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A Decisional Analysis of the Effects of Inferotemporal Lesions in the Rhesus Monkey

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Modified signal detection procedures were used to analyze the effects of inferotemporal cortical resections. The results demonstrated (a) a severe difficulty in responding to differences in luminance; (b) a small but consistent change in sensitivity (d'), which is attributed to an increased sensitivity to noise or a deficiency in the suppression of irrelevant aspects of the environment; and (c) an enhanced bias to respond to a nonrewarded stimulus (a lowering of criterion). This altered bias contrasts with the results obtained from limbic resections in a previous experiment which produced a marked increase in bias to a rewarded contingency without influencing discrimination or detection.

A recurring problem in neuropsychology is the teasing apart of cognitive from primary sensory disturbances. The question was posed by von Monakov (1914) as to whether agnosias and aphasias ever occur without some concomitant change in sensory capacity. Pros and cons have been argued, perhaps most persuasively in the clinical literature by Bay (1953), who claimed that careful testing of patients with lesions of the "association" areas *always* turns up *some* sensory deficiency. This finding raises the question whether these minimal sensory disturbances are due to the invasion by the lesion of primary sensory projection systems or whether the sensory disturbances are part and parcel of any "higher level" dysfunction. One of the hopes of making animal models of agnosia was to help clarify this issue.

However, the behavioral analysis of the effects of lesions of "association" areas of the cortex in nonhuman primates has yielded a myriad of results which have been difficult to interpret.

Three basic findings have been obtained: (a) The posterior and frontal "association" cortex serve different functions; (b) the posterior "association" cortex can be subdivided into areas each of which shows modality specificity, i.e., resections of a subdivision drastically impair discriminations in a specific sensory mode and only in that mode; and (c) a variety of disturbances are produced when different sorts of discrimination problems are used. These findings raise the same issue as that raised by von Monakov on the basis of clinical testing. For instance, lesions of the cortex of the inferior temporal gyrus result in deficits in retention and in relearning of visual discriminations of all sorts (patterns, color, brightness, etc.) when they are presented in a two-choice task (Blum, Chow, & Pribram, 1950; Chow, 1951; Mishkin & Pribram, 1954). The impairment is limited to the visual mode—somesthetic, gustatory, and auditory dis-

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criminations remain unaffected (Blum et al., 1950; Dewson, Pribram, & Lynch, 1969; Pribram, 1954; Pribram & Barry, 1956; Wilson, 1957). Further, the amount of impairment is a function of the difficulty of the task (Pribram, 1958). Finally, differences in effect on more complex discriminations are obtained when the anterior, middle, or posterior portion of the gyrus is resected (Bolster & Crowne, 1979; Christensen & Pribram, 1977, 1979; Gross, Cowey, & Manning, 1971; Iwai & Mishkin, 1969; Wilson, 1968).

When these tests of greater complexity are used to determine the nature of the visual disturbance, a variety of results are obtained. Thus, in a multiple choice task, Pribram (1960a) found that monkeys with inferotemporal resections sampled fewer cues than did their controls. Butter (1968, 1969) then demonstrated that such lesions also impaired the number of features of a cue which were sampled. However, when a two-choice simultaneous task was changed to a successive version, the operated monkeys were even more impaired despite the fact that cue number and features remained identical, which suggests that task difficulty rather than cue difficulty was the problem (Pribram & Mishkin, 1955). Wilson (1968) reconciled these results by showing that the sampling difficulty was related to spatial redundancy and was processed by the posterior portions of the gyrus whereas task difficulty was related to the temporal redundancies originating in the distribution of reinforcing events and was processed in the anterior parts of the gyrus.

The results of these experiments and their analyses are of the same order as those that define agnosias in human patients. But another set of experiments led to findings that are not readily fitted into such a category. Thus when parametric variations on cue dimensions such as size are made in a two-choice task, the operated monkey also shows a deficit, albeit not a severe one (Mishkin & Hall, 1955). Further, when such variations are made in a situation in which behavior is continuously varied as a function of the stimulus, no deficit at all is found (Ettlinger, 1959).

In short, the question remains unanswered as to whether the cognitive deficit is based on some change in sensitivity to cues, changes in attention or bias to those cues, or some other exclusively cognitive disturbance. To answer this question, we chose a signal-detection approach because it allows a distinction to be made between difficulties attributable to cue sensitivities (expressed as d') and those attributable to response biases (expressed as β) that influence attention to cues. A psychophysical task was used in which luminance (i.e., contrast) and the probability of rewarded target presentation were varied. The two experiments reported below present variations of this task.

Experiment 1

Method

Subjects and lesions. The subjects were six adolescent rhesus monkeys previously tested in simple auditory pattern, and visual pattern discrimination tasks (see Dewson et al., 1969) but without experience in tasks in which the intensity of stimulation or probability of reinforcement was varied. Throughout the experiment the monkeys were individually housed with free access to water. They were fed, after behavioral testing, once per day with Purina monkey pellets and fruit of sufficient quantity to maintain normal growth and reliable responding for the duration of their daily test sessions. Three monkeys had been subjected to bilateral removal of the inferotemporal cortex through direct visual identification and constituted the IT group. Details of the surgical procedure and anatomical verification of the lesions have been reported elsewhere (Dewson et al., 1969). The remaining three monkeys were unoperated and served as a normal control group.

Apparatus. All testing was carried out with the DADTA III system described in detail elsewhere (Pribram, 1969). The animal testing unit consisted of an enclosure lit with a lamp giving ambient light of .7 ftL. (2.4 cd/m^2), with one of the sides of the enclosure being made up of a 4 × 4 matrix of 16 translucent panels. The surround of these panels reflected approximately .4–.7 ftL. (1.37–2.4 cd/m^2). A food cup was centered below the stimulus array. The stimuli were large red and green circles, back projected onto each panel. The red stimuli were maintained at a constant intensity level of approximately 7 ftL. throughout the experiment. The green stimulus intensity (which, when exposed at maximum luminance, also measured approximately 7 ftL.) was controlled by changing the duration of the "on" phase of the stimulus within a flicker with a base period of 24 msec, which is above the fusion threshold (Mishkin & Weiskrantz, 1959). The intensity numbers (6, 5, 4, 3, 2) used hereafter refer to the duration in milliseconds of this "on" phase.

Stimulus color, brightness, and position were controlled by a PDP-8 computer, which also recorded and collated response parameters, such as stimulus choice, and position of the panel pressed, the latency of the response, and whether the response was correct and rewarded.

Experimental design. For Experiment 1, a yes-no (go–no-go) procedure was instituted so that the theory of signal detectability (TSD) could be applied to the resulting data (Green & Swets, 1966; McNicol, 1972). The two stimulus alternatives were the occurrence or nonoccurrence of a green circle on the display matrix. If a green circle appeared during the trial, the appropriate response was to press the panel on which the light appeared (yes or go). If a green circle did not appear, the appropriate response was to withhold a response, i.e., not to press any of the panels (no or no-go).

d', reflecting sensitivity, and β, representing a monkey's criterion, or response bias, are the two independent parameters of TSD. d' was experimentally manipulated by changing the intensity level of the green circle, and β was manipulated by changing the probability of a go trial, inducing a monkey to change its response bias. When intensity remains constant throughout a series of trials while the probability is altered, d' would be expected to remain constant, whereas β might change.

