SOME EFFECTS OF POSTERIOR PARIETAL AND DORSOLATERAL FRONTAL CORTICAL LESIONS ON LEARNING AND PERFORMANCE OF SPACE-PERCEPTION TASKS UNDER VISUAL GUIDANCE IN MONKEYS AND RATS.

Thesis presented for the degree of Master of Science at the University of St. Andrews.

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DECLARATION

I declare that this thesis was composed entirely by myself. The research, the results of which appear in this thesis, was carried out by myself except where collaboration was necessary as indicated in the text.

Neither this thesis nor any other representation of the research carried out has been accepted in fulfilment of the requirements of any other degree or professional qualification.

SUMMARY

A review of the clinical effects of damage in the posterior parietal area of the brain was given followed by a comparative anatomical and psychological survey of the problem of definition of association cortex.

Five experiments with 12 Macaca arctoides as subjects were described. Four of the monkeys were given bilateral posterior parietal cortical lesions and 4 bilateral dorsolateral frontal cortical lesions. The remaining 4 monkeys served as unoperated controls. The 5 experiments were designed to measure performance on aspects of visual perception of spatial relationships. All tasks were presented in a Wisconsin General Testing Apparatus. It was predicted from human and other animal studies that parietal monkeys would be impaired on tasks of this type.

Parietal monkeys were impaired only on a stylus maze task and control tests failed to eliminate the possibility that this may have been due to sensory motor incoordination rather than a higher order perceptual defect.

The failure of the present study to demonstrate parietal impairment on stimulus-response separation tasks or tasks involving completion of fragmented patterns was attributed to the initial training given and it was suggested that all monkeys had learned to orient to a relevant stimulus regardless of position.

On a task involving grouping of lines by proximity in two groups of two, it was demonstrated that results can be misleading and individual animals use different cues to learn the same discrimination.

Dorsolateral frontal monkeys were shown to have generally shorter response

latencies on two choice visual discrimination problems. It was suggested that this was due to a disturbance of response inhibition.

An attempt was made to replicate the anomolous transfer effect in rats and to affect the organization of dot arrays presented visually, by lesions in prestriate cortex. The anomolous transfer effect was shown to be less reliable than has been hitherto reported and the possibility that slight overtraining or strain differences may affect it was raised.

Attention was drawn to the anatomical connections of superior colliculus and pulvinar nucleus and some behavioural consequences of superior collicular ablation were mentioned. The possibility that the superior colliculus and the posterior parietal cortex may be jointly involved in spatial perception was put forward. It was suggested that the deficit described by some following posterior parietal ablation in monkeys may be due to selective attention loss.

The question of the usefulness of lesion experiments to psychology at the present stage of physiological knowledge was raised.

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CHAPTER I INTRODUCTION.

1. BRIEF REVIEW of the CLINICAL LITERATURE

Clinical disturbances in perception of spatial relationships and in spatial orientation after brain damage have been reported since early this century. Holmes in 1918 described symptoms of "visual disorientation" ("an affection of the power of localizing the position in space and the distance of objects by sight alone") following damage to the posterior parietal region of the brain. Kleist (1912) and Pötzl (1928) also described similar syndromes, their terms for which have been translated respectively as "optic apraxia" and "geometric optic agnosia".

More recent case reports of patients with posterior parietal damage include descriptions of symptoms not confined to the visual sense modality but none the less related to space perception. There has been little agreement as to the correct interpretation of these changes in spatial orientation and hence on a meaningful nomenclature but the symptoms generally manifested by human patients after damage to the parieto-occipital region are well documented and will now be enumerated as follows:

(i) Constructional apraxia

(ii) Left-right disorientation

(iii) Loss of topographical orientation

(iv) Difficulty in finding the relative positions of things to one another and to the self

(v) Unilateral visual neglect (not associated with hemianopia)

(vi) Denial and neglect of limbs

(vii) Finger agnosia

(viii) Dressing apraxia

(ix) Loss of visual imagery

(x) Upset of body image appreciation

The diverse explanations put forward for the mechanism or mechanisms underlying these symptoms have as their basis the alternative views that the symptoms can all be explained by sensory changes, or that a complex integrative function is involved. These two extreme views have arisen from the classical view of there being two distinct levels of organization of sensory input in the brain; sensation in the primary projection fields and elaboration of sensation and hence discrimination in the anatomically adjacent associative areas. Historically agnosias (that is deficiencies in recognition not explainable by sensory changes) have been assumed to follow destruction of these associative zones. However anatomical, physiological and psychological evidence collected over the last twenty or thirty years has indicated the inaccuracy of the assumption of a sensory-associative dichotomy hence indicating that neither of the extreme views in the debate over the nature of the parietal impairment is well grounded.

There is considerable evidence that functions mediated by several sense modalities are impaired by the same lesion in the posterior parietal region in man. Semmes, Weinstein, Ghent and Teuber (1955 and 1963) found subjects with parietal damage impaired on performance of a map following task when presented either visually or tactually and hence concluded that the parietal deficit was not due to disturbance

of a high level visual function. In their 1955 paper, Semmes et al. were also critical of the use of the terms agnosia and apraxia for the complex of symptoms classically associated with parietal damage and in 1963 they suggested that a general inattention to background features of the environment may underly all the difficulties encountered by the patient with parietal damage. Ettlinger, Warrington and Zangwill (1957) favoured keeping the term "visuo-spatial agnosia", despite the non-specifically visual nature of the defect, for want of a more explicit term. Ettlinger, Warrington and Zangwill were particularly critical of Denny-Brown's term "amorphosynthesis" and its implications. The visual constructive disorder following parietal lesions could not according to Ettlinger, Warrington and Zangwill be wholly attributable to unilateral neglect (Denny-Brown states that defect in recognition of form, which is the function of the parietal cortex, is the "basis of unawareness of part of extrapersonal space" (Denny-Brown, Meyer and Horenstein 1952)) since the constructive disorder is still there when stimuli are presented tachistoscopically to the intact visual half field.

The definition of the deficit following parietal lesions in man is further complicated by evidence of the nature of the impairment differing according to whether the right or left hemisphere or both are involved. There is considerable support for the importance of the right (minor) posterior parietal cortex (assuming the oversimplified case of cerebral asymmetry with language represented on the left "dominant" side). Whitty and Newcombe (1965) reported six cases with right sided posterior parietal damage who showed spatial disorientation,

constructional apraxia, loss of topology and distance judgement. Piercy, Hécaen and Ajuriaguerra had previously reported in 1960 significantly more common constructional apraxia after right than after left sided lesions and suggested that the functions impaired in constructional apraxics are more fully organized in the right hemisphere than in the left. Teuber (1965) also favoured the explanation that a different mode of organization in the two posterior parietal areas gives rise to the asymmetry of lesion effects. Teuber suggested that the more diffuse representation of some functions in the right parietal area than in the left may give rise to the complex of impairments which follow large right sided posterior parietal lesions.

Right posterior lesions impaired the tactual maze of Corkin (1965) and the visual maze of Milner (1965) and while there was no clear distinction between anterior and posterior damage on the perceptual maze tasks (visually presented) of Benton, Elithorn, Fogel and Kerr (1963) and Elithorn (1964), patients with right sided brain damage found these tasks harder than did those with left sided damage. Humphrey and Zangwill in 1952 reported a case of a left handed man with right sided occipitoparietal damage who showed some symptoms usually attributed to left and right unilateral lesions but not the usual bilateral picture. This case was more interesting when it was reported than it is now since it pointed to a difference in the localization of function in the two hemispheres in a left handed man from a right handed one and we now have considerable evidence that these differences usually exist.

The concept of "extrapersonal space" has already been mentioned

in association with Denny-Brown's definition of the parietal impairment. The question of a personal/extrapersonal space dissociation was investigated by Semmes, Weinstein, Ghent and Teuber in 1963 and some evidence for the anatomical basis of such a dichotomy was advanced. Two tests were given to 76 brain damaged war veterans and 32 controls with peripheral nerve damage. There was a test of personal orientation which involved touching named parts of the body and an extrapersonal test which required the subject to walk through paths in a room indicated on maps which he held in constant orientation to his body (hence varying orientation to the testing room). Partial dissociation of anterior and posterior lesioned patients was found on these two tests; left anterior, right anterior and left posterior(paradoxically not bilateral anterior) patients were impaired on the bodily orientation test whereas left posterior and bilateral posterior patients were severely impaired on the extrapersonal orientation test.

2. SOME COMPARATIVE ANATOMICAL CONSIDERATIONS.

It was mentioned in the preceeding section that evidence from human studies points away from the existence of anatomically discrete association areas in the cortex which serve to elaborate information received by the adjacent primary sensory areas. In 1953 Chow and Hutt wrote a criticism of the methods used to define "association" areas in the rhesus monkey and concluded that a revision of the whole concept of association cortex was needed since as a physiological entity it was unrealistic in view of the general trend of failure to demonstrate a dichotomy of integrative and receptive functions

separating association cortex from adjacent projection areas.

The early anatomical studies of the so called association pathways in primates and other animals are however none the less valuable in the light of the evidence against the sensory-association dichotomy and some of the results of these will be considered in this section.

There have been few recent studies of the anatomy of rat association cortex, much of our present knowledge still being the results of the classical work of Lashley and Krieg in the first half of this century. Lashley (1941) identified a pathway from nucleus lateralis posterior of the thalamus to a region on the lateral cerebral cortex anterior to the striate area, corresponding with Krieg's areas 7 and 39 (Krieg 1946).

Lund also more recently (Lund 1964a) found evidence of posterior association cortex in the rat (Lashley and Frank (1934) doubted its existence on behavioural grounds). Lund found projections from parts of the cortex designated by Krieg as visual cortex to deeper layers of the superior colliculus than those to which the striate area projects (Lund 1964b), hence showing a similarity between this area in the rat and area 7 in the monkey. In 1954, Nauta and Bucher published results of a study of cortico-cortical connections of the visual cortex in the rat and reported fibres from area 17 to 18a only, with additional tangential intracortical fibres joining 17, 18, 18a and other parts of 17. Nauta and Bucher referred to Krieg's cortical map, and if, as Lund's more recent study indicated, part of Krieg's area 17 is really homologous with area 7 in the higher mammals, this may be an indication of connections between area 17

and area 7 which is a part of the posterior "association" area.

