We examined the nature and the selectivity of the motion deficits produced by lesions of extrastriate areas MT and MST. Lesions were made by injecting ibotenic acid into the representation of the left visual field in two macaque monkeys. The monkeys discriminated two stimuli that differed either in stimulus direction or orientation. Direction and orientation discrimination were assessed by measuring thresholds with gratings and random-dots placed in the intact or lesioned visual fields. At the start of behavioral testing, we found pronounced, motion-specific deficits in thresholds for all types of moving stimuli, including pronounced elevations in contrast thresholds and in signal-to-noise thresholds measured with moving gratings, as well as deficits in direction range thresholds and motion coherence measured with random-dot stimuli. In addition, the accuracy of direction discrimination was reduced at smaller spatial displacements (i.e. step sizes), suggesting an increase in spatial scale of the residual directional mechanism. Subsequent improvements in thresholds were seen with all motion stimuli, as behavioral training progressed, and these improvements occurred only with extensive behavioral testing in the lesioned visual field. These improvements were particularly pronounced for stimuli not masked by noise. On the other hand, deficits in the ability to extract motion from noisy stimuli and in the accuracy of direction discrimination persisted despite extensive behavioral training. These results demonstrate the importance of areas MT and MST for the perception of motion direction, particularly in the presence of noise. In addition, they provide evidence for the importance of behavioral training for functional recovery after cortical lesions. The data also strongly support the idea of functional specialization of areas MT and MST for motion processing.

Introduction
Extrastriate area MT is one of the more extensively studied components of the dorsal cortical stream (Felleman and Van Essen, 1991; Merigan and Maunsell, 1993). The large proportion of directionally selective neurons in this area, the complex nature of their receptive fields, and the discovery that they integrate low-level, localized motion signals (Movshon et al., 1985; Rodman and Albright, 1989; Britten et al., 1992) has led to the widely accepted notion that this area is specialized for processing of visual motion information. Additional support for this notion comes from physiological studies showing that the sensitivity of MT neurons to visual motion is similar to that measured psychophysically, and that local microstimulation of MT and MST neurons alters the visual choices of a monkey towards the direction to which the stimulated neurons are most sensitive (Salzman et al., 1992; Celebrini and Newsome, 1994, 1995). Similarly, the motion responses of neurons in area MST, which receives major inputs from MT, also suggest a role in motion processing, specifically in the analysis of object motion and optic flow (Duffy and Wurtz, 1991; Tanaka and Saito, 1989) and in pursuit eye movements (Komatsu and Wurtz, 1988).

Additional evidence implicating areas MT and MST in the processing of motion information comes from lesion studies (Newsome and Paré, 1988; Schiller, 1993; Pasternak and Merigan, 1994; Schiller and Lee, 1994; Orban et al., 1995). These studies have revealed deficits in discriminations involving complex motion stimuli masked by noise and a reduction in the accuracy of discrimination of speed and direction of motion. In addition, a number of studies reported abnormalities in smooth pursuit eye movements (Dursteller and Wurtz, 1988; Newsome et al., 1985; Yamasaki and Wurtz, 1991). In a previous study from this laboratory, Pasternak and Merigan (1994) reported that monkeys with large bilateral lesions of areas MT and MST showed permanent deficits in direction discrimination of complex random-dot stimuli masked by noise, but no lasting deficits in contrast sensitivity for direction, measured with moving gratings. This result led to the conclusion that neurons in areas MT/MST contribute primarily to processing of more complex motion masked by noise. The present study was undertaken to determine whether previously observed permanent deficits can be attributed to the presence of motion noise or to the use of complex random-dot stimuli. The second goal was to determine the specificity of the deficits and to examine some of the factors involved in commonly observed improvements of motion thresholds after the lesions. This was accomplished by placing unilateral lesions in monkeys whose fixation was controlled with scleral search coils and comparing thresholds for a variety of types of visual motion stimuli placed in the intact and the lesioned visual. We found large deficits in direction discrimination with both simple and complex motion stimuli early in the post-lesion training. With continued testing, the longest enduring threshold elevations were in the accuracy of direction discrimination and in the discrimination of stimulus direction in the presence of motion noise. This increased susceptibility to noise was specific to tasks involving direction discrimination.

Materials and Methods
Subjects
Two young adult male monkeys (Macaca nemestrina) were used. On weekdays, water was restricted for a period of 23 h before testing and their daily water ration, in the form of fruit juice, was provided during behavioral testing. On weekends, the monkeys were not tested behaviorally and had unrestricted access to water. Food was continually available in the home cage and monkeys received supplements of fresh fruit and vitamins daily. Body weights were measured twice weekly to ensure good health and normal growth. Experiments were carried out in accordance with the guidelines published in the NIH Guide for the Care and Use of Laboratory Animals (NIH publication no. 86–23, revised 1987).