Changing the probability of appearance of one of the stimulus alternatives also allows Receiver Operating Characteristics (ROC) curves to be constructed from the experimental data, provided the assumptions of the signal detection method are not violated. In ROC curves, the probability of a hit (pressing the panel when the green circle appeared) is plotted against the probability of a false alarm (pressing the panel when no green circle appeared) for each probability condition.

In addition, reaction time (RT) was recorded, and the conventional processing of RT through the analysis of variance was supplemented by using the RTs to normalize the choice data. In humans, RT data are often interpreted as providing estimates of the confidence with which choices are made. In the current experiments such "confidence" ratings were used to normalize the results of the choice data in the following manner: The normalized RT latency under each probability condition was expressed as a fraction of the total (i.e., the sum of the latencies of hits for each group of monkeys), normalized for 1. Thus the normalized RT latency also expresses 1 minus the sum of latencies for false alarms. In this way, the differences in absolute cumulative RT among the various intensity conditions were eliminated which allowed direct comparison of hit and false alarm confidence indices across intensity cells. With this procedure, the ratio between the means of these two RT distributions was assumed to be analogous to d' and is labeled d'_{RT}. Under this assumption, as the criterion is moved from high to low values, it generates a curve based on the normalized latencies of the two distributions. Figure 1 illustrates this, and the theoretical curves for different d'_{RT}. This graph reveals the form of the curves as d'_{RT} increases.

Procedure. Since the monkeys had considerable experience with the DADTA III system, the course of pretraining to establish asymptotic performance as a baseline for the TSD experiment could be substantially expedited. During the first 2 pretraining days (50 trials daily), red circles were simultaneously displayed on all the bottom panels of the display matrix. A press of any of these four panels produced the delivery of a 190-mg Noyes banana pellet and initiated a 5-sec intertrial interval followed by a return of the stimulus display. The depression of an unlit panel (false alarm) was recorded, but it neither advanced the program nor produced a reward. Two pretraining sessions were sufficient to ensure that all monkeys were responding consistently with short response latencies to the red circles.

For the next six pretraining test sessions (50 trials daily), the red circles were continuously displayed on the four bottom panels until terminated by a red panel press. On 50% of the trials (go trials) bright green circles (intensity 5) *subsequently* appeared on the second panels of the second and third columns of the display matrix. A press of either green panel within 3 sec of display produced a reward, and 5 sec later another trial was initiated. (If no press occurred, the display extinguished in 3 sec.) During the remaining 50% of these trials (no-go trials), no green circles display followed red light termination. If the monkeys did not press any panel during the ensuing 3-sec period, a reward was delivered. A press of either the red circle or the green circle panel (now unlit) during this 3-sec period was recorded, and the 5-sec intertrial interval was immediately instituted without presentation of reward. For

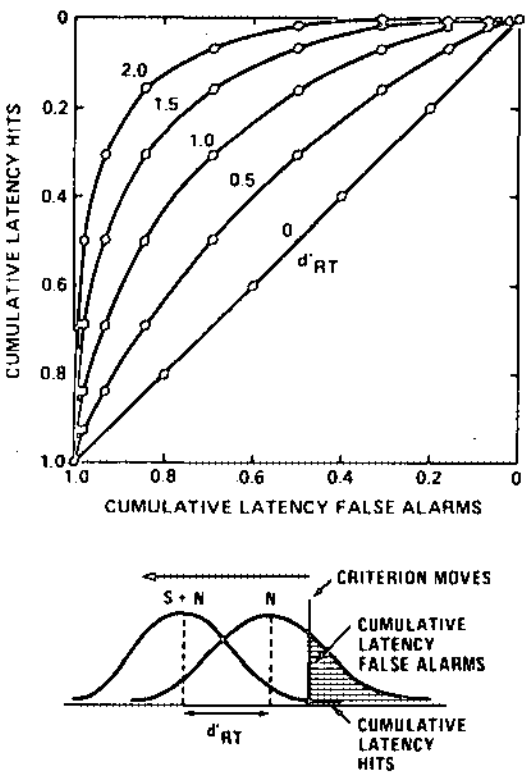

Figure 1. Theoretical d'_{RT} curves constructed for selected values of d'_{RT}. (Lower panel shows model that generates the curves on the graph.)

Table 1
Normalized Mean False Alarm Latency of Individual Subjects, and Mean and Standard Deviation for Each Group in Experiment 1

	Latency normalized false alarms (1 − *p*FA)															
	.8				.6				.4				.2			
	Intensity level				Intensity level				Intensity level				Intensity level			
Subject	2	3	4	5	2	3	4	5	2	3	4	5	2	3	4	5
Normal																
293	.76	.20	.04	.02	.63	.15	.03	.01	.43	.10	.02	.01	.20	.05	.01	.00
294	.74	.08	.11	.00	.60	.05	.09	.00	.36	.02	.06	.00	.19	.01	.04	.00
243	.78	.80	.60	.13	.64	.55	.19	.02	.44	.17	.12	.01	.08	.07	.05	.00
M	.76	.36	.25	.05	.623	.25	.103	.01	.41	.096	.066	.0066	.16	.043	.033	.00
SD	.028	.386	.305	.07	.033	.264	.081	.01	.042	.076	.037	.006	.053	.031	.023	.00
IT																
274	.84	.35	.05	.04	.58	.08	.04	.03	.30	.04	.03	.02	.15	.03	.02	.01
284	.86	.86	.80	.67	.68	.65	.53	.46	.44	.44	.35	.16	.25	.25	.15	.07
295	.80	.81	.88	.22	.60	.65	.67	.01	.44	.40	.16	.01	.24	.17	.07	.00
M	.83	.673	.576	.31	.62	.46	.413	.166	.39	.283	.18	.063	.21	.15	.08	.026
SD	.096	.282	.459	.324	.053	.328	.333	.255	.102	.221	.161	.084	.071	.111	.066	.039

both go and no-go trials, a press of any panel other than those used for red circle or green circle presentation was recorded, but it did not advance the program or produce a reward. These six pretraining sessions were sufficient to ensure that all subjects were producing a criterion of 90% correct and asymptotic short response latencies.

The same general procedure used during pretraining was used during the 50-trial daily test sessions for 48 days, with the following modifications: During each of these test sessions, one of four green stimulus intensities (5, 4, 3, 2) was used in conjunction with one of three a priori probabilities of go trials (80%, 50%, 20%). The stimulus intensity/a priori probability combinations were selected from a scrambled set so that each stimulus intensity/a priori condition was replicated four, and only four, times. For the 80% a priori condition, 40 of the 50 test trials were go trials, and so forth. The sequence of go and no-go trials within each session was arranged randomly.

