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The role of the lateral suprasylvian visual cortex of the cat in object-background interactions: Permanent deficits following lesions

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Abstract. The contribution of the lateral suprasylvian cortex to pattern recognition was studied by behavioural detection experiments in combination with bilateral lesions of different parts of the lateral suprasylvian areas (LSA) and area 7 in seven cats. In a two-alternatives forced choice task the cats had to discriminate simple outline patterns which were additively superimposed on a structured visual background made up of broadband Gaussian noise. For various stimulus conditions (moving or stationary patterns and/or background) the detection probability (P_D) of the cats was measured as a function of the signal-to-noise ratio (S/N). Each cat was tested before and after the lesion. Four different types of lesion could be distinguished depending on their extent: (1) lesion of parts of the (LSA); (2) lesion of parts of the LSA with undercutting of areas 17, 18 and 19; (3) lesion of area 7; (4) lesion of area 7 and parts of the LSA.

1. We found that a large bilateral lesion of the LSA led to significant deficits in all test situations which were dependent on the existence of relative velocity of moving patterns against a structured background. The ability of the cats to discriminate simple outline patterns which were kept stationary was not reduced. On the contrary, when they were tested with stationary and moving patterns on unfocused (empty) backgrounds, we found, to our great surprise, that the performance of the lesioned cats was significantly improved compared with intact animals. As these lesioned cats had no deficits with moving patterns on a uniformly grey background, we conclude that the deficits with the moving patterns must have been caused by interactions between patterns and background, and not by movement of a pattern per se.
2. As soon as the lesion of the LSA was extended by a bilateral undercutting of areas 17, 18 and 19 we found very severe deficits in all test situations, regardless of whether the patterns were moving or kept stationary, or whether they were superimposed on a background or not. The most substantial deficits occurred when the patterns were moving on a stationary background. In these situations the cats were no longer able to reach the 84% correct criterion. Again, the cats were able to reach criterion with moving patterns on a uniformly grey background indicating that this deficit is probably caused by the interaction of patterns and background and not by motion of the patterns per se.
3. A large lesion of area 7 led to modest but significant deficits of more or less the same degree in all test situations with the exception of quickly moving patterns on a structured background. In contrast, a much smaller lesion of area 7 yielded significant deficits only when the background was moved and there was a low relative velocity between the patterns and the background.
4. In brief, the combination of a lesion of area 7 with that of the LSA roughly provoked a combination of the effects of the two lesions (1 and 3) alone. We found significant deficits in all test situations.

We did not find evidence for any type of functional recovery in any of the lesions described. All deficits were permanent. Our results support the idea of a functional segregation between the LSA and area 7. They confirm that the LSA are involved in pattern recognition whenever it is associated with motion in combination with object-background interactions. This suggests an involvement of the LSA in the analysis of object- and self-induced motion. An interpretation of the results of lesioning area 7 is based on the conjecture that the mechanism of vergence movements or of binocular fusion of both retina images might be impaired. Under this condition it seems conceivable to expect double images or a reduced visual acuity because the images are out of focus. This would have a similar effect on the detection performance in each stimulus configuration. In addition, the results of lesion 4 provide evidence that both functional subunits (LSA and area 7) are not able to compensate for one another. Finally, we conclude from the results after lesion 2 that the

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LSA is part of a system which is based on the cooperation with striate cortex and requires intact primary cortex for its full function.

Key words: Detection performance – Visual noise – Pattern recognition – Behavioural experiments – Lesion of area 7 – Cat

Introduction

The lateral suprasylvian cortex, which comprises the region along the suprasylvian gyrus and the region on the lateral suprasylvian sulcus, represents a large part of the cat visual system, and, in contrast to visual areas 17, 18 and 19, is mainly characterized by extrageniculate inputs (Sprague et al. 1977; Rosenquist 1985). Based upon its retinotopic organization, Palmer et al. (1978) subdivided the region of the suprasylvian sulcus into six different areas: anterior medial lateral suprasylvian (AMLS), anterior lateral LS (ALLS), posterior MLS (PMLS), posterior lateral LS (PLLS), dorsal LS (DLS), ventral LS (VLS); and we will use their nomenclature. Since the region on the middle suprasylvian gyrus of the cat is believed to be homologous to the monkey's inferior parietal lobe (IPL), the area generally agreed to be important in directing the monkey's visual attention (see review by Hyvärinen 1982), we follow the nomenclature used by Hassler and Muhs-Clement (1964) and Heath and Jones (1971) and call it area 7. The same region is sometimes called the middle suprasylvian area (MS; Yin and Greenwood 1992ab) or anterior portion of the middle suprasylvian gyrus (AMSS; Dow and Dubner 1969, 1971)

While the lateral suprasylvian visual areas (LSA) have been studied rather intensively in the last decade (see review of Spear 1991), there are only a few reports on electrophysiological studies in area 7 (Dow and Dubner 1969, 1971; Straschill and Schick 1974; Robertson et al. 1975; Joseph and Giroud 1986; Yin and Greenwood 1992ab).

Based upon electrophysiological data, the most likely function of the LSA is that it is important for processing information about image motion. This includes an involvement in the estimation of object movement, in processing figure/ground motion (von Grünau and Frost 1983; Dinse and Krüger 1987; Krüger and Dinse 1988), optical flow-fields for self-motion (Rauschecker et al. 1987a; von Grünau et al. 1987), movement in three-dimensional space (Toyama and Kozasa 1982; Toyama et al. 1985), optokinetic nystagmus (Hamada et al. 1988; Herdman et al. 1989), accommodation and the near response (Bando et al. 1984, Bando 1985), in the regulation and direction of visual attention (Rizzolatti and Camarda 1975, 1977) and visuomotor integration of saccadic eye movements (Kennedy and Magnin 1977; Vanni-Mercier and Magnin 1982, Komatsu et al. 1983; Yin and Greenwood 1992b).

Unfortunately, there have been few behavioural tests of these hypotheses and the understanding of the behavioural functions of the LSA is still only rudimentary. So far, cortical lesions combined with the available behavioural studies suggest that the LSA cortex is not important for cats to discriminate simple patterns and forms (Wood et al. 1974; Sprague et al. 1977; Spear et al. 1983) or for visual localization and attention (Spear et al. 1983; Hardy and Stein 1988). In contrast, the involvement of the LSA in learning and interhemispheric transfer (Berlucchi et al. 1979; Ptito and Leporé 1983), in approach behaviours (Hardy and Stein 1988) and in the generation of the optokinetic nystagmus (Ventre 1985a, Tusa et al. 1989) has been discussed. The results of Pasternak et al. (1989) indicate an involvement in processing velocity and/or temporal frequency information. Other motion-processing functions have not been tested directly in behavioural experiments.

Our knowledge about the behavioural function of area 7 is much more rudimentary. From electrophysiological data we know that a high percentage of cells respond to moving visual stimuli and that many cells are polymodal (Dubner and Brown 1968; Robertson et al. 1975) and discharge in synchrony with saccadic eye movements (Straschill and Schick 1974; Joseph and Giroud 1986; Komatsu et al. 1983; Yin and Greenwood 1992b). These findings suggest area 7 is involved in the construction of the behavioural space and its maintenance during movements, however, this function has not been tested directly in behavioural experiments. There are very few behavioural experiments involving a lesion of area 7. Fabre and Buser (1980, 1981) tested the involvement of area 7 in the visually guided forelimb movement and found a deficit only when it was lesioned together with area 5. Unilateral lesions of area 7, encroaching on some surrounding visual areas, produced an asymmetric vestibulo-ocular nystagmus (Ventre 1985b). In both cases, the deficits were compensated within a few weeks or months. Preliminary results indicate an involvement of area 7 in depth perception of the cat. Krüger et al. (1990, 1993) found permanent deficits in a binocular but not a monocular depth discrimination task following a lesion of area 7, indicating that the ability to use binocular depth cues such as retinal disparity or vergence angle was impaired.

In this paper we present the results of an analysis of the behavioural function of the LSA and area 7 in the cat using detection experiments in combination with object and background motion before and after cortical lesions. In a visual detection experiment, an animal first learns to discriminate two visual patterns (signals) without background, and then is required to recognize the same patterns disturbed by a background of Gaussian visual noise additively superimposed. So far, the performance of normal cats and those lacking areas 17 and 18 (Krüger et al. 1986), areas 17, 18 and 19 (Krüger et al. 1988) and area 19 (Dinse and Krüger 1990) have been analysed. Moreover, we described preliminary results in two cats with lesions of the lateral suprasylvian cortex (Kiefer et al. 1989). These cats showed mild deficits when patterns and background were kept stationary and a severe impairment in performance when patterns were moving on a stationary background. Movement of the background relative to a stationary pattern resulted in an intermediate deficit. Al-

though this was a first behavioural indication for an involvement of the LSA in the discrimination of moving patterns and the analysis of dynamic scenes, many questions remained unclear. One of these questions was which parts of the cortical lesion, which included part of the LSA, part of area 7, and a partial undercutting of areas 17, 18 and 19, were responsible for the behavioural deficits? Therefore, we started a new series of detection experiments in combination with lesions of different parts of the lateral suprasylvian cortex. This article is the fifth in a series of papers designed to explore pattern recognition after ablation of different visual areas and presents the results of six cats with different lesions of the LSA and area 7 together with the results of the experiments with the cat named Cäsar. Some of the results with Cäsar have already been described (Kiefer et al. 1989).

Materials and methods

This study is based on results obtained from two female (Sara, Cassy) and five male (Donald, Felix, Gandhi, Lancelot, Cäsar) adult domestic cats, which were tested before and after lesion of different parts of the LSA and area 7.

Stimuli and test procedure

The methodological approach has been described in detail elsewhere (Krüger et al. 1986). Briefly, the cats were trained and tested binocularly with a two-alternatives forced choice between two bright outline patterns, a triangle and a circle, with equal area. The outer base of the triangle measured 26 mm, the outer diameter of the circle measured 20 mm and the width of the outlines was 2.7 mm. The two patterns were projected onto small doors (13 × 10 cm) in the front of the test box and additively superimposed on a Gaussian broadband noise, which was projected from a second projector. The noise pattern had uniform spectral power density and included spatial frequencies from 0.002 to 0.27 cycles/deg, which covered the entire frequency range of the test patterns as determined from their Fourier transforms. The ratio of the standard deviation to the mean intensity of the noise (σ/m) was 0.314. The intensity of the test patterns was altered by neutral density filters in a range from 25 to 900 lx, while the noise was kept constant at values around 90 lx. A continuous set of signal-to-noise (S/N) ratios was thus produced by applying the formula

$$\frac{S}{N} = \frac{\text{intensity of signal (lx)}}{\text{intensity of noise (lx)} \times 0.314}$$

The cats were food deprived for 12 h, with water continuously available. They were motivated by food rewards, which they received in the test box only when they had opened the door onto which the correct pattern (triangle) was projected. If they chose incorrectly they were not allowed to correct, found the door locked, and received no food. Each cat was trained and tested in four daily sessions, each consisting of 36 pattern presentations, 6 days/week. Within each session the left/right position of the triangle was changed randomly. In each of the four daily sessions within one task one different S/N value was given in a sequence that made sure that the same S/N ratio was once tested in the 1st, 2nd, 3rd and 4th session each day. In order to get the theoretical Gaussian amplitude density distribution of each point of the pattern, masking should be obtained with different noise specimen or by shifting the same noise specimen with respect to the pattern. We achieved the same effect by a shift of the patterns with respect to the noise for each presentation. The intertrial interval was 10–15 s. During this time a door covered the screens, the patterns were changed and the empty food dish behind the screens was replenished. Olfactory and auditory cues were controlled by filling both dishes with food and by changing the slides and moving the lock in the same sequence before each trial. The cats could move about freely in the test box and appeared to make their choice at a mean viewing distance of 10 cm from the screen. A wooden wedge at the bottom of the test box in front of the screens prevented a closer approach. Though this distance was within the nearpoint of accommodation and convergence of the cat, it seemed improbable to us that the cats picked out a distance which made it more difficult for them to make a correct choice and to get the food rewards. Our aim was to measure differences rather than absolute values. Thus, even if this close distance had an effect on the cat's detection performance, this effect would have been the same for each of the different parameters and for the preoperative performance as well as for the postoperative one. In the dynamic conditions, patterns or noise were moved horizontally 7 cm back and forth across the doors with the aid of a galvanometric mirror system controlled by a triangular signal from a wave-generator. Depending on their velocity, the direction the patterns moved in was reversed at most twice before the cats made their decision.

The training phase lasted 4–5 weeks (1200–1300 pattern presentations) and was started with a triangle-versus-blank discrimination (contrast 375 : 1). After reaching the 90% correct criterion in three consecutive sessions, the training was continued with a triangle-versus-circle discrimination (contrast 375 : 1) without background, and finally with a triangle-versus-circle discrimination with additively superimposed background (S/N = 13). After the training, a phase of pre-testing followed to familiarize the animals with the experimental situation, in which they had to discriminate the same stimuli but with lower S/N ratios. When the cats had reached this training level, they were able to transfer to moving patterns or moving background without additional training.

In the preoperative tests the cats were tested binocularly in the following situations, which were run in the sequence A, B, C, G, D, E, F and H:

A. Patterns and background both kept stationary (all seven cats)
B. Patterns moved at 2 cm/s (12°/s), background kept stationary (all seven cats)
C. Patterns moved at 10 cm/s (60°/s), background kept stationary (all seven cats)
D. Patterns kept stationary, background moved at 10 cm/s (60°/s; cats Donald, Felix, Gandhi) or moved at 2 cm/s (12°/s; cats Cäsar, Cassy, Lancelot, Sara)
E. Patterns and background moved simultaneously in phase with one another, both at 10 cm/s (60°/s; cats Donald, Felix, Gandhi) or at 2 cm/s (12°/s; cats Cassy, Lancelot, Sara)
F. Patterns and background moved in anti-phase to one another, both moved at 5 cm/s (30°/s; cats Donald, Felix, Gandhi) or at 1 cm/s (6°/s; cats Cassy, Lancelot, Sara)

To decide whether the deficits after the lesions were related to the pattern movement themselves or to the interactions between patterns and background, the seven cats were also tested with stationary and moving patterns on an empty uniformly grey background made up of unfocused visual noise which was no longer perceived as background. For these test series the percentages of correct decisions were plotted as a function of the contrast between the patterns and the uniformly grey background.

