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Rittenhouse, Cynthia D., Beth A. Siegler, Courtney A. Voelker, Harel Z. Shouval, Michael A. Paradiso, and Mark F. Bear. Stimulus for rapid ocular dominance plasticity in visual cortex. *J Neurophysiol* 95: 2947–2950, 2006. First published February 15, 2006; doi:10.1152/jn.01328.2005. Although it has been known for decades that monocular deprivation shifts ocular dominance in kitten striate cortex, uncertainty persists about the adequate stimulus for deprivation-induced losses of cortical responsiveness. In the current study we compared the effects of 2 days of lid closure and 2 days of monocular blur using an overcorrecting contact lens. Our finding of comparable ocular dominance shifts in visual cortex indicates that deprived-eye response depression is not a result of reduced retinal illumination. The quality rather than the quantity of retinal illumination is the key factor for ocular dominance plasticity. These data have implications for both the mechanism and treatment of amblyopia.

INTRODUCTION

Ocular dominance (OD) plasticity in visual cortex is a striking example of how the qualities of sensory experience influence receptive field development in the neocortex. Monocular deprivation (MD), usually produced experimentally by unilateral lid closure, rapidly causes cortical neurons to lose responsiveness to the deprived eye. Closing an eye *1*) greatly reduces the amount of light falling on the retina and *2*) degrades image formation. The relative contributions of these two effects of eyelid closure on the ocular dominance shift are important to understand because this knowledge constrains possible mechanisms.

Over the past four decades, beginning with the seminal experiments of Wiesel and Hubel (1963), there have been a number of reports that merely diffusing or blurring images in one eye is sufficient to cause an ocular dominance shift. However, most of these observations have been cursory (e.g., few animals or absence of controls for statistical comparisons) and/or complicated by prior dark rearing (Blakemore 1976; Christen and Mower 1987; Eggers and Blakemore 1978; Maguire et al. 1982; Tieman et al. 1983). There has been only one systematic and quantitative comparison of monocular diffusion and occlusion. In kittens dark reared to 4 wk of age, Christen and Mower (1987) found comparable ocular dominance shifts after ≥15 additional weeks of visual experience with one eye covered with either a diffusing contact lens or an occluding lens. However, the long duration of the deprivation does not allow any conclusions to be reached about how rapidly these shifts occurred, and the prior dark rearing complicates the interpretation (selective monocular experience vs. monocular deprivation). The problem of dark rearing was avoided in studies by Ikeda and Tremain (1978), who blurred the images in one eye by repeated daily treatment of three kittens with atropine, and by Von Noorden and Crawford (1977), who removed the lens from one eye in three monkeys. However, neither study directly compared the consequence of monocular blur and lid closure and, again, the long duration of the treatment makes it difficult to discern whether the rate of the OD shift was different.

Renewed interest in this question has come from two recent findings. First, it is now clear that the OD shift produced by temporarily silencing all activity in one retina is significantly less (Rittenhouse et al. 1999) and qualitatively different (Frenkel and Bear 2004) from that caused by lid closure. These findings suggest a much greater role for the residual activity in the deprived retina in triggering deprived-eye depression than previously suspected. Second, it is now clear that the OD shift can be accounted for by both deprived-eye depression and open-eye potentiation (Frenkel and Bear 2004; Mioche and Singer 1989; Sawtell et al. 2003). Only after brief periods of deprivation is the OD shift accounted for solely by deprived-eye depression. Our aim in the present study was to perform a quantitative comparison of the consequences of brief monocular deprivation by blur and lid closure in young normally reared kittens.

METHODS

Monocular deprivation

Kittens were reared in a normal 12/12 light cycle until postnatal day P45–P61, and then divided into three groups: those receiving monocular blur (MB) for 2 days, those receiving monocular lid suture (MS) for 2 days, and those with normal binocular vision (NR). Animals in the MB group had a hard contact lens inserted into the left eye. These lenses were made in the strongest available prescription correction for myopia (planar lens with −10 diopters correction), resulting in a focal distance beyond the limit of the walls of the animal room (Fig. 1). MS kittens had the lids of their left eye sutured under isofluorane anesthesia (3% in 1 liter/min O_2) after placement of a plano (noncorrecting) hard contact lens in that eye. Lenses were 12 mm in diameter, large enough to cover the iris and pupil. Animals remained on a 12/12 light cycle, but during each dark phase were moved to a darkroom. MS kittens had fully recovered from anesthesia at the onset of their first 12-h light phase. After 48 h of restricted visual experience,

