ON AND OFF CHANNELS IN PRIMATE VISION

by

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B.S., Biology, Cornell University 1984

Submitted to the Department of
Brain and Cognitive Sciences
in Partial Fulfillment of the
Requirements for the
Degree of

Doctor of Philosophy

at the

Massachusetts Institute of Technology

September 1992

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July 8, 1992

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Submitted to the Department of Brain and Cognitive Sciences
on July 8, 1992 in partial fulfillment of the requirements
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Abstract

Three investigations into the roles of the ON and OFF channels in the processing of luminance information in the primate visual system have been undertaken. The abstracts from each of these studies follows:

The mammalian rod bipolar, for which only one class has been identified, has been described as being hyperpolarizing by some investigators and depolarizing by others. We now report the effects of 2-amino-4-phosphonobutyrate (APB), a potent blocker of depolarizing bipolar cells, on visual behavior in the dark adapted monkey. While in mesopic and photopic conditions only the monkeys' ability to detect incremental stimuli is impaired, under scotopic conditions all light mediated response in the monkey is eliminated. Assuming APB is acting on rod bipolars in the same fashion as it does on cone bipolars, we conclude that the primate rod bipolars all depolarize to light and that the ON and OFF channels are formed by the amacrine cell network.

ON channel blockade with the glutamate analog 2-amino-4-phosphonobutyrate (APB) under photopic conditions has been shown to severely compromise monkeys' ability to detect stimuli brighter than background while only slightly affecting their ability to detect stimuli darker than background (Schiller et al. 1986a; Dolan & Schiller 1989). This is a direct confirmation of Jung's 1961 proposal (Jung 1973) that the "B" and "D" channels correspond to the ON and OFF channels as first described by Kuffler (1953). We further explore the perceptual consequences of selectively blocking the ON channel in alert monkeys. We find that ON channel blockade reduces the brightness of incremental stimuli, but this reduction is not enough to account for the deficit in detection. Simultaneous contrast is preserved following ON channel blockade, thus making it unlikely that perceptual lateral processes are comprised of opposing ON and OFF subunits. The detectability of stimuli that appear by virtue of changes only in background luminance is determined both by the polarity of the

temporal contrast change as well as by the spatial polarity of the stimulus contrast, indicating that a higher order mechanism is involved in detection of luminance stimuli. Finally, we test the hypothesis that temporal ramp changes are differentially processed by the ON and OFF channels to a higher degree than are step changes. We find, however, that the detection of incremental ramps is no more affected than the detection of incremental steps following APB administration.

Adaptation to stimuli modulated in luminance with a sawtooth temporal profile has been reported to induce two effects: it produces a dynamic luminance aftereffect in the direction away from the adaptation ramp change and it selectively raises detection threshold for contrast steps of the same polarity as the adaptation step change. Both mechanisms have been explained by a non-linearity in adaptation of incremental and decremental channels; however, this non-linearity works inversely in the two cases. In this study these two phenomena are examined using spatially distinct stimuli and test subjects across a range of adaptation and test stimulus temporal profiles. The results indicate that the detection of incremental and decremental step changes is equally affected by sawtooth adaptation, but that the detection of ramp changes of the same polarity as the adaptation ramps is selectively impaired. In addition it was found that the dynamic luminance aftereffect appears to sum with Troxler-like fading. Both phenomena show a peak effect with the same adaptation profile; this peak is defined by rate of contrast change in the ramp portion of the adaptation and appears to be less dependent on its temporal frequency. I conclude that sawtooth luminance modulation selectively adapts luminance channels of varying temporal properties, and that these correspond to the ON and OFF channels of early vision.

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Acknowledgements

To Peter Schiller for providing me with enough support to make it to the end. To my rest of my thesis committee: Richard Andersen, Patrick Cavanagh, Ellen Hildreth, and Mriganka Sur for their help, time and energy.

To Jan Ellertsen - no other person here has been more important to me and has taken better care of me. As far as I'm concerned you are the department's greatest asset.

To the German psychophysicists whose work I am undoubtably failing to cite.

To Bob Marini, a true healer, who walks the inherent fine lines of his position with strength, humanity, and personality. To Carson, Clinton and all the animal caretakers, and in fond memory of Johnson, who have all earned my fullest respect. They've handled the enormous responsibility of creating comfort and dignity for the monkeys, with enthusiasm and insight. To Sylvester, the craftsman and artist, always available to get the job done yesterday, and with only a few grumbles. To Steve, who has helped me with enumerable electronic projects.

To Bonnie, Mary, Julie, Julia, and Tracy, for their technical and administrative assistance. And most of all to Karen, whose friendship and warmth has helped the lab become more personal, relaxed, and fun. To Warren, whose ability to make order out of chaos is critical to the lab's continued health and success. To Ed, the traveller, who knows that people are only as interesting as our imagination allows them to be. To Karl, whose fondness for Snickers bars is the only thing that kept his eyes in his head during long hours of staring at my monitor. To all of the Schiller group over the years who were able to work with openness and respect for others. To my office mates Cecilia and Diana who have put up with continuous attacks at the frontiers of personal space. To everyone on the sixth floor who made my walk from office to lab to office not only diverting but entertaining and supportive.

To Checkers, Marcel, Quark and PeeWee - I hope I see you all on the other side and that you're not too upset with me. Lets find some trees to climb in and live the way we're all really supposed to live.

To my special friends here over the years - Sabina, Todd, Corrie, Laurie, Young, Henry, Stefan, Hemai, Tony, Lynne, Gail, Eric, Lyle, Jonathan, Charo, Martin, Laurie, Manny, Alain, Leo, Reha, Terry, and Joachim - you are all gems. To my family, who've watched on with love, support, guidance, and patience. To Lori Lynn, who has been giving me continuous love and hope - I cherish my thoughts of our future together.

And to everyone who decided for themselves to see who I am.

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Introduction

The work presented in this thesis represents three levels of analysis of the parallel processing of luminance information in the primate visual system: pharmacological, physiological, and perceptual. The channels involved in this parallel processing have been described psychophysically as the light increment and decrement channels and physiologically as the ON and OFF channels. The purpose of my thesis is to understand how the signals from these two parallel channels is combined at all three levels. The basic technique I use is to selectively impair activity in one of these two channels, either through pharmacological or visual manipulation, and to assess changes psychophysically.

Chapter 1 is an investigation into the nature of the rod bipolar cell response in the monkey. I examine the effect of the glutamate analog 2-amino-4-phosphonobutyrate (APB) on scotopic vision to infer whether there is only one type of rod bipolar cell and whether it depolarizes or hyperpolarizes to light. The retinal circuitry involved in the creation of the ON and OFF channels from this single type of bipolar cell is discussed.

In chapter 2 I examine the perceptual consequences of ON channel blockade using APB in the monkey. It is already known that ON channel activity is necessary for the detection of stimuli brighter than background. Here I further explore the role of the ON channel in

brightness and contrast perception and relate it to the psychophysically defined luminance channels.

In chapter 3 I report on a method of differentially adapting the luminance channels via visual stimulation. Adaptation to a stimulus modulated in luminance with a sawtooth temporal profile selectively disrupts detection of light stimuli, and induces a percept of gradually changing brightness. The nature of the adaptation as it relates to the luminance channels, as well as the temporal properties of this phenomenon, are explored.

Chapter 1 has been published in Visual Neuroscience (1989), volume 2, pages 421 - 424. Chapter 2 is to be submitted to Visual Neuroscience in July 1992; portions of it have already been presented at Society for Neuroscience and ARVO meetings. Chapter 3 is to be submitted to Vision Research in July 1992.

Chapter 1

Evidence for Only Depolarizing Rod Bipolar Cells in the Primate Retina

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Evidence for Only Depolarizing Rod Bipolar Cells in the Primate Retina

Abstract

The mammalian rod bipolar, for which only one class has been identified, has been described as being hyperpolarizing by some investigators and depolarizing by others. We now report the effects of 2-amino-4-phosphonobutyrate (APB), a potent blocker of depolarizing bipolar cells, on visual behavior in the dark adapted monkey. While in mesopic and photopic conditions only the monkeys' ability to detect incremental stimuli is impaired, under scotopic conditions all light mediated response in the monkey is eliminated. Assuming APB is acting on rod bipolars in the same fashion as it does on cone bipolars, we conclude that the primate rod bipolars all depolarize to light and that the ON and OFF channels are formed by the amacrine cell network.

Introduction

A general attribute of the mammalian visual system is the segregation of visual information into the ON and OFF channels at an early processing stage (Kuffler, 1953; Schiller, 1986). The mechanisms that accomplish this differ for the cone and rod systems. In the cone system, two general classes of cone bipolars can be distinguished from the variety of existing types, namely those that depolarize (ON) and those that hyperpolarize (OFF) to light (Famiglietti and Kolb, 1976). The morphology of the two classes is distinct: ON cone bipolars form invaginating synapses with photoreceptor synaptic ribbons and connect with retinal ganglion cells in sublamina *b* of the inner plexiform layer while OFF cone bipolars form flat synapses with the photoreceptors and terminate to connect with ganglion cells in sublamina *a* of the IPL (Famiglietti and Kolb, 1976; Nelson et al., 1978).

In the rod system, by contrast, only one morphological class of bipolar cells has been identified (Boycott and Kolb, 1973; Kolb, 1979; Kolb and Nelson, 1983). These rod bipolars do not make direct connections with the retinal ganglion cells; instead, amacrine cells, particularly the AII amacrine, are interposed (figure 3) (Kolb and Famiglietti, 1974). The AII amacrines in turn make multiple connections, including electrical gap junctions with ON cone bipolars and glycinergic synapses with OFF cone bipolars (Famiglietti and Kolb, 1975; Kolb, 1979; Pourcho, 1980; Pourcho and Goebel, 1985). In this way the rod bipolar can produce action potentials to both light increment and light decrement in the ganglion cells.

Intracellular recordings from the retina in the inverted eye-cup preparation have supported the anatomical evidence for the existence

of a single class of rod bipolar cells. The nature of its light response, however, remained unclear. Nelson et al. (1976) recorded from rod bipolar cells in the cat and found them all to be hyperpolarizing, while Dacheux and Raviola (1986), recording from the rabbit, found them all to be depolarizing. There are three possible explanations for these conflicting findings. The first is that the rod bipolar has different responses in these two animals. In view of the seemingly consistent anatomy of the outer retina across all vertebrate species (Famiglietti, 1981), this would be surprising. The second explanation is that interspecies differences could accentuate electrode sampling biases (Dacheux and Raviola, 1986). The third explanation is that due to the enormous difficulty inherent in intracellular recording from mammalian retina the cells were either damaged or misidentified.

Müller et al. (1988) have recently shown that intravitreal application of 2-amino-4-phosphonobutyrate (APB), a potent agonist of ON bipolars, blocks all light mediated response of retinal ganglion cells in the dark adapted cat. Furthermore, application of strychnine blocks activity in OFF ganglion cells. Shiells et al. (1981) have reported that APB blocks the response of ON rod bipolars in the dogfish. Together, these findings support the notion that the rod bipolar through the AII amacrine cell as an intermediary makes inhibitory, glycinergic synapses with the OFF pathway and excitatory connections with the ON pathway. This assumes rod bipolar cells to have receptor pharmacology analogous to cone bipolar cells.

We have previously reported that under photopic conditions, blockade of the ON channel with APB drastically impairs the monkeys' ability to detect incremental light stimuli while leaving the ability to

detect decremental stimuli relatively unimpaired (Schiller, 1982; Schiller et al., 1986). The present study examines how light mediated behavior in the dark adapted monkey is affected by APB.

Materials and Methods

Three adult rhesus macaque monkeys were used in this study. Monkeys were trained to make saccades to stimuli appearing on a CRT. Eye movements were recorded using a scleral search coil; a PDP 11/74 governed the presentation of stimuli, monitoring of eye movements, and storage of data. A trial began with the appearance of a central fixation spot presented against an achromatic background. Following foveation of the fixation spot for 100-150 msec, a target stimulus appeared in one of four randomly chosen positions. Monkeys were rewarded with apple juice if they made a single, direct saccade to the target within 650 msec. To minimize guessing, catch trials were used in which no target appeared and the monkey was rewarded for maintaining fixation.

The fixation spot consisted of a 0.5° black square within a 1.0° white square. The target stimulus was a 3° square at one of the following incremental (brighter than background) or decremental (darker than background) contrasts: -100, -50, -20, +14, +25, +33, and +74%. Since the dynamic range of a CRT is limited, overall luminance of the display was controlled with Kodak neutral density filters in one log incremental steps; in this way the background luminance was varied from 1.2×10^{1} to 1.2×10^{-3} cd/m^2. The neutral density filters were placed in a specially designed holder fitted in front of the monkey's face that prevented ambient light from reaching the monkey's eyes. One eye of the monkey was occluded at random under computer control with a pneumatic shutter mounted on the frame of the monkey chair. A monkey was considered to be adapted to a particular illumination level after spending at least 15 minutes at that illumination; since dark

adaptation was done in one log unit steps, after this amount of time at a given illumination level no further change in performance was observed. Performance was assessed for each eye separately by determining the percentage of correct trials and the reaction time latencies for the correct trials.

Prior to the APB applications monkeys were anaesthetized with a mixture of 4% halothane, 67% nitrous oxide, and 29% oxygen. Sterile intravitreal injections of 0.05 to 0.10 ml of either R/L-APB (20 mM) or L-APB (5-10 mM) were made to yield vitreal concentrations of 750 μM or 250-375 μM, respectively. Testing of monkeys began within one hour of anaesthesia; the effects of APB lasted at least four hours, with complete recovery from the drug's affects by the next day. A total of five injections were made in the three monkeys.

Results

Results for all three monkeys were similar. Reaction time and percent correct for one monkey are shown in figure 1 as a function of stimulus contrast and mean luminance of the display. As is shown, the monkeys' performance decreased with both stimulus contrast reduction and with mean luminance reduction.

As we have previously reported, following APB administration monkeys showed a dramatic reduction in their ability to detect incremental stimuli under photopic illumination, as measured by a 60-142% increase in response latency and a 9-55% reduction in correct trials for the stimuli we used. There was only a slight effect on the detection of decremental stimuli (17-30% increase in reaction time, 1-4% decrease in percent correct).

As mean luminance was lowered towards scotopic conditions, monkeys showed an increasing deficit in their ability to detect both incremental and decremental stimuli. At background luminance of 1.2×10^{-2} cd/m^2 [log 3.0], the monkey's performance was equally impaired for incremental and decremental stimuli. At background luminance of 1.2×10^{-3} cd/m^2 [log 4.0], the monkey had difficulty in detecting even the fixation spot; the monkey probably guessed the position of the target for the reaction times plotted.

Figure 2 summarizes the results obtained from the two other monkeys; these results, which are similar to those of the first monkey (figure 1), show the following: (1) Percent correct performance and response latencies for stimuli presented to the intact eye are comparable for both incremental and decremental stimuli in both the light and the dark adapted states. Normal reaction times are greater in the dark

adapted state. (2) In the light adapted state APB decreases percent correct performance and increases latencies for incremental stimuli; only minor changes in performance can be seen for decremental stimuli. (3) In the dark adapted state percent correct performance is severely impaired for both incremental and decremental stimuli. Mean response latencies for detected stimuli almost doubled for both incremental and decremental stimuli.