In this and the succeeding experiments, the actual number of trials varied somewhat from that specified by the theoretical design because of computer malfunction and other problems. The percentage of hits and false alarms, therefore, reflects the actual number of trials within an experimental condition and differs slightly from those that would be calculated on the basis of the numbers given above.

Results

Choice data. Table 1 presents the normalized means and standard deviations of false alarms for each group under all conditions, and Table 2 depicts the results of the choice data. d' and β were calculated (according to Egan, 1975, pp. 57–74) for individual subjects for intensities 4 and 5 taken together, and intensities 2 and 3 taken together, at each of the three different a priori probability levels and averaged for the normal and for the IT groups. Intensity levels 4 and 5 were labeled "high" intensity, and intensity levels 2 and 3, "low" intensity. The data from the normal monkeys and IT monkeys were separately pooled.

Table 2
d′ and β Over All Conditions in Experiment 1

Intensity level/ condition	Normal		IT		d' difference
	d'	B	d'	B	
High					
80%	4.32[a]	.03	2.63	.04	1.69
50%	2.62	.59	1.81	.22	.81
20%	3.86	.06	2.07	.13	1.79
Low					
80%	1.14	.53	.71	.40	.43
50%	.89	.89	.45	.59	.44
20%	1.08	.88	.28	.84	.80

Note. d' differences between normal and IT groups are all significant, $p < .01$. IT = inferotemporal cortex.
[a] A z score of +3 was arbitrarily assigned to the hit probability of 1 in this condition in determining d'.

The most important result of these data is that d' is greater in every case for the normal group. The d' values were compared statistically by using the technique described by Gourevitch and Galanter (1967) and were significant at $p < .01$ if one takes d' per se as a rough measure of sensitivity. This suggests that the IT cortex lesion had a deleterious effect on sensitivity.

As was expected, d' was lower for both groups when the intensity level was lower. The overall d' for both groups at the high intensity was 2.88, and .76 at the low intensity. The difference is also significant ($p < .01$).

According to TSD assumption, d' should have remained relatively constant within each intensity level for both groups. However, in this experiment, d' is lower in the 50% condition at the high intensity for both the normals and the ITs. This effect is also present at the low intensity for the normals but not for the ITs. Some dependence of d' upon the experimental manipulation of changing the probability of signal occurrence is indicated. The monkeys tend to maximize their opportunity for obtaining reward by increasing their false alarm rate at the lower intensities. Thus the task fails to be a time forced-choice procedure and as a result violates the TSD assumption that response bias is independent of sensitivity, and ROC curves could not be constructed on the basis of these data, which are presented in Table 2.

A simple alternative to the ROC analysis was therefore attempted. When the number of hits is divided by the number of total responses (hits and false alarms), a rough measure is obtained of the "efficiency" with which the normal and IT subjects were responding. Table 3 presents a summary of this analysis and shows a loss in efficiency due largely to an increase in false alarms over the asymptotic level of performance achieved in the pretraining sessions. The response of both groups of monkeys to the introduction of the TSD procedure in which

Table 3
Efficiency Scores

Intensity level/ condition	Normal				IT			
	Hit	FA	Total	Efficiency	Hit	FA	Total	Efficiency
High								
80%	1.00	.093	1.093	.92	.995	.478	1.473	.67
50%	.935	.133	1.068	.87	.960	.479	1.439	.67
20%	.996	.113	1.109	.90	.979	.485	1.464	.67
Low								
80%	.868	.494	1.362	.64	.950	.824	1.774	.53
50%	.720	.379	1.099	.65	.921	.832	1.753	.53
20%	.746	.336	1.087	.68	.777	.683	1.460	.53

Note. Efficiency = hits/(hits + false alarms). IT = inferotemporal cortex; FA = false alarm.

intensity and probability of reinforcement are varied is to produce more false alarms, and this tendency is greater in the IT group. Note that the loss in efficiency is greater for the monkeys with IT lesions than for the controls and that the efficiency of the IT group is remarkably uniform at each intensity level. This suggests that some factor influencing efficiency is operating rather than one that influences the production of false alarms per se.

RT data. The mean latency values for correct detections at each intensity level and probability condition for the normals and ITs are presented in Table 4 and illustrated in Figure 2.

A three-factor analysis of variance (ANOVA) with repeated measures on two factors was carried out on these RT data. The three factors were group (normal, IT), stimulus intensity (5, 4, 3, 2), and a priori probability (80%, 50%, 20%).

Only the probability main effect was significant, $F(2, 8) = 11.14$, $p < .01$. The RT was significantly greater as the a priori probability of a signal decreased. Neither of the remaining main effects was statistically significant ($F < 1$). However, both the

Table 4
Mean False Alarm Latency for Individual Subjects, and Mean and Standard Deviation for Each Group, Under All Conditions in Experiment 1

	Intensity level 5			Intensity level 4			Intensity level 3			Intensity level 2		
	Probability schedule			Probability schedule			Probability schedule			Probability schedule		
Group	80%	50%	20%	80%	50%	20%	80%	50%	20%	80%	50%	20%
Normal												
243	.929	1.011	1.109	1.075	.992	1.154	.999	1.218	1.342	.991	.813	1.170
294	.922	.944	1.050	1.055	1.110	1.200	1.263	1.404	1.562	1.308	2.271	1.745
293	1.017	1.063	1.087	1.122	1.237	1.255	1.434	1.389	1.545	1.098	2.256	2.122
Total	2.868	3.018	3.246	3.252	3.339	3.609	3.696	4.011	4.449	3.397	5.340	5.037
M	.956	1.002	1.082	1.083	1.113	1.203	1.232	1.337	1.483	1.132	1.780	1.679
SD	.045	.101	.030	.034	.122	.051	.219	.103	.122	.161	.686	.479
IT												
295	.998	1.071	1.067	.844	.836	1.372	.682	.873	1.300	.657	.728	.873
284	1.001	1.010	1.069	.841	1.216	1.128	.761	.972	1.187	.817	1.022	1.234
274	1.045	1.000	1.167	1.065	1.109	1.309	1.433	1.396	1.639	1.537	1.425	1.057
Total	3.044	3.081	3.303	2.750	3.161	3.809	2.876	3.241	4.126	3.011	3.175	3.164
M	1.015	1.027	1.101	.917	1.053	1.289	.959	1.080	1.375	1.003	1.050	1.055
SD	.026	.038	.057	.128	.196	.127	.413	.278	.235	.468	.349	.180

Note. IT = inferotemporal cortex.