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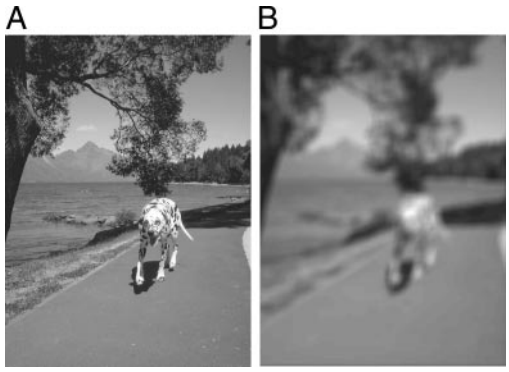

FIG. 1. Simulated visual image viewed through a diffusing contact lens. *A*: a natural visual image as it would be seen through a nondeprived eye. *B*: same image seen through a contact lens with a corrective power of minus 10. Assuming an eye diameter of 15 mm with an appropriate natural lens system (power 1/0.015), an object at infinity with focus at 0.013, instead of 0.015 m (13 mm), a point at infinity would have a physical size on the retina of (pupil size) × (0.002/0.013) = 15% of pupil size. For a 0.2-cm pupil, the point radius is 0.03 cm. If an image falls on a 1-cm^2 area of retina, then a point is 3% of image length. For a 256 × 256 pixel image, this means convolving the image with a spot of radius of nearly 8 pixels; the result is blurred details with retention of large object shapes.

animals were immediately prepared for extracellular recording of cells in area 17 as described previously (Rittenhouse et al. 1999). NR animals did not wear contact lenses. In all cases, the optics were confirmed to be clear in both eyes before recording.

Cortical electrophysiology and analyses

Recordings were made in area 17 of the right hemisphere, contralateral to the deprived eye, as described by Rittenhouse et al. (1999). Briefly, single-unit recordings were made using glass-covered tungsten electrodes (Alan Ainsworth, London, UK or FHC, Bowdoinham, ME). Successive units were recorded, ≥125 μm apart, along penetrations that ran tangentially through striate cortex down the medial bank of the lateral gyrus. Cells from all layers of cortex were sampled. Unit responses were isolated and the cell's receptive field was mapped using a handheld light bar. Each eye then independently viewed moving sinusoidal gratings at 16 evenly spaced orientations and one blank screen while the other eye was covered close to the eye to eliminate scattered light entering the eye. Responses to five trials were collected in peristimulus time histograms (PSTHs). The visual stimuli were high-contrast sinusoidal luminance gratings with a spatial frequency of 1 cyc/deg drifting at 1 Hz. Experience in both our laboratory and that of other investigators shows that gratings with these parameters drive the majority of neurons in striate cortex neurons. Ocular dominance measurements with fixed stimulus parameters (i.e., nonoptimized) are reliable because receptive field structure as well as orientation and spatial frequency selectivity are matched in the two eyes (Skottun and Freeman 1984) and responses fall off monotonically from the peak response. Because our aim was to quantitatively measure ocular dominance in as many neurons as possible, we did not go to any lengths to optimize stimuli for individual neurons, which would have slowed our rate of data collection. Therefore we do not report any data on possible differences in peak firing or orientation selectivity.

A computer-generated ocular dominance (OD) score for each unit was calculated according to the following equation: OD = (R − L)/(R + L), where "R" is the right eye response to the preferred stimulus (orientation and direction) minus spontaneous activity (blank screen trial), and "L" is the left eye response to the preferred stimulus minus spontaneous activity. Thus scores ranged from −1.0, driven by the contralateral (left) eye, to 1.0, driven by the ipsilateral (right) eye. To ensure accuracy, scoring was corroborated subjectively by auditory monitoring of responses to a handheld light bar. A five-category system was used. Briefly, cells in category 1 responded only to stimulation through the contralateral eye; cells in category 5 responded only to stimulation of the ipsilateral eye; and those in category 3 responded equally well to stimulation of either eye, with categories 2 and 4 representing intermediate responses. The computer OD scores (−1.0 to 1.0) were later binned into five bins for comparison. When a computer score was not within ±1 OD category of the experimenter's score, the data for that cell were examined by a third party, blind to the deprivation condition, who decided whether the computer score should stand or whether the cell should be disregarded. If the quantitative measure of OD was obviously corrupted by differences in spontaneous activity across trials or by a poor signal-to-noise ratio, these data were excluded; on this basis, 14% of the recordings in each group were disregarded.