Discussion

Our findings show that all light mediated behavior is abolished in the dark adapted monkey following administration of APB. This is noted in reference to our previous findings that in light adapted conditions only the detection of incremental stimuli is compromised (Schiller et al., 1986). The conclusion we draw from these findings is that there is only one physiological class of rod bipolar cells in the monkey that is depolarizing in nature, which when blocked by APB eliminates scotopic vision.

The action of APB on cone bipolars is well documented: a glutamate agonist selective for depolarizing (ON) bipolars, APB maintains these cells at hyperpolarization, rendering them silent (Slaughter and Miller, 1981; Slaughter and Miller, 1985; Arkin and Miller, 1987). Although APB has been recently reported to have some effect on hyperpolarizing (OFF) bipolars, the actions are subtle (Arkin and Miller, 1987). The existence of a hyperpolarizing rod bipolar that is affected by APB cannot be ruled out at this point. However, given that i) rod bipolars make invaginating synapses with the rod synaptic ribbon as do depolarizing cone bipolars (Boycott and Kolb, 1973), ii) that the ERG of a dark adapted retina shows a prominent b-wave that in the light adapted state represents the ON pathway (Evers and Gouras, 1986), and iii) AII amacrines show a rapid depolarization to light (Nelson, 1982), it is certainly simpler to model the rod bipolar as being depolarizing. Still disturbing is the finding of a hyperpolarizing response in rod bipolars in the cat (Nelson et al., 1976).

The simplest circuit that fits with these and related findings (Dacheux and Raviola, 1986; Müller et al., 1988) is one proposed by Kolb

and Famiglietti (1974) and Müller et al. (1988) for the cat in which the rod pathway relies on the AII amacrine to create a double-ended response from the single class of rod bipolar cells. This could be accomplished via the AII cell's inhibitory glycinergic connections (Pourcho, 1980; Pourcho and Goebel, 1985; Pourcho and Goebel, 1987) with OFF cone bipolars and OFF ganglion cells and excitatory electrical connections with the ON cone bipolars (figure 3). Although ambiguities remain, this is clearly the most straightforward model for the monkey that can be proposed based on current evidence.

Acknowledgements

Supported by NIH EY00676 and Training Grant 2T32-GM07484.

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Figure Legends

Figure 1. Plot of monkey's percent correct performance and reaction time latencies as a function of stimulus contrast and overall display intensity. Each row represents data for a single illumination level; the background illumination for the top row is $1.2x10^1$ cd/m^2 and each subsequent row is one log unit dimmer than the one above. Vertical lines bisecting each column show background illumination level. Normal data are plotted with solid lines, APB data with dashed lines. At photopic illumination (top row), detection of only incremental stimuli is greatly impaired. At lower (mesopic-scotopic) illumination levels, monkey's performance for decremental stimuli becomes impaired as well. At the lowest illumination level (bottom row), virtually all light adapted response is eliminated from the APB injected eye. APB data is summed from two injections, one in each eye. Standard error of the mean is shown for reaction times. Total number of trials: normal: 4,800; APB: 1,159.

Figure 2. Summary graph of another monkey's performance for one APB injection. APB affects only the detection of incremental stimuli in the light adapted state, but affects both incremental and decremental stimuli detection during dark adaptation.

Figure 3. Proposed pathway for scotopic vision in the mammalian retina; diagram is highly schematic with many connections not represented. Depolarizing (ON) cone bipolars make invaginating connections with cones and connect directly with ON ganglion cells in IPL(a); hyperpolarizing (OFF) cone bipolars make flat connections

with cones and connect directly with OFF ganglion cells in IPL(b). Rod bipolars, which make invaginating connections with rods, do not connect directly with ganglion cells and instead connect with AII amacrine cells. AII amacrines form electrical gap junctions with the ON cone bipolars and glycinergic synapses with the OFF cone bipolars and ganglion cells. OPL, outer plexiform layer; IPL, inner plexiform layer.

Figure 1

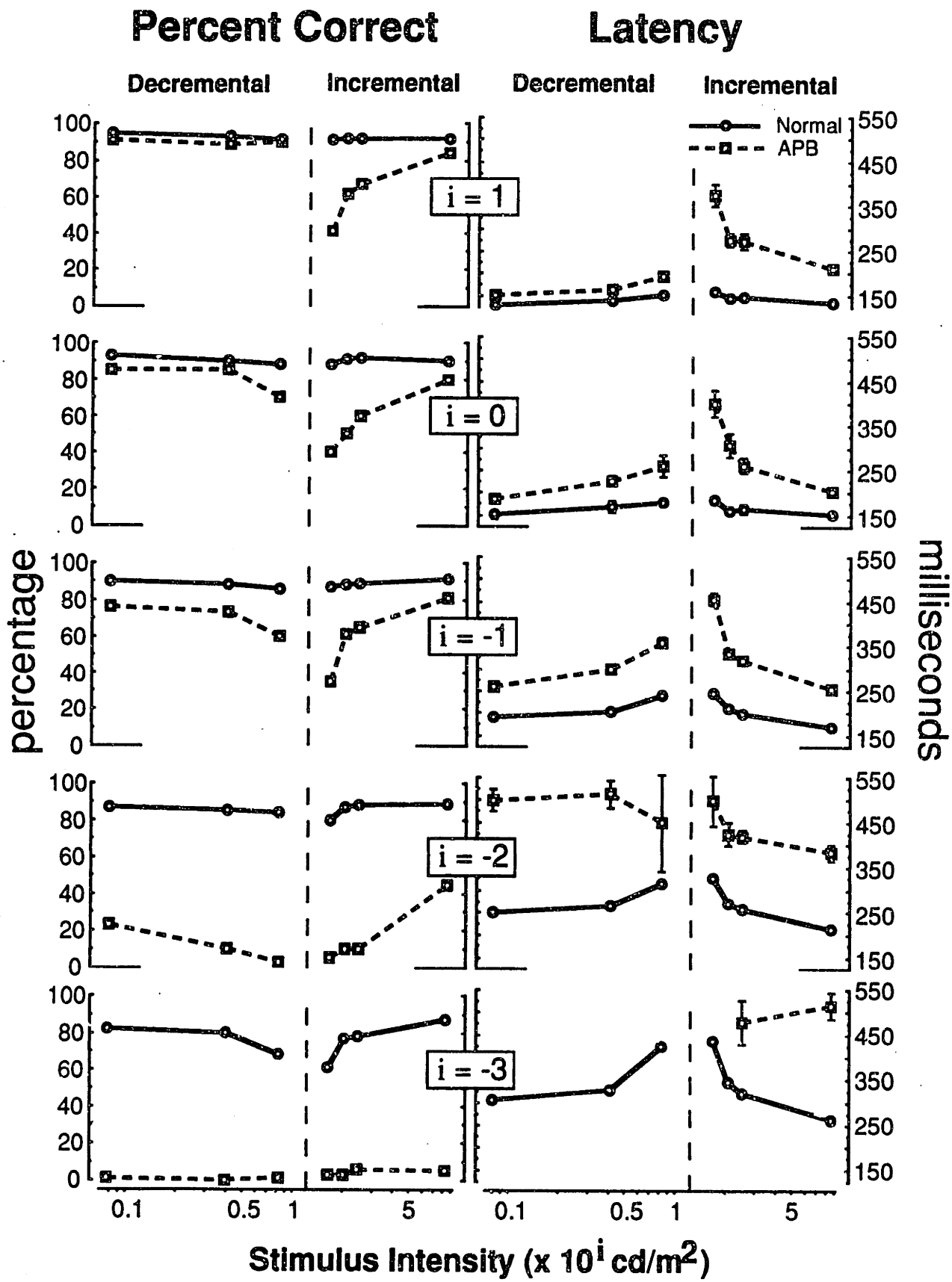

Percent Correct
Latency
Decremental
Incremental
Decremental
Incremental
Normal
APB
i = 1
i = 0
i = -1
i = -2
i = -3
percentage
milliseconds
100
80
60
40
20
0
550
450
350
250
150
0.1
0.5
1
5
Stimulus Intensity (x 10^i cd/m^2)

Figure 2

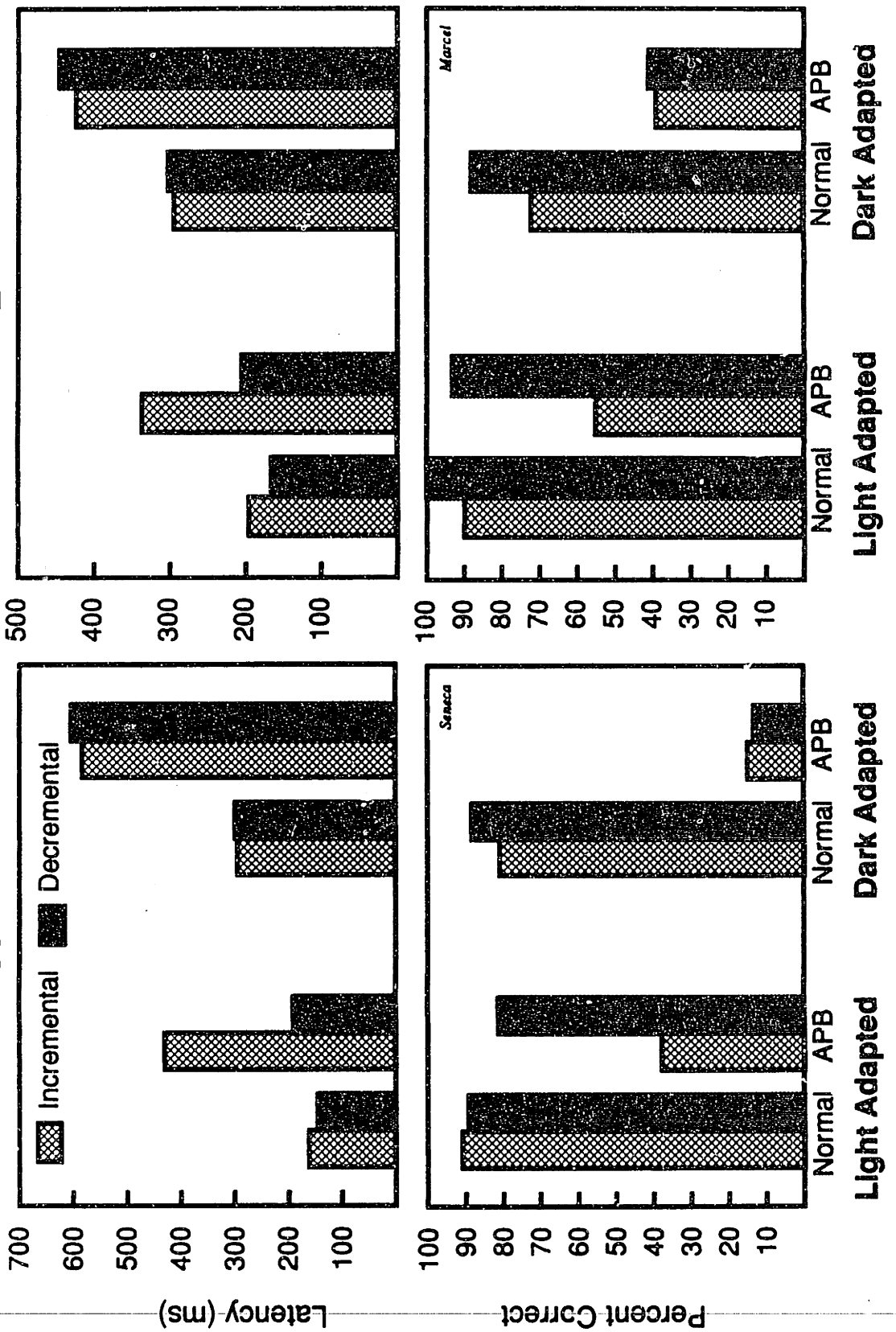
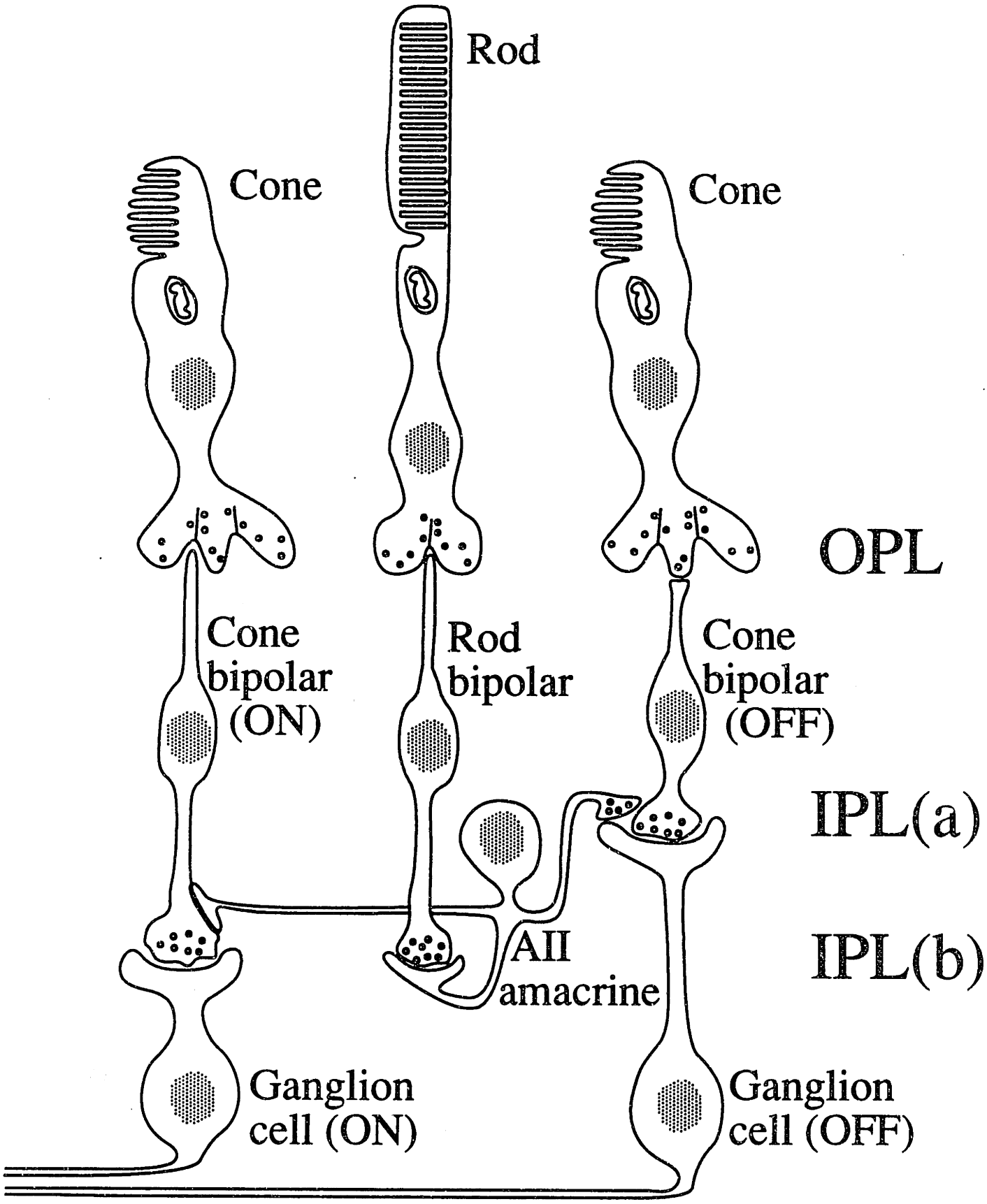

A
B
Incremental
Decremental
Latency (ms)
Percent Correct
Seneca
Marcel
Normal
APB
Light Adapted
Dark Adapted

Figure 3


Rod
Cone
Cone
OPL
Cone bipolar (ON)
Rod bipolar
Cone bipolar (OFF)
IPL(a)
AII amacrine
IPL(b)
Ganglion cell (ON)
Ganglion cell (OFF)

Chapter 2

Effects of ON Channel Blockade with 2-amino-4-phosphonobutyrate (APB)on Brightness and Contrast Perception in Monkeys

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Effects of ON Channel Blockade with 2-amino-4-phosphonobutyrate (APB) on Brightness and Contrast Perception in Monkeys

Abstract

ON channel blockade with the glutamate analog 2-amino-4-phosphonobutyrate (APB) under photopic conditions has been shown to severely compromise monkeys' ability to detect stimuli brighter than background while only slightly affecting their ability to detect stimuli darker than background (Schiller et al. 1986a; Dolan & Schiller 1989). This is a direct confirmation of Jung's 1961 proposal (Jung 1973) that the "B" and "D" channels correspond to the ON and OFF channels as first described by Kuffler (1953). We further explore the perceptual consequences of selectively blocking the ON channel in alert monkeys. We find that ON channel blockade reduces the brightness of incremental stimuli, but this reduction is not enough to account for the deficit in detection. Simultaneous contrast is preserved following ON channel blockade, thus making it unlikely that perceptual lateral processes are comprised of opposing ON and OFF subunits. The detectability of stimuli that appear by virtue of changes only in background luminance is determined both by the polarity of the temporal contrast change as well as by the spatial polarity of the stimulus contrast, indicating that a higher order mechanism is involved in detection of luminance stimuli. Finally, we test the hypothesis that temporal ramp changes are differentially processed by the ON and OFF channels to a higher degree than are step changes. We find, however, that the detection of incremental ramps is no more affected than the detection of incremental steps following APB administration.

