Visual Motion Aftereffects:
A Review

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Major Area Paper

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Dear Committee Members,

Well, here it is. Yes, it's obnoxiously long and I apologize for that. I did cut down some of the longer more tedious sections, so you can imagine what it was like before. (The longer, more detailed version is available upon request. The main sections which got shortened were the direction selectivity of adaptation and the intro section on effects of various stimulus parameters. There may be a few others that underwent less drastic revisions.) Anyway, if you find it too long and overwhelming to read in its entirety, I would be glad to meet and help explain some of the more complicated sections. I do actually have some valid reasons for making this so long, and probably some not so valid ones as well. One reason is my obsessive nature, which compels me to try to explain everything about MAEs and put it all together. For some subjects that would be possible in a review paper; for MAEs it's an impossible task. Another reason is that the issues relating to MAEs are all so interrelated that it's really impossible to discuss some of the topics without reference to others, which means those secondary issues must be explained as well. And as you'll find from reading this, there are quite a few aspects of MAEs which are as yet unaccounted for. Yet another reason I wanted to make this as complete as possible is that I am hoping to publish it in some form, and I think what's needed in the literature is a paper which really covers the topic thoroughly, not one which addresses one aspect of MAEs while ignoring others which have impact on interpretations of that aspect. If you have any suggestions as to additions or modifications I might want to make before submitting it, I'd love to hear them. I'm sure most of that will come out at my meeting, though! (Maybe it would be better as two separate papers: one on the physiological results and the failure of the model to account for such things as storage, and another on the psychophysical evidence regarding the ratio model and the characteristics of MAEs.) Anyway, believe it or not I've enjoyed writing this, so I hope you enjoy reading it. Meeting day is April 18th, at 2 pm. Let me know if you have any comments or questions before then.

Thanks,

Errata Affecting Content

p. 13m - conventional "MAEs" -> "CMAEs"
p. 11b - "tested eye occluded" -> "adapted eye occluded"
p. 35b - "low N" -> "high N"
p. 87t- change second "rapidly" to "gradually"
p. 120b - "rivalry" -> "MAE" (oops!)

Other Errata

References - Schiller, Finlay & Volman (1976b) should be Finlay, Schiller & Volman (1976)
p. 5m - "techniques are" -> "techniques were"
p. 6m - "evidence suggested" -> "evidence suggesting"
p. 7m - Wiesenfelder & Blake reference goes after "studies of binocular rivalry"
p. 8m - no comma after linear motion
p. 8b - "even though there" -> "even though light"
p. 9t - Smith and Over "found" -> "found that"
p. 10b - "large" -> "major"
p. 11m - "literature would" -> "literature would be"
p. 15b - "'...as'" -> "since '...as'"
p. 16b - "an MAE" -> "a MAE"
p. 18b - "blinding" -> "blinded"
p. 22b - "1977;" -> "1977"
p. 25t - "difference" -> "distinction"
p. 25t - take out one "representing"
p. 28m - "deactivation" -> "Deactivation"
p. 30b - "done" -> "conducted"
p. 31t - "et al" -> "Fiorentini and Bisti"
p. 31m - "Fiorentini and Bisti" -> "et al"
p. 45t - "especially be" -> "be especially"
p. 45b - comma after "investigators"
p. 50b - "behind" -> "underlying"
p. 53t - take out "or at least...contradict"
p. 58m - "area 18" -> "area 18 cells"

p. 60m - "and 18 cells" -> ", area 18 cells"
p. 61b - "luminance threshold" -> "luminance threshold following adaptation"
p. 67t - "1975N" -> "1975a" (this occurs twice)
p. 76m - remove the "did" after "large (10-degree) shift"
p. 77b - "when" -> "with" and add "added" after "motion" in last line
p. 80m - after "passage of time", change "and" to "a mechanism which"
p. 89b - add "0.50 or" before "0.75"
p. 89m - remove unfinished sentence of paragraph!
p. 90b - comma after "say, to the right"
p. 91m - add "and relative" after "both absolute"
p. 99t - add "However," before "Absence", and "Absence" -> "absence"
p. 101t - remove one "that" from line 6
p. 102b - period after "1987)"
p. 103t - "compared MAE" -> "compared MAEs"
p. 109m - "lend" -> "lends"
p. 114m - add "the" before "retinal motion signal"
p. 120m - remove extra period after "aftereffects"

Acknowledgments

Many thanks to my advisor, Randy Blake, for comments on earlier versions of this paper, for getting me to this point, and for trusting me to write this without constant consultation and despite the rarity of my presence in the department during normal hours. (I'm also grateful to Randy for finding me a computer to use at home, and to Dr. Margolin for not using it so that I could.) Thanks also to Jeff Schall for helpful discussions, for comments on an earlier draft of this paper, and for his bountiful supply of physiology articles. I am indebted to Mollie Maginn for countless trips to the library and copy machine to get me mass quantities of articles, and for not even griping about it (at least not in my presence). I thank my friends for putting up with my ridiculous and busy work schedule, and for keeping me sane and entertained while I was not working. And of course, thanks to Mishka for tolerating my crazy schedule, for companionship while I slaved away at all hours of the night, and most of all, for quickly learning to walk across my desk and keyboard without stepping on any keys (well, most of the time anyway). During the writing of this paper I was supported by an NSF Graduate Fellowship; by Good&Plenty, chocolate peanut butter cookies, various other junk food, Ensure Plus, macaroni & cheese, NoodleRoni, and frozen dinners; and by friends and family.

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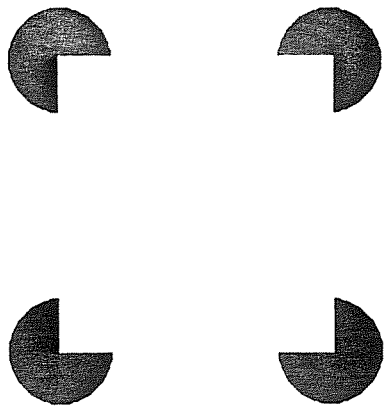

Figure 1. Common example of a stimulus which demonstrates the phenomenon of subjective contours. The presence of a complete square occluding the four disks should be compelling, even though the sides of the square are defined only by the collinear contours of the "pacman" figures.

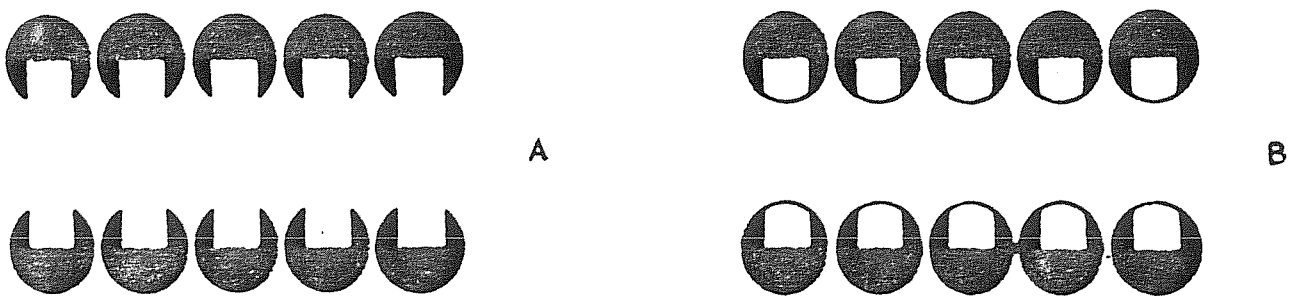

Figure 2. Stimuli used by Smith and Over (1979)√ in their study of motion aftereffects following adaptation to subjective contours. MAEs were observed following adaptation to motion of the grating depicted in (A), but not following adaptation to the grating in (B), which does not evoke the perception of subective contours. The MAEs observed after adapting to stimulus (A) were similar to those produced by an equivalent real motion stimulus.

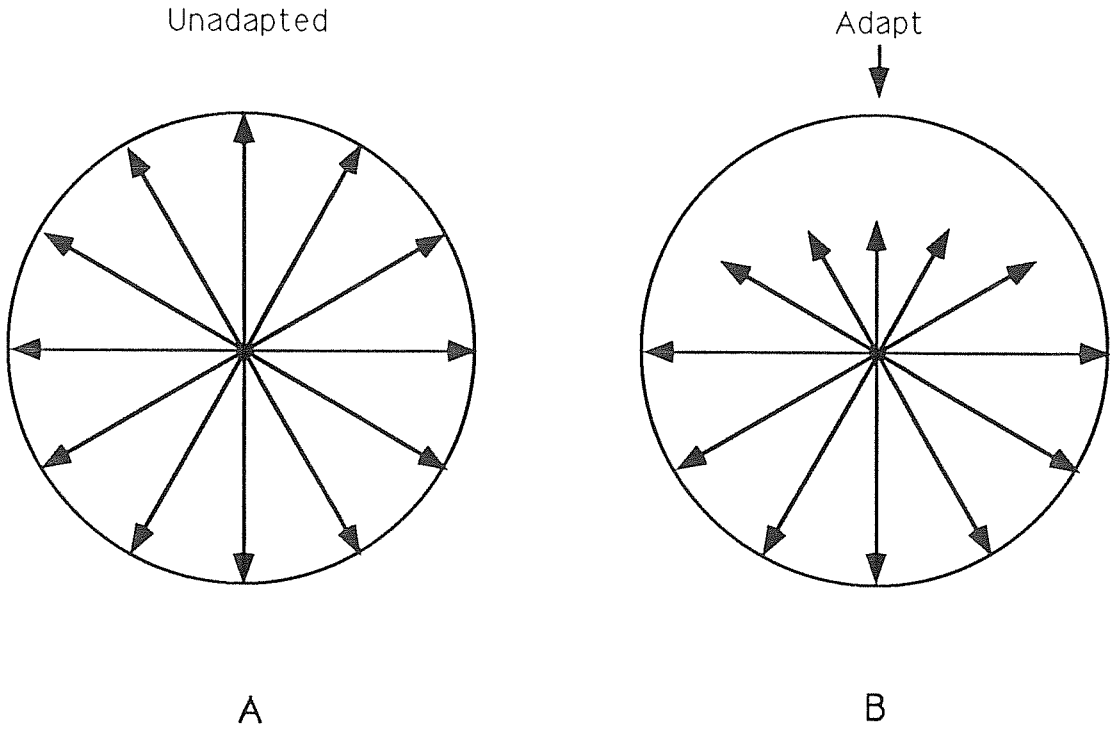

Figure 3. Diagrams representing hypothetical distribution of responses of cells selective for different directions of motion. These distributions represent the assumptions of an extended version of the ratio model of MAE generation: the overall distribution of directional responses, rather than the relative responses of opposite directions, determines the direction of motion perceived. The number of arrows shown was deemed sufficient to illustrate the changes in the response profile, and should by no means be taken as representing the actual number of direction-selective channels. (A) In the unadapted condition, spontaneous activity of direction-selective cells representing all directions of motion is equal. Thus, when a stationary stimulus is viewed, there is no imbalance in responses among the various directions, and motion is not perceived. (B) Following adaptation to upward motion, however, responses to upward motion are reduced. Responses to similar directions are also diminished, although the magnitude of the decrease is lessened for directions farther from the adapted direction. In this diagram I have depicted the adaptation effects as altering responses to directions within 90 degrees from the adapting direction. While this value is not completely arbitrary, it is used here as only a representative example; in actuality individual cells may show broader or narrower directional tuning. Note that the response to upward motion is now substantially lower than that to downward motion. Responses to similar directions are decreased as well, but in a symmetrical manner, so that the net imbalance is in favor of the downward component. If you imagine each individual response vector as consisting of horizontal and vertical components, it becomes apparent that the reductions in response in the horizontal direction are counterbalanced, so that the only unbalanced decrease is in the upward direction. Thus, the response profile shown would correspond to apparent motion in the downward direction when the stimulus is in fact stationary, due to the imbalance in activity of upward- vs. downward-selective cells.

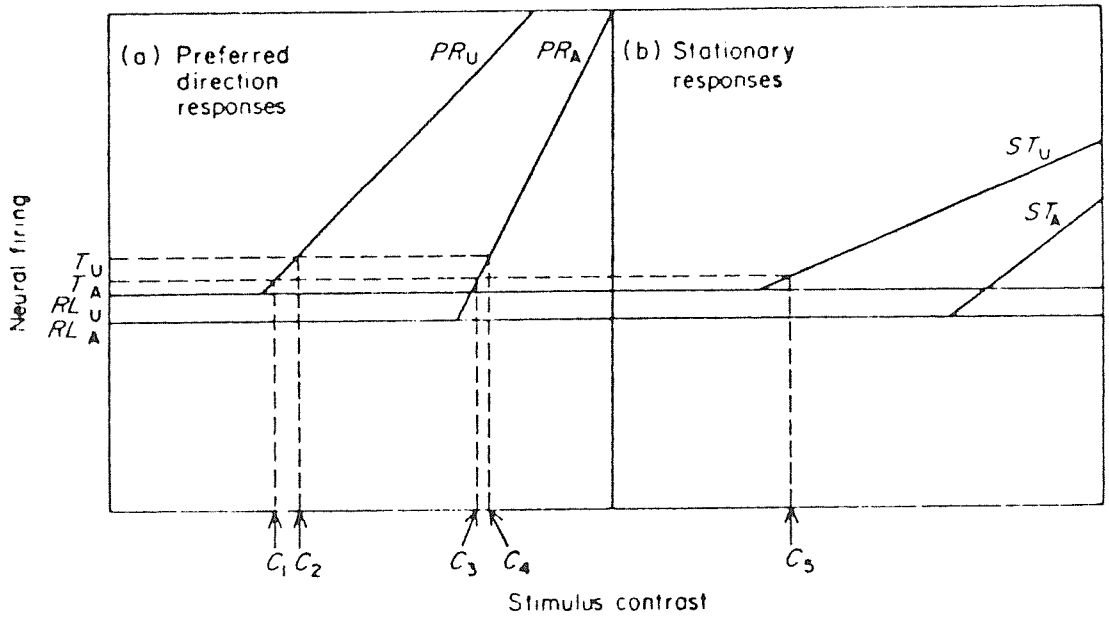

Figure 4. Moulden & Mather's (1978) interpretation of the ratio model, which takes into account only opposite directions of motion. Thus, adapted units are units adapted to the preferred direction of motion; unadapted units are selective for the opposite direction of motion and are therefore not adapted. The curve labeled PR_U represents the response of an unadapted unit to motion in the preferred direction. The curve labeled PR_A shows the similar response for an adapted unit, and reflects the decrease in response to preferred motion following adaptation. Note that the adapted response curve is both shifted to higher contrasts (indicating a type of gain control), and increased in slope, relative to the unadapted curve. RL_U is the spontaneous firing rate for an unadapted unit; the rate for an adapted unit is RL_A. RL_A is lower than RL_U, reflecting the fact that adaptation decreases maintained discharge. The responses of unadapted and adapted cells to stationary stimuli are shown by the curves labeled ST_U and ST_A; again, the adapted response is reduced relative to the unadapted response, with both a shift to higher contrasts and an increase in slope. The threshold firing rate for an unadapted unit is T_U. This is the firing rate at which motion will be signalled; it exceeds the resting rate by a fixed proportion. Likewise, the threshold for an adapted unit is T_A; Note that the absolute threshold firing rate is lower for an adapted unit than an unadapted unit. However, the contrast required to achieve that lower threshold is higher, as it is derived from the adapted response curve, PR_A. The contrast corresponding to threshold for an unadapted unit is C_2, which is quite a bit lower than that for an adapted unit, C_4. C_4 represents the contrast at which the firing rate in the adapted unit exceeds the unadapted unit's resting level by the fixed proportion required for motion detection. While the threshold contrast is increased following adaptation, the threshold ratio of firing rates of adapted and unadapted units is unchanged. C_1 is the contrast required for an unadapted unit stimulated by motion opposite to the adapting motion to exceed the adapted unit's resting level by the threshold ratio; thus a decrease in threshold for motion opposite to the adapting motion should obtain. C_5 is the threshold contrast at which the response of an unadapted unit (selective for motion opposite the adapting motion) will exceed the adapted unit's resting rate by the threshold ratio. Thus, this is the minimum contrast at which a MAE will be observed following motion adaptation. Finally, C_3 is the threshold contrast for motion following adaptation to motion in both the same and opposite directions. It represents the contrast required for an adapted unit to exceed the resting level of another adapted unit (selective for opposite motion) by the threshold firing ratio. The ratio model can thus explain the finding that the threshold elevation resulting from adaptation to opposite directions of motion (C_3) is nearly identical to that produced by adaptation to one direction alone (C_4), as demonstrated by Sekuler, Lehr, Stone & Wolf (1971).

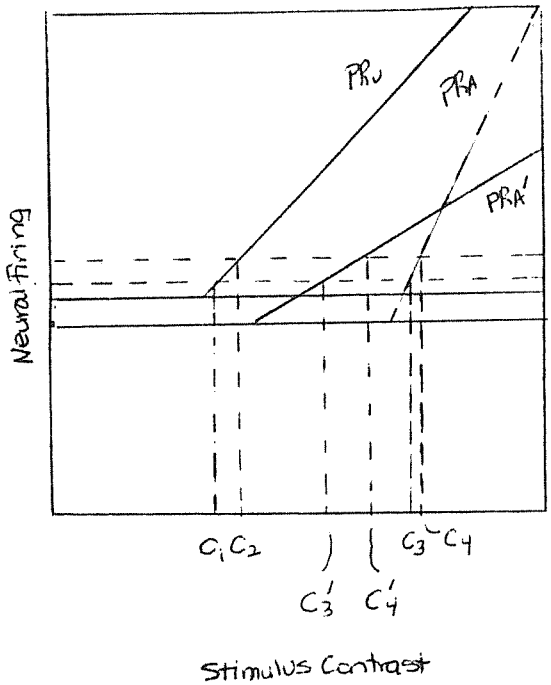

Figure 5. Modification of Moulden & Mather's (1978) Figure 1, showing the effect of varying the parameters of the adapted response curve (PR_A). In the original figure, the threshold elevation resulting from adaptation to opposite directions of motion is nearly identical to that produced by adaptation to one direction alone, as shown by the threshold contrasts C3 and C4. However, it is demonstrated here that with an adapted response curve of lesser slope, the difference between these two measures of elevation is increased, such that threshold elevation following adaptation to both directions (C_3') is decreased relative to that produced by unidirectional adaptation (C_4').

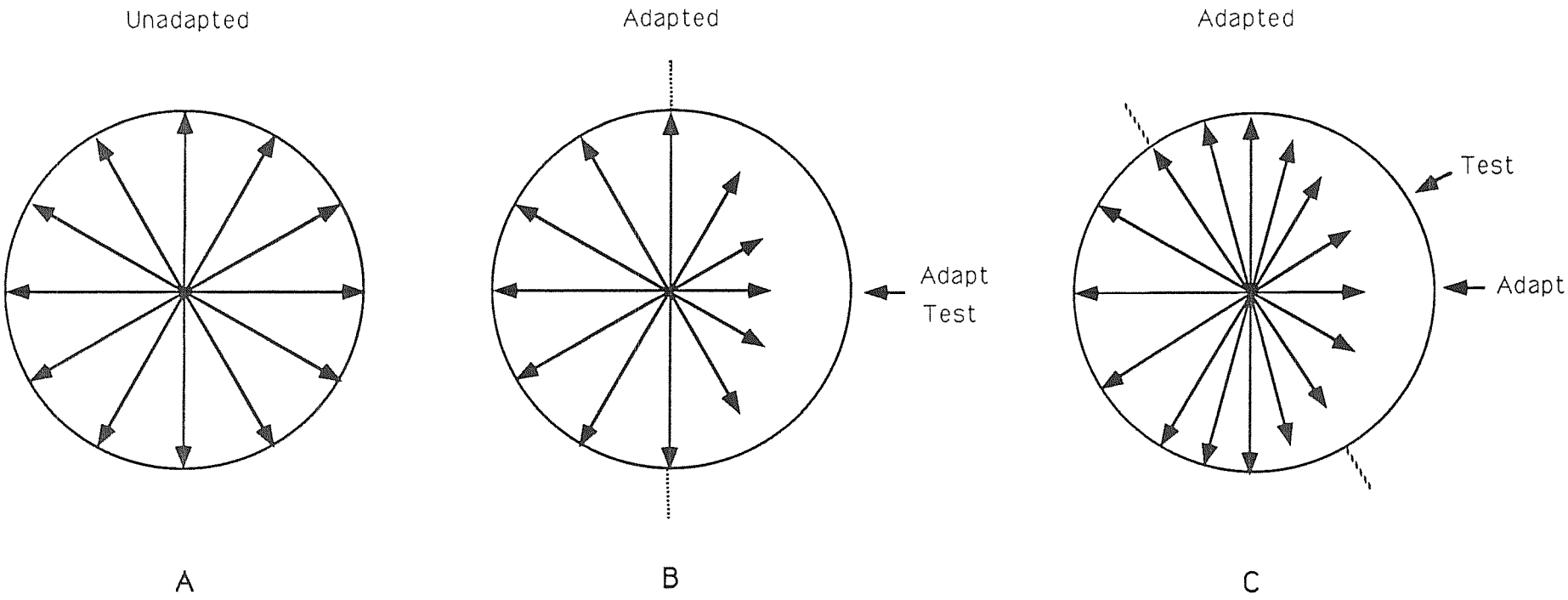

Figure 6. Response profiles accounting for the pattern of perceived direction shifts described by Levinson & Sekuler (1976). (A) In the unadapted state, responses to all directions of motion are equal, as in Figure 3. (B) Following adaptation to rightward motion, the response to rightward motion is decreased, as are responses to similar directions. Note that while cells selective for a range of directions are adapted, the decreases in responsiveness are symmetrical with respect to rightward motion. Thus, when rightward motion is subsequently viewed, no imbalance in responses relative to rightward motion exists, so no misperception of direction occurs. (C) Now suppose we adapt to rightward motion, but test the perceived direction of motion 30 deg counterclockwise from rightward. (It may help to focus your attention solely on the half of the diagram to the right of the dashed line, as only cells selective for these directions are involved in detecting the test motion.) In this case, there is an asymmetry in the responses of the cells involved in detecting the test motion: responses of cells with preferred directions counterclockwise from rightward are higher than those with preferred directions clockwise from rightward. Thus rightward motion is actually perceived as being counterclockwise from rightward, indicating a shift away from the adapting direction, as demonstrated by Levinson and Sekuler.

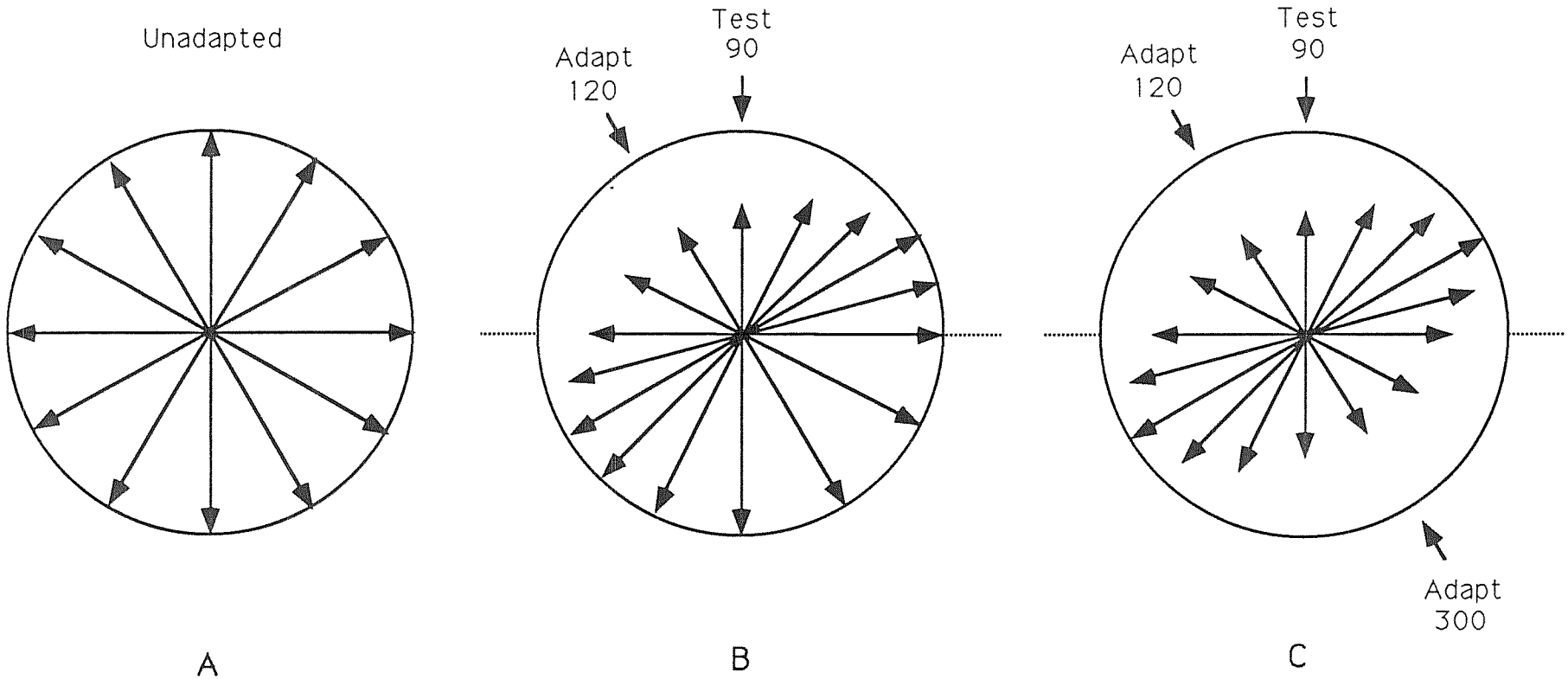

Figure 7. Response profiles demonstrating pattern of perceived direction shifts obtained psychophysically by Levinson & Sekuler (1976) for adaptation to opposite directions of motion. Rightward motion corresponds to a direction of 0 degrees. (A) Unadapted response profile, as in Figures 3 and 7. (B) Adaptation to motion at 120 deg (30 deg CCW from upward) is followed by assessment of perceived direction for upward motion (90 deg). The situation is essentially identical to that illustrated in Figure 7c, with the relevant test directions being those in the upper half of the diagram. Upward motion is perceived as being slightly CW from upward (away from the adapted direction), due to the imbalance in responses relative to upward motion which favors CW motion. (C) Simultaneous adaptation to opposite directions of motion (120 deg and 300 deg) produces identical, symmetrical reductions in response, yielding the response profile shown. When upward motion is subsequently viewed, the situation differs little from the previous condition. Again, the directional responses relevant for the perceived test motion direction are those above the horizontal meridian of the profile. The only difference is in the region CCW of rightward (0-30 deg), which shows slightly reduced responses relative to (B). Thus, the shift in perceived direction is essentially the same as in (B), as reported by Levinson & Sekuler.

MOTION AFTEREFFECTS: DESCRIPTION OF PHENOMENON

If motion in a single direction is viewed for a prolonged period, a subsequently viewed stationary pattern will appear to move in the opposite direction. This phenomenon, called the motion aftereffect (MAE), is frequently reported following viewing of a waterfall, with other portions of the landscape appearing to move upwards when viewed after the waterfall. Thus the MAE is also referred to as the "waterfall illusion". In recent times, the MAE is probably better known in relation to movie credits. After watching movie credits roll upward for up to several minutes, the stationary credits shown at the end frequently appear to be moving downward. This effect is often very vivid, although the observer is still aware that these final credits are stationary, so the MAE is not identical in appearance to real motion. The waterfall illusion involves linear, or unidirectional, motion. In a variant of the MAE, if a spiral is viewed rotating, say, clockwise and expanding, a subsequently viewed stationary spiral will be seen to rotate counterclockwise and to contract. The spiral MAE (often called the SAE) is generally much more compelling and easier to produce than the linear MAE, and indeed much research has focused on this type of MAE. Spiral MAEs also generally persist longer than do linear MAEs. Both of these aftereffects tend to decay within a short period of time (seconds or minutes), so that the vividness or magnitude of the phenomenon decreases over time until it is no longer apparent. But as will be apparent in later sections, there are some rather drastic exceptions to the rule that MAEs decay rapidly.

The motion aftereffect is a well-known phenomenon with a long history. It was discovered as early as the time of Aristotle, who published the first known report of the phenomenon in his treatise on Dreams (*Parva Naturalia*). Wohlgemuth (1911) published a detailed review of all work conducted in relation to MAEs up until that time, and described a large set of his own experiments extensively investigating the characteristics of the MAE. The MAE has continued to be a popular topic of research, and recent physiological findings which suggest a possible neural basis for the MAE render it an even more timely subject of study.

Much of the work on MAEs in the past few decades has revolved around its use as a diagnostic tool in clinical populations. The use of MAEs, specifically spiral MAEs, in clinical research was widespread in the 1950's and 1960's. Holland (1965) and Over (1973) provide a review of work in the field during that time. These studies attempted to relate differences in subject population ("organic" patients vs. "nonorganic" patients vs. controls) to differences in magnitude, duration, or mere presence of spiral MAEs. Overall, however, consistent findings were not obtained. Studies addressing the use of MAEs in distinguishing different patient populations do continue, however, with studies reporting such findings as correlations between psychoticism scores and spiral MAE duration, (Rawlings, 1985), low spiral MAE durations in grossly obese patients, who also showed more anxiety and an immature sense of self (Rydén & Danielsson, 1983), positive correlations of duration with introaggression, depressive inhibition, and anxiety, and negative correlations with repression and projected introaggression (all as measured by the Defense Mechanism Test), and a higher prevalence of disavowal in subjects at the extremes of the distribution of MAE durations (Andersson & Bengtsson, 1985), and lower spiral MAE durations in subjects with high intolerance of ambiguity (Srivastava, 1987). McFarland (1985) provides a recent review of the use of the spiral MAE in assessing brain damage. A previous study (Spitz & Lipman, 1959) also found that mentally retarded subjects are less likely to report observing MAEs, and that those who do not tend to have lower IQs than those who do.