Comparisons across groups were made using nonparametric statistics. Cumulative probability distributions were analyzed using Kolmogorov–Smirnov tests, group analyses with Kruskal–Wallis tests, and, where appropriate, Mann–Whitney *U* tests were used for post hoc comparisons between groups.

RESULTS

Extracellular single-unit recordings of area 17 neurons were obtained from kittens reared normally (n = 7 animals) or visually deprived for 48 h by either MS (n = 10 animals) or MB (n = 11 animals). All recordings were from the right hemisphere, contralateral to the deprived (left) eye. Because OD plasticity declines with age (Hubel and Wiesel 1970; Mower 1991), we intentionally studied older animals, whose OD shifts would be reduced or slower compared with animals at the peak of the critical period, and we kept the deprivation periods brief to examine plasticity before it reached maximum levels. Animal ages were matched across groups: the average age (in days) for the MB group was 53.3 ± 1.5, for the MS group was 52.6 ± 1.5, and for the NR group, 52.1 ± 1.7.

Cortical plasticity was assessed using a quantitative OD score for each recorded unit (see METHODS). Scores ranged from −1.0 for cells responsive only through the contralateral eye to 1.0 for cells responsive only to stimulation of the ipsilateral eye; cells with an OD score of 0 responded equally well through each eye. Cumulative probability distributions of OD were plotted for all units in each experimental group (NR; n = 144 units; MS: n = 220 units; MB: n = 307 units; Fig. 2*A*). As expected, in NR animals there was a higher proportion of units dominated by the contralateral eye (OD scores <0). However, in both deprivation groups the distributions were shifted toward the ipsilateral (nondeprived) eye (OD scores >0). There was a significant effect of group ($P < 0.0001$, Kruskal–Wallis test), and post hoc analyses confirmed that both deprivation groups had OD score distributions that were significantly different from those of the normally reared animals ($P < 0.0001$, Kolmogorov–Smirnov tests), whereas the MB and MS distributions were not significantly different from each other.

For more conventional visual examination of the data, OD histograms were derived for each group of animals by dividing the pooled OD score distribution into five equal-sized bins (Fig. 2, *B–D*). These OD histograms reveal unmistakable shifts in OD of cells from MB (Fig. 2*C*) and MS (Fig. 2*D*) animals compared with normal controls (Fig. 2*B*).

OD data pooled from many animals can be biased toward those individual cases where the largest number of units were studied. Therefore we also analyzed the results by case (Fig. 2,