Apart from the very few studies of the rat and the wealth of studies of the monkey which will be discussed shortly, there have been studies in the cat stressing the subcortical connections between the superior colliculus and the pulvinar nucleus of the thalamus (the importance of this nucleus will become clearer later). Altman and Carpenter (1961) reported ipsilateral projections from the superior colliculus to the caudal part of the pulvinar identified by Nauta and Gygax staining, and a review by Meikle and Sprague in 1964 pointed out the presence of a reciprocal pathway between pulvinar and superior colliculus in the cat. A study by Lyubimov in 1965 of six dogs also indicated a fibre connection between superior colliculus and pulvinar nucleus in this species.

An interesting evolutionary approach was made in a series of studies of the tree shrew (Tupaia glis) by Diamond and coworkers at Duke University. These were lesion studies of the visual system with associated anatomical and behavioural observations. This species, while showing many of the characteristics of the very primitive mammalian order Insectivora, is often classified in the primate suborder Lemuroidea. Snyder and Diamond (1968) proposed that the greater role played by the extrastriate visual system in the tree shrew than in the rat, cat or monkey was based on the different connections of the pulvinar (acting as a relay for all input to extrastriate (but sensory) belt cortex) in this species from the more familiar mammalian forms (Killacky, Diamond, Hall and Hudgins 1968). In 1969 Diamond and Hall published a comparative discussion of neocortical evolution based partly on the experiments

of their group and they concluded that after the arboreal insectivore stock, cortical evolution diverged and has converged again resulting in the similarities in modern forms between, for example, the tree shrew (a primitive primate) and the squirrel, which have both evolved extrastriate cortical visual sensory areas along separate paths from a common insectivorous ancestor.

To return now to anatomical considerations of the posterior "association" cortical areas of monkeys. Again, many important studies were carried out several decades ago. In 1935 Mettler identified corticofugal fibres by the Marchi technique for staining degenerating nerve fibres from various dorsolateral loci in the posterior cortex, although his lesions unfortunately did not include the tissue in the sulci. Apart from the extensive connections of the pulvinar, Mettler found pathways between the occipitoparietal, occipitotemporal, posterior superior parietal, angular and the upper parts of the superior and middle temporal gyri and the "eye nuclei" (the interstitial, occulomotor, trochlear and abducens nuclei). The pathways Mettler reported between the angular, occipitoparietal and occipitotemporal gyri and the lateral geniculate nucleus may be attributable to the subcortical damage made with these lesions.

A little more recently in 1942, Peele made a study of the distribution of efferent fibres, also using the Marchi method. Most of the parieto-thalamic fibres from areas 5 and 7 went to the nucleus lateralis posterior, the nucleus medialis dorsalis also receiving some fibres from area 5 while the pretectal area received from 5 and 7 and the superior colliculus from area 7. Chow published a more complex retrograde cell degeneration study of the cortical

projection field of the pulvinar nucleus (Chow 1950). He identified connections between the prestriate and posterior parietal cortical regions and most parts of the pulvinar but with an orderly spatial arrangement of the various cortical divisions' representation in the pulvinar. Chow was however particularly critical of the Marchi method in a review he wrote three years later (Chow and Hutt 1953), since it is impossible to tell whether efferent or afferent degenerating fibres are stained.

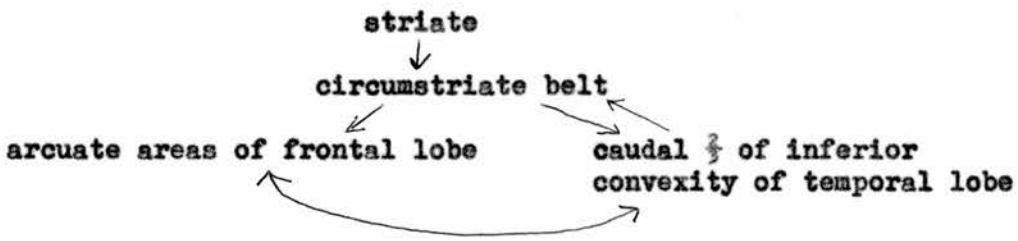
Peele's finding of connections between parietal cortex and superior colliculus was given some support in 1963 by Ebner who found degeneration in the stratum intermedium of the superior colliculus by the Nauta technique after complete parietal lobe removal in monkeys. Cragg (1969) in another study using the Nauta technique reported that the lateral geniculate nucleus does not project outside the striate area and while the pulvinar projects to areas 18 and 19, and 17 projects to 18, there was no established connection between area 17 and the prestriate area 19.

The earliest studies already mentioned, (Mettler 1935, Peele 1942), reported pathways from one part of the parietal cortex to others, particularly to adjacent areas and the homologous contralateral ones. Fibres between parietal cortex and the superior temporal gyrus were also recorded. Peele (1942) also reported degeneration in the anterior occipital gyri after ablation of areas 5 and 7 but none in the frontal lobe except the precentral gyrus after ablation of areas 1,2 and 3. Bailey, Von Bonin, Davis,Garol and McCulloch (1944) added the following pathways of interest to this study from the results of physiological neuronography: from area 39 to just

above the posterior part of the sulcus principalis, and from area 18 to just above the supero-anterior extremity of the arcuate sulcus.

In 1949, Petr, Holden and Jirout were able to surgically explore the ventral surface of the brain and apply strichnine to the third temporal gyrus. The resultant firing was in an area of parietal cortex above and below the intraparietal sulcus in an area closely corresponding to the lateral extent of the posterior parietal lesion experimentally produced in monkeys and associated with behavioural deficits such as misreaching (to be discussed in the next section). Strichnine on the sulcus principalis and angular gyrus also produced firing in parietal cortes, the former apparently a demonstration of a reciprocal pathway to that found by Bailey et al. (1944). Chow and Hutt (1953) also criticised the method of physiological neuronography employed by Bailey et al. and Petr, Holden and Jirout on the grounds that the effects of strichnine were not fully understood.

More recent studies of cortico-cortical connections include those of Kuypers, Szwarcbart, Mishkin and Rosvold (1965), Pandya and Kuypers (1969) and Zeki (1971). Kuypers et al. found evidence for connections between the banks of the lunate sulcus and the caudal bank of superior temporal sulcus (a similar pattern of degeneration was found as after striate lesions) and between the inferior temporal convexity and parts of the circumstriate cortical belt and prefrontal cortex. Kuypers et al. suggested the following scheme:



The direct projection from the circumstriate belt to the inferior temporal area was later denied by Zeki (1971), although Mishkin's work (1966) supports the existence of this pathway as a diffuse projection system.

Pandya and Kuypers (1969) brought the purely anatomical results back into the realm of psychology and probably oversimplified the situation by stating that somatosensory, auditory and visual cortical areas abut in the inferior parietal lobule with visual projections dominating. They also stressed the convergence in the premotor area and the reciprocal connections between this area and the cingulate gyrus. Hence the possible importance of the cingulate gyrus in connection with the integrative function of the parietal cortex was suggested.

To summarize the current anatomical situation of the posterior parietal cortex in the monkey: the main subcortical connections are with pulvinar nucleus in the thalamus. Intracortical connections are within the parietal area and via the corpus callosum to the contralateral parietal cortex, also to the frontal lobe above the sulcus principalis. The prestriate area probably also acts as a relay station from striate cortex to inferotemporal cortex.

3. COMPARATIVE PSYCHOLOGICAL REVIEW.

Attempts to produce experimentally behavioural changes in the lower primates to parallel those found in man with posterior parietal lesions have produced varied results with very few consistent findings to indicate that this area in the monkey does serve an essential

function in normal behaviour.

An early behavioural analysis following bilateral posterior parietal lesions in the monkey was published by Jacobsen in 1936. He had observed a difficulty in grasping food and in tactile recognition which he thought was reduced under visual guidance (contrary to what would be predicted if this deficit mimicked the clinical picture). In what seems now an obviously simplistic explanation of his results, Jacobsen assumed that his lesion had removed a specialized association area which normally controlled complex integrative function.

An impairment of reaching was also reported by Peele in 1944 and Peele went further to say that ablation of area 5 in the monkey impaired reaching except under visual guidance, whereas ablation of the part of area 7 which lies on the lateral surface caused overshooting with visual guidance. Fleming and Crosby (1955) also reported that unilateral ablation of area 5 resulted in contralateral impairment of grasping and placing which was most marked when the animal was blindfolded. Such localization of function has not been found generally but the misreaching phenomenon has been found to be a reliable characteristic of the larger monkey posterior parietal lesions (for example Blum, Chow and Pribram 1950, Bates and Ettlinger 1960, Ettlinger and Kalsbeck 1962, and Moffett, Ettlinger, Morton and Piercy 1967). In the Moffett et al. 1967 paper, no small area was found to be critical for accurate reaching but a regional gradient was found. Drewe, Ettlinger, Milner and Passingham (1970) discussed the reaching impairment produced in monkeys by parietal cortex ablation in relation to human difficulties after parietal lesions.

Other functions which have been reliably affected after posterior

parietal lesions in monkeys have been concerned with somatic sensation or learning based on somaesthesis. Jacobsen (1936) found defect in tactile sensation. Ruch, Fulton and German (1938) failed to demonstrate any defect in weight or roughness discrimination after bilateral posterior parietal cortex ablation in monkeys, but more recent studies have produced positive results. Peele (1944) found an inability to recognize food by touch after area 7 lesions. Blum (1951), Ettlinger and Kalsbeck (1962), M.Wilson (1965) and Ettlinger, Morton and Moffett (1966) found deficit on tactile discrimination problems after posterior parietal lesions and Moffett, Ettlinger, Morton and Piercy (1967) identified a critical region for tactile discrimination in the intraparietal sulcus along the border of areas 7 and 5 with area 2. M.Wilson (1965) suggested that tactile information could not be fully utilized in the absence of posterior parietal cortex, but Ettlinger, Morton and Moffett (1966) suggested that the posterior parietal tactile defect may be secondary to selective motor retardation.