Current work tends to focus on the neural mechanism of motion perception and of MAE generation. A plethora of both psychophysical and physiological studies have been undertaken with the goal of determining the neural correlate of the MAE. The relevant physiological work has been conducted using animal models, generally cat and monkey. It is thus important to assess the extent to which human and animal vision are similar, and in particular to determine whether the physiological processes thought to underlie MAEs in humans might have analogous functions in the animal models used to investigate them. One might therefore wonder whether only humans see MAEs, or whether other animals might perceive them as well.

Scott & Powell (1963; see also Scott, Jordan & Powell, 1963) trained a 3-year old rhesus monkey to discriminate expanding vs. contracting stimuli. They then investigated the effect of adaptation to motion on the responses of this monkey to subsequent stimuli which expanded or contracted slowly. The shifts in performance were in accord with the predictions made on the assumption that the monkey would experience MAEs, and the pattern of results was very similar to that found in an analogous experiment (Scott, et al, 1963) which employed human subjects. This finding is especially important in light of the fact that all of the physiological work conducted on the MAE to date involves nonhuman animals, with much of the investigation being centered on cat and monkey visual systems.

In the current paper I review both the psychophysical and physiological literature relating to MAEs and their neural basis. It is a rather substantial field, however, so in many cases it will be necessary to limit the discussion to specific issues, and to discuss only a sample of the relevant studies. I by no means claim to present a complete review of the MAE literature, as the topic is so broad that it would be inconceivable to attempt to cover the whole subject in a single review paper. Hopefully the information presented here comprises a comprehensive overview of the field, which will both provide the reader having general interests with a broad exposure to various issues in our understanding of MAEs, and provide a general review and a reference base for those interested in more specific aspects of MAEs.

I begin with a description of the effects of various stimulus manipulations on the strength of the MAE, followed by a discussion of some general characteristics of MAEs and a few associated phenomena. Next I will briefly discuss some of the evidence used to determine the general level at which MAEs arise. Then I will examine in more detail the physiological studies which have been conducted with the goal of determining the neural concomitants of the MAE, and address some of the issues involved in interpreting these studies. Following this elaboration of the relevant physiological findings, I will describe the similarities between the MAE and the cellular processes believed to underly it, as a means of assessing the feasibility of this proposed relationship as a "linking proposition" connecting the realm

of the phenomenal with that of the physiological. Next I will describe the prevailing model for the basis of MAEs, and review the relevant psychophysical evidence in order to further evaluate the model.

In the next major section, I discuss the phenomenon of storage of MAEs, and discuss the problem this effect poses for the current model of MAEs. This section also contains a discussion of related characteristics of MAEs, such as the role of a surrounding region in generation of MAEs. Unfortunately very few experiments relating to a physiological explanation of storage have been conducted, but I will address the significance of the relevant physiological findings to an explanation of storage and of MAEs themselves. Finally, I will discuss the role of eye movements in MAEs, and present a summary of our understanding of the MAE as it stands currently.

Stimulus parameter effects

Much research on MAEs has been designed to assess the effects of certain stimulus manipulations on the strength of the MAE. There are several methods for measuring MAE strength. The most common involve measures of duration, whereby an observer merely signals the end of the MAE either verbally or by pressing a button. A magnitude estimation task can also be used to measure initial MAE magnitude, or to continuously assess both strength and duration of the MAE. Subjects are asked to estimate some characteristic of the MAE, generally velocity, on a scale from 1 to 10, with readings taken over an extended period in order to measure the decay of the MAE as well as obtaining a measure of duration.

Another procedure involves nulling the MAE: a low-velocity moving stimulus is presented moving in the direction opposite to the MAE, and the subject continuously adjusts the grating velocity so that it appears stationary. Thus a record of apparent velocity over time is obtained. A comparison procedure can also be employed, which requires the observer to adjust the velocity of a comparison stimulus until its velocity is equivalent to the MAE velocity.

One unique method is a variant of the nulling procedure. It involves viewing a pattern of concentric circles which are successively expanding or contracting at low velocity. The subject's task is simply to report whether the circles appear to be expanding or contracting. A

variety of such patterns are presented, with variable direction and velocity, so that the apparent velocity of the MAE can be inferred from the velocity at which the circles either appear stationary (if that response is allowed) or are judged to be expanding on half of the trials. Thus it is not a direct nulling procedure, as the subject does not control the nulling motion; the theoretical basis is identical, however. The apparatus used for this procedure is called a metamethograph; it was used in the studies of MAEs in monkeys discussed above.

A recently developed method is also a nulling procedure of sorts. It employs counterphase gratings: a combination of two gratings moving in opposite directions, which appears to flicker and reverse phase rather than move. When the contrast of one component is decreased, however, it appears to drift in the direction of the higher contrast component. Following adaptation, a counterphase grating appears to drift in the direction opposite the adapting direction. Thus, it is possible to adjust the contrast of one grating so that the MAE is just cancelled by the apparent motion of the counterphase grating, providing another measure of MAE strength.

Most of the studies to be discussed in this paper used simple measures of duration and/or magnitude; when other techniques are employed, the specific procedure will be reported.

Illumination Increasing stimulus illumination overall may have little effect on MAE strength (Cords & von Brücke, 1907; Wohlgemuth, 1911; Day, 1957; Holland, 1958; Pickersgill & Jeeves, 1958; Buckingham & Freier, 1985). However, increasing luminance of only the adapting stimulus was shown to increase the MAE (Kinoshita, 1909; Ross & Taylor, 1964). Increasing test luminance, however, produces a reduced MAE (Szily, 1905; Ross & Taylor, 1964; but see also Wohlgemuth, 1911). This latter finding is likely related to the phenomenon of storage, which will be discussed later.

Contrast Increasing the contrast of the adapting target usually results in a longer and stronger MAE, although this effect saturates at a low contrast (0.03, Keck, Palella & Pantle, 1976). Test contrast has the reverse effect: MAEs are best at low contrasts, and the effect is present for a larger range of contrasts. Like the analogous result with varying luminance, this effect is probably related to the phenomenon

of storage. Also, increasing the contrast in the middle of the test period causes a weakening of the MAE, whereas decreasing it strengthens it (Keck & Pentz, 1977).

Spatial frequency The MAE is specific for the spatial frequency (SF) of the test stimulus relative to that of the adapting stimulus (Over, Broerse, Crassini & Lovegrove, 1973; Brigner, 1982). In addition, Brigner and Berks (1985) showed that removing the high SFs from the test spiral by blurring the target results in a longer MAE than when the SF composition of the stimulus is not altered. A nonsignificant trend in the same direction was found for removing the high SFs from the adapting spiral. They conclude that high SFs diminish the MAE.

Velocity Studies have generally shown that as the velocity of the adapting motion is increased within a limited range, the velocity and magnitude of the MAE increase as well; at higher velocities however, a decrease in MAE strength is observed (Kinoshita, 1909; Wohlgemuth, 1911; Pickersgill & Jeeves, 1958; Taylor, 1963a; Scott & Medlin, 1962; Buckingham & Freier, 1985; Brigner, 1986). However, other reports have found no effect of velocity (Johansson, 1956; Holland, 1958), or only a decreasing effect (Goldstein, 1957). There is also evidence suggested that the effect of adapting velocity is spatial frequency-specific (Over, et al, 1973), suggesting that temporal frequency (TF) may be the critical factor, rather than velocity. Other researchers have reported effects of TF on MAE strength (Pantle, 1974; Moulden, 1974), and it is likely that both velocity and TF are influential as determinants of MAE magnitude.

Adaptation duration The general consensus is that MAEs exhibit higher apparent velocity and longer persistence when the adaptation duration is increased to a limiting level, at which saturation occurs (Cords & von Brücke, 1907; Kinoshita, 1909; Holland, 1958; Bakan & Mizusawa, 1963; Sekuler & Pantle, 1967; Strelow & Day, 1971; Lehmkuhle & Fox, 1975; Wiesenfelder & Blake, 1990). Most reports find this limit to be on the order of 30-120 seconds, although reports of increases with longer durations exist as well (Pickersgill & Jeeves, 1958; Taylor, 1963a; Hershenson, 1989). Some investigators failed to find a significant effect of adapting duration, or have reported the

reverse effect (Goldstein, 1957). If the adapting motion is presented intermittently, the MAE is decreased, and may even be weaker than it would be if it were actually presented for the same amount of time in a continuous manner, suggesting that the MAE decays between intermittent presentations (Holland, 1962; Lehmkuhle & Fox, 1975; O'Shea & Crassini, 1981).

Stimulus size Increases in the size of the target generally produce increases in the magnitude of the MAE (Granit, 1927; Pickersgill & Jeeves, 1958), although there are also conflicting reports (Holland, 1958). However, the effect of varying stimulus size by manipulating viewing distance is controversial. Granit (1928) reported that the MAE decreases when the size is increased by decreasing distance. Freud (1964), however, found that this manipulation did increase the MAE. There may thus be a complicated interaction between these two variables. This idea is supported by Costello's (1960) finding that if distance is decreased with visual angle kept constant, the MAE tends to be reduced. This author (Wiesenfelder & Blake, 1990, 1991) has observed that one disadvantage of using the small targets required for studies of binocular rivalry is that the MAEs observed for such targets are weaker and harder to obtain than those with larger targets. It thus seems clear that when target size is increased independent of distance, MAEs are enhanced. Note, however, that in the case of an adapting target consisting of the entire visual field, MAEs are seldom observed (Aitken, 1878; Wohlgemuth, 1911). Likewise, Aitken (1878) reported that if the test target is much larger than the adapting target, no MAE occurs following adaptation to a rotating disk.

Grating orientation Over, Broerse, Crassini and Lovegrove (1973) found that the maximal MAE occurred when the adapting and testing orientations were identical, with decreasing MAEs for differences up to 45 degrees. Beyond this value, MAEs were not reliably obtained. This latter value may not be completely accurate, however, as considerable variability was present in their data. It is possible that with less variable data, substantial MAEs would be found for larger differences between adapting and test orientation.

Direction of adapting motion Mather (1980) reported that MAEs seen for a stationary random dot field following adaptation to drifting visual noise are longest for upward and downward motion, and lowest for leftward and rightward motion. The use of dot patterns rather than gratings removes the influence of orientation, which may affect measures of the variation in MAE strength with adapting direction. No other such studies are known to this author.

Eccentricity Peripherally viewed MAEs have been consistently reported to differ in both quality and magnitude from those viewed foveally. While peripheral MAEs appear more intense initially (Szily, 1905; Cords & von Brücke, 1907; Wohlgemuth, 1911), they tend to dissipate more quickly (Wohlgemuth, 1911). Granit (1928) also reported that the peripherally viewed MAE tends to end less abruptly. Peripheral MAEs have smaller durations and magnitudes (Pickersgill & Jeeves, 1958; Freud, 1964), and require larger stimuli to reach their maximal level (Richards, 1971).

Characteristics of MAE

Adapting motion

As apparent from the above discussion, MAEs can be produced by both linear motion, and spiral motion. The latter involves both radial and rotational components, as well as perceived motion-in-depth (that is, motion toward or away from the observer). There is a great deal of similarity between the two types of MAE, but there are also some basic differences between these two types of motion, the significance of which will be addressed in later sections. MAEs can also be produced by a variety of types of stimulation other than adaptation to real motion.

If a series of collinear lights is flashed in sequence, it appears as though a single light actually traverses the entire path including the space between the lights, even though there is emitted only from discrete points along the path. This phenomenon is known as apparent motion, or stroboscopic motion, and can take various forms. MAEs do result from adapting to apparent motion (Szily, 1905; Wohlgemuth, 1911; Deutsch, 1950; Anstis & Moulden, 1970; Anstis, 1980; von Grünau, 1986), although they are apparently reduced in magnitude relative to equivalent MAEs produced by real motion (Banks

& Kane, 1972). On the other hand, stroboscopic illumination of the test stimulus severely reduces or abolishes the MAE (Anstis, Gregory, Rudolf & MacKay, 1963).

Adapting to motion produced by subjective contours, also called illusory contours (Schumann, 1904), can also generate MAEs. With such stimuli, edges are defined not by real continuous contours, but by subjective contours, which are produced by collinear but noncontinuous line segments. The pattern shown in Figure 1 is a common example of subjective contours. Smith and Over (1979) found after adapting to a moving set of subjective contour stimuli (see Fig 2a), subjects observed MAEs. MAEs were not seen when the stimuli were essentially the same except for a few added thin contours to close the figures so that no subjective contours were seen (see Fig. 2b).

MAEs area also obtained following adaptation to phantom motion (Weisstein, Berbaum & Maguire, 1977; Hershenson, 1989). Phantom motion is seen within regions that do not actually contain moving contours, when these regions are adjacent to regions which do contain moving contours. For example, imagine that a large vertical grating pattern has a horizontal band covering the middle portion of it, so that the individual bars of the grating are discontinuous, as if two separate gratings are present. Now imagine that only these two separate gratings are presented to the observer. If these gratings are set into motion in the same direction, observers frequently report that they see bars moving in the middle section between the two gratings as well (Tynan & Sekuler, 1975). After adapting to such motion, if a stationary grating is presented only in the previously empty region, MAEs are also observable (Weisstein, et al, 1977). Phantom MAEs were even produced when the gratings consisted not of real contours, but of illusory contours formed by columns of X's. Similarly, Hershenson (1984) found that when subjects adapt to the upper half of a rotating spiral, an MAE can be observed on the lower half of a set of concentric circles. The recovery rate was similar to that of the normal MAE observed on the upper semicircles.

Another phenomenon involving motion in regions which do not actually contain moving contours is induced motion (Duncker, 1929). When a large annulus slowly rotates, while a central disk within the

annulus remains stationary, the disk often seems to rotate in the opposite direction. Likewise, if a large grating pattern moves behind a fixation point, the fixation point may appear to drift in the opposite direction. (The illusion that the moon seems to be moving behind the clouds is thus an example of induced motion.) Note that this effect is different from phantom motion, whereby motion of nonmoving regions is in the *same* direction as adjacent motion. Many studies have shown that adapting to induced motion can result in MAEs (Szily, 1905; Wohlgemuth, 1911; Anstis & Reinhardt-Rutland, 1976; Reinhardt-Rutland, 1981, 1983a, 1983b, 1984, 1987a). Anstis and Reinhardt-Rutland (1976) showed that an MAE can be observed for a region which exhibited induced motion during adaptation, even when the adapting region does not produce an MAE. This was determined by adapting alternately to opposite directions of motion, with the region of induced motion visible only during one direction of adaptation. They also demonstrated that an MAE can induce an opposite MAE in an adjacent region, even in the absence of induced motion during the adapting period. This was accomplished by having alternate sectors of the adapting region move in opposite directions, so that induction did not occur. In fact, as will be discussed later, induced motion is often invoked to explain characteristics of MAEs which would otherwise seem paradoxical, such as an MAE within the adapting region in the *same* direction as the adapting motion.

Especially intriguing is the recent report (Anstis, 1990) that MAEs can be observed even when no moving contours are presented during the adapting or test periods. The phenomenon involved adaptation to regions of constant dimming or brightening, followed by a shift in fixation after the adaptation period. The phenomenon is probably related to the finding that luminance modulation can produce apparent motion (Mather, 1984; Moulden & Begg, 1986); as just discussed, apparent motion can cause MAEs. Further work will be necessary to fully establish the details and implications of this phenomenon.

Contingent MAEs

A large area of research involves contingent aftereffects. These aftereffects are elicited only when the test stimuli possess certain

characteristics which were present in the adapting stimuli. Thus, an observer might adapt simultaneously to a region of horizontal stripes on a green background, and a region of vertical stripes on a red background. Upon subsequent viewing of an identical pattern without the colored backgrounds, observers usually report appropriate color afterimages depending on the orientation of the stripes. Thus the region containing vertical stripes will appear slightly greenish, while the horizontal striped region will appear slightly reddish. This effect was first discovered by McCollough (1965); thus it is referred to as the McCollough effect. A wide variety of other contingent aftereffects have also been discovered, including contingent MAEs (CMAEs). They were first reported simultaneously by Favreau, Emerson and Corballis (1972) and Mayhew and Anstis (1972). The first group of researchers in fact found that these CMAEs were still present 24 hours following adaptation. Potts and Harris (1975) subsequently demonstrated that CMAEs contingent on characteristics of the surrounding regions can even occur, with the actual moving region unchanged during the contingent adaptation conditions.

Contingent MAEs represent a complex phenomenon in and of themselves, so a detailed review of the relevant literature would beyond the scope of this paper. Yet some studies of CMAEs relate to topics of interest regarding simple MAEs, and I will therefore limit my discussion of the phenomenon to such studies.

Interocular transfer

If one eye is adapted to stimulus motion, the MAE is evident in the other eye, even with the tested eye occluded so that it is unexposed to the test stimulus. The first to make this observation was apparently Dvorák (1870), and several contemporaries confirmed this result (Exner, 1888; Szily, 1905; Wohlgemuth, 1911; Ehrenstein, 1925; Durup, 1928), as have recent investigators (Holland, 1957; Pickersgill & Jeeves, 1958; Freud, 1965; O'Shea & Crassini, 1981). Others, however, failed to find evidence for transfer (Budde, 1884). Holland (1957) reported that the duration of the transferred MAE is only 60-70% of the monocular MAE duration, and Freud (1964) also demonstrated that the transferred MAE is weaker. Pickersgill and Jeeves (1958), however, noted that while for some subjects the

transferred MAE was stronger than the monocular MAE, it was weaker for others. Physiological evidence for interocular transfer of MAEs also exists. Maffei, Berardi and Bisti (1986) found that the adaptation effects observed in both area 17 and area 18 cells of cat could be elicited either monocularly or interocularly. Thus interocular transfer of MAEs is now a commonly accepted phenomenon, although, as will be discussed shortly, the interpretation of such transfer is more controversial.

Monocular, binocular, and dichoptic MAEs

In most observations of MAEs, monocular and binocular viewing produce identical results. That is, if you adapt to a moving stimulus with either one or both eyes, the resulting MAE will be identical, except perhaps for a difference in strength, whether viewed with the adapted eye, the unadapted eye, or both. It is however possible to dissociate the monocular MAEs of the two eyes, or to dissociate the monocular from the binocular MAE, by presenting different directions of motion during adaptation, either simultaneously or in alternation.

If the two eyes view different directions of motion simultaneously, two distinct monocular MAEs can be seen following adaptation. These are known as dichoptic MAEs. Dichoptic MAEs can actually be thought of as contingent MAEs, with the direction of MAE being contingent upon the eye with which the test stimulus is viewed. Gates (1934) was the first to report the phenomenon, using spirals rotating in opposite directions. Anstis and Moulden (1970) demonstrated dichoptic MAEs for oppositely rotating sectored discs, and reported that no MAE was apparent under binocular viewing conditions; a similar result was reported by Anstis and Duncan (1983) for rotating random dot fields. Favreau (1976) has carried out an extensive investigation of dichoptic MAEs, which will be discussed in a later section.

Anstis and Moulden further showed that if the stimulus characteristics are such that motion is not seen by either eye alone, but is seen by both eyes together, an appropriate MAE results with either monocular or binocular viewing. They used a series of flashing lights, with the spatiotemporal parameters designed to produce apparent rotation when viewed binocularly, but only oscillation or

flashing when viewed monocularly. They also found that if the displays are arranged so that rotational motion is in one direction for either eye alone, but in the opposite direction when viewed binocularly, the MAE corresponding to the binocularly viewed motion is seen both monocularly and binocularly. They noted that it was not possible to determine whether the result for monocular viewing of the test target indicates that a monocular MAE was not generated, or whether it was overcome by the binocular MAE.

Anstis and Duncan (1983) reported that when the adaptation period consists of alternating periods of left eye, right eye, and binocular motion, with opposite directions for monocular vs. binocular viewing, distinct monocular and binocular MAEs can be observed. Thus MAEs can also be demonstrated as being contingent upon viewing condition in terms of ocularity. As the authors point out, however, the results do not allow us to decide whether these are truly contingent MAEs, in the sense that they exhibit characteristics of conventional MAEs, or whether they are only simple MAEs.

Influence of attention

The effect of attention on the generation of MAEs has not been unequivocably determined. Szily (1905) argued that the MAE occurs independent of conscious awareness, as subjects observed MAEs even when unaware of the adapting motion. Likewise, Wohlgemuth (1911) reported that MAEs were obtained even in the absence of conscious attention. Few such studies have been conducted recently. Chaudhuri (1990) reported that, in fact, distracting subjects by engaging them in an additional task during the adaptation period significantly reduced the resulting MAEs. When the subject was required to respond whenever a number, rather than a letter, appeared in a small window, MAE duration was reduced relative to the passive viewing condition, regardless of whether the window was fixated or not. However, requiring subjects to respond whenever the background of the moving texture pattern became a specific color did not cause a noticeable change in MAE duration.

Associated phenomena

A variety of other effects can be observed following adaptation to motion, including a decrease in perceived velocity of subsequently

viewed motion (Cords & von Brücke, 1907; Wohlgemuth, 1911; Gibson, 1937; Goldstein, 1957; Carlson, 1962; Scott, Jordan & Powell, 1963; Rapoport, 1964; Thompson, 1981; Smith, 1985), a shift in perceived direction of motion away from the adapted direction (Levinson & Sekuler, 1976; Marshak & Sekuler, 1979; Mather & Moulden, 1980), and an increase in thresholds for subsequently viewed stimuli (Sekuler & Ganz, 1963; Pantle & Sekuler, 1969; Sharpe & Tolhurst, 1973; Tolhurst, 1973; Moulden & Mather, 1978). Clarke (1974) also found that following adaptation to motion in one direction, visual evoked potentials (VEPs) elicited by same and opposite directions of motion were different.

While a complete explanation of these related effects is beyond the scope of this paper, a complete model of the neural basis of the MAE must take these phenomena into account as well. For the most part I will deal solely with the phenomenon whereby a stationary target is seen to move in the direction opposite to previously viewed motion. To the extent that these other phenomena provide evidence relevant to an explanation of this basic phenomenon, however, a discussion of these effects will be warranted.

PROPOSED LOCUS OF MAE: CENTRAL VS. RETINAL

Early proposed mechanisms of MAE production

The MAE was discussed by a variety of researchers during the nineteenth century, as summarized by Wohlgemuth (1911). Various explanations for the MAE were proposed, including eye movements (voluntary or involuntary), the fading of afterimages in the order generated by a moving target, a continuing impression of movement which causes seen objects to seem to move in the opposite direction relative to this impression (a form of induced motion), retinal blood flow, fatigue (retinal or central), and psychical or pseudo-cognitive processes.

Historically there has been a great deal of disagreement as to whether the MAE is produced retinally or more centrally. Many early investigators believed that the MAE arises due to any of a variety of retinal processes, including those listed above. However, others began to suspect that the phenomenon could not be accounted for by retinal processes alone. Oppel (1860) gave up his initial theory that the MAE

is a "reaction-activity of the eye with reference to moving retinal images," (Oppel, 1956) for the explanation that the basis of the MAE "must not be looked for in the mechanical nor in the optical apparatus, but that it lies beyond the obscure bridge which connects the retina with the sensorium." Aitken (1878) found that when the test target (a stationary disk) is much larger than the adapting target, no MAE occurs. He concluded that the locus of the MAE must therefore be beyond the retina, as factors other than retinal processes must underly this interaction between retinal locations.

Clues from interocular transfer

One factor which is commonly taken as evidence for the central locus of the MAE is the interocular transfer of the MAE. As mentioned above, when one eye is adapted, the other eye can frequently observe a MAE. Although Dvorák (1870) was the first to report interocular transfer, he still attributed the MAE to a retinal process. Exner (1888) noted that while the MAE shows transfer, an analogous effect is not present in the realm of color aftereffects. He also points out that stimulating the unadapted eye with motion in the opposite direction decreases the magnitude of the MAE, and therefore concluded that the MAE must arise beyond the retina. Szily (1905) also found interocular transfer, and reported that dichoptic viewing of opposing motions in the two eyes resulted in no binocular MAE. However, monocular MAEs could still be seen in the appropriate directions, albeit in weakened form. He concluded that the MAE must be mediated centrally. Wohlgemuth (1911) found interocular transfer, but notes that this does not necessarily mean the MAE is mediated centrally, "...as there is an after-effect in the subjective field of the closed eye, the contents of this field probably fuse with that of the open eye. As a result of this, the field of the open eye is in an agitated condition." (p. 28)

The existence of an interocularly transferred MAE is now well established (Holland, 1957; Pickersgill & Jeeves, 1958; Barlow & Brindley, 1963; Freud, 1964; Favreau, 1976; O'Shea & Crassini, 1981), as summarized earlier. However, some controversy remains as to the validity of using this finding as proof that the MAE must be centrally mediated. One of the main objections to such an argument has been

elaborated by Day (1958). He points out that since the two monocular visual fields overlap, it is conceivable that the aftereffect seen by the adapted eye is projected onto the visual field of the unadapted eye even when the adapted eye is closed, so that it appears as though the aftereffect is being seen by the unadapted eye. This occurs in the case of negative afterimages, for which there is general acceptance of a retinal mechanism (e.g. MacLeod & Hayhoe, 1974; but see also Davies, 1973). This objection is essentially the same as that put forth by Wohlgemuth (1911) almost fifty years earlier. Barlow and Brindley (1963) note that "...transfer would be explicable on the hypothesis that it is due to expression of maintained discharge even if this occurred only in the retina," (p.1347).

This claim can be addressed at several levels. One point which can be made is that negative afterimages are visible regardless of subsequent stimulation (or lack thereof) of the adapted or unadapted eye. Thus an afterimage is seen even with the eye closed, and under this condition it is in fact not possible to determine which eye was originally stimulated. The MAE, however, is usually reported as an effect specific to the adapting target or a similar stimulus. On the other hand, while current reports of MAEs do not address the issue, many early investigators discussed the observation of an MAE with the eyes closed, or on a homogeneous field without a suitable test target (e.g. Wohlgemuth, 1911). It is therefore possible that the MAE is simply projected onto the visual field of the unadapted eye, as it is projected onto a homogeneous field or onto the "subjective field" when the eyes are closed.

A strong piece of evidence for the failure of the "projected aftereffect" theory is based on the finding that the MAE can in fact be stored or prolonged if the appropriate test target is not presented immediately (e.g. Wohlgemuth 1911, Spigel 1960). That is, if the test target is not presented to the adapted eye until the duration of the usual MAE has already elapsed, an MAE is frequently still visible, and can last well beyond the end of the normal decay period. Thus, the absence of an appropriate test target immediately following adaptation somehow inhibits the decay of the MAE, so that it persists for longer than it would if testing were immediate. (This phenomenon will be

discussed in full in the section on storage.) Thus, a MAE is not a passive phenomenon such as an afterimage, which simply occurs and then decays even with the eyes closed. (Note however that there is an active component to afterimages, in that blinking can often be used to prolong the afterimage.) This represents an obstacle in acceptance of the explanation that the events underlying the MAE occur even when no test stimulus is presented to the adapted eye, and result in projection of the MAE onto objects seen by the unadapted eye.

In addition, Spigel (1964) investigated the effect of interocular testing on storage of the MAE, as a means of determining the peripheral or central locus of the MAE. Observers all adapted for 30 seconds to a rotating radial pattern. They then either remained in the dark for the mean MAE duration, had the unadapted eye exposed to a homogeneous field, or had the unadapted eye exposed to the stationary test stimulus. When this comparison is made for the adapted eye, the MAE shows storage under both conditions in which the test target is not immediately presented; that is, if a period of darkness or viewing of a homogeneous field follows adaptation, the MAE can still be elicited after a span of time equal to the duration of the normal MAE. Spigel's main finding was that even when the test target was presented to the unadapted eye, the MAE was less than occurred under the complete storage condition (darkness), and was in fact negligible. Clearly the visual stimulation in the unadapted eye is affecting the decay of the MAE in the adapted eye, indicating that retinal processes in the adapted eye are not the sole basis for the MAE. This implies that the MAE is at least partially centrally mediated.

Kalfin and Locke (1972) also employed a storage paradigm as a means of addressing the issue of interocular transfer. They reiterated Day's (1958) concern that interocular transfer could reflect the projection of the MAE in the adapted eye onto the field of the unadapted eye. They suggest, therefore, that a good way to determine whether interocular transfer truly occurs would be to test the unadapted eye after the MAE in the adapted eye has completely decayed. Subjects adapted monocularly to a rotating spiral for 15 minutes, then returned in 24 hours. Both eyes were tested, in random order with the untested eye occluded. Fifteen of 29 subjects saw the

MAE with both eyes, 9 saw it only with the adapted eye, and none saw it only with the unadapted eye. The authors conclude that since two-thirds of the observers saw the MAE in the unadapted eye (actually only one-half, or 15 of 29, since five subjects saw no MAE in either eye and can't be discounted), a central locus of the MAE is indicated.

At first glance, it is unclear how this finding differs from previous findings that MAEs are seen interocularly. No observers saw the MAE only with the unadapted eye, which would seem to be the desired outcome for concluding that projection of the adapted eye's MAE does not play a role. This fact is not surprising, in that interocularly transferred aftereffects are usually weaker than monocular aftereffects (e.g. Holland 1957, Freud 1964, Favreau 1976), a result which can be explained by the presence of cortical cells with differing ocular dominance (Hubel & Wiesel, 1962, 1968). But this does not detract from the lack of apparent support for true interocular transfer. The key here is not the use of stored MAEs, as the authors seem to suggest. Rather, it might be inferred from the fact that observers had both eyes tested in random order that some observed the interocular MAE after the adapted MAE had been tested and therefore decayed. However, the results are not presented in terms of which eye was tested first, so a definitive conclusion can not be reached. Also, the authors do not state that the MAE in the first eye tested was allowed to decay completely prior to testing of the other eye. It is unclear why the authors chose to use a stored MAE at all. It would be just as valid to determine whether an interocular MAE is present following an immediate test and decay of the monocular MAE, without needing to allow for storage. This would seem to be a critical experiment for determining if interocular MAEs can be observed without the possibility of projection from the adapted eye onto the visual field.

Further, Barlow and Brindley's (1963) finding that interocularly transferred MAEs can be observed even when the adapted eye has been completely pressure blinding prior to testing strongly supports a central role in MAE mediation. Pressure blinding is thought to abolish all retinal ganglion cell activity (Bornschein, 1958), so that the MAE must be due to adaptive processes beyond the retinal level.