Introduction

Slaughter and Miller (1981) first demonstrated that the glutamate agonist 2-amino-4-phosphonobutyrate (APB) blocks the light response of ON bipolars, while having only minor effects on photoreceptor, horizontal cell, or OFF-bipolar responses in the mudpuppy retina. Additional researchers have confirmed the selective effects of APB on ON channel activity in the rabbit retina (Massey et al. 1983; Bloomfield & Dowling 1985a; Bloomfield & Dowling 1985b; Knapp & Schiller 1984) and LGN (Knapp & Mistler 1983), cat retina (Shiells et al. 1981; Bolz et al 1984), lateral geniculate nucleus (LGN) (Horton & Sherk 1984), and cortex (Sherk & Horton 1984), and the monkey retina (Knapp & Schiller 1984), LGN (Schiller 1982; Schiller 1984a) and cortex (Schiller 1982). Although most researchers have reported subtle effects of APB on OFF bipolar cell response (Arkin & Miller 1987 for example), these effects appear minor in comparison to the effect on the ON bipolars. Additionally, although there may be cross-channel interactions between the ON and OFF channels in the retina in agreement with a "push-pull" model (Levine & Shefner 1977; McGuire et al. 1984), the predominant signals feeding into ON and OFF ganglion cells are presumed to be from the ON and OFF bipolars, respectively.

Schiller et al. (1986a) first reported the perceptual consequences of intravitreal administration of APB in the alert monkey. They found that ON channel blockade with APB severely impaired the detection of stimuli lighter than background (incremental) while leaving the detection of stimuli darker than background (decremental) largely unaffected. This impairment is manifested as an increase in both

detection threshold and reaction time latency. This demonstration not only confirmed the independent nature of processing of light increments and decrements but confirmed earlier theories (Jung 1961, in Jung 1973) that the physiological ON and OFF channels as first described by Kuffler (1953) are the corresponding neural substrate to the light increment ("B" or brightening) and light decrement ("D" or darkening) channels. The reputed roles of these two luminance processing channels, however, extend beyond detection. In the current study we further examine the perceptual consequences of ON channel blockade on processing of luminance information in the monkey.

Although it is clear that the ON channel mediates the detection of incremental stimuli (and the OFF channel presumably for decremental stimuli), the roles of the ON and OFF channels in the judgement of brightness is not understood. Psychophysical evidence supports the notion of separate channels for assessing brightness and darkness (Broca & Sulzer 1902; Anstis 1967; Hanly & MacKay 1979; Krauskopf 1980; Cavanagh & Anstis 1986; Magnussen & Glad 1975), but it is unknown if the perception of brightness and darkness is carried 1) in a dedicated fashion by the ON and OFF channels, respectively, 2) by both channels as a comparison in activity, or 3) by both channels individually and nonselectively. In cases 1 and 2, ON channel blockade should cause stimuli to look less bright. Such a decrease in perceived brightness might explain the impairment in detection of incremental stimuli produced by APB. Alternatively, if the ON and OFF channels are able to process brightness independently, ON channel blockade might not produce any brightness shift. In this case, one could argue that the brightness of a stimulus is not a factor in its detectability.

Single cell recording from OFF center geniculate and striate cells following ON channel blockade with APB have demonstrated that receptive field surrounds remain intact (Schiller 1984a; Horton & Sherk 1984; Sherk & Horton 1984). Thus it appears that both center and surround are created predominantly by either the ON or OFF channel and not by opposing both channels. As phenomena such as simultaneous contrast, Mach bands, the Craik-O'Brian-Cornsweet illusion, and the Chevreul illusion demonstrate, both absolute luminance and edge contrast are critical factors in the perception of brightness. A parallel can be drawn between the lateral interactions involved in the role of contrast in brightness perception and the lateral inhibition responsible for creation of the receptive field. Jung (1973) in fact proposes that the "B" and "D" channels combine to produce this lateral inhibition. If lateral inhibition in perception requires both the ON and OFF channels, blocking the ON channel should result in a breakdown of simultaneous contrast. Brightness assessments would then be made more by virtue of direct luminance comparisons and less by comparison of local contrasts of the stimuli.

The onset of an incremental stimulus causes a modulation in activity for both the ON and OFF channels. The fact that incremental stimuli are not detected following ON channel blockade implies that the OFF channel information is not as effective to some higher level mechanism responsible for detecting the stimulus. If a decremental stimulus is presented by increasing the luminance of the background only, the same edge contrast events occur as when an incremental stimulus appears by virtue of its luminance increasing. If object detection is mediated solely by the appearance of edges, then increments

in either the foreground or background should be equally processed by the ON channel and equally compromised by APB, and neither decrements in foreground nor background should be effected. If, on the other hand, the spatial contrast of the stimulus is important in determining which channel is responsible for carrying it, APB may produce deficits for detecting incremental stimuli that appear via decreasing background luminance.

Adaptation to a stimulus modulated in luminance with a sawtooth temporal profile produces a dynamic brightness aftereffect; this aftereffect appears as gradual brightness shift in a direction opposite to the direction of the ramp phase of the sawtooth (Anstis 1967). Sawtooth adaptation also differentially affects the detection of subsequently viewed step-changes (Krauskopf 1980) and sawtooths (Hanly & MacKay 1979). In these two cases, if the polarity of the test stimuli matches that of the corresponding phase of the adaptation stimulus, detection will be selectively impaired. Presumably the step and ramp phases of the sawtooth differentially adapt the luminance channels responsible for detection and the percept of brightness. In particular, Cavanagh and Anstis (1986) propose the existence of a temporal channel for processing the step phase of the sawtooth and a sustained channel for processing the ramp phase, and that the brightness percept results from a combination of these signals. They argue that there is greater saturation in the processing of the step phase of the adapting stimulus, and thus the percept is biased toward the percept carried by the ramp phase.

There is another way of creating a nonlinearity such that the sawtooth differentially adapts the ON and OFF channels without

presupposing multiple temporal channels. Step changes could be less selectively processed by a single channel than ramp changes. For example, the ramp phase of a ramp up/step down sawtooth might be detected to a greater extent by the ON channel than would the step phase be detected by the OFF channel; this would result in a greater adaptation of the ON channel. This would also explain a finding by Dolan (1992) that the detection of ramps is far more sensitive to sawtooth adaptation than is the detection of step changes; if ramps are processed more exclusively by one channel they would be more sensitive to differential adaptation of the channels.

Experiments 1 and 2 in the current study examine the question of whether ON channel blockade results in a shift in perceived brightness and/or contrast. In experiment 1, the apparent brightness of incremental and decremental stimuli following APB administration is assessed by having a monkey compare the brightness of a stimulus presented to the APB-injected eye to one presented to the non-injected eye. In experiment 2, the effect of APB on simultaneous contrast is assessed by having a monkey compare the apparent brightness of stimuli presented on backgrounds of different luminance, to determine if APB decreases simultaneous contrast. In experiment 3 we explore the effects of APB on the detection of stimuli that appear by virtue of changes in background luminance. Experiment 4 tests the hypothesis that temporal luminance ramps are detected more exclusively by a single class of channel than are luminance step changes.

Methods

Experimental control, stimulus generation, and data collection were done with a PDP 11/73 computer. Stimuli were presented on a color CRT, at a viewing distance of approximately 60 cm. High-luminance borders drawn on the CRT, as well as ambient lighting behind the CRT and on its frame, ensured photopic conditions. The basic paradigm for all experiments is as follows: monkeys were required to fixate a small centrally located fixation spot for a period of time, during which a single stimulus (detection task) or multiple stimuli (discrimination task) were presented. Following a cue, the monkey had to make a single direct saccade to the target stimulus. Monkeys were rewarded with a drop of apple juice for making a correct response within an allotted period of time. Performance was measured in terms of mean reaction time latencies for correct responses and percentage of correct responses. Stimuli were always achromatic and were either incremental (more luminant than background) or decremental (less luminant than background). Unless otherwise noted stimulus contrast was measured as $(lum_{stim} - lum_{bkg}) / lum_{bkg}$. Stimulus positions were varied randomly from trial to trial.

Three adult rhesus monkeys were used in these studies. The monkeys' heads were fixed and eye position was monitored with a scleral search coil. Monkeys were water deprived but were given free access to fluids following each day's testing.

Prior to APB administration, monkeys were anaesthetized with a mixture of 4% halothane, 67% nitrous oxide, and 29% oxygen. Either R/L-APB or L-APB were used for the intravitreal injections; we found the latter to be about twice as effective as the former, but no qualitative

differences in effect were seen. The APB was administered in sterile saline at concentrations ranging from 10-20 μM (R/L-APB) or 5-10 μM (L-APB) and in volumes of either 0.05 or 0.10 ml; this yielded intravitreal concentrations of approximately 500-750 μM (R/L-APB) or 250-375 μM (L-APB). Monkeys were tested within one hour of injection, and effects were present for at least four hours.

Contrast sensitivity data were collected periodically for all monkeys and confirmed that the injections had no long term effects. Performance levels on all tasks returned to baseline on the day following the injections. Saline injections were performed as a control for all monkeys; no effect was seen for these injections.

Assessment of APB effect

The efficacy of all injections was classified by a pair of indices representing the change in reaction time latency for detection of a light increment and decrement at a contrast of 0.20. Thus an efficacy index of 2.3/1.2 indicates that reaction time latency increased by 2.3 for incremental stimuli and by 1.2 for decremental stimuli. These stimuli were 1.0° - 1.5° circles and presented randomly in 1 of 4 positions at the same eccentricities as the experimental stimuli. Figure 1 shows representative data for reaction times and percent correct as a function of stimulus contrast under normal and APB conditions.

Experiment 1 - Apparent brightness shift

A monkey was trained to choose the brighter (or darker) of two incremental (or decremental) stimuli. Stimuli were 1° squares presented at 3° eccentricity in 2 of 4 possible positions along the diagonals. For incremental stimuli, the background luminance was 0.1 cd/m^2, the target stimulus was 48.0 cd/m^2, and the comparison stimulus varied from 0.6 to 38.0 cd/m^2. For decremental stimuli, the background luminance was 36.0 cd/m^2, the target stimulus was 6.0 cd/m^2, and the comparison stimulus varied from 7.8 to 33.0 cd/m^2.

The target and comparison stimuli were presented separately to each eye by drawing separate left and right images on the CRT and having the monkeys view the CRT through a pair of prisms. For the APB injections the target stimulus was always displayed to the injected eye. The fixation spot was presented to both eyes. One monkey was used in this study; injection data represents a total of 4 injections, 3 at one efficacy (2.1/1.2, 2.1/1.1, 2.3/1.3) and 1 at another particularly high efficacy (3.1/1.6).

Data are presented as percent correct performance. For the low efficacy APB and normal conditions these values are a mean of three sessions and are shown with standard deviations.

The left graph on figure 2 plots the results for incremental stimuli. Under normal conditions, the monkey performance reached chance when the luminance difference between the two stimuli was between approximately 10.0 and 10.5 cd/m^2. Following the lower efficacy APB administrations, the curve shifted rightward, indicating that the apparent brightness of the target decreased since the luminance of the "matching" comparison stimulus decreased. For the

higher efficacy injection, the decrease in apparent brightness of the target is dramatic; even stimuli of very low luminance presented to the non-injected eye appeared to be as bright as the stimulus presented to the APB-injected eye. Thus for incremental stimuli ON channel blockade does result in a decrease in brightness.

The effect of APB on the brightness of decremental stimuli is plotted on the right graph of figure 2. On this graph, as we move along the abscissa from left to right, the luminance of the non-target (comparison stimulus) increases away from that of the target and thus its contrast decreases. Following the lower efficacy APB administrations, the curve shifts slightly to the left, indicating that the monkey is matching darker (higher contrast) stimuli to the injected eye. Following the higher efficacy injection, even stimuli of very low contrast (i.e. high luminance) presented to the non-injected eye appeared as dark as the stimulus presented to the APB-injected eye. The significance of these findings is discussed below.

Experiment 2 - Simultaneous contrast

As in Experiment 1, the monkey was trained to choose the brighter (or darker) of two incremental (or decremental) stimuli. Stimuli were 1° squares presented at 5° eccentricity in 2 positions along the horizontal. The background was either homogeneous (40 cd/m^2) or ramped linearly in luminance (28 - 52 cd/m^2) along the horizontal. The contrast of the target stimulus was fixed at 0.4 with respect to its immediate background, while the contrast of the comparison stimulus varied from 0 to 0.4, with respect to its immediate background (see figure 3). Although the position of the target stimulus was random,

data for ramped background trials was analyzed only when the target stimulus luminance equalled the background luminance at the distractor stimulus position. One monkey was used in this study; injection data represent a total of 3 APB injections of similar efficacy (2.4/1.2, 2.6/1.4, 2.3/1.2).

Data are shown in the form of percent correct performance. These values are a mean of three sessions, and standard deviations are shown.

The top graphs in figure 4 show the monkey's performance plotted as a function of the difference in contrast between the two stimuli. The monkey's performance became random as the contrast difference between the two stimuli decreased, regardless of whether the background was homogeneous or graded. This is the case with both incremental and decremental stimuli. Thus it is clear that the apparent brightness of the stimuli is based on their local contrasts, rather than their absolute luminances, demonstrating simultaneous contrast.