G. Patterns stationary and superimposed on a uniformly grey background (all seven cats)
H. Patterns moved at 2 cm/s (12°/s) and superimposed on an uniformly grey background (but Cäsar tested only after the lesion, other cats tested both before and after)

Corresponding values in the degree of visual angle were calculated from a mean viewing distance of 10 cm during testing. In situations A and E there was no relative velocity between patterns and background, in situation B the relative velocity was 2 cm/s, in situation C it was 10 cm/s, in D and F, 2 cm/s (cats Cassy, Lancelot, Sara) or 10 cm/s (cats Donald, Felix, Gandhi), respectively.

Test I. Cats Donald, Felix and Gandhi were tested monocularly with an opaque soft contact lens (Titmus) closing the other eye in the test situations A (left and right eye tested before lesion, left eye tested after lesion), C (left eye tested before and after lesion), D (left eye tested before and after lesion), E (right eye tested before and after lesion), F (right eye tested before and after lesion) and H (right eye tested before lesion).

After finishing the preoperative program, control tests were resumed in each task to ensure that the preoperative data represented the final performance level in each case. If there was an improvement compared with the first runs of the task, this second run was expanded to obtain a new detection curve.

Detection curves

The detection performance was described with a detection curve, in which the percentage of correct decisions within a series of pattern presentations was plotted as a function of the S/N ratio. For each of the 4–8 S/N values on a curve, a minimum of 144 measurements per point were carried out in not less than four sessions (36 presentations in one session). We expected the detection curves to be theoretically describable by error functions (Krone et al. 1983) whose slopes and positions were determined by two free parameters:

$P_D(S/N) = \mathrm{erf}[(S/N\text{-}m)/\sigma]$
$P_D(m) = 0.5$
$P_D(m+\sigma) = 0.841$
$\sigma = 1/\text{slope} = \text{standard deviation of the curve}$

where P_D is the detection probability.

The slope of the error function was estimated for each session. To determine the significance of the deficits, the samples of the preoperative and postoperative slope values were subjected to a nonparametric analysis of variance using the Kruskal-Wallis test (Larsen and Marx 1986).

Different lesions led to deficits of different degrees. As a measure for the extent of a deficit we took the shifts in the positions of the preoperatively and the postoperatively measured detection curves. The point of reference of each curve was the S/N value at a P_D of 84.1% of correct decisions as determined by a least square fit of the data.

Surgery

After completion of the preoperative test program, different parts of the lateral suprasylvian visual cortex were lesioned by subpial aspiration of the grey matter by Prof. G. Berlucchi (University of Verona), with the cats under pentobarbitone sodium anaesthesia (35 mg/kg), using routine sterile precautions (method described by Sprague et al. 1977). Following aspiration, the defect was covered with gelatin film (Gelfilm). We intended to damage the LSA in cats Cäsar, Donald, Felix and Gandhi, area 7 in cats Lancelot and Sara, and the LSA together with area 7 in cat Cassy.

Postoperative testing

Since all cats recovered rapidly and without complications, it was possible to resume behavioural testing within 14 days after the operation. After the operation, cats Felix, Donald and Gandhi were tested in the sequence A, C, D, E, F, I, B, G and H, while cats Sara, Lancelot and Cassy were tested in the sequence A, B, C, E, F, D, G, H. After finishing the postoperative test program, control tests were resumed in each task to find out whether the deficits were constant or the cats showed an improvement of the performance.

Histology

Six to twelve months after the operation, the cats were given a lethal dose of pentobarbitone sodium and were perfused intracardially with 4% formaldehyde. The brains were drawn, removed for histological verification of the lesions, embedded in paraffin and cut coronally at 12.5 μ m. The extent of each cortical lesion was reconstructed from projection drawings of selected coronal Nissl-stained sections through the cortex and the thalamus and extrapolated using surface landmarks and cortical maps as described by Reinoso-Suarez (1961), Otsuka and Hassler (1962), Niimi and Sprague (1970), Sanderson (1971), Palmer et al. (1978) and Tusa et al. (1981). Retrograde degeneration in the thalamus was studied at a magnification which allowed identification of surviving large- and medium-sized (but not small) cell bodies, which were plotted. For details see Sprague et al. (1977).

Results

Reconstruction of the lesions

The reconstruction of the cortical lesion of cat Cäsar has already been illustrated and described in Kiefer et al. (1989). The location and extent of the lesion in each of the six other cats is reconstructed and plotted in a surface view of the hemispheres and selected coronal sections through the cortices in Figs. 1 – 5. Since the lesions differed in extent and location, we decided to illustrate all six lesions in detail. The estimates of the visual coordinates of the spared tissue were extrapolated using the maps prepared by Tusa et al. (1981) and Palmer et al. (1978). The retinotopic limits of degeneration in the different laminae of the lateral geniculate nucleus (LGN) were approximated by using the physiological maps of Sanderson (1977) and are shown for cats Donald (Fig. 1), Felix (Fig. 2) and Gandhi (Fig. 3). Figure 6 summarizes the extent of the damage of each area that was involved for the left and right hemisphere of each cat. An evaluation of the destroyed tissue was related to the extent of the intact area according to the maps of Tusa et al. (1981) and Palmer et al. (1978). Quantification of the undercutting was related to its antero-posterior extent.

Cat Donald. The most complete lesion of areas PMLS, PLLS and DLS was that in cat Donald. The lesion extended from A12.5 in both hemispheres to about P5 on the right and P2.8 on the left side (Fig. 1). The damage to the cortical tissue started at the top of the lateral bank of the suprasylvian sulcus, leaving areas AMLS and ALLS nearly intact, and extended from anterior to posterior over both banks of the middle and posterior suprasylvian sulcus to the fundus of the sulcus, damaging most parts of areas PMLS and PLLS and even undercutting partially areas 17, 18 and 19. Some islands at the fundus of the right suprasylvian sulcus were spared, possibly leaving intact parts of the area centralis representation in areas PMLS and PLLS. The undercutting of areas 17, 18 and 19 was only slight on the right hemisphere, extending antero-posteriorly from A1 to P0.4, which corresponds to the representation of the lower visual field near the horizontal meridian, and very extensive on the left side, extending from A7.4 to A0.4, which corresponds to the

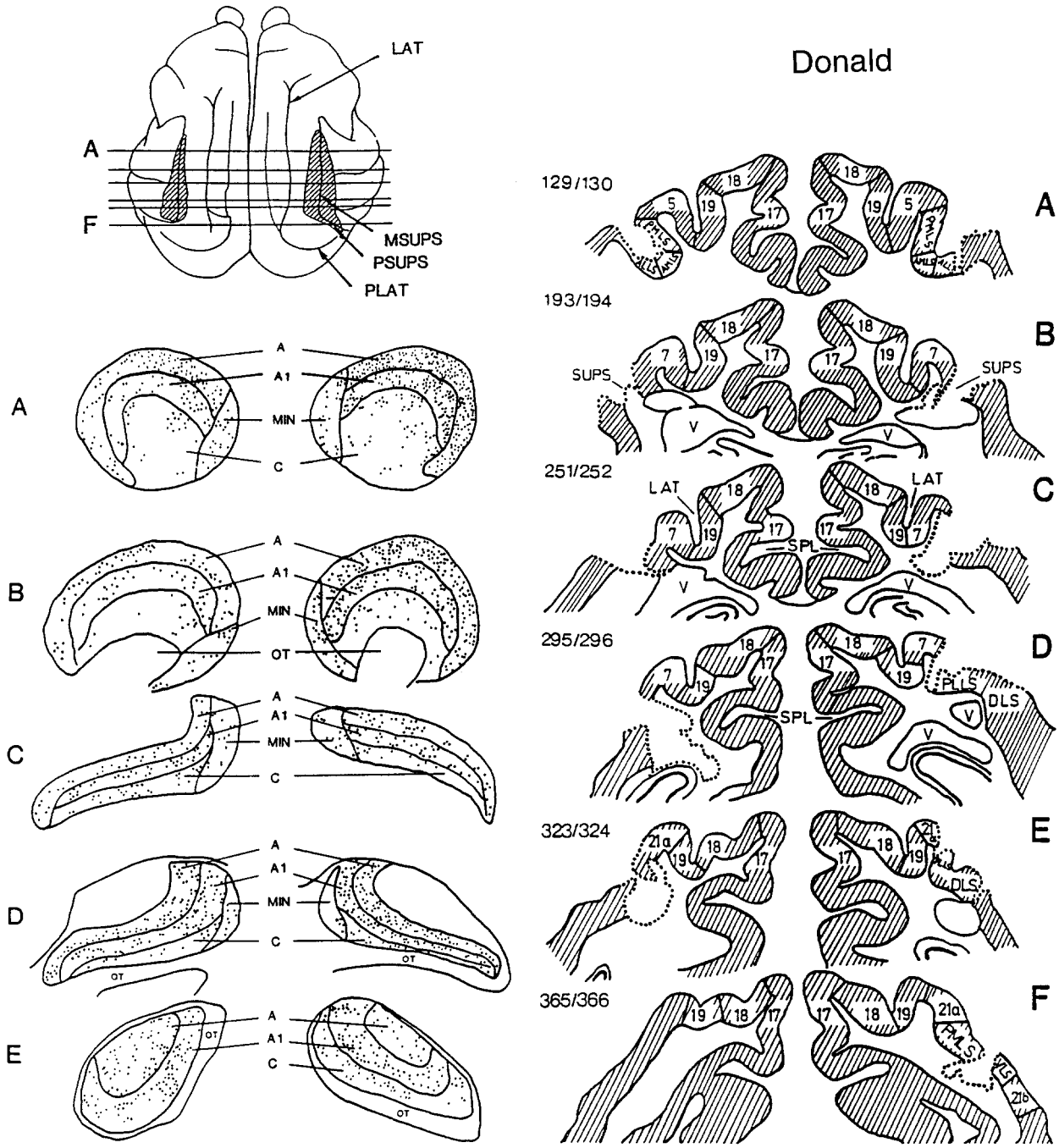

Fig. 1. Reconstruction of the cortical lesion in cat Donald. The surface view of the cortical lesion was reconstructed by photographs and microscopic examination of coronal sections. *Lettered lines A-F*, levels at which selected coronal sections are drawn to show the details of the lesions in cortex and white matter (*right side*) and the dorsal lateral geniculate complex (LGN_d; *left side*). Cyto- and myeloarchitectural areas are indicated by *numerals*. *Dotted lines*, removal of pia mater. Retinotopic coordinates are extrapolated from Tusa et al. (1981) and Palmer et al. (1978). The position of surviving cell bodies of medium and large cells in laminae A, A1, C of the LGN_d and the medial interlaminar nucleus (*MIN*) are shown as *dots*. *AMLS*, anterior medial lateral suprasylvian area; *ALLS*, anterior lateral lateral suprasylvian area; *DLS*, dorsal lateral suprasylvian area; *LAT*, lateral sulcus; *MSUPS*, middle suprasylvian sulcus; *OT*, optic tract; *PMLS*, posterior medial lateral suprasylvian area; *PLAT*, postlateral sulcus; *PLLS*, posterior lateral lateral suprasylvian area; *PSUPS*, posterior suprasylvian sulcus; *SPL*, splenial sulcus; *SUPS*, suprasylvian sulcus; *V*, ventricel; *VLS*, ventral lateral suprasylvian area

representation of more peripheral parts of the lower visual field.

The lesions in areas PMLS, PLLS and DLS were reflected by a severe atrophy and marked cell loss in lamina C of the LGN_d and the medial interlaminar nucleus (MIN) in both hemispheres. The undercutting of areas 17, 18 and 19 was reflected by a severe atrophy and marked cell loss in laminae A and A1 of the anterior part of the LGN_d in the left hemisphere, and of the middle part of the LGN_d in the right hemisphere.