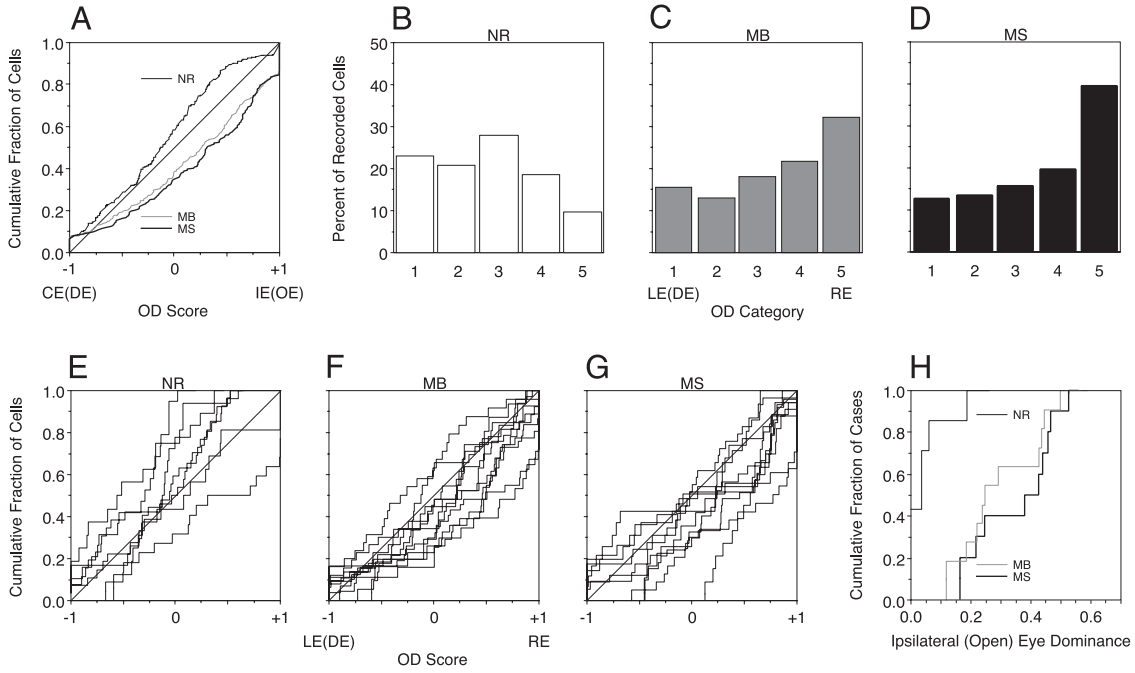

FIG. 2. Analysis of the ocular dominance data. *A*: cumulative fraction of the total number of recorded units with each ocular dominance (OD) score is plotted for each group: normal (thin black line), monocular blur (MB, thick gray line), and monocular deprivation (MD, thick black line). Two distributions of scores from deprived animals are significantly different from the scores from normal binocular vision (NR) animals ($P < 0.0001$, Kolomogorov–Smirnov test). *B–D*: percentage of recorded cells in each of 5 OD categories pooled for each of the 3 groups of animals NR ($n = 144$), MB ($n = 307$), and MD ($n = 220$). Cells in category 1 responded solely through the left (deprived) eye, cells in category 5 responded solely through the right (nondeprived) eye, category 3 cells were driven equally well through either eye, cells in groups 3 and 6 fell in between. *E–G*: analysis of ocular dominance by individual case. Stepped cumulative probability distributions for the units with each OD score from each NR ($n = 7$), MB ($n = 11$), and MD ($n = 10$) animal is plotted against OD score. From these distributions, an ipsilateral-eye dominance (IED) was determined for each animal as the percentage of cells with OD scores between 0.5 and 1.0. *H*: cumulative fraction of animals with each IED score from NR, MB, and MD animals.

E–G). Cumulative probability distributions of OD scores for each animal reveal a clear tendency for MS and MB animals to have a larger proportion of positive OD scores [i.e., more cells dominated by the ipsilateral (nondeprived) eye] than NR animals. To quantify this effect, an ipsilateral-eye dominance (IED) score, reflecting the percentage of neurons with OD scores >0.5, was determined for each case (Rittenhouse et al. 1999). Figure 2*H* shows the cumulative probability distribution of the IED values of the individual animals in each group. The MS and MB groups are significantly different from NR ($P < 0.01$, Mann–Whitney *U* test), but not different from each other.

DISCUSSION

Compared with their normally reared counterparts, striking changes in OD were observed in kittens for which pattern vision in one eye during a brief period of monocular deprivation was distorted but not eliminated. That is, we were able to induce significant OD shifts comparable to lid suture, simply by blurring visual input to one eye, blurring similar to the image seen by a person with normal vision looking through the lens of a very myopic friend's eyeglasses.

The OD shift after monocular deprivation during the critical period can be accounted for by deprived-eye depression, open-eye potentiation, or both. Definitive determination of the basis for the OD shift requires chronic recordings. In mice this is easily achieved with recordings of visually evoked potentials (VEPs), and such studies reveal that the OD shift occurring over the first 3 days of deprivation is accounted for solely by deprived-eye depression (Frenkel and Bear 2004). As a result of cortical anisotropies such as ocular dominance and orientation columns, chronic recordings are much more challenging in kittens. Nonetheless, Mioche and Singer (1989) were able to follow the activity of some neurons in kitten visual cortex over the course of monocular deprivation. They state that "during the shift in ocular dominance, the responses to the open eye remained remarkably stable" (p. 189). Moreover, by 48 h of deprivation, the majority of cells in their sample lacked *any* response to the deprived eye, which can be accounted for only by deprived-eye depression. Finally, in their description of the effects of reverse suture, they state that "invariably, the first effect of reversal was a decrease of the response to the newly deprived eye" (p. 191). Thus taken together, it seems safe to assume that the OD shift after brief monocular deprivation is largely attributable to deprived-eye depression. Therefore we used 2 days of deprivation in this study—the minimum time required to get a reliable shift in kittens at 7–8 wk, an age range that is well within the critical period, but after the peak sensitivity to deprivation to avoid saturation effects.