Stimuli
Gratings
Drifting sinusoidal gratings were generated online by a Macintosh computer and displayed on a 17 in. Nanano monitor. The stimuli had a
Circular aperture, were 4° in diameter, and were viewed at a distance of 42 cm. The mean luminance of the display was set at 20 cd/m². For Monkey 1, the presentation of the sample grating was terminated by pressing a middle button and the sample onset and the offset followed a 200 ms cosine. Since the response latency for that monkey ranged between 600 and 800 ms, of which ~250–300 ms accounts for a motor response. (Beck and Chambers, 1970; Schieber and Poliakov, 1998), sample duration for this monkey was ~350–500 ms. The duration of the sample grating for the other monkey was not limited by the animal and followed a 350 ms smooth cosine temporal envelope. For both monkeys, the timecourse of the test stimulus followed a 300 ms raised cosine envelope. The gratings were set to 1 c/deg and moved or flickered in counterphase at 1, 5 or 10 Hz. Direction discrimination thresholds were measured by varying the grating contrast (Fig. 1A), or by introducing one-dimensional dynamic spatial noise (Fig. 1B). Orientation discrimination thresholds were measured by varying the contrast of the grating or by adding two-dimensional static spatial noise to a high-contrast sinusoidal grating (Fig. 1C). Stimuli masked by one- and two-dimensional noise were developed by Dr Charlie Chubb, University of California, Irvine, who generously provided the software for their generation.

Dynamic one-dimensional noise consisted of a pattern of randomly appearing bars (2 min in width) with orientation identical to the sample grating; the intensities of the bars were jointly independent and uniformly distributed between 10 and 30 cd/m². Stimulus degradation was achieved by randomly replacing a specified proportion of pixels in the grating by such noise bars. The contrast of the grating masked by dynamic noise was set to 20% while the contrast of the noise bars ranged from 0 to 50%.

Signal-to-noise was computed as follows:

\[
\text{signal-to-noise} = \frac{|S - I|}{\sqrt{(S - S)(I - I) - (S - I)^2}}
\]

where

\[
S - I = \sum_{p} S(p)I(p)
\]

\[
S - S = \sum_{p} S(p)S(p)
\]

\[
I - I = \sum_{p} I(p)I(p)
\]

where \( S \) is a degraded sinusoidal grating and \( I \) is an undegraded sinusoidal grating.

Two-dimensional spatial noise was generated by adding a sinewave luminance modulation of a fixed contrast (50%) to the two-dimensional, three-pixel square noise elements. The intensities of the noise elements were uniformly distributed over the same range of intensities as the grating.

\[
\text{Signal-to-noise} = k \left( \frac{\text{SignalProportion}}{1 - \text{SignalProportion}} \right)
\]

for \( k = \sqrt{(3\pi/2)} \), where SignalProportion is a number between 0 and 1 that controls the relative proportions of signal and noise additively combined to produce the stimulus.

Contrast and signal-to-noise thresholds were measured either when sample and test moved in opposite directions or when their orientations were orthogonal to each other.

**Random-dot Stimuli**

The dot stimuli were identical to those used previously (Pasternak et al., 1990; Pasternak and Merigan, 1994) and consisted of dots repeatedly displaced with a direction of motion chosen randomly from a uniform distribution of directions. The dots moved at a speed of 5°/s (\( \Delta t = 13 \) ms; \( \Delta x = 0.065° \)) and, like the gratings, were displayed on a Nanao monitor placed at a distance of 42 cm. Each stimulus contained 75 dots, the dot density in all experiments was 1.5 dots/deg² and the mean luminance of the display was 0.1 cd/m². Each dot was 0.03° in diameter and its luminance was set to ~3.5 log units above detection threshold for human observers. The lifetime of individual dots was either equal to the duration of stimulus presentation (350–500 ms) or was set at 100 ms. Discrimination difficulty was changed by varying the range of the direction distribution (Fig. 1D) or the proportion of the dots moving in non-random directions (Fig. 1E).

**Behavioral Procedures**

The monkeys were seated in a primate chair with two pushbuttons for Monkey 2 and three buttons for Monkey 1. They were trained to fixate a small spot at the center of the display while performing the discrimination task in a chosen location of the visual field as shown in Figure 2. Two comparison stimuli appeared in succession in the same retinal location and the monkeys were required to judge them as the ‘same’ or ‘different’ by pressing the right or the left response button. The monkey initiated the trial by fixating a small spot for 500–1000 ms. This resulted in a tone signaling the start of the trial and presentation of the ‘same’ and then the test stimulus. The duration of the sample was limited for one monkey (Monkey 2) and was terminated by the middle button press for
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plates with dental cement consisting of a brass rod attached to the monkey's skull by means of a 20 cm field coil and its head held firmly by a head-restraint system. Behavioral testing, each monkey was placed in a magnetic field generated by a 200 ms delay. Fixation was maintained until the test stimulus disappeared. The monkey indicated whether the test was either same or different than the sample by pressing the right or left button respectively. Direction discrimination: one of eight possible directions of motion was selected at random as a sample stimulus and test stimulus moved either in the same or different direction. Orientation discrimination: one of six possible orientations of flickering gratings was selected at random as a sample and the test was either the same or orthogonal to the sample.

