellular system, has only approximately 10% contrast in dynamic range³⁵ and consequently requires rapid adjustment.

In experiment 3, no effect of stimulus orientation was seen in the PERG, as expected, because there are no oriented receptive fields in the retina.²⁴ This is in contrast to the observations in the VEP recordings and emphasizes the differences between cortical and retinal contrast-adaptation mechanisms. The actual site of retinal contrast adaptation can be within or before the retinal ganglion cells. Findings by Brown and Masland⁶ in rabbit retina suggest that some contrast adaptation already takes place in bipolar cells.

Testing the contrast sensitivity by itself is likely to shift the current adaptation state, depending on the exposure duration relative to the time constants involved, the current adaptational state, and the difference from the preceding contrast. Rapid gain control occurs over a few hundred milliseconds and played only a small role in our experiments, in which longer times were studied. The slower part of contrast adaptation ($\gg 1$ second) may be on the order of 10 seconds, or even several minutes, if comparable to motion.³⁶ In any case, the duration of the initial adaptation period in our experiments (10 minutes) should have introduced marked adaptation and, during the test, there was a 1:2.2 ratio of test to readaptation. For low adaptation contrasts, this duty cycle could have introduced a slightly higher adaptation level, possibly reducing the strengths of the effects we could observe (see Hoffmann et al.³⁶ for a discussion of the dynamic issues of ramping up and down the adaptation level). Specifically, it may be fruitful to assess the time constants of contrast adaptation in humans.

Data on retinal contrast adaptation in humans have been scarce and controversial. Our findings suggest that different types of contrast adaptation mechanisms occur in retina and cortex, respectively. In the VEP, a contrast-divisive effect—a

shift along the log (contrast) axis—was dominant (Fig. 2B). In the PERG, adaptation effects occurred mainly in the temporal domain, as indicated by the reduced latency under high-contrast adaptation.

The differences between PERG and VEP effects fit well with the findings of Carandini et al.,¹¹ which they obtained by intracellular recordings from cortical cells. Whereas contrast adaptation left visual input largely unaffected (PERG), it reduced the cells' spiking activity (VEP) through tonic hyperpolarization.

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