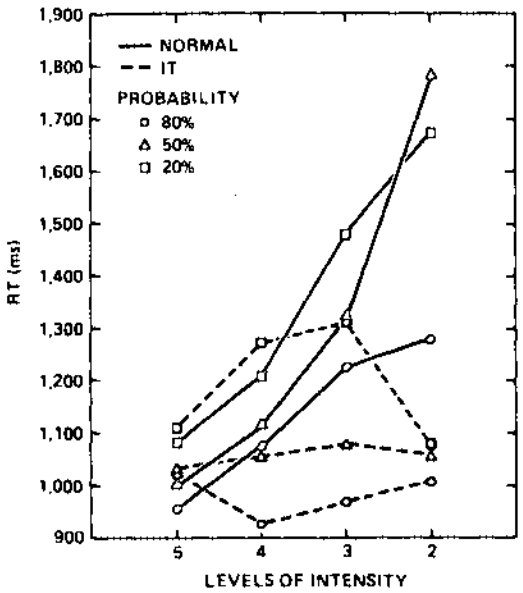

Figure 2. Intensity × Probability × Group interaction for reaction time (RT) in Experiment 1. (Mean RT for normal and IT animals is plotted as a function of the probability of signal occurrences. IT = inferotemporal cortex.)

two-way interaction between probability and intensity and the trip interaction (Groups × Probability × Intensity) were statistically significant, $F(6, 24) = 2.45$, $p < .05$ and $F(6, 24) = 2.70$, $p < .05$, respectively.

Figure 2 clarifies these relations. It can be seen that both groups are affected similarly by the probability of the stimulus occurrence, showing increased RTs with decreased probability. While the normals show a slowed RT with decreased intensity, this parameter has no effect on ITs. Thus the IT data (showing no change in reaction time to stimulus intensity) cancel out the intensity main effect. In addition, these data explain the Probability × Intensity interaction (which is also produced by the failure of IT subjects to respond to stimulus intensity parameters). Finally, note that the intensity levels chosen produce a nearly uniform ratio scale. The normal animals show an equivalence to the psychophysical findings for human subjects (Stevens, 1961).

The ANOVA computation based solely on RT data does not include error scores. It was therefore decided to construct ROC curves according to the procedure outlined in Method in order to determine whether an analysis that takes into account error scores, in terms of hits and false alarms, would provide additional information. Noted above was the spurious nature of the two-way interactions in the ANOVA due to the differences in the responses of each group whereas the data for each group generally fit the assumptions, necessary for the construction of such curves, that response bias is independent of sensitivity. Table 4 shows the means and standard deviations of false alarms for each group under all conditions, and Figure 3 shows ROC curves constructed on the basis of the normalized latency distributions for hits and false alarms on go trials. Because the major difference between groups was found at the 50% probability level, only data from this level are plotted. This restriction also resolves any possible remaining problem that might arise because of the Probability × Intensity interaction.

The purpose of the ROC analysis is to determine whether the normal subjects with their slowed RT are in fact performing more sensitively than the IT group. The curves shown in Figure 3 show that both groups appear to be equally sensitive at high-intensity levels and equally insensitive at low intensities (despite the more cautious approach of the normal subjects). However, the normal group appears to be considerably more sensitive than the IT group at the intermediate range of intensities (3 and 4).

The question arises as to what mechanism might underlie the reduced sensitivity of the IT group. Once again, the method of signal detection can provide an answer. When the ROC curves of Figure 3 are combined, as shown in Figure 4, it is clear that the data of neither the normal nor the IT group show the idealized distribution shown in Figure 1. Such distributions, which are symmetrical about the negative diagonal of the unit square, are characteristic of results obtained in auditory experiments but not those obtained in visual experiments which commonly show a skewed distribution. When the diagonal is symmetrical (i.e., it would show a slope of 1.0 on a normal-normal plot), this indicates that the variances of the signal

and the noise distributions are equal. When the distribution is skewed as in the curves shown in Figure 4, creating slopes in normal-normal plots of less than 1.0, this indicates that the variance of the noise distribution is greater than the variance of the signal distribution. Note that the distribution of the points for the IT group shows an asymmetry even greater (i.e., slope even shallower, if on a normal-normal plot) than that of the control group. This indicates that the lesions have resulted in an increase in the variance of the noise distribution. Thus the apparent decreased sensitivity of the IT monkeys appears to be based on a greater sensitivity to noise or on an inability to suppress irrelevant aspects of the visual environment.

Discussion and A Pilot Left-Right Forced-Choice Experiment

In this experiment, the appropriate response to an absent signal was to withhold pressing the panel. As noted, correct no-go responses were rewarded. This avoids the objection (see Weiskrantz, 1968) that nonrewarded no-go responses bias the subject