Our finding that MB rapidly shifts OD is inconsistent with the pervasive dogma that response depression in cortex is a result of diminished light stimulation of the retina (reviewed by Sengpiel and Kind 2002). With a blurring contact lens, effective image contrast is reduced, but average luminance is unaffected. Thus the current result, considered together with previous experiments using monocular inactivation with tetrodotoxin (TTX) (Frenkel and Bear 2004; Rittenhouse et al. 1999), supports an alternative idea that it is the *pattern* of activity—not the *rate*—that drives response depression during

monocular deprivation (Blais et al. 1999). According to the assumptions of this theory, correlated activity arising from an open eye viewing high-contrast patterns is more likely to elicit a postsynaptic response than uncorrelated activity in a deprived eye. If synaptic depression occurs when afferent activity occurs at times of low postsynaptic activity, and synaptic potentiation at times of high postsynaptic activity (reviewed by Bear 2003), depression of the deprived-eye synapses would occur in the MB case even if the open- and deprived-eye retinas were to have the same average activity level (Blais et al. 1999; Sengpiel and Kind 2002).

A molecular basis for rapid deprivation-induced response depression has recently been identified. Heynen et al. (2003) showed in rats that MS causes the same changes in postsynaptic glutamate receptors in rat visual cortex as occurs in the slice model of homosynaptic long-term depression (LTD) (Heynen et al. 2003). One of these changes is a significant loss of surface-expressed α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors. Interestingly, this molecular change did not occur when they inactivated the deprived eye with TTX, a procedure that eliminates any residual retinal activity. Thus we suggest that MB and MS are equally effective in shifting OD because they both provide the conditions necessary to drive glutamate receptors away from synapses: the presence of presynaptic activity that fails to correlate with strong postsynaptic activation.

Regardless of the mechanism, however, we can now conclude in kittens that two different conditions produce rapid OD shits, monocular lid suture and blur; and two different conditions fail to shift OD, monocular inactivation and normal rearing. Understanding how these four types of experience affect the amount and timing of activity in the lateral geniculate nucleus and cortex is likely to reveal the natural basis for response depression during cortical development.

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REFERENCES

Bear MF. Bidirectional synaptic plasticity: from theory to reality. *Philos Trans R Soc Lond B Biol Sci* 358: 649–655, 2003.

Blais BS, Shouval HZ, and Cooper LN. The role of presynaptic activity in monocular deprivation: comparison of homosynaptic and heterosynaptic mechanisms. *Proc Natl Acad Sci USA* 96: 1083–1087, 1999.

Blakemore C. The conditions required for the maintenance of binocularity in the kitten's visual cortex. *J Physiol* 261: 423–444, 1976.

Christen WG and Mower GD. Effects of monocular occlusion and diffusion on visual system development in the cat. *Brain Res* 415: 233–241, 1987.

Eggers HM and Blakemore C. Physiological basis of anisometropic amblyopia. *Science* 201: 264–267, 1978.

Frenkel MY and Bear MF. How monocular deprivation shifts ocular dominance in visual cortex of young mice. *Neuron* 44: 917–923, 2004.

Heynen AJ, Yoon BJ, Liu CH, Chung HJ, Huganir RL, and Bear MF. Molecular mechanism for loss of visual cortical responsiveness following brief monocular deprivation. *Nat Neurosci* 6: 854–862, 2003.

Hubel DH and Wiesel TN. The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *J Physiol* 206: 419–436, 1970.

Ikeda H and Tremain KE. Amblyopia resulting from penalisation: neurophysiological studies of kittens reared with atropinisation of one or both eyes. *Br J Ophthalmol* 62: 21–28, 1978.

Maguire GW, Smith EL 3rd, Harwerth RS, and Crawford ML. Optically induced anisometropia in kittens. *Invest Ophthalmol Vis Sci* 23: 253–264, 1982.