PHYSIOLOGY: DIRECTION-SELECTIVE CELLS

Discovery of direction-selective cells

The discovery of direction-selective cells greatly furthered neural modles of the MAE. These cells were first discovered in cat striate cortex (Hubel, 1959; Hubel & Wiesel, 1959, 1962) and in frog retinal ganglion cells (Lettvin, Maturana, McCulloch & Pitts, 1959; Maturana, Lettvin, McCulloch & Pitts, 1960). Rodieck and Stone (1965) found that cat retinal ganglion cells do not show analogous direction selectivity. Rather, they exhibit radial selectivity, such that the preferred response is for motion either toward or away from the receptive field center, as defined by on and off regions. Stone and Fabian (1966), however, found cells that were direction-selective, even for reversed contrast stimuli. Such cells were subsequently demonstrated in the area 17 of monkey (Hubel & Wiesel, 1968; Schiller, Finlay & Volman, 1976a). Orban, Kennedy and Maes (1981a, 1981b) demonstrated that these cells also exist in both area 18 of cat, in highter proportion than in area 17. They are also found in area 19 (Orban, Duysens & van der Glas, 1980; Duysens, Orban, van der Glas & de Zegher, 1982). The existence of such cells in cortex has consistently been verified both in monkey (Wurtz, 1969; Orban, Kennedy & Bullier, 1986) and in cat (Campbell, Cleland, Cooper & Enroth- Cugell, 1968; Pettigrew, Nikara & Bishop, 1968; Henry, Bishop & Dreher, 1974; Emerson & Gerstein, 1977; Albus, 1980; Peterhans, Bishop & Camarda, 1985; Orban, et al, 1986; Hammond & Pomfrett, 1989, 1990a, 1990b; Crook, 1990; Gizzi, Katz, Schumer&Movshon, 1990).

Dubner and Zeki (1971) found that area MT in monkeys contains a high proportion of direction-selective cells. Many studies have confirmed the prevalence of direction-selective cells in this area (Zeki, 1978; Baker, Petersen, Newsome & Allman, 1981; Maunsell & van Essen, 1983; Albright, 1984; Al right, Desimone & Gross, 1984; Felleman & Kaas, 1984; Allman, Miezin & McGuinness, 1985; Mikami, Newsome & Wurtz, 1986; Albright, 1989; Rodman & Albright, 1989). Further work has suggested that these cells may be involved in processing more complex types of motion than simple linear motion. This topic will be addressed shortly. Direction-selective cells have also

been demonstrated in the cat's lateral suprasylvian area (LS), which shows characteristics similar to area MT in monkeys (e.g. Rauschecker, von Grünau & Poulin, 1987; Rauschecker, 1988).

These cells are characterized by being most responsive to a given direction of motion ("preferred" direction), the opposite of which ("null" direction) produces a greatly reduced response. In addition, responses to motion orthogonal to the preferred direction are usually negligible. The direction-selectivity of these cells is of course bandpass, so that a range of directions of motion can activate a given cell. The most extremely direction-selective cells exhibit no response at all to motion in the null direction, and may show a suppression of spontaneous activity for such motion. Hubel & Wiesel (1962, 1968), however, noted that motion in the null direction usually did not cause a suppression of spontaneous activity. Cells with less dramatic selectivity show decreased responsiveness in the null direction, yet this response is greater than that for motion in the orthogonal directions. In addition, some cells have been reported to be bidirectional: they respond equally well, and most strongly, to two opposite directions of motion, with minimal responses to motion at 90 degrees to these preferred directions. These cells were first characterized by Campbell, Cleland, Cooper and Enroth-Cugell (1968). Recent researchers (Hammond & Pomfrett, 1989, 1990b) have adopted the convention of referring to cells with minimal response in the null direction as "direction-selective", while those with less asymmetric responses to preferred and null motion (including bidirectional cells) are termed "direction-biased". Hubel and Wiesel (1959) pointed out that direction-selective cells usually do not respond to stationary stimuli. Wurtz (1969) confirmed this, demonstrating that direction-selective cells usually show only transient initial responses to stationary stimuli; thus their responses to such stimuli are very brief.

Distribution of preferred directions

Oyster and Barlow (1967) found that for on-off type retinal ganglion cells of rabbit, a distribution of preferred directions of motion showed four lobes, near the horizontal and vertical axes. For on type cells, however, only three lobes were apparent, which the authors

suggest is the minimum necessary to signal unambiguously the direction of motion.

Campbell et al (1968) reported that the distribution of preferred directions of motion for cat striate cells did not show a bias toward vertical and horizontal motion as opposed to oblique motion. Pettigrew, Nikara and Bishop (1968), on the other hand, found that simple cells with high direction selectivity tended to prefer vertical and horizontal orientations.

Subsequent investigations of area 17 cells (Payne, Berman & Murphy, 1980) and MT cells (Albright, Desimone & Gross, 1984) suggest that cells with similar preferred directions are usually grouped together, and seem to be organized in columns. The size of a preferred direction column is about half the size of an orientation column, a finding which may reflect the relationship between optimal orientation and preferred direction. That is, for a given orientation, there are two preferred directions, which may each be represent in half of an orientation column.

Direction-selectivity vs. orientation-selectivity

Note that when bars or gratings are used in studies of direction-selectivity, direction preference is actually confounded with orientation bias. (This issue will also be addressed in a later section in relation to psychophysical measures of direction-selectivity.) However, evidence suggests that this confound does not have severe consequences for the interpretation of most studies of direction-selective cells. Henry, Bishop and Dreher (1974) systematically examined the independent contributions of orientation-selectivity and direction-selectivity on cell responses, by comparing response patterns for stationary bars (which possess orientation but not direction), moving bars, and moving spots (which have direction but not orientation). The authors determined that orientation selectivity does not differ for moving or flashing bars, and that the optimal axis of motion is always orthogonal to the optimal orientation. Likewise, the optimal axis and direction of motion are the same for both moving bar and moving spot. The general direction-selectivity is unchanged with variations in axis of motion. Maunsell and van Essen (1983), however,

found that while the preferred orientation is usually perpendicular to the preferred direction, there are exceptions.

Schiller, Finlay and Volman (1976b, 1976c) also reached the conclusion that orientation- and direction-selectivity are independent characteristics of cells, based on converging evidence of several types, and suggest that these two forms of selectivity arise from distinct neural mechanisms. Eysel, Wörgötter and Pape (1987) presented strong evidence for the same conclusion, in their study of the mechanism of direction selectivity. It was possible to abolish direction selectivity without blocking orientation selectivity. (These results will be discussed in detail shortly.) Likewise, Zhang (1990) found that if a cell's response pattern is portrayed as a polar plot, a one-dimensional Fourier decomposition of the polar plot along its angular dimension can be used to dissociate the orientational and directional components of the response.

Also, psychophysical studies (Pantle & Sekuler, 1969; Moulden & Mather, 1978) have suggested the independence of direction-selective and orientation-selective mechanisms. Thus, it seems that despite the confounding of orientation and direction of motion in many studies, the results and conclusions are probably not compromised by this confounding. (In fact, for cells in MT, directional preference and optimal orientation do not always covary.)

"True" direction-selectivity

Campbell, Cleland, Cooper and Enroth-Cugell (1968) reported that direction-selectivity is independent of stimulus spatial frequency. Hammond and Pomfrett (1990b), however, found that two of four categories of cells, including the group with the largest proportion of cells, showed systematic variations in directionality with changes in spatial frequency, with some even reversing their directional preference. In addition, orientation/direction tuning was enhanced at high spatial frequencies, as measured by width of tuning.

Many studies (e.g., Schiller, et al, 1976a; Emerson & Gerstein, 1977;) have shown that some direction-selective cells change, and may even reverse, their preferred direction with changes in contrast. In fact, cells may appear direction-selective when in fact the observed directionality is purely a function of contrast, due to the presence of

"on" and "off" regions (Hartline, 1940) in the receptive field. However, Hubel and Wiesel (1959) determined that the directional characteristics of some of their cells could not arise simply from the interaction of excitatory and inhibitory receptive field regions. Maturana & Frenk (1966) found that direction-selective cells in pigeon retina exhibited selectivity which was invariant to illumination, contrast, and color, and Peterhans, Bishop and Camarda (1985) reported that 81% of simple cells exhibited the same preferred direction for both light and dark edges.

Barlow and Hill (1963a) also demonstrated the invariance of direction-selectivity for cells with changes in contrast, and delineated four characteristics of "true" direction-selective cells. In addition to showing direction selectivity, they show the same directional preference regardless of contrast, even if contrast is reversed. And their responses vary little with stimulus intensity changes and with variation in stimulus location within the receptive field.

This distinction between cells showing absolute direction-selectivity and those showing direction-selectivity dependent on contrast has led some (Emerson & Gerstein, 1977) to distinguish between "direction-selective" cells, representing the former category, and "direction-asymmetric" cells, representing the latter group. Recent work indicates that the basic mechanisms of direction-selectivity in these groups of cells may differ (Emerson & Gerstein, 1977).

Mechanism of direction selectivity

While the mechanism of direction selectivity has not yet been fully elucidated, a variety of relevant evidence exists. As mentioned above, direction selectivity of some cells can be predicted merely on the basis of the existence of excitatory and inhibitory regions of the receptive field. Generally, however, this is not the case (e.g. Hubel & Wiesel, 1959; Barlow & Levick, 1965; Bishop, Goodwin & Henry, 1974; Ganz, 1984; Ganz & Felder, 1984; Peterhans, et al, 1985).

It is currently believed that inhibition plays a major role in producing direction selectivity. Some of the evidence derives from studies examining the responses of cells to stimulation with varying spatial and temporal parameters (e.g. Barlow & Levick, 1965; Henry &

Bishop, 1971; Benevento, Creutzfeldt & Kuhnt, 1972; Goodwin & Henry, 1975; Goodwin, Henry & Bishop, 1975; Emerson & Gerstein, 1977; Movshon, Thompson & Tolhurst, 1978; Ganz, 1984; Ganz & Felder, 1984). Barlow and Levick (1965) conducted the first such investigation in the rabbit retina, and proposed that direction selectivity is due to suppression of responses to null motion, which is in turn due to inhibition. Their results supported this type of mechanism, as opposed to the alternative hypothesis involving conjunction of excitation (which would produce facilitation of responses in the preferred direction). They noted, however, that some facilitation in the preferred direction may play a minor role as well.

Other researchers have reported evidence supporting the role of inhibition in direction selectivity (Benevento et al, 1972; Henry & Bishop, 1971; Goodwin, et al, 1975; Goodwin & Henry, 1975; Emerson & Gerstein, 1977), and also found evidence for a lesser contribution of facilitation as well (Henry & Bishop, 1971; Emerson & Gerstein, 1977; Movshon, Thompson & Tolhurst, 1978). Pennartz and van de Grind (1990) describe a model for direction selectivity in rabbit and squirrel retina, which accounts for the characteristics reported by other researchers (e.g. Barlow & Levick, 1965), and correctly predicts cellular responses to stationary flashes and to moving spots or slits. This model includes a pathway for lateral inhibition, providing further evidence for the role of inhibition in direction selectivity.

Other studies have demonstrated the contribution of inhibition by investigating the consequences of application (intravenous or topical) of antagonists to the inhibitory transmitter GABA (gamma-aminobutyric acid), such as bicuculline and picrotoxin (Pettigrew & Daniels, 1973; Rose & Blakemore, 1974; Daniels & Pettigrew, 1975; Silitto, 1977; Caldwell, Daw & Wyatt, 1978). These studies have generally shown that direction selectivity is greatly reduced or completely eliminated when the activity of GABA is blocked.

Ariel & Daw (1982) studied the effect of ACh (acetylcholine, an excitatory transmitter) agonists such as nicotine and physostigmine on direction selectivity, and suggested that the increase in excitatory stimulation produced can overcome the inhibitory effects of GABA on

responses in the null direction, resulting in a loss of direction selectivity.

Eysel, Wörgötter and Pape (1987) examined the effects on cat simple cells of inactivating nearby regions, either by heat lesions, localized cooling, or GABA application. (Note the difference between applying GABA directly to direction-selective cells, as above, and applying it to cells which provide input to these cells.) Two of these approaches yielded reductions of direction selectivity without affecting orientation selectivity; cooling affected orientation selectivity also. When regions representing representing null motion towards the recorded cell's receptive field were inactivated, responses to null motion increased, but when regions representing preferred motion toward the receptive field of the recorded cell were inactivated, direction selectivity was not affected. Further work with inactivation via GABA application (Eysel, Muche & Wörgötter, 1988) showed that the results for simple and complex cells differ. Some complex cells showed increases in response to the preferred direction of motion, leading the authors to propose that an imbalance between inhibition at either end of the directional axis, rather than the existence of unilateral inhibition, determines the direction and magnitude of direction selectivity. Evidence for facilitation in complex cells was found as well; increased null responses were more prevalent in simple cells, whereas reductions in excitation were more frequent in complex cells.

Several of these studies (Caldwell, et al, 1978; Ariel & Daw, 1982; Pennartz & van de Grind, 1990) examined direction-selective retinal ganglion cells of rabbit, for which there is no analog in the primate visual system. Thus the implications of those particular studies may not be fully applicable to cortical direction-selective cells in cats or monkeys.

The effects of antagonists of inhibitory transmitters differ somewhat with cell type, and some inconsistencies between various studies exist. The role of GABA in the mechanism of direction selectivity has received strong support. But the finding that strychnine (a blocker of non-GABA inhibitory transmitters) activates simple cells suggests that another transmitter system may also contribute to

adaptation, especially in simple cells (Daniels & Pettigrew, 1975). A thorough discussion of the topic would be inappropriate here; the reader is therefore referred to the reports cited here and a variety of other investigations of the topic for further details.

Direction selective cells in MT

Pattern vs. component direction selectivity

Movshon, Adelson, Gizzi and Newsome (1985) found that cells in V1 were selective for component motion only, while MT cells could either be direction-selective for component direction or for pattern direction. That is, all direction-selective cells showed a directional preference for a single drifting grating. However, if cells were stimulated by a plaid pattern consisting of two drifting gratings of different orientation, V1 cells and some MT responded to the direction of motion of individual components, whereas some MT cells actually responded to the overall direction of the pattern rather than to the component directions. Gizzi, Katz, Schumer and Movshon (1990), however, determined that direction-selective cells in area 17 and in the lateral suprasylvian area (LS) of cat were all selective only for the direction of component motion, and not for the direction of pattern motion. This discrepancy highlights the necessity to take inter-species differences into account when interpreting the results of physiological studies.

Direction-selective cells in superior colliculus

Direction-selective cells are also found in the superior colliculus (e.g. Sterling & Wickelgren, 1969; Wickelgren & Sterling, 1969; Cynader & Berman, 1972; Mandl, 1974).

Sterling and Wickelgren (1969; Wickelgren & Sterling, 1969) examined the characteristics of cells in the superior colliculus of cats, and also assessed the influence of cortical input on the responses of collicular cells. These authors (Sterling & Wickelgren, 1969) reported that three-fourths of the 160 cells they examined exhibited direction selectivity. This is drastically different from the value of 5% determined later for monkey superior colliculus (Cynader & Berman, 1972). Unlike cortical direction-selective cells, they often showed responses to orthogonal directions of motion, with the least response always occurring for the null direction rather than orthogonal

directions. Thus orientation is not as critical for these cells as for cortical cells. Directional tuning was much broader than for comparable cortical cells, with variations of at least 30 degrees in direction of motion necessary to elicit changes in responsiveness. About 90% of these cells showed preferred directions within 30 degrees of horizontal (representing only 30% of possible directions).

Most of these cells preferred motion away from the area centralis. They suggest that this bias may be related to the role of the colliculus in coordinating eye movements, and the requirement to adjust fixation when an object moves away from, but not toward, the area centralis. Like cortical cells, the direction selectivity of collicular cells was unaffected by changes and reversals in contrast, and was unrelated to the presence of on and off subregions. In fact, these cells were different from retinal, geniculate, and simple cortical cells in that they did not exhibit discrete subregions. Most of the cells (90%) resembled hypercomplex cortical cells, showing end-inhibition, binocular input, and positional nonspecificity like their cortical counterparts. Most had receptive fields in the contralateral visual field, so that the ocular dominance distribution was rather asymmetrical. The authors found no evidence of orientation- or direction-selective input from retinal elements.

Wickelgren and Sterling (1969) employed three methods of assessing cortical input to the colliculus. Part of the cortex was lesioned or reversibly cooled, or cats were raised with one eye sutured from birth. Following cortical inactivation, no cells showed direction selectivity, with only three cells showing even slight directional preferences. In addition, 70% of the cells became exclusively or largely monocular, being responsive only to contralateral input, and spatial summation was evident. Collicular cells are normally binocular, and do not show summation. Previous studies suggested that the cortical input to the colliculus is ipsilateral, whereas the retinal input is contralateral (e.g. Altman, 1962). In addition, many more cells resembled optic nerve fibers rather than cortical cells in their response properties. Thus it is apparent that many of the distinguishing characteristics of collicular cells are derived from cortical, rather than direct retinal, input. The authors suggest that

these cells may receive input from cortical cells with specific preferred directions, to account for the bias seen in collicular preferred directions. (The anisotropies in preferred direction discussed above, however, suggest that this characteristic may already be present at the cortical level; the bias may be enhanced in the colliculus, however.) The broad directional and orientational tuning of collicular cells may be attributed to input from cortical cells with a range of directional and orientational preferences, yielding overall selectivity with shallower tuning.

Similar results were obtained by Berman and Cynader (1976). These researchers reported that direction selectivity of superior colliculus cells in cat was abolished by cortical lesions regardless of age at lesion. Like Wickelgren and Sterling, they found that the ocular dominance distribution of these cats was biased toward representation of the contralateral eye. Ogasawara, McHaffie and Stein (1984) found more complex results than did the above studies. They reported that while deactivation of areas 17 and 18 of cat minimized both direction selectivity and binocularity in the superficial layers of the colliculus, they did not affect cells in deeper lamina. deactivation of the posterior regions of the suprasylvian cortex (PSSC), however, produced equivalent modifications of response for cells in deeper layers. These results support the notion that the direction selectivity of collicular cells is derived from nonretinal input, but point out the complexity involved, as well as the probability of dual sources of this input.

ROLE OF DIRECTION-SELECTIVE CELLS IN MAE

As discussed in an earlier section of this paper, a variety of explanations for the MAE have been proposed through the years. The discovery of direction-selective cells opened up a whole new area of research, aimed at establishing whether these cells are somehow responsible for the perceptual phenomenon of MAEs. It was Sutherland (1961) who first proposed the selective fatigue of specific direction-selective cells as the basis for the MAE. He suggested that direction-selective cells might be present in the human visual system as in other animals, and that their adaptation might be the neural concomitant of the phenomenal MAE. He proposes that, "...the direction in which something is seen to move might depend upon the

ratios of firing in cells sensitive to movement in different directions, and after prolonged movement in one direction a stationary image would produce less firing in the cells which had just been stimulated than normally, hence apparent movement in the opposite direction would be seen to occur," (p.227).

This model of the neural basis of the MAE has come to be known as the ratio model, in that it postulates that the visual system determines the direction of motion seen by calculating a ratio between the outputs of cells responding to opposite directions of motion. (It is unclear whether Sutherland's original intention was to suggest ratios of responses of cells selective for two opposite directions only, or rather a comparison of responses of cells representing all possible directions. This issue will be discussed in detail later.) Following adaptation to a given direction of motion, cells selective for that direction of motion are fatigued, so that upon viewing a stationary stimulus, the signal from the opposite-direction cells is stronger than that of the adapted-direction cells. This imbalance results in the perception of motion of a stimulus which is actually stationary. MacKay (1961) concurrently suggested a similar but less detailed mechanism of MAE production, suggesting that the visual system "...at some stage incorporates detectors of motion as such, which become adapted . . . and for some time afterwards adopt a 'zero level' that is negative."

The idea that the MAE is due to fatigue was not new. The first to propose some form of fatigue or exhaustion of neural processes as the basis of the MAE was Mach (1875): "...we have to think that with the movement of a retinal image a special process is set up which is not present in the resting stage; and that in opposite movements similar processes in similar organs are excited but these processes exclude each other in such a way that with the occurrence of the one, the other is counterbalanced, and with the exhaustion of one the other occurs," (quoted in Wohlgemuth, 1911, p. 7). Thompson (1880,1881) attributes the MAE to fatigue at the retinal level. Exner (1888) is apparently the first to explain this effect of fatigue at the cellular level. At this time the existence of direction-selective cells in the visual system was of course not known. However, Exner postulates the existence of populations of "summation cells", which seem to be

differently affected depending on the direction of motion, so that with one direction of motion one type of summation cell becomes more fatigued than the other. Thus after prolonged viewing of one direction of motion, "...it will appear as if [one summation cell] is most strongly and [the other] least strongly excited, *i.e.* as if the objects had a movement opposed to the original one," (Wohlgemuth, 1911, p.17).

Cornsweet (1970) proposed that the MAE would result from the presence of direction-selective cells accompanied by recurrent inhibition in the outputs of these cells. Thus, during prolonged viewing of motion in a given direction, cells selective for that direction would first increase responsiveness, then, because of inhibition, decrease responsiveness. At the cessation of the motion, the excitatory activity would immediately cease, whereas the inhibition would persist longer, resulting in a decrease below the resting firing rate. Coupled with the lack of such effect in cells selective for the opposite direction, this would lead to the perception of motion in the opposite direction. Thus, inhibition provides a reasonable explanation both of decreased responsiveness due to adaptation and of the existence of the MAE.

Before examining the psychophysical evidence relating to the ratio model, let us take a look at the physiological data, in order to evaluate the feasibility of an explanation of MAEs based on the known characteristics of direction-selective cells. All studies examined cells in cat striate cortex, unless otherwise noted.

Physiological studies of adaptation of direction selective cells

The first physiological study of the adaptation effects on single direction-selective cells was done by Barlow and Hill (1963b) in rabbit retinal ganglion cells. They found that, as predicted by Sutherland's (1961) model, prolonged stimulation of direction-selective cells resulted in a reduction of their spontaneous level of activity. They reiterate Sutherland's point that a temporary imbalance in the maintained activity of cells tuned to opposite directions might result from adaptation to one direction, and that this might be the basis of the MAE. In fact, they point out, the temporal characteristics of the adaptation effects at the cellular level seem compatible with the psychophysically determined characteristics of the MAE. (This

similarity will be discussed later.) However, as they point out, direction-selective cells have not been found in primate retinas, suggesting that such a process must occur at a higher-level site, either LGN or cortex.

Maffei, et al (1973) noted that while cortical cells showed strong adaptation effects, geniculate fibers showed either no adaptation effects, or effects lasting no more than 2 or 3 seconds. While not a definitive result, since slight adaptation was found, this finding provides some evidence that adaptation occurs beyond the LGN, and thus beyond the retina, especially in light of the other evidence to date. Movshon and Lennie (1979) similarly reported a lack of adaptation effects in the LGN. Thus it seems that striate cortex is the earliest site at which physiological adaptation to moving stimuli occurs.

Maffei Fiorentini and Bisti (1973), though not addressing the issue of MAEs, demonstrated that the responsiveness of cat area 17 cells to a low-contrast grating pattern moving in the cell's preferred direction is decreased following adaptation to a similar high-contrast grating. Then Vautin and Berkley (1977) studied the adaptation effects in single cells of cat area 17 using motion in both the preferred and null directions. They indeed found decreases in cell responsiveness to the preferred direction of motion following adaptation to such motion. Only some cells showed decreased responses to null motion following adaptation to preferred motion, or to preferred motion following adaptation to null motion. This study was followed by quite a few other studies of cat area 17 cells (Hammond, Mouat & Smith, 1985, 1986, 1988; Hammond & Mouat, 1988; Hammond 1989; Maffei, Berardi & Bisti, 1986; Movshon & Lennie, 1979; Maffei, et al, 1973; Albrecht, Farrar & Hamilton, 1984; Hammond, Pomfrett & Ahmed, 1989; von der Heydt, Hänny & Adorjani, 1978; Marlin, Hasan & Cynader, 1988), and one of monkey MT cells (Petersen, Baker & Allman, 1985). These studies investigated such aspects of cellular adaptation as direction selectivity, effect of cell type (simple vs. complex), effect of velocity, and effect on spontaneous level of activity. The results of these studies were not always consistent, as will be readily apparent in the next sections.

Effects of adaptation on spontaneous activity

One issue which is as yet unresolved is the question of whether adaptation produces a decrease in a cell's spontaneous activity, or whether it is manifested only in altered driven responses. One study found that for the majority of cells examined, resting activity was decreased by adaptation (Vautin & Berkley, 1977). This same study found a decrease in spontaneous activity for some cells after adaptation to motion in the opposite direction as well. Another reported that spontaneous activity is decreased only for some cells (Movshon & Lennie, 1979). Others, however, have found no such effect (Hammond, et al, 1988; von der Heydt, et al, 1978), while another (Hammond, 1989) found that some special complex cells exhibited increases in resting rates and some exhibited decreases, while other cell types showed no changes. Finally, Marlin et al (1988) found that after adaptation in the preferred direction, most cells decreased spontaneous activity, and after adaptation in the nonpreferred direction, many increased spontaneous rates.

Must spontaneous levels be decreased by adaptation according to the ratio model?

Sutherland (1961) did not specify whether the ratio model as he envisioned it required a reduction in spontaneous firing rates following suppression, or a reduction in stimulus-driven activity. Barlow and Hill (1963b) and Barlow and Brindley (1963) suggested that it is the observed decrease in spontaneous levels that explains the imbalance following adaptation in one direction. Actually Barlow and Hill (1963b) used a stationary version of their adapting stimulus (rotating disk) as the preadaptation and test stimulus. This isn't actually spontaneous activity *per se*, but the response of the cells to the stationary target was essentially unchanged from spontaneous levels, being characterized by the authors as, "the normal, irregular maintained discharge."

Clearly a reduction in the level of maintained firing would be sufficient to explain the fact that a stationary stimulus is seen to move following adaptation in a single direction, if we accept the finding (e.g. Barlow & Hill, 1963b) that direction-selective cells generally do not respond to stationary stimuli. It may not be as obvious how a decrease

in spontaneous rates could explain the related phenomena of increased contrast thresholds for a moving stimulus and of decreased apparent velocity following such adaptation. These phenomena require not only that spontaneous levels of response be reduced by adaptation, but that driven responses be reduced as well. However, it is a likely consequence that a reduction in resting rate would be accompanied by a reduction in driven activity, which would account for all the observed effects.

The fact that changes in spontaneous firing rates *can* account for observed effects does not, however, answer the question of whether the reduction of spontaneous rates is a *necessary* part of a physiological explanation of MAEs. The critical question is: Is a reduction in spontaneous activity necessary, in addition to responses to moving stimuli, to explain the generation of the MAE as envisioned by the ratio model? There seems to be no question that driven activity is affected by adaptation. But as discussed above, many studies reported that spontaneous rates of firing were unaffected by prior adaptation. This poses a problem for the theory that the MAE, manifested as perceived motion of a stationary stimulus, occurs as the result of adaptation of direction-selective cells. If spontaneous rates are unaffected, how do we explain the proposed imbalance in activity of direction-selective cells which would need to occur with a stationary stimulus? It does seem as if changes in maintained firing must play a role in the MAE; however, it is still conceivable that a mechanism of adaptation could be proposed which would explain MAEs independent of spontaneous rate effects.

Direction selectivity of adaptation

Clearly adaptation causes changes in the responses of cat striate cells, but there is some disagreement as to details. One specific point of debate is the direction selectivity (or lack thereof) of adaptation effects. That is, are the responses of direction-selective cells altered for both preferred and null directions of motion, representing a general fatigue effect, or is only the response in one direction affected? This question can be addressed by looking at two patterns of adaptation effects: the effect of adaptation to preferred motion on null vs. preferred responses, and the effect of adaptation to preferred vs.

null motion on preferred responses. That is, does adaptation to preferred motion have a differential effect on preferred and null responses? And are responses to preferred motion differentially affected by preferred and null adaptation?

Evidence relating to the differential effect of adaptation to preferred motion on responses for opposite directions of motion has been inconsistent. While motion adaptation almost always results in a decrease in responses to subsequent preferred motion, the effect of null adaptation is not as clearcut. In particular, some cells have shown facilitation (increased response relative to preadaptation levels) for the preferred direction after adaptation to motion in the null direction (Hammond, et al, 1985, 1988; Hammond & Mouat, 1988; von der Heydt, et al, 1978; Marlin, et al, 1988), some have shown no change (Hammond, et al, 1985, 1986, 1988; Hammond & Mouat, 1988; Vautin & Berkley, 1977; Marlin, et al, 1988), and some have shown decreased responsiveness after such adaptation (Vautin & Berkley, 1977; Hammond, et al, 1985, 1988; Hammond & Mouat, 1988; Marlin, et al, 1988). Different researchers found greatly different proportions of cells in each of these categories. For instance, von der Heydt et al (1978) reported that many cells showed enhanced responsiveness following adaptation to the null direction. Contrarily, Vautin and Berkley (1977) and Marlin et al (1988) found that many cells showed a decrease, while Hammond et al (1986) reported that cells showed no change at all. Most of these studies found cells in at least two of the three categories of possible responses to null adaptation. Note that only von der Heydt et al (1978) and Marlin et al (1988) used stationary test gratings, and they obtained opposite results.

The issue of directional selectivity of adaptation can also be addressed in terms of the effects of adaptation to preferred motion on subsequent responses to null motion vs. preferred motion. Note that this is merely another way of looking at the direction selectivity of adaptation, and that these two issues are so clearly interrelated that it would be ludicrous to attempt to completely dissociate them. It thus seems particularly interesting that only two types of cell responses have been found (as opposed to the three listed above): some cells

showed decreased responsiveness in the null direction following preferred adaptation (Hammond, et al, 1985, 1988; Hammond & Mouat, 1988; Vautin & Berkley, 1977; Marlin, et al, 1988), while others were unaffected by such adaptation (Hammond, et al, 1988; Hammond & Mouat, 1988; Hammond, et al, 1989; Marlin, et al, 1988). Thus, while adapting to null motion sometimes causes enhanced responsivity in the preferred direction, this has not been demonstrated for the reverse case. (However, see results for MT cells below.)