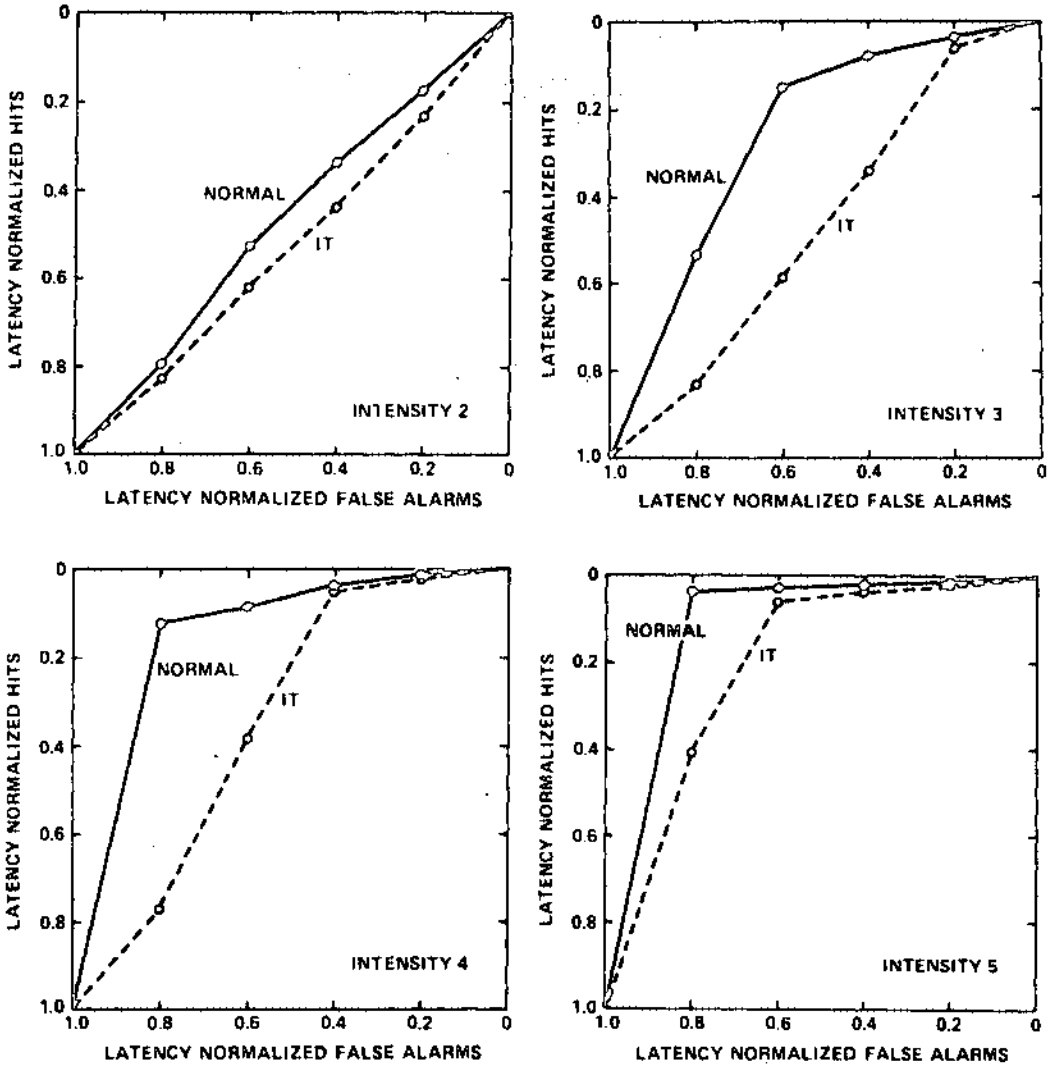

Figure 3. d'_{RT} curves for normal and IT animals at intensities 2, 3, 4, and 5 in Experiment 1. (IT = inferotemporal cortex.)

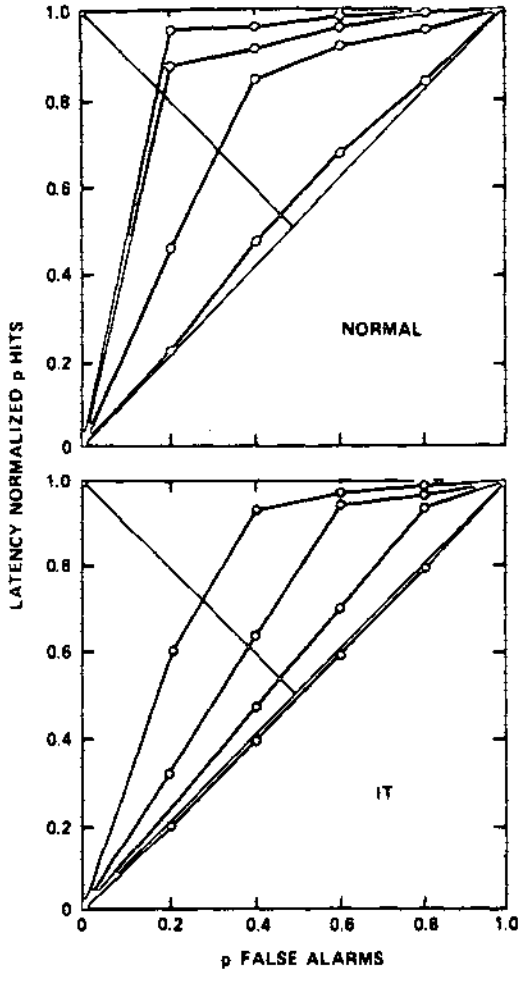

Figure 4. Combined plot of ROC functions shown in Figure 3, demonstrating the asymmetry of distribution of points. (The distribution for the IT monkeys is skewed more than that of the controls, which indicates an even greater difference in variance between signal and noise distributions. ROC = Receiver Operating Characteristics; IT = inferotemporal cortex.)

toward inappropriate go responses which would be confounded in turn with any deficit in stimulus detection. Thus, the number of rewards was maximized by producing the appropriate go or no-go response on the appropriate go or no-go trial, a procedure that has the advantage of producing more accurate measures of reaction time and, together with pretraining to a criterion of asymptotic performance and the self-pacing procedure, of minimizing the possibility that the deficit of the IT monkeys was due to some overall failure in attention.

Rather, as can be seen from Table 2, IT subjects were less efficient than their controls in restraining their tendency to "go" in the absence of a signal. This loss in efficiency could possibly account for the inferior d' values obtained in this experiment. To eliminate this possible source of confounding bias and detection, we next trained the monkeys in a task designed to elicit an overt response on *every* trial: a two-alternative (left-right forced-choice) TSD procedure. There were two simultaneous presentations on each trial: One contained the signal; the other, noise. Subjects indicated where the signal appeared by pressing the appropriate panel. During the course of one trial, the signal (green circle) might appear either on the right or on the left panel.

This pilot experiment was undertaken in order to develop an appropriate left-right TSD procedure and also to test whether a higher intensity signal would overcome the inferior detection (d') of the IT subjects shown in Experiment 1. Thus, five intensities (luminance level determined as noted in Apparatus in Experiment 1) of the green stimulus (15, 6, 5, 4, 3) were presented 20 times each in random sequences during a test session. To simplify the procedure, we eliminated the probability factor, and the signal appeared equally often on the right- and left-hand displays. No bias to respond to either side was assumed, and none was found. The dependent variable was percentage correct when the green circle appeared on the left side.

Table 5 shows the results obtained in this

Table 5
Mean Percentage of Correct Responses in Pilot Experiment With Corresponding d′ for Two-Alternative Forced Choice

	Intensity level				
Group	15	6	5	4	3
Normal					
%	100.	99.0	94.8*	83.3*	55.0
d'	8.49	6.59	4.61	2.74	.40
IT					
%	99.8	96.0	86.8	72.2	54.0
d'	8.15	4.94	3.16	1.67	.28

Note. IT = inferotemporal cortex.
* $p < .01$.

pilot study. The percentages correct at intensity 5 and intensity 4 were significantly higher for the normal than for the IT subjects (Mann-Whitney, $p < .01$). The percentage correct at intensity 6 was marginally significantly higher for the normal subjects ($p < .10$). There was no significant difference between the normal and the IT subjects at the highest intensity (15) or at the lowest intensity level (3) where both groups were performing at chance. Also evident in the data is a monotonic decrease in percentage correct as stimulus intensity decreases.

The results of this pilot experiment are thus similar to those obtained in Experiment 1, even though withholding a response was not required. Further, IT subjects do overcome their deficit in detection at the highest intensity level, which suggests that the defect is not an absolute one.

Experiment 2

Method

Subjects and lesions. Experiment 2 was undertaken in order to allow a complete ROC analysis while using the procedure adopted in the pilot left-right forced-choice experiment. To this end, eight additional preadolescent rhesus monkeys served as subjects. The inferotemporal cortex was removed bilaterally from four of these animals. The remaining four monkeys served as controls. All had been used in a previous study; details of the surgery and of the anatomical verification of the lesions have been described (Bagshaw, Mackworth, & Pribram, 1970).

Design. The experimental design remained the same as in the pilot left-right forced-choice experiment except that now the location of the green circle on the right- and left-hand panels was manipulated according to a probability schedule: In addition to the 50% probability condition used in the pilot study, a 90% and a 10% condition at each intensity level were also included in the design. Reaction time data were also collected in order to construct ROC curves based on the normalized RT latency of hits and false alarms.