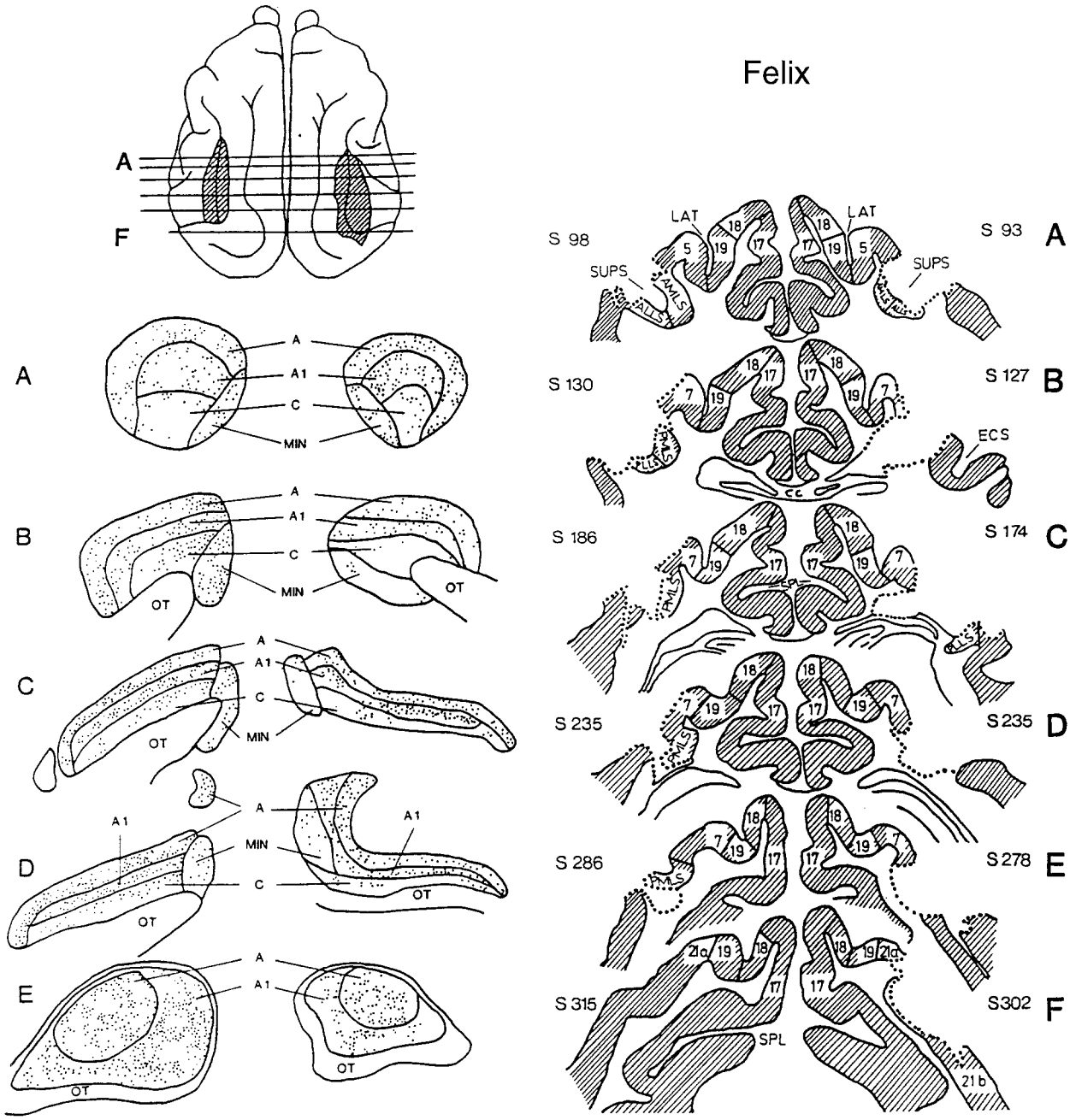

Fig. 2. Reconstruction of the cortical lesion in cat Felix. *Symbols and abbreviations* as in Fig. 1

Altogether the lesion destroyed about 65% of PMLS, 80% of PLLS and 90% of DLS, 5% of area 7 and 15% of area 21a on the right hemisphere, and about 10% of area AMLS, 15% of ALLS, 80% of PMLS, 90% of PLLS and DLS, respectively, and about 10% of 21a in the left hemisphere (Fig. 6).

Cat Felix. In cat Felix the damage to the left and the right hemisphere was rather different. The lesion started from around A11 in both hemispheres and extended to P4 on the right side and P1.5 on the left side (Fig. 2). The lesion in the right hemisphere was much more complete than that in the left one. It started at the top of the lateral bank of the suprasylvian sulcus, extended posteriorly into the fundus of the sulcus, included the lateral bank of the sulcus and led to an undercutting of areas 17, 18 and 19 extending from A8 to A4.5, which corresponds to the representation of peripheral parts of the lower visual field. In the anterior part of the left hemisphere however, the lesion spared islands in the fundus of the suprasylvian sulcus (Fig. 2, coronal sections A, B), which represent parts of the central visual field of the PMLS and the PLLS. The extent of the undercutting of areas 17, 18 and 19 was comparable with that on the right hemisphere, but was more posterior, reaching from A4.5 to A1.7, which corresponds to more horizontal parts of the lower visual field.

The lesion of areas AMLS, ALLS, PMLS and PLLS

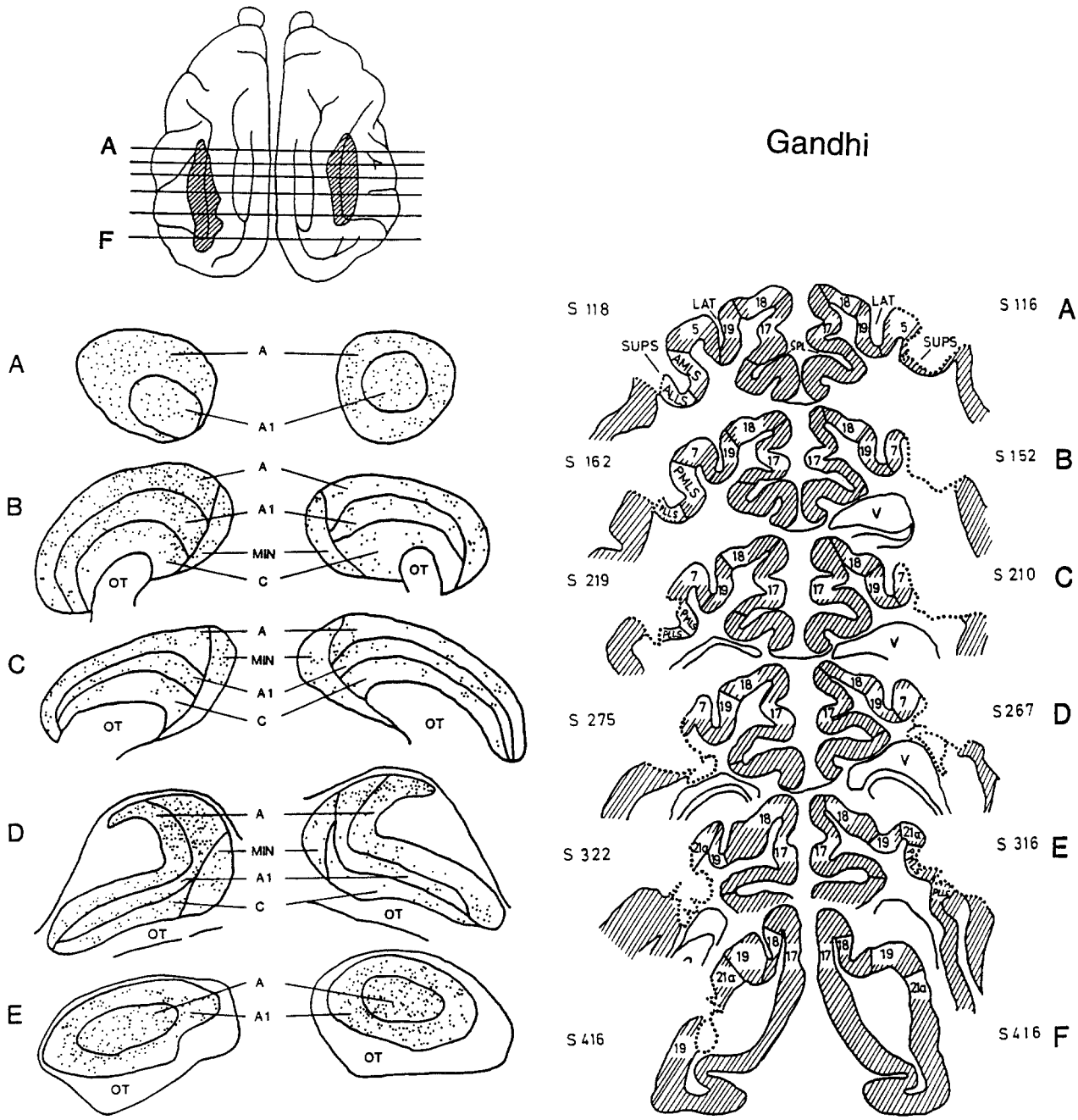

Fig. 3. Reconstruction of the cortical lesion in cat Gandhi. *Symbols and abbreviations* as in Fig. 1

of the right hemisphere was reflected by a severe atrophy and marked cell loss in lamina C throughout all parts of the LGN_d and the MIN. In the left hemisphere, this atrophy was much less severe, corresponding to the more incomplete lesion. The undercutting of the lower visual field representation of areas 17, 18 and 19 was reflected by a severe atrophy and marked cell loss in laminae A and A1 of the anterior parts of the LGN_d in both hemispheres (Fig. 2, sections A, B).

Altogether, in the left hemisphere, the lesion removed about 15% of area 5, 10% of area 7 and ALLS, 30% of PMLS and PLLS, 20% of DLS and about 10% of VLS, leaving the area centralis representation intact in all areas. In the right hemisphere, the lesion totally damaged areas ALLS and PLLS and destroyed about 20% of AMLS, 75% of PMLS including its area centralis representation, 20% of DLS, 10% of area 7 and less than 5% of area 5 (Fig. 6).

Cat Gandhi. The lesion in cat Gandhi was comparable with that of cat Donald concerning the damage to areas PMLS and PLLS. The main difference between the lesions in the both cats was the degree of bilateral undercutting of areas 17, 18 and 19. In cat Gandhi the undercutting extended from A9 to A0.5 on the right side and from A3.8 to P0.7 on the left one (Fig. 3), corresponding

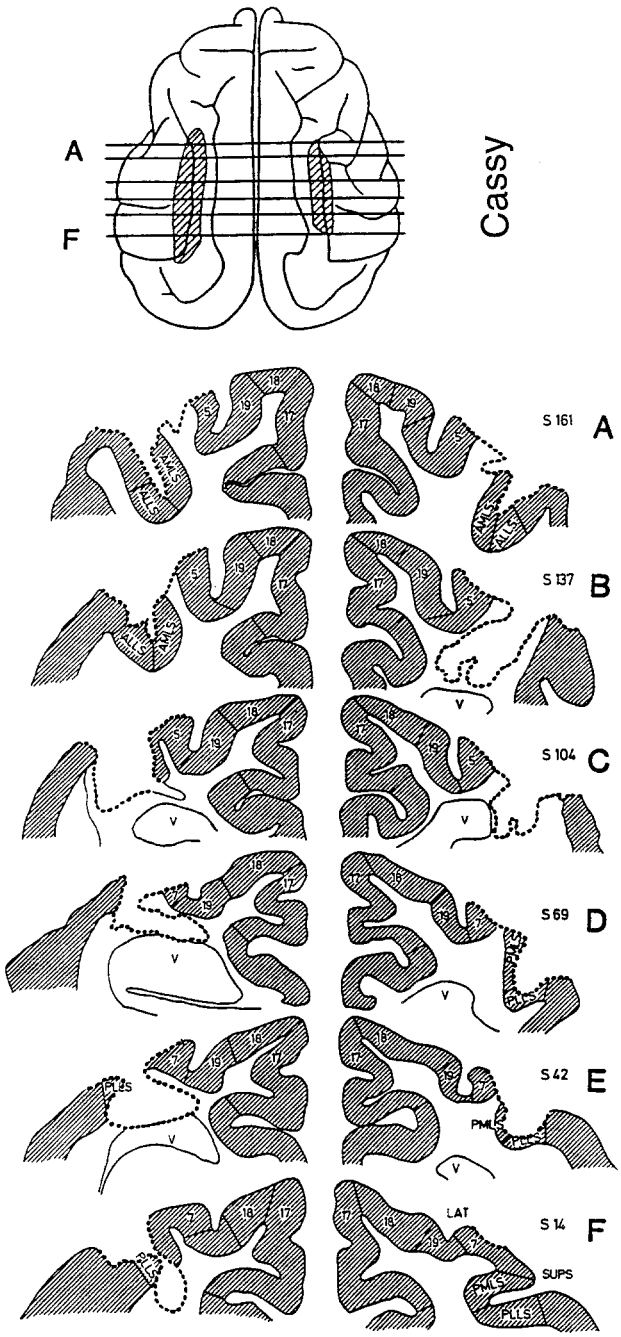

Fig. 4. Reconstruction of the cortical lesion in cat Cassy. *Symbols and abbreviations* as in Fig. 1

to representations of the lower visual field in both hemispheres.

The large lesion in areas PMLS and PLLS in both hemispheres was reflected by a severe atrophy and marked cell loss in lamina C throughout all parts of the LGN_d and the MIN. The more extended undercutting of areas 17, 18 and 19 in the right hemisphere, corresponding to a large part of the lower visual field representation, was reflected by a severe atrophy and marked cell loss in laminae A and A1 of the anterior and middle parts of the LGN_d. In the left hemisphere, the atrophy in laminae A and A1 was mainly restricted to the middle part of the LGN_d, corresponding to the undercutting of more horizontal parts of the lower visual field representation.

Altogether, the lesion removed roughly 25% of area 7, 12% of AMLS, 25% of ALLS, 80% of PMLS and 75% of PLLS on the right side, and 6% of ALLS, 80% of PMLS, 65% of PLLS, 13% of area 7 and 40% of area 21a on the left side (Fig. 6), including the entire central visual field representation of PMLS and PLLS on the left side and part of it on the right side.

Cat Cassy. Measuring the extent of the damage to cortical tissue, cat Cassy had the largest lesion, corresponding to the intention to combine a LSA-lesion and a lesion of area 7 in this cat. The lesion in her left hemisphere extended from A17.8 to P1.8, in her right hemisphere from A16.5 to A1.5 (Fig. 4). In both hemispheres, the lesion started at the crown of the suprasylvian gyrus, destroying about 50% of area 5 and 40% of area 7, and more posteriorly included the medial and lateral bank of the suprasylvian sulcus, corresponding to areas AMLS, ALLS, PMLS and PLLS. In the left hemisphere the lesion destroyed about 45% of AMLS and 40% of ALLS, including the central representation, and about 65% of PMLS and 60% of PLLS. In the right hemisphere, the damage to areas AMLS and ALLS started more anteriorly and destroyed about 85% of AMLS and 75% of ALLS, including area centralis representation, whereas the damage in PMLS and PLLS was restricted to the anterior parts of these areas, corresponding to about 45% of each area (Fig. 6) and leaving intact parts of the central representation.

An undercutting of areas 17, 18 and 19 occurred mainly in the left hemisphere from A6.3 to A2.5 (Fig. 4, sections D-E; about 3.8 mm), corresponding to peripheral parts of the lower visual field representation, whereas in the right hemisphere it extended from A10.5 to A9 (only 0.5 mm), representing the far periphery of the lower visual field.