The bottom graphs in figure 4 show the monkey's performance with a graded background under normal and APB administered conditions. Although APB causes a drop in performance for both incremental and decremental stimuli, the monkey's choice of brighter or darker stimuli is still based on the stimuli's contrast with their immediate background. If the choice was based on a direct luminance comparison of the stimuli, then the curves would be displaced rightward. ON channel blockade therefore does not eliminate simultaneous contrast.

Experiment 3 - Target versus background changes

Monkeys were trained to detect the appearance of a target stimulus presented at 1 of 4 positions. Stimuli consisted of 1° circles (monkey Q) or gaussians ($contrast_{1°} = 0.10 * contrast_{center}$) (monkey P) presented at 2° eccentricity along the horizontal and vertical. Background luminance was 50 cd/m^2. Stimuli were defined by a step change in either their luminance or the background luminance. The luminance steps were either incremental or decremental and were of the following contrast series: 0.2, 0.26, 0.34, 0.4 (monkey Q) or 0.14, 0.16, 0.34, 0.50 (monkey P). Contrasts were measured as the temporal change in luminance divided by the initial (background) luminance. Two monkeys were used in this study; injection data represents 2 APB injections in each monkey, all of similar efficacy (monkey Q: 2.0/1.1, 2.2/1.2; monkey P: 2.3/1.2, 2.4/1.1).

Data are presented in the form of percent correct and reaction time latencies, and are shown separately for the two monkeys. Standard deviations are not shown for the percent corrects since only two injections were done in each monkey. Mean and standard deviations for the reaction time latencies are pooled across the two APB injections and across two days of normal testing.

The top graphs in figure 5 show the results for changing target luminance for both monkeys. As we have found before, APB results in a marked increase in latency and decrease in performance for the detection of incremental stimuli, and only slightly effects the detection of decremental stimuli. The bottom graphs in figure 5 show the results for changes in background luminance. For both monkeys the effect of APB is intermediate between that for incremental and decremental target changes. In monkey Q, both incremental and decremental

background changes are equally effected, while in monkey P incremental changes are slightly more affected. Thus the polarity of the temporal contrast change that defines a stimulus does not alone determine the stimulus' detectability following administration of APB.

Experiment 4 - Temporal contrast ramps

A monkey was trained to detect the appearance of a target stimulus presented at 1 of 4 positions. Stimuli consisted of 1° gaussians ($contrast_{1°} = 0.10 * contrast_{center}$) presented at 2° eccentricity along the horizontal and vertical. Background luminance was 50 cd/m^2. Stimulus luminance was either stepped or ramped up over a period of 500 or 1000 msec. The step or ramps were always centered within a 1000 msec window that occurred at a set time following the monkey's acquisition of the fixation spot. Following the 1000 msec stimulus window the monkey was allowed an additional 800 msec to initiate a saccade to the target window, during which the stimulus remained at its final luminance. Only incremental stimuli were shown; contrasts used were 0.067, 0.10, 0.13, 0.20, 0.27, 0.33, 0.50, and 0.83. Contrasts were measured as the final luminance divided by the initial (background) luminance. One monkey was used in this study; injection data represents 2 APB injections of similar efficacy (2.3/1.2, 2.4/1.1).

Data are shown in the form of percent correct and reaction time latencies. Standard deviations are not shown for the percent corrects since only two injections were done in each monkey. Mean and standard deviations for the reaction time latencies are pooled across the two APB injections and across two days of control testing.

The left graph in figure 6 shows the percent correct performance as a function of stimulus contrast for the different step/ramp types. Under normal conditions, the monkeys' ability to detect a stimulus of all but the highest contrasts is impaired by ramping the stimulus appearance. This can also be seen in the right graph in figure 6 as an increase in reaction time latency; note, however, that these values are confounded by the fact that low contrast stimulus ramps reached the monkey's contrast detection threshold later than did step changes. Following APB administration, the performance in all step/ramp types decreased, but not selectively for the ramps. In fact, there may be a saturation of the effect, such that the difference between the different step/ramp conditions is lessened. Thus ON channel blockade does not selectively impair the detection of incremental ramps over incremental steps.

Discussion

Many visual aftereffects, such as the various tilt and motion aftereffects, have be explained by proposing that the percept is carried in a distributed fashion by two or more opposed channels that are being differentially affected by the adapting stimulus. The resulting percept becomes biased toward the percept carried by the less adapted channel(s). If such a model applies to the perception of brightness, with the ON channel carrying the brightness percept and the OFF channel the darkness percept as proposed by Jung (1973), then a decrease in the activity of the ON channel, such as from APB, should cause stimuli to look less bright. Our data for the perception of incremental stimuli fits this model. However, if the ON channel carries the brightness percept alone, without comparison with the OFF channel, we might also expect compromise of the ON channel to lead to a decrease in perceived brightness. This latter hypothesis is unlikely given that the aftereffects produced by sawtooth adaptation are best explained by assuming a comparison is being performed between the ON and OFF channels (Cavanagh & Anstis 1986). What can be stated with certainty is that brightness information cannot be carried equally as well by either the ON or the OFF channel.

In the brightness shift experiment, we presented the incremental stimuli against a dark background intentionally to eliminate contrast information. Obviously this was not possible for decremental stimuli, and it's reasonable to assume that the brightness of this background field differed in the two eyes; the background may have differentially adapted the APB and control eyes. Thus it is difficult to say if the monkey was comparing brightness or apparent contrast. With the low

level APB administrations the differential adaptation was likely small, and ON channel blockade probably causes decremental stimuli to appear darker, as it does for incremental stimuli. If this is the case, it would indicate that the ON and OFF channels convey brightness and darkness, respectively, regardless of whether the stimulus is brighter or darker than background. At higher levels of ON channel blockade the difference in the adaptation states of the two eyes is larger, and the interocular comparison paradigm may not be suitable due to binocular rivalry. With such a dosage the monkey in fact may have been making a contrast comparison, and the apparent contrast of the injected eye's stimulus may have been quite low.

The moderate decrease in brightness of stimuli following APB administration cannot alone account for the impairment in detecting incremental stimuli. Furthermore, even high contrast incremental stimuli that are readily detected show a large increase in reaction time latency. The properties of a stimulus that determine its brightness then are not the same as those that determine its detectability.

The contrast at the edges of a stimulus remains an important factor in determining its brightness following ON channel blockade. Had this not been the case, the assessment of brightness would have been made more on the basis of absolute luminance than on a comparison of contrast. This implies that the lateral interactions subserving simultaneous contrast continue to function following impairment of the ON channel, in contrast to what Jung (1973) proposed. This parallels the findings for single unit receptive fields in LGN and cortex in the monkey: the center and surround are not created by opposing the ON and OFF channels (Schiller 1984a; Horton

& Sherk 1984; Sherk & Horton 1984). Whether it is really the center/surround nature of these early stages in vision that are responsible for contrast or brightness assessment is unknown. Certainly, however, if APB had a large differential effect on center and surround responses, contrast assessments would be greatly impaired and phenomena such as simultaneous contrast should be decreased.

As demonstrated by the target versus background experiment, temporal contrast change alone does not determine the detectability of a stimulus following APB administration. If this were the case, then APB would equally affect the detection of incremental foreground and background changes, and the detection of decremental background changes would have been little affected. The ON channel must convey "gestalt" information pertaining to the coherency of incremental stimuli in addition to temporal contrast increments. Thus an incremental stimulus, regardless if it appeared via an incremental or decremental temporal event, is carried at least in part by the ON channel. It would be interesting to test the effects of APB on detection of disembodied edges (Savoy & Burns 1989) since they are relatively devoid of the "gesalt" cues that define a stimulus as a bounded object.

Adapting to stimuli modulated in luminance with a sawtooth function has a large differential affect on the detection of ramped versus stepped luminance changes (Dolan 1992). The detection of ramps of the same direction as the ramp phase of the sawtooth is greatly impaired, whereas the detection of step changes is not. If luminance ramps are processed more exclusively by one channel than are luminance steps, there exists a simple explanation for this phenomenon. However, experiment 4 demonstrates that ON channel

blockade with APB does not affect the detection of ramps more than the detection of steps. Thus the effect of sawtooth modulation is unlikely to be just one of generically adapting the ON channel; the adaptation must occur differentially across temporal channels, as proposed by Cavanagh & Anstis (1986).

The general finding that light increment information is not conveyed to detection mechanisms by the OFF channel raises an obvious question: assuming that comparisons in activity between channels are being conducted, why would higher visual centers not make use of "cross-channel" information, that is light decrement information from the ON channel and light increment information from the OFF system? The most likely explanation is that such information is inherently less accurate than information carried by the "proper" channel. For one thing, given the low maintained discharge of retinal ganglion cells, the dynamic range carried by decreases in firing rate is lower than that carried by increases. Any push-pull interactions designed to increase overall gain should occur in the outer retina where such signals are graded. As mentioned previously, there is some evidence that this exists to a limited extent. More importantly, beyond the outer retina the ON channel conveys light increments with an increase in firing rate and provides better temporal resolution than can the OFF channel, which would signal such events by decreasing firing rate. As pointed out by Levick (1973), with a maintained discharge rate of 30/sec, approximately 5% of the interspike intervals are longer than 80 msec. This allows for unacceptably long delays, and combining such a signal with a more temporally accurate one may in fact compromise overall accuracy. Thus it may be that higher visual areas sacrifice

cross-channel information to retain optimal temporal specificity in each individual channel. This would be particularly important in the processing of fine moving patterns, where light increments and decrements must be processed equally quickly to prevent degradation of the image.

Acknowledgements

This work was supported by National Eye Institute research grant No. EY00676 to PHS, and by NIH training grant No. T32 GM07484 and NIMH training grant No. T32 MH15761 to RPD. We wish to thank Karen MacDonell for her technical and administrative assistance and Edward Tehovnik for his helpful comments on the manuscript.

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Figure legends

Figure 1. Representative data for detection under normal and APB-injected conditions (monkey P). The left graph shows reaction time latencies with standard deviations. The right graph shows percent correct performance; the horizontal line indicates the 25% performance lower limit for when the monkey is performing at chance. The left side of each graph shows decremental stimuli, the right side shows incremental stimuli. Note that APB selectively increases reaction time and decreases percent correct performance for incremental stimuli. Efficacy index (ratio of reaction time increase for increments vs. decrements at contrast = 0.2) for this injection was 2.3/1.2.

Figure 2. Percent correct performance for the brightness shift experiment (monkey P). Standard deviations are plotted. Normal and two APB-injected conditions of different efficacy are plotted; the target stimulus was presented to the APB-injected eye, the non-target (comparison stimulus) to the non-injected eye. The horizontal lines indicate the 50% performance lower limit for when the monkey is performing at chance. The abscissa plots the luminance difference between the target (fixed) and the non-target (variable). The left graph plots data for discrimination of two incremental stimuli. When the luminance difference between the target and non-target was highest (i.e. the non-target luminance was low), the monkey performed well under all conditions. However, following APB administration, the monkey's performance decreased faster as the non-target stimulus luminance increased, especially for the higher efficacy injection. The

right graph plots data when the stimuli were both decremental. When the luminance difference between the target and non-target was highest (i.e. the non-target luminance was high and thus its contrast was low), the monkey performed well under all conditions. Following the lower efficacy APB administration, the monkey's performance decreased less quickly than normal as the non-target luminance was increased. Following the higher efficacy injecting, the monkey's performance decreased much faster than normal as non-target luminance was increased.

Figure 3. Schematic showing luminance profile for ramped background used in simultaneous contrast experiment. Stimuli shown here are incremental. The target luminance was fixed and the comparison stimulus (non-target) varied across trials. The position of the target varied randomly for each trial, but data were analyzed only when the target was positioned such that its luminance equalled the background luminance at the comparison stimulus, as shown.

Figure 4. Percent correct performance for the simultaneous contrast experiment (monkey C). Left graphs show incremental stimuli, right graphs show decremental stimuli. The horizontal lines indicate the 50% performance lower limit for when the monkey is performing at chance. Upper graphs compare performance for homogeneous and graded background. In both cases, the monkey's performance reaches chance as the contrast of the comparison stimulus approaches the contrast of the target. If the monkey was making the brightness or darkness assessment based on absolute luminance, the curve for the

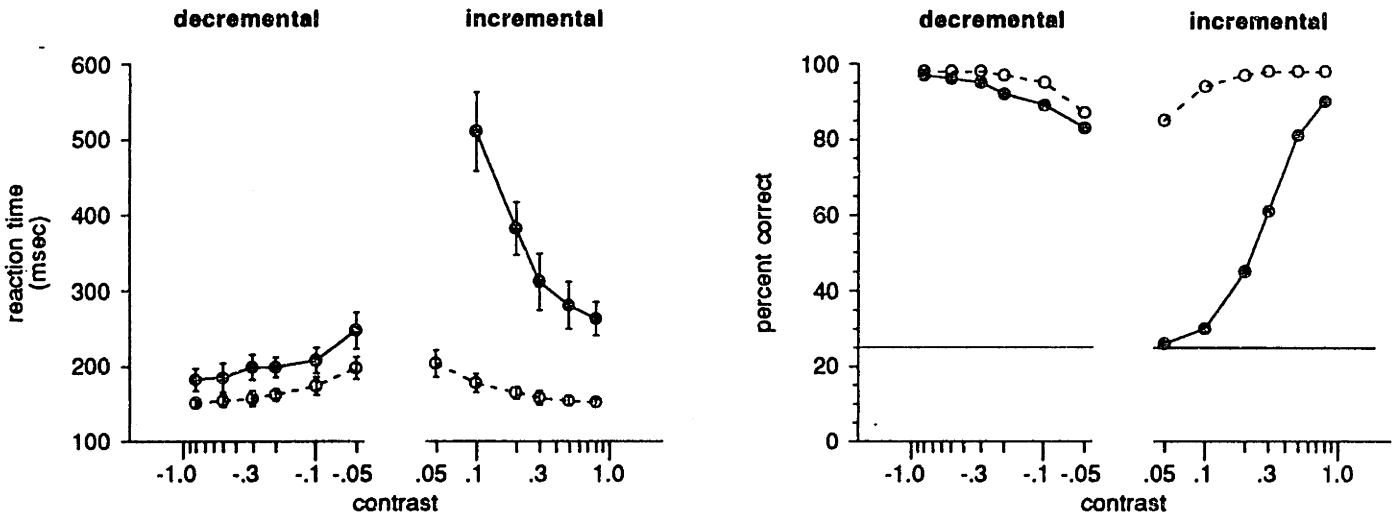
graded background trials would be shifted far to the right. Lower graphs show performance for graded background trials under normal and APB-injected conditions. For both incremental and decremental stimuli, the monkey's assessment of the relative brightness or darkness of stimuli is still based on contrast, rather than on absolute luminance.

Figure 5. Reaction time latencies and percent correct performance for changing target vs. background experiment. Left graphs show data for monkey Q, right graphs for monkey P. Standard deviations are plotted for reaction times. The horizontal lines on the percent correct plots indicate the 25% performance lower limit for when the monkey is performing at chance. Upper graphs show data for trials in which the target luminance increased (right side of each graph) or decreased (left side of each graph. APB increased reaction times and decreased percent correct for increments more than for decrements. Lower graphs show data for trials in which the background luminance increased (right side of each graph) or decreased (left side of each graph). APB's effect on reaction time and percent correct is intermediate between the effects on incremental and decremental target changes.