Procedure. The procedure for pretraining the subjects to asymptotic performance was similar to that used in Experiment 1. Now, however, pressing a red bottom panel invariably produced a bright green stimulus in one of the two center panels (the end panels of the four-panel row were unused) just above the bottom row. The green stimulus appeared in pseudorandom order in the left and right center positions and were initially distributed equally (50% right, 50% left). The monkeys were trained to press on every trial. A press of the green panel invariably produced a reward and another trial 5 sec later; a press of the other panel (unlit) produced no reward but did turn off the green stimulus and gave rise to a new trial, also in 5 sec. (Pressing any other than the two center panels in the row above the bottom was recorded but had no effect on the trial.) At the end of this pretraining period, all monkeys were responding at the 90% criterion to the pertinent panels, with short latencies.

To minimize any overall effects of changes, for two test days after pretraining, we followed the same procedures used during the last five pretraining test sessions except that now the intensity of the green stimulus was randomly varied among five different intensities, each intensity being presented for 25 trials daily. The intensity values used were 9, 6, 5, 3, and 0, determined as described in Apparatus for Experiment 1.

The following 24 days of testing provided the experimental data. Behavioral testing was performed as in Experiment 1 and consisted of randomly presenting three intensities of the green stimulus (6, 5, and 3) under three different a priori probabilities of stimulus position. On each of these 24 days, each green stimulus intensity occurred on 20 trials according to a restricted randomization schedule. For half of the subjects, on Days 1–8 the green stimulus appeared randomly on the right in 90% of the daily 100 trials, on Days 9–16, 10%, and on Days 17–24, 50%. For the remaining subjects, the green stimulus appeared on the left with the same probabilities.

Results

Choice data. The results for intensity levels 9 and 0 failed in all instances to differentiate between the operated and the control groups. Figure 5 shows this for the probability hits at the 50% probability level. The three intermediate intensity levels were therefore chosen for presentation. Table 6 presents d' and β for the intermediate intensity levels and different probability conditions for the combined data of normals

Table 6
d' and β Over All Conditions in Experiment 2

Intensity level	Condition	Normal		IT	
		d'	β	d'	β
6	90%	4.31	.16	3.77	.95
	50%	6.03**	.02	4.94	462.25
	10%	7.40**	1.00	4.47	741.50
5	90%	3.69*	.02	2.77	.43
	50%	2.70	.30	2.39	1.63
	10%	4.67	623.00	2.65	7.77
3	90%	.63	.62	.52	.71
	50%	.21	.92	.15	1.04
	10%	.86	2.32	.36	1.61

Note. IT = inferotemporal cortex.
* $p < .08$. ** $p < .01$.

Table 7
Mean Hit and False Alarm Rates Over All Conditions in Experiment 2

Intensity level	Group	Measure	Probability schedule 90%	50%	10%
6	Normal	Hit	.995	1.000	1.000
		FA	.042	.010	0
	IT	Hit	.971	.892	.781
		FA	.031	0	0
5	Normal	Hit	.998	.963	.833
		FA	.208	.181	0
	IT	Hit	.953	.839	.625
		FA	.137	.081	.010
3	Normal	Hit	.868	.696	.292
		FA	.688	.622	.079
	IT	Hit	.821	.417	.125
		FA	.657	.358	.066

Note. FA = false alarm; IT = inferotemporal cortex.

and ITs. When the normal and IT groups were compared, d' was significantly different at intensity 6 for the 50% and 10% conditions and at intensity 5 for the 10% condition ($p < .01$). The other d' differences were not statistically significant.

This finding is in agreement with the results at the lowest intensity in the pilot experiment. It appears that only at the intermediate intensity levels does the normal group exhibit a reliable superiority over the groups with IT lesions. This superiority could be dependent on fairly small differences in the proportion of hits and false alarms. However, the result obtained in this experiment is consonant with that obtained in Experiment 1 in which a somewhat greater difference in d' was obtained, and with the results of other experiments in which physical stimulus differences are parametrically varied (e.g., Mishkin & Hall, 1955) and differences between groups appear only in the intermediate ranges of stimulus differences.

Table 7 shows the hit and false alarm rates for all probability conditions at the intermediate intensity ranges. Figure 5 shows the distribution of hits of all animals for all intensities at 50% a priori probability of reinforcement. It can be seen from this figure and this table that there is a monotonic increase in hit rate with intensity and in false alarm rate as the probability of a signal occurring in the designated panel increases. Therefore, ROC curves were constructed for these data.

Figure 6 shows the separate ROC curves at intermediate intensity levels 3, 5, and 6. The hit and false alarm axes are marked off in terms of z scores according to the procedures outlined in Egan (1975, p. 61). On such a linear deviate scale, the data points would fall along a straight line if the assumption of normality for the signal and noise distributions were true. The theoretical straight line for a particular d' that comes close to fitting the data is sketched in on each ROC graph.

From both the results in the table and the ROC curves it can be seen that d' decreases as the intensity level decreases. As in Experiment 1 and the pilot to this experiment, this confirms the expectation that as the intensity level of the green light decreases, stimulus detection becomes more difficult for both groups and is manifest earlier in the IT subjects.

RT data. The mean response latencies produced by the normal and IT groups for each stimulus intensity/probability combination are shown in Table 8.

As in the previous two experiments, a re-

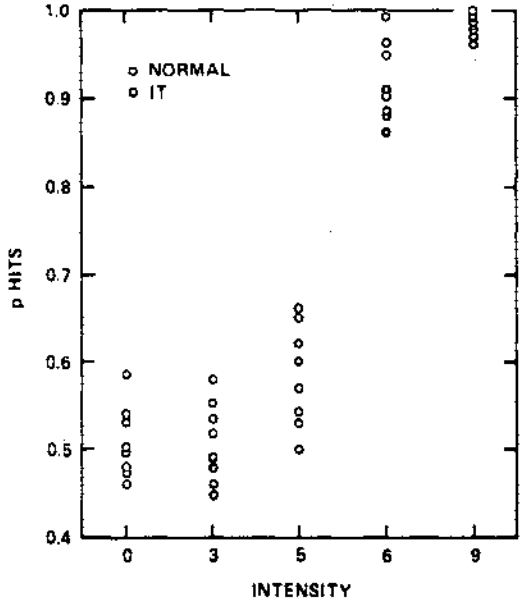

Figure 5. Graph of the probability of hits made by each monkey at each intensity at 50% a priori probability of reinforcement. (IT = inferotemporal cortex.)