Marlin, Hasan and Cyander (1988) also addressed the direction selectivity of adaptation by looking at changes in two other measures: direction selectivity itself (ratio of preferred to null response, P/N), and total responsivity (P+N). Direction selectivity decreased for most simple cells, and was sometimes even reversed. For complex cells, no general trend of change in direction selectivity was found, with 57% of these cells actually increasing their direction selectivity. Following null adaptation, simple cells usually showed increased direction selectivity, while complex cells showed no change.

Following preferred adaptation, all but two cells had decreased total responsivity (value<100%), and most were below 60% of preadaptation levels. This effect was not related to initial degree of direction selectivity. For adaptation to null motion, however, the decrease in responsivity was correlated with initial level of direction selectivity. Specifically, most cells with low direction selectivity (low P/N, and likely low N) showed decreases in responsivity, while highly direction-selective cells showed only slight decreases or even increases. The decrease in responsivity was lower than that following preferred adaptation. This finding is not surprising, as some cells showed increased responsivity following null adaptation.

Adding confusion to the issue is the fact that many authors did not explicitly state all the specific results of their studies of the effects for null adapt and null test. This is understandable in that there is such variability in response patterns among cells, making it more useful to report findings in terms of consistent and common results. Sometimes, however, the figures show individual cell records which show trends that are not mentioned. For instance, Figure 1 of

Hammond et al (1986) seems to illustrate a slight increase in preferred response following null adaptation for one cell, and a decreased response for another cell. This same figure shows a decreased response for null test following preferred adaptation for one cell. None of these results is specifically mentioned by the authors, which may suggest their unrepresentativeness and relative infrequency. However their existence should not be ignored, despite their rarity.

One potentially enlightening situation involves the directional selectivity of adaptation for truly bidirectional cells. These cells are characterized by equivalent optimal responses for two opposite directions of motion, with minimal response to motion in the orthogonal directions. Thus the direction selectivity of adaptation can be addressed without confounding by differences in initial magnitude of response. The results to date are inconclusive. One study (Vautin & Berkley, 1977) found that for these cells, following adapting to one preferred direction of motion, responses were decreased for both preferred directions. However, Hammond et al (1985, 1986) stress the differential susceptibility of these cells to adaptation for only the adapted, and not the unadapted, direction. (One additional study (von der Heydt, et al, 1978) examined a group of cells which were not classified as direction selective cells, as they did not meet the criterion of having a response in the preferred direction that was at least three times that in the null direction. It is unclear whether these cells are truly direction non-selective or are bidirectional. In these cells, adapting to both directions of motion caused equal reductions in responsiveness.)

One factor which may have contributed to this discrepancy (and possibly to the general confusion in studies of direction-selective adaptation) is the relative magnitude of adaptation effects on responses for the adapted versus the unadapted direction of motion. Marlin et al (1988) have reported that regardless of a cell's preference for direction of motion, adaptation effects tend to be greater for the adapted than the unadapted direction. That is, in cells which show decreases in response to both null and preferred motion following adaptation to preferred motion, more adaptation will be evident in

responses to preferred motion. On the other hand, following adaptation to null motion, responses to null motion will be reduced more than will preferred motion responses. Thus it is possible that for a bidirectional cell, the response in the unadapted direction would be less affected by adaptation than the adapted direction. Without the use of statistics, it is conceivable that researchers might view the unadapted direction response as being unchanged, in comparison to the more drastic change apparent in the adapted direction. (This highlights the need for more objective appraisals of experimental results, an issue which will be addressed in a later section.)

Only one study of adaptation in monkey MT cells has been conducted to date (Petersen, Baker & Allman, 1985). The authors found that facilitation in the null direction occurred in these cells after adaptation to motion in the preferred direction. The same result was obtained for responses to the preferred direction following adaptation to null motion.

Does adaptation have to be direction selective at the single cell level?

Clearly the issue as to whether adaptation is direction-selective is still an open question. While many studies found evidence supporting such selectivity, the existence of evidence to the contrary can not be ignored. How crucial is the direction selectivity of adaptation to our ability to explain the MAE with reference to physiological processes? In particular, is it possible that selectivity is an unnecessary assumption?

In his brief suggestion that the MAE might be the result of differential adaptation of cells selective for different directions of motion, Sutherland (1911) did not discuss whether the adaptation effects he predicted would involve general fatigue or direction-specific changes in response. Barlow and Hill (1963b) and Barlow and Brindley (1963) refer solely to changes in spontaneous activity following adaptation. The first study which seems to address the issue in terms of direction-selective adaptation rather than simply decreased general responsiveness was conducted by Vautin and Berkley (1977), who investigated the differential effects of preferred and null adaptation on both types of responses.

As evident from the sections above, many recent physiological studies have examined the direction selectivity of adaptation effects at the cellular level. However, is a direction-selective alteration of cellular responses necessary to explain the MAE and its known characteristics? That is, is it a prerequisite of our neural theory of MAEs that adaptation to a cell's preferred direction of motion result in differential effects on responses to preferred and null motion, or that adaptation to preferred motion produce different changes in response than adaptation to null motion? Or is a decrease in maintained activity, which would manifest itself in decreases for both directions of motion, sufficient to account for the phenomenon?

The theory as originally conceived (Sutherland, 1961; Barlow & Hill, 1963b) seems to rely solely on spontaneous activity, with the corresponding result that responses to all stimulation would be decreased following adaptation. Likewise, null motion would be expected either to produce no change in cell response (since it often produces no response at all and thus would not promote fatigue), or perhaps slight decreases. However, at some point the belief that preferred motion should decrease preferred responses, while not affecting or perhaps increasing null responses, has become popular. Likewise, null motion is expected to facilitate responsiveness for preferred motion while decreasing null motion responses.

For some researchers the point of interest in studying direction-selective cells is the expected differential change in a cell's driven response following direction-specific adaptation. That is, some researchers expected that individual cells would show increased response to preferred motion following adaptation to the null direction, and decreased response following adaptation to the preferred direction. Indeed, cells exhibiting this pattern of results were found by several researchers (Hammond, et al, 1985, 1986, 1988; Hammond & Mouat 1988; Petersen, et al, 1985; von der Heydt, et al, 1978; Marlin, et al, 1988), but note that not all cells in each of these studies exhibited this characteristic.

By this mechanism, not only are cells for the adapted direction operating at a lower level of responsiveness than in the unadapted condition, but those selective for the opposite direction are above

their unadapted responsiveness. This is certainly not inconsistent with the ratio model of MAE generation, but is it a necessary aspect of its explanatory value? That is, does the lack of observation by many researchers of differential effect of adaptation at the single cell level pose a serious problem for the ratio model? It seems not. In fact, as discussed above, the phenomenon of MAEs could be fully explained solely in terms of reductions in spontaneous firing rates based on a simple fatigue process independent of direction of motion. The overall balance of responses for cells selective for all directions would be biased toward the direction opposite the adapted direction, since cells in the adapted direction would have decreased responsiveness and those for the opposite direction would be unchanged, with all others being balanced out (see Figure 3). This resulting imbalance in spontaneous levels in cells adapted (i.e. selective for the adapted direction) as compared to cells which were not adapted would produce the MAE. Consistent with this idea, some researchers have found that single cells show reduced response in both preferred and null directions following adaptation in the preferred direction (Hammond, et al, 1985, 1988; Hammond & Mouat, 1988; Vautin & Berkley, 1977; Marlin, et al, 1988). This could be explained by a simple fatigue process, whereby the cell's overall responsiveness to all stimulation is reduced following prolonged activity/activation.

So why do we expect single cells to show direction-selective adaptation? One reason is the existence of reports showing that general fatigue is not a valid explanation of adaptation effects. Maffei, Fiorentini and Bisti (1973) argued against cell fatigue as the mechanism of adaptation, as the cells they examined generally showed stable response to the high-contrast adapting grating, following a brief initial transient. Adaptation also occurred when the cell's response to the adapting grating was reduced below the level of response to the low-level test grating by occluding part of the cell's receptive field. Dealy and Tolhurst (1974) concluded that adaptation produced by prolonged stimulation of cells by stationary high-contrast gratings was due to inhibitory processes, rather than mere fatigue following prolonged activity. They noted the findings of Maffei et al (1973), and suggested that the spatial adaptation mechanism may be the same for

moving stimuli. Ohzawa, Sclar and Freeman (1985) examined contrast adaptation in cat striate cells, and reported that a mechanism of adaptation based on simple fatigue was unlikely. Adaptation to a stimulus which filled the whole receptive field produced a larger response than a stimulus which only partially filled it; yet adaptation effects were equivalent. In addition, they reported preliminary evidence that adaptation to stimuli of nonoptimal parameters can result in decreased responsiveness to optimal stimuli, even though the adapting stimuli elicited no response from the cell whatsoever. These authors also suggest inhibition as the mechanism of adaptation.

Maffei, Berardi and Bisti (1986) determined that the strength of a physiologically demonstrated interocular MAE is not related to the strength of input from the contralateral eye. Interocular transfer was even found for cells with no apparent response to the contralateral eye. Thus the notion that adaptation represents fatigue due to repeated stimulation and activity seems inappropriate. These authors note, however, that they cannot exclude the possibility of fatigue in neurons at other sites, that may contribute to the responsiveness of the cells examined.

Likewise, Marlin et al (1988) reported that dramatic changes in responsivity and direction-selectivity were obtained following null adaptation, even though adaptation itself caused minimal responses. They suggest that adaptation is not proportional to the amount of activity of a given cell, but rather results from adaptation of pools of afferent neurons. Vautin and Berkley (1977), however, suggested that the extent of reduction in responsiveness to repeated stimulation *is* related to the extent to which prior stimulation produced cell responses, as adapting to preferred motion could reduce responses to null motion, and that moderate activity produced by adapting to null motion was followed by decreased responding to preferred motion.

Further evidence for the insufficiency of fatigue as an explanation of adaptation derives from Vidyasagar's (1990) study of the effects of GABA on cell response in area 17 of cat. This author demonstrated that while application of GABA prevents cell firing, it does not reduce the magnitude of adaptation. Fatigue can not account for adaptation in

this situation, because GABA has blocked cell firing and thus the opportunity for fatigue due to prolonged stimulation and activity.

Another reason to expect neural adaptation to exhibit direction-selectivity is the existence of psychophysical evidence suggesting that adaptation must be direction-selective (e.g Moulden & Mather, 1978). This evidence will be discussed in detail later; suffice it to say at this point that there is substantial reason to suspect that adaptation occurs in a direction-selective manner.

Simple vs. complex cells

One factor which may be partly responsible for the lack of consistency in investigations of direction selectivity of adaptation is that results have shown to differ for simple as opposed to complex cells. Most studies have found that complex cells are less susceptible to adaptation than are simple cells (Hammond, et al, 1985, 1988; Hammond & Mouat, 1988; Maffei, et al, 1973; Albrecht, Farrar & Hamilton; Maffei, et al, 1986), with special complex cells being especially resistant to adaptation (Hammond, et al, 1985, 1986, 1988; Hammond, et al, 1989). One study (Albrecht, et al, 1984), though, actually found that complex cells were *more* adaptable. Another (Maffei, et al, 1986) found that interocular transfer of adaptation effects in area 17 was greater for simple than complex cells, while in area 18 cells simple-like cells and complex-like cells were equally adaptable and showed equal interocular transfer. The time course (rate of buildup and decay) of adaptation has generally been reported as being more rapid for simple cells than for complex cells (Hammond, et al, 1988; Hammond & Mouat, 1988; Maffei, et al 1986), although the opposite result has also been obtained (Maffei, et al, 1973; Albrecht, et al, 1984), as has a finding of equivalent time courses (Vautin & Berkley, 1977).

Marlin et al (1988) examined a variety of measures of adaptation in both simple and complex cells, as discussed in the section on direction-selectivity of adaptation. They found no difference between simple and complex cells in terms of total responsivity (null response plus preferred response). Simple cells showed decreases in direction selectivity following preferred adaptation, while complex cells showed increases or no change. For null adaptation, complex cells again

showed no change, while simple cells increased selectivity. Decreases in response to null motion occurred following null adaptation for both simple and complex cells, but the effect was greater in simple cells. Following preferred adaptation, decreases in null responsivity occurred for both simple and complex cells, but correlated with preadaptation level of direction selectivity for simple cells only. Preferred responsivity was decreased for both simple and complex cells following preferred adaptation, with the change being greater for simple cells. Following null adaptation, simple and complex cells showed equivalent decreases in preferred responses, but the correlation between change in responsivity and initial direction selectivity was significant for simple cells, only.

Von der Heydt et al (1978) found that while adapting to a moving grating caused a direction-selective effect for a moving test grating in both simple and complex cells, testing on a stationary grating revealed effects for simple cells only. They propose that complex cells receive input both from direction-selective simple cells, whose dominant response is to moving stimuli, and from non-selective simple or geniculate cells, which account for responses to stationary stimuli. These latter cells are unaffected by motion adaptation; thus the responses of complex cells to stationary targets is unchanged following motion adaptation.

Methodological causes of discrepancy

Clearly the results of recent studies of physiological adaptation effects have contributed invaluable information regarding the possible neural basis of MAEs. Still, a number of issues remain unresolved. The issue of whether a decrease in spontaneous rates following adaptation is necessary to account for the MAE, and the question as to the necessity of direction-selective adaptation effects at the single cell level, may be crucial in terms of developing a comprehensive mechanism to explain the observed characteristics of MAEs and related phenomena. The answers to these questions, however, remain elusive. The lack of consistency of the results to date in terms of the direction selectivity of adaptation effects and the effect of adaptation on spontaneous rates is both puzzling and disturbing, and needs to be addressed in future research. One likely factor in the discrepancies

observed between different studies and different researchers lies in methodological differences, including such variables as type of stimulus and general procedure. Other discrepancies may reflect the need to assess adaptation effects and their direction selectivity in other ways. Following is a discussion of some of these variables.

Small sample sizes

One problem is the small number of cells used in some of these studies. On occasion experimenters reported results from only a few cells of a given type in a given condition. Thus it is often impossible to determine whether the responses observed for that class of cells are actually characteristic of that class, or whether responses are actually either generally different or quite variable. This limits the generalizability of the results of a given study, and may be partially responsible for the inconsistency of findings among different studies.

Procedural differences

In addition, differences in procedures employed by different research groups no doubt account for some of the differences in results observed. Since experimenters were frequently conducting their experiments with a particular issue in mind, their choice of adapting and test stimuli, adaptation durations, and adaptation protocols no doubt reflected this purpose, and led to differences in these variables between studies. It is unfortunate that so few studies have been carried out to date, as the limited focus of each study results in a need for more numerous studies to address all the issues.

Lack of stationary test patterns

One particular aspect of the physiological studies has special relevance for a discussion of the neural basis of the MAE. All but three (Barlow & Hill, 1963b; von der Heydt, et al, 1978; Marlin, et al, 1988) of the studies reported here investigated adaptation effects solely in terms of the response to a moving stimulus following adaptation to motion in the same or the opposite direction. Barlow and Hill (1963b) used a stationary version of the adapting stimulus for testing in their experiments of rabbit retinal ganglion cells; however, the applicability of their results in explaining MAEs in humans is probably not as great as for results in species more similar to ourselves. Yet their results do support the general possibility of adaptation effects being manifested

in a change in responsiveness to a stationary stimulus, and are not in contradiction with similar studies in cats and monkeys.

One study (von der Heydt, et al, 1978) tested responses to stationary gratings which were turned on and off every 0.5 sec and which reversed phase every 1 sec. Interestingly, this test stimulus was effective only for simple cells; complex cells showed no adaptation effects for stationary gratings. Marlin et al (1988) investigated the effect of adaptation on the response to a stationary grating, although their analysis was not as extensive as their analysis of responses to moving stimuli, and in fact they tested only 19 cells in this manner. However, their results are informative, and are as yet the only results of such an investigation. For the test of responses to a stationary stimulus, the cell's preferred grating was flashed into the cells receptive field for 1-4 seconds. The basic finding was that responses to the stationary stimulus were decreased following adaptation in the preferred direction, and unchanged following null adaptation.

The compellingness of the MAE itself involves the perception of motion when viewing a *stationary* test target. Thus, while the phenomena studied physiologically are of course relevant to understanding the MAE, they do not represent the actual conditions under which the MAE is seen. It is thus important to evaluate the utility of such studies in building a model of the MAE. In particular, it is likely that many experimenters did not consider testing with a stationary target for the simple reason that the cells studied did not respond to non-moving stimuli. What does this say about the neural basis of the MAE, and about the role of single cell responses? This is a factor which must be considered if we are to truly understand the basis of the MAE.

One point made by Marlin et al (1988) addresses the role of adaptation of responses to stationary vs. moving targets in the motion aftereffect. They note that changes in both spontaneous activity and responses to stationary stimuli are small relative to the effects on responses to moving stimuli. They suggest that involuntary eye movements during viewing of a stationary stimulus result in the involvement of cell responses to moving stimuli, even though no stimulus motion is present. That is, when an observer views the

stationary grating, involuntary eye movements cause the image to be alternately moved across the retina in opposite directions, activating cells selective for both directions as well as non-selective cells. Thus after adaptation to motion in a given direction, the combined signal for leftward motion from both direction selective and non-selective cells will be higher than that for rightward motion. Thus, it is possible to account for the MAE without relying solely on the adaptation effects on cell responses to stationary stimuli. However, this interpretation requires that we assume the visual system does not separate motion signals produced by image motion from those produced by eye movements.

Evidence suggests that this separation actually does occur. Bridgeman (1972), for example, studied awake behaving monkeys trained to track moving targets with eye movements, and found that some cortical cells respond to motion relative to the background, while others respond to absolute motion. (This and related studies will be discussed in the section on eye movements.) He concludes that the cortex may employ these two types of cells to accomplish a distinction between eye movement and stimulus movement. Galletti, Squatrito, Battaglini and Maioli (1984) obtained similar results, but estimated that only about 10% of monkey V1 cells were differentially responsive to motion in the presence vs. the absence of eye movements. Thus it is unclear whether the suggestion of Marlin et al is actually feasible.

Role of preadaptation response levels

I have already discussed the fact that adapting to one direction of motion usually affects responses for that direction more than those for the other direction, regardless of a cell's directional preference. Another issue relating to the direction-selectivity of adaptation involves the possible influence of the preadapted response level on the extent of adaptation effects. This may especially be important in assessing effects on responses to motion in the null direction, which are frequently negligible even prior to adaptation. To illustrate this point, I will discuss the results of Marlin et al (1988), who, unlike other investigators examined the entire spectrum of adaptation effects with respect to direction selectivity. This study thus provides the most comprehensive analysis of the direction selectivity of adaptation in

cortical cells to date. They analyzed effects in terms of changes in preferred responses, changes in null responses, changes in overall responsivity (null response + preferred response), and changes in direction selectivity (ratio of preferred response to null response).

In their figures 2 and 3, it is apparent that the cells which show relatively negligible preadaptation responses to null motion also show negligible adaptation effects. Likewise, those cells with substantial null responses also show significant decreases in these responses following adaptation. This suggests that a relationship between preadaptation responses and response changes due to adaptation may exist. It is possible that this relationship illustrates some form of floor effect, in that responses which are sufficiently small can not be decreased further by adaptation. Since the precise mechanism of adaptation of direction-selective cells is not known, we can not yet evaluate this possibility, but it is possible that a floor effect might result if inhibition were involved. That is, if decreases in response following adaptation are due to increased inhibition, it is possible that cells with no noticeable response to null motion are fully inhibited, such that no further inhibition of null responses can occur. (It thus represents a neural ceiling effect, manifested as a floor effect due to the inverse nature of inhibition.)

Both simple and complex cells decreased null responses following both preferred adaptation and null adaptation. Cells with low direction selectivity (low P/N) showed the strongest effect. One possible interpretation of this result is that the low ratio of these cells is due to larger null responses. Thus, these cells, by virtue of their larger preadaptation responses, would be more susceptible to adaptation, and would show a greater decrease in null response. Of course, preadapted preferred response magnitude contributes to this ratio as well, so it is unclear how feasible this explanation is. Interestingly, the authors report that only cells with strong null responses (P/N<5) showed a decrease in total responsivity (P+N) following null adaptation. They have therefore associated a low ratio with high null response, suggesting that the scenario just described might be appropriate. In addition, if cells with higher null responses (and thus lower ratios) are more likely to show decreases in null

response, the relationship between response ratio and decrease in total responsivity might be attributable to the prior level of null response.

Petersen, Baker and Allman (1985) found that direction-selective adaptation occurred only in MT cells that were strongly direction-selective. However, cells with selective decreases in response to null motion would not be characterized as showing direction-selective adaptation according to their adaptation index. Cells with less strong direction-selectivity are more likely to have substantial preadaptation null responses, which might show larger decreases in null responses, but would not be characterized as showing direction-selective adaptation.

One suggestion is thus that future researchers endeavor to report adaptation effects at the cellular level in relation to preadaptation levels, in addition to other desired measures. This would allow an assessment of the extent to which preadaptation responses need to be taken into account in interpreting direction-selective adaptation effects.

Lack of statistical testing

Another relevant issue is the lack of statistical testing in the physiological studies. Experimenters tend to reach conclusions regarding cell responses and changes in those responses merely by "eyeballing" frequency distributions or traces of the responses through time. This may affect the validity of their conclusions, in that variability representing chance factors is not systematically accounted for. Granted, researchers do generally report when cells show inconsistent response patterns over time. Yet it is still questionable whether valid answers regarding the presence or absence of an effect on cellular responses can be achieved merely by inspection of response records. One main exception is the study by Marlin et al (1988), who employed statistical tests of response changes. The use of similar statistical analyses in the future would ensure the validity of the conclusions reached from physiological studies.

Effect of anaesthesia

One criticism of the physiological studies performed to date is that most involved paralyzed, anaesthetized cats (or in one case,

monkeys). Thus, it is questionable to what extent the cellular responses observed are representative of normal cellular responses under unanaesthetized conditions (Poggio, 1972). Vautin and Berkley (1977) addressed this issue by assessing the effects of different levels of anaesthesia on the responses of V1 cells, and also by comparing responses in anaesthetized and unanaesthetized cats. They found that responses under the two conditions were similar, except that higher variability was found for cells in unanaesthetized cats. The general patterns of responding were unchanged. Increasing the level of anaesthesia above the levels generally used in the experiments caused reductions in the magnitude and variability of cell responses, yet the time course did not seem to be altered. They thus conclude that the use of anaesthesia did not alter the results of their study.

Schiller, Finlay and Volman (1976a) also reported similar results for awake behaving and for anaesthetized monkeys. Similarly, Vogels, Spileers and Orban (1989) found that the relationship between response variability and response magnitude is similar in awake behaving animals and paralyzed anaesthetized animals. Their experiment with awake monkeys showed that this relationship was best described by a power function with power approximately equal to one. They note that the same relationship was found previously for anaesthetized cats by Dean (1981), and for both anaesthetized cats and monkeys by Tolhurst, Movshon and Dean (1983). There is thus at least some evidence that response changes under anaesthesia may be a valid measure of unanaesthetized response patterns.

Role of inhibition in adaptation

The cellular mechanism of adaptation has not yet been established, despite the multitude of investigations of adaptation. Fatigue due to prolonged stimulation and activity can not account for many of the reported characteristics of adaptation, as discussed earlier. As mentioned in a previous section, Cornsweet (1970) proposed that the MAE is attributable to recurrent inhibition in the outputs of direction-selective cells. During adaptation, cells selective for that direction would first increase responsiveness, then decrease it due to inhibition. Prolonged inhibition following adaptation would depress the cell's resting firing rate, producing an imbalance in the

outputs of directional detectors and yielding an MAE. A variety of existing evidence points to a possible role of inhibition in the mediation of adaptation effects.

Vautin and Berkley (1977) describe a model of adaptation effects, which can account for all types of responses they observed. This model basically involves decay of both excitatory and inhibitory responses. Decay of both types of responses follows an exponential function; manipulating the time constants of decay and the initial magnitude of excitation and inhibition yields combined responses similar to the cellular responses exhibited physiologically. They noted that the time constants for both excitation and inhibition are in the range of observed values. In addition, the transient increase in response shown by some cells when the stimulus was removed at the cessation of the adaptation period can be attributed to a sudden decrease in inhibition.

Further evidence regarding the involvement of inhibitory processes in adaptation was recently presented by Vidyasagar (1990). As mentioned earlier, GABA administration prevented cell firing in cat striate cells, but did not block adaptation. We can thus conclude that fatigue is not the cause of adaptation. Vidyasagar also showed that addition of glutamate (an excitatory transmitter) or GABA did not reduce cell sensitivity in the absence of an adaptation stimulus. Thus adaptation does not result from excitatory or inhibitory activity of the adapted cell, further debunking the notion that adaptation is the result of prolonged cell activity. In addition, bicuculline did not prevent adaptation, suggesting that adaptation does not occur via GABA inhibition of the cell. The author suggests that adaptation is the result of changes in a cooperative cortical network, with the effect on single cells mediated by intracortical excitatory connections.

Role of direction-selective collicular cells in MAE

Richards and Smith (1969) have presented evidence suggesting that the MAE may arise from processes in the midbrain, rather than in the cortex. The basis for this investigation was Richards' (1968) previous finding that the geniculostriate pathway is altered by convergence changes, as the mapping of the retina onto the cortex is altered. This leads to micropsia: the change in apparent size of a fixed

retinal image. They thus suggest that if the MAE is indeed cortically based, then it should show this characteristic. If on the other hand it is processed in the colliculus, no such result should obtain. Subjects adapted to a rotating spiral disk surrounded by a spiral annulus rotating in the opposite direction. A small border between the two regions existed, such that no MAE was apparent in the region. Adaptation and test could each occur for a viewing distance of 25 or 200 cm. The 25 cm distance was created using a lens and prism combination, and measurements were corrected for optical reduction. It was found that at these distances, the relative change in apparent size (assessed by setting the separation between two pinpoints of light to be perceptually equal at the two distances) was 1.3. Thus, a similar change in apparent size of the MAE border would indicate the involvement of the geniculostriate pathway. The subject was required to position two pins in the border region of the MAE so that they were seen neither to move inward or outward. No difference in settings was found for the two distances, leading the authors to conclude that the MAE must be mediated in the collicular pathway rather than the geniculate pathway.

These results must be viewed cautiously at this point, however. While direction-selective cells have been consistently observed in the colliculus, no investigations of possible adaptation effects in these cells have been conducted as yet. Thus it would be premature to actually attribute the MAE to adaptation at the level of the colliculus. Nor have cells in superior colliculus been shown to be selective for more complex forms of motion, such as the various components of spiral motion, as has been demonstrated in cortex. (Those findings will be discussed in later sections.) In addition. the technique used in this study was a rather indirect method of localizing the MAE, and it is unclear whether any flaws are inherent in the design or in the rationale behind the design.

Richards (1971) reported that the range of sizes of motion detectors inferred from his psychophysical experiments are comparable to the receptive field sizes in the midbrain, although they are also similar to receptive field sizes at other levels. Likewise, while other characteristics of MAEs examined were commensurate with

processing at the level of the colliculus, other areas could not be ruled out.

SIMILARITIES BETWEEN PSYCHOPHYSICS & PHYSIOLOGY OF MAE'S

Stimulus specificity

As discussed in the introductory sections, the psychophysically measured MAE varies with the parameters of the adapting and test targets. In particular, MAEs are stronger when adapting and test targets possess similar spatial frequency and orientation. The orientation-specificity is paralleled by the physiological observation that adaptation effects are greater when adapting and test orientations are similar, even taking into account the differential baseline response to stimuli of varying orientation. (e.g. Hammond, et al, 1989). Likewise, physiological studies suggest that the magnitude of adaptation decreases as the difference between adapting and test spatial frequencies increases. Movshon and Lennie (1979) reported that this relationship was consistently found for complex cells, but less so for the smaller sample of simple cells examined. Albrecht, Farrar and Hamilton (1984) similarly found that adaptation is maximal when adapting and test spatial frequencies are identical, as did Hammond et al (1988).

Effect of adaptation duration

One clear and consistent finding of psychophysical studies of the MAE is that the strength of the MAE increases as the duration of adaptation increases, at least up to a limiting level (Cords & von Brücke, 1907; Kinoshita, 1909; Holland, 1958; Pickersgill & Jeeves, 1958; Bakan & Mizusawa, 1963; Sekuler & Pantle 1967; Strelow & Day, 1971; Hershenson, 1982). This has in fact formed the basis for several studies of MAEs, including studies of the effect of binocular rivalry on the buildup of different types of MAEs (Lehmkuhle & Fox, 1975; Wiesenfelder & Blake, 1990), and is currently widely accepted as a characteristic of MAEs in general. Do the physiological studies show evidence for this pattern at the single cell level? In fact, two of the three studies which address this issue failed to find such an effect (Hammond, et al, 1985, 1988). Only Barlow and Hill (1963b) reported that the minimum duration of adaptation necessary to elicit adaptation effects is similar to the psychophysical minimum duration, and they

did not address the effect of increasing adaptation duration beyond this level.

How then do we explain the psychophysical result (assuming that the physiological results will hold up after attempts at replication)? One possibility is that increasing the duration of adaptation results in an increase in the number of cells affected.

Studies have found that the duration of the MAE increases as the size of the adapting/test field increases (Granit, 1928; Pickersgill & Jeeves, 1958; Freud, 1964). Likewise, Sekuler & Pantle (1967) found that if only part of the adapted area is tested due to a shift in fixation during the test period, the duration and velocity of the MAE is reduced relative to the case in which the test pattern covers the whole adapted area of the visual field. However, it is arguable whether their experiment really addresses the issue of the number of cells adapted, since it really manipulates the number of cells tested relative to the number adapted, not the number of cells adapted *per se*. In fact, their results can be taken merely as a byproduct, so to speak, of the storage process, in that the cells not tested are storing the MAE. (This result will discussed in relation to storage in a later section.) Their subjects in fact reported that after the MAE under shifted fixation had ceased, they were still able to see an MAE upon shifting back to the original fixation point.