the other monkey (Monkey 1) (see below). Incorrect responses resulted in a 3–6 s tone and no reward. A break in fixation or a response during the presentation of the sample or the test stimuli resulted in a brief tone and the termination of the trial. The sequence of presentation of same/different stimulus pairs was randomized from trial to trial. To avoid position biases, a correction procedure was used: after three consecutive errors on one button, the same trial was repeated until the animal made a correct response (which was not included in the data analysis). Each testing session consisted of 300–500 trials separated by a 3 s intertrial interval.

Initial training was performed with two comparison stimuli presented simultaneously, one below the other. In this version of the task, the sample remained on the screen and the test was placed below it. After the animals reach criterion performance (three consecutive sessions >90% correct or four sessions >80%), they were trained with a delay condition in which the sample was turned off and the test stimulus turned on. Gradually, the locations of the sample and the test were moved closer together until they appeared in the same retinal location.

Threshold Measurements

Thresholds were measured after the monkeys reached criterion performance on the task. A staircase procedure was used: three correct responses resulted in a less discriminable stimulus (e.g. reduced contrast or increased noise), and each incorrect response decreased the difficulty of discrimination. Thresholds were measured by varying only the sample stimulus along one of several dimensions (contrast, signal-to-noise, etc.). The test stimulus always remained highly discriminable. Thresholds were estimated by fitting a Weibull function to the resulting psychometric data, and determining the stimulus value corresponding to 75% correct. At least three or four threshold determinations were made for each stimulus condition after performance reached stability (i.e. within ±10% of the mean).

Control and Calibration of Eye Position

Eye position was monitored with magnetic search coils. A scleral search coil and head restraint device were implanted after the monkey had adapted to the apparatus and learned the basic behavioral task. During behavioral testing, each monkey was placed in a magnetic field generated by a 20 cm field coil and its head held firmly by a head-restraint system consisting of a brass rod attached to the monkey's skull by means of titanium orthopedic plates and screws. The brass post was attached to the plates with dental cement -1 cm above the skull. Eye position was calibrated prior to each daily psychophysical session by rewarding the monkey for positioning its gaze within an electronically defined 0.75–1° window centered on the fixation spot and maintaining it within this window for 700–1000 ms. The spot was positioned in random locations on the display screen within the central 15°. This allowed the adjustment of signal gain and offset. Approximately 10 trials were usually needed for this calibration.

Lesions

Ibotenic acid lesions were made using procedures identical to those reported by Pasternak and Merigan (1994). Ibotenic acid (10 µg/µl) was injected at multiple sites along the superior temporal sulcus (STS) in one hemisphere. Two magnetic resonance image (MRI) brain scans, one before the lesion and one several months after the lesion, were made for each monkey. Injections were guided by the MR images and direct visualization of the STS. Each injection (1.5 µl) was made with a Hamilton syringe with -2.5 mm separation at several depths at each location. Both monkeys received injections into the right hemisphere (Monkey 1, 90 injections; Monkey 2, 73 injections).

Magnetic Resonance Imaging

The brains were scanned on 2 and 1.5 T GE magnets. T2-weighted images with high signal-to-noise ratio were obtained with a small surface coil. Coronal and horizontal scans were performed with the following parameters: T E/T R 5000/90 or 3000/85; 1.5 mm thick slices 0.2 mm apart; 256 × 256 array, field of view 10 or 15 cm. This method provided sufficient gray matter/white matter contrast to visualize many of the anatomical details and the location and extent of ibotenic acid lesions. The images were examined and analyzed with NIH Image software.

For this procedure, the monkeys were anesthetized with sodium pentobarbital (25 mg/kg i.v.) to effect, placed in a specially constructed MRI-compatible stereotaxic holder, intubated and continuously monitored by EKG. The ear bars were filled with water or vitamin E, which was visible on the MR images. This allowed the determination of stereotaxic coordinates in the Horsley–Clark coordinate system.