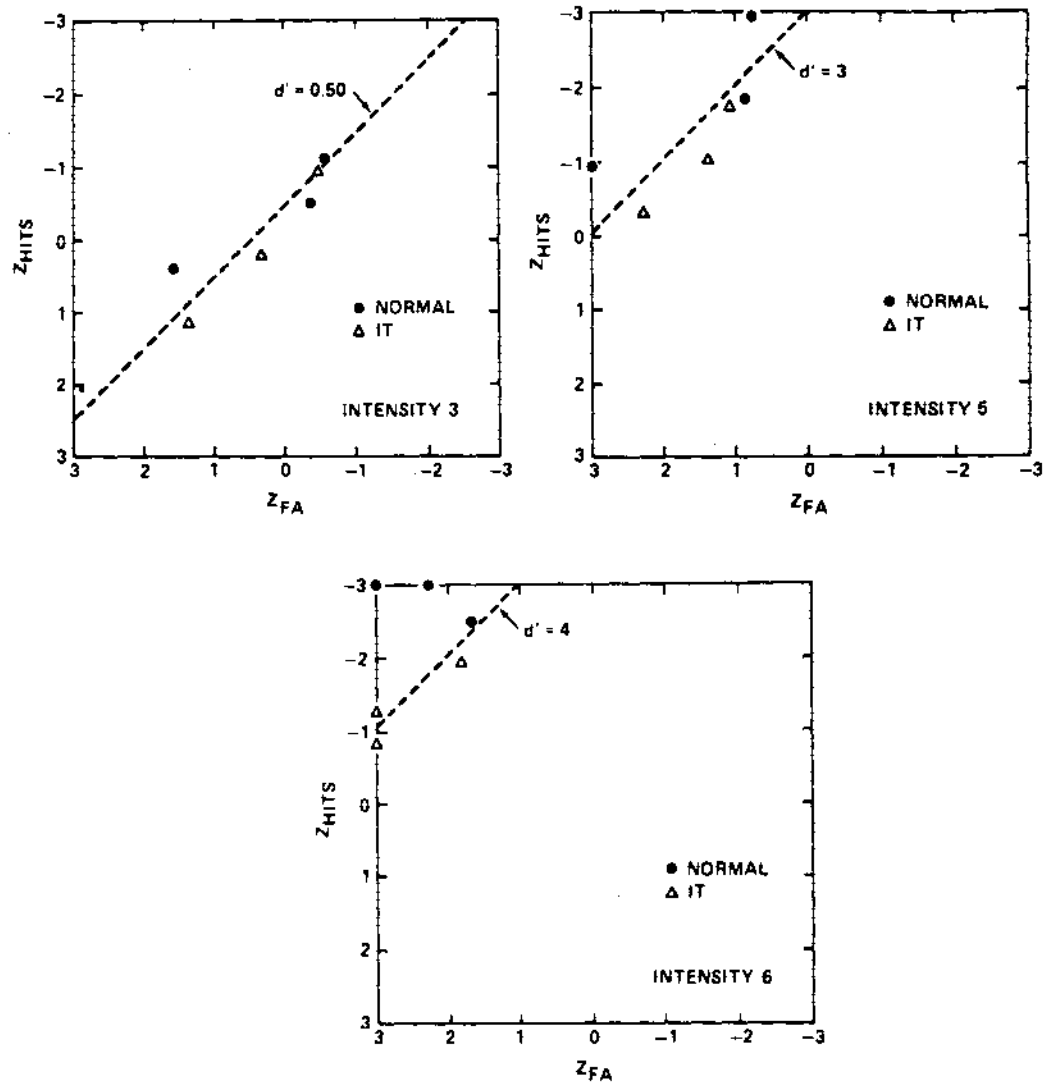

Figure 6. d' curves for normal and IT animals at intensities 3, 5, and 6 in Experiment 2. (Dashed line is the theoretical curve for $d' = .50$. IT = inferotemporal cortex; FA = false alarm.)

peated-measures ANOVA was used to assess any differences in correct trial latencies. The factors were group (normal, IT), intermediate intensity level (6, 5, 3), and probability level (90%, 50%, 10%), with the repeated measures occurring over the stimulus intensity levels and the a priori probability conditions. Once again, no significant differences between groups was obtained when averaged over all levels of intensity and a priori probability.

However, the analysis indicated that the differences in correct response latencies produced at different stimulus intensities were reliable, $F(2, 8) = 4.83$, $p < .05$. The Intensity × Group interaction was significant, $F(2, 8) = 7.86$; $p < .025$, as was the Probability × Group interaction, $F(2, 8) = 17.72$, $p < .01$. The Group × Intensity × Probability interaction was also significant, $F(8, 24) = 3.24$, $p < .01$.

The triple interaction is illustrated in Figure 7. Note that there is a monotonic decrease in RT under *decreasing* probability conditions for the IT group. This finding makes it unlikely that the results obtained

Table 8
Mean Correct Reaction Time Over All Conditions in Experiment 2

Intensity level	Group	Probability schedule 90%	50%	10%
6	Normal	.784	.879	.871
	IT	.862	.715	.625
5	Normal	.805	1.071	.958
	IT	.884	.892	.684
4	Normal	.892	1.479	1.348
	IT	.973	.727	.619

Note. Each point in this table represents the combined data for the four test sessions administered at each intensity condition. IT = inferotemporal cortex.

in this experiment or in Experiment 1 are due solely to the increased tendency toward false alarms of the IT group obtained in both experiments.

As was found in Experiment 1, and confirmed here by the significant Intensity × Group interaction, the normal subjects were more able to respond appropriately to differences in intensity level than were the IT monkeys. Changes in intensity made little impact on the RTs of the ITs. Also, as in Experiment 1, this insensitivity accounts for the two-way and three-way interactions which in a sense are spurious since they do not reflect on the TSD assumptions when each group is taken separately.

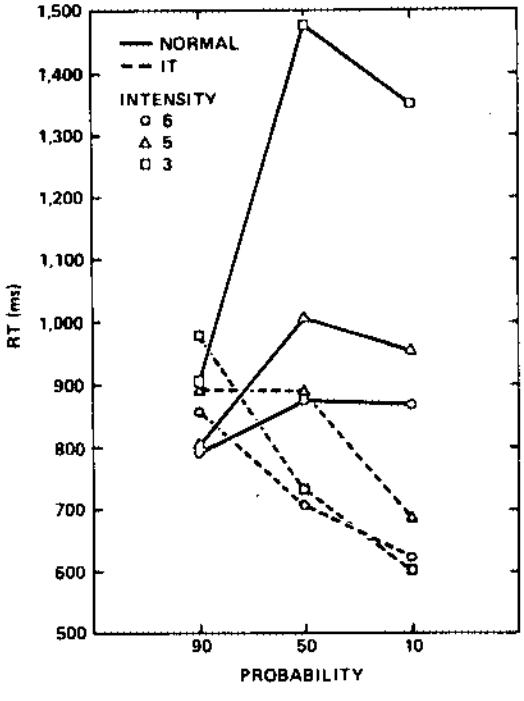

Figure 7. Probability × Intensity × Group interaction for reaction time (RT) in Experiment 2. (Mean RT for normal and IT animals is plotted as a function of stimulus intensity. For clarity, only probabilities 90%, 50%, and 10% are plotted. IT = inferotemporal cortex.)