Figure 6. Reaction time latencies and percent correct performance for detection of step- vs. ramp-change experiment (Monkey P). Standard deviations are plotted for reaction times. The horizontal lines on the percent correct plots indicate the 25% performance lower limit for when the monkey is performing at chance. Reaction time increases and percent correct decreases as the period over which the stimulus

changes is increased (i.e. steps are more readily detected than ramps). Following APB administration, performance decreases overall, but not selectively for ramps.

Figure 1


decremental
incremental
reaction time (msec)
600
500
400
300
200
100
-1.0
-.3
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.05
.1
.3
1.0
contrast
decremental
incremental
percent correct
100
80
60
40
20
0
-1.0
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-.05
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.3
1.0
contrast

Figure 2

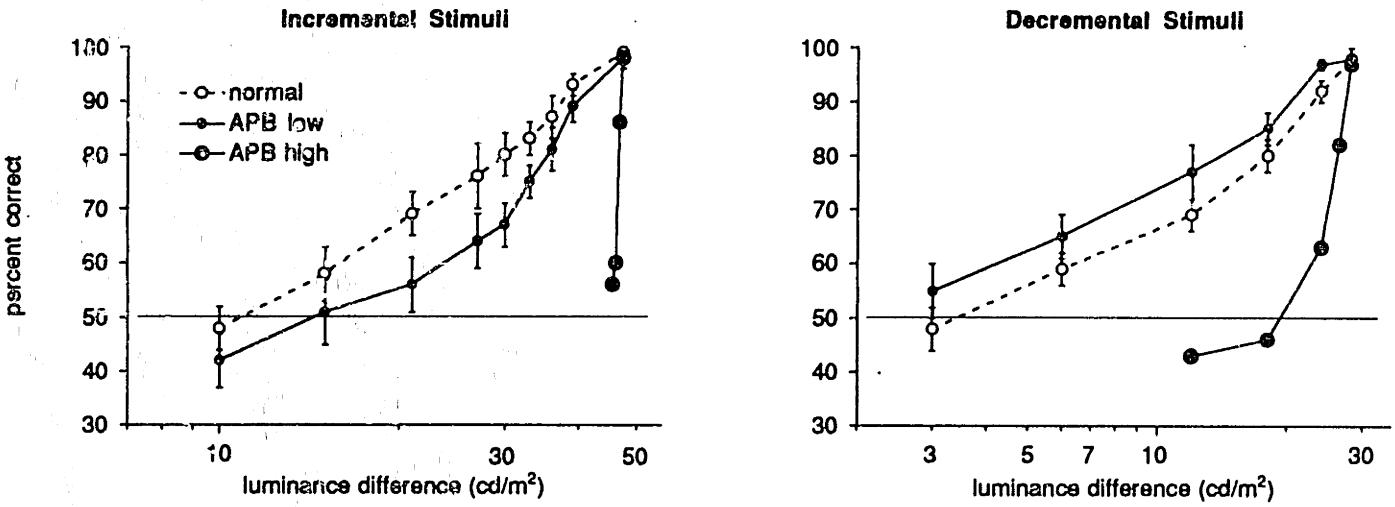

Incremental Stimuli
normal
APB low
APB high
percent correct
100
90
80
70
60
50
40
30
10
30
50
luminance difference (cd/m²)
Decremental Stimuli
100
90
80
70
60
50
40
30
3
5
7
10
30
luminance difference (cd/m²)

Figure 3

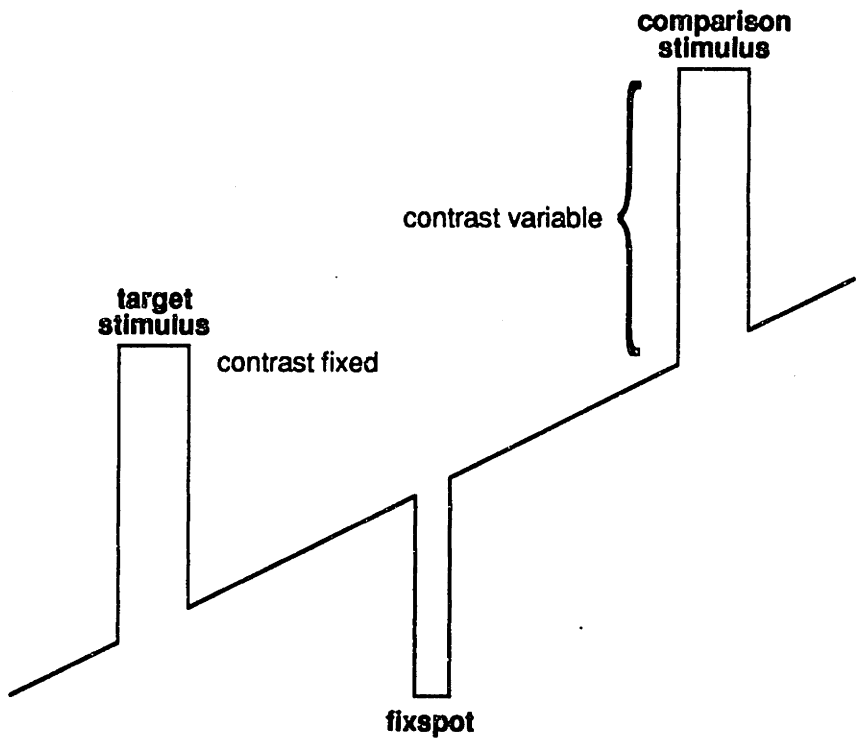
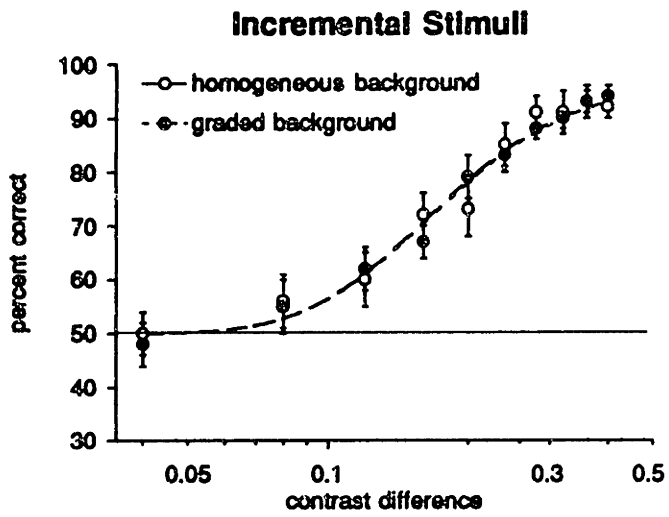
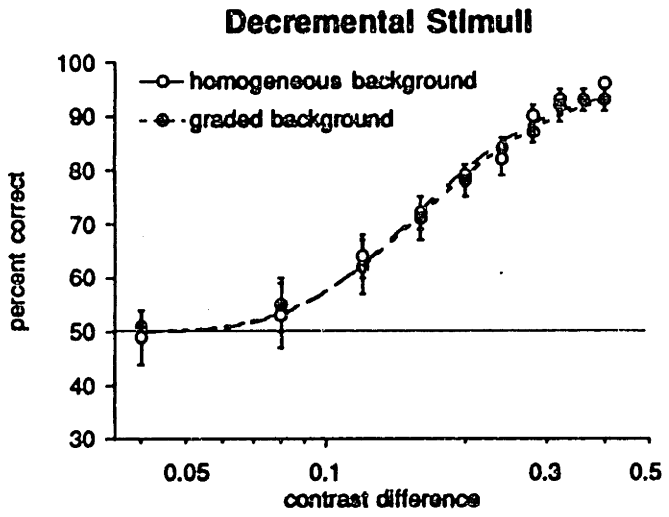

comparison stimulus
contrast variable
target stimulus
contrast fixed
fixspot

Figure 4


Incremental Stimuli
homogeneous background
graded background
percent correct
100
90
80
70
60
50
40
30
0.05
0.1
0.3
0.5
contrast difference


Decremental Stimuli
homogeneous background
graded background
percent correct
100
90
80
70
60
50
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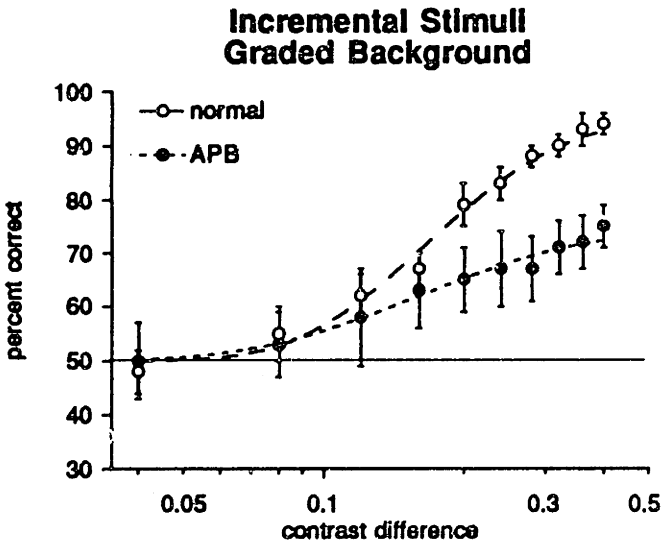
30
0.05
0.1
0.3
0.5
contrast difference


Incremental Stimuli
Graded Background
normal
APB
percent correct
100
90
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70
60
50
40
30
0.05
0.1
0.3
0.5
contrast difference

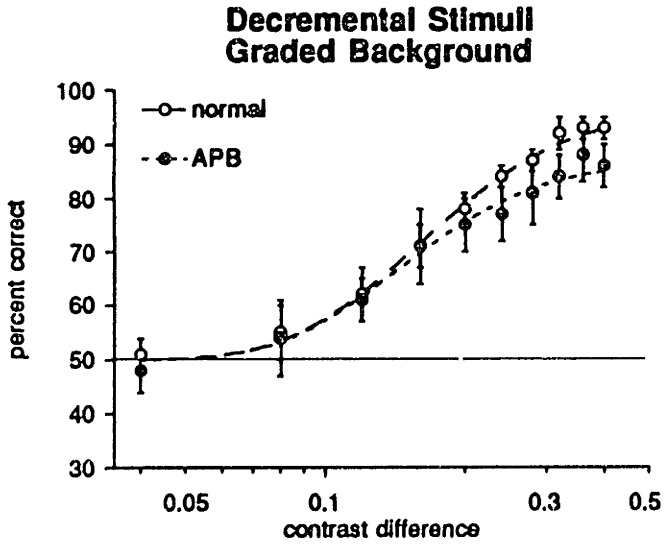
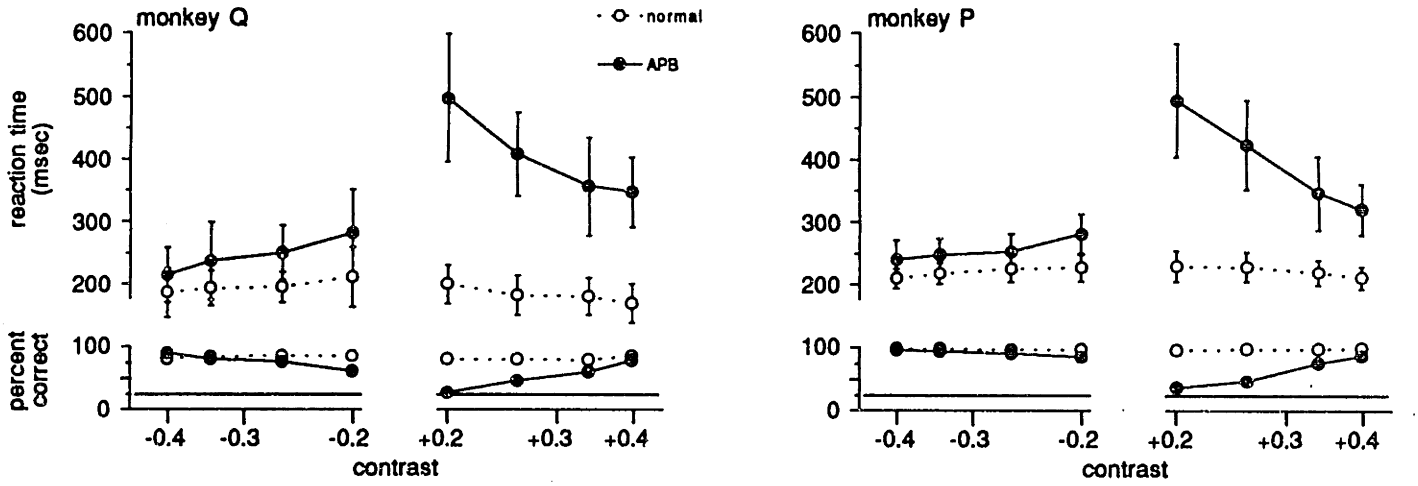

Decremental Stimuli
Graded Background
normal
APB
percent correct
100
90
80
70
60
50
40
30
0.05
0.1
0.3
0.5
contrast difference