Reports of an orientation deficit in monkeys which appears to be similar to that observed in humans came from Ettlinger and Wegener (1958), Bates and Ettlinger (1960) and Blum, Chow and Pribram (1950) (in two monkeys with large parieto-temporo-preoccipital lesions), in the form of an inability to find the home cage.

Other more clearly visual functions affected by parietal lesions are less clear. The lesions experimentally produced to investigate these functions fall into two categories: those confined to the preoccipital belt essentially attempting to interrupt cortico-cortical fibres from the striate region, and the larger lesions sometimes even including some temporal cortex but usually referred

to as posterior parietal lesions and which as Bates and Ettlinger (1960) pointed out should be called "superior parieto preoccipital". Some of the experiments used parietal lesions differing in some respects from those used by Ettlinger and his coworkers, notably in the amount (if any) of tissue removed from the medial surface.

In a summary of a series of experiments, Wm.A.Wilson Jr.(1965) wrote that posterior parietal cynomolgous monkeys were deficient on visual problems. Loss of colour and pattern perception were reported by Chow in 1952 in one monkey but the lesion in this animal destroyed much of the visual field (as shown by the considerable degeneration in the lateral geniculate nucleus). Pasik, Pasik, Battersby and Bender (1958) failed to find an effect of posterior parietal ablation alone in visual discrimination although their results were complicated by combining different lesions in the same animal.

Smaller prestriate lesions have often failed to produce any observed changes in visual behaviour (e.g. Lashley 1948, Evarts 1952), suggesting that prestriate cortex is not a necessary relay from the striate cortex to other cortical visual areas but that subcortical pathways are important. It was however suggested by Mishkin in 1966 that "the negative or negligible effects of prestriate lesions can be traced to a failure to disrupt completely the critical striate-inferotemporal interaction" and that "the fact that an impairment can be produced by a sufficiently extensive ablation favours the view that the prestriate region is an essential part of the system". A consideration of prestriate lesion experiments is not directly relevant since the area has been investigated for the part it plays in linking the striate and inferotemporal areas in visual discrimination and

not with reference to spatial perception. However the posterior parietal area includes part of the prestriate belt so the results of prestriate lesions must be considered. Positive findings have been reported by Ades and his coworkers (Ades 1946, Ades and Raab 1949, Riopelle and Ades 1953) and Zeki (1967) who suggested impairment of information processing after circumstriate lesion since he was able to demonstrate transfer of learned visual information from an intact hemisphere to a contralateral circumstriate lesioned hemisphere in the absence of corpus callosum but not vice versa. No details of the lesions were given in this paper. Meyer, Harlow and Ades (1951) suggested on the basis of the nature of the prestriate impairment found by their group that "habits applicable to the solution of a particular problem are lost but acquisitions applicable to the solution of a class of problems are spared after preoccipital ablation". Lashley (1948) criticised Ades'(1946)results and suggested that the prestriate deficit could be explained by interference of successive "dated" memories. His findings of impairment on delayed response and conditional reaction have not been supported. Partly on account of Pohl's finding (Pohl 1970) of impairment of posterior parietal (including dorsal prestriate) monkeys on landmark reversal, Mishkin (1972) proposed that prestriate cortex may be found to have a specialized function in extrafoveal vision, that is, in perception of spatial relationships.

For experiments on preoccipital function in animals other than primates, we again turn to the work of Karl Lashley. Lashley (1931) and Lashley and Frank (1932) concluded that lesions in the rat other than those which disrupt the optic radiations do not interfere

with performance of visual tasks. Lashley and Frank (1934) wrote that since lesions right around the striate area of the rat did not interfere with retention of visual tasks, either there was no visual association area functional in habits based on discrimination of simple geometric figures (comparable with the inferotemporal area in monkeys),or (which brings the rat in line with the monkey as envisaged by Mishkin (1966)), its parts were equipotential. However Kirk (1936) doubted Lashley's interpretation since he found amnesia for a harder visual discrimination task than those used by Lashley after an extensive unilateral cortical lesion independant of the striate area. Lashley was highly critical of Kirk's work (Lashley 1942) and reported a further experiment to back up his own earlier conclusion that visual cortical function in the rat was confined to the striate area. Thomas (1966 and 1970) also found evidence against Lashley's ideas of equipotentiality and in favour of the involvement of the parietal region of the brain of the rat in spatial functions. Thomas found a temporary impairment in maze performance of rats following parietal lesions.

An interesting finding related to spatial function in the rat was made by Potegal (1969). Potegal found that rats with damage to the caudate nucleus were unable to perform egocentric food localization although they could do harder tasks using relative positions of external cues. Potegal points out that the caudate in the rat is not strictly comparable with monkey or cat but the similarity between this dissociation of spatial function into egocentric and allocentric in the rat and the suggestions made by Pohl's work on the monkey (Pohl 1973) and that of Semmes et al. (1963) on man

is interesting.

The pulvinar target on the cortex of the tree shrew was ablated by Killackey, Diamond, Hall and Hudgins (1968) after which there was a deficit in visual learning. The organization of the visual system in the tree shrew is however unusual, with more detailed vision in the extrageniculostriate system than in other animals (Snyder, Hall and Diamond 1966). However Killackey (unpublished) observed that it was only under certain conditions that tree shrews could learn a horizontal-vertical discrimination in the absence of striate cortex. These animals could not abstract visual information from the surroundings and could only perform this discrimination with the stimuli alone backlit. The selective attention mechanism was apparently upset.

4. AIMS of the PRESENT STUDY.

The series of experiments to be described in the next chapter was devised in an attempt to clarify the situation with regard to posterior parietal cortex. It was felt that a better understanding of the posterior parietal deficit in visuo-spatial perception in the monkey was needed to identify areas of similarity and difference between these animals and man.

The human situation has been outlined and the few attempts to define the effects of posterior parietal damage in other animals in comparable terms have been mentioned. A brief comparison of the human and monkey data in this area was made by Drewe, Ettlinger,

Milner and Passingham (1970) who concluded that comparable damage in man and monkey may give rise to different disorders, and pointed to the need for more detailed study of the monkey.

The experiments contained in this study were designed to investigate the visuo-spatial perceptual capacity of monkeys with posterior parietal lesions and to compare this with that of monkeys with dorsolateral frontal lesions and unoperated controls.

One aspect of treatment of spatially relevant information in the rat has been also included for study of the effects of various lesions for comparative purposes.

CHAPTER II. EXPERIMENTS.

1. MATERIALS and METHODS.

In this section will be described the materials and methods common to all the work performed on monkeys which is included in this thesis. Testing methods used in each experiment will be described under the headings for individual experiments (sections 2a to 2e of this chapter).

(a) Subjects. Twelve "stumptailed" monkeys (*Macaca arctoides*) took part in the experiments. Ten were male and two female. These animals were born in the wild. This species was used in preference to rhesus monkeys since "*M. speciosa* ... are docile and easy to manage in captivity in comparison with *M. mulatta*" (Hall 1968). Symmes and Anderson (1967) found the stumptailed monkey equal or superior to rhesus in acquisition of behavioural tasks and in response to and recovery from surgical intervention. It was felt that the brain lesions performed on these animals would be comparable to those reported by other experimenters working with rhesus monkeys since Kling and Orbach in 1963 wrote, "A gross examination of the fissural pattern on the brain surface revealed no marked differences in the two species. Our studies of the brain to date suggest that the available stereotaxic atlases for *M. mulatta* could be employed for subcortical placements in *M. speciosa*."

The monkeys weighed between three and four and a half kilograms

at the start of the behavioural testing programme. They were weighed on the first day of every month.

(b) Maintenance. The monkeys were normally housed two to a cage three feet by three feet by two feet eight inches. Daily food intake was approximately 200 grams of calf weaner pencils and 175 grams of peanuts. An additional 175 grams of fresh vegetables was given weekly. Feeding was carried out immediately after testing for that day was finished. Water was freely available during all the time spent in the home cage.

(c) Behavioural testing. All testing was carried out using a version of the Wisconsin General Testing Apparatus (WGTA) (Harlow 1959). In the version of this apparatus used, there were two one way screens between the experimenter and the subject so that the experimenter never lost sight of the monkey between trials. Both screens were never opened simultaneously so the monkey never saw the experimenter during the testing session. Except where otherwise stated, the forward screen (nearer to the monkey) triggered a micro-switch when it was lowered, which started a timer. The monkeys were in transport cages on portable trolleys during testing. The floor of the cage was at the same height as the horizontal working surface of the WGTA (28 inches); this surface was 24 inches square. All testing apparatus used was screwed centrally on this surface. Illumination was provided by two 30W strip lights, one on either side of the subject, and there was a light excluding screen behind the monkey's cage. White noise was generated in the test room

which was situated across a passage from the housing room so as to minimize noise.

The behavioural testing covered by this thesis took place during the twelve month period from August 1972. For most of this time six animals were tested by Mr. W.Dewar and six by myself. Dr.A.D. Milner took over from either of us during holidays or illness. Testing was normally carried out five days a week.

Animals were given preliminary training in the WGTA on a two-choice junk-object discrimination task and food preferences were ascertained. All monkeys preferred either raisins or sultanas to peanuts, and the preferred fruit was used as reward there after.

(d) Grouping. Monkeys were divided into the following three groups of four:

(i) those to have lesions of posterior parietal cortex (text fig.1)

(ii) those to have dorsolateral frontal cortical lesions (text fig.2)

(iii) unoperated controls.

Assignment to a group was carried out according to performance on preoperative tasks. Monkeys completing the preoperative training schedule were allotted in threes in the order parietal, frontal, control groups so as to eliminate any bias towards apparent impairments in post-operative performance of the lesioned animals.