Results

Lesion Extent

The two monkeys are currently participating in other experiments, and therefore the histological assessment of their lesions is not yet available. We used T2-weighted MRI scans to determine the location and the extent of the lesions. The damaged regions appeared brighter than the surrounding tissue with a distinctly fuzzy, defocused appearance. Figure 3 shows a series of coronal slices, 1.5 mm thick separated by 0.2 mm, spanning the extent of the lesion for both monkeys obtained before and after the lesions. We were able to obtain a fairly close match between the corresponding preoperative and postoperative scans. The slight differences in the appearance of the sulcal pattern between the matching pre- and postoperative sections are most likely due to a slight variation in the positioning of the brain in the scanner, and in the choice of the image plane. Moreover, the slices are fairly thick, and thus average signals spanning across regions may contain fairly large variation in shape of the sulcus. The lesions are visible as brighter regions in both monkeys, although the images of the lesions in Monkey 2 are more distinct. This difference is most likely due to the better signal-to-noise ratio in the 1.5 T magnet used for postoperative scan of that monkey.

To identify specific cortical areas, we matched the appearance of the sulcal pattern to published physiological and anatomical data indicating the relative of specific visual areas (Boussaud et al., 1990; Desimone and Ungerleider, 1986; Ungerleider, 1995). The approximate locations of visual cortical areas FST, STP, MT, MST, V4 and 7a are shown on the pre-lesion slices in both monkeys.

Monkey 1

The damaged region of cortex included large portions of MT and
MST in the right hemisphere. Cortex on the inferior parietal gyrus and inside the STS was damaged over a 12 mm region extending in the posterior and dorsal direction from the anterior margin of the lesion. There was also damage to some of the adjacent regions including portions of STP, FST and 7a. In addition, a portion of the representation of the lower contralateral quadrant of the visual field in V4 and V4t was damaged.

**Monkey 2**  
The damaged region of cortex extended over ~12 mm along the STS of the right hemisphere. The lesion included most of areas MT and MST, but appears to have spared the more anterior portions of both areas. These spared portions of MT and MST are likely to contain the representation of more central visual field (Desimone and Ungerleider, 1986; Komatsu and Wurtz, 1988). Portions of areas 7a, and the representation of the lower quadrant in V4t and V4 also sustained some damage.

**Psychophysical Mapping of Lesions**  
To ensure that the stimulus was placed entirely within the damaged portion of the visual field, we measured motion signal thresholds at multiple adjacent positions in the intact and lesioned portions of the upper visual field. This measure has been shown to be permanently affected by MT/MST lesions (Pasternak and Merigan, 1994). Threshold measurements were limited to the upper visual field to prevent contamination of results by unintended damage to the adjacent representation of the lower field in area V4. Motion signal thresholds in both monkeys were elevated throughout the upper quadrant contralateral to the lesion as compared to the corresponding locations in the ipsilateral, intact field. In Monkey 1, the mapping was performed with a 4° target in ten overlapping locations within the central 10°. A 1.5- to 2-fold deficit was present throughout the this portion of the visual field with the maximal deficit localized at an azimuth of 5°, 5° above the horizontal meridian. In Monkey 2, the thresholds were measured with a 3° target in nine adjacent non-overlapping locations within the central 10° and compared to the corresponding locations in the intact quadrant. A 3- to 5-fold deficit was present in all eight locations with the greatest loss found at 8° azimuth just above the horizontal meridian. For further psychophysical testing we selected the locations of the maximal threshold elevation in the lesioned hemifield and the corresponding locations on the opposite (intact) side of the vertical meridian.

**Direction Discrimination**

**Grating stimuli**

**Contrast Thresholds.** Contrast sensitivity for discriminating the direction of gratings drifting at 5°/s is shown for both monkeys in Figure 4D. These data represent stable performance measured at the completion of the study, many months (12–24 months) after the lesion. Monkey 1 showed no appreciable residual loss, while Monkey 2 showed a small depression in sensitivity (0.1 log units). The same pattern of results was found when contrast thresholds were measured at lower and higher speeds (1 and 10°/s). At these speeds, the intact and lesion thresholds for Monkey 1 were identical, while Monkey 2 showed a small depression in sensitivity, identical to that observed at 5°/s.

Sensitivity loss measured early in post-lesion training and at the end of the experiment, computed as a ratio between the intact and lesioned thresholds, is shown in Figure 5A. For Monkey 1, ‘early’ testing was performed in the lower visual field. The initial loss in sensitivity (left plots) nearly disappeared within the next four testing sessions and the performance reached the intact levels. The subsequent shift in the testing location to the upper field did not reveal substantial deficits in contrast thresholds during the early stages of testing in that location. This suggests that the recovery in contrast sensitivity observed in the lower visual field may have transferred to the upper visual field.

**Signal-to-noise Thresholds.** Thresholds for discriminating the direction of drifting gratings, masked by dynamic one-dimensional noise, are shown in Figure 4B. Unlike contrast sensitivity, which after the initial deficit showed substantial recovery, the residual deficit in signal-to-noise thresholds remained pronounced in both monkeys. These data represent stable performance, measured ~13–24 months after the initiation of training in the lesioned visual field. Earlier in training (Monkey 1: 52 days of training and 3 months after the surgery; Monkey 2: 10 days of training, 6.5 months after surgery), the deficits were more severe (see Fig. 5B) and with continued training improved somewhat, although never reaching intact levels.