The lesion of areas AMLS, ALLS, PMLS and PLLS of both hemispheres was reflected by a severe atrophy and marked cell loss in lamina C throughout all parts of the LGN_d and the MIN. The undercutting of areas 17, 18 and 19 of the left hemisphere was reflected by a severe atrophy and marked cell loss in laminae A and A1 of the anterior parts of the LGN_d; the undercutting in the right hemisphere was not reflected by atrophy or cell loss in the LGN_d.

Cat Lancelot. The most complete lesion of area 7 was that in cat Lancelot. The lesion in the left hemisphere extended from A13 to P6, in the right hemisphere it extended from A11.5 to P6. In both hemispheres the lesion completely included the crown of the suprasylvian gyrus (Fig. 5), extending to the top of both the lateral bank of the lateral sulcus and the medial bank of the suprasylvian sulcus, but leaving the banks of both sulci intact. Altogether, in the left hemisphere, about 25% of area 5 and 70% of area 7 and a small part (10%) of area 21a were destroyed. In the right hemisphere about 15% of area 5, 70% of area 7 and 40% of area 21a were damaged (Fig. 6). We found no undercutting of areas 17, 18 and 19,

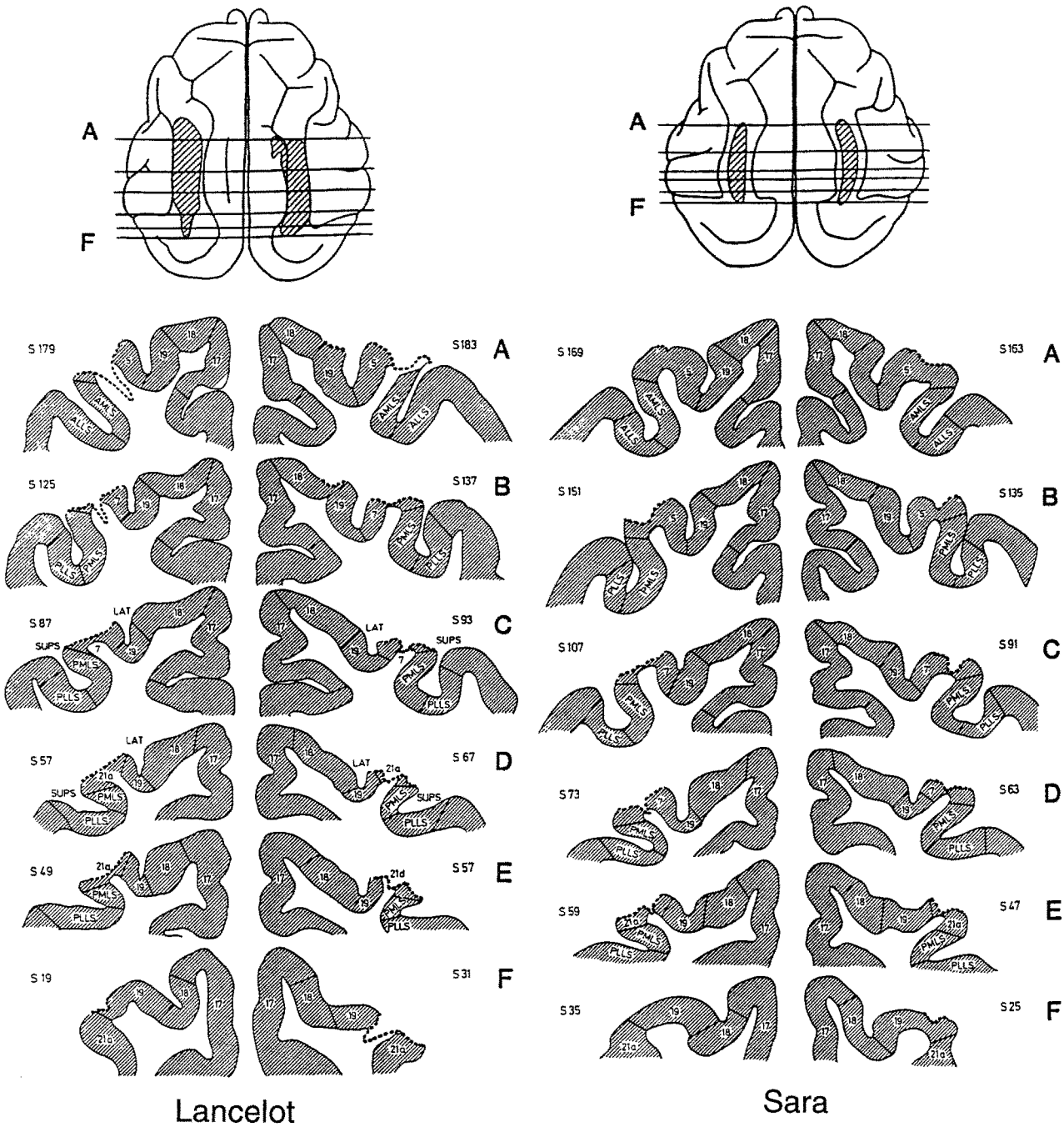

Fig. 5. Reconstruction of the cortical lesion in cats Lancelot (*left*) and Sara (*right*). *Symbols and abbreviations* as in Fig. 1

which was reflected by no atrophy or cell loss in the LGN_d.

Cat Sara. Cat Sara had the smallest lesion, much smaller than that of cat Lancelot. In the left hemisphere it extended from A10.3 to P3, and in the right, from A11.5 to P4.5 (Fig. 5). In contrast to the lesion of cat Lancelot the lesion destroyed only the medial part of the crown of suprasylvian gyrus. The lateral bank of the lateral sulcus and the medial bank of the suprasylvian sulcus remained completely intact and there was no undercutting of areas 17, 18 or 19. Altogether, in the left hemisphere around 10% of area 5, 35% of area 7 and 25% of area 21a were destroyed, in the right hemisphere about 10% of area 5, 25% of area 7 and 15% of area 21a were damaged (Fig. 6).

Cat Cäsar. A careful evaluation of the undercutting of areas 17, 18 and 19 in cat Cäsar (Kiefer et al. 1989) showed that it extended from A9.5 to A6.9 and from A 4.6 to A3 on the left side and from A9 to P1.8 on the right side, on both sides corresponding to the representation of parts of the lower visual field.

The large lesion of areas ALLS, PMLS, PLLS, DLS

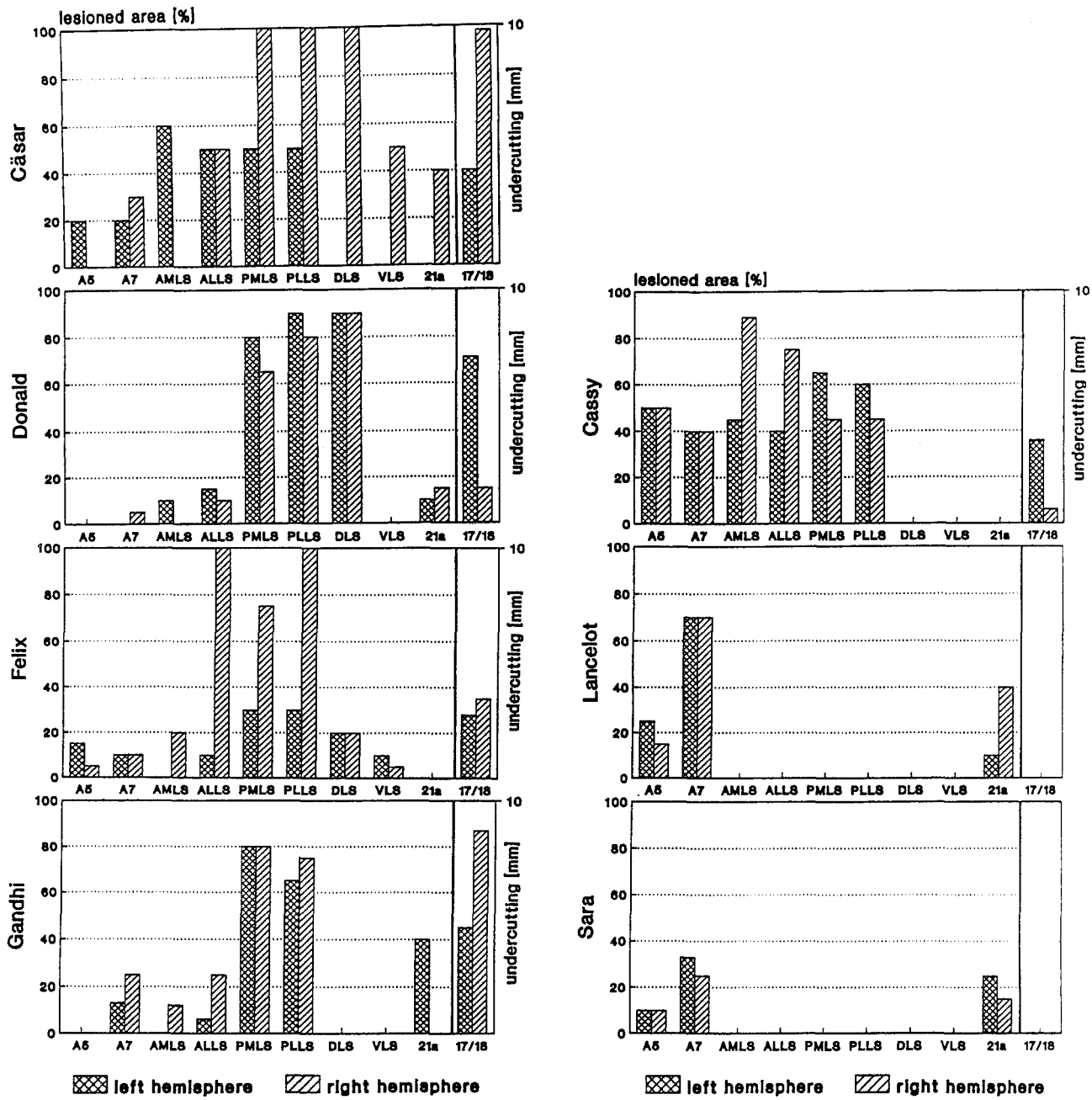

Fig. 6. Quantification of the extent of the damage of each area for the left and right hemisphere of each cat. Evaluation of the destroyed tissue was related to the extent of the intact area. Quantification of the undercutting of areas 17/18 is indicated in millimeters of the antero-posterior extent of the undercutting. *Abbreviations* as in Fig. 1

and VLS and the extended undercutting of areas 17, 18 and 19 on the right hemisphere were reflected by a severe atrophy and marked cell loss in laminae A, A1 and C throughout all parts of the LGN_d and the MIN. The less extended undercutting in the left hemisphere was reflected by a less severe atrophy in laminae A and A1 which was mainly restricted to the anterior part of the LGN_d, corresponding to the undercutting of more peripheral parts of the lower visual field representation. The smaller lesion of the LSA was reflected by a less severe atrophy in lamina C throughout all parts of the LGN_d and the MIN.

To summarize, the lesion in Cäsar destroyed roughly 20% of area 5 and area 7, respectively, 60% of AMLS, 50% of ALLS, PMLS and PLLS in the left hemisphere, leaving intact parts of the area centralis representation in the latter areas, and 30% of area 7, 50% of ALLS, 100% of areas PMLS, PLLS and DLS, 50% of VLS and about 40% of area 21a in the right hemisphere (Fig. 6).

Postoperative behaviour

During the first 3 days after the operation the cats appeared blind. It was difficult to gain their attention with an object moving in front of their eyes and for the search of food they appeared to rely primarily on olfaction. Af-

ter 3 days they were able to move around in their familiar housing without bumping into obstacles.

One week after the operation the cats started spontaneously jumping onto slightly elevated planes, but depth perception was still poor as they had difficulty reaching the floor again. This was especially striking in cat Lancelot who was able the reach his sleeping place at 180 cm height over a slanted board but was not able to leave this place without help.

Two weeks following the operation the cats were able to move freely in unknown surroundings, their visual placing was normal and they were again able to stop accurately a moving piece of food with their paw. The behaviour in the test box was unchanged concerning motivation and cooperativity compared with the preoperative observations, except for cat Gandhi, who was less cooperative after the operation. Three weeks after the operation the jumping behaviour towards lower planes was completely normal and the cats' behaviour could hardly be distinguished from their preoperative behaviour.

Detection performance of lesioned cats

The deficits we found in the earliest possible test sessions (2 weeks after the operation) were constant over the whole period of 4–6 months, with no indication of relearning or recovery. This was confirmed with control tests at the end of the postoperative test program. On the other hand, none of the cats – not even the cats with suprasylvian lesions undercutting areas 17, 18 and 19 – showed any retention deficits of the learned pattern discriminations.

Binocular tests. To demonstrate the degree of the deficit in each cat detection curves for all seven cats are shown in Fig. 7. Each test situation is shown for both the preoperative (continuous lines) and postoperative case (dashed lines). The significance level between the preoperative and postoperative set of data is given for each pair of curves only if the deficit was significant, i.e. if the significance level is less than 0.05. Additionally, the S/N value for a P_D of 84.1%, the shifts between pre- and postoperative detection curves, and the levels of significance for the deficits are listed in Table 1.

The preoperative (black squares) and postoperative (empty squares) curves for stationary (G) and moving patterns (H) on an uniformly grey background made up of unfocused visual noise are shown in Fig. 8. Again, the significance level between the preoperative and postoperative set of data is given for each pair of curves only if it was significant. Table 2 shows the corresponding contrast values for a P_D of 84.1% positive responses, the shifts between the preoperatively and the postoperatively measured curves, and the levels of significance of the deficits.