Surgery was performed on the first suitable day following completion of preoperative training. Two weeks recovery time was allowed. Control animals rested for two weeks at the same stage in the testing

Figure 1. a lateral and b medial drawings of right cerebral hemisphere of Macaca mulatta showing main sulci. Redrawn from von Bonin and Bailey (1947). The striped area is the intended extent of the posterior parietal lesion.

ai inferior ramus of arcuate sulcus

ai superior ramus of arcuate sulcus

ca calcarine fissure

cc corpus callosum

ce central sulcus

ci sulcus cinguli

ip intraparietal sulcus

l lunate sulcus

la lateral fissure

oi inferior occipital sulcus

ot occipitotemporal sulcus

p sulcus principalis

po parieto-occipital incisure

rh rhinal fissure

sp subparietal sulcus

ts superior temporal sulcus

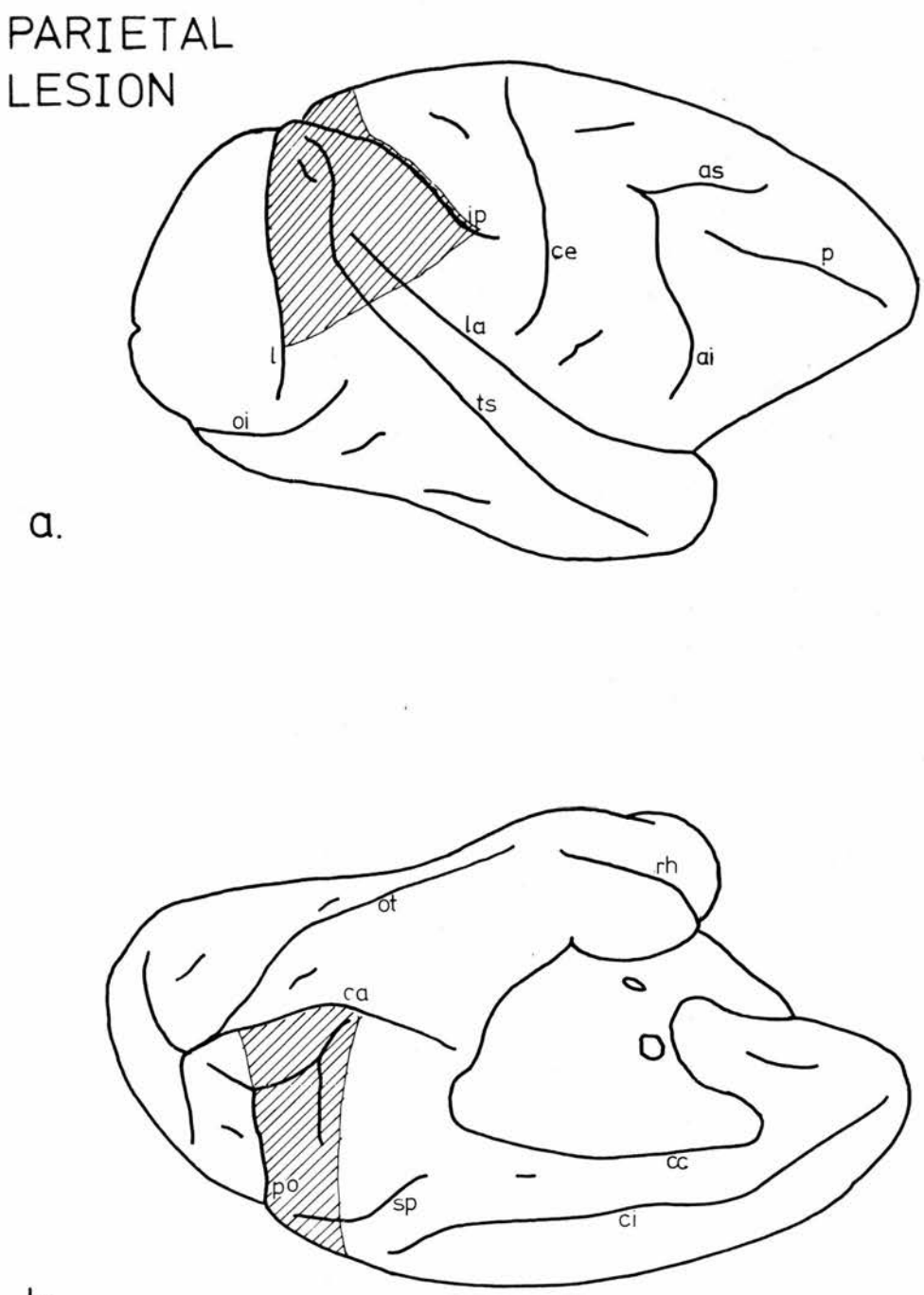


Fig 1

Figure 2. Lateral view of right cerebral hemisphere of Macaca mulatta redrawn from von Bonin and Bailey (1947) with sulci marked as in fig. 1. The intended extent of the dorsolateral frontal lesion is shown striped.

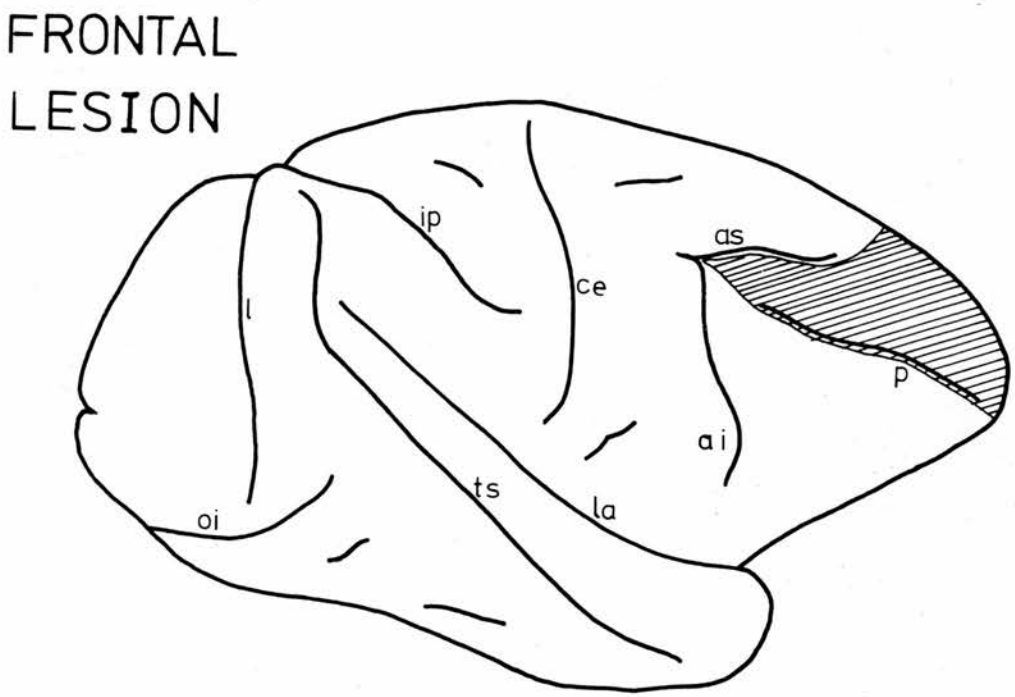


Fig 2

schedule.

(e) Surgery. Anaesthesia was induced by intramuscular injection of Ketamine (McCarthy, Chen, Kaump and Ensor 1965) and was maintained with pentobarbital (Nembutal) administered intravenously. This method was described by Drake in 1972. Supplementary doses of Nembutal were given as necessary (Domino, McCarthy and Deneau 1969) either intraperitoneally or intravenously using an Abbott disposable Butterfly-21 infusion set.

All operations were performed as single stage bilateral subpial aspirations and the aseptic ablation techniques described for the monkey by Meyer and Meyer in 1971 were adhered to except in the following respects:

(i) The head holder used had no ear bars.

(ii) A 3M company "Steridrape" with a circular aperture was used.

(iii) A single bone flap was turned laterally to expose both cerebral hemispheres. Skull holes were drilled on either side of the sagittal suture with a diamond burr and joined across the midline by cutting the bone with a de Vilbiss' cutter to avoid the possibility of causing bleeding from the medial sagittal sinus.

(iv) Bleeding was normally controlled with light pressure and cottonoid patties. Electrocautery was used where necessary, if for instance a blood vessel traversing the subdural space and adhering to the dura was in

the path of the desired dural incision. In fact the cautery was only used twice.

(v) No wire was used in reapproximating the bone flap, since thin steel saws used obviated the necessity for this.

(vi) The wounds were dressed with BDH "Nobecutane".

The following points about individual operations should also be noted:

(i) The first monkey to be operated (P1) had a large swelling which had been increasing in size over most of the cranium. It was noted that the head was spongy to palpate before surgery. When the temporal muscle was cut, it was found that this swelling was a haematoma apparently in the pericranium and containing an estimated 50 ml. of blood. The temporal muscles were found to be unusually thick in this animal. The haematoma did not return after surgery and no other abnormalities were noted.

(ii) An accidental burn was caused with the diathermy in P3. This was in the left post-central gyrus at the position estimated to be the hind limb sensory area. The contralateral hind limb was noted to be used awkwardly for some time following the operation and this monkey took rather longer to recover from surgery than did the others, requiring hand feeding for a few days.

(iii) F4 was noted to have a very short upper limb of

arcuate sulcus bilaterally.

Surgery was carried out by Dr.A.D.Milner assisted by Mr.W.Dewar and myself.

The monkeys (and rats) were still participating in further behavioural studies at the time of writing so no histological verification of the lesions had been made.

Informal observation revealed marked misreaching by parietal monkeys for several days postoperatively.

2. EXPERIMENTAL REPORTS.

(a) Experiment 1. Stylus maze task.

Introduction. This experiment was prompted by the reports of impairments of human subjects with posterior brain lesions on perceptual mazes. Milner (1965) presented a stylus maze task resembling the one used here to 79 human subjects with surgically produced brain lesions. The lesions limited to parietal lobe were (except in one very interesting case) small, and these cases showed no impairment in learning the maze task. The one case with a larger right sided parietal lobectomy and the right parieto-temporo-occipital cases however were the most severely impaired in terms of a tendency in these cases to lose bearings in the maze. This deficit suggested a qualitatively different difficulty from that of the right temporal lobectomy patients who suffered a short term memory impairment associated with hippocampal damage.