Figure 5

Changing Target



Changing Background

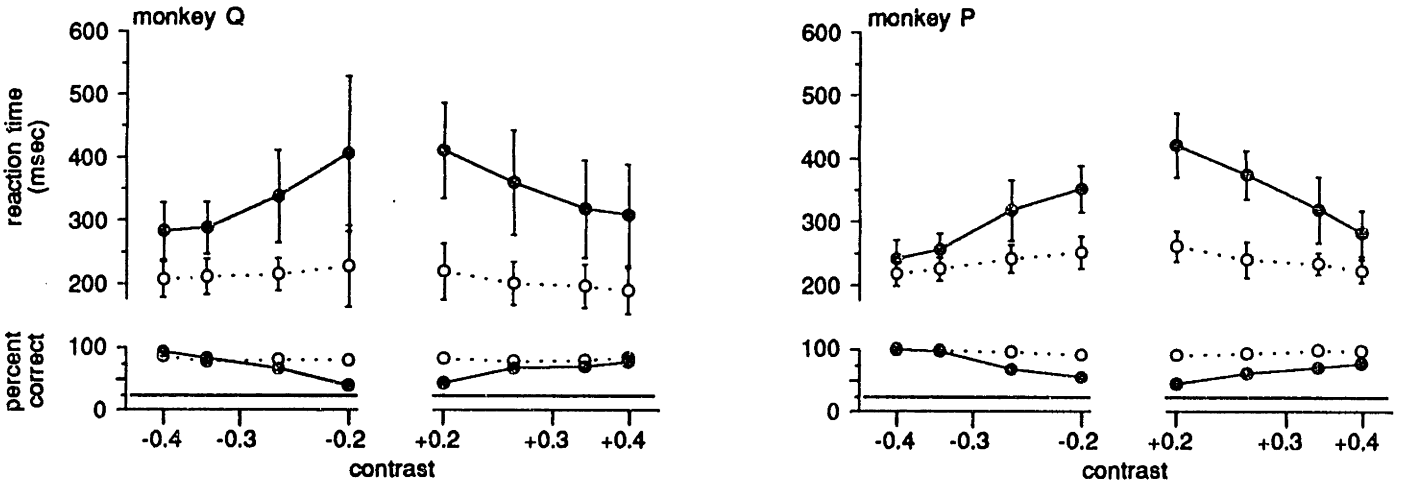
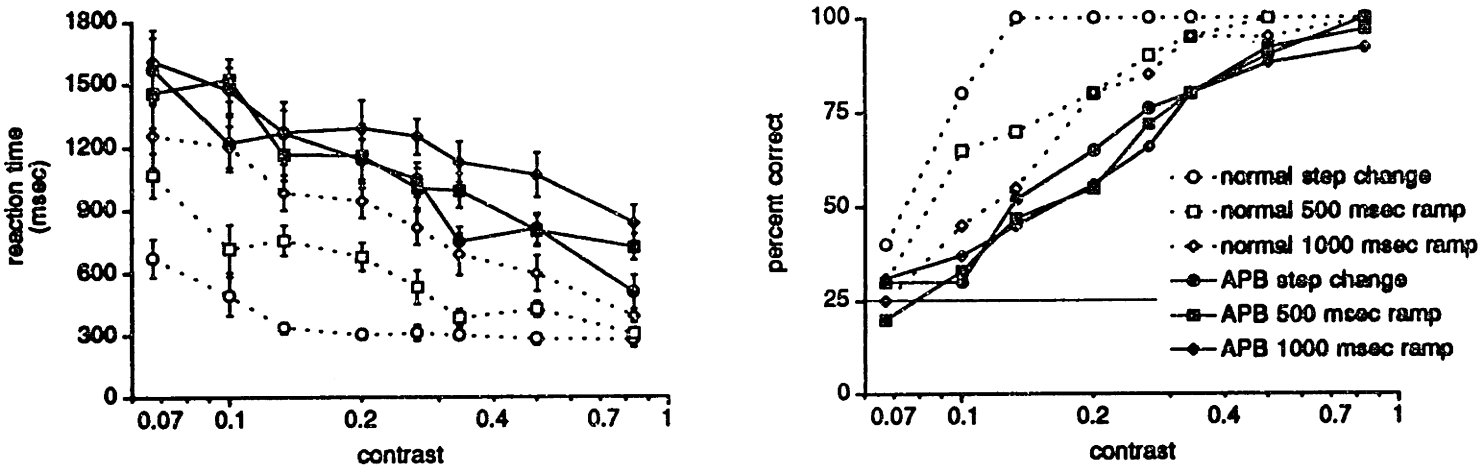


Figure 6

Ramp Detection



Chapter 3

Temporal Factors in Luminance Channel Adaptation Following Sawtooth Modulation

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Temporal Factors in Luminance Channel Adaptation Following Sawtooth Modulation

Abstract

Adaptation to stimuli modulated in luminance with a sawtooth temporal profile has been reported to induce two effects: it produces a dynamic luminance aftereffect in the direction away from the adaptation ramp change and it selectively raises detection threshold for contrast steps of the same polarity as the adaptation step change. Both mechanisms have been explained by a non-linearity in adaptation of incremental and decremental channels; however, this non-linearity works inversely in the two cases. In this study these two phenomena are examined using spatially distinct stimuli and test subjects across a range of adaptation and test stimulus temporal profiles. The results indicate that the detection of incremental and decremental step changes is equally affected by sawtooth adaptation, but that the detection of ramp changes of the same polarity as the adaptation ramps is selectively impaired. In addition it was found that the dynamic luminance aftereffect appears to sum with Troxler-like fading. Both phenomena show a peak effect with the same adaptation profile; this peak is defined by rate of contrast change in the ramp portion of the adaptation and appears to be less dependent on its temporal frequency. I conclude that sawtooth luminance modulation selectively adapts luminance channels of varying temporal properties, and that these correspond to the ON and OFF channels of early vision.

Introduction

We have all experienced luminance afterimages in which prior exposure to a bright or dark adapting field induces an illusion of a field opposing in contrast. An interesting phenomenon first reported by Anstis (1967) occurs when the luminance of the adapting stimulus changes linearly over time; in this case the afterimage changes in a contrast direction opposite to the adaptation. Repeated presentations of such an adaptation in the form of a sawtooth luminance profile produces a robust aftereffect; thus adapting to a stimulus with a ramp up/step down luminance profile produces an aftereffect that appears as a single ramp down in luminance. Anstis also reported the absence of any interocular transfer of this phenomenon, and later demonstrated the perceptive fields for this phenomenon to be quite large (Anstis & Harris 1987).

The notion that separate neural mechanisms process light increments and decrements (Jung 1973) yields a simple explanation of this dynamic luminance aftereffect. Differential adaptation of these two luminance channels produces a neural bias toward the less adapted channel and thus a perceptual bias toward the information it carries (Anstis 1967). This differential adaptation occurs due to greater saturation in the channel processing the step phase of the adaptation than in the channel processing the ramp phase, owing to the higher temporal rate of luminance change in the latter (Cavanagh & Anstis 1986). The luminance channels being adapted are assumed to be transient in nature, as they are responsible for carrying information relating to changing luminances, possibly as a result of object motion.

If there is only one incremental and one decremental channel, one would predict from the above model that a ramp up/step down sawtooth adaptation, which produces a ramp down aftereffect, would also selectively impair detection of light increments more than light decrements. However, if there are multiple incremental and decremental channels with different temporal properties, the effect of sawtooth adaptation on detection could depend on the temporal nature of the test stimulus. Krauskopf (1980) found a selective increase in the detection threshold of step change stimuli of the same luminance polarity as the step change in the adaptation sawtooth. He proposes, in contrast to Anstis, that convolution of sawtooth stimuli with an impulse response function (Sperling & Sondhi 1968) produces a non-linearity that biases adaptation of the step change channel over the ramp change channel. The only way to relate these two notions is by assuming that multiple temporal channels exist for processing each luminance polarity, and that they are being differentially adapted by sawtooth luminance modulations. Thus, a ramp up/step down sawtooth might selectively adapt a mechanism for processing of incremental ramps, producing an aftereffect of a ramp down, and selectively adapt a mechanism for processing of decremental steps, producing an impairment in detection of decremental steps.

In order to test this hypothesis, it is necessary to test the effect of sawtooth adaptation not only for the detection of luminance steps but luminance ramps as well. Hanly & MacKay (1979), adapting and testing with sawtooth stimuli, found a selective impairment for detection of a stimulus of the same polarity as the adaptation. However, since the test stimulus contains both step and ramp components, there is

no way of determining what phase of the adaptation affected what phase of the test stimulus.

If stimuli differing in rate of luminance change indeed undergo differential processing in the visual system, it is important to understand whether this occurs via distinct sets of temporally tuned channels. Also, it should be demonstrated whether the two effects of sawtooth adaptation, namely the polarity specific increase of detection thresholds and the dynamic aftereffect itself, are part of the same process. In the present study I test the effect of varying sawtooth temporal profiles on (1) detection of test stimuli of varying luminance slope and (2) magnitude of the induced afterimage.

Methods

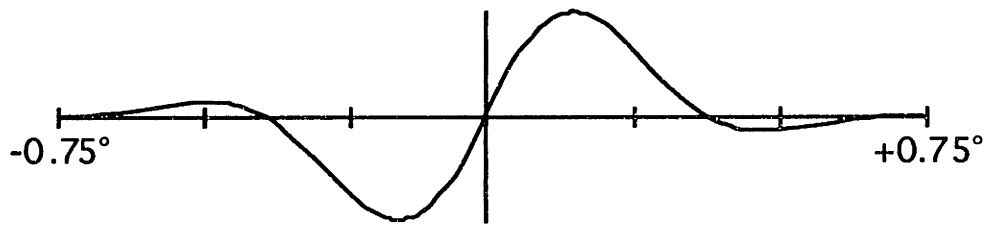
Stimuli

Stimulus generation, experimental control, and data collection were done on an Macintosh microcomputer. The 3 8-bit channels of the computer's internal video controller were run together through a passive resistor network (ISR video attenuator) (Pelli & Zhang 1991) and used to control the green gun on a Sony 1302 color monitor with a 60.0 Hz frame rate. Following gamma correction this produced an effective 12 bit linear control of gun luminance. Background luminance was fixed at 45.5 cd/m^2, yielding CIE coordinates of {.280, .598}.

Adaptation and test stimuli consisted of gabor functions with the following luminance profile:

$$Lum_x = Lum_{bkg} + 1.24 * \sin(x * 2.7 * \pi) * \exp(-8.0 * x^2)$$
$$Lum_y = Lum_{bkg} + \exp(-8.3 * x^2)$$

where x is expressed in degrees and Lum_{bkg} is the background (mean) luminance. The horizontal spatial profile is sketched here:



Choice of stimulus spatial frequency and envelope was constrained by findings of Anstis and Harris (1987). At an eccentricity

of 1°, they found the perceptive fields governing the luminance aftereffect to be approximately 0.34°. Thus the full period of the stimuli was set to approximately twice this value to minimize the possibility of having the two contrast opposed halves of the stimuli falling into one perceptive field. In fact, at 1° eccentricity with stimuli on the diagonal axes, stimulus diameter is limited to approximately this value. These stimuli appeared as a pair of flanking vertically oriented blurred oval regions, 0.7° in height and 0.35° in width, of opposing contrast. Two additional outer flanking regions were discernable at high contrasts. Stimuli were presented at 1° eccentricity, along the diagonals to avoid orientation specific effects that were apparent when the stimuli were positioned along the horizontal and vertical meridians.

Stimulus contrast changes occurred in spatial counterphase. = Contrasts are expressed as $(Lum_{stim} - Lum_{bkg}) / Lum_{bkg}$. For simplicity, the remainder of this paper will refer only to changes in the right flanking region of the stimulus. Adaptation stimuli varied in contrast over time as a function of either a triangular wave or a sawtooth wave, with temporal frequencies of 1, 3, 6 and 12 Hz. Total contrast range of the adaptation was either 2.0 (yielding contrast slopes on the ramp portion of 2.0/sec, 6.0/sec, 12.0/sec, and 24.0/sec) or 1.0 (yielding contrast slopes of 1.0/sec, 2.0/sec, 6.0/sec, and 12.0/sec). Sawtooth adaptation stimuli were always ramped up and stepped down in luminance, and were modulated around the mean background luminance. Test stimuli varied in contrast over time as a single ramp function, with contrast slopes of 0.25/sec, 0.75/sec, 1.5/sec, 3.0/sec and ∞/sec (step change) of both polarities. Adaptation stimuli whose ramp phase was in the same direction as the test stimulus contrast will be

referred to as same-sign adaptations, those in the opposite direction as opposite-sign adaptations (figure 1).

Stimuli were viewed from a distance of 60 cm. Identical left and right eye images were generated separately on the monitor and were viewed as a single fused image through a prism assembly. This configuration proved more comfortable for prolonged viewing because of the relaxed vergence. A bezel assembly containing the prisms fitted over the monitor and served as a light box to eliminate ambient light. Subjects refrained from blinking except during intertrial periods to reduce artifacts during the adaptation period.

Psychophysical procedures

Two different tasks were employed in this study: a 4 position detection task and a method of adjustment task. Both tasks were presented either with or without an adaptation period prior to test stimulus appearance.

In the 4 position detection task subjects fixated a small bullseye pattern for 1500 msec (4200 msec for adaptation trials) (figure 1). The final 1000 msec of this period was the test stimulus time window, during which a stimulus appeared in 1 of 4 fixed positions. The test stimulus was always centered in the 1000 msec time window. At the end of the test period the fixation pattern was reversed in contrast as a cue for the subject to respond by depressing a key corresponding to the test position. Subjects were required to respond within 700 msec of the cue, during which period the test stimulus maintained its final contrast value. As this was a forced choice paradigm, trials in which the subject either failed to respond or responded too late were discarded. 1800 msec

following the cue a new trial began. Test stimulus contrast ranges were chosen using the BestPEST method (Lieberman & Pentland, 1982). Thresholds and standard deviations were determined from an end-weighted running average of the final 90 trials of a 100 trial series. Test stimulus contrast slopes remained fixed for a given trial series.

In the method of adjustment tasks subjects were required to adjust the slope of a test stimulus temporal contrast ramp so that it appeared to be constant contrast. Contrasts at the start of the ramp were either -0.5 or +1.5 of background; effects observed for test stimuli of 0 contrast (i.e. changes to a uniform background) were too small to be accurately quantified. Test ramp duration was fixed at 500 msec, after which the test contrast was stepped back to background and the cue appeared. Adjustments were made in the intertrial time following the cue and were done blindly, thus affecting the next trial's test stimulus. Once subjects were satisfied with their adjustments they depressed a key indicating that the next adjustment trial series should begin. Each run consisted of 10 such sets of adjustments. Test contrast slopes for the first trial of each run were chosen randomly.

Adaptation trial runs for both tasks began with a 30 sec pre-adaptation period. Adaptation duty cycle was 50%; greater duty cycles improved the aftereffects only marginally, but the preadaptation period proved critical for maintaining a stable aftereffect. Longer preadaptation periods did not improve the effects significantly. Adaptation temporal frequency and contrast range were fixed for a given run of trials.

On any given day, non-adaptation trials were performed prior to adaptation trials, minimizing any possible long-term adaptation effects.

In addition, trials of a given adaptation type were run in groups, again to minimize potential cross-interference.

Subjects' ability to maintain fixation during adaptation and test stimulus presentation was confirmed periodically by having the subjects attend to the strong afterimage generated by the fixation pattern. For all adaptation conditions subjects reported the afterimage to be well defined and in alignment with its contrast reversed image.

Subjects

One author (RD) served as one of the two subjects, and is a 30 year old male with normal visual acuity. The other subject is a 23 year old male with normal visual acuity using usual refractive correction, which he wore for the experiments.

Results

Detection threshold changes

Figure 2 (top panel) shows that detection thresholds for step change targets increase as temporal frequency increases. There is no differential effect between the three adaptation profiles.

In contrast, the bottom panel of figure 2 shows that there are large differences between the adaptation conditions for detection of ramp stimuli (∂ contrast = 0.25/sec). Same-sign sawtooth adaptation produces the largest threshold increase, with a peak effect at 3 Hz. Both opposite-sign sawtooth adaptation and triangular adaptation cause a monotonic rise in threshold with increasing temporal frequency, as with step stimuli; thresholds following adaptation to opposite-sign sawtooth adaptation are slightly higher than for triangular adaptation.

Figure 3 plots data for all four test ramp slopes, plotted as change in log threshold contrast. As the slope of test ramp decreases, the differential effect of the same and opposite-sign sawtooth adaptation becomes more pronounced. For the 2 slower ramp stimuli, same-sign adaptation produces a peak effect with temporal frequency of 3 Hz.

To determine whether the peak in the adaptation tuning curves is due to optimizing the temporal frequency or the contrast change rate of the adaptation, the adaptation contrast range was halved. Figure 4 shows the effect of the different adaptation conditions on detection of ramp stimuli (∂ contrast = 0.25/sec). Under these conditions the optimal adaptation temporal frequency has shifted to 6 Hz. At half contrast, this represents the same contrast change rate as the 3 Hz. adaptation at full contrast (∂ contrast = 6.0/sec). These results suggest that the rate of change of ramp portion of the adaptation stimulus, and

not its temporal frequency, determines its effectiveness for producing polarity specific detection impairment.