According to the different extent and location of the cortical lesions in the seven cats described above, their deficits in the behavioural tests were very different. *Donald*, the cat with the most complete bilateral lesion of areas PMLS, PLLS and DLS, showed small (Table 1, S/N shifts of 0.7–1.4) but significant deficits in only three test situations: when patterns were quickly (10 cm/s) or slowly (2 cm/s) moving on a stationary background and when patterns and background were moving in anti-phase to one another at 5 cm/s (Fig. 7B,C,F), the last case creating the largest deficit. However, moving the patterns on a uniformly grey background led to no deficit (Fig. 8B). On the contrary, in both test series with uniformly grey background (Fig. 8), this cat showed a better performance when it was lesioned than as an intact animal. The shift of the postoperative detection curve into the lower S/N range was significant for the moving patterns.

Felix, a cat with a much smaller bilateral lesion of areas PMLS, PLLS and DLS than cat Donald, showed only one significant and very small (Table 1, S/N shift 0.5) deficit: when patterns were quickly moving at 10 cm/s on a stationary background (Fig. 7C). In all other test situations the cat had no deficits (Fig. 7, Table 1) and, like Donald, in both test situations with uniformly grey background it postoperatively had better performance than as an intact animal (Fig. 8, Table 2). In this cat the shift of the curves with the stationary patterns was significant.

Cat *Gandhi*, who had a lesion comparable with that of Donald, but with an additional bilateral undercutting of areas 17, 18 and 19, showed large and significant deficits in all test situations (Figs. 7,8, Tables 1,2). The deficits were very severe and the cat was not able to reach the 84% correct level when the patterns were moving no matter if the background was either kept stationary (Fig. 7B,C), or was moving simultaneously in phase (Fig. 7E) or in anti-phase to the patterns (Fig. 7F). Gandhi reached the 84% correct level only with stationary patterns on a stationary (Fig. 7A) or moving background (Fig. 7D) and in the test series with a uniformly grey background (Fig. 8). In these test situations the deficits were large in comparison with the deficits of the other cats (Table 1, S/N shift 9.5–9.6; Table 2, contrast shift 2.3–3.3).

Cat *Cäsar*, who had not been tested with patterns and background moving simultaneously in phase (E) or moving in anti-phase to one another (F) showed significant deficits in all other test situations. The detection curves of this cat, though already shown in Kiefer et al. (1989), are again shown in Figs. 7 and 8 and Tables 1 and 2. We were not able to determine the shift in the pre- and postoperative detection curves for quickly (Fig. 7C) and slowly (Fig. 7B) moving patterns because in these situations the cat was not able to reach the 84% correct level. The smallest deficit occurred when patterns and background were both kept stationary (Fig. 7A, S/N shift 3.3). For stationary patterns on a quickly moving (10 cm/s) background the deficit was intermediate (Fig. 7D, S/N shift 8.2). In addition, Cäsar showed a significant deficit with stationary patterns on a uniformly grey background (Fig. 8A, Table 2). The test series with moving patterns on a uniformly grey background was carried out only with the lesioned animal and is shown in Fig. 8B.

Cat *Cassy*, with a much larger lesion than Donald, Felix, Lancelot and Sara, consistently showed significant deficits in all test situations. All the deficits were larger (Table 1, S/N shifts 1.3–5.0) than the deficits of the other

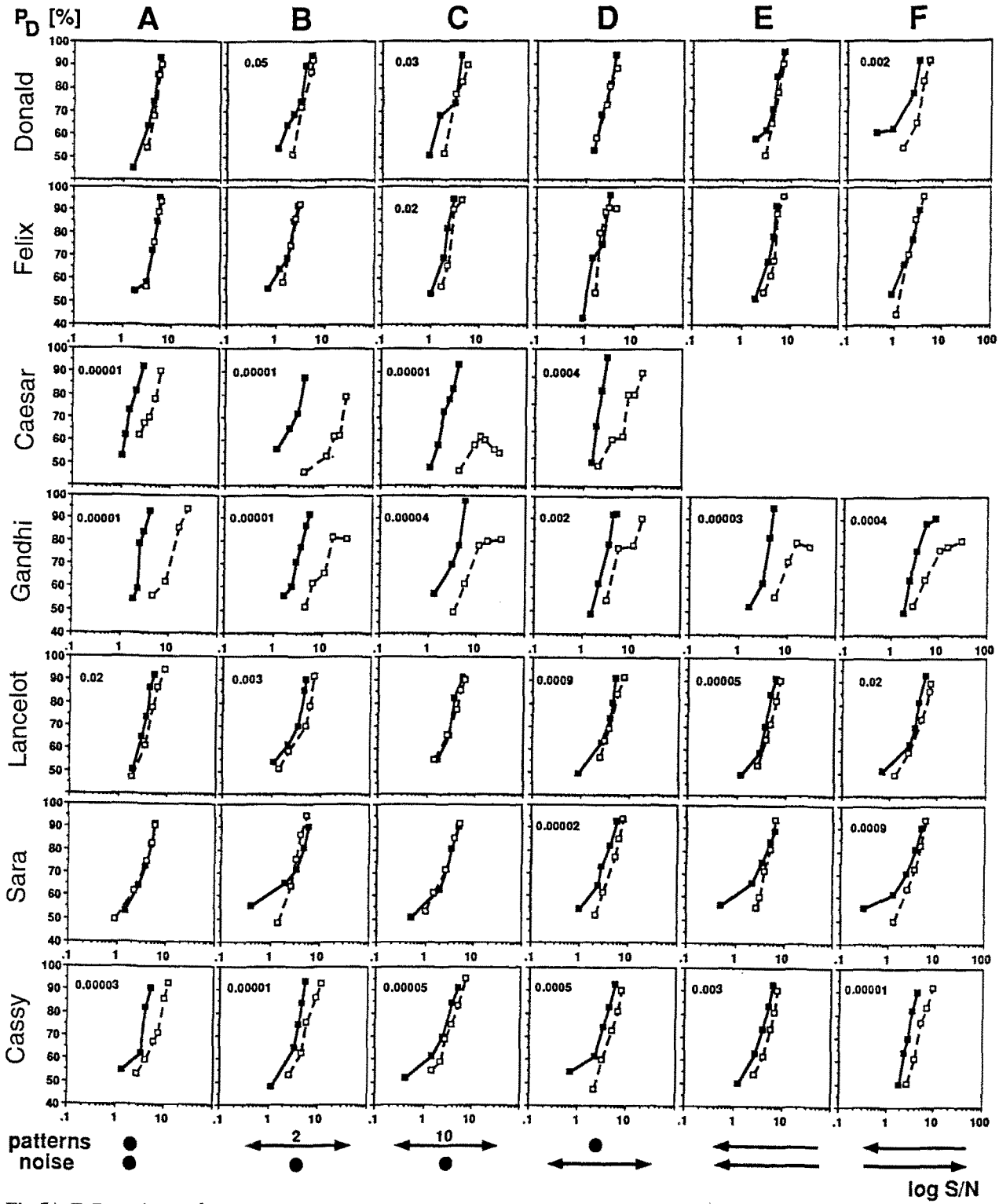

Fig. 7A–F. Detection performance of seven cats before (*continuous lines*) and after (*dashed lines*) the lesion. The cats were tested with small patterns superimposed on broadband noise. Patterns and noise were kept stationary (**A**), the patterns were moved relative to the noise at 2 cm/s (**B**) and 10 cm/s (**C**) while the noise was kept stationary, or the noise was moved relative to the patterns at 2 cm/s (Cassy, Lancelot, Sara) or at 10 cm/s (Donald, Felix, Gandhi) while the patterns were kept stationary (**D**), or patterns and noise were both moved at 2 cm/s (Cassy, Lancelot, Sara) or 10 cm/s (Donald, Felix, Gandhi) simultaneously in phase to one another (**E**), or patterns and noise were both moved at 1 cm°/s (Cassy, Lancelot, Sara) or 5 cm/s (Donald, Felix, Gandhi) in anti-phase to one another (**F**). Each point of the curve is the probability of a positive response P_D (%) computed over at least 144 trials subdivided in four sessions. The percentage of positive responses is plotted on the *ordinate* in a linear scale and the signal-to-noise ratio (S/N) on the *abscissa* in a logarithmic scale. The *numbers at the top* correspond to the significance level of the deficits with $P < 0.05$

Table 1. Test series performed with intact and lateral suprasylvian visual area-lesioned cats

Test	Cat	S/N at 84.1%		Shift	$P<$	Significant deficits
		Intact	Lesioned			
Patterns stationary, background stationary	Donald	5.0	5.4	0.4	0.06	
	Felix	4.6	4.9	0.3	0.09	
	Gandhi	3.0	12.6	9.6	0.00001	x
	Cäsar	2.2	5.5	3.3	0.00001	x
	Cassy	4.3	9.3	5.0	0.00003	x
	Lancelot	4.3	5.9	1.6	0.02	x
	Sara	5.1	5.2	0.1	0.4	
Patterns moved at 2 cm/s, background stationary	Donald	3.6	4.3	0.7	0.05	x
	Felix	2.3	2.4	0.1	0.2	
	Gandhi	4.1	n.r.	≫	0.00001	x
	Cäsar	3.7	n.r.	≫	0.00001	x
	Cassy	4.5	8.0	3.5	0.00001	x
	Lancelot	4.4	6.3	1.9	0.003	x
	Sara	5	3.8	−1.2	0.2	
Patterns moved at 10 cm/s, background stationary	Donald	3.6	4.6	1.0	0.03	x
	Felix	2.3	2.8	0.5	0.02	x
	Gandhi	4.5	n.r.	≫	0.00004	x
	Cäsar	3.0	n.r.	≫	0.00001	x
	Cassy	3.7	5.0	1.3	0.00005	x
	Lancelot	3.8	4.7	0.9	0.09	
	Sara	3.6	3.5	−0.1	0.5	
Patterns stationary, background moved at 2 or 10 cm/s	Donald	2.7	3.0	0.3	0.8	
	Felix	2.6	2.3	−0.3	0.3	
	Gandhi	3.6	13.1	9.5	0.002	x
	Cäsar	2.3	10.5	8.2	0.0004	x
	Cassy	4.5	6.9	2.4	0.0005	x
	Lancelot	4.5	5.6	1.1	0.0009	x
	Sara	4.5	6.1	1.6	0.00002	x
Patterns and background moved simultaneously in phase	Donald	5.0	6.0	1.0	0.1	
	Felix	4.7	5.0	0.4	0.09	
	Gandhi	4.2	n.r.	≫	0.00003	x
	Cäsar	–	–	–	–	–
	Cassy	4.9	6.9	2.0	0.003	x
	Lancelot	4.7	6.4	1.7	0.00005	x
	Sara	4.8	5.1	0.3	0.2	
Patterns and background moved in anti-phase to one another	Donald	2.8	4.2	1.4	0.002	x
	Felix	2.9	2.7	−0.2	0.9	
	Gandhi	4.5	n.r.	≫	0.0004	x
	Cäsar	–	–	–	–	–
	Cassy	3.5	7.0	3.5	0.00001	x
	Lancelot	4.4	6.4	2.0	0.002	x
	Sara	3.9	4.8	0.9	0.0009	x

Numbers correspond to the signal-to-noise (S/N) values at a detection probability of 84.1% of correct decision determined by a least square fit of the corresponding detection curves, to the shift in the position of the pre- to the postoperative detection curves and to the level of significance of the deficits ($P<$). Negative shift values mean that the postoperative detection performance was better than the preoperative one

≫, the lesioned cats were not able to reach (n.r.) the 84% correct level

cats (Fig. 7, Table 1). Interestingly, the largest deficit occurred (S/N shift of 5.0) when the patterns and background were kept stationary (Fig. 7A). The smallest deficits occurred when the patterns were quickly moved (10 cm/s) on a stationary background (Fig. 7C), slowly moved (2 cm/s) on a uniformly grey background (Fig. 8A, Table 2) or kept stationary on a uniformly grey background (Fig. 8B, Table 2).

Sara, the cat with the smallest cortical lesion (area 7), consistently had the smallest deficits. This cat showed significant deficits only in three test situations: (1) when the patterns were moving at 2 cm/s on a uniformly grey background (Fig. 8B, Table 2); (2) when the patterns were kept stationary and the background was moved at 2 cm/s (Fig. 7D); and (3) when patterns and background were moved at 1 cm/s in anti-phase to one another (Fig. 7F). These deficits were significant but small (Table 1, S/N shifts 0.1–1.6). Movement of the patterns on a stationary background led to no significant deficit (Fig. 7B,C). The largest deficit occurred when only the background was moved (Fig. 7D).