Elithorn and coworkers (e.g. Elithorn 1964) have performed a

series of experiments using a lattice-maze task which requires the same type of visual memory for spatially relevant information. These workers did not find the focal aspect as important in defining an impairment as the hemisphere which was damaged but they also found a qualitative difference between the impairments of anterior and posterior lesion patients, the posterior patients showing a greater impairment on the easier tasks.

These findings of there being an impairment on these perceptual mazes in human patients with posterior brain lesions, which is unrelated to sensory deficit or generalized short term memory loss , along with the classical difficulty in performing manual skills under visual guidance following parietal lobe damage in humans (Critchley 1953) suggested that we should look at the performance of monkeys with parietal cortex lesions on a stylus maze task.

Materials and Methods. The maze used in this experiment is shown in plate 1. The lower right quadrant of the maze only was used. The internal area of the whole maze was 10 inches square and each block of the four by four matrix was two inches square. Styli were made with two metal discs held together by a screw thread and separated by a nut. The metal discs held the stylus in position in the groove of the maze so that it could only be removed at the end of the pathway. There was a plastic covered knob at one end of the screwthread for the monkey to hold and the reward was on the other end. Errors (wrong turns at choice points) were recorded electrically and removing the stylus at the end of the pathway stopped the timing device, hence giving the time taken to complete a trial. Ten trials were

Plate 1. Stylus maze apparatus used in experiment 1.

s stylus in starting position

e exit position

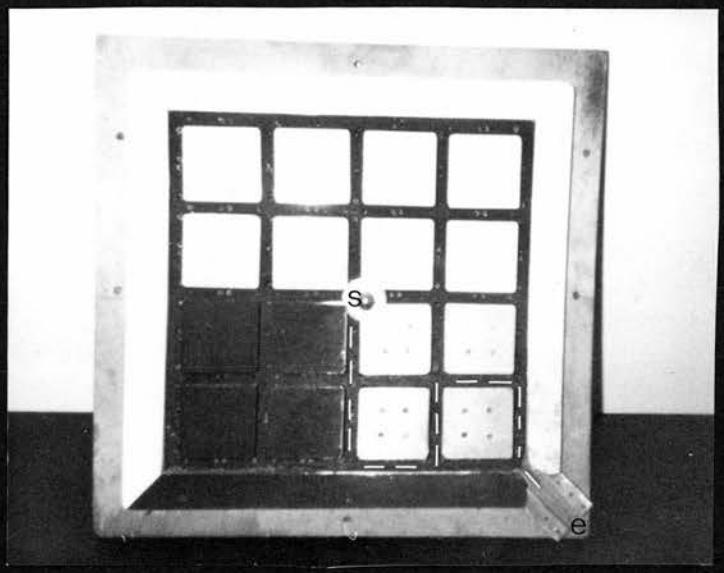
White dotted line marks correct solution pathway.

Plate 2. Pointer apparatus used in experiment 2.

p pointer

d doors (concealing food wells)

1



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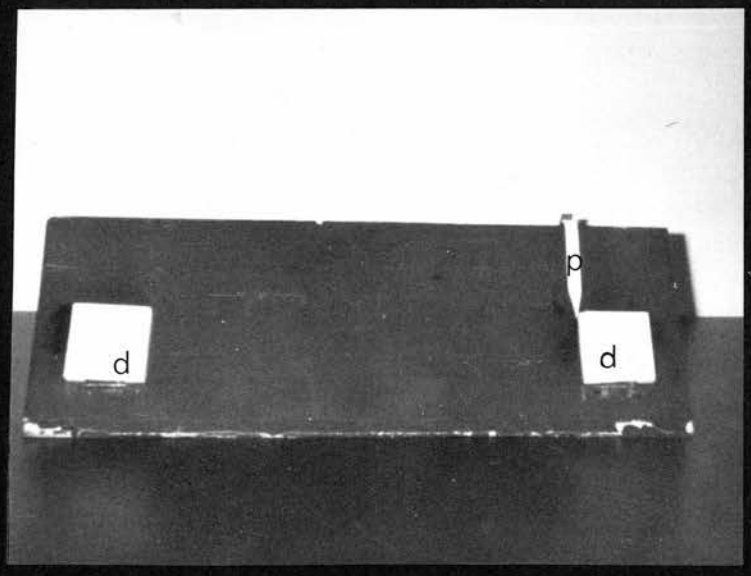


given daily.

The animals were trained preoperatively on this task to a criterion of three consecutive correct errorless trials. Training was achieved by starting near the end of the maze and moving the stylus one stage further from the goal after two consecutive errorless trials were performed, until the whole of the chosen pathway was in use.

This was the first task to be given preoperatively and retraining to the same criterion was given on the days immediately prior to surgery. Retraining again was the first postoperative task.

Results. Pre- and postoperative trials and errors to criterion and savings scores (assessed from errors) are presented in table I. The reduced savings in the parietal monkeys compared with the unoperated controls is not statistically significant (Mann-Whitney U=4, p=0.171). The very great detriment in postoperative relearning of P3 and P4 however, and to a lesser extent P1, indicates that these animals do have an experimentally induced impairment on this task.

Mean times per trial pre- and postoperatively are compared in table II. Inter-subject comparisons of absolute time scores are not made. The post- minus preoperative difference for the parietal animals is significantly different from that for the unoperated controls (U=0, p=0.014) and for the frontal group (U=0, p= 0.014). The frontals and controls do not differ significantly (U=4, p=0.171).

Discussion. There are two obvious possible explanations of the deficit some of the parietal monkeys demonstrated on this task. The

SUBJECT	TRIALS TO CRITERION		ERRORS TO CRITERION		SAVINGS
	Pre-op	Post-op	Pre-op	Post-op	$\left(\frac{\text{pre-post}}{\text{pre+post}}\right)$ errors
P1	2	129	4	789	-0.99
P2	70	6	168	76	0.38
P3	45	150F	128	923	$<$ -0.76
P4	185	150F	889	1333	$<$ -0.20
					$<$ -0.39 MEAN
F1	16	11	49	34	0.18
F2	137	62	569	213	0.46
F3	271	85	1167	312	0.58
F4	236	105	1130	525	0.37
					0.40 MEAN
C1	44	50	169	223	-0.14
C2	13	86	72	303	-0.62
C3	42	25	168	120	0.17
C4	300F	66	1929	382	$>$ 0.67
					$>$ 0.02 MEAN

Table I: Pre- and postoperative trials to criterion and errors to criterion and savings scores for the stylus maze task. The results for each group do not differ significantly in any respect.

SUBJECT	PRE-OP	POST-OP	DIFFERENCE POST- PRE-OP
P1	7.36	17.88	+10.52
P2	8.57	105.50	+96.93
P3	9.42	18.39	+8.97
P4	13.53	35.87	+22.34
			34.69 MEAN
F1	4.34	6.14	+1.80
F2	9.48	11.79	+2.31
F3	11.41	6.51	-4.90
F4	15.79	22.28	+6.49
			5.70 MEAN
C1	12.93	13.74	+0.81
C2	11.91	11.83	-0.08
C3	16.80	22.24	+5.44
C4	23.03	19.82	-3.21
			2.96 MEAN

Table II Mean times per trial on stylus maze task in seconds.

first is that there was an impairment of visuo-motor coordination. The animal may have been manifesting an articulative problem causing him to be simply unable to move the stylus accurately along the desired path.

The second explanation is that there was a deficit of spatial orientation. An impairment of this kind is suggested after parietal lobe damage in humans and was demonstrated experimentally in the locomotor maze type task employed by Semmes and coworkers (Semmes, Weinstein, Ghent and Teuber 1955 and 1965). How far analogies should be drawn between this locomotor task and the stylus maze tasks used here and by Milner (1965), Corkin (1965) and Orbach (1959) in which one limb is moved relative to the rest of the body, is uncertain (Teuber 1965). Milner (1965) however suggested that her parietal patients became disoriented while performing the stylus maze task and since the postoperative task in the present experiment did not involve new learning, it was considered that the parietal impairment could have been of a disorientation nature.

Two control experiments were carried out in an attempt to eliminate one of these two explanations and so to define the parietal impairment more clearly. These control experiments will now be outlined.

(i) Marked maze.

The task was to follow a marked pathway through the maze; there was no necessity to remember the correct solution. If the performance of parietal monkeys improved under this condition, it would be concluded that the impairment on the original task was not one of sensory-motor coordination but reflected a lack of spatial orientation.

Materials and methods. White nylon cord was threaded through holes drilled in the pathway of the maze used in the original task. The cord showed up clearly. Fifty trials were given under this condition followed by a further 100 trials with marked and unmarked conditions on alternate days. Six animals only were used.

Results. Mean errors per trial and mean times per trial are given in table III.

Discussion. Since performances on the marked maze were if anything poorer than those on the original task (probably the nylon cord served to distract the monkeys), this alteration in task was not simplifying the problem as was expected, so no conclusions as to the nature of the parietal impairment on maze performance could be drawn.

(ii) Pathway task.

The task was again altered to one of moving a stylus through a track where no incorrect moves could be made. Time scores would reflect dexterity of performance of this type of task.

Materials and methods. The apparatus had the same overall dimensions as the maze shown in plate 1. There was a single track cut through a metal plate in which the styli could be moved, hence there were no choice points and no errors could be made. The shape of the pathway was the same as the correct solution to the maze (plate 1). The same styli as before were used. The timer

SUBJECT	MARKED (1st 50 TRIALS)		MARKED		UNMARKED	
	ERRORS	TIME	ERRORS	TIME	ERRORS	TIME
P1	4.72	14.38	2.72	12.58	2.38	12.01
P2	8.60	11.81	2.72	9.58	3.16	9.97
F1	5.30	10.36	4.40	13.50	2.77	9.26
F2	2.74	20.75	3.60	12.47	2.98	10.34
C1	8.03	14.73	3.80	17.49	2.92	16.33
C2	4.78	17.00	3.62	17.02	2.16	12.29

TABLE III Mean time (in seconds) and error scores for marked maze control experiment.

was started by the monkey moving the stylus from the starting position and was stopped by his withdrawing it at the end of the track, thus a recording was made of the actual time an animal took to draw the stylus through the track. Fifty trials were given (10 daily). This task was given after about seven postoperative months.