It seems clear that increasing the size of the adaptation field, and thus the number of cells adapted, results in an increase in the strength of the MAE. How is this effect of target size explained? The effect of adaptation of larger numbers of cells on the magnitude of the MAE could help to explain the psychophysical relationship between adaptation duration and MAE duration, which does not seem to hold at the single-cell level (Hammond, et al, 1985, 1988). I propose two possible ways in which increasing the duration of adaptation could increase the number of cells adapted.

One possible explanation is that some cells have a temporal threshold, so to speak, for adaptability. That is, there may be cells which do not adapt immediately at the commencement of activity, and that show reduced activity only after a period of time has elapsed. Of course, this idea is only of interest if the physiological evidence

supports, or at least does not contradict, the predictions. While this possibility has not been explicitly addressed, some of the physiological reports support at least the existence of cells with delayed adaptation. Figure 1 of Marlin et al (1988) illustrates a cell whose response seems stable for the first 12 or perhaps 15 seconds of adaptation, then begins to show the decreased responsiveness characteristic of prolonged stimulation. Some of the cell responses presented by Albrecht, Farrar and Hamilton also support such a possibility. Figure 1a shows a cell with a shallow slope for the decrease in responsiveness; it is possible that had a longer adaptation period been used, the cell would have decreased its activity even further. Even more convincing are the cell responses shown in their Figure 2. One cell has just reached its peak firing rate at the end of the 30 second adaptation period, and thus might show a decrease with longer adaptation. Another doesn't reach its peak until 8 seconds, finally decreasing to a plateau level near the end of the 30 second adaptation period.

Also, as mentioned previously, complex cells were found to be less adaptable than simple cells, and some complex cells were found to show no adaptation effects at all. Is it possible that some of these cells had a temporal threshold which was not reached? This question can only be answered by further physiological study, and in fact these thresholds would have to be on the order of tens of seconds to account for the psychophysical increases in MAE with durations up to a minute. If this does turn out to be the case, the relationship between adaptation duration and MAE duration might be at least partially explained by the increased recruitment of complex cells as adaptation progresses, up to a certain point.

An alternative explanation is that as more cells are included in the adaptation process, the probability of cells with long time courses (which would thus maintain the adapted state for a longer period) being adapted is increased. Thus, as more cells are recruited by the adaptation process, the number of cells with a large time constant increases, and therefore more cells remain partially adapted following a given period of time, so that the MAE lasts longer overall. This hypothesis is consistent with Taylor's (1963a) finding that increasing adaptation duration alters both initial magnitude and decay rate of the

MAE, while increasing velocity only affects initial magnitude. This explanation is of course not mutually exclusive with the previous one, and it is indeed possible that both processes could have a role in the effect of adaptation duration.

The lack of agreement between physiological and psychophysical findings is especially disturbing in light of the ease and frequency with which the psychophysical relationship between adaptation duration and MAE strength has been shown, as it would be difficult to suggest that the psychophysical studies were all confounded in a manner which could explain the discrepancy. The ratio model as proposed does seem to suggest a simple basis for the effect of adaptation duration: in particular, one would predict that as the adaptation duration increases, the responsiveness of activated cells would continually decrease, yielding an increased imbalance in the responses of these cells relative to cells signalling other directions, and thus producing a larger MAE. This explanation also explains the saturation observed at a certain level of adaptation (typically about one minute), as responsiveness cannot fall below zero, and thus limits the imbalance which can result from continued adaptation. It is thus puzzling that the physiological findings do not support this prediction. It may be premature, however, to throw in the towel on the ratio model, as very few studies have actually addressed the effect of increased adaptation on strength of neural aftereffects, and it is possible that further investigation will yield results which provide a physiological explanation for this effect. Clearly the discrepancy between physiological and psychophysical findings relating to the effect of adaptation duration on MAE strength is one which must be resolved if we are to fully understand the basis of the MAE.

Time course

While the relationship between adaptation duration and MAE strength has not been established physiologically, the time course of adaptation and of the neural aftereffects of adaptation seem to be consistent with the psychophysically observed time course of adaptation and MAEs. Psychophysical experiments have consistently illustrated that the time course of decay of the MAE follows an exponential function (Taylor, 1963a; Keck & Pentz, 1977; Beverley &

Regan, 1979; Hershenson, 1989), and physiological studies have exhibited a similar relationship. Barlow and Hill (1963b) noted that the initial buildup of adaptation at the neural level fits well with the decrease in motion sensitivity which occurs during about the first 15 seconds of adaptation. They also pointed out that the duration of suppression of maintained firing is similar to the duration of the short-term MAEs, and, as mentioned above, that the duration of adaptation necessary to show adaptation is appropriate in relation to the psychophysical phenomenon as well.

Vautin and Berkley (1977) also examined the similarity between the time course of recovery from neural adaptation and that of the MAE itself. They compared their physiological data with the psychophysical data of Blakemore and Campbell (1969), and determined that the time constants were similar. (Note, however, that Blakemore and Campbell employed stationary gratings flashed at 2 Hz, not drifting gratings.)

There has not as yet been a physiological analogue of the multiple decay phases reported in the psychophysical literature (Taylor, 1963a; Hershenson, 1989), however. These multiple phases may be related to the phenomenon of storage, to be discussed shortly; thus the discovery of a physiological correlate would be of great utility.

Contrast response

Another characteristic of adaptation effects which is illustrated similarly by both physiological and psychophysical investigations is the contrast response of the motion mechanism. One consistent physiological finding is the saturation of responsiveness at relatively low contrast, with further increases in contrast causing no further increase in response. Barlow and Hill (1963a) demonstrated that the response of direction-selective cells shows a substantial increase with increases in the ratio of stimulus luminance (variable) to background luminance (0.2 mL) from .1 to 1.5, but very little further increase up to a ratio of 6. This suggests that these cells saturate at a contrast level of about 0.2 ($[L_{max}-L_{min}]/[L_{max}+L_{min}] = [.3-.2]/[.3+.2] = .2$). Maffei and Fiorentini (1973) examined the contrast response of cat striate cells, and found that the discharge rate saturates, although the contrast at

which saturation occurred was variable and not consistently as low as in these other studies.

Meanwhile, the psychophysical results support a similar effect of contrast. Pantle and Sekuler (1969) measured luminance threshold elevation following adapting motion in the same direction and in the opposite direction. The elevation following opposite motion was taken as a measure of the adaptation of orientation-selective (i.e. non-direction-selective) units, whereas the difference between this level and the elevation produced by motion in the same direction was taken as the degree of adaptation of direction-selective units. They reported that adaptation of direction-selective elements, increases up to a contrast of about 0.16, and then is independent of contrast. For the orientation-selective mechanism, on the other hand, adaptation increases up to the maximal level employed, 0.83.

Keck, Palella and Pantle (1976) examined the effect of varying adapting contrast on MAE duration and initial magnitude, and found that both measures increased as contrast increased up to 0.03, then showed little further increase. They point out that their threshold contrast was 0.005, whereas that of Pantle and Sekuler (1969) was 0.024, a factor which would explain the difference in saturation contrast between the two studies. They thus suggest that foveal direction-selective motion mechanisms possess a limited or compressed contrast response, a notion supported by the physiological studies cited above.

Albrecht, Farrar and Hamilton (1984) also presented evidence regarding the similarity in contrast response as measure psychophysically and physiologically. They compared the extent of contrast adaptation measured psychophysically by Blakemore and Campbell (1969) and Blakemore, Muncey and Ridley (1973) with those measured physiologically in that study. The two measures were similar, as were the recovery curves.

Directional asymmetry of MAEs

A number of researchers have reported that centrifugal (also called foveofugal) spiral MAEs are stronger than are centripetal (foveopetal) MAEs. Wohlgemuth (1911) first reported that the expanding MAE is stronger than the contracting MAE for the same

adapting stimulus rotated in opposite directions. This finding has been replicated on numerous occasions (Spitz & Lipman, 1959; Costello, 1960, 1961; Bakan & Mizusawa, 1963; Scott, Lavender, McWhirt & Powell, 1966; Reinhardt-Rutland, 1987b; see also reviews by Holland, 1965 and Hershenson, 1982). Richards (1971) also found that less contrast is needed at maximum sensitivity to observe an expanding MAE than for a contracting MAE. Interestingly, Spitz (1958) found similar results, but for a group of mentally retarded adolescents. Longer adaptation duration was required for these subjects to see a contracting MAE than for an expanding MAE. This result was replicated by Spitz and Lipman (1959), who obtained the same result for both normal and mentally retarded subjects.

Other researchers, however, have reported that asymmetry does not occur (Pickersgill & Jeeves, 1958; Hershenson, 1982). Hershenson (1982) found that for each duration of adaptation, an equal number of trials showed larger centrifugal MAEs as showed larger centripetal MAEs. Asymmetry was also reported by Truss and Allen (1959).

Several authors (e.g. Bakan & Mizusawa, 1963) have suggested that the greater MAE under the centrifugal condition may be attributable to the greater ease of fixation for a contracting adapting spiral. Scott et al (1966), however, claim to have shown that this is not the case. Subjects adapted while either fixating the center of the target, fixating with lights flashed on and off in sequence around the periphery of the target, or while tracking these flashing lights. They found decreased MAEs in both flashing light conditions relative to the fixation-only condition, but no difference in eye movements for expanding versus contracting MAEs. They noted that the impaired fixation reduced MAEs as much as the change in direction of rotation, yet the eye movements did not differ with the change in direction. They thus concluded that a difference in fixation does not explain the asymmetry.[1]

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1. However, they did not do a simple comparison of eye movements for the two directions under the rotation condition, as they only compared eye movements across fixation conditions. Their data show identical results for the different directions in the other two conditions, but some difference is evident for the fixation condition. It is possible that this difference was

In addition, these authors demonstrated that the asymmetry holds for linear MAEs as well as spiral MAEs. Following adaptation to linear motion toward the fovea, MAEs were larger than those that occurred following adaptation to motion away from the fovea. Thus, like spiral MAEs, centrifugal linear MAEs are stronger than centripetal ones. Scott et al (1966) further reported that after long exposure to the adaptation stimulus over several days, the asymmetry decreased and sometimes disappeared completely. This effect was due both to increases in centripetal and decreases in centrifugal MAEs. They had reported earlier (Scott & Powell, 1963) that for monkeys, centripetal MAEs tend to be larger than centrifugal MAEs. They suggest that since the monkeys need more practice, this extended exposure reverses the asymmetry. It would be interesting to determine if this reversal is apparent for human observers who have spent much time observing motion adaptation stimuli.

Some support for a physiological basis of this asymmetry exists at the single-cell level. Orban, Kennedy and Maes (1981a) reported that a large number of area 18 are tuned to centrifugal motion. Area 17 cells respond optimally and equally to both centrifugal and centripetal motion. Area 18 contains a higher proportion of direction-selective cells, though, and these authors propose that it is more specialized for motion processing, with areas 17 and 19 playing lesser roles. Albright (1989) reported a bias for preference of centrifugal motion in MT cells as well.

These findings are paralleled by the report of Reinhardt-Rutland (1988) that observers perceived approaching motion in depth of a spiral more than retreating motion in depth of the same spiral rotating in the opposite direction. Expansion is accompanied by motion toward the observer, whereas motion away from the observer corresponds to contraction. It is unclear, however, whether these results relate directly to expansion/contraction asymmetry, as size-change and motion-in-depth components of MAEs may be mediated separately (Beverley & Regan, 1979).

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manifested as an interaction between direction and fixation condition, but was not sufficient to produce an overall difference between the two directions. It is unclear whether they tested for an interaction or not.

Realize, however, that the interpretation of these findings is ambiguous. The finding that centrifugal MAEs are stronger might lead us to suspect a majority of cells preferring *centripetal* motion, which would be adapted by such motion yielding a centrifugal MAE. How does a bias toward centrifugal motion, evident both physiologically and psychophysically, produce the asymmetry observed?

One possibility is that since there is a predominance of cells selective for centrifugal motion, the adaptation of a proportion of these cells produces less of an overall decrease in responsiveness than would occur for equivalent adaptation of the less numerous centripetal-selective cells. Thus, the imbalance in favor of centripetal motion following centrifugal adaptation is less than that in favor of centrifugal motion following centripetal adaptation. An alternative explanation relates to the differential occurrence of centrifugal vs. centripetal motion in the optic flow field. The importance of centrifugal motion in everyday experience has been duly noted (e.g. Gibson, 1950, 1966), as forward motion of the observer results in an expanding flow field with a predominance of centrifugal motion vectors. It might thus be proposed that elements specialized for detection of centrifugal motion may be less prone to adaptation effects than are elements detecting other directions of motion, a characteristic which could be viewed as an evolutionarily adaptive trait. On the other hand, this hypothesis presupposes that adaptation is nonadaptive, when in fact it may be viewed as adaptive. Adaptation in the realm of velocity of motion for instance, which is clearly related to directional adaptation, might serve as a gain control mechanism, so that animals would still be sensitive to a range of velocities, and to differences in velocities, when exposed to continuous high-velocity motion. That is, the decrease in response accompanying prolonged viewing of motion would allow the sensitivities of neural analyzers to be reset, so as to optimize sensitivity at a level at which saturation would normally occur. This notion is supported by the finding that MAEs to tend to increase in strength as adaptation velocity increases, suggesting that adaptation might be more advantageous at higher velocities, at which saturation might occur. It will be interesting to see whether cells selective for centrifugal motion are less susceptible to

decreased responsiveness due to prolonged adaptation, as a possible means of distinguishing between these two alternatives.

Eccentricity

Another characteristic of the MAE for which an explanation based on neural processes should be sought is the differential strength of the MAE in foveal versus peripheral vision. Recall that most researchers (Szily, 1905; Cords & von Brücke, 1907; Wohlgemuth, 1911; Granit, 1928; Freud, 1964) reported that while peripheral MAEs were initially stronger, they were shorter in duration, and that there were qualitative, not simply quantitative, differences in central and peripheral MAEs. Some researchers have related this difference to the differential contributions of rods and cones to perception at different eccentricities (e.g. Wohlgemuth, 1911; Granit, 1928).

Currently, attempts at explanations based on characteristics of direction-selective cells seem more desirable, yet remain elusive. Orban, Kennedy and Bullier (1986) found that direction-selectivity of cells in V1 of monkey tends to decrease with increasing eccentricity. In addition, they found that direction-selective cells at greater eccentricities are sensitive to higher velocities than their less peripheral counterparts. This was also reported in studies of cat area 17 cells (Orban, et al, 1981b; Orban, et al, 1981a) and 18 cells (Orban, Kennedy & Maes, 1981b) and area 19 cells (Duysens, Orban, van der Glas & de Zegher, 1982). Cells possessing receptive field in the center of the visual field, on the other hand, retain direction-selectivity at lower velocities than do those with peripheral receptive fields. Differential adaptability or recovery of peripheral versus foveal direction-selective cells, however, has not yet been reported. And Orban, Kennedy and Maes (1981a) note that fewer direction-selective cells are found as eccentricity increases. Thus it would be premature to attribute MAE differences with eccentricity to the physiological results to date, but hopefully further research will enable this relationship to be elucidated.

PSYCHOPHYSICAL EVIDENCE REGARDING THE RATIO MODEL

As mentioned above, the first to propose the so-called ratio model as an explanation of MAEs was Sutherland (1961), who postulated differential fatigue of cells selective for different directions

of motion (already reported by Hubel & Wiesel, 1959), yielding an imbalance in the outputs of these direction channels. The study by Barlow & Hill (1963b) presented the first physiological evidence of direction-selective effects of adaptation, and thus provided the first opportunity for an evaluation of the model. Sekuler and Pantle (1967) were the first to elaborate the model and some of its predictions, and to conduct experiments aimed at evaluating the model's validity. The ratio model is currently the major (and possibly only) proposed mechanism for the neural basis of MAEs. Perception textbooks and courses commonly teach as gospel this explanation of MAEs based on direction-detectors and differential fatigue following adaptation to a single direction. However, the literature is equivocal in its evaluation of the model. The relevant physiological work has already been discussed above. Now I will review some of the major psychophysical findings and their implications for acceptance of the ratio model.

Threshold elevation: direction-selectivity

One phenomenon related to motion adaptation which can be investigated without reference to actual MAEs is threshold elevation. Following adaptation to a grating moving in a given direction, the luminance threshold and contrast threshold for the same grating in the same direction are found to increase, indicating that it is more difficult to detect a grating following adaptation. Several studies have investigated threshold elevation following motion adaptation, and many of their results have implications for acceptance of the ratio model as the basis underlying direction discrimination and MAE generation. One might predict that threshold elevation would be direction-selective, based on the evidence discussed earlier suggesting that adaptation at the single cell level is direction-selective. On the other hand, the existence of bidirectional cells has been suggested as reason to expect elevation in the opposite direction as well (Vautin & Berkley, 1977).

Sekuler and Ganz (1963) found using a stabilized adapting stimulus that the luminance threshold is higher in the adapted than in the unadapted direction of motion. They did not, however, measure preadaptation threshold levels, so the degrees of absolute elevation in each direction can not be determined, nor can it be determined

whether threshold was actually affected for the opposite direction. Pantle and Sekuler (1969), however, measured absolute, rather than relative threshold elevation. They showed that the luminance threshold increases as adapting grating contrast increases. This increase occurred for both adapted and unadapted directions, but was larger in the adapted direction. Likewise, Tolhurst (1973) and Sharpe and Tolhurst (1973) found that contrast threshold elevation does occur after adaptation to motion in the opposite direction; Tolhurst also noted the lesser degree of elevation for the opposite direction.

Moulden and Mather (1978), however, claimed that according to the ratio model, the threshold for motion in the direction opposite to the adapted direction should actually be *decreased* relative to the unadapted condition. This facilitation had not been found psychophysically (e.g. Pantle & Sekuler, 1969), and as discussed earlier, the physiological studies of single cell responses to motion following adaptation to motion in the opposite direction are contradictory (e.g. Hammond, et al, 1985, 1988; Hammond & Mouat, 1988, Vautin & Berkley, 1977; Hammond, et al, 1989). The authors suspected that previous failures to find psychophysical facilitation could be attributed to the absence in the previous studies of a measure of orientation-specific adaptation. They thus included a condition neglected by previous studies: adaptation to a stationary grating. The threshold elevation produced by adapting to the stationary grating represents orientation-specific adaptation, independent of direction. It is to this condition that the other conditions should be compared to determine whether adapting to motion in either direction produced threshold elevation or facilitation.

Subjects adapted to either a homogeneous field (of the same space-averaged luminance as the other stimuli), a stationary grating which reversed phase every 1 sec, a rightward-moving grating, or a leftward-moving grating. Post-adaptation thresholds were measured for a rightward-moving grating. The authors reported that thresholds were elevated for adaptation in the same direction, relative to those for adaptation to the stationary grating. The key finding was that facilitation was in fact observed: the threshold elevation produced by adaptation to motion in the opposite direction was less than that

produced by adaptation to the stationary grating. Thus it is possible that had the previous studies accounted for orientation-selective effects, facilitation would have been found, supporting the ratio model.

Interactions of motion adaptation in opposite directions

Independence of direction detection at threshold

In order to evaluate the validity of the ratio model as the basis for the MAE, we must also evaluate its validity as the basis of motion detection, or more specifically, direction detection. One major controversy is the issue of whether the ratio model holds at threshold. That is, are motions of opposite directions detected independently at threshold, or do interactions occur as predicted by the ratio model? Several studies examined the implications of the effects of adaptation to motion on subsequent thresholds, with conflicting results.

Sekuler and his colleagues (Sekuler, Lehr, Stone & Wolf, 1971) claimed that the ratio model cannot hold at threshold, based on their experiments suggesting that the visual system shows independence at threshold. The basis for their experiment is the claim (Sekuler & Levinson, 1974) that adaptation to both leftward and rightward motion should reduce the sensitivities of both direction channels equally, so that both components of the ratio between the two responses are reduced, leaving the ratio itself unaffected.[2]

In one condition, subjects adapted alternately to grating motion in opposite directions. In another, subjects adapted alternately to motion in the tested direction and to a stationary grating. The authors report that alternate adaptation to the two opposite directions produces the same contrast threshold elevation for the tested direction as does adapting alternately to motion and a stationary target. They thus claim that movement of different directions is

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2. This prediction is of necessity based on the premise that the "ratio" of responses which determines the perceived direction of motion is actually a ratio or quotient, rather than a difference, and that adaptation results in a reduction by division of a cell's response. Their conclusions would not hold if in fact it is the difference, rather than the ratio, of responsivity that determines motion detection, as multiplying or dividing the two components by the same amount would change the resulting difference (by that same amount). Likewise, if one conceives of adaptation as subtracting from a cell's response, rather than dividing it, then an actual ratio between two responses *would be* affected by adapting each direction equally, while a difference between the two would not.

detected independently at threshold, since otherwise the adaptation to motion in the opposite direction should counteract, so to speak, the adaptation from motion in the original direction and result in no threshold elevation. They do not, however, object to the applicability of a ratio model for movement detection at suprathreshold levels, suggesting that while the MAE depends on the relative responsiveness of opposite direction channels, the threshold for detecting a particular direction of motion depends only on the sensitivity of that direction channel. Thus the applicability of the ratio model at threshold does not necessarily relate to its utility in explaining the MAE.

Note that in the experiment of Sekuler et al (1971), subjects were required merely to detect the grating, not to discriminate its direction of motion. As pointed out by Sekuler, Ball, Tynan and Machamer (1982), detection of a moving target may be easier than an actual direction-discrimination task. It is thus possible that a different result would obtain were the threshold for discrimination measured, so that directional interactions predicted by the ratio model might be observed.

In fact, Mather and Moulden (1983) obtained results supporting this notion. Subjects viewed a field of moving random dots, either with all dots moving in one direction, or half in one direction and half in the opposite direction. Dot density was double in the bidirectional case. The subject's task was to adjust the contrast until the direction of motion for the unidirectional stimulus was just visible, or until either direction was visible for the bidirectional case was just visible. (Note that no adaptation is involved; subjects measured unadapted thresholds only.) Thresholds were found to be higher in the bidirectional than the unidirectional case, an effect which was not attributable to the increased dot density. These results thus suggest that detection of opposite directions of motion is not independent. The authors do suggest that detection may be independent when only contrast detection, rather than direction detection, is examined.

Watson, Thompson, Murphy and Nachmias (1980) had also provided evidence that detection of a moving stimulus and identification of its direction of motion must be distinguished. Unlike a previous experiment by Levinson and Sekuler (1975b), which

determined that opposite directions are detected independently, their analysis also took into account probability summation between independent channels perturbed by uncorrelated noise. They determined that for high TF and low SF (high velocities), detection of opposite directions is direction-selective, and identification threshold is equal to detection threshold. For high SF and low TF (low velocities), however, detection is not direction-selective, and the threshold for identifying direction of motion is higher than that for detecting motion. That is, at low velocities, summation between opposite directions occurs, indicating nonindependence. At higher velocities, however, summation does not occur, and detection of opposite directions is independent.

These results support the notion that independence applies only to mere detection of a moving stimulus, and not to detection of actual direction of motion. Sekuler (1975) suggests that detection of motion depends solely on the response of the most sensitive mechanism, and not on interactions between direction-selective mechanisms.

Watson et al (1981) pose two possible interpretations of their results. It is possible that a given direction-selective mechanism may possess a small degree of sensitivity to motion in the opposite direction, which declines as velocity increases. Thus at lower velocities, some summation would occur, yielding nonindependence. At higher velocities, summation would not occur, and detection would be independent. On the other hand, distinct direction-selective and direction-nonselective mechanisms might exist. In this case, selective mechanisms might be more sensitive at high velocities, whereas nonselective mechanisms would determine detection at low velocities. Therefore at low velocities, detection of motion would be determined by the sensitivity of the nonselective mechanism, whereas discrimination of direction would of necessity rely on the selective mechanism, yielding different thresholds for the two tasks. At higher velocities, the direction selective mechanism would be responsible for both detection tasks, resulting in equivalent thresholds.

The latter possibility (separate mechanisms) is supported by Watson's (1977) previous finding that no summation between high and low TFs occurs. Watson et al (1981) also point out the similarities

between the proposed characteristics of these two mechanisms and those proposed for the putative transient and sustained mechanisms (Kulikowski & Tolhurst, 1973).

Other studies do show that detection of opposite directions of motion is not independent. Levinson and Sekuler (1975a) wondered if the threshold elevations found for adaptation in the same and opposite directions would be combined, so that adapting simultaneously to gratings moving in opposite directions would produce higher threshold elevation than would adapting to either direction alone. They found, rather, that *less* elevation resulted when subjects adapted to such a composite grating. This supports the notion inherent in the ratio model, that adaptation effects are due to imbalances in responses to different directions resulting from adaptation to motion in a given direction. Thus, this finding is easily explained, in that adaptation due to the added opposite grating counteracted, or balanced, the adaptation effects produced by the original grating. That is, since cells for the opposite direction also became somewhat adapted, the imbalance was not as great as when the original grating was presented alone. This also explains their finding that less threshold elevation occurred as the contrast of the opposite grating increased, since the adaptation of the cells for opposite motion would be increasingly adapted, narrowing the gap between the responses of the opposite direction "channels".

Similar reasoning can explain their additional finding that this inhibition of threshold elevation due to presence of motion in the opposite direction can be reversed by prior adaptation to the opposite direction. In this case, the adaptation conditions were the same: a grating moving rightward, or this same grating combined with a grating moving leftward. However, a preadapting period of exposure to the leftward moving grating or a homogeneous field preceded adaptation. In this case it was found that the effect of the added leftward grating during adaptation was decreased. This can be explained on the basis that preadaptation to the leftward grating resulted in an initial decrease in responsiveness to leftward motion, so that further adaptation during the adaptation period produced little further decrease, whereas adapting to the rightward grating was still

effective at adapting the rightward motion channel, so that the amount of threshold elevation observed for the combined gratings was similar to that observed for the rightward grating alone.

There seems to be a discrepancy between the results of Sekuler et al (1971) and those of Levinson and Sekuler (1975N). The alternating adaptation employed in the experiment of Sekuler et al (1971) did not yield differential threshold elevation when motion was combined with opposite motion as opposed to being combined with a stationary grating. Simultaneous adaptation to opposite directions of motion in the experiment of Levinson and Sekuler (1975N), however, *did* show an interaction between opposite motions. It is unclear how this discrepancy can be resolved. The paradigms are equivalent, except for the temporal factor in the adaptation period. Thus it is not possible to explain the difference in results in terms of Sekuler's (1975) claim that while motion perception at threshold shows independence, motion perception at suprathreshold levels shows inhibitory interactions. Both experiments utilized adaptation at suprathreshold levels, combined with subsequent measurements of threshold detection.

One possible explanation is that summation can only occur when the two directions of motion are presented simultaneously. This is conceivable, if one considers the presence of cells in the visual system whose responses are modified by the presence of motion in the opposite direction (e.g. Frost & Nakayama, 1983). Thus, presentation of opposite motions in alternation would differentially adapt cells such as these, while more conventional directional-selective cells would be adapted equally by simultaneous or alternating adaptation (except of course for the opportunity for dissipation of effects in the alternating case, which is not present in the simultaneous condition, and the need to control for total duration of adaptation to each direction). These cells respond preferentially to motion in opposite directions, and might thus be adapted to a greater extent in the simultaneous condition. This greater adaptation would be manifested as a greater degree of threshold elevation. This is a preliminary suggestion, however, and as the cells investigated by Frost and Nakayama were in

the pigeon tectum, it remains to be seen whether analogous cells exist in primates.

Moulden and Mather (1978) have claimed not only that Sekuler et al's (1971) results fail to disprove the ratio model, but that they are in fact consistent with it and fulfill the model's predictions. They explain that it is necessary to differentiate between a change in contrast thresholds and a change in the ratio of firing rates. That is, just because contrast threshold is raised (or not) does not mean that the firing rate ratio is changed (or not). In addition, they point out a fallacy of most adaptation experiments, which do not take into account the orientation-specific component of adaptation, which contributes to adaptation both for same and opposite directions of motion. Sekuler apparently accepted this argument in a personal communication to the authors, but still held his results as disproof of the ratio model (Sekuler & Levinson, 1974). However, Moulden and Mather showed, based on predicted relationships between contrast and firing rate for unadapted and adapted units, that the ratio model actually predicts Sekuler et al's (1971) result. In their interpretation of the ratio model, the contrast-response pattern of a direction-selective unit is decreased overall by prolonged activity, such that the response shown as a plot of firing rate versus contrast is either shifted to the right, decreased in slope, or both, following adaptation (see Fig. 4).

They claim that the particulars of the decreased response -- in terms of a shift to the right vs. a change in slope in a plot of response as a function of contrast -- are not important for the model, so long as the response profile of the adapted unit lies completely below the unadapted profile. That is, the response at any given contrast is always lower for an adapted than an unadapted unit, as in their Figure 1 (Fig. 4). Several studies do provide some evidence for the nature of the change in contrast response properties following adaptation. Figure 2A of Movshon and Lennie's report (1979) depicts a change in contrast response characterized by both a shift to the right and a change in slope. Likewise, Albrecht, Farrar and Hamilton (1984) found that adaptation affects the contrast response function of cells via both a sensitivity shift (a shift to the right on a plot of response vs. contrast)

and a compression shift (a decrease in the maximum response and thus a downward shift).

In order for motion to be detected, Moulden and Mather propose, a threshold value of the ratio between firing rates for units for opposite directions of motion must be exceeded (refer to Fig 4). They proceed to show that for an unadapted system, the contrast necessary to elicit a motion response is constant, at a level C_2, representing the unadapted contrast threshold for motion in a given direction. Next, they show that adaptation of units selective for one direction of motion results in an increase (from C_2, to C_4) in the contrast necessary to produce elevation in firing rate of these units relative to units for the opposite direction. Thus, the firing rate ratio is unchanged, yet threshold elevation in the adapted direction occurs. They further show that adaptation to one direction of motion results in a specific contrast above which a stationary target will seem to move in the unadapted direction. (One can thus predict that if MAEs are measured at near-threshold contrast levels, there will be a threshold contrast level below which no MAE will be observed, although the stimulus will be visible. This would be an interesting test of their predictions.)