Cat *Lancelot*, who had much greater damage of the same cortical areas than cat Sara, showed small but significant deficits in all test situations (Figs. 7, 8) except

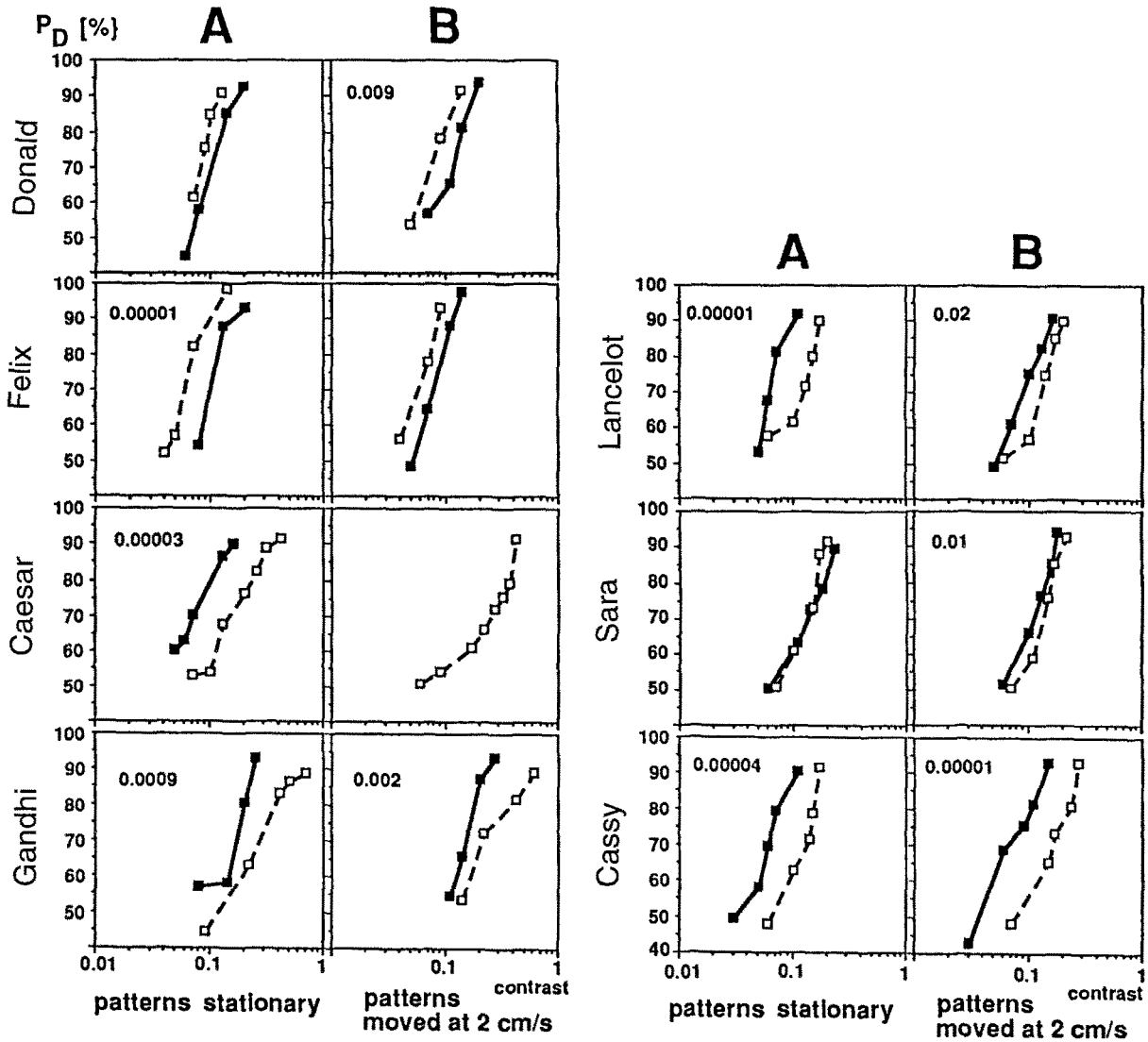

Fig. 8. Performance of seven cats before (*continuous lines*) and after the operation (*dashed lines*) tested with stationary patterns (**A**) and patterns moved at 2 cm/s (**B**) on a uniformly grey background made up of unfocused visual noise. The percentage of positive responses is plotted on the *ordinate* in a linear scale and the contrast between patterns and background on the *abscissa* in a logarithmic scale. The *numbers at the top* correspond to the significance level of the deficits with $P < 0.05$

Table 2. Test series with uniformly grey background performed with intact and lateral suprasylvian visual area-lesioned cats

Test	Cat	Contrast at 84.1%		Shift	$P<$	Significant deficits
		Intact	Lesioned			
Patterns stationary, background unfocused	Donald	1.4	1.0	−0.4	0.06	
	Felix	1.3	0.7	−0.6	0.00001	x
	Gandhi	2.1	4.4	2.3	0.0009	x
	Cäsar	1.3	2.9	1.6	0.00003	x
	Cassy	0.9	1.7	0.8	0.00004	x
	Lancelot	0.8	1.7	0.9	0.00001	x
	Sara	2.0	1.8	−0.2	0.8	
Patterns moved 2 cm/s, background unfocused	Donald	1.6	1.1	−0.5	0.0009	x
	Felix	1.0	0.8	−0.2	0.3	
	Gandhi	1.9	5.2	3.3	0.002	x
	Cäsar	–	4.0	–	–	
	Cassy	1.2	2.5	1.3	0.00001	x
	Lancelot	1.4	1.8	0.4	0.002	x
	Sara	1.5	1.8	0.3	0.01	x

Numbers correspond to the contrast values at a detection probability of 84.1% of correct decision determined by a least square fit of the corresponding detection curves. See notes to Table 1

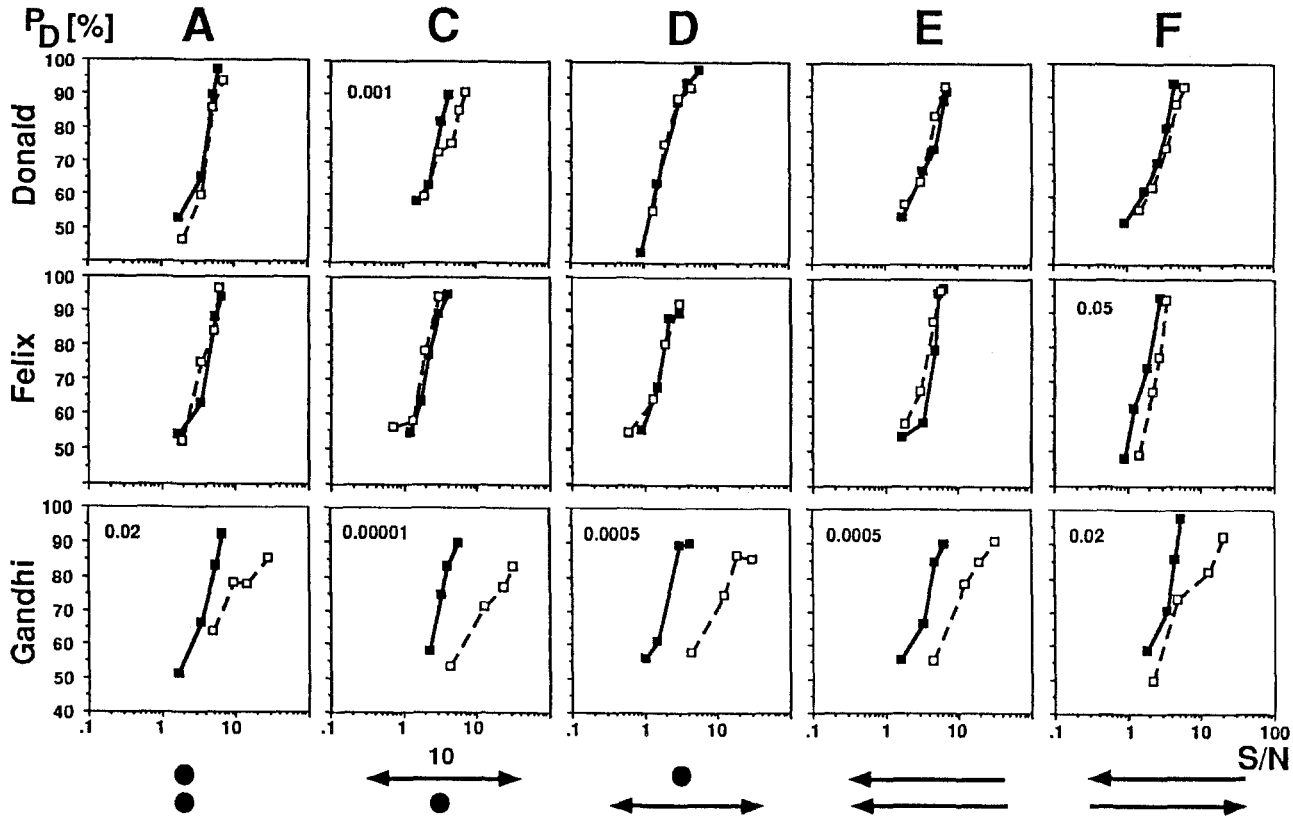

Fig. 9A,C-F. Monocularly tested detection curves of three cats before (*continuous lines*) and after the operation (*dashed lines*). **A** Patterns and background both kept stationary (left eye tested); **C** patterns moved at 10 cm/s, background kept stationary (left eye); **D** background moved at 10 cm/s, patterns kept stationary (left eye); **E** patterns and background both moved at 10 cm/s simultaneously in phase to one another (right eye); **F** patterns and background both moved at 5 cm/s in anti-phase to one another (right eye). *Abscissa* in logarithmic scale

Table 3. Monocular test series performed with intact and lateral geniculate visual area-lesioned cats

Test	Cat	S/N at 84.1%		Shift	$P<$	Significant deficits
		Intact	Lesioned			
Patterns stationary,	Donald	4.5	4.6	0.1	0.4	
background stationary,	Felix	4.8	4.8	0	0.3	
left eye	Gandhi	5.1	11.4	6.3	0.02	x
Patterns moved at 10 cm/s,	Donald	3.2	5.5	2.3	0.001	x
background stationary,	Felix	2.7	2.3	−0.4	0.02	x
left eye	Gandhi	3.9	n.r.	≫	0.00001	x
Patterns stationary,	Donald	2.5	2.5	0	0.3	
background moved at 10 cm/s,	Felix	1.9	2.2	0.3	0.3	
left eye	Gandhi	2.5	10.7	8.2	0.0005	x
Patterns and background	Donald	5.8	4.8	−1.0	0.4	
moved simultaneously in phase,	Felix	4.8	4.0	−0.8	0.7	
right eye	Gandhi	4.6	10.8	6.2	0.0005	x
Patterns and background moved	Donald	3.5	4.1	0.6	0.2	
in anti-phase to one another,	Felix	2.2	2.9	0.7	0.05	x
right eye	Gandhi	4.1	10.4	6.3	0.02	x

See notes to Table 1

one: the test with quickly moving patterns (Fig. 7C). The extent of all deficits were very similar (Table 1, S/N shifts 1.1–2.0; Table 2, contrast shifts 0.4 −0.9).

Monocular tests. The pre- (black squares) and postoperative (empty squares) detection curves of the monocular test series carried out with the cats Donald, Felix and Gandhi are shown in Fig. 9. Again, the significance level for the deficit is given only if it was significant. Additionally, the S/N values for a P_D of 84.1% positive responses, the shifts between pre- and postoperative detection curves, and the levels of significance of the deficits are listed in Table 3.

Cat *Donald* showed a small but significant deficit only

for moving patterns on a stationary background which was tested with his left eye (Fig. 9C). Cat *Felix* had a significant deficit only when patterns and background were moved in anti-phase to each other, which was tested with his right eye (Fig. 9E). Cat *Gandhi*, however, showed significant deficits in all five test situations, regardless of whether his left or right eye was tested. However, he was not able to reach the 84% correct level when he was tested with moving patterns on a stationary background.

Discussion

Evaluation of the lesions

An evaluation of the lesions described above should take into account the fact that unilateral cortical lesions do not affect the binocular behavioural performance in a pattern discrimination task (Berlucchi et al. 1978, 1979; Ptito and Lepore 1983). The only relevant portion of the lesion is that which bilaterally affects the visual field. Moreover, the extent of the damage of a cortical area is important for the evaluation of its behavioural consequence. Therefore, we decided that cortical areas from which more than 80% remained bilaterally intact were not taken into consideration for an evaluation of the postoperative deficits. Under these constraints, in the case of cat Cassy, a damage of 50% of area 5, 40% of area 7, 45% of AMLS, 40% of ALLS, and 45% of PMLS and PLLS only can be considered for an interpretation of the deficits (Fig. 6).

This is also true for an evaluation of the undercutting of areas 17, 18 and 19. Accordingly, the extent of the concurrent undercutting of both hemispheres is considered for the behavioural performance of the cats and might lead to the clarification of the great differences in the postoperative performance: in cat Donald the coexisting undercutting of both hemispheres extended from A1 to A0.4, i.e. only 0.6 mm cortex was concurrently undercut; in Felix and Cassy there occurred no range of concurrent undercutting of both hemispheres; in Gandhi, however, this was the case over an extent of 3.3 mm; and in Cäsar the coexisting undercutting of both hemispheres came to 3.7 mm. In all cats the undercut parts of areas 17 and 18 represent different parts of the lower visual field, while the representations of the upper visual field seemed to remain intact in all cats.

This consideration is reinforced by the deficits found in the binocular test series with stationary patterns (A). Cats Donald and Felix had no deficits in this situation, in which cats with bilateral lesions of areas 17/18 showed severe deficits (Krüger et al. 1986). This is a clear indication that the unilateral undercutting of areas 17/18/19 in cats Donald and Felix can be neglected because it occurred without behavioural consequence.

Three cats (Gandhi, Cäsar, Cassy) were affected on all tests. From this it would seem apparent that neither area 21a nor area 5 are implicated in these deficits since each of the three seriously impaired cats had one or both of these areas intact. The problems in these cats must therefore be due to damage to area 7, to undercutting of areas 17 and 18 and/or to LSA damage.

Based on the considerations mentioned above, four subgroups can be distinguished, depending on the size and extent of the lesions and the postoperative deficits:

1. Lesion of lateral suprasylvian visual areas without undercutting areas 17, 18 and 19 (Felix and Donald)
2. Lesion of lateral suprasylvian visual areas with undercutting areas 17, 18 and 19 (Cäsar and Gandhi)
3. Lesion of area 7 (Sara and Lancelot)
4. Lesion of area 7 and the lateral suprasylvian visual areas without undercutting areas 17, 18 and 19 (Cassy)

Postoperative behaviour

None of the cats described above had deficits in retention of the preoperatively learned pattern discriminations. This statement is in agreement with earlier experiments on cats with bilateral lesions relatively confined to PMLS cortex, for which little or no effect on postoperative retention or new learning of brightness, form or pattern discriminations was reported (Sprague et al. 1977; Spear et al. 1983; Spear 1991). Our results, however, conflict with the experiments of Berlucchi and Sprague (1981), who found that cats with suprasylvian lesions undercutting areas 17, 18 and 19 had devastating deficits in retention, no such animal was able to perform above chance in a pattern problem. The disagreement can easily be attributed to the different extent of the cortical lesions and the undercutting. The lesions described by Berlucchi and Sprague (1981) seemed to involve parts of area 19 and to undercut areas 17, 18 and 19 to a much larger extent than the lesions of cats Gandhi and Cäsar. Accordingly, the lesions described by Berlucchi and Sprague (1981) resemble a lesion of areas 17, 18 and 19. It is well known that cats with removal of areas 17, 18 and 19 show devastating deficits in retention and are unable to carry out such a pattern discrimination task as described above (Doty 1971; Cornwell et al. 1980; Berlucchi and Sprague 1981; Krüger et al. 1988).