Results. Results of this experiment are shown in table IV. Differences between groups are not significant. Parietal animals all show improvement with testing.

Discussion and Conclusion. The improvement the parietal monkeys show with practice on this stylus in tracktask suggests that they do have a difficulty in performing the task compared with other animals. If P2 is eliminated (this animal was not impaired on the original maze task), we do see a pattern of impairment in the parietals. It was conluded that the possibility that the parietals' impairment on the maze task was caused by a sensory motor coordination lack could not be eliminated.

(b) Experiment 2. Pointer task.

Introduction. This experiment was concerned with the effects of parietal and frontal lesions on the solution of a problem with cue and response/reward spatially separated.

Although there is considerable disagreement as to the exact nature of the mechanism underlying the impairment, it is well

SUBJECT	OVERALL MEAN TIME PER TRIAL	MEAN TIME FIRST 10 TRIALS	MEAN TIME LAST 10 TRIALS	"IMPROVEMENT"
P1	14.28	9.35	6.06	3.29
P2	2.98	3.63	2.07	1.56
P3	5.54	9.88	2.94	6.94
P4	7.13	7.58	5.86	1.72
F1	5.48	4.61	7.61	-3.00
F2	5.32	6.76	6.20	0.56
F3	1.85	2.20	1.61	0.59
F4	9.36	7.77	7.75	0.02
C1	3.41	4.13	3.55	0.58
C2	16.05	36.85	11.98	24.87
C3	3.57	3.34	8.71	-0.37
C4	7.32	9.08	5.31	3.77

TABLE IV Time scores for stylus in track control task in seconds.

documented that humans with parietal lobe damage have difficulty in localizing objects in space, hence in assessing the relative distances between them and in accurately differentiating left from right (e.g. Holmes 1918, McFie and Zangwill 1960, and Whitty and Newcombe 1965).

It has been shown to be difficult for normal monkeys to learn a task in which stimulus,response and reward are separate spatially in any combinations (Murphy and Miller 1958, Meyer, Pollidora and McConnell 1961, Schuck, Pollidora, McConnell and Meyer 1961, Stollnitz and Schrier 1962, Davis, Lampert and Rumelhart 1964, Pollidora and Fletcher 1964, Medin 1968, Sayner and Davis 1972). Sayner and Davis (1972) found that a raisin attached to the stimulus (which was spatially separated from the reward/response) facilitated mediation of the stimulus-response gap. This facilitation could be explained by the fact that the raisin forced response to both the stimulus and the response manipulandum, a condition which has been shown to improve performance and is known as double responding (Murphy and Miller 1958).

The effects of brain lesions on the performance of monkeys on stimulus-response separation tasks have been observed in three studies. Ettlinger and Wegener in 1958 found monkeys with bilateral parietal cortical lesions to be impaired on retention of a task requiring them to open the door nearer to a certain configuration of absent pegs from a seven by three matrix of pegs on a board. Bates and Ettlinger (1960) published a further investigation of the conditions responsible for this deficit in the parietal animals and ascertained that it was due to a disability relative to normal animals to associate cue and either response or reward when they were separate.

A more recent study in this area was that of Pohl in 1970. Pohl found parietal monkeys impaired on a landmark reversal task, that is, an impairment of choice in terms of the spatial relation to an external landmark.

In the present experiment, it was hoped to define more clearly the parietal deficit on a stimulus-response separation task. It was suggested that if the impairment was one of directing fixation or response, the presence of a raisin on the stimulus (pointer) would improve the performance of these parietal monkeys to a greater degree than it did that of normal animals by directing their (otherwise inadequate) attention.

Materials and Methods. A board six by 18 inches painted matt black and supported at 52° to the horizontal was used. Two white doors 12 inches apart and two inches square were hinged to this board covering one inch square foodwells. A moveable white metal pointer slotted into a metal runner behind this board in positions one inch apart. This apparatus is shown in plate 2.

Preoperative training consisted of training to a criterion of 80% correct (27 out of 30 consecutive trials correct) with the pointer (stimulus) touching the manipulandum, that is zero separation condition. Position habits persisting over a row of ten trials were corrected. This was followed by further preoperative training to a criterion of 48 out of 60 consecutive trials correct with the pointer position varying randomly between zero and five inches from either door. Thirty six trials were given daily on this task. Left-right positions of the pointer were varied according to

the Gellerman schedule.

Postoperatively 144 trials were given in the random separation condition as before and this was designated condition a. A hundred and forty four trials were then given with a raisin attached to the end of the pointer (condition b) and this was followed by a further 144 trials without the raisin again (condition c). Errors were recorded in each case and it was also recorded if the monkey touched the pointer when there was no raisin on it or failed to take the raisin in condition b.

Results. Postoperative total error scores are presented in table V and summarized over lesion groups in text fig.3. Parietal animals were not impaired on performance of this task as measured by total error scores. Condition b did not affect the error scores of any group differently from any other group. Errors over all subjects under condition b were fewer than under condition a (Wilcoxon matched pairs signed ranks test, T=5.5, significant at 0.005 level). Condition c scores however were not significantly different from condition b scores.

Frontal animals were impaired on initial postoperative performance (condition a) compared with controls (U=0, p=0.014).

Error scores were also examined in terms of the distance of stimulus-response separation at which they were made. Summaries of percentage of all errors which were made at each separation distance are presented in table VI and graphically in text fig.4. Analysis of variance yielded significant effects of distance of separation of pointer from response site (F=87.14, df=5,159) and of interaction

SUBJECT	PRE-OP	POST-OP ERRORS a(no raisin)	b(with raisin)	c(no raisin)	TOTAL
P1	47	39	29	24	92
P2	20	45	41	31	117
P3	112	28	22	45	95
P4	47	26	21	31	78
MEAN		34.5	28.25	32.75	95.5
F1	31	40	26	23	89
F2	11	39	26	33	98
F3	20	34	27	19	80
F4	50	41	30	27	98
MEAN		38.5	27.25	25.5	91.25
C1	11	25	31	18	74
C2	115	31	11	16	58
C3	29	24	23	28	75
C4	99	32	23	38	93
MEAN		28.0	22.0	25.0	75

Table V. Error scores for preoperative learning and postoperative performance on pointer task. Errors under condition a for the frontal group significantly exceed those for the control group (Mann-Whitney U=0, p= 0.014).

Figure 3. Histogram summarizing errors in postoperative performance of monkeys on pointer task under the three conditions a. no raisin, b. with raisin and c. no raisin attached to the pointer.

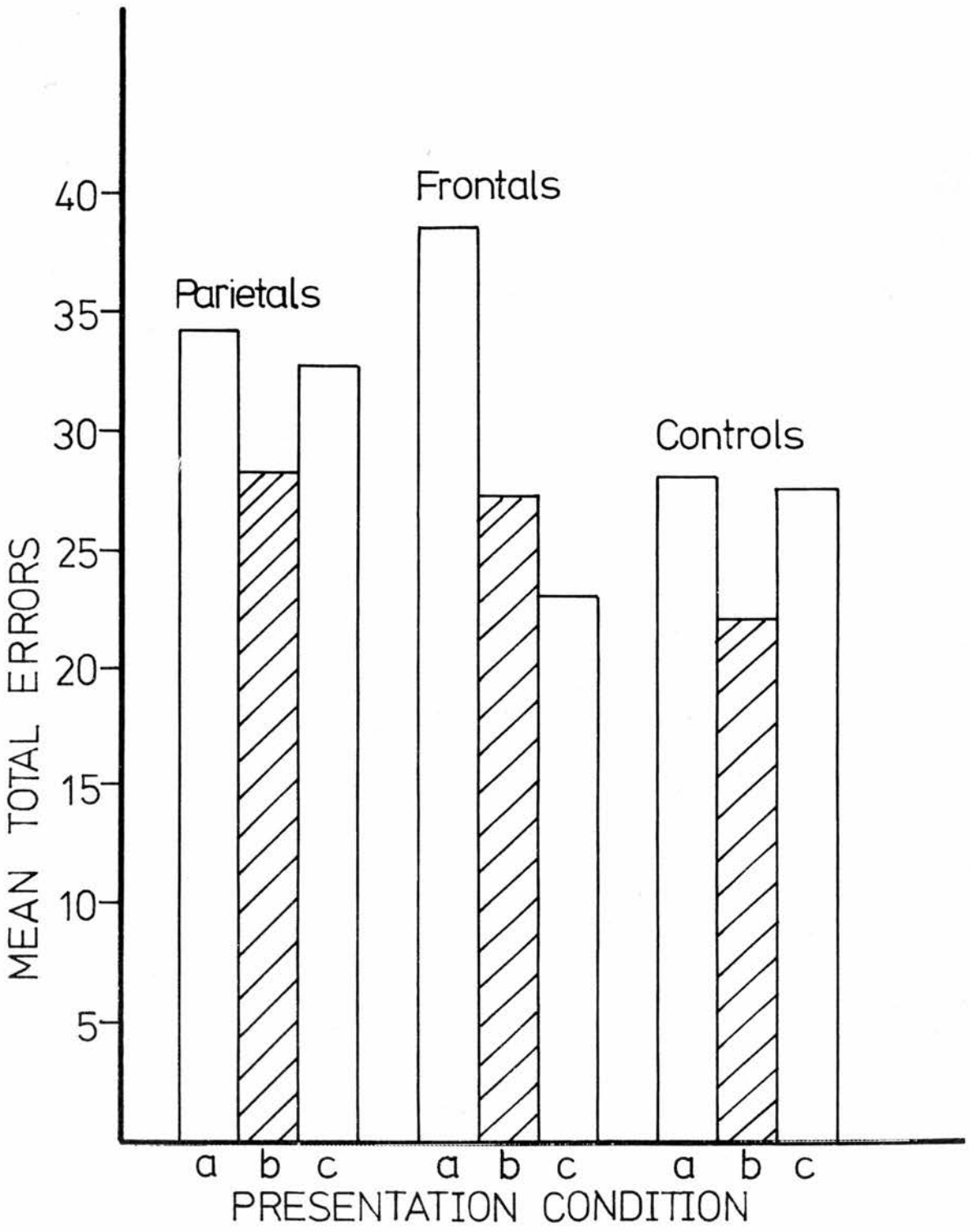

Parietals
Frontals
Controls
40
35
30
25
20
15
10
5
MEAN TOTAL ERRORS
a b c
a b c
a b c
PRESENTATION CONDITION

Fig 3

S/R SEPARATION	PARIETALS			FRONTALS			CONTROLS		
	a	b	c	a	b	c	a	b	c
0	6.2	2.7	0.6	6.1	3.8	3.9	9.3	0.8	0.9
1 (inch)	9.9	6.2	6.8	9.4	3.8	1.9	9.1	3.3	1.3
2 "	15.4	10.6	10.0	12.4	6.4	10.7	10.5	8.7	6.7
3 "	18.8	11.2	18.9	18.4	21.4	10.5	15.9	17.1	13.3
4 "	22.8	27.5	25.6	24.3	28.2	23.8	23.4	41.8	38.1
5 "	26.9	41.8	38.2	29.5	36.3	46.2	32.2	30.5	39.8

Table VI. Percentage of total errors made at each S/R separation distance under each condition.