Mioche L and Singer W. Chronic recordings from single sites of kitten striate cortex during experience-dependent modifications of receptive-field properties. *J Neurophysiol* 62: 185–197, 1989.

Mower GD. The effect of dark rearing on the time course of the critical period in cat visual cortex. *Dev Brain Res* 58: 151–158, 1991.

Rittenhouse CD, Shouval HZ, Paradiso MA, and Bear MF. Monocular deprivation induces homosynaptic long-term depression in visual cortex. *Nature* 397: 347–350, 1999.

Sawtell NB, Frenkel MY, Philpot BD, Nakazawa K, Tonegawa S, and Bear MF. NMDA receptor-dependent ocular dominance plasticity in adult visual cortex. *Neuron* 38: 977–985, 2003.

Sengpiel F and Kind PC. The role of activity in development of the visual system. *Curr Biol* 12: R818–R826, 2002.

Skottun BC and Freeman RD. Stimulus specificity of binocular cells in the cat's visual cortex: ocular dominance and the matching of left and right eyes. *Exp Brain Res* 56: 206–216, 1984.

Tieman DG, Tumosa N, and Tieman SB. Behavioral and physiological effects of monocular deprivation: a comparison of rearing with diffusion and occlusion. *Brain Res* 280: 41–50, 1983.

Von Noorden GK and Crawford ML. Form deprivation without light deprivation produces the visual deprivation syndrome in *Macaca mulatta*. *Brain Res* 129: 37–44, 1977.

Wiesel TN and Hubel DH. Single-cell responses in striate cortex of kittens deprived of vision in one eye. *J Neurophysiol* 26: 1003–1017, 1963.

Corrigenda

Volume 89, April 2003

Pages 1748–1760: Schäfer SS, Berkelmann B, and Dadfar F. "Magnitude of Oscillations in the Response of Ia Muscle Spindle Endings Under a Static γ Stimulation of Increasing Frequency" (http://jn.physiology.org/cgi/content/full/89/4/1748; doi:10.1152/jn.00952.2001). During production, the DOI number was misrepresented in this article. The correct DOI number is presented here: doi: 10.1152/jn.00952.2001.

Volume 94, July 2005

Pages 119–135: Frechette ES, Sher A, Grivich MI, Petrusca D, Litke AM, and Chichilnisky EJ. "Fidelity of the Ensemble Code for Visual Motion in Primate Retina" (doi:10.1152/jn.01175.2004; http://jn.physiology.org/cgi/content/full/94/1/119). In the METHODS (in the first paragraph of *Stimuli*), the mean light intensity was misrepresented: "intensity 9,200 (8,700; 7,100)" should read "intensity 4,300 (4,200; 2,400)."

Volume 95, February 2006

Pages 862–881: Larsson J, Landy MS, and Heeger DJ, "Orientation-Selective Adaptation to First- and Second-Order Patterns in Human Visual Cortex" (doi:10.1152/jn.00668.2005; http://jn.physiology.org/cgi/content/full95/2/862). During production, the URL to direct readers to Supplemental Data was misrepresented. This has been corrected and replaced in the on-line version of the final published article (http://jn.physiology.org/cgi/content/full/00668.2005/DC1). Therefore the on-line version now deviates from the print journal with regard to this correction.

Volume 95, March 2006

Pages 1380–1396: Jiang W, Jiang H, and Stein BE. "Neonatal Cortical Ablation Disrupts Multisensory Development in Superior Colliculus" (doi:10.1152/jn.00880.2005; http://jn.physiology.org/cgi/content/full/95/3/1380). Important information was inadvertently omitted from the legends of Figs. 2 and 4. This information is presented here. Figure 2: lesion designations at *bottom right* ("LS Lesion" and "AES & LS Lesion") should read "rLS Lesion" and "AES & rLS lesion," respectively. All lesions are shown on the right hemisphere for illustrative purposes. Figure 4: all receptive fields are plotted on the right hemifield for illustrative purposes.