Detection performance of lesioned cats

For an evaluation of the results of the detection experiments, the characteristics of the different test situations must be taken into consideration. Owing to the lack of background, the test series with moving patterns on uniformly grey background are characterized by a lack of relative velocity and a lack of interaction between patterns and background. By comparison of this test situation with that consisting of moving patterns on a focused background, it is possible to decide wheather a deficit is caused by movement of the patterns per se or if it is caused by the interaction between patterns and background. This will be discussed for the four different lesions mentioned above.

Lesion of LSA without undercutting areas 17, 18 *and* 19 (*Donald and Felix*). The large bilateral lesion of LSA in cat Donald led to significant deficits in all test situations with moving patterns on a structured background, no

matter what the pattern velocity was, and only in the cases with relative velocity (Fig. 7B, C, F). The results from the monocular test series (Fig. 9C, F) confirmed those from the binocular ones, though the deficit with patterns and background moving in anti-phase to one another (F) was not significant.

The smaller bilateral LSA lesion in cat Felix led to a small but significant binocular deficit only in the case with quickly moving patterns on a stationary background (Fig. 7C). However, in the monocular test series, Felix also had a significant deficit with patterns and background moving in anti-phase to one another (Fig. 9F). This confirmed that the extent of the lesions and the behavioural deficits in these two cats were very similar.

In the situation with the patterns and background moving simultaneously in phase with one another, without relative velocity both cats had no deficits (Fig. 7E). Furthermore, we found no deficits in these cats when they were tested with moving patterns on a uniformly grey background (Fig. 8B). This provides a strong argument that the deficits must have been caused by the interaction of patterns and background, and not by movement of a pattern per se. When the two cats were tested with stationary or moving patterns on uniformly grey background (Fig. 8), we found that they performed much better after the lesion than as intact animals. Though the possibility of a practice effect cannot be excluded completely, we think it is rather improbable, because these two particular animals were completely overtrained in a training and testing phase of 11 months before they were lesioned. In addition, both cats seemed very keen to make no mistakes.

The deficits we found in the cats with LSA lesions were restricted to cases where pattern motion was associated with relative movements between patterns and background. These cases are comparable with situations in the natural environment in which an object is moving while the cat is stationary or follows the object. Therefore, we believe our results indicate an involvement of the LSA in the analysis of object-background interactions and in the discrimination of object-induced and self-induced motion. We propose that the LSA might be the place in which the sensory input of background motion from the retina, which always is caused by self-motion, is balanced with the actual motor signals of this self-motion, stabilizing vision and making the system invariant to self-induced motion.

There are many findings from single cell recordings in the LSA that correspond with our ideas. The large receptive fields found in areas PMLS and PLLS with their centre-surround organization (Spear and Baumann 1975; Camarda and Rizzolatti 1976; Smith and Spear 1979; Zumbroich et al. 1986; Rauschecker et al. 1987b) are very well suited for processing large stimuli such as background and are a prerequisite for establishing invariance performances.

Several authors reported saccade-related discharges in the PMLS (Kennedy and Magnin 1977; Vanni-Mercier and Magnin 1982, Komatsu et al. 1983; Yin et al. 1986), and recently Yin and Greenwood (1992b) presented evidence of saccade-related responses in areas PMLS and PLLS of behaving cats. Such responses could be corollary discharges or efferent copy signals from the superior colliculus relayed through the pulvinar. Additionally, Donaldson (1979) reported that some PMLS cells respond to stretch of extraocular muscles. If the LSA is the place to calculate such a corollary discharge with the sensory input of background motion, LSA lesioned cats would no longer be able to determine the correct object motion in combination with self-induced motion, and consequently their visual acuity of objects would be diminished. Indeed, Pasternak et al. (1989) found permanent deficits in cats with lesions in the lateral suprasylvian cortex who had to discriminate differences in the speed of moving gratings. These deficits were most pronounced at higher temporal and spatial frequencies and at lower contrasts. This corresponds with our findings; they suggest that the LSA may provide speed information to oculomotor areas.

Following the double-opponent model of von Grünau and Frost (1983), the presence of a structured background, but not a corollary discharge or efferent copy signal, is essential for the determination of object- and self-induced motion in which cells with so-called double-opponent, centre-surround direction selectivity could be involved (Frost 1985). Those cells were found in area PMLS (von Grünau and Frost 1983; Dinse and Krüger 1987) and also in areas 17, 18 and 19 of the cat (Dinse and Krüger 1987). All the same, such a mechanism of double-opponent, direction selective cells calculates the relative velocity between object and background and is therefore in agreement with the reafference principle, because both methods make the animal insensitive to self-induced motion and help to determine the object motion. Our behavioural results described above are consistent with both methods, making the animal invariant of self-induced movements, but are not sufficient to decide which might be implemented in the cat.

For the detection of a moving object, which in a natural situation is always paralleled by movement of the background, as the animal tries to focus the object the LSA processing requires a cooperation with areas 17/18. As we have shown, areas 17/18 alone are able to process moving patterns on a structured background, but with a degraded performance (see Fig. 7). The smaller receptive fields in areas 17 and 18 are much less qualified for processing large stimuli such as background than those in LSA. Cooperation with the LSA might cause an increase in efficiency in determining the relative velocity between the object and either the background or the object velocity. We believe that there is a trade off between the improvement of the performance in situations with moving patterns and background, and a deterioration of the perceptual performance in scenes without background. In this view, owing to its cooperation with the LSA, area 17 is hampered from reaching its maximal function. This assumption might explain the improvement of the performance in the situations with uniformly grey background (Fig. 8) when the LSA are eliminated.

Our results do not support an involvement of the LSA in the generation of the optokinetic nystagmus (OKN;

Ventre 1985a; Hamada et al. 1988; Herdman et al. 1989; Tusa et al. 1989), because we found no deficits in the situation with patterns and background moving simultaneously in phase with one another (Fig. 7E) which should have elicited a strong OKN.

Tusa et al. (1989) found the reduction in gain of the slow phase of OKN after unilateral LSA lesions was not necessarily due to a deterioration of the evoked eye movements, it could also result from a miscalculation of the sensory retinal input with the motor information concerning self-motion, which results in the calculation of a reduced pattern velocity and so affects the gain. Therefore, the deficits they reported may be caused by a very similar effect to the one we described above.

Lesion of LSA with undercutting of areas 17, 18 *and* 19 (*Cäsar and Gandhi*). As soon as a bilateral undercutting of areas 17/18/19 was added to the lesion of the LSA, we found very strong deficits in all test situations. These additional deficits seem to be mainly due to the undercutting of areas 17 and 18, as 17/18-lesioned cats are known to have strong deficits of comparable extent. However, while the deficits with stationary and moving patterns are more or less equal in 17/18-lesioned cats (Krüger et al. 1986), the LSA-lesioned cats showed much more pronounced deficits in the binocular test series with moving than with stationary patterns. As soon as the patterns were moving on a background, the two cats were no longer able to reach the 84% correct criterion (Fig. 7B, C, E, F; see also Kiefer et al. 1989). As the two cats were able to reach criterion viewing moving patterns on a uniformly grey background (Fig. 8B), these deficits seem mainly to be caused by the interaction of patterns and background and not by motion of the patterns per se. A comparison with the results of cat Cassy, who had a very similar lesion to Cäsar's, including parts of area 7 and LSA but without any undercutting of areas 17 and 18, shows that in Cassy the deficits with the moving patterns are smaller than those with stationary ones. These findings provide additional support for the assumption that the more pronounced deficits with moving patterns seem not to be due to damage of areas 7 or AMLS and ALLS but rather to the undercutting of areas 17 and 18, and that the LSA is part of a system which is involved in the detection of moving objects but which requires the cooperation of LSA and striate cortex for its full function.

After finishing the postoperative detection tests (about 6 months after the operation) in cat Cäsar, the horizontal (OKN) was measured using the search coil technique (Robinson 1963), in the laboratory of Prof. K.-P. Hoffmann. During optokinetic stimulation with a binary noise the gain (slow-phase velocity/stimulus velocity) of OKN in cat Cäsar was in the range of normal animals (unpublished results of Prof. Hoffmann). We take this finding as an indication that the deficits in these cats are unrelated to the motor processing of eye movements and are mainly based on sensory information processing. Since Cäsar was tested 6 months after the operation, these results agree with the findings of Tusa et al. (1989) that cats recover nearly to their preoperative values in 6 months.

Removal of area 7 (*Lancelot and Sara*). Cat Lancelot, an animal with no apparent damage to the LSA and no undercutting of areas 17/18, had a larger lesion of area 7 than cat Sara. He showed small but significant deficits of more or less the same extent in all test situations except one: the cat had no significant deficit with quickly moving patterns on a structured background (Fig. 7C). This seems to implicate area 7 in detection of both stationary and moving targets. From electrophysiological data this would be very surprising because only about two-thirds of the neurons in area 7 are responsive to visual stimulation (Dow und Dubner 1969, 1971; Straschill und Schick 1974; Joseph and Giroud 1986; Yin and Greenwood 1992a), but the deficits we found in the visual detection tasks were comparable with those following a lesion of area 19 (Dinse and Krüger 1990). Moreover, the intact areas of the lateral suprasylvian cortex and the intact areas 17, 18 and 19 were obviously not able to compensate for this loss of area 7 completely. Therefore, we suppose the participation of area 7 in a mechanism, since its loss or impairment seems to affect the detection performance in a way that is not directly related to sensory processing of visual information.

Some indications come from the results in a depth-perception task on the jumping stand with the same cats (Krüger et al. 1990, 1993). For Lancelot we found that his monocular performance was nearly unaffected after the lesion, whereas his binocular performance was severely impaired. This means that the lesion in this cat mainly impaired the ability to use the binocular depth cues such as retinal disparity or vergence angle for the perception of depth, which results in a loss of binocular fusion. Therefore, we propose that after this lesion the mechanisms of vergence movements or of binocular fusion of both retinal images might be impaired. Such an impairment can cause unstable perception, double images for the cat or a reduced visual acuity, because the images are out of focus. The consequence for the detection performance would be that the cats showed a somewhat poorer performance in all test series, regardless of whether the patterns or the background were kept stationary or were moving, which was in fact the case.

A similar effect of monocular superiority in a series of visual pattern discriminations has already been described for monkeys with mid-brain lesions involving the pretectum and superior colliculi. Also in this case, the abnormality of vergence eye movements was proposed to be one, if not the only, explanation for the animals being significantly less accurate when viewing binocularly (Lawler and Cowey 1986).

This assumption corresponds to electrophysiological findings from area 7 in the cat. This area is thought to participate in the construction of the behavioural space and the maintenance of space perception during and after movements. Although Yin and Greenwood (1992a) reported a lower percentage of direction selective cells in area 7 than in areas PMLS and PLLS, the visual-responsive neurons in area 7 show a preference for high velocities and they are highly direction selective (Dow and Dubner 1969, 1971; Straschill and Schick 1974). However, there are a higher percentage of cells in area 7 than in

areas PMLS and PLLS whose activity is saccade-related (Yin and Greenwood 1992b; Straschill und Schick 1974; Joseph and Giroud 1986). Such neurons are able to update the eye position in the perceptual space. The so-called fixation and vergence neurons might be the neuronal substrate for the construction of the behavioural space; other neurons contribute to the constancy of the spatial perception during a movement (Yin and Greenwood 1992b; Joseph and Giroud 1986). It seems therefore conceivable that area 7 of the cat contributes to the control of compensatory eye movements or to keeping the plane of fusion focused.

The results from cat Sara, who had a much smaller lesion of area 7 than Lancelot, reveal the type of test series that were most sensitive to the deficit following a lesion of area 7. In the detection experiments Sara showed small but significant deficits when the background was moved, given that there was a low relative velocity between patterns and background (Fig. 7D,F). It appears reasonable to assume that these situations give rise to the sensory information "I am moving", which can be compared with situations in the natural environment in which the animal makes an eye or head movement to produce no (D) or only small (F) gain to a simultaneously moving object. This appears to make the background move. On the other hand, Sara had a significant deficit with patterns moving slowly on a uniformly grey background (Fig. 8B), which indicates that, in this case, the deficit seems to be caused by movement of the pattern per se and not by the interaction of pattern and background. However, whenever the patterns were moving on a structured background (Fig. 7B, C), Sara had no deficits. Additionally, one of these situations (C) was the only one in which cat Lancelot had no deficit (Fig. 7C). Compared with the LSA-lesioned cats who had significant deficits in these situations, we conclude that, unlike lesions in area 7, the LSA seem to be involved.

Lesion of area 7 plus LSA (Cassy). Surprisingly, the combination of the lesion in area 7 with that in the LSA in cat Cassy roughly leads to a summation of the effects of the two lesions alone. Cassy had significant deficits in all test situations (Fig. 7) and the shifts in her pre- and postoperative detection curves approximately correspond to a summation of the shifts in cats Donald and Lancelot (see Tables 1, 2). This correspondence was not consistent in all test series, particularly the stationary patterns on the stationary background (Fig. 7A). In that case, Cassy had her strongest deficit (S/N shift of 5.0), which was much larger than those of cats Lancelot and Donald. But, comparing the extent of the lesion of cat Cassy with those of cats Felix, Donald, Lancelot and Sara (Fig. 6), one has to admit that in cat Cassy areas AMLS and ALLS are much more affected than in any one of the other four cats. This might be the cause for the discrepancies in this case. The results from cat Cassy again give support to the hypothesis that area 7 and the LSA are functionally different.