**Random-dot Stimuli**

**Range Thresholds.** The final direction range thresholds (Fig. 4C) were measured ~8–12.5 months after the start of behavioral training in the lesioned portion of the visual field. A comparison of early and final thresholds is shown in Figure 5C. The early training data for Monkey 1 were measured 7 weeks after the lesion and after 7 days of training within the lesioned quadrant. The early data for Monkey 2 were measured ~30 days after the beginning of training within the lesion. The initial 2.5- to 5-fold threshold elevations returned to near normal levels within the next several months of training. These results show that after extensive training, initial severe deficits in motion integration produced by MT/MST lesions recovered almost completely.

**Motion Signal Thresholds.** Figure 4D shows the final motion signal thresholds obtained from the two monkeys many months after the surgery. The loss measured at the start of behavioral testing in the lesioned field compared to the loss measured at the completion of testing is shown in Figure 5D. Both monkeys showed final threshold elevations on the order of 30–40%, although early in training the deficits in both monkeys were much more pronounced. Monkey 1 was tested in the lesioned portion of the visual field 7 weeks after the surgery and showed a 3.2-fold threshold elevation. Monkey 2, when tested ~1 month after the lesion, showed nearly a 5-fold elevation in motion signal threshold. Although the magnitude of the deficit decreased dramatically during the first month of training on this task, performance never reached control levels.

**Direction Difference Thresholds.** The effect of MT/MST lesion on the accuracy of direction discrimination was assessed with coherently moving random-dot stimuli. The data in Figure 6A show the relationship between the spatial displacement of individual dots (Δx) and direction difference thresholds measured in Monkey 1.

The deficit in accuracy of discrimination was greatest for the smallest spatial displacement (0.065°) and decreased with...
Figure 3. MT/MST lesions. Coronal $T_2$-weighted scans through the brains of the two monkeys. The slices are 1.5 mm thick and separated by 0.2 mm. Preoperative scans for both monkeys and the postoperative scan for Monkey 1 have been obtained in a 2 T magnet ($T_E/T_R$ 2000/60; FOV 100 mm). Postoperative scans of Monkey 2 were obtained in a 1.5 T magnet ($T_E/T_R$ 5000/85; FOV 160 mm). The lesions appear brighter than the surrounding tissue with a distinctly fuzzy, defocused appearance. Abbreviations: ios, inferior occipital sulcus; ips, intraparietal sulcus; lat, lateral sulcus; lu, lunate; opts, occipital temporal sulcus; sts, superior temporal sulcus; pmts, posteromedial temporal sulcus.
increasing step size. The rightmost point on the plot shows the
maximal spatial displacement ($D_{\text{max}}$) measured by setting
the difference in direction to 90° and varying $\Delta x$. The relative
improvement in the accuracy of discrimination at larger step
sizes in the lesioned location suggests an increase in the spatial
scale of the residual directional mechanism (Rudolph
et al., 1994). Since the change in spatial displacement was accom-
panied by a change in speed, it is not clear whether it is the
increase in $\Delta x$ or in speed that determined the improvement in
the accuracy of discrimination. To resolve this question we
examined the effect of changing speed on the accuracy of
discrimination without changing the step size. This was
accomplished by varying the temporal interval ($\Delta t$) between
individual dot displacements and setting $\Delta x$ to 0.45° (Fig. 6B).
The deficit measured previously at a speed of 5°/s ($\Delta t = 13$ ms; $\Delta x = 0.065$°) (see Fig. 6A) reappeared with this
larger step size when the temporal interval was increased so that
the speed was lowered to 7°/s. Thus, after MT/MST lesion, the
residual directional mechanism appears to prefer larger spatial
displacements and shorter temporal intervals, i.e. higher speeds.

Measurements of direction difference thresholds began ~14
months after the lesion. At that time, the direction difference
threshold measured in the lesioned location was a factor of 1.6
higher than that measured on the intact side. Although the
deficit decreased somewhat with training, it never reached the
control levels (Fig. 6C).

**Orientation Discrimination**

To assess the selectivity of the lesion effects, we also measured
the ability of the monkeys to discriminate stimulus orientation.
We used flickering gratings of the same spatial and temporal
frequency as the moving gratings in the direction discrimination
experiment. Thresholds were measured by varying grating
contrast or masking a 20% contrast grating with two-dimensional
spatial noise. The results are shown in Figure 7.