Dynamic aftereffect assessment

Figure 5 shows the effect of the varying adaptation waveform and temporal frequency on the dynamic luminance aftereffect. With no adaptation, it was a test ramp that gradually increased in luminance that appeared to be of constant in brightness.

incremental luminance ramp appeared to subjects as constant. This presumably represents the contrast change necessary to offset Troxler-like fading. Adaptation to triangular waveforms of all temporal frequencies did not increase in the amount of contrast added (dotted line).

Adaptation to sawtooth stimuli in which the ramp was of the same contrast polarity as the test stimulus induced an aftereffect of decreasing contrast in the test stimulus; thus subjects had to add contrast to the stimulus (figure 5 top panel, solid line). Adaptation to sawtooth luminance profiles in which the ramp was of opposite contrast polarity as the test stimulus again induced an aftereffect of darkening, but this now acted to increase apparent contrast in the test stimulus; thus subjects subtracted contrast (added luminance) from the stimulus to keep its appearance one of constant contrast (dashed line). In both cases the peak effect was observed when the adaptation temporal frequency was 3 Hz, corresponding to a ramp contrast change rate of 6.0/sec. The effect is stronger when adaptation ramp and test stimuli were of the same contrast polarity. In these cases there appears to be a cumulative effect of the fading.

As with the detection task, the importance of adaptation temporal frequency versus adaptation ramp slope was assessed by halving the adaptation contrast range. The bottom panel of figure 5 shows that under these conditions the peak effect is observed when the adaptation has a temporal frequency of 6 Hz, corresponding to a ramp contrast change rate of 6.0/sec.

Discussion

Adapting to a stimulus modulating in time with a sawtooth luminance profile differentially affects the detection of incremental and decremental ramps. This polarity specific effect becomes more pronounced as the slope of the test ramp decreases and is not seen for detection of step change stimuli.

If increments and decrements regardless of temporal profile are processed by only two luminance channels, it is necessary to explain how sawtooth adaptation differentially affects these two channels. The proposal of Cavanagh & Anstis (1986), that a saturation in response to the step change portion of the sawtooth adaptation creates a non-linearity which biases the effect of the adaptation in favor of the ramp change direction, does not alone explain why ramped stimuli are differentially affected and step stimuli are not. One possibility is that step changes are detected to a large degree by both the incremental and decremental channels, while ramp stimuli are detected more exclusively by only one channel and thus more susceptible to adaptation of the channel that carries them preferentially. However, recent evidence has shown that ON-channel blockade by APB does not increase threshold for detection of incremental ramps more so than it does for detection of incremental steps (Dolan and Schiller 1992).

Alternatively, and more in agreement with current theories of early vision (e.g. Thompson 1984; Hammet & Smith 1992; Snowden & Hess 1992), increments and decrements could each be processed by multiple temporal channels. If this were the case, one might expect to find the opposite effect for step stimuli as for ramp stimuli, namely that step stimuli with the same contrast polarity as the sawtooth step phase

would be more difficult to detect. Although no evidence for this was found in this study, it is possible that the step-specific effect is much smaller in magnitude and was masked by the ramp-specific effect. Krauskopf (1980), who has found such an effect, unfortunately did not test the detection of luminance ramps in his study.

Relating this adaptation phenomenon to existing notions of temporal channels is not straightforward. For one thing, the test stimuli and induced aftereffect are non-periodic. Although the adaptation is periodic, even single ramps can induce the aftereffect (Anstis 1967). Furthermore, the adaptation's rate of contrast change, not its temporal frequency, was found to be important for determining the magnitude of the aftereffect. Unlike conventional studies of temporal channels using periodic oscillations for masking and testing stimuli, there is no direct match between rates of luminance change of the adaptation and the test or aftereffect; the peak effects are produced with adaptation ramp rates far higher then either the most-affected detection test stimulus or the strongest induced dynamic aftereffect.

Still, there might be some notion of tuning of the adaptation stimulus. In this study stimuli were intentionally confined to a specific spatial frequency and position; I am currently investigating the effect of changing eccentricity and spatial frequency to see if spatial and temporal sensitivities covary, as Hess and Snowden (1992) have done for masking and detection with sinusoidal oscillations.

One explanation for the selective increase in threshold in detection of ramps following same-sign adaptation is that the afterimage of decreasing contrast subtracts from the actual contrast in a fashion that lessens its detectability. This would not explain the

smaller threshold increase following opposite-sign adaptation; in fact if the aftereffect added to the actual stimulus one might expect an improvement in detection. Also, it would be difficult to explain why the detection of steep ramps is impaired. It is likely that differential adaptation puts the entire system into a compromised and thus less sensitive state. In any case, given that both the luminance aftereffect and the ramp detection effect are maximal with the same adaptation contrast change rate, it is reasonable to assume that they are part of the same process.

Apparently there is an interaction between Troxler-like fading and the brightness shifts produced by the adaptation condition. Although Troxler (1804) described the fading of stabilized images as occurring in the periphery, foveal images undergo fading when properly stabilized; Gerrits (1978) in fact found fading to occur faster in the fovea than in the periphery for well stabilized images. While the stimuli in the current study were by no means retinally stabilized, they were spatially blurred enough that small eye movements did not produce large enough local contrast changes to reverse the fading effect; the small but significant contrast ramp needed to "null" the steady luminance stimuli attests to this fact. Given that it was possible to induce a steady contrast stimulus to *not* fade with some of the ramp down adaptations it is relevant to ask if the two effects combine. The contrast-decreasing afterimage (figure 5, solid line) is stronger than the contrast-increasing afterimage (dashed line), when comparing the absolute values of the contrast shifts. If the contrast value required to stabilize steady stimuli is subtracted from the two curves, their absolute values match quite well.

In agreement with previous reports (Hanly & MacKay 1979; Anstis & Harris 1987), no interocular transfer of the polarity specific adaptation effect was found. It is reasonable then to assume that eye of origin information is still available at the level at which this adaptation takes place, thus placing the adaptation mechanism in the retina, LGN, or striate cortex.

It is interesting to note that the contrast changes for the two polarities of aftereffect are fairly symmetrical when contrasts are measured in terms of background luminance as opposed to test stimulus luminance. If contrasts were represented in terms of test stimulus luminance, the contrast values for the contrast-decreasing stimuli would be one-half their values as plotted in figure 5, while those of the contrast-increasing stimuli would be twice their values as plotted. Apparently the aftereffect adds or subtracts luminance rather than contrast to a stimulus.

As noted previously, Krauskopf (1980) found a polarity specific adaptation effect on detection of step changes. However, his stimulus configuration differed greatly from ours: he used a solid 9.5° disk for adaptation and test stimulus. Given the sharp edges of such a stimulus, it is likely that detection was mediated at the edges, at an eccentricity (assuming steady fixation) of 4.25°. This is quite different from the stimuli used in this study (blurred contrast edges centered at approximately 1° eccentricity). Still, I have no clear explanation on why this difference in stimulus would cause such a difference in results.

How reasonable is it to assume that these incremental and decremental luminance channels correspond to the physiological defined ON and OFF channels? Previous findings using the

pharmacological agent 2-amino-4-phosphonobutyrate (APB) to block the ON pathway clearly demonstrate the role of the ON channel in selectively carrying light increment information (and presumably likewise for the OFF channel) (Schiller et al. 1986; Dolan & Schiller 1992). Given the evidence that the dynamic luminance aftereffect shows no interocular transfer, it is straightforward to assume the adaptation is occurring somewhere between the bipolar cells and striate cortex, and probably as a result of differential adaptation of temporal channels within the ON and OFF pathways.

Acknowledgements

This work was supported by National Eye Institute research grant No. EY00676 to Peter H. Schiller. I wish to thank Edward Tehovnik for his helpful comments on the manuscript and Karl Zipser for his time as a subject. I am indebted to Peter H. Schiller for his support during this study.

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Figure legends

Figure 1. Schematic showing trial paradigms for the method of detection and method of adjustment tasks. The horizontal scale represents time (not to scale), with luminance represented on the vertical dimension. In both tasks a fixation pattern is illuminated to commence each trial, and remains constant until it reverses contrast as a cue for the subject to respond. The adaptation stimulus, if present, is a ramp up/step down staircase and begins once the subject has indicated that fixation has begun. In the detection task, a step or ramp stimulus is presented after a 200 msec gap and is centered in a 1000 msec stimulus window; it is followed by the appearance of the cue. The subject has an additional 700 msec to respond by indicating the position in which the test stimulus appeared. In the adjustment task, the subject is shown a 500 msec test ramp following the 200 msec gap, and is then allowed to adjust the contrast slope of the next test stimulus. Intertrial period brings each trial up to total of 6 seconds. Note that adaptation stimuli are referred to as same-sign or opposite-sign depending on the polarity of the test stimulus with respect to the ramp phase of the adaptation.

Figure 2. Results for detection of step change and slow ramp (∂ contrast = 0.25) test stimuli. Test stimulus contrast thresholds (∂Lum_{stim} / Lum_{bkg}) are plotted against temporal frequency of the adapting stimulus. For step change stimuli, thresholds increase slightly as adaptation frequency increases, regardless of the adaptation waveform. For slow ramp stimuli, thresholds following triangular and opposite-sign sawtooth adaptation increase as for step change stimuli; thresholds

following same-sign sawtooth adaptation show peak at adaptation frequency of 3 Hz.

Figure 3. Results for detection of full series of slow ramp test stimuli. Change in log thresholds ($log\ (\partial Lum_{Stim} / Lum_{bkg})_{adapt} - log\ (\partial Lum_{Stim} / Lum_{bkg})_{normal}$) are plotted against temporal frequency of the adapting stimulus. Effect of triangular and opposite-sign sawtooth adaptation increases as adaptation frequency increases. Same-sign sawtooth adaptation shows peak effect at 3 Hz for slower test stimulus ramps. In general the effect of adaptation, especially the difference between the two sawtooth polarities, increases as test stimulus ramp slopes decrease.

Figure 4. Results for detection of slow ramp test stimulus (∂ contrast = 0.25) following adaptation at half previous contrast range. Peak adaptation effect has shifted from 3 Hz to 6 Hz, preserving sawtooth ramp phase contrast slope of 6.0/sec.

Figure 5. Results for afterimage matching task. Following no adaptation and triangular adaptation, subjects added contrast to the test stimulus to counteract fading. Following same-sign sawtooth adaptation (polarity of test stimulus equals adaptation ramp direction), subjects added more contrast to counteract aftereffect of decreasing apparent contrast. Following opposite-sign sawtooth adaptation, subjects subtracted contrast to counteract aftereffect of increasing apparent contrast. For both types of sawtooth adaptation, peak effect is observed when adaptation temporal frequency is 3 Hz. Lower panel

shows same effect following adaptation at half previous contrast; peak effect is observed when adaptation temporal frequency is 6 Hz, preserving sawtooth ramp phase contrast slope of 6.0/sec.