Finally, Moulden and Mather show that when units sensitive to opposite directions of motion are equally adapted, the contrast thresholds for both directions are elevated (from C_2 to C_3), although the ratio of firing rates is unchanged. In fact, for the specific situation illustrated, the threshold elevation produced by adaptation to both directions is only slightly lower than that produced by adaptation to the tested direction alone. Recall that Sekuler, et al (1971) showed that adapting alternately to opposite directions of motion resulted in essentially the same threshold elevation as did adapting alternately to motion and a stationary target. Moulden and Mather suggest here that this result does not indicate that the ratio model does not hold at threshold, and is in fact a result that the ratio model would predict. However, one must note that this might be in part an artefact of the specific response characteristics illustrated by Moulden and Mather, in which case it is likely that with somewhat different parameters (specifically a shallower slope for the adapted response), these two

types of adaptation would be expected to produce different degrees of threshold elevation (see Fig 5). (In fact, the change in slope illustrated by Movshon and Lennie (1979) was a decrease in slope, rather than an increase as suggested by Moulden and Mather.) If so, the problem with Sekuler et al's result would be not that there *was* a threshold elevation for adaptation to opposite directions of motion, but that this elevation did not differ at all from that produced by adapting to one direction alone.

Direction-selectivity of perceived velocity shift

Another phenomenon related to MAEs is the change in perceived velocity occurring after prolonged adaptation to motion. It is unclear whether this effect is solely the function of adaptation to a given direction of motion, or whether adaptation to a specific velocity also has differential effects. However, many findings regarding perceived velocity shifts are relevant to a discussion of the ratio model and of the direction-selectivity of the effects of motion adaptation.

As Barlow and Hill (1963b) mentioned, the velocity of the MAE is rather slow and phenomenally weak, a characteristic easily explained (as they suggest) by the fact that the magnitude of adaptation effects is limited by the zero level of firing. The spontaneous activity of unadapted units is not high to begin with, and thus the difference between these and adapted cells' firing rates can not be large. However, the imbalance does of course produce a substantial MAE with noticeable velocity, and also is sufficient to alter our perception of the velocity of objective motion.

Cords and von Brücke (1907) were the first to report that adaptation to motion can alter the perceived velocity of real motion, showing that the MAE can slow or cancel objective motion in the opposite direction and even reverse it. They apparently pursued this investigation following a statement by Kleinert (1878) suggesting that the MAE could add to objective motion. Wohlgemuth (1911) confirmed their findings. Gibson (1937) also found a decrease in velocity following prolonged viewing of motion. Goldstein (1957) systematically investigated the changes in perceived velocity as a function of duration and velocity of motion adaptation. He found that the magnitude of the decrease in apparent velocity increased with

adaptation durations of 2 to 22 seconds, then leveled off for durations up to 60 seconds. Taylor (1963b) also examined the perceived velocity decrease which accompanies prolonged viewing of motion. He found that the mean log rate of perceived velocity change increased as the square root of adapting inspection duration up to at least 10 minutes. He also reported that as actual velocity increased from 2.2 to 133.2 deg/sec, the rate of change in perceived velocity decreased.

Carlson (1962) was the first to show that the change in perceived rotational velocity depended on the velocity of the test stimulus relative to that of the adapting stimulus. He also examined the direction-specificity of the effect. For test motion in the same direction as adapting motion, decreased apparent velocity resulted for test velocity below or equal to the adapting velocity, while increased apparent velocity was found for test velocities greater than the adapting velocity. No significant effects were seen for motion in the opposite direction, suggesting that the effect was direction-selective.

Carlson speculated that the recently discovered direction-selective cells (Hubel & Wiesel, 1959) might be involved in this phenomenon. Thus, adapting to motion in one direction would fatigue those cells selective for that direction, so that velocity would be underestimated for motion in the same direction. Velocity perception in the opposite direction would be unaffected. Note, however, that if velocity coding interacts with direction coding, with the decreased response in the opposite direction affecting velocity in a direction-specific manner, then velocity might be expected to increase for the opposite direction. This is not as obscure an idea as it may first seem, as the subjective motion in the direction opposite to adaptation, characteristic of the MAE, might conceivably add to the objective motion in that opposite direction, resulting in an increase in perceived velocity. Thus, velocity would be coded in terms of both magnitude and direction, with magnitude coded on a continuum, and direction in an opposing manner as with direction-selective cells. On the other hand, the contributions of bidirectional and partially direction-selective (direction-biased) cells, whose responses for null motion might actually be depressed along with those for preferred

motion (see section on direction-selective cells), might result in decreases in perceived velocity even for the opposite direction.

The issue was further explored by Scott, Jordan and Powell (1963) , who found that the perceived velocity of expansion or contraction is indeed increased following adaptation to motion in the opposite direction. This is contrary to the results of Carlson (1962), who found no change for motion in the opposite direction, and suggests that the MAE actually adds to objective motion. Their results with similar experiments on a rhesus monkey also supported the notion of algebraic additivity of MAE to objective motion.

Rapoport (1964) , like Carlson, found that the direction of perceived velocity shift for motion in the same direction depended on the test velocity relative to adapting velocity. However, unlike Carlson, she found that at the highest of the three adapting velocities used (72 deg of rotation per second), apparent velocity for motion in the opposite direction changed in the same manner as for motion in the same direction. That is, for test velocities less than or equal to the adapting velocity, velocity was reduced, while for higher test velocities it was increased. She also noted that a minimum separation of adapting and test velocities seemed necessary in order to obtain a shift in perceived velocity.

These results suggest that the observed velocity shifts may be the result of velocity-specific adaptation, corresponding to a "place-coding" mechanism for velocity coding, and not simply a result of direction-specific adaptation. The place-coding scheme was given its name because of its original use in explaining frequency coding in the auditory system, which involves position along the basilar membrane (von Békésy, 1960). According to this scheme, velocity is coded along a continuum by velocity-selective units with bandpass velocity responses. The adaptation of one of these sets of cells or velocity "channels" results in a decreased responsiveness of those cells, so that when the outputs of the various channels are compared, the balance is shifted away from that velocity. This would explain the differential effects of test velocities above or below the adapting velocity, as a shift away from the adapting velocity would result in an increase for higher velocities, while producing a decrease for lower velocities. This would

also account for Rapoport's observation that a minimum separation between adapting and test velocities is necessary, as the effects of adaptation of a velocity channel will only be manifested for objective velocities somewhat above or below the adapting velocity.

Note that Sekuler and Pantle (1967) reached the conclusion that adaptation to motion does not involve velocity-selective adaptation. Their reasoning (based on the assumption that MAE velocity should be independent of adaptation duration if velocity-specific adaptation is involved) is not obvious, however, and it may be possible to find a flaw. Thus their findings may not actually be in contradiction with those of Rapoport.

Thompson (1981) reported the same results as Carlson and Rapoport for velocities below the adapting velocity. However, he found that there was either a decrease or no change in apparent velocity for test velocities above the adapting velocity, and suggests that in previous studies, the results were confounded by contrast effects, as adaptation has been shown to cause decreases in apparent contrast (Blakemore, Muncey & Ridley, 1973). He specifically proposes that at high velocities, the decrease in perceived contrast can lead to overestimation of velocity. Subsequently, Smith (1985) confirmed Thompson's finding that perceived velocity never increased, and that it decreased only for test velocities less than or equal to the adapting velocity. He also found that there is a minimum velocity, below which the velocity shift does not vary with test velocity. He further noted that the decrease in apparent velocity was only half the magnitude for the opposite direction as for the same direction, and suggests that the effect must be partially direction-selective.

Clearly the results of these studies are somewhat contradictory, and an explanation of all observed effects remains to be proposed. However, it seems apparent that the velocity shift must result both from direction-selective and velocity-selective adaptation. The interactions between these two types of selectivity at the single cell or cell aggregate level might explain the various observed effects. The adaptation of velocity-sensitive units certainly has ramifications in terms of the basis of the MAE, but a thorough examination of the role of such adaptation is beyond the scope of this paper.

Resultant MAEs for adaptation in opposite directions

Wohlgemuth (1911) found that if one adapts in alternation to motion in opposite directions, the MAE obtained is a combination of the two MAEs produced under separate adaptation conditions. When adaptation in one direction was brief, and thus produced a shorter MAE, the resultant MAE was a sum of the two individual MAEs, with no MAE apparent for a few seconds, and then a MAE corresponding to the direction of longer adaptation.

Similarly, Woolsey and Newman (1973) showed that simultaneous adaptation to stimuli rotating in opposite directions produces little or no MAE, with the MAE obtained attributable to the slightly higher level of adaptation produced by the front rotating disk, which, although partially transparent, did somewhat occlude the disk behind it. This confirms the operation of a mechanism for MAE production by which adaptation to opposite motions counterbalance each other, as the ratio model would predict. Adaptation to opposite directions of motion results in equal adaptation of cells selective for each direction. Thus, little or no net imbalance in the outputs of these two groups of cells occurs, and little or no MAE results.

Interactions of motion adaptation in nonopposite directions

In addition to interactions between adaptation to opposite directions, one needs to consider the predicted result for motions in various other combinations, in order to fully evaluate the ratio model. It is unclear whether the ratio model as originally envisioned by Sutherland (1961) entailed merely a ratio between outputs for opposite directions of motion, or whether he intended that the model should comprise a comparison among outputs for all directions. A model of the latter type would be similar to a place-coding scheme (von Békésy, 1929) for variables such as SF, except that it is possible to have opposite directions of motion, but not opposite SFs. Thus, the distribution of SF-selectivity can be envisioned as a linear continuum, with some cells responsive to low SF, some to high SF, and some to intermediate SFs, each with bandpass characteristics. The distribution of direction-selectivity is similar, except that it is more properly illustrated as circular (as in Fig. 3), due to the opponent nature of direction. Regardless of whether the model encompasses all

directions or only two opposite directions, the predictions regarding motion in opposite directions are identical. The predictions for interactions between other motion combinations, however, depend on one's interpretation of this aspect of the model.

Threshold elevation

Levinson and Sekuler (1980) demonstrated that following adaptation to a coherently moving field of random dots, luminance thresholds for detecting similar patterns moving in various directions are elevated. Note that this measure is unconfounded by differences in orientation, as would occur for drifting gratings. This elevation was maximal for identical adapting and test directions, but was still evident even with differences of 45 deg. Thus, detection of the test motion was affected by motion in directions other than the same and opposite direction, indicating that detection involves interactions between detectors of nonopposite motions. An alternative interpretation is that a single direction channel was involved, but had broad tuning (showing responses for up to 45 degrees from optimal direction), and was adapted to a decreasing degree as the direction of the adapting pattern was increasingly different from the test direction.

Composite MAEs

If only outputs for opposite directions are compared, then adapting to two orthogonal motions might be expected to produce two separable MAEs, which might perhaps be visible simultaneously. On the other hand, if all direction-selective outputs are compared, we would expect the resultant MAE to be an average, or vector sum, of the individual MAEs.

Wohlgemuth (1911) reported that after adapting to several directions simultaneously or successively, the resulting MAE is in fact what would be predicted on the basis of a combination of the individual MAEs. Riggs and Day (1980) found that following alternate adaptation to two directions of motions, the MAE observed was opposite the resultant of the two adapting motion vectors. This result obtained for both gratings and dot patterns, so that orientation was not a confounding factor.

Mather (1980) obtained similar results. When subjects adapted to two superimposed motions with varying degrees of difference in

direction of motion, the direction and duration of the subsequent MAE were always in accordance with a model which compares the outputs of the entire distribution of direction-selective cells and yields an MAE which is an average of the individual MAEs. The author suggests that summation of MAEs occurs when the two motion directions are within the same bandwidth, and so are presumably activating the same set of direction-selective cells. Thus, the MAE duration decreases as the angular separation between the two adapting directions increases, and at a certain separation, summation no longer occurs and the MAE duration corresponds to a zero summation function, which predicts even shorter durations. The use of random dot patterns instead of gratings negates the possible objection that orientation effects confound the results.

Perceived direction shift

Another piece of evidence supporting the role of the entire distribution of directional responses in motion detection is the existence of shifts in perceived direction of motion following adaptation to a given direction. Levinson and Sekuler (1976) reported such a shift following adaptation to a random dot field drifting uniformly in a given direction. This shift did not occur for motion in the same direction as the adapting motion, but a large (10-degree) shift did was obtained for motion in a direction differing by 30 degrees from the adapting direction. That is, if subjects adapted to motion 30 degrees clockwise (CW) or counterclockwise (CCW) from rightward, subsequent rightward motion seemed to be moving 10 degrees CCW or CW from rightward.

This finding can be explained with reference to the type of diagram employed earlier to illustrate the imbalance due to adaptation which is thought to produce the MAE. In Figure 6a, the unadapted responses for all directions are equal and at a maximum. That is, prior to adaptation, an equivalent stimulus in any direction will produce an equivalent response (except perhaps for the presence of anisotropy, which should not affect the predictions). Following adaptation to motion to the right, responses are most depressed for the adapted direction, but are also diminished for directions within a certain range on either side. For our purposes the specific range need not be exact,

but let us say that adaptation affects cells responsive to directions up to 90 degrees from the adapted direction, with the effect reduced for directions increasingly removed from the adaptation direction. Thus, adaptation to rightward motion would produce a distribution of responses resembling that shown in Fig 6b. If rightward motion is then viewed, cells responsive to directions up to 90 degrees away on either side will be stimulated. Note, however, that while these cells have all been adapted, the effects of adaptation are symmetrical, so that the reduction in responsiveness of cells for directions equally removed from rightward on either side are equally reduced. Thus, a comparison of the relevant cell outputs will result in the perception of rightward motion, as no shift in responses relative to the tested direction has occurred.

Now consider the situation, depicted in Figure 6c, where motion 30 degrees CCW from rightward is tested. In this situation, the decrease in responsiveness relative to the tested direction is asymmetrical. Thus, while motion in this direction would normally produce equivalent responses for cells selective for directions equally removed on either side of this direction, the responses are now biased in the CW direction (away from the adapted direction). Motion therefore appears to be shifted in the CW direction from rightward. The authors found that this shift was lower for directions 60 degrees away from the adapted direction, with only minimal shifts at 90 or 120 degrees away.

Levinson and Sekuler further determined that this shift in perceived direction is not due merely to a perceptual addition of an MAE to the objective motion direction. They measured the perceived direction shift produced by adapting to motion 30 degrees CCW from upward, and testing upward motion. They then measured the shift produced for identical test motion, but with simultaneous adaptation to motion 30 degrees CCW from upward and 150 degrees CW from upward -- that is, to equal and opposite motions. In this case, no MAE is produced, as the adaptation produced by each direction cancels out, as discussed above. They found that no reduction in perceived direction shift occurred when the opposite adapting motion,

indicating that the presence of an MAE was not necessary for this shift.

Their findings can be explained with reference to the distributions of responses for different directions resulting from the two types of adaptation. In Figure 7a, we see the usual unadapted response pattern. In Figure 7b, the response distribution resulting from adaptation to the 30 degree CCW motion alone is shown. Again, responses decrease most for the adapted direction, with lesser adaptation for directions further from this direction up to orthogonal directions, at which no reduction occurs. If motion in the upward direction is now viewed, responses are asymmetrically affected relative to the test direction, so that responses for motions CW of the test direction (again, away from the adapted direction) are higher. Thus, upward motion actually appears to move slightly CW of upward.

Next, consider adaptation to opposite directions. The resulting response pattern is illustrated in Figure 7c. Note that responses are decreased in all directions, except those orthogonal to the adapting directions, and the adaptation effects produced by the opposite adapting directions are equivalent. In comparing this to Figure 7b, it is apparent that the responses to directions near the test direction (upward) are virtually identical in the two cases. The only difference lies in the region from 60-90 degrees CW from upward, which is only minimally involved in detection of upward motion. Thus the perceived direction shift in this situation is virtually unchanged from that produced by adaptation to the single direction of motion alone. (The adaptation in the region differing for the two situations would be expected to produce a slight decrease in the magnitude of the shift, but again, this effect is minimal due to the large difference in these directions from the tested direction.)

Marshak and Sekuler (1979) reported a similar effect in the domain of perception of simultaneous motions. They showed that when two drifting random dot fields are presented moving in different directions, a misperception of the difference in direction occurs, so that the directions seem to be more different than they actually are. This perceived error increased as the actual difference in direction increased up to about 22.5 degrees, with no error for differences

above 90 degrees. Mather and Moulden (1980) obtained similar results; however, they did not examine differences below 20 degrees, and thus reported a simple decrease in perceptual error as the difference in direction increased up to 90 degrees, with no error for greater differences. They further noted that results were identical for fixating and tracking conditions. These findings emphasize the interactions of nonopposite directions, and suggest the existence of inhibition between direction-selective mechanisms.

Ratio model vs. distribution-shift model

Mather (1980) proposed that his evidence shows the inadequacy of the ratio model as an explanation of MAE generation, and suggested instead the distribution-shift model, which entails a comparison of the responses for direction-selective elements tuned to all directions, not just to opposite directions. This model does in fact seem to account for the observed findings regarding direction interactions, as discussed above. Realize, however, that this model is more of an extension of the ratio model than a substitute for it, especially in light of our inability to determine the originator's (Sutherland, 1961) intent regarding non-opposite directions of motion. Mather and Moulden (1983) in fact referred to the distribution-shift model as an extended ratio model, rather than a replacement for the ratio model.

It seems difficult to envision a process whereby the visual system would independently code opposite directions of motion, without any interaction between these direction pairs. What *would* one see under this mechanism after adaptation to two directions of motion simultaneously? It seems rather unintuitive to suggest that a stationary object would at once appear to move in two separate directions. The only plausible result would be a form of monocular rivalry, whereby the two MAEs would be observed in alternation, a finding which still seems less parsimonious than a simple resultant MAE. Thus it is not surprising that motion detection and generation of MAEs involve comparisons across the entire continuum, rather than being restricted to comparisons of opposite directions alone.

STORAGE OF MOTION AFTEREFFECTS

In addition to observing MAEs on a stationary version of the adapting stimulus or some other stationary object (an "objective field"),

researchers have also reported that an MAE can be observed in the subjective field, that is, with the eyes closed. Brewster (1845) was the first to note this phenomenon. Wohlgemuth (1911) claimed that while the MAE is easier to observe in the objective field, it lasts longer when viewed in the subjective field. This observation is related to another phenomenon reported by Wohlgemuth (1911): if the eyes were closed immediately following the adaptation period, so that the test stimulus was not viewed until a period of time equal to the normal duration of the MAE had elapsed, an MAE was still observed at the time. That is, the MAE was still apparent at a time when, had the eyes been left open and the test target viewed continuously, the MAE would have already completely decayed. This phenomenon was extensively studied by Spigel (1960, 1962, 1964), who termed it "storage", as the MAE seems to be stored during the period of eye closure.

One fault inherent in the ratio model is its failure to account for the phenomenon of storage. The model asserts simply that the MAE results from fatigue of direction-selective neurons due to prolonged stimulation, leading to an imbalance of neurons selective for different directions. Thus recovery from adaptation, corresponding to decay of the MAE, is conceptualized as a passive recovery of neuronal responsiveness due to the passage of time, and cannot explain the effect of differential post-adaptation stimulation. Storage thus poses a definite problem for a simple neural fatigue and recovery model of MAEs, and as such merits a detailed discussion.

Spigel (1960) first measured the mean MAE duration for each subject. At the next session, subjects adapted to a rotating spiral, then sat in the dark for the amount of time representing their mean MAE duration. Spigel reported that in this situation, subjects reliably saw a substantial MAE at this time, although it should have decayed almost fully by the time testing began. He concluded that the decay of the MAE is not simply a passive recovery process, in that stimulation following adaptation affects the rate of decay, such that the absence of stimulation produces an inhibition of the decay process. He noted that it was unclear whether the critical factor was the absence of light stimulation or the absence of contours.

Spigel (1962) examined storage further by measuring MAEs under five different conditions of postadaptation stimulation. Following adaptation, subjects were exposed to either (1) the test target (regular MAE), (2) darkness for the mean MAE duration, (3) a homogeneous field, (4) a dim homogeneous field, or (5) the test spiral for half the mean MAE duration followed by darkness for the remainder of that duration. The residual MAE (the duration of MAE following this pretest period) was significantly lower in the first condition than in all other conditions, indicating that storage had occurred to some degree in all other conditions. There was also a significant difference between the bright and dim homogeneous fields, and between the bright field and complete darkness, indicating that storage occurred more readily in the absence of bright light. (The difference between the dark and dim conditions was not significant.) These findings suggest that the presence or absence of contour is the critical factor in the decay of the MAE. (Spigel postulates that the bright field condition produced less storage due to the presence of some texture, yielding a stimulus that was not contour-free.) This finding is consistent with Griffith and Spitz's (1959) report that following adaptation to rotating spiral, an MAE can be observed on textured surfaces such as a plastered wall or a cloud-filled sky, but not on a cloudless bright region of the sky. The stored MAE is not as long as the immediate MAE, suggesting that there is some loss of adaptation effects independent of postadaptation stimulation.

Ross and Taylor (1964) found that the MAE decays more slowly when the illumination of the test pattern is decreased. This can be integrated with Spigel's (1962) findings, to suggest that illumination does play a role in storage, with lower illumination of a test stimulus prolonging decay of the MAE.

Spigel (1964) went on to determine that stimulation of the unadapted eye also contributes to the decay of the MAE (as discussed above regarding interocular transfer as in indicator of central mediation). Storage as measured in the adapted eye was decreased (that is, decay proceeded more normally) when the test stimulus was presented only to the unadapted eye, with the adapted eye in darkness, than when both eyes were in darkness. Spigel concluded

that the MAE must be mediated at a central locus, since, "if peripheral events predominated in the aftereffect phenomena, post-exposure conditions at the eye unstimulated by movement should have had relatively little or no influence on the" decay of the MAE in the adapted eye (p.244).

Sekuler and Pantle (1967) demonstrated another aspect of the storage of MAEs: storage can occur in delimited portions of the adaptation region. Subjects adapted to a rotating disk of radial lines, then shifted fixation for testing, so that only part of the region containing the test target during testing had been previously adapted. The MAE decreased as less of the adapted portion of the retina was exposed to the test stimulus. Subjects reported that when the tested MAE had ceased, they could still observe an MAE when they returned fixation to the point of fixation during adaptation. Thus, it is possible for the MAE to be stored in regions not tested, yet expressed in tested regions.

Long-term storage of MAEs

Masland (1969) was the first to demonstrate long-term storage of MAEs. Subjects adapted for 15 minutes to a rotating spiral, then returned in 20-26 hours, at which time they were still able to observe a MAE. (Subjects who had adapted to a stationary spiral did not see a MAE at this time.) As the storage period increased up to 15 minutes, the apparent speed of the MAE (measure using a nulling procedure) decreased, then remained constant with further increases up to 24 hours of storage. Masland suggested a habituation process as the cause of this phenomenon. He did not, unfortunately, report whether a significant difference was found between the group of subjects that were tested both immediately and after delay, and the group that was tested only after delay. However it seems obvious that an immediate MAE would not last 24 hours; therefore his results can be taken as further evidence for storage of the MAE.

Subsequently, Kalfin and Locke (1972) found that after adapting for 15 minutes to spiral motion, most of their subjects saw a MAE after 24 hours in the adapted eye, and half saw it in the unadapted eye. Thus, like the immediate MAE, the long-term stored MAE shows interocular transfer. In addition, Hershenson (1985) found that

subjects observed MAEs up to 72 hours after adapting to a rotating spiral for a mere 30 seconds, providing further evidence for the existence of long-term MAEs. Meeker and La Fong (1988) reported a much lower incidence of long-term MAEs. They conclude that long-term MAEs may be neither as general nor as easy to produce as others would have us believe. However, their brief report leaves out relevant details, such as the size and parameters of the adapting spiral, so it would be advisable to treat their results with caution. They point out that MAEs were reduced following a 5-minute pre-test delay relative to the immediate test condition, but do not comment on the relevance of the presence of an MAE after 5 minutes as evidence of storage. And a highly important detail is left out: the authors tested some subjects both immediately and after a 48-72 hour delay, but fail to mention whether there was any relationship between presence of a long-term MAE and occurrence of prior testing. It is thus difficult to draw any conclusions about storage from these results.

Cause of recovery: time vs. testing

The existence of storage in itself suggests that prior testing influences the decay and subsequent occurrence of the MAE. Clearly the passage of time also affects MAE decay: while MAEs are easily observed immediately after adaptation, the passage of time can diminish the occurrence or vividness of the MAE. Thus, Meeker and La Fong's (1988) result is evidence for the effect of time passage, as is Favreau's (1979) finding that while MAEs were still observed 186 hours after adaptation without prior testing, they were not apparent when the first test occurred 336 hours after adaptation. In addition, Keck and Pentz (1977) found that the initial velocity of the MAE decreased as the storage duration increased, supporting the role of time passage in MAE decay.

Keck and Pentz (1977) used storage of the aftereffect as a means of measuring the recovery rate of direction-selective analyzers in the absence of stimulation. They proposed that MAE decay results in part from spontaneous recovery of adapted cells, and in part due to stimulation by the test pattern. Keck, Palella and Pantle (1976) had previously determined that the duration of the MAE decreases with increasing test contrast. Thus zero contrast is the limiting case,

representing decay of the MAE due only to recovery of direction-selective analyzers. In order to indirectly assess this decay rate, MAE velocity was estimated following storage periods (during which subjects viewed a dim homogeneous field) ranging from 2 to 50 seconds. This initial velocity was taken as a measure of the initial MAE strength following a given duration (the storage period) of testing with exposure to zero contrast. This value was found to decrease with increasing storage duration, indicating some decay of MAE due to the passage of time, in the absence of stimulation. The decay followed an exponential function, with time constants of about 20 seconds. (The authors note that some illumination was present during the test period, and that the time constant increased when the eyes were closed during the storage period.) Unfortunately, comparable magnitude estimates for the nonstorage condition were not obtained, so that the degree of storage actually shown can not be assessed. The authors conclude that the time course of the MAE and the time course of recovery of direction-selective analyzers are not identical, as test conditions affect MAE decay. This is further support for the notion that the MAE involves more than simple adaptation and recovery of neural components.

Favreau (1979), in a study investigating the differences between contingent and simple MAEs, conducted a set of experiments in which some subjects were tested on a single occasion, and some on multiple occasions, for delays up to 168 hours. Thus the effect of repeated testing could be assessed. For each duration of delay, subjects were tested twice, with a 7-minute period between tests. It could therefore be determined whether passage of time alone affected decay of MAE, independent of prior testing.

Six of twelve subjects observed a MAE after a 168-hour delay in the single test group. In a subsequent experiment, no subjects observed MAEs at 336 hours. Favreau compared the difference in MAE duration between two consecutive tests within one session (delay condition) to the difference between the second test in one session and the first test in the next session. If the passage of time contributes to MAE decay, we would expect a greater difference in the latter case than in the former, since one additional test has occurred for both

conditions, but a greater amount of time has passed for the between-session comparison. For the repeated tests group, there was a nonsignificant trend for differences to be higher in the between-session comparison. (This trend also held, though not at a significant level, for the single-test groups. However the appropriateness of this analysis is questionable, in that the within-session comparison is also within-subjects, while the between-session comparison is between subjects.) The author suggests that the trend indicates a decline due to both time passage and effects of prior testing.

In a similar vein, she found that the differences between MAEs observed at a given delay duration for the single-test group and those for the repeated-test group were significant for only 2 of the seven durations (both tests for 7 hr, first test for 48 hr). Other durations resulted in nonsignificant trends in the appropriate direction. Thus the effect of repeated testing, or lack thereof, cannot be unequivocably established. While many of Favreau's results lack statistical significance, they are consistent with results of other studies (e.g. Keck & Pentz, 1977; Spigel, 1960, 1962, 1964), which indicate the contributions of both time passage and repeated testing to the decay of the MAE.

Clues from the role of the surround region

All of the studies of storage mentioned thus far have examined storage of spiral or rotational MAEs. Very few researchers have investigated the storage of linear MAEs, but several studies of one particular aspect of storage of linear MAEs have been conducted. Strelow and Day (1971) published two studies of the effect of surround characteristics on the MAE. In the first (Day & Strelow, 1971), they determined that MAEs are significantly reduced in the absence of a patterned surround. MAEs were somewhat reduced when either the adapting or test target was presented without a surround, and were even more reduced when both targets were presented in the absence of a surround. The reduction in MAE strength was greater when the test-stimulus as opposed to the adapting stimulus lacked a surround. Thus the effect of the absence of a surround was more extreme during the test period, but was also observed for the adaptation period. The luminance of the surround had very little effect through the range

0.11-11.17 cd/m^2, leading the authors to conclude that it is the absence of contour, not the absence of light that causes the decrement in the observed MAE. They also found that more MAE was observed for a patterned surround even when a plain surround was more luminous. Moreover, Bell, Lehmkuhle and Westendorf (1976) found that if a patterned surround is introduced halfway through the test period, the MAE magnitude increases. This effect is enhanced if a patterned surround is present during adaptation as well. Unlike Day and Strelow (1971), they found that increasing the luminance of the homogeneous surround during the test period did increase the MAE. When a homogeneous surround of luminance 3.1 cd/m^2 was introduced halfway through the test period, MAE velocity immediately increased. This increase was less than that produced by adding a patterned surround, however, and the nature of the surround during adaptation had no influence.

One possible explanation for the observed surround effects is that in the absence of a surround, the MAE is not perceived, although the neural process underlying the MAE continues normally. This seems conceivable in that motion is generally perceived in a relative manner (Gibson, 1966), so that motion of the target might not be observed without a stationary surround with which to compare it. This is thought to be the explanation for the fact that MAEs are generally not observed when the entire visual field is adapted (e.g. Wohlgemuth, 1911). A related suggestion, proposed by Strelow and Day (1971), is that in the absence of a surround, the threshold for detecting real and apparent motion could increase, so that the MAE fails to reach the threshold and is therefore not seen. They note that this is consistent with Brown's (1931) finding that the threshold for motion detection increases with surround homogeneity, although they have been unable to replicate Brown's finding of a reduction in apparent velocity of real movement in the absence of a surround.