Volume 95, January 2006

Pages 379–400: Victor JD, Mechler F, Repucci MA, Purpura KP, and Sharpee T. "Responses of V1 Neurons to Two-Dimensional Hermite Functions" (doi:10.1152/jn.00498.2005;http://jn.physiology.org/cgi/content/full/95/1/379).As a result of a normalization error, the values of the filters *L* (but not *E*) plotted in Figs. 3, 5, and 6 are twice as large as they should be. Consequently, cells described as "under-rectified" (Figs. 3, *A* and *B*; 5, *A* and *B*; and 6*A*) should have been described as "half-wave rectifying," consistent with the illustrated poststimulus histograms. Correction of this error shifts values of the index I_{sym} (quoted in the text and plotted on the abscissa of Fig. 11) toward 1, i.e., the "complex" end of the simple versus complex spectrum. The correct value of the index and the published value are related by

$$I_{corrected} = (3 + 5I_{published})/(5 + 3I_{published})$$

Correction of this error also alters the values of $I_{c\text{-}p}$ by decreasing the weighting of contributions from the *L*-component, although these changes are slight (Fig. 9). The error does not affect the index I_{shape}, which quantifies the difference between the responses to Cartesian and polar stimuli and thus does not alter any of the conclusions of the paper. Corrected Figs. 9 and 11, with legends, are presented here.

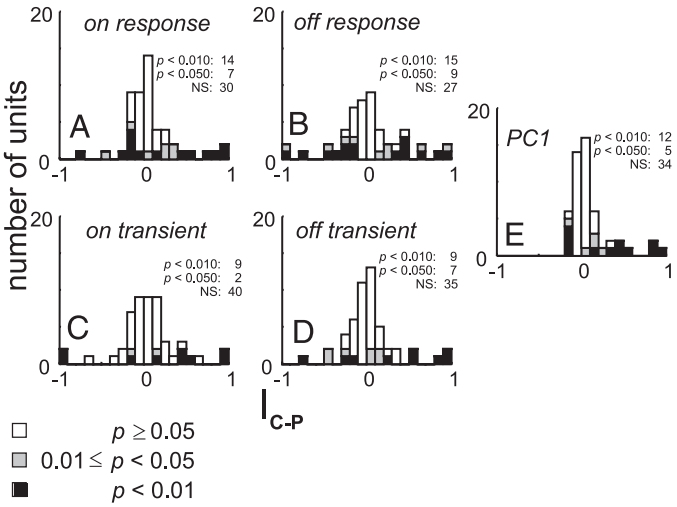

FIG. 9. Distribution of relative responsiveness to Cartesian and polar stimuli, $I_{c\text{-}p}$ (*Eq. 11*). Values >0 indicate larger responses to Cartesian stimuli; values <0 indicate larger responses to polar stimuli. Significance levels are calculated by jackknife and are shown as in Fig. 7. Each panel contains calculations based on a different response measure.

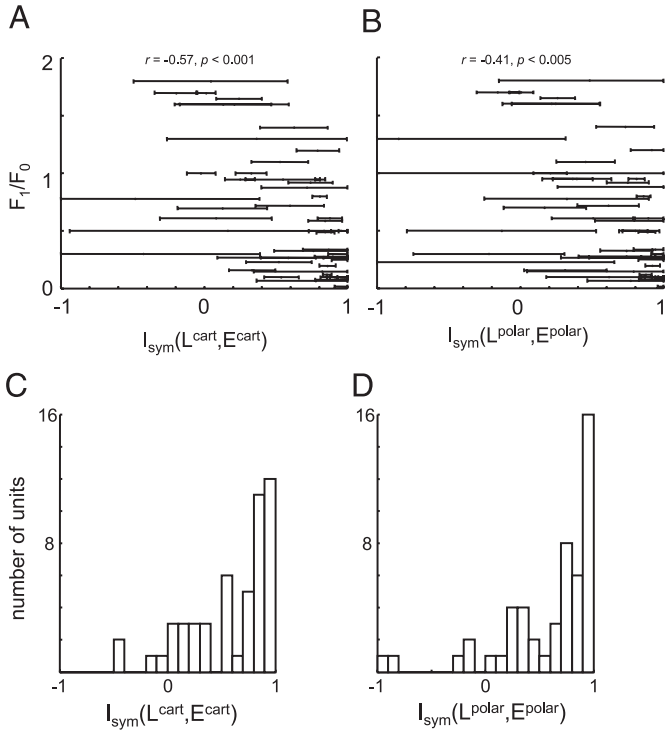

FIG. 11. Relationships of indexes of overall nonlinearity $I_{sym}(L, E)$ (*Eq. 5*) determined from Cartesian (*A*) and polar (*B*) responses to the F_1/F_0 ratio used to classify cells as simple and complex. For both Cartesian and polar measurements, units with I_{sym} close to 1 tended to have small values ("complex") of the F_1/F_0 ratio. *C* and *D*: distribution of these indexes across the population. Distributions for Cartesian and polar responses are similar.