Figure 1

Task Descriptions

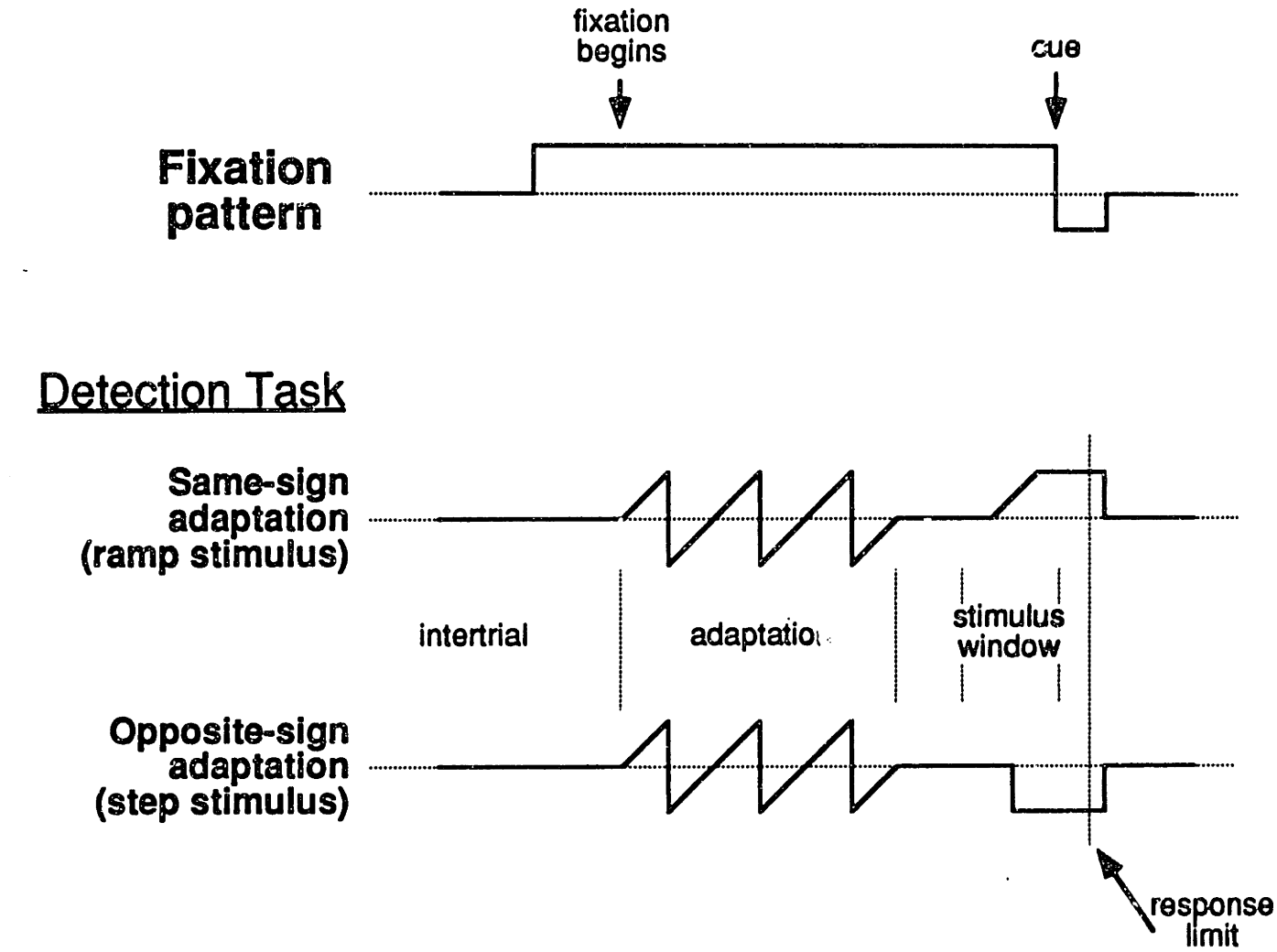


Figure 2

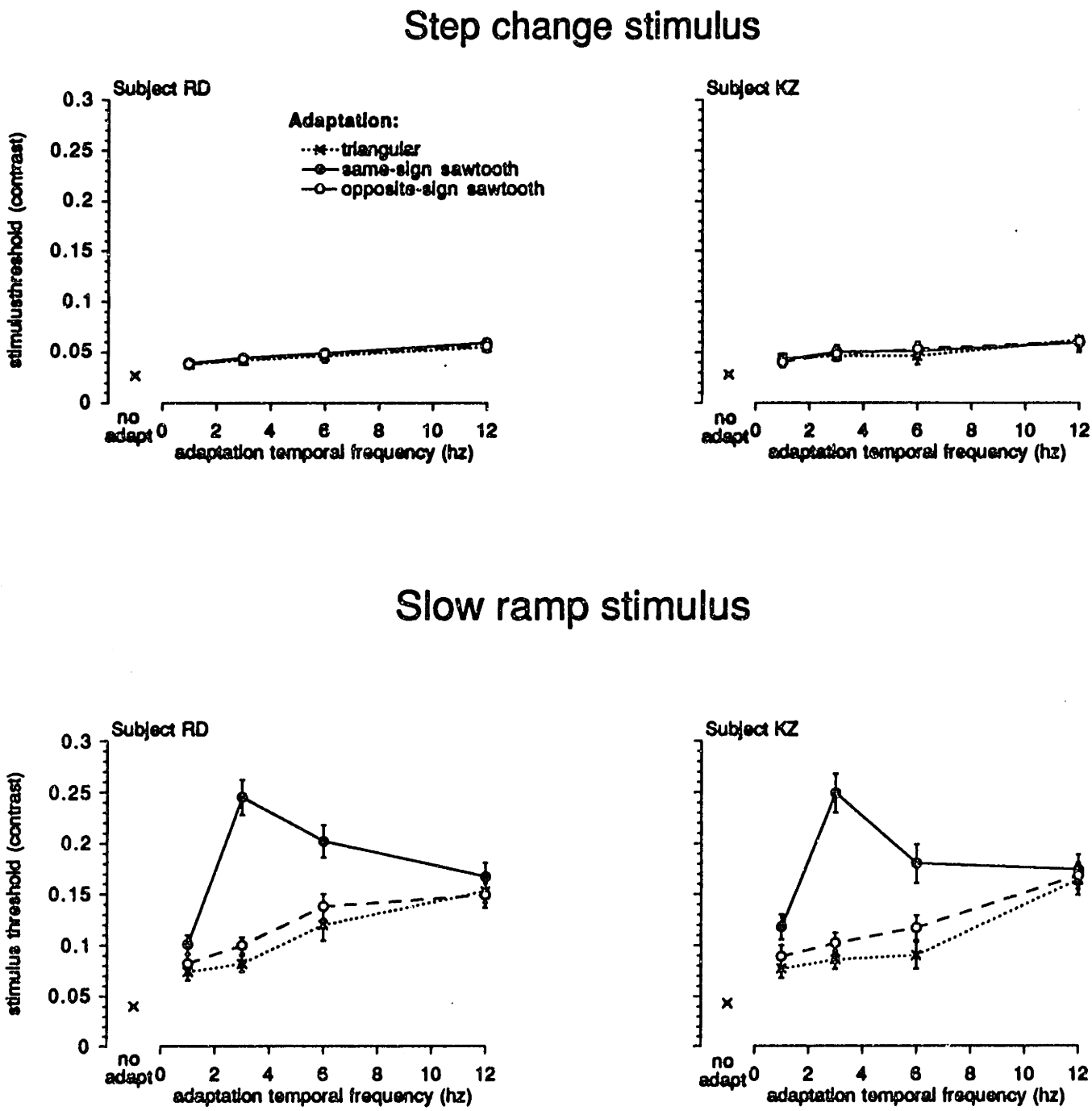

Step change stimulus
Subject RD
Subject KZ
Adaptation:
triangular
same-sign sawtooth
opposite-sign sawtooth
stimulusthreshold (contrast)
0.3
0.25
0.2
0.15
0.1
0.05
0
no adapt
0
2
4
6
8
10
12
adaptation temporal frequency (hz)
Slow ramp stimulus
Subject RD
Subject KZ
stimulus threshold (contrast)
0.3
0.25
0.2
0.15
0.1
0.05
0
no adapt
0
2
4
6
8
10
12
adaptation temporal frequency (hz)

Figure 3

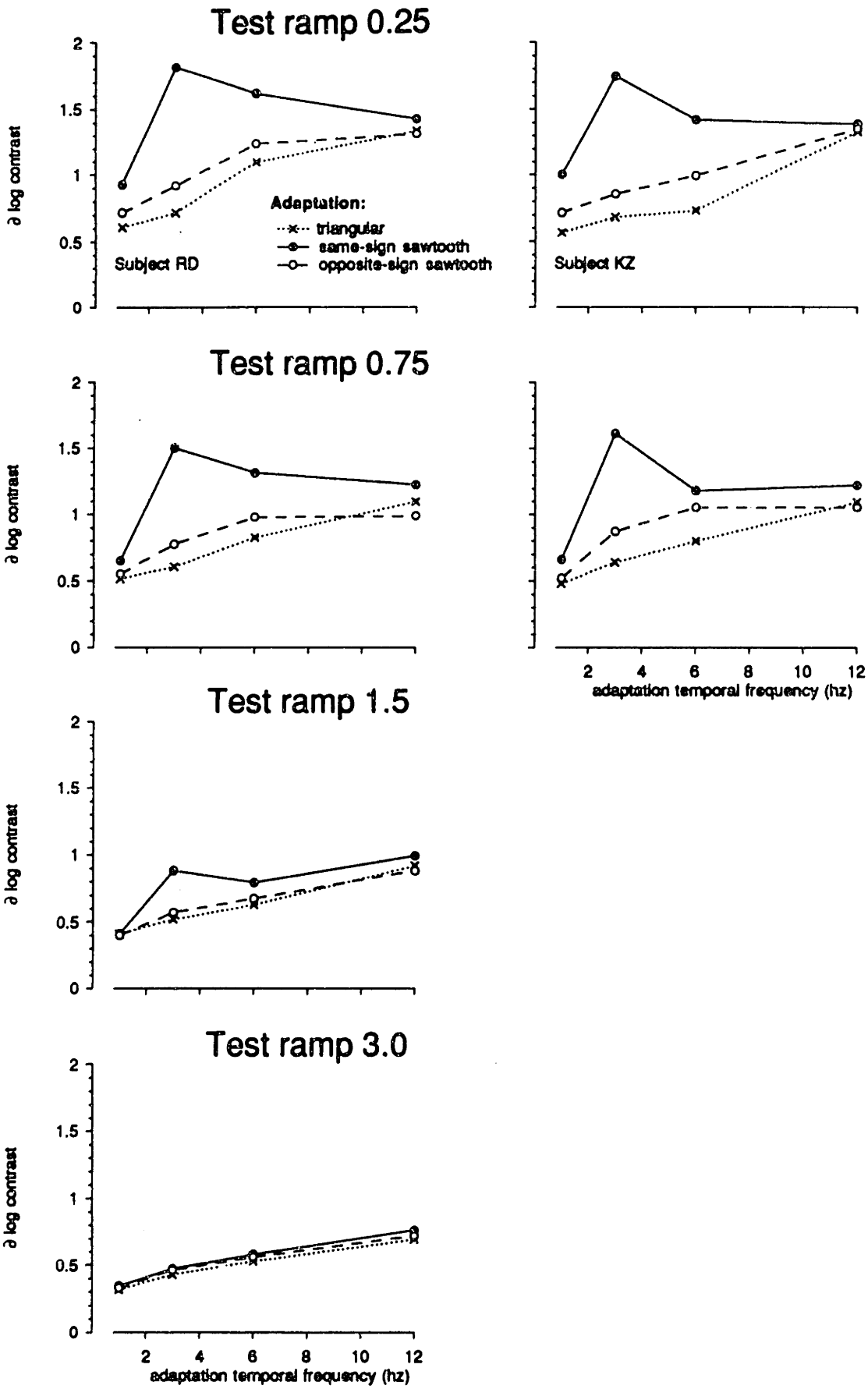

Test ramp 0.25
Test ramp 0.75
Test ramp 1.5
Test ramp 3.0
∂ log contrast
Adaptation:
triangular
same-sign sawtooth
opposite-sign sawtooth
Subject RD
Subject KZ
adaptation temporal frequency (hz)

Figure 4

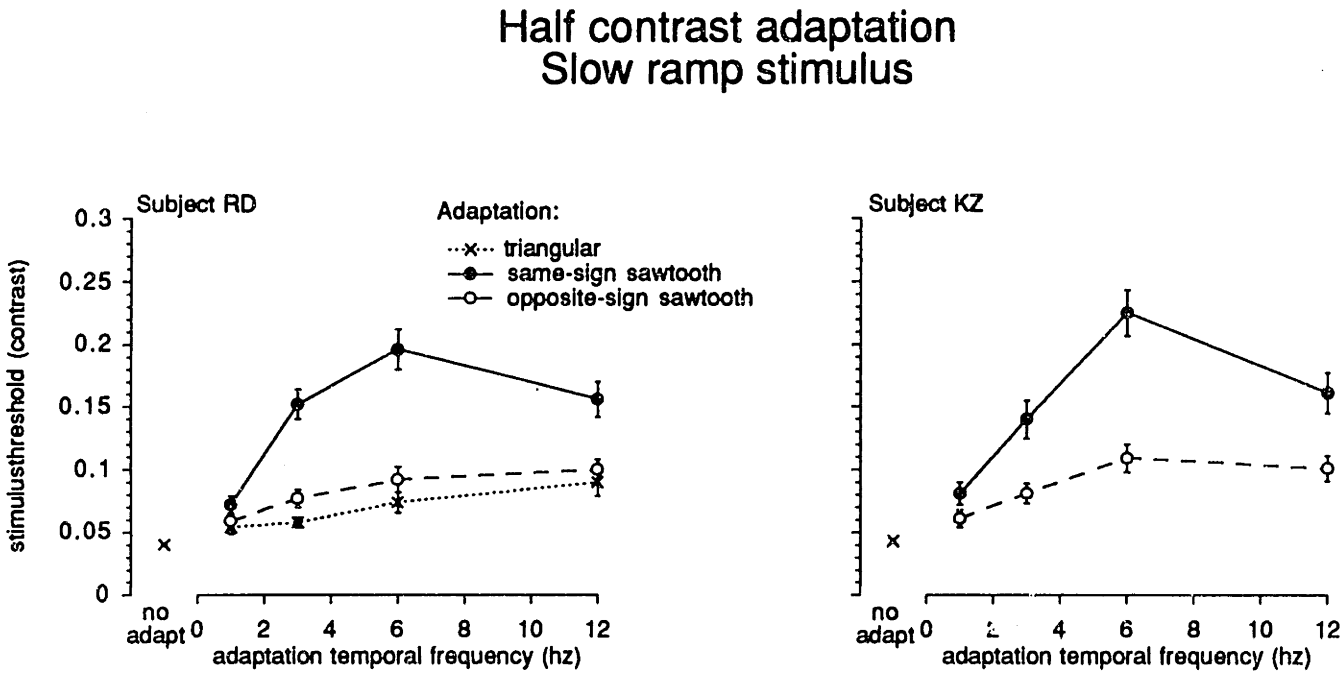
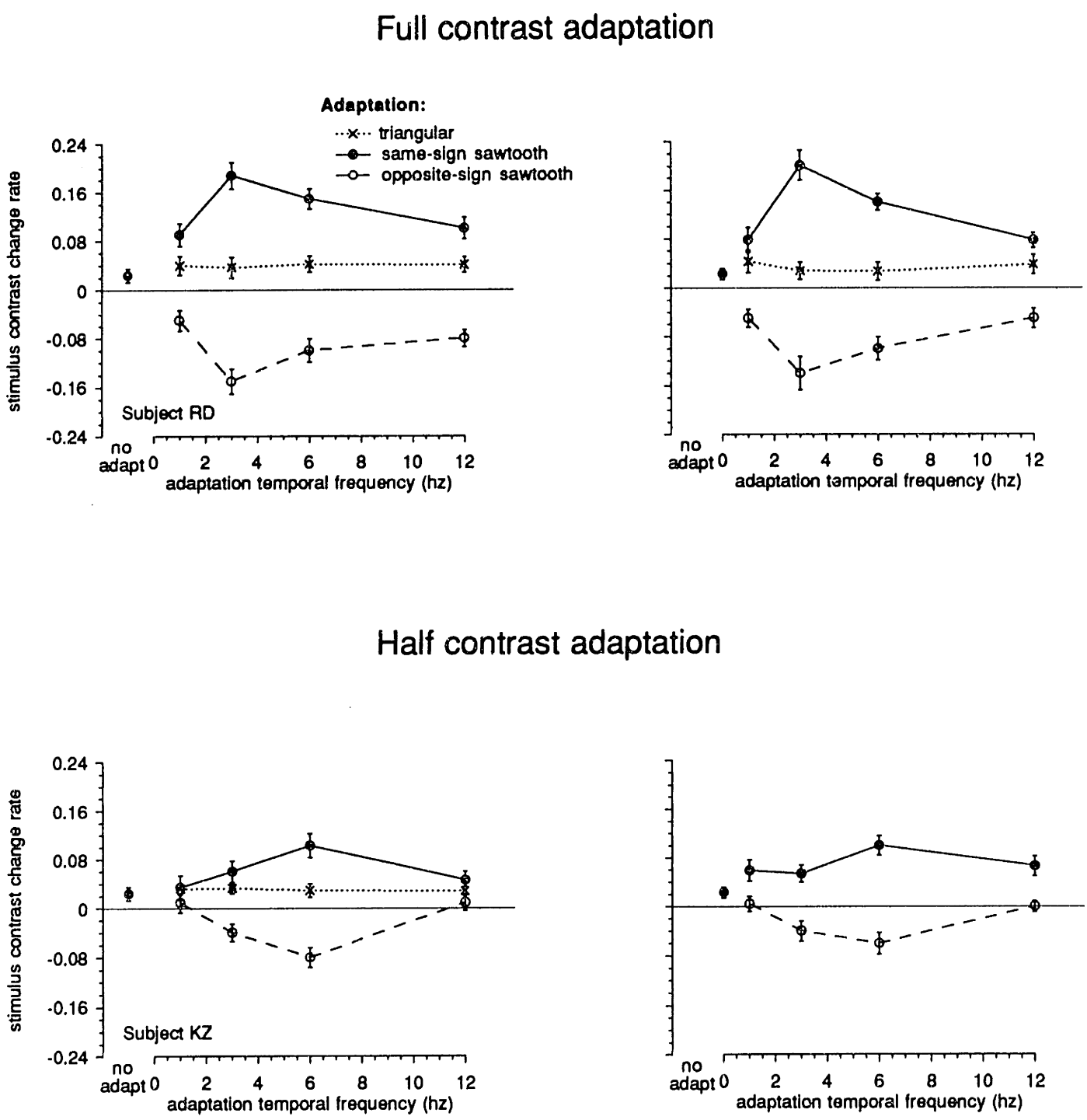

Half contrast adaptation
Slow ramp stimulus
Subject RD
Adaptation:
triangular
same-sign sawtooth
opposite-sign sawtooth
stimulusthreshold (contrast)
0.3
0.25
0.2
0.15
0.1
0.05
0
no adapt
0
2
4
6
8
10
12
adaptation temporal frequency (hz)
Subject KZ
no adapt
0
4
6
8
10
12
adaptation temporal frequency (hz)

Figure 5


Full contrast adaptation
Adaptation:
triangular
same-sign sawtooth
opposite-sign sawtooth
stimulus contrast change rate
0.24
0.16
0.08
0
-0.08
-0.16
-0.24
Subject RD
no adapt
0
2
4
6
8
10
12
adaptation temporal frequency (hz)
Half contrast adaptation
Subject KZ