However, the second study by Strelow and Day (1971) indicates that these explanations are not correct. They found that in the absence of a surround, the MAE is in fact stored. In one condition the surround was continuously visible during both adaptation and test. In the other

condition, the surround was absent for the first half of the test period, and replaced for the second half. The initial MAE magnitudes are similar in both conditions, but in the absence of a surround, the MAE decreases in magnitude rapidly, while the MAE with the surround present decreases rapidly. When the surround is reintroduced after half of the 20-second test period has elapsed, the magnitude of the MAE again increases, so that it is higher than that in the continuous-surround condition. This MAE is also prolonged relative to the regular MAE. Thus it seems that the absence of a patterned surround results in storage of the MAE.

The finding that storage occurs in the absence of a surround renders inadequate any explanations of the surround effect based on the inability to perceive motion in the absence of a surround, with the motion signal actually generated. For the neural expression of the MAE is in fact delayed or inhibited in the absence of a surround, and can later be expressed by reintroducing the surround, with the result that the MAE lasts longer than it would have had the surround been present continuously.

Eye movements and induced motion

How then do we explain the effect of surround absence on the MAE? Clearly the answer to this question would provide valuable clues to the mechanism of storage of MAEs. Yet that answer has so far remained elusive. One possible explanation for the observed effect of the absence of a patterned surround is that eye movements play a role in MAEs. Specifically, it is possible that observers usually track a moving grating with pursuit eye movements, so that the surround moves relative to the retina. Thus, cells receiving input from surround regions will be adapted, so that an MAE in the surround will result. It may thus be that the observed MAE in the target is actually an induced motion effect due to the actual MAE in the surround. Some MAE may actually be due to adaptation of cells stimulated by the target, of course, since tracking is rarely perfect and will result in some retinal motion of the target. As mentioned briefly in the introductory sections, many studies have shown that induced motion can produce MAEs (Szily, 1905; Wohlgemuth, 1911; Anstis & Reinhardt-Rutland, 1976; Reinhardt-Rutland, 1981, 1983a, 1983b, 1984, 1987a). Thus,

when the surround is absent, the surround MAE is stored, and therefore the only MAE seen corresponds to the weaker target MAE. The eye movement theory would also explain the finding that the reduction in MAEs in the absence of a surround is less striking for a rotating stimulus than for linear motion. But since Day and Strelow say only that the effect is reduced for rotational motion, we must infer that some effect was seen. It is possible that such an effect could be attributable to torsional eye movements.

Several findings support the possibility that eye movements during adaptation result in the buildup of an MAE for the surround region, with the central MAE attributable to an induced motion effect. Morgan, Ward and Brussell (1976) found that an MAE is observed when a subject tracks the motion of stripes across a stationary pattern of stripes or across stripes moving in the opposite direction. This MAE is in the same direction as the retinal motion of the untracked stripes, opposite to the direction of tracking. They determined that this effect was due to adaptation produced by retinal motion of the background. In the test period, an MAE for the background region was obtained, in the direction opposite to adaptation. This MAE induced an opposite MAE on the test stimulus. This was shown by an experiment in which the background was presented to both eyes during adaptation, while the target was seen only by one eye. In the fixation condition, where no retinal surround motion occurs, the unadapted eye showed only a weak MAE, typical of interocular MAEs. After tracking, however, there was no difference in the MAEs for the two eyes, suggesting that adaptation was equivalent for the two eyes and therefore due to the background.

They subsequently showed that the MAE is reduced in the tracking condition, but not the fixation condition, when the striped region contains stationary stripes superimposed, relative to the case in which only the moving stripes are present. This effect is due to the presence of a motion signal from the target during tracking, so that the surround no longer produces an excess signal, and is not as effective at inducing motion in the target. Morgan et al also found that this MAE shows storage, indicating it could explain the effects of surround absence discussed above. They concluded that the neural

process which is responsible for the MAE is influenced by stimulation by both moving and stationary contours. The role of stationary stimuli will be addressed shortly.

In a set of experiments which will be discussed further in the section on the general role of eye movements in MAEs, Mack, Goodwin, Thordarsen, Benjamin, Palumbo and Hill (1987), used three vertically separated rows of vertical stripes. Subjects either tracked a fixation target which moved along with the central grating, or fixated the central grating while the flanking gratings moved. Although retinal motion occurred for the flanking gratings in both conditions, an MAE in these regions was seen only in the fixation condition. And while retinal motion of the central grating occurred in neither case, MAEs were seen in both conditions. This central MAE was in the same direction as the previous motion of this grating, whether the motion was real or due to tracking. The authors conclude that the MAE seen in the flanking regions is due to induced motion. They hypothesized that the MAE produced during tracking is weaker than the MAE with real motion and fixation, and in fact found that with a short adaptation period in the fixation condition, the regular MAE was rarely seen, making it equivalent to the tracking condition. They suggest that their results can explain the findings of.

But several factors render the eye movement/induced motion hypothesis inadequate as an explanation of surround effects. For one thing, in most MAE experiments, including those investigating surround effects, subjects are instructed to fixate the center of the adapting stimulus, not to track the motion. While it is conceivable that some difficulty in fixating accurately might occur, the amount of tracking occurring as a result would probably not be great. Seidman, Thomas, Huebner, Billian and Leigh (1990) found a pursuit gain of 0.1 when subjects were attempting to fixate linear motion at velocities of 5 or 7.5 deg/sec. (This indicates that the velocity of pursuit movements was about 0.75 deg/sec.) The argument that linear MAEs may be due to induced motion as a result of tracking the adapting motion has been raised as an objection to studies finding that the linear MAE is undiminished by suppression due to binocular rivalry (Lehmkuhle & Fox, 1975). (The implications of this result will be

discussed in a later section.) According to this view (O'Shea, Timney, Wilcox & Symons, 1990), the MAE observed after adaptation to linear motion may actually correspond to adaptation of surround regions due to tracking eye movements, with an induced MAE seen in the target region. If this is true, then the region generating the MAE is not actually suppressed, explaining the lack of effect of suppression on the MAE. The argument that very little tracking occurs when subjects attempt to fixate would also insubstantiate this hypothesis.

Another problem with attributing surround effects to induced motion due to tracking is that existing evidence contradicts some of the predictions of the hypothesis. In the experiments of Day and Strelow (1971), one would expect that when the surround is absent or unpatterned in the adapt period, the MAE generated for the surround would be minimal. This explains the finding that the MAE is reduced by absence of a surround during adaptation, but it does not explain how a reasonable MAE can be observed in the case when the surround is present only during testing, as it would seem that the surround MAE would be negligible in the case of a dark surround during adaptation. Also, Day and Strelow found that if the surround is dark during the test period, then using a dark surround for adaptation causes minimal further decrease in the MAE. This may be a floor effect, though.

Comparison to stationary stimuli

Another possible explanation of surround influence on MAEs is that responses to motion are enhanced by comparison to stationary stimuli. The idea that detection of stationary targets may influence detection of motion is not new. Braddick (1980) examined the effects of several stimulus manipulations on the percept produced by Ternus displays. In these displays, the first frame consists of three targets (usually lines or small spots), equally displaced horizontally. The second frame contains these same targets, shifted, say, to the right so that the first target occupies the original position of the second target, and so on. Two percepts are possible: the first target of the first frame can be seen to jump to the last position in the second frame, with the other two seeming to remain stationary, or all three targets can be seen to move to the right by one position. The details of Braddick's

experiment are not critical here; the relevant result is that it is the signal generated by the "short-range" (low-level) process indicating the presence of stationary targets, or the lack of such a signal, that determines which form of motion is seen. Thus the response to stationary targets seems to have a definite role in motion perception. How does this fact apply here?

Bell, Lehmkuhle and Westendorf (1976) suggest that the surround effect indicates the operation of neural units sensitive not only to motion, but also to static properties of the visual field. Several physiological findings may shed some light on the effect of stationary stimuli on motion perception, and therefore of the effect of surround characteristics on the MAE. Bridgeman (1972) found in awake behaving monkeys that some cells (39%) respond to both absolute motion, while others (39%) respond only to relative motion. That is, one set of neurons is activated by any motion relative to the retina, while another set responds only to motion relative to a background. Bridgeman suggests that these two cell types may allow a comparison in order to distinguish eye movements (which result in absolute but not relative image motion) from object motion. Likewise, as mentioned earlier, Galletti, Squatrito, Battaglini and Maioli (1984) showed with a larger cell sample that about 10% of monkey V1 cells respond differentially to stimulus motion depending on the presence or absence of pursuit eye movements. Most cells responded only in the absence of eye movements, but one actually responded more during eye movements.

In addition, Bums and Webb (1971) found that in cat cortical cells, responses to motion can be modulated by the presence of a stationary stimulus in the cell's receptive field. Thus, while cells were relatively unresponsive to presentation of a stationary light/dark edge, the response to a moving white line could be significantly altered by the addition of a such an edge parallel to the line.

It seems therefore that the visual system possesses the ability to compare the responses to objects that move relative to other objects with a "baseline" of responses to non-relative motion, and also to modify its responses to moving stimuli when stationary stimuli are also present. These factors may be related to the effect of surround

characteristics on MAE, in that the presence of stationary pattern may modify responses to the adapting and test targets. However, a complete model of how this might occur has yet to be proposed. One possible objection to the role of these physiological processes in mediating surround effects on MAEs is that the stationary pattern is only adjacent to the outer edges of the adapting and test targets. We would therefore need to assess the feasibility of the effect being mediated largely by cells with receptive fields near the border between the target and surround.

Potts and Harris (1975) further investigated the role of surround characteristics on MAEs. They were able to generate MAEs contingent on the spatial frequency, orientation, or color of the stationary surround. (In this case the surround consisted of both an outer surround region and an inner central fixation area, so that the target was an annulus.) This effect was independent of luminance and contrast differences. (They did not examine the possibility of storage in the absence of the appropriate surround, as they were comparing MAEs observed for two different surrounds, e.g. magenta vs. green or vertical stripes vs. horizontal stripes). They propose that some cells may be sensitive to relative motion between achromatic moving patterns and adjacent stationary regions of a specific pattern or color. Thus the proposed role of cells such as those found by Bridgeman (1972) and Burns and Webb (1971) might be modified to suggest that in addition to being selective for motion adjacent to a stationary region, these cells are also selective for other characteristics -- SF, color, orientation -- of the stationary stimulus.

Relative motion

An explanation related to a comparison of moving stimuli with adjacent stationary stimuli involves the hypothesis that MAEs require relative motion, as opposed to just absolute motion (e.g. Reinhardt-Rutland, 1987/88). Motion relative to a stationary background of course fits this requirement; thus the hypothesis accounts for any observations which can be explained by a comparison to stationary stimuli. But this hypothesis also suggests that motion in several directions simultaneously, even in the absence of stationary contours, would be sufficient to generate a MAE. Unlike the eye movement and

stationary contour hypotheses, an explanation due to interaction of motion in different directions can account for the fact that the absence of background has little effect on the spiral aftereffect, which contains motion components in all directions.

This idea does have some support in the physiological literature. Frost and Nakayama (1983) found that in the pigeon optic tectum, some cells responded maximally when a textured background moved in the direction opposite that of a moving spot. Complete response inhibition occurred when the texture and spot moved in the same direction. It is possible that the cells examined by Bridgeman (1972) would also show facilitation when opposite motions were present, although this situation was not investigated. Note, however, the possible difference in the situation whereby a stimulus moves over a background, as in these studies, as compared to the situation present in the studies of surround effects on MAEs, where target and background are in adjacent regions. Conventional direction-selective cells might be expected to reduce their responses to a stimulus in the presence of an oppositely moving background, as the opposite motion would counteract to some extent the response produced by the original motion. Motion in an adjacent region, however, might enhance responses, perhaps through facilitation or disinhibition. Hammond and MacKay (1981) in fact found that a mask of moving texture reduced cell responses to a moving bar when the texture was presented within the receptive field, but enhanced responses when presented at either end of the directional axis of the receptive field.

The role of neurons sensitive to motion relative to a background was suggested by Strelow and Day (1975). They determined that independent adaptation of surround and target occur. As the duration of adaptation with both target and surround present increases, the MAE reached a peak at 60 seconds and then reached a plateau. At this duration of target adaptation, however, the peak MAE occurred for a surround duration of only 30 seconds, indicating that adaptation to the surround is faster. The authors do not relate this independence to the possible involvement of relative-motion cells. It remains unclear how this independent adaptation could arise, and how it could produce the observed effects on MAE strength.

Apparently more work will be required in order to determine conclusively how the effects of surround characteristics on MAEs are mediated. Clearly the answer will allow advances in our understanding of the mechanism of storage as well. Let me return now to a discussion of the general phenomenon of storage itself.

Other aftereffects showing storage

Storage has also been demonstrated for other types of aftereffects, such as the McCollough effect (Jones & Holding, 1975; Holding & Jones, 1976; MacKay & MacKay, 1977; but see also Skowbo, Garrity & Michaud, 1985). Thompson and Movshon (1978) reported that storage of the threshold elevation aftereffect (following prolonged viewing of a high-contrast grating) occurs only in darkness, and not when the observer views a homogeneous field during the storage period. They also noted that this aftereffect is not stored for the long durations reported for MAEs (Hershenson, 1985; Masland, 1969). I will not attempt to incorporate these related findings into my discussion of storage of MAEs, as the topic would become much too expansive. However, the reader should be aware of the apparent ubiquity of the storage phenomenon, and any future model of stored MAEs will also need to consider applicability to storage of other aftereffects.

Neural basis of storage of MAEs

There is a glaring lack in the literature of suggestions as to how storage of MAEs can be explained at the neural level. The ratio model in its current form clearly can not provide such an explanation, but alternative explanations have not been forthcoming. Unfortunately, very few physiological studies have even presented results relating to the neural correlate of storage of MAEs, and none were designed expressly to investigate this basis.

Vautin and Berkley (1977) conducted an experiment to determine the time course of recovery following adaptation in the absence of stimulation. Cells were adapted for one minute, then an interval of 0-60 seconds passed before the cells were exposed to the adapting stimulus again. The initial response following the delay interval was used to assess recovery. Recovery of adaptation for the cell illustrated was exponential, with a time constant of about 7 seconds,

and was 95% complete in about 22 seconds. The cell seems to have fully recovered in 30 seconds. The two other cells examined showed similar results, with time constants of about 4 and 6 seconds. Unfortunately, the authors do not present a comparison of the cells' responses to a test stimulus following adaptation to their responses when storage occurs, a comparison which would allow an evaluation of the existence and extent of storage at the single-cell level.

Note that the time constant reported here differs from that measured psychophysically by Keck and Pentz (1977), which was on the order of 20 seconds. Since only a few cells were examined, however, it would be premature to accept this difference as fact. It is likely that the time constant varies among neurons, and psychophysical evidence (Taylor, 1963a; Hershenson, 1989) suggests that adaptation conditions as well as test conditions can affect the rate of decay. Thus direct comparisons among time constants measured in different studies may not be appropriate. It is unclear whether stimulus manipulations affect the time constants of individual cells, or whether they affect the makeup of the population of adapted cells, so that the distribution of time constants is altered.

One physiological finding which may relate to the storage of MAEs is the frequent report that spontaneous rates of firing in direction-selective cells are not depressed following motion adaptation (see earlier section). This lack of effect may correspond to the storage of the aftereffect, in that adaptation effects are only expressed upon stimulation by an appropriate test target. At the physiological level this is manifested as a lack of reduction in spontaneous rates, combined with a decrease in driven activity produced by stimulation with a test stimulus. At the psychophysical level, the parallel result is the storage of MAEs in the absence of a test target, followed by observation of an MAE when a test target is presented beyond the time at which the MAE would have completely decayed. Thus, although the basis of storage is not made explicit by the elaboration of this relationship, the two phenomena at least seem consistent, and this similarity may aid in the development of an appropriate explanation of the mechanism of storage. Hopefully future physiological work will reveal that in cells which do not decrease their

spontaneous firing in response to adaptation, the physiological analog of storage can be observed. That is, these cells should, if unexposed to a test stimulus for a period equivalent to the duration of their driven adaptation effects, show no change in spontaneous firing during this time. They should, however, exhibit adaptation effects following this period. Such a finding would at least provide support for the operation of a mechanism similar to that proposed to explain immediate MAEs.

ARE THERE TWO MOTION AFTEREFFECT MECHANISMS?

One issue which has been raised in relation to MAEs is the possibility that there may be two different types of MAE, with different mechanisms, possibly at different levels of the visual system. That is, some MAEs may result from processes at a lower level, whereas others may arise at a higher level. A variety of evidence exists to support this notion: the existence of both long- and short-term MAEs, the phenomenon of storage, the presence of multiple phases of decay of MAEs, differences in decay characteristics for different types of MAEs, differences in contrast response, and the differential effects of rivalry on various MAEs. Each of these findings will be discussed in turn.

Multiple decay phases

Taylor (1963) was the first to demonstrate the presence of distinct phases in the decay of the MAE. Recall that he found that the time course of MAE decay for adaptation durations of 5 and 20 seconds could be fit by a single exponential function. For durations of 80 and 320 seconds, however, a second, slower decay period followed the first, with the first phase similar to that found with shorter durations. Within subjects, no relationships between the MAE magnitude (intercept of decay function) or decay rate for the first phase and those for the second phase were found, nor was there a relationship between intercept and time constant. Velocity of adaptation affected only the intercept, and the two phases differed in their velocity effects, with the second phase reaching maximum at a lower adapting velocity (60 vs. 120 deg/sec).

Masland (1969) also found two phases of decay of MAE -- an initial rapid decay for 5-10 minutes, followed by a phase with a slower decay rate. Hershenson (1989) found that three phases of MAE decay could be discerned, only one of which actually represented decay of

the MAE. The first phase was a period of maximum magnitude, with no decay. Next was the actual decay phase, during which the MAE magnitude decreased exponentially. The decay phase was followed by a tail phase, whereby minimal magnitude was reported for a continuous period. The durations of all three phases, and the time constant of the decay phase, were affected by adaptation duration up to 15 minutes. He concludes that the phase durations and the time constant are not fixed properties of motion channels.

Bonnet and Pouthas (1972) found two phenomenally different phases of MAEs, using an adapting spiral with a mask revealing only certain sectors of the spiral. When subjects adapt to rotation of such a spiral, and then test on a full spiral, two phases are evident. The initial portion of the MAE is seen only in the regions of the test spiral corresponding to the regions visible during adaptation. Then, however, areal spread occurs, so that the MAE is observed on the entire spiral. They refer to the first phase as the kinetic phase, and the second as the figural phase. Interestingly, they determined that the duration of the kinetic phase is the same as the duration of the first phase of vividness or faster decay (corresponding to the first phase of Taylor, 1963) when the test stimulus was a noise pattern, for which no areal spread occurs. Observers were consistently able to distinguish the two phases both for the spiral test target and the noise target.

The authors also reported that increasing the number of sectors from two to four, while keeping total visible area constant (either 45 or 90 deg), caused a decrease in the duration of the first phase, and in the total MAE duration. The duration of the second phase was unaffected, again supporting different mechanisms for the two phases. No areal spread occurred when only one sector of 45 or 90 degrees was used, however, suggesting that increasing the number of sectors both increases the duration of the second phase and decreases that of the first phase. Areal spread was observed when a single sector of 135 or 180 degrees was employed.

Short-term vs. long-term MAEs

Mahmud (1987) demonstrated the existence of both short-term (ST) and long-term (LT) MAEs, using a reverse adaptation procedure.

Subjects adapted to a rotating spiral for 10 minutes, then were immediately tested for presence and direction of MAE. Five minutes later, subjects adapted to the spiral rotating in the opposite direction for one minute, and were subsequently tested each minute for five minutes. After the first adaptation period, subjects all observed MAEs in the appropriate direction. After the second adaptation period, they again observed appropriate MAEs, in the direction opposite the original MAE. During the five-minute period, though, the MAE actually reversed back to the original direction. Thus the reverse adaptation temporarily, but not completely, reversed the expression of the original MAE. The process responsible for the reversed, or ST MAE, must therefore differ from that underlying the LT MAE seen after the ST MAE decayed. A simple ratio-type model would predict that the reverse adapting motion would counteract the adaptation effects of the original motion. Thus if the reverse motion were strong and prolonged enough, it would produce enough adaptation to reverse the original MAE. If it were not strong enough to completely counteract the original MAE, the original MAE would be observed, though weaker in magnitude. If the reverse motion were just strong enough to just counteract the original adaptation, no MAE would occur. The finding that the original MAE returns after decay of the reversed MAE can not be explained by the ratio model and a single adaptation mechanism. Thus, in addition to providing yet another reason to question the validity of a simple ratio model, these results suggest the existence of two MAE mechanisms, which exhibit different time courses.

Mahmud also found that the ST MAE shows interocular transfer, whereas the LT MAE does not. However, it is possible that transfer was not found for the LT MAE simply because it is initially weaker, and thus does not last as long even though the time course is similar. (This argument will be discussed in more detail in the next section.) Since only the ST MAE is visible immediately following reverse adaptation, it is possible that the neural processes underlying the LT MAE are also taking effect at that time, but are overpowered by the ST MAE, so that only the ST MAE is observed until it decays. If this is the case, it is also possible that the LT MAE would be observed interocularly if the

ST MAE were minimized so that the LT MAE could be measured earlier, before decaying completely.

Mahmud suggests that the ST MAE is the result of adaptation at an early level of motion processing, whereas the LT MAE arises from a more complex mechanism at a higher level. Absence of interocular transfer is generally associated with lower-level, rather than higher-level processes.

Differences in decay characteristics for different MAEs

Size-change vs. motion-in-depth

Adaptation to a stimulus which appears to expand or contract when rotated may also seem to move in depth. The MAE which succeeds such adaptation also possesses these components (e.g. Hershenson, 1987; Beverley & Regan, 1979). Beverley and Regan (1979) reported that size-change and motion-in-depth MAEs represent distinct MAE types, as evidenced by their differing decay characteristics. Both MAEs showed exponential decay, but the time course of the size-change MAE was much more rapid. The authors propose that these MAEs are processed at hierarchical stages of motion processing, with the motion-in-depth analyzed at a higher level than linear motion or size-change information. They relate this hypothesis to the previous report (Regan & Cynader, 1979) that some area 18 cells are sensitive to changing-size, and may as a population be capable of signalling information about size-change. Cynader and Regan (1978) reported cells in cat area 18 that are selective for the direction of motion in depth, and Regan and Cynader (1982) described similar cells in cat cortex near the border between areas 17 and 18. The role of these cells in MAEs was not discussed, however.

Interocular vs. dichoptic

Favreau (1976) found that interocular MAEs are evident immediately after adaptation, but not when tested again 7 minutes following adaptation. She did, however, find that dichoptic MAEs were evident after 7 minutes. Masland's (1969) report of two MAE phases led Favreau to propose that these two phases represent the return to preadaptation firing levels in two different populations of neurons. She attributes the interocular MAE to binocular neurons, and the dichoptic MAE to monocular neurons.

However, it is possible that her finding of decreased decay rate was confounded by the initial level of neural adaptation. She points out that comparing the results of the two experiments nonparametrically is impractical, and since the results are "obvious", does not statistically analyze the differences. However, it would be helpful to assess whether the initial magnitudes of dichoptic and interocular MAEs were similar. The means are difficult to interpret, largely because in the dichoptic condition the two eyes are adapted alternately, and the means for dichoptic MAE duration are affected by which eye was adapted just prior to testing. Thus the mean initial magnitude for the eye not just adapted is lower than that for the eye just adapted, with the mean for the interocular condition intermediate. It is unclear which measure should be compared to the interocular condition, or whether an average of the two is most appropriate. If interocular MAEs are in fact initially weaker than are comparable dichoptic MAEs, the difference in persistence of the two MAEs could be attributed to this difference. This initially lower level of adaptation and thus of aftereffect for the interocular condition would be manifested as a decreased total duration, even if the decay rate is the same in both conditions. (Imagine two parallel decay curves, with equal slope and thus equal decay rates, but with different y-intercepts. The curve with the lower intercept will reach the x-axis, representing complete decay of MAE, at a shorter duration.)

Favreau interprets her results as supporting two parallel systems for perceiving motion. She claims that different decay rates can not occur at hierarchical levels for the two MAEs, because central units (representing the interocular MAE) get their input from earlier units (representing the dichoptic MAE), and therefore the dichoptic MAE should disappear at least as soon as the interocular MAE. However, it is possible that the two cell populations may merely be monocular and binocular cells at the same locus, which does not necessarily constitute a pair of parallel systems.

Another possibility is that two rates of decay might represent two different adaptation processes within the same neurons. As yet there have been few studies of the precise cellular mechanism underlying adaptation of direction-selective cells. It is conceivable,

however, that two processes might mediate adaptation, with one occurring rather rapidly and being counteracted rapidly, and the other taking longer to build up and longer to disappear. This duality might take the form of two different inhibition systems mediated by different transmitters. Support for this idea lies in the report by Daniels and Pettigrew (1975) discussed earlier, which suggests that that there may be more than one inhibitory transmitter system involved in the mechanism of direction selectivity of cortical cells. Rose and Blakemore (1974) also proposed the possible involvement of an additional, non-GABA inhibitory transmitter, which would explain the different time courses of some of the effects they observed in complex cells as opposed to simple cells.

Another objection to the overzealous acceptance of Favreau's conclusions involves the lack of consistency between her and others' methodologies. For instance, the adaptation velocity used was only 5 rpm, which differs greatly from the velocities commonly used for studies of MAEs (i.e. 80 rpm for Masland, 1969). It is also unclear whether the adaptation paradigms for interocular and dichoptic MAEs can actually be considered as equivalent, a limitation not specific to this study.

Contrast response of stored MAE

Kirita (1987) demonstrated that while the contrast response of an MAE that immediately follows adaptation saturates at about 0.1, the duration of the stored MAE increases continuously for contrasts up to at least 0.5. The former value is slightly higher than that reported by Keck, Palella and Pantle (1976); however, this difference is probably attributable to procedural differences, and clearly illustrates the contrast saturation of the MAE. The lack of such saturation for the stored MAE suggests that different mechanisms are involved in production of the two types of MAE. Kirita proposes that there are two direction-selective mechanisms, with differing contrast responses, and relates his findings to those of Maffei and Fiorentini (1973), which Kirita interprets as suggesting that complex cells exhibit a saturation in contrast response, whereas simple cells show no such saturation. This is not a clear-cut distinction, however, as the contrast at which saturation occurred, according to the report of Maffei and Fiorentini,

was variable, and moreover, was at a relatively high contrast (e.g. 0.5). One problem with interpretation of Kirita's results is that for the immediate MAE, an adaptation duration of 30 seconds was employed, whereas for the stored MAE, the adaptation duration was 10 minutes. It could thus be argued that the difference observed might be related to the difference in time and extent of adaptation, rather than to the difference in mechanisms. It would be desirable to see if the same result is obtained when a 10-minute adaptation period is used for the immediate MAE as well.

Low-level and high-level MAE processes?

What could explain the observed differences in different types of MAEs? Is it possible that the differences all relate to procedural variables, or do these differences actually reflect the operation of two separate MAE processes? The former suggestion seems unlikely, in light of some of the differences discussed above. A variety of evidence, both psychophysical and physiological, suggests that motion is processed at hierarchical levels, with linear motion processed at an earlier site, and more complex types of motion -- spirals with their rotational and size-change components, and plaids whose direction of motion is different from that of the individual components -- processed at higher levels. Movshon, Adelson, Gizzi and Newsome (1985) showed that while V1 cells respond only to component direction for a moving plaid, some MT cells respond to overall pattern direction. Saito, Yukie, Tanaka, Hikosaka, Fukada and Iwai (1986) and Tanaka and Saito (1989) found that cells in MST respond to rotational motion and to expansion/contraction. Cells in posterior parietal cortex (area PG) were also found to be responsive to rotational motion (Sakata, Shibutani, Ito & Tsurugai, 1986) and to expansion/contraction (Motter & Mountcastle, 1981; Motter, Steinmetz, Duffy & Mountcastle, 1987; Steinmetz, Motter, Duffy & Mountcastle, 1987) Thus V1 can be thought of as a lower-level motion-processing site, which extracts only the linear component of motion, whereas higher areas extract more global types of motion such as plaid motion, rotation, and size-change.

Studies of MAEs have similarly suggested a multi-level setup, possibly corresponding to the different types of motion processing discussed above.

Local vs. global rotational MAEs: different decay characteristics

Cavanagh and Fabreau (1980) compared MAE for different test stimuli following adaptation to a spiral target. Their main comparison was between a test target identical to the adapting stimulus, and one which was the mirror image of the adapting stimulus. The rationale for this manipulation is that an MAE observed on a mirror-image spiral must be due to global processes, rather than local adaptation, because the contours of a mirror-image spiral are all orthogonal to those in the original, a difference beyond the orientation-tuning limit of most cortical motion detectors (Schiller, Finlay & Volman, 1976b). The normal MAE, on the other hand, could conceivably be mediated at either the local or the global level.

They found that MAEs were observed for the mirror-image test target, as well as on a radial-line pattern (used for a different group of subjects) with contours differing--by 45 deg from the original spiral contours. This was true for immediate tests and a second pair of tests 7 minutes later. No difference was observed between MAEs for mirror-image and radial targets, although both were significantly smaller than for the original test stimulus. The authors conclude that the MAEs for the mirror-image and radial targets represent a global process of adaptation. For a second experiment in which tests were conducted only after the 7-minute delay, a different result was obtained. Only the MAE for the original spiral target was significant. However, the variabilities in this experiment for both mirror and radial targets seem somewhat higher, which may have contributed to the lack of a significant result. This seems especially possible in light of the fact that the two curves for the mirror spiral in the delay condition (Expt. 1 vs. Expt. 2) appear identical, except for the slight increase in standard error in Expt. 2. In both cases this MAE is rather insubstantial, being under 1 second in both experiments. Note, however, that even under the optimal condition (Expt. 1, immediate test), the mean MAE is only about 4 seconds. The lack of a significant Mae in the delay condition of Expt 2 is especially surprising, in that

we would expect a greater MAE relative to Expt 1 since no prior test occurred in Expt 2. The standard MAE, however, did show a significant increase in the delay condition of Expt 2 relative to that of Expt 1, indicating that storage was occurring.