Functional implications

Though we trained and tested seven animals, the number of animals for each type of lesion was low, and the lesions of each type showed interindividual variability. Therefore the following conclusions should be met with appropriate caution.

1. Our results give support to the idea of a functional segregation between the LSA on the one hand and area 7 on the other. This idea is further supported by the results of a depth-perception task with the same cats (Krüger et al. 1990, 1993) in which the monocular performance was nearly unaffected after lesion of area 7, but impaired after lesion of LSA, while the binocular performance deteriorated after both types of lesion.
2. The permanent deficits found after lesion of the LSA confirm the view that the LSA are involved in pattern recognition whenever it is associated with motion, and in object-background interactions. They also indicate an involvement of the LSA in the discrimination of object-induced and self-induced motion. We propose that the LSA might be the place in which the sensory input of background motion from the retina, which is always caused by self-motion, is balanced with the actual motor signals of this self-motion, making the system insensitive to self-induced motion and thus stabilizing vision.
3. Area 7 seems to be involved in a mechanism which has a similar effect on the detection performance in each stimulus configuration. This can be due to impaired binocular fusion of both retinal images, or an impaired mechanism of vergence movements which would lead to unstable perception, resulting in double images for the cat or reduced visual acuity because the images are out of focus.
4. We conclude that the LSA is part of a system which is involved in the detection of moving objects, but which requires cooperation with striate cortex for its full function. Our findings do not support the existence of a one area-one function relation. On the contrary, they are in agreement with the assumption of a pronounced interaction of different areas.

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References

Bando T (1985) Pupillary constriction evoked from the posterior medial lateral suprasylvian (PMLS) area in cats. Neurosci Res 2:472–485

Bando T, Yamamoto N, Tsukuhara N (1984) Cortical neurons related to lens accommodation in posterior lateral suprasylvian area in cats. J Neurophysiol 52:879–891

Berlucchi G, Sprague JM (1981) The cerebral cortex in visual learning and memory, and in interhemispheric transfer in the cat. In: Schmitt FO, Worden FG, Adelman G, Dennis SG (eds) The organization of the cerebral cortex. MIT, Cambridge, Mass.,pp 415–440

Berlucchi G, Sprague JM, Lepore F, Mascetti GG (1978) Effects of lesions of areas 17, 18 and 19 on interocular transfer of pattern discriminations in split-chiasm cats. Exp Brain Res 31:275–297

Berlucchi G, Sprague JM, Antonini A, Simoni A (1979) Learning and interhemispheric transfer of visual pattern discriminations following unilateral suprasylvian lesions in split-chiasm cats. Exp Brain Res 34:551–574

Camarda R, Rizzolatti G (1976) Visual receptive fields in the lateral suprasylvian area (Clare-Bishop) of the cat. Brain Res 101:427–443

Cornwell P, Overman W, Ross C (1980) Lesions of visual cortex impair discrimination of hidden figures by cats. Physiol Behav 24:533–540

Dinse HRO, Krüger K (1987) Sensitivity of cat cortical neurons (areas 17, 18, 19, PMLS) to interaction between object and background - local vs global processing revisited. Soc Neurosci Abstr 13:1624

Dinse HRO, Krüger K (1990) Contribution of area 19 to the foreground-background interaction of the cat: an analysis based on single cell recordings and behavioral experiments. Exp Brain Res 82:107–122

Donaldson IML (1979) Responses in cat suprasylvian cortex (Clare-Bishop Area) to stretch of extraocular muscles. J Physiol (Lond) 296:60

Doty RW (1971) Survival of pattern vision after removal of striate cortex in the adult cat. J Comp Neurol 143:341–370

Dow BM, Dubner R (1969) Visual receptive fields and responses to movement in an association area of cat cerebral cortex. J Neurophysiol 32:773–784

Dow BM, Dubner R (1971) Single-unit responses to moving stimuli in middle suprasylvian gyrus of the cat. J Neurophysiol 3:47–55

Dubner R, Brown JF (1968) Responses of cells to restricted visual stimuli in an association area of cat cerebral cortex. Exp Neurol 20:70–86

Fabre M, Buser P (1980) Structures involved in acquisition and performance of visually guided movements in the cat. Acta Neurobiol Exp (Warsz) 40:95–116

Fabre M, Buser P (1981) Effects of lesioning the anterior suprasylvian cortex on visuo-motor guidance performance in the cat. Exp Brain Res 41:81–88

Frost BJ (1985) Neural mechanisms for detecting object motion and figure-ground boundaries contrasted with self-motion detecting systems. In: Ingle D, Jeannerod M, Lee D (eds) Brain mechanisms of spatial vision. Nijhoff, Dordrecht

Grünau M von, Frost BJ (1983) Double opponent process mechanism underlying the RF-structure of directionally specific cells of cat's lateral suprasylvian visual area. Exp Brain Res 49:84–92

Grünau M von, Zumbroich TJ, Poulin C (1987) Visual receptive field properties in the posterior suprasylvian cortex of the cat: a comparison between areas PMLS and PLLS. Vision Res 27:343–356

Hamada T, Flandrin JM, Furuya N (1988) Latent defect in monocular optokinetic nystagmus after neonatal removal of the lateral suprasylvian area in the cat. Behav Brain Res 27:109–114

Hardy SC, Stein BE (1988) Small lateral suprasylvian cortex lesions produce visual neglect and decreased visual activity in the superior colliculus. J Comp Neurol 273:527–542

Hassler R, Muhs-Clement K (1964) Architektonischer Aufbau des sensomotorischen und parietalen Cortex der Katze. J Hirnforsch 6:377–420

Heath CJ, Jones EG (1971) The anatomical organization of the supraslvian gyrus of the cat. Ergeb Anat Entwicklungsgesch 45:3–64

Herdman SJ, Tusa RJ, Smith CB (1989) Cortical areas involved in horizontal OKN in cats: metabolic activity. J Neurosci 9:1150–1162

Hyvärinen J (1982) The parietal cortex of monkey and man. Springer, Berlin Heidelberg New York

Joseph JP, Giroud P (1986) Visuomotor properties of the anterior suprasylvian gyrus of the awake cat. Exp Brain Res 62:355–362

Kennedy H, Magnin M (1977) Saccadic influences on single neuron activity in the medial bank of cats' suprasylvian sulcus (Clare-Bishop area). Exp Brain Res 27:315–317

Kiefer W, Krüger K, Strauss G, Berlucchi G (1989) Considerable deficits in the detection performance of the cat after lesion of the suprasylvian visual cortex. Exp Brain Res 75:208–212

Komatsu Y, Shibuki K, Toyama K (1983) Eye movement related activities in cells of the lateral suprasylvian cortex of the cat. Neurosci Lett 41:271–276

Krone G, Kunz D, von Seelen W (1983) On the analysis of the cat's pattern recognition system. Biol Cybern 48:115–124

Krüger K, Dinse HRO (1988) Background dependence of direction selectivity in cortical neurons of areas 17, 18, 19, 7 and PMLS of the cat. Soc Neurosci Abstr 14:602

Krüger K, Heitländer-Fansa H, Dinse HRO, Berlucchi G (1986) Detection performance of normal cats and those lacking areas 17 and 18: a behavioral approach to analyse pattern recognition deficits. Exp Brain Res 63:233–247

Krüger K, Donicht M, Müller-Kusdian G, Kiefer W, Berlucchi G (1988) Lesion of areas 17/18/19: effects on the cat's performance in a binary detection task. Exp Brain Res 72:510–516

Krüger K, Groh A, Kiefer W (1990) Lesion of area 7 and the lateral suprasylvian areas impair depth perception of cats (abstract). Invest Ophthalmol Vis Sci 31:399

Krüger K, Kiefer W, Groh A (1993) Lesion of the suprasylvian cortex impairs depth perception of cats. Neuroreport 4: 883–886

Larsen RJ, Marx ML (1986) An introduction to mathematical statistics and its applications. Prentice-Hall, Englewood Cliffs, NJ

Lawler KA, Cowey A (1986) The effects of pretectal and superior collicular lesions on binocular vision. Exp Brain Res 63:402–408

Niimi K, Sprague JM (1970) Thalamo-cortical organization of the visual system in the cat. J Comp Neurol 138:219–250

Otsuka R, Hassler R (1962) Über Aufbau und Gliederung der corticalen Sehsphäre bei der Katze. Arch Psychiatr Z Neurol 203:212–234

Palmer LA, Rosenquist AC, Tusa RJ (1978) The retinotopic organization of lateral suprasylvian visual areas in the cat. J Comp Neurol 177:237–256

Pasternak T, Horn CM, Maunsell JHR (1989) Deficits in speed discrimination following lesions of the lateral suprasylvian cortex in the cat. Visual Neurosci 3:365–375

Ptito M, Lepore F (1983) Effects of unilateral and bilateral lesions of the lateral suprasylvian area on learning and interhemispheric transfer of pattern discrimination in the cat. Behav Brain Res 7:211–227

Rauschecker JP, Grünau MW von, Poulin C (1987a) Centrifugal organization of direction preference in the cat's lateral suprasylvian visual cortex. J Neurosci 7:943–958

Rauschecker JP, Grünau MW von, Poulin C (1987b) Thalamo-cortical connections and their correlation with receptive field properties in the cat's lateral suprasylvian visual cortex. Exp Brain Res 67:100–112

Reinoso-Suarez F (1961) Topographischer Hirnatlas der Katze. Merck, Darmstadt

Rizzolatti G, Camarda R (1975) Inhibition of visual responses of single units in the cat visual area of the lateral suprasylvian gyrus (Clare-Bishop area) by the introduction of a second visual stimulus. Brain Res 88:357–361

Rizzolatti G, Camarda R (1977) Influence of the presentation of remote visual stimuli of visual responses of cat area 17 and lateral suprasylvian area. Exp Brain Res 29:107–122

Rosenquist AC (1985) Connections of visual cortical areas in the cat. In: Peters A, Jones EG (eds) Visual Cortex. (Cerebral Cortex vol 3) Plenum, New York, pp 81–117

Robertson RT, Mayers KS, Teyler TJ, Bettinger LA, Birch H, Davis JL, Phillips DS, Thompson RF (1975) Unit activity in posterior association cortex of cat. J Neurophysiol 38:780–794

Robinson DA (1963) A method of measuring eye movement using a scleral search coil in a magnetic field. IEEE Trans Biomed Eng 10:137–145

Sanderson KJ (1971) The projection of the visual field to the lateral geniculate and medial interlaminar nuclei in the cat. J Comp Neurol 142:101–118

Smith DC, Spear PD (1979) Effects of superior colliculus removal on receptive-field properties of neurons in the lateral suprasylvian visual area of the cat. J Neurophysiol 42:57–75

Spear PD (1991) Functions of extrastriate visual cortex in nonprimate species. In: Leventhal A (ed) The neural basis of visual function. (*Vision and visual dysfunction, vol.* 4) Macmillan, Basingstoke, UK, pp 339–370

Spear PD, Baumann TP (1975) Receptive field characteristics of single neurons in lateral suprasylvian visual area of the cat. J Neurophysiol 38:1403–1420

Spear PD, Miller S, Ohman L (1983) Effects of lateral suprasylvian visual cortex lesions on visual localisation, discrimination, and attention in cats. Behav Brain Res 10:339–359

Sprague JM, Levy J, DiBerardino A, Berlucchi G (1977) Visual cortex areas mediating form discrimination in the cat. J Comp Neurol 172:441–488

Straschill M, Schick F (1974) Neuronal activity during eye movements in a visual association area of cat cerebral cortex. Exp Brain Res 19:467–477

Toyama K, Kozasa T (1982) Responses of Clare-Bishop neurones to three dimensional movement of a light stimulus. Vision Res 22:571–574

Toyama K, Komatsu Y, Kasai H, Fujii K, Umetani K (1985) Responsiveness of Clare-Bishop neurons to visual cues associated with motion of a visual stimulus in three-dimensional space. Vision Res 25:407–414

Tusa RJ, Palmer LA, Rosenquist AC (1981) Multiple cortical visual areas: visual field topography in the cat. In: Woolsey CN (ed) Cortical sensory organization, vol 2. Humana, Clifton, NJ, 1–61

Tusa RJ, Demer JL, Herdman SJ (1989) Cortical areas involved in OKN and VOR in cats: cortical lesions. J Neurosci 9:1163–1178

Vanni-Mercier G, Magnin M (1982) Retinotopic organization of extra-retinal saccade-related input to the visual cortex in the cat. Exp Brain Res 46:368–376

Ventre J (1985a) Cortical control of oculomotor functions. I. Optokinetic nystagmus. Behav Brain Res 15:211–226

Ventre J (1985b) Cortical control and oculomotor functions. II. Vestibulo-ocular reflex and visual-vestibular interaction. Behav Brain Res 17:227–234

Wood CC, Spear PD, Braun JJ (1974) Effects of sequential lesions of suprasylvian gyri and visual cortex on pattern discrimination in the cat. Brain Res 66:443–466

Yin TCT, Medjbeur S, El Barbary A, Felsten G (1986) Visual response properties of cells in the middle and lateral suprasylvian cortices on the behaving cat. Invest Ophthalmol Vis Sci 27:155

Yin TCT, Greenwood M (1992a) Visual response properties of neurons in the middle and lateral suprasylvian cortices of the behaving cat. Exp Brain Res 88:1–14

Yin TCT, Greenwood M (1992b) Visuomotor interactions in responses of neurons in the middle and lateral suprasylvian cortices of the behaving cat. Exp Brain Res 88:15–32

Zumbroich TJ, Grünau M von, Poulin C, Blakemore C (1986) Differences of visual field representation in the medial and lateral banks of the suprasylvian cortex (PMLS, PLLS) of the cat. Exp Brain Res 64:77–93