Monkeys 1 showed no detectable deficit in the discrimination
of stimulus orientation; its contrast and signal-to-noise thresholds
were in the normal range. Monkey 2 had a small deficit in
contrast thresholds, similar to that found in the direction
discrimination task, suggesting that the modest deficit in
contrast sensitivity in the two tasks may be a reflection of
generalized depression in sensitivity following the lesion. In
contrast to the signal-to-noise thresholds for direction, we found
no deficit in signal-to-noise thresholds for orientation. These
thresholds were measured 18 months and 7 months after the
lesion in Monkey 1 and Monkey 2 respectively. Although
contrast thresholds improved gradually with training, the extent
of this improvement was the same in the intact and the lesioned

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**Figure 4.** Permanent effects of MT/MST lesions on direction discrimination. Thresholds measured at the completion of the study, ~12–24 months after the lesions. On each trial the
direction of the sample was chosen at random from a set of eight directions. Sample and test stimuli always moved in the same or opposite directions. Stimulus position: Monkey 1 — target centered at 5° azimuth and 5° elevation; Monkey 2 — target centered at 8° azimuth and 2° elevation. Stimulus size, 4° diagonally. (A) Contrast sensitivity (1/contrast
thresholds × 100). Contrast thresholds were measured in the intact and the lesioned portions of the visual field. Spatial frequency, 1 c/deg; speed, 5°/s (temporal frequency, 5 Hz). (B) Signal-to-noise thresholds measured by varying the proportion of pixels in the grating that assumed random intensities. Spatial and temporal (speed) frequency, target size and
position the same as in (A). (C) Direction range thresholds. Speed = 5°/s ($\Delta t = 13$ ms; $\Delta x = 0.065$°). (D) Motion signal thresholds. Speed = 5°/s ($\Delta t = 13$ ms; $\Delta x = 0.065$°). Error
bars are ±1 SEM. See text for other details.
fields. The lack of measurable effects on orientation discrimination in the presence of two-dimensional noise suggests that the increased susceptibility to noise observed in direction discrimination is specific to the domain of motion.

**Discussion**

The most pronounced and permanent effect of the lesions was on the discrimination of motion direction in the presence of noise. This increased and lasting susceptibility to noise was evident with simple moving gratings as well as with random-dot stimuli. In addition, the accuracy of direction discriminations was permanently reduced, particularly at smaller spatial and larger temporal displacements (i.e. lower speeds). The long-term effect on integration of local directional signals was minimal, although much more severe deficits were observed earlier in post-lesion training and these were not limited to stimuli masked by noise. The improvements in thresholds appeared to be a function of continued training in the lesioned portion of the visual field, rather than time since the lesion. On the other hand, orientation discrimination assessed by measuring contrast thresholds and signal-to-noise thresholds was largely unaffected, demonstrating that neurons in areas MT/MST contribute uniquely to motion discrimination.

**Lesion Location and Extent**

The comparison of preoperative and postoperative MRI scans obtained from both monkeys allowed a fairly detailed evaluation of the extent and location of cortical damage. This evaluation was facilitated by the availability of a direct comparison of the ibotenic acid lesions visualized in the MR scans and subsequent histological analysis of the same tissue obtained from a monkey and several cats involved in other studies in the laboratory (T. Pasternak, unpublished observations). This comparison provided us with the information about the appearance in the MR scans of cortical tissue devoid of neurons. In addition, in our previous study of the effects of MT/MST lesions, the lesions were made using the same approach as here and were reconstructed histologically (Pasternak and Merigan, 1994). On the other hand, it is important to keep in mind that MRI does not provide the precision of the histological analysis. The limitations are imposed by the thickness of the MR slices (1.5 mm) and the inability to visualize the precise location of MT, which in the histological section stained for myelin can be seen quite readily (Van Essen et al., 1981). Although the location of MT can vary from animal to animal with respect to the Horsley–Clark stereotaxic coordinates, its position within STS appears to be fairly constant (Van Essen et al., 1981). We thus compared the sulcal pattern visible in the MRI scans to the published reports (Desimone and Ungerleider, 1986; Boussaoud et al., 1990; Ungerleider, 1995) to estimate the location of MT, MST and other major visual areas.

According to these estimates, the lesions in both monkeys were marked throughout areas MT and MST, and there was some involvement of adjacent areas, including area 7a and the representation of portions of the lower visual field in area V4. The damage to V4, however, is unlikely to complicate the interpretation of our results, since we collected our data primarily with stimuli placed in the upper visual field. Psychophysical
mapping of the extent of the lesions showed the largest deficits at locations beyond the central 3–4°. This relative sparing of the central representation is consistent with the pattern of cortical damage revealed by MRI, which shows sparing of cortical tissue in the more anterior end of MT and MST, which contains more central representation of the visual field (Desimone and Ungerleider, 1986).