The authors conclude that the standard MAE, which likely involves both local and global processes, shows different storage properties than does the global MAE. Both MAEs seem to be affected by the passage of time, but only the local MAE is affected by previous testing. Cavanagh and Favreau suggest that the global MAE is mediated by global rotation detectors.

It is possible, however, that the storage effects were confounded by the initial level of MAE in each case. (This same argument was noted above for the results of Favreau, 1976.) If the two types of MAE have identical decay rates (slopes on a plot of magnitude as a function of time), they may still show different levels of persistence (that is, the maximum duration at which an effect is observable without prior testing), simply due to differing initial levels (y-intercepts). Thus it is necessary to support this result with further data showing that the time courses actually differ for the two types of MAE.

Local vs. global rotational MAEs: differences in retinal specificity

Goldstein (1958) stressed the similarities between spiral and linear motion, showing that if a mask were placed over a rotating spiral so that only four narrow sectors of the spiral were visible, the visible motion clearly resembled four separate regions of linear motion. Thus it is likely that while spiral motion may be specifically extracted at a higher level, some processing, and thus some adaptation, may occur at the lower site of linear motion processing. Thus the differences observed between the two types of MAEs are likely due to the contribution of the higher-level process to spiral MAEs.

Bonnet and Pouthas (1972) made use of Goldstein's demonstration to illustrate the presence of two distinct phases of spiral MAEs, as discussed earlier. Recall that they found that when subjects adapt to such a masked spiral and test on a full spiral, the MAE first exhibits a phase of retinal specificity, with the MAE only visible in the adapted region. A second phase then follows, during

which areal spread occurs so that an MAE is observed over the whole spiral. The existence of these two phases strongly suggests the operation of two mechanisms, one of which exhibits retinal specificity and a rapid time course, and the other of which is more global in nature and possesses a longer time course.

Interestingly, the authors report that areal spread does not occur if the test stimulus is identical to the adapting stimulus but is rotated so that the adapting sectors do not overlap the testing sectors. This result poses somewhat of a problem for the role of global expansion/contraction detectors in the second phase, as one would expect that a MAE would be seen in this case. However, additional experiments reported in the same study suggest that configural similarity between adapting and test stimuli is necessary for areal spread, a factor which could explain the above result.

Evidence for extrastriate MAEs

As well as reporting the existence of cells in MT selective for direction of pattern motion, rather than component motion, Movshon et al (1985) reported the results of a psychophysical experiment supporting the role of these cells in the generation of MAEs. For a plaid pattern composed of two gratings oriented at 120 deg, the component directions each differ from the pattern direction by 60 deg. The plaid appeared to move in a coherent manner to the right, whereas the individual gratings appeared to move toward 30 deg CW of upward and 30 deg CCW of downward. Following adaptation to a vertical grating drifting rightward, the detection threshold of a similar grating is elevated. Will this occur for a plaid, whose components are each oriented 60 degrees away from vertical?

These authors found that following adaptation to the plaid, the contrast threshold for a vertical grating was not elevated. Likewise, adapting to the vertical grating did not elevate the detection threshold for the plaid. These results were obtained despite the fact that the overall direction of motion was identical for the plaid and the grating, and therefore suggest that detection thresholds are a component-level phenomenon. That is, adaptation to one of these targets does not result in threshold elevation for the other because the component

directions differed by 60 degrees, and thus are detected by different groups of direction-selective cells, such as those found in V1.

But the story is not yet complete. Adelson and Movshon (1982) demonstrated previously that if one component of a plaid pattern is at much lower contrast than the other, the two gratings are seen to slide over one another with their own separate directions of motion, rather than moving as a coherent plaid. As the contrast of this component is raised, the probability of coherence increases. It is thus possible to determine the coherence threshold of a plaid, that is, the contrast of the lower-contrast grating at which the plaid is seen to cohere rather than slide. As just mentioned, the detection threshold of a plaid is *not* elevated, consistent with the notion that detection of the plaid depends merely on detection of the individual components, which is not affected by prior adaptation to a vertical grating. However, the coherence threshold for the plaid is elevated. This effect cannot be attributed do adaptation at the level of the components; thus the authors conclude that adaptation of pattern-selective MT cells must be responsible for this phenomenon. Note that pattern-selective MT cells do respond to the direction of motion of a single grating in the appropriate orientation and direction when presented alone. Thus, they seem to extract the overall direction of motion of a stimulus, regardless of whether one or more individual directional components are present. Adaptation of such cells would therefore result in decreased responsiveness, manifested as a decrease in sensitivity for any pattern with the same overall direction, be it a grating or a plaid. These authors do not report on the effects of adaptation to gratings and plaids on subsequently presented *stationary* stimuli. Thus, their results do not demonstrate pattern-selective MAEs *per se*. However, the observation of elevated thresholds is consistent with adaptation of pattern-selective elements, which would be expected to yield a MAE. This author is currently conducting a set of experiments involving such MAEs. Observers have actually obtained MAEs on a stationary horizontal grating following adaptation to a plaid pattern moving downward with components each oriented at 60 deg from horizontal. In fact, this MAE is usually reported as being stronger than is the MAE observed on a single component of the plaid following

adaptation to the plaid. Direct observation of pattern-selective MAEs provides strong evidence for the existence of a higher-level site of MAE production.

Wenderoth, Bray and Johnstone (1988) also reported evidence supporting the existence of MAEs mediated at a level beyond striate cortex. Subjects adapted either to pairs of gratings of differing orientation presented in alternation, or to those same pairs of gratings presented simultaneously, composing a plaid pattern with a direction of motion differing from the individual component directions. The difference in orientation, and thus direction, between the components of a pair was varied, but the pattern direction (horizontal) remained constant. Testing always involved a vertical grating. Cells selective for component direction would be adapted similarly during both simultaneous and alternating presentation of the two components. The increase in orientation difference between the adapting and test components would thus result in a decrease in MAE strength, as MAEs are orientation selective. This result was obtained in the alternating condition. For the simultaneous condition, however, this trend was absent or minimized. This is attributed to adaptation at the level of cells selective for pattern direction, such as those reported in MT (Movshon, et al, 1985). Since the direction of pattern motion is unchanged when the component orientations were varied, no reduction in adaptation of pattern direction-selective elements occurred when the component orientations were increasingly different from vertical. Alternating adaptation yielded only component-specific adaptation effects, presumable mediated by both V1 and MT cells. Simultaneous adaptation, however, produced pattern-selective adaptation in MT cells. These results therefore support the notion that MAEs are not a homogeneous phenomenon.

Further evidence for a unique contribution of MT to MAE generation comes from a set of experiments by von Grünau. He reported (1989) the results of an investigation of MAEs measured on areas either overlapping the adapted area or separate from the adapted area. The adapting pattern was either a single grating, or a plaid composed of two gratings. The test stimulus was a counterphase grating, which was seen to flicker in the unadapted condition, and was

seen to drift in the opposite direction following adaptation to a drifting grating. Von Grünau reported that when adapting and test areas were overlapping, MAEs were best for test gratings with the same orientation as the adapting grating, and were similar for test gratings with either the same orientation as one of the adapting plaid components, or the orientation perpendicular to the direction of plaid motion. This measure presumably taps into responses of both V1 and MT cells, with V1 cells showing component-selective adaptation, and MT cells showing pattern-selective adaptation, so that both types of MAEs are observed. When the adapting and test regions were nonoverlapping, however, a different pattern of results obtained. For both gratings and plaids, the resulting MAE was best for a counterphase grating with the orientation perpendicular to the overall direction of adapting motion. Thus, measuring MAEs for nonoverlapping regions corresponds to the higher-level motion process, corresponding to MT cells selective for overall pattern motion regardless of whether the pattern is a single grating or a combination of gratings.

Effect of binocular rivalry on MAEs

When the two eyes simultaneously view different targets, an alternation between these two targets is perceived, so that each target is periodically rendered invisible ("suppressed") for several seconds at a time. Thus, the information from a physically present stimulus is blocked at some point in the visual pathway. Studies of the effect of rivalry on the buildup of different MAEs provide evidence for different sites of the neural processes underlying these MAEs. Lehmkuhle and Fox (1975) found that the duration of the linear MAE is undiminished when adaptation occurs under conditions of binocular rivalry, so that the adapting motion is intermittently suppressed and thus visible for only part of the adaptation period. O'Shea and Crassini (1981) confirmed this result, and additionally reported that interocular transfer of the linear MAE is not reduced by suppression due to binocular rivalry. If the MAE is generated at a site beyond the site of binocular rivalry suppression, stimulus information would be intermittently blocked from reaching this site during suppression phases of rivalry, so that the effective adaptation duration would be

reduced, yielding a decrease in the observed MAE. Since rivalry had no effect on the MAE, they concluded that the linear MAE must arise at a site prior to binocular rivalry suppression.

For spiral MAEs, however, the story seems different. Wiesenfelder and Blake (1990) demonstrated that suppression does interfere with the buildup of the spiral MAE. When subjects adapted during rivalry, with the spiral motion visible during no more than half the adaptation period, the MAE duration was substantially reduced relative to the nonrivalry condition. Thus we can conclude that the site of spiral MAE generation is beyond the site of rivalry, as suppression did prevent the adapting motion signal from reaching the MAE site. The site of rivalry is as yet not clearly established (see Wiesenfelder & Blake, 1990, for a discussion of the site of binocular rivalry). However, the differential effect of suppression on the two types of MAE is strong evidence that the two are mediated at different sites, either with spiral motion at a higher and linear motion at an earlier level, or with linear motion in a parallel pathway, separate from that involving both spiral motion and rivalry.

Effect of binocular rivalry on storage of linear MAE

A recent study by Wiesenfelder and Blake (1991), in combination with the results of Lehmkuhle and Fox (1975), lend further support to the hypothesis that MAEs do not represent a single phenomenon with a single site and mechanism. Subjects adapted monocularly to linear motion under nonrivalry conditions, so that adaptation was equivalent in all conditions. Immediately following adaptation, a stationary grating was presented to the adapted eye, while a dynamic field of large dots was presented to the other eye. This rival target was capable of suppressing the grating for the entire storage period used (15-30 seconds, chosen to be optimal for each individual). During the storage period, subjects either covered both eyes (storage condition), viewed just the grating (normal MAE), viewed just the dots (control condition), or viewed both targets (rivalry condition).

Storage occurred when the eyes were closed, as evidenced by a longer MAE in that condition than in the normal MAE condition. The MAE in the rivalry condition was identical to that in the storage condition, and was thus longer than that in the normal MAE condition.

This effect could not be attributed to the effect of the dots alone. We therefore concluded that suppression prevented the information from the stationary grating from reaching the MAE site. This result was surprising, in light of the previous report (Lehmkuhle & Fox, 1975) that information from the adapting target does reach the MAE site during suppression. It thus seems feasible that the stored MAE represents a higher-level process occurring at a site beyond the rivalry site, whereas the normal MAE is an earlier process occurring prior to the rivalry site.

As discussed above, the neural processes underlying storage of MAEs remains a mystery, and there is a complete paucity of physiological evidence relating to the possibility of different sites of the normal and stored MAEs. The current finding is thus especially useful, as it represents the only evidence to date clearly supporting processing of the immediate and stored MAEs at different sites.

Low-level vs. high-level MAEs: physiological evidence

A great deal of physiological evidence exists which indirectly supports the notion that MAEs may correspond to motion processes at different levels, although studies of adaptation at these different levels remain scarce. The study by Petersen, Baker and Allman (1985) of adaptation of MT cells was discussed earlier, and will thus not be included in this section. Note, however, that their study involved linear motion only, and can therefore not be used as a direct assessment of the role of MT in mediating higher-level MAEs.

Albright (1989) determined that the preferred directions of motion of MT cells are biased, with a large proportion preferring foveofugal motion. Brenner and Rauschecker (1990) reported a similar bias in area LS of cat. This finding provides preliminary evidence that MT may be involved in extracting expansion and contraction, or that it may be the first stage of a process performing this function. This latter possibility seems likely, and is supported by studies of cells in the middle superior temporal area (MST), which receives direct input from MT, and of area PG, which receives input from MST. These studies, discussed earlier, demonstrated that many cells in MST and PG respond preferentially to rotational motion or to expansion/contraction. These cells could conceivably be adapted

during stimulation by a rotating spiral or disk, and thus underlie spiral and rotational MAEs.

ROLE OF EYE MOVEMENTS IN MOTION AFTEREFFECTS

Some early investigators believed that MAEs were due to eye movements, while others felt eye movements hindered MAE production. Purkinje (1825). Adams (1834), and Helmholtz (1867) all espoused the eye movement theory of MAE generation, and Helmholtz asserted that tracking the moving target is necessary for MAE production. The discovery by Plateau (1850) of an MAE following adaptation to a rotating spiral, which would seem not to accommodate an explanation due to eye movements, provided preliminary evidence against this hypothesis. Dvorak (1870) showed that with concentric spirals, appropriate MAEs in opposite directions occurred, and Kleinert (1878) showed that adapting simultaneously to several spirals moving in opposite directions also yielded simultaneous MAEs. This would seem to negate the possibility of eye movements mediating the MAE. A similar result was obtained by Hoppe (1894), for simultaneous adaptation to a spiral and its mirror image. This was apparently enough evidence to convince most later researchers (e.g. Exner, 1888; Szily, 1905) that eye movements were not the cause of the MAE.

Since that time, quite a few studies have been conducted with the goal of determining the role of eye movements in the generation of MAEs. Several of these were mentioned earlier in relation to the feasibility of the induced motion/eye movement explanation of surround effects. This will by no means be a complete examination of the role of eye movements in MAEs, or of the interactions of the oculomotor and motion-detecting systems, which could in itself provide the basis for an entire review paper. However I will touch on some of the relevant studies in order to provide some perspective on the issues involved.

Fixation

Wohlgemuth (1911) emphasized the need to maintain fixation during adaptation in order to observe a maximal MAE. More recently, Holland has demonstrated this requirement. In 1957, he showed that the production of MAEs is dependent upon fixation, in that random changes in fixation throughout the fixation period result in the

absence of an MAE. He then showed (Holland 1959,1960) that the degree to which an observer maintains fixation is correlated with the duration of the subsequent MAE. When viewing a spiral rotating at a speed above the fusion threshold (in this case 600 rpm), observers sometimes see brief glimpses of the spiral. These glimpses are taken as evidence of tracking the motion, which reduces the effective velocity of motion below the fusion threshold. Holland found that the higher the frequency of such glimpses during adaptation, the lower the MAE observed.

Retinal stabilization

Other evidence against the requirement for eye movements as a determinant of MAEs derives from experiments utilizing stabilized images. When the adapting or test stimulus is retinally stabilized, eye movements do not result in retinal motion of the target, and therefore can not account for any observed MAE. Sekuler and Ganz (1963) found that MAEs, along with corresponding elevation of luminance thresholds for motion detection, occurred when the adapting target was retinally stabilized. They did not, however, employ a stabilized test target. Moulden (1975) found that MAEs are obtained when the test target is stabilized as an afterimage. This experiment used a spiral stimulus, with opposite 90-degree sectors visible. In a key experiment, Drysdale (1975) used linear motion in the form of a squarewave grating, with both adapting and test stimuli stabilized, and found that MAEs were still observed.

Fixating vs. tracking: direct comparisons

Another method of investigating the role of eye movements in MAE production has been to compare the MAEs observed following adaptation to a fixated moving stimulus, with those seen following adaptation by tracking of a moving target over a stationary stimulus. Kinoshita (1909) found that an MAE could be observed if the eyes were moved over a stationary target or if a moving target was fixated. Anstis and Gregory (1965) claimed that the MAEs produced in these two conditions are equivalent. They concluded that motion detectors are unable to distinguish motion produced by eye movements from that due to actual object motion, with MAEs resulting from retinal motion regardless of eye movements. However, assuming that the

length of the direction vectors in their figures represents magnitude of MAE, then the MAEs produced in the two conditions were about equal, even though the retinal motion was 3.75 times faster in the tracking condition. Thus it seems that the MAE produced by tracking a fixation spot over a stationary grating would be weaker than that for conventional adaptation if the adapting velocities were equal.

The same conclusion was reached by Tolhurst and Hart (1972), who found no difference in the threshold elevation produced for moving stimuli produced by adaptation in the two conditions (fixation vs. tracking). They concluded that the mechanism responsible for motion adaptation can not distinguish object motion from motion due to eye movements, and that therefore the corollary discharge from eye movement control centers, if it exists, must arise at a site beyond the adaptation site. The only alternative they propose is that while adapted units may be capable of this distinction, the adapting mechanism is not. Note, however, that threshold elevation may be produced by orientation-specific as well as direction-specific adaptation, so that they might have found different results had they discounted the proportion of adaptation in each case which was actually due to orientation-specific adaptation. Also, as discussed above, it is possible that threshold elevation and MAEs are mediated separately.

Later studies failed to replicate the findings of Anstis and Gregory (1965). Mack, Goodwin, Thordarsen, Benjamin, Palumbo and Hill (1987), in a study discussed earlier in relation to the role of stationary stimuli on MAEs, were unable to duplicate the results of Anstis and Gregory despite repeated and varied attempts. They in fact found that the MAE produced by tracking generally occurs for regions unstimulated by adapting motion and is in the same direction as the tracking, and attribute the effect to induced motion. Recall that they had used adapting and test targets consisting of three rows of vertical stripes. In the tracking condition, subjects tracked the leftward motion of a tracking target and the central grating, while the upper and lower (flanking) gratings remained stationary. In the fixation condition, subjects fixated a target in the center of the central grating, while the flanking gratings moved rightward. Thus in both cases, the central grating was stationary relative to the retina, while the flanking

gratings moved relatively rightward. They found that while MAEs for the central grating occurred in both conditions, MAEs for the flanking gratings were only seen in the fixation condition. In addition, the MAEs for the central grating following tracking were in the same direction as their prior motion. The authors conclude that this MAE represents the result of induced motion from the flanking gratings, for which normal MAEs occur.

They also determined that the MAE produced for the flanking gratings is weaker in the tracking condition (similar to the result illustrated but not mentioned by Anstis and Gregory, 1965). This was supported by the finding that short adaptation durations with fixation also fail to yield such MAEs. They suggest that some damping of the motion signal occurs as a compensation process during tracking eye movements. This would of course imply that the motion system does have access to the signal produced by tracking eye movements, leading to a discrepancy with Anstis and Gregory.

Similar results were obtained from a subsequent experiment (Mack, Hill & Kahn, 1989), intended to determine whether it is retinal motion signal or the signal from the compensation process comparing retinal and eye movement which produces the MAE. In the first part of the experiment, the adapting stimulus was the same as for Mack et al (1987). However, subjects either fixated a central fixation point while the central bars moved rightward, or tracked the fixation point continually upward while the central bars moved rightward. Thus, in the first case, both retinal motion and motion relative to eye movements were in the horizontal direction, leading to the prediction of a horizontal MAE. In the tracking condition, however, the adapting motion was oblique with respect to the retina, but was horizontal relative to eye movements. Thus, in this condition, the MAE would be oblique if it were caused by retinal motion alone, but horizontal if it were produced by a comparison of retinal motion and eye movements. The MAE in both fixation and tracking conditions was found to be horizontal. To discount the objection that the MAE may have appeared horizontal due to the vertical orientation of the test contours, as motion of lines tends to be seen in the direction orthogonal to orientation (Wallach, 1976), they replicated the experiment using a

moving random dot field in place of the grating. The results were identical.

The authors admit that an alternative explanation (suggested to them by Anstis) may be advocated based on the fact that relative motion is more effective for MAE production than absolute motion (Day & Strelow, 1971; see also Reinhardt-Rutland, 1987/88). Thus, while the horizontal component of the adapting pattern motion is relative to all other contours in view, the vertical component of its motion in the tracking condition is relative to the tracking point only. Accordingly, one might explain the lack of an oblique MAE on the weakness of the relative motion of the vertical component relative to the horizontal component. One fallacy of this explanation is that the mere fact that relative motion is more effective at producing MAEs than is absolute motion, suggests that retinal motion alone is not the basis of the MAE. But that does not necessarily mean that it is a comparison of retinal motion with eye movements that produces the MAE. Mack et al (1989), however, do claim that the relative motion explanation of their results is untenable, in light of the previous finding (Mack et al, 1987) that no MAE was produced by tracking, while fixation produced a strong MAE, despite the equivalence of the two conditions in terms of relative motion.

Consequences of imperfect fixation

In a recent report, Seidman, Thomas, Huebner, Billian and Leigh (1990) concluded that while eye movements sometimes accompany MAEs, neither they nor their consequences are the cause of MAEs. For linear motion, they found that pursuit eye movements occur during adaptation with attempted fixation, with a gain (ratio of pursuit velocity to motion velocity) of 0.1. During the subsequent test period, MAEs were observed in the direction opposite to the adapting motion, but no eye movements accompanied them. For rotational motion, torsional nystagmus was observed during the adaptation period with fixation, with a gain of 0.05. Following adaptation, an MAE was seen opposite to the adapting motion, and torsional eye movements in the same direction as the MAE also occurred. The eye movements did not last as long as the MAE, however. The MAE was opposite to the

direction of retinal slip due to eye movements, negating the possibility that the MAE resulted from retinal slip.

MAEs and pursuit after-nystagmus

Chaudhuri (1990) recently discovered an interesting effect relating to eye movements and MAEs. But let me preface a discussion of the phenomenon with the caveat that the author does not take this to be a "normal" MAE, suggesting that his findings may not relate to normally observed MAEs. Observers tracked a moving target in an otherwise dark room. If the motion ceases and the target is removed, pursuit eye movements in the same direction continue for a brief period following target removal. Muratore and Zee (1979) originally discovered this effect, and termed it "pursuit after-nystagmus" (PAN), emphasizing its similarity to previously observed optokinetic after-nystagmus (OKAN) (e.g. Cohen, Matsuo & Raphan, 1977; Raphan, Cohen & Matsuo, 1977; Waespe & Henn, 1977). Chaudhuri found that if a stationary test target is presented following the tracking period, an appropriate MAE is seen. Note that in his tracking condition, the only visible object is the tracked target, so that the only motion relative to the retina is the minimal relative motion due to the inaccuracy of tracking. Thus we would expect that an MAE would not result from such adaptation. In addition, Chaudhuri found that PAN is abolished when a stationary target is present following the tracking period.

Chaudhuri suggests the following mechanism to explain these results. When a stationary target is visible, PAN results in retinal slippage. The visual system thus generates pursuit movements in the opposite direction in order to maintain fixation. However, PAN and OKAN are believed to be reflexive, resulting from discharge in a subcortical component of the oculomotor system (Cohen, et al, 1977; Waespe & Henn, 1977; Cannon & Robinson, 1987; Chaudhuri, 1990). Thus no efferent signal corresponding to PAN reaches the motion system, while the pursuit signal is available to the motion system. Therefore, the visual system interprets the pursuit movements as signalling actual motion, so that motion is perceived in the direction opposite to PAN, and therefore opposite to the prior tracked motion.

This theory is supported by Chaudhuri's finding that with a test target in the form of an afterimage, PAN does occur, and the MAE

does not. Since retinal stabilization prevents retinal slip due to PAN, pursuit movements to counteract it are unnecessary. Further evidence lies in the finding that the temporal velocity profiles of the MAE (as assessed by a psychophysical nulling technique) and of PAN are correlated for the first two-thirds of the test period. Chaudhuri suggests that the difference toward the end of the test period may be attributable to the difficulty of accurate nulling at low MAE velocities, such that MAE velocity is underestimated toward the end of the decay period. He also established that the phenomenon also occurs for OKAN, whereby the tracked target is embedded in a field of random dots. He notes that Heckmann and Post (1988) have postulated perceptual registration of an efferent signal in the realm of induced motion as well.

How can this phenomenon be integrated with other results pertaining to the interaction of eye movements and MAEs? At this point it is not clear. Recall that this MAE is observed in the absence of any retinal motion during the adapting period. It is possible that this phenomenon only occurs in the absence of a normal MAE. Note however that this MAE is in the appropriate direction, and as such may be inseparable from a simultaneous MAE. It would be interesting to find a situation in which the possible presence of this effect concurrent with a normal MAE could be assessed. Another aspect of the PAN-related MAE warrants mention: after tracking a single target, an entire field of targets all appear to be moving in the direction of the MAE, even though all but the fixated target are in unadapted regions. This is not an instance of induced motion, which would entail motion in opposite directions for the central and other targets, or more likely, motion of either central or other targets, but not both. Likewise, the motion of the targets in unadapted regions can not be attributed to visible surround contours during tracking, as this would be manifested as a MAE in the same direction as prior tracking, rather than the opposite direction.

Another factor which may limit the relevance of this finding for an explanation of MAEs is that MAE studies generally employ fixation during adaptation, so PAN is unlikely to result. It would be interesting to know whether Seidman et al (1990) would have observed PAN

following adaptation with their linear motion stimulus. The gain of pursuit during adaptation was .1, indicating that the velocity of pursuit was about .75 deg/sec. If PAN were observed during the postadaptation phase in the absence of a test target, one could claim that the MAE observed is due to the mechanism suggested by Chaudhuri (1990), since, as predicted, eye movements were not observed during the MAE. It would also be informative to determine whether torsional eye movements result in residual movements analogous to PAN. Seidman et al (1990) found that unlike the linear MAE, the rotational MAE was accompanied by eye movements in the same direction. (While the gain was again small, the motion velocity was high, yielding substantial velocities for torsional movements during tracking of 3-9 deg/sec.) Note that these eye movements are opposite to the torsional movements during adaptation. Thus if we were to apply Chaudhuri's mechanism in this case, we would have to postulate an overcompensation by the torsional eye movement system in its attempt to counteract the after-nystagmus, resulting in these opposite movements. This overcompensation might eventually corrected, explaining the longer duration of the MAE than the eye movements. Of course at this point this is all speculation, and future work will be needed to ascertain whether Chaudhuri's MAE can be at all related to the mechanism of normal MAEs.

CONCLUDING REMARKS

This paper has presented an expansive and varied array of experimental evidence relating to the MAE and its neural basis. Rather than attempting to summarize the entire paper, I will briefly list the topics which must be addressed if we are to establish a functional model of MAE production which can account for all the relevant findings. In the process I will indirectly elaborate the status of our understanding of MAEs, by pointing out the unresolved issues.

At this point, the role of direction-selective cells and of adaptation of these cells in MAEs would be questioned by very few vision researchers. However, the process by which this adaptation occurs has remained elusive. Surely if we are to propose a model of MAEs based on cellular adaptation, we must learn how adaptation is mediated.

Also, the specific requirements of the role of direction-selective cells in mediating MAEs remain controversial. Are physiological adaptation effects directionally selective? Do decreases in spontaneous rates follow prolonged adaptation? Are the effects different for different cell types or in different areas? These and other questions must be answered if we are to fully explain the MAE. The development of a comprehensive model with specific, testable predictions regarding the cellular effects would expedite the investigative process, allowing the appropriate experiments to be designed in order for these issues to be addressed. Our progress in addressing these issues would also of course be aided by a resolution of the previous issue.

One finding not directly addressed in this paper is the finding that after a MAE initially decays, it can sometimes be elicited again at a later time. This phenomenon was illustrated in the discussion of the existence of short-term and long-term MAEs, whereby one MAE can be observed, then reversed, and then made to reappear again. These and related findings must be incorporated into a model of MAE production, as the current model can not account for them. I will get to the related phenomenon of storage of MAEs shortly.

Ample evidence exists to support the notion that adaptation occurs at different sites in the visual system. It will thus be necessary to assess the relative contributions of these various sites, and to determine how adaptation effects at different sites are related and how they are integrated. Clearly adaptation does occur at hierarchical levels. But is adaptation at each level responsible for MAEs, or is only the highest level involved? Are the adaptation effects at higher levels derived from those at lower levels? It is also possible that adaptation may occur in parallel pathways. Could binocular and monocular cells represent two functional adaptation processes? The finding that distinct binocular and monocular MAEs can be obtained supports this notion. In addition, it remains to be established whether direction-selective cells in the superior colliculus show adaptation effects. If so, we must then determine whether these effects are a byproduct of cortical adaptation, or whether they arise independently. If the latter is found to be true, then we must establish whether this system

represents a parallel pathway in MAE production, or whether the processes in this region are incorporated with those in cortical regions as determinants of the MAE.

Another issue mentioned only briefly is the existence of contingent MAEs. Several researchers have postulated that contingent aftereffects in general represent a basically different phenomenon than do simple MAEs. Thus it is possible that a more extensive model will be required to incorporate CMAEs as well, as an explanation of the mechanism of simple MAEs might not be sufficient to account for the complex stimulus interactions characteristic of contingent aftereffects..

It would also be desirable and useful to determine how various forms of adaptation not involving contour movement in the adapting region can generate MAEs. How does adapting to phantom motion result in an MAE in a region where no moving contours were present? (This issue is of course related to the question of why phantom motion is observed in the first place.) And how can adapting to a stimulus with no moving contours whatsoever yield an MAE?

The phenomenon of storage is a clear example of the fact that a simple ratio model can not fully explain the mechanism of generation of MAEs. Passive fatigue and recovery just can not be invoked as the process by which MAEs arise, since recovery is clearly altered by postadaptation conditions. How do these conditions interact with the rivalry mechanism(s)? What specifically occurs when a test stimulus is presented to cause decay of the MAE, and how is that process prevented in the absence of stimulation? How do the characteristics of the surrounding region affect the decay and storage of MAEs? It is essential that we answer these questions in order to develop a truly comprehensive explanation of the neural basis of MAEs. At this time no attempts have been made to explain storage in terms of cellular processes. Hopefully progress in establishing the mechanism of storage of other aftereffects will facilitate this process.

In closing, I would like to emphasize the fact, which should be readily apparent, that MAEs are a complex phenomenon, and that whatever their basis, it is far removed from the types of simple explanations posed by the earliest investigators. We have come a long way in our understanding of the MAE, but we still have a long way to go.

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