Figure 4. Graph showing percentage of errors made at near (zero and one inch), medium (two and three inches), and far (four and five inches) separation distances of pointer from nearer response door by members of the three groups of monkeys under the three conditions a, b and c.

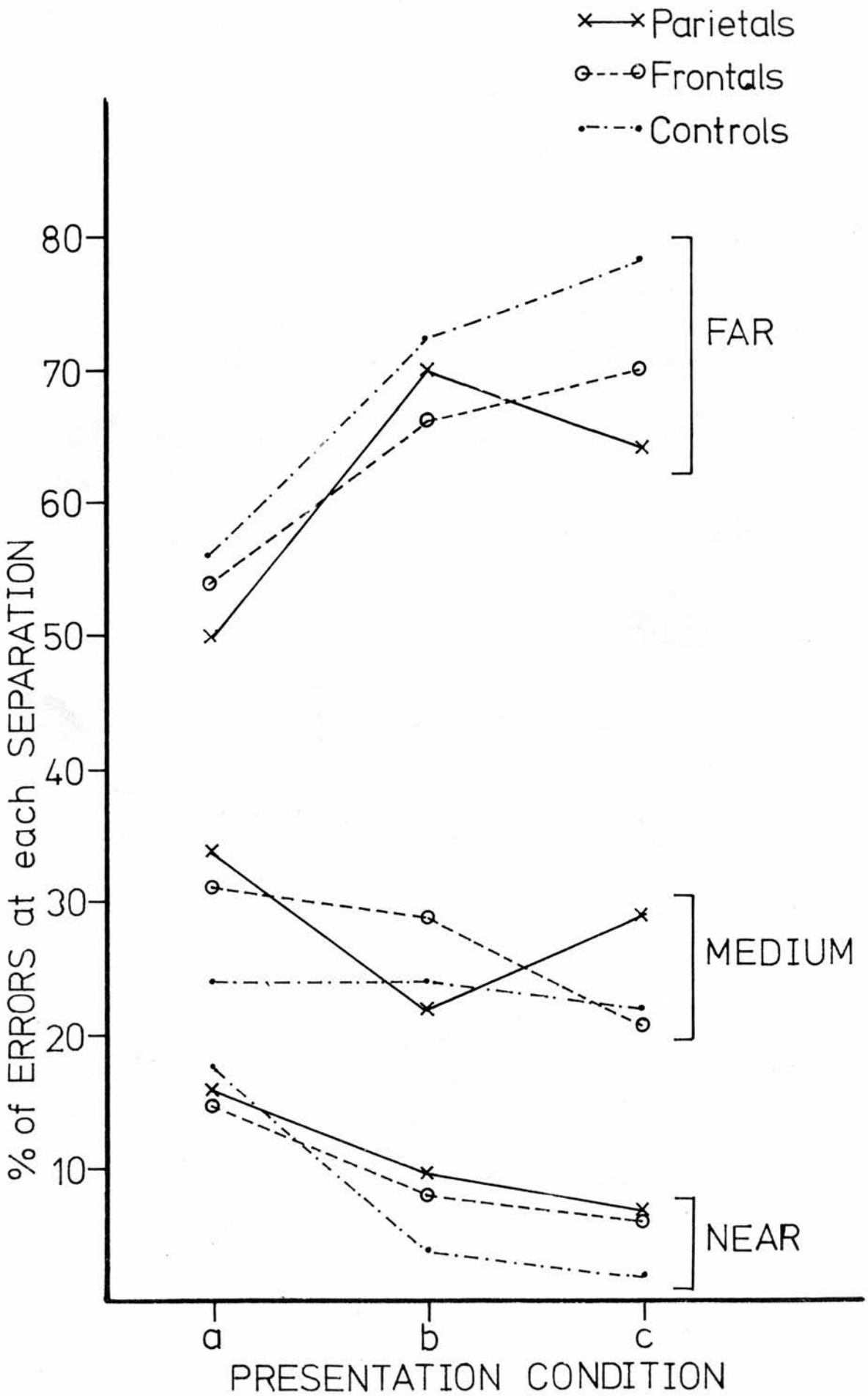

Parietals
Frontals
Controls
80
70
60
50
40
30
20
10
% of ERRORS at each SEPARATION
FAR
MEDIUM
NEAR
a
b
c
PRESENTATION CONDITION

Fig 4

of stimulus-response separation distance with the presence or absence of raisin condition ($F=2.90$, $df=10,159$) but not with lesion group.

Discussion. The failure of the parietal monkeys in the present experiment to demonstrate an impairment on postoperative performance of this task was unexpected in view of the findings of previous studies. The finding of an initial impairment in the frontal group was also surprising in view of Pohl's finding (Pohl 1970) that frontal animals were not impaired in his landmark task (in fact they performed significantly better than did unoperated controls on initial trials) and Bates and Ettlinger's finding (Bates and Ettlinger 1960) that frontal monkeys were not impaired on a stimulus-response discontiguity problem.

The pointer task used here depended on the use of an extrapersonal referent spatially separate from the response locus. It is this function which Pohl (1973) and Mishkin (1970) suggested was impaired by removal of posterior parietal cortex. The finding that the frontal monkeys in the present experiment were initially impaired on post-operative performance was not interpreted as an inability on their part to make use of the extrapersonal referent. The fact that these animals improved with practice (and not just with a raisin on the pointer) suggests that the initial difficulty may have been due to some other cause, the most likely of which is probably that the hyperactivity of these animals prevented them from attending fully to the situation before responding. It is suggested that the frontal animals responded to the door they looked at first or to the one nearer to where they sat, as do normal monkeys when first confronted

with a choice situation (Oscar-Berman, Heywood and Gross, 1971) and which is suggested for the orbital frontal impairment on visual discrimination found by Passingham (1972).

An important difference between the present study and Pohl's earlier work (Pohl 1970) was that Pohl looked at initial learning scores and reversals whereas the account given here is of performance on a preoperatively learned task. However Bates and Ettlinger (1960) and Pohl in a later study (Pohl 1973), found parietal monkeys impaired on learned stimulus-response separation problems. An explanation for the apparent discrepancy in these results will now be offered.

After Bates and Ettlinger's parietal monkeys had mastered postoperative relearning of the stimulus-response separation task, the stimulus-response gap was increased and the parietals were not impaired by this increase any more than were controls. Bates and Ettlinger proposed on the strength of this finding that the initial impairment shown by the parietals postoperatively was due to an impairment in learning to examine the cues before responding. Once the technique of examining the cues had been learnt, there was no impairment even when the task was made harder. I would suggest that all the animals in the present study had learned before surgery to attend to the stimulus regardless of its position, whereas Bates and Ettlinger's monkeys had not done so in the same way.

It could however be argued that if the subjects in the present study had learned the technique of double responding preoperatively, which seems likely to be how problems of this type are solved (Fellows 1968 chap.7), and were applying this postoperatively, the presence

of the raisin on the pointer forcing overt double responding would not significantly improve performance as it in fact did. I would argue that this improvement was due to practice rather than forcing double responding since in half the cases, on the first day of testing with the raisin, more errors were made than on the last day of testing without the raisin. Final testing without the raisin also did not reduce performance level, a finding contrary to that of Sayner and Davis (1972). This last finding also supports the notion that the animals in the present study had already learned to attend to the stimulus without its being baited with food.

The method of training used in this experiment differed from that used by Pohl in 1973 on what appears otherwise to be a very similar task. It will be remembered that in the present study animals were shifted directly from the zero separation of stimulus from response site to a random arrangement of zero to five inch separations. In Pohl's study (Pohl 1973), he taught the task to the animals by stepwise approximation to the final condition of two inch separation of cue and response site. In Bates and Ettlinger's study also, the stimulus-response distance was small. I would suggest that Pohl's monkeys may have been responding generally to the overall configuration of response plaque, object, response plaque rather than having learned to orient to the stimulus regardless of position as I suggest the monkeys in the present pointer task had learned to do. If this is the case, it seems reasonable to suggest that the severe impairment which Pohl observed in two out of three parietal monkeys on this task could have been caused by a simple sensory defect (which seems unlikely in view of the lack of defect on other tasks)

or else an impairment in ability to interpret a complex input of dissociated items.

One anomolous finding of the present experiment in the context of the literature on double responding which should be mentioned, although I do not propose to attempt an explanation of it, was that one monkey (C4), when tested in the final condition (without the raisin-condition c) failed to open the corect door every time he touched the pointer. This finding was contrary to all the evidence suggesting that orienting towards the stimulus and double responding improve performance.

(c) Experiment 3. Discrimination learning with spatially separate irrelevant cue.

Introduction. This experiment, like experiment two, was concerned with the effects on performance of spatially separating a stimulus from the site of the reward/response. In this experiment however, the relevant cue was never separated from the response manipulandum which was always directly above the reward.

The animals were first trained to respond to a colour cue which later became irrelevant in favour of a shade of grey. The irrelevant colour was displaced to the side of the relevant grey cue which covered the manipulandum and reward.