Volume 95, March 2006

Pages 1571–1587: Vingerhoets RAA, Medendorp WP, and Van Gisbergen JAM. "Time Course and Magnitude of Illusory Translation Perception During Off-Vertical Axis Rotation" (doi: 10.1152/jn.00613.2005; http://jn.physiology.org/cgi/content/full/95/3/1571). During production, *Eq. 1* was misrepresented. This equation as been corrected and replaced in the online version of the final published article. Therefore the online version now deviates from the print journal with regard to this correction.

Pages 1843–1852: Neusch C, Papadopoulos N, Müller M, Maletzki I, Winter SM, Hirrlinger J, Handschuh M, Bähr M, Richter DW, Kirchhoff F, and Hülsmann S. "Lack of the Kir4.1 Channel Subunit Abolishes K^+ Buffering Properties of Astrocytes in the Ventral Respiratory Group: Impact on Extracellular K^+ Regulation" (doi:10.1152/jn.00996.2005; http://jn.physiology.org/cgi/content/full/95/3/1843). C. Neusch and N. Papadopoulos contributed equally to this article. A statement affirming this fact appeared in the first-published version at Articles in PresS (http://jn.physiology.org/cgi/reprint/00996.2005v1); however, the statement was inadvertently left out of the final-published version.

Pages 1966–1975: Stark E, Drori R, and Abeles M. "Partial Cross-Correlation Analysis Resolves Ambiguity in the Encoding of Multiple Movement Features" (doi:10.1152/jn.00981.2005; http://jn.physiology.org/cgi/content/full/95/3/1966). During production, *Eqs. 6*, *8*, and *15–17* were incorrectly cited within the text. These in-text citations have been corrected and replaced in the online version of the final published article. Therefore the online version now deviates from the print journal with regard to these corrections.

Volume 95, April 2006

Pages 2314–2325: Wicher D, Agricola H-J, Söhler S, Gundel M, Heinemann SH, Wollweber L, Stengl M, and Derst C. "Differential Receptor Activation by Cockroach Adipokinetic Hormones Produces Differential Effects on Ion Currents, Neuronal Activity, and Locomotion" (doi:10.1152/jn.01007.2005; http://jn.physiology.org/cgi/content/full/95/4/2314). During production, the URL to direct readers to Supplemental Data was misrepresented. This has been corrected and replaced in the on-line version of the final published article (http://jn.physiology.org/cgi/content/full/01007.2005/DC1). Therefore the on-line version now deviates from the print journal with regard to this correction.

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Pages 2947–2950: Rittenhouse CD, Siegler BA, Voelker CC, Shouval HZ, Paradiso MA, and Bear MF. "Stimulus for Rapid Ocular Dominance Plasticity in Visual Cortex" (doi:10.1152/jn.01328.2005; http://jn.physiology.org/cgi/content/full/95/5/2947). In the author line, the middle initial of Courtney Voelker was misrepresented. Corrected name is presented here: Courtney C. Voelker.

Pages 3001–3011: Isokawa M and Alger BE. "Ryanodine Receptor Regulates Endogenous Cannabinoid Mobilization in the Hippocampus" (doi:10.1152/jn.00975.2005; http://jn.physiology.org/cgi/content/full/95/5/3001). Twice in the article (in the third paragraph of the INTRODUCTION and in the second paragraph of the DISCUSSION), the citation for "Straiker and MacKie 2005" should have been presented as "Straiker and Mackie 2005," as listed in the REFERENCES.

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Pages 3948–3954: Ziburkus J, Cressman JR, Barreto E, and Schiff SJ. "Interneuron and Pyramidal Cell Interplay During In Vitro Seizure-Like Events " (doi:10.1152/jn.01378.2005; http://jn.physiology.org/cgi/content/full/95/6/3948). During production, the URL to direct readers to Supplemental Data was misrepresented. This has been corrected and replaced in the on-line version of the final published article (http://jn.physiology.org/cgi/content/full/01378.2005/DC1). Therefore the on-line version now deviates from the print journal with regard to this correction.