**Discrimination of Grating Direction**

In both monkeys, measurements of contrast thresholds in the lesioned visual field began ~2 months after the lesion. At that time, we found approximately a 2-fold loss in sensitivity in both animals. This loss decreased with continued testing over the next several months as both monkeys were periodically retested. The performance of Monkey 1 reached normal levels relatively rapidly (within a week) while that of Monkey 2 remained slightly depressed even after several months of testing. In that monkey, a small loss in sensitivity was also evident in the orientation discrimination task (see Fig. 7), suggesting that the sensitivity loss was not specific to motion and may be a reflection of a generalized depression in overall sensitivity in the lesioned portion of the visual field. This effect did not appear to be related to the extent of the lesion, as there was no long-term effect in Monkey 1, whose lesion was comparable to that of Monkey 2. The decreased loss of contrast sensitivity with training and the lack of large permanent effects of the lesion on contrast thresholds confirms a previous report that large bilateral MT/MST lesions produced early deficits, but little or no lasting effect on contrast sensitivity (Pasternak and Merigan, 1994). One of the monkeys in that study also showed a small generalized decrease in sensitivity, which did not appear to be motion specific. An analogous finding of largely preserved contrast sensitivity for direction has also been reported after damage to cortical area LS in the cat (Pasternak et al., 1989) and in a patient with damage to parieto-occipital cortex (Plant et al., 1993).

While the deficits in contrast thresholds were relatively modest and largely transient, the deficit in signal-to-noise thresholds, measured with high contrast gratings, was more severe initially and the recovery more limited. This finding of increased susceptibility to noise is consistent with previous results for random dot patterns from this laboratory (Pasternak and Merigan, 1994), as well as those of Newsome and Paré (1988). The present result extends the range of stimuli that reveal a reduced ability to extract motion from noise in the absence of MT/MST neurons from complex dots to grating stimuli. Moreover, it shows that the increased susceptibility to noise after MT/MST lesions is motion specific, since signal-to-noise thresholds for orientation discrimination measured with gratings under the same spatiotemporal conditions were unaffected by these lesions.

The transient depression in contrast sensitivity for direction suggests that MT/MST neurons contribute to the processing of even simple directional signals, such as those generated by drifting gratings. However, our results suggest that with continued testing this function may be assumed quite effectively by directionally selective neurons in other visual areas. In addition, we cannot rule out the contribution of neurons in spared portions of MT and/or MST, some of which have been shown to expand their receptive fields into the lesion (Sober et al., 1997). In contrast, the ability to discriminate grating direction in the presence of noise appears to be less robust and remained deficient despite extensive retraining. This result,
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Random-dot Stimuli
The initially measured deficit in discriminating the direction of random-dots was severe in both monkeys: one monkey was unable to reliably perform the task even with completely coherent motion, while the other could only do so with stimuli containing very low levels of noise or a narrow range of directions. When performance improved sufficiently to allow the determination of thresholds, both monkeys showed large deficits in thresholds for both direction range and motion signal. With further testing, range thresholds improved quite rapidly and almost reached intact levels. On the other hand, although the magnitude of the deficits in motion signal thresholds decreased in both monkeys, these thresholds did not recover completely despite extensive additional training. A similar pattern of early deficits and subsequent recovery in the two types of thresholds was reported for monkeys with extensive bilateral lesions of MT and MST (Pasternak and Merigan, 1994).

The initial defects in motion integration and in the extraction of motion signals from noise suggest that MT/MST neurons may normally contribute to both functions. However, the nearly complete recovery in motion integration suggests that the mechanisms in MT/MST are not unique in their ability to integrate local direction signals. Indeed, it has recently been shown that an earlier visual cortical area, area V3, contains a population of neurons that perform this computation, responding to the global direction of motion of a pattern, rather than the direction of its component vectors (Gegenfurtner et al., 1997).

The persistent deficit in discriminating motion in the presence of motion noise suggests that areas MT/MST could be uniquely specialized for processing motion in the presence of noise. Alternatively, this increased susceptibility to motion noise may be a consequence of the motion pathway losing a significant number of directionally selective cells. This latter possibility is supported by the finding that cats with widespread loss of directionally selective neurons showed a similar reduction in motion signal threshold (Rudolph and Pasternak, 1994). Whatever the locus of the mechanism responsible for sifting the veridical directional signal out of a noisy background, it is likely to be of major functional significance, since scenes containing a moving object of interest are likely to contain other, irrelevant direction vectors. A similar deficit for ‘noisy’ motion stimuli has also been reported in patients with damage to posterior temporal cortex (Vaina, 1989; Baker et al., 1991; Vaina and Cowey, 1996; Braun et al., 1998) and in cats with lesions of the lateral suprasylvian cortex (Rudolph and Pasternak, 1996), areas implicated in the processing of motion information in the two species.