The ROC curves shown in Figure 8 were also plotted with RT normalized data as in Experiment 1. At intensity 3, $d' = .50$ for both groups and is nearly at chance. However, at intensity level 5, the curve for the normal group is more inclined toward the upper right-hand corner of the graph than is that for the IT group. Referring back to Figure 1, we see that this indicates a greater separation between the means of the signal RT distribution. Thus, the latency data again support the choice data at intensity 5 where two of the three d' values were greater for the normal group. The detection performance of the normal group appears superior at this intensity. Notice also that detection performance, as revealed by this type of RT normalized ROC curve, improved for the IT group as intensity increased from level 3 to level 5. Thus, improved sensory performance results in a lower mean RT distribution for signal presentation than for noise presentation.

No RT ROC curves are shown at intensity 6 because there were too few false alarms at this intensity level.

General Discussion

The results of these experiments show once again that monkeys with resections of inferotemporal cortex are deficient in their response to differences in stimulus luminance. This deficiency is most clearly demonstrated in Figure 2 by the flat slopes produced by the monkeys with IT lesions in response to differences in luminance, even at suprathreshold levels. Note that in this figure, normal subjects produce perfect power slopes since the luminance ratios are essentially equal. The flat slope with an exponential near zero, means that the subjects with lesions simply did not *discriminate* one signal from another along the dimension of intensity. This finding in the

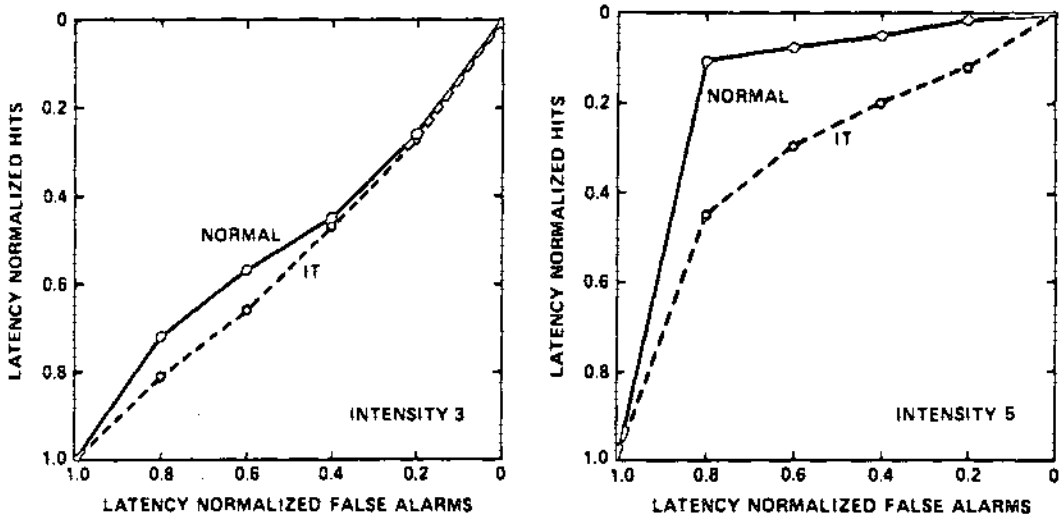

Figure 8. d'_{RT} curves for normal and IT animals at intensities 3 and 5 in Experiment 2. (IT = inferotemporal cortex.)

current study is completely consistent with those of all previous studies that have shown discrimination deficits after IT lesions (see reviews by Gross, 1973; Pribram, 1974).

A deficit in discrimination can be completely independent of threshold (d') as has been found, for instance, by McGuinness (1972). Nonetheless, the results of the present experiments using both go–no-go and right-left forced-choice procedures show a consistent albeit minimal defect in d' in monkeys with lesions of the inferotemporal cortex. Because the effect is minimal, it is of little general interest; however, it does have a bearing on the question posed at the outset of the introduction to this article. In keeping with the results obtained in humans (Bay, 1953), a small but significant change in sensitivity often accompanies lesions of intrinsic "association" cortex even when the primary sensory projection systems remain undamaged.

Our findings appear to contradict those reported by Bender (1973), but this is an apparent contradiction only. Despite Bender's disclaimer, his lesions did in fact have an effect, also minimal, which was opposite in sign to that found in the current study. Perhaps this difference can be accounted for by the fact that Bender tested his monkeys under scotopic conditions whereas our monkeys were run phototopically. Thus, Bender's two monkeys with IT lesions (essentially similar to those studied in the present report) and two with foveal prestriate resections showed a slightly enhanced scotopic sensitivity, whereas we have shown that IT lesions produce a slightly decreased phototopic sensitivity.

It should be emphasized once again that, though present, neither effect is large and certainly neither is sufficient to account for the marked change in discriminative capacity that follows IT (and to a lesser extent, foveal prestriate) lesions. The question thus arises as to the possible meaning of this small change in sensitivity. As indicated by the ROC analysis (see end of Results for Experiment 1 and Figure 4), the IT lesion appears to make the monkeys less efficient and more sensitive to noise. That this inefficiency may be due to an inability to suppress irrelevant aspects of the visual environment is borne out by the results of another study (Bagshaw et al., 1970) performed on the same group of monkeys in which eye movements were recorded. In that experiment, the monkeys with IT lesions cast their gaze much more often than did the controls to irrelevant parts of the scene.

One explanation for the consistent minimal shift in sensitivity after intrinsic "association" area lesions such as those of the IT cortex can be suggested in terms of the ef-

ferent hypothesis of IT function. This hypothesis (Pribram, 1958, 1960b, 1971; Spinelli & Pribram, 1966, 1967) proposes that the visual effects of IT cortex manipulations are due to corticofugal influences on processing in primary visual systems. In other modalities (e.g., audition, Dewson, 1968; somesthesis, Granit, 1955), such efferent controls have been found to be primarily sensitivity adjusting mechanisms. Perhaps one of the main influences of IT cortex is to regulate photopic vision based in large part on foveal processes (Gross, 1973). Hence, if IT lesions impair contrast thresholds in foveal vision, a slight shift toward a scotopic sensitivity could result.

Finally, the problem of the brain mechanisms involved in response bias (β) has been addressed by this set of experiments. Resection of IT cortex rendered the monkeys less able to withhold their responses to the unreinforced cue when the probability of occurrence of a stimulus was altered. The increase in false alarms was manifest in both the go–no-go and the right-left TSD procedures. This result is in marked contrast to that obtained after resection of the limbic formations (amygdala and hippocampus) on the inner medial portion of the temporal lobe. After such resections monkeys are strongly biased to go to the rewarded cue and as a result manifest a higher criterion and an apparent lowered motivation to respond (Pribram, Douglas, & Pribram, 1969; Spevack & Pribram, 1973). Furthermore, monkeys with limbic lesions show no defect in discrimination performance or in detection (d'), which enhances the validity of finding such deficits in the current study.

The question remains whether the severe difficulty in making discriminations in the visual mode, which is experienced by monkeys with lesions of the inferotemporal cortex, is related to this change in criterion and, if so, which is the primary deficit.

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