Accuracy of Direction Discrimination
In a previous study, we found that monkeys with large bilateral lesions of areas MT and MST are less accurate in discriminating differences in motion direction of two simultaneously presented random-dot patterns (Pasternak and Merigan, 1994). This deficit decreased at larger step sizes and was not detectable at the largest spatial displacements. The present study confirmed the existence and extent of the previously reported deficit. This function was first measured 14 months after surgery, following extensive training on other tasks in the lesioned visual field. Nevertheless, we did observe some training induced improvements in thresholds in the lesion (see Fig. 6C). This result suggests a role for neurons in area MT and/or MST in mediating this function in the intact brain. The residual threshold elevation probably reflects the assumption of this ability by surviving directionally selective neurons with suboptimal directional tuning or greater variability, the two factors implicated in determining the accuracy of discrimination (Wilson and Gelb, 1984).

We determined the spatiotemporal properties of the residual directional mechanism by manipulating the size of spatial displacements and the temporal interval between those displacements. In the lesion, a 7-fold change in step size eliminated the direction threshold loss, suggesting a shift towards larger optimal spatial displacement. The same manipulation in the intact field produced no change in threshold. This shift in the optimal spatial displacement was accompanied by a shift towards shorter preferred temporal intervals. Thus, the residual motion mechanism mediating the task appears to operate at larger spatial scales (Baker and Braddick, 1985) and shorter temporal intervals. This result confirms and extends the previous finding of the increased spatial scale of the directional mechanism after MT lesions (Pasternak and Merigan, 1994).

The initial deficits in motion integration and in the extraction of motion signals from noise suggest that MT/MST neurons may play a key role in the extraction of motion signals from noise.

Random-dot Stimuli
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Specificity of the Deficit
The gratings used to measure thresholds for discriminating stimulus direction and orientation were of the same spatial and temporal frequency. The speed of the random-dot targets was also matched to the speed and the flicker rate of the gratings. The spatiotemporal equivalence of all stimuli used in this study permits an easy comparison of the effects of lesions on different tasks. The permanent deficit in noise-masked discriminations was present when the animal discriminated stimulus direction and not when it discriminated stimulus orientation. Previous studies have shown specificity of MT lesions on some discriminations. For example, Newsome and Pare (1988) reported that MT lesions resulted in transient deficits in motion coherence thresholds but did not affect contrast sensitivity for discriminating orientation of stationary gratings. We previously showed deficits in the accuracy of direction discrimination but intact
accuracy in the discrimination of spatial frequency discrimination (Pasternak and Merigan, 1994).

The intact signal-to-noise thresholds for orientation discrimination suggest that increased susceptibility to noise is not simply due to an increase in the amount of noise throughout the visual system. They also suggest that deficits in motion processing cannot be attributed to a general decline in performance in the lesioned portion of the visual field. Rather, the selectivity of the signal-to-noise deficit suggests that the two major stimulus features, orientation and direction of motion, are likely to be processed by separate cortical mechanisms.

**Behavioral Training and Improvements in Thresholds**

One of the most striking findings of this study is the extent to which many aspects of motion perception had returned to nearly normal levels by the time the experiments were completed. This is in stark contrast to the initial evaluation of lesion effects when we observed relatively large deficits in nearly all aspects of direction discrimination. Although the present experiments were not designed to study postoperative recovery, some observations suggest that this recovery did not result from the passage of time since the lesion, but rather that it was induced and/or facilitated by behavioral training. For example, the initial deficits in range and motion signal thresholds in Monkey 1 (see Fig. 5C,D) were first measured 7 weeks after the surgery, during the first few days of testing in the lesioned quadrant. During the 7 week period that followed the surgery, the monkey was tested with stimuli placed in the intact hemifield. This experience did not appear to have any beneficial effect on initial performance in the lesioned quadrant, which started to improve only after the animal began working with stimuli placed in that portion of the visual field. Similarly, measurements of direction difference thresholds in the same monkey began 14 months after the placement of the lesion and following extensive training and recovery on other motion tasks. While the thresholds measured in the intact field improved somewhat with continued training, the improvements in the lesioned field were substantially more pronounced, resulting in a small residual deficit at the conclusion of testing.

A number of studies have previously reported that monkeys with MT lesions show improvements or even complete functional recovery within a few days after the lesion (Newsome et al., 1985; Durstberger and Wurtz, 1988; Yamashita and Wurtz, 1991). This relatively rapid recovery appears to be quantitatively different from our observations of deficits detectable even a year after the lesion. The most likely explanation of this apparent inconsistency is the time at which threshold measurements began in the lesioned portion of the visual field. In studies where the recovery was observed within days after the lesion, the damage was produced by injecting ibotenic acid into a retinotopically localized portion of MT while the monkey was performing a behavioral task in the corresponding portion of the visual field. Thus, immediately after the lesion and in the days that followed, the animal received extensive behavioral training in the damaged visual field. On the other hand, in our experiments this type of training occurred many days or even months after the lesion. This suggests that the necessary condition for partial or complete recovery after lesion is behavioral training within the lesioned portion of the visual field. It remains to be determined what type of visual stimuli and behavioral tasks are most effective in inducing functional recovery after extrastriate cortical damage.

**Notes**

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