It was argued that if parietal monkeys were impaired in associating spatially discontiguous stimuli and rewards, they may be better than other monkeys at ignoring spatially displaced cues which they had previously learned to be meaningful.

Materials and Methods. The two-choice apparatus shown in plate 3 was used. Lids four inches wide and ten inches apart covered one and three-eighths inches diameter foodwells. The foodwells were under one side of the lids as shown. Opening a lid triggered a microswitch which stopped the timing device (which had been started by lowering the one way screen nearer to the monkeys in the WGTA) and hence recordings of response latencies were obtained.

All training on this task was given postoperatively only. Animals were first trained to a criterion of 90% correct (three errors over 30 consecutive trials) on a red/green discrimination. Humbrol Enamels numbers 19 and 2 were used. Half the animals from each lesion group had each colour positive. Left-right positions were varied according to the traditional Gellermann schedule (Gellermann 1933) and a non correction procedure was used. The day after criterion was reached on this colours discrimination, the monkey was taken to the task of discriminating between two shades of grey (Humbrol Authentic Colours HB3 and HB6). The grey paint covered the half of the front of the lid which covered the food well and held the knob by which the monkey opened the lid. The other half of the lid was painted with the irrelevant red/green colour. The colour cue was one inch from the manipulandum (and reward). Hence two pairs of lids were used as illustrated in text fig. 5. A double Gellermann schedule was used to alter left-right and stimulus pair in use in each trial. The same criterion as for the colours discrimination was use.

Response latencies, errors and trials to criterion were recorded.

Plate 3. Two choice apparatus used in experiment 3.

l lid with stimulus configuration painted on

f foodwell

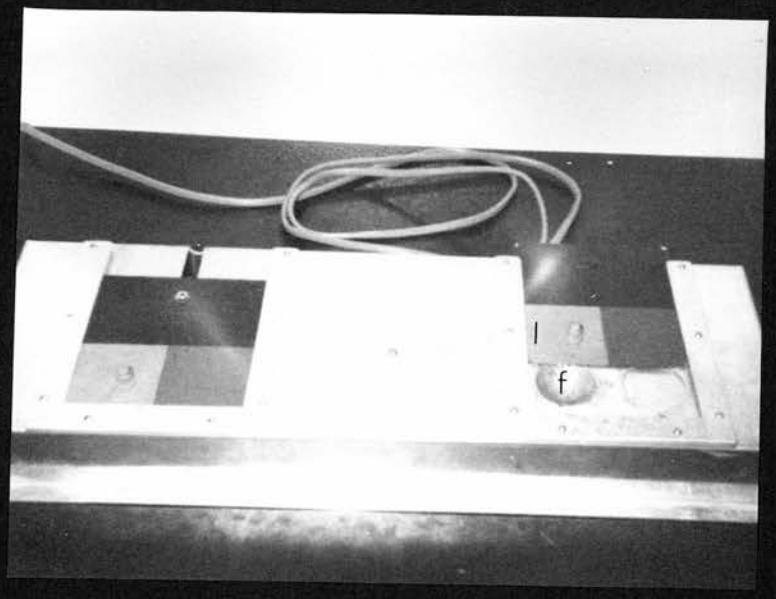
Plate 4. Two choice apparatus used in experiments 4 and 5.

f foodwell

l lid

s stimulus on wooden block

3



4

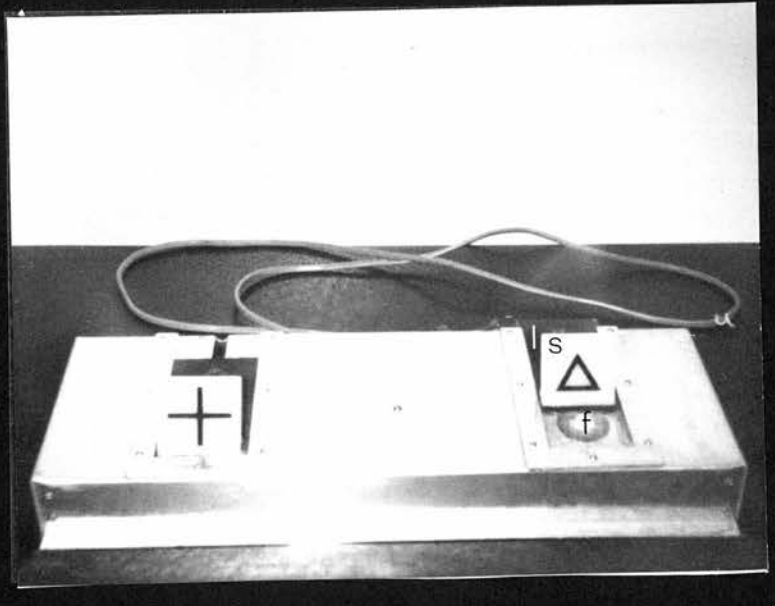


Figure 5. Diagram showing stimuli used in experiment 3.

a. Red and Green lids

b and c. Stimulus (lid) pairs with irrelevant colour cue.

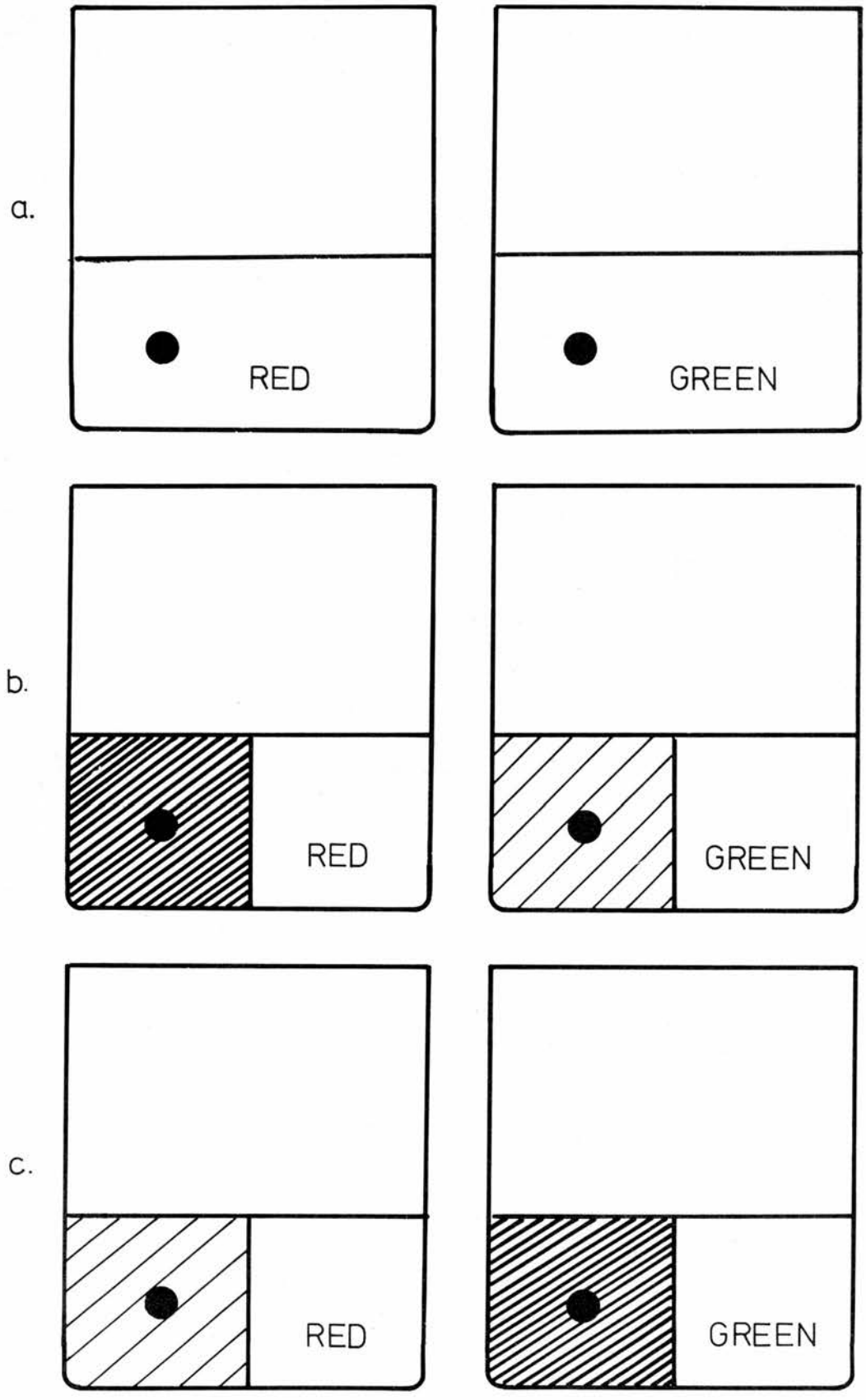


Fig 5

Results. Trials to criterion and mean response latencies for both discriminations are presented in table VII. Total errors and percentage of errors which were made by responding to the previously positive colour cue are also given for the shades of grey discrimination task.

The monkeys from each lesion group were not differentially affected by the presence of the irrelevant colour cue.

The only result to attain significance was that the frontal group of monkeys were quicker to respond than controls on the light/dark grey discrimination (Mann Whitney U=1, p=0.029). Their latencies compared with parietals also nearly reached significance (U=2, p=0.057) as they did compared with controls on the red/green discrimination.

Discussion. The tendancy for dorsolateral frontal monkeys to have shorter response latencies on this task than did parietals or unoperated controls was not entirely unexpected. Passingham found dorsolateral prefrontal monkeys responded more quickly than control animals on a visual discrimination task although the difference was more marked following orbital prefrontal lesions (Passingham 1972). The explanation of this finding is not clear and there seems some confusion over the similarities between monkey and man in this respect (Milner 1964).

The failure to demonstrate any intergroup difference in the ability to ignore the previously relevant colour cue when it was displaced to the side of the grey stimulus and the response/reward site requires an explanation. One explanation of this could be that

SUBJECTS	RED/GREEN		SHADES of GREY			%ERRORS to
	TRIALS to CRIT.	MEAN TIME (secs)	TRIALS to CRIT.	MEAN TIME (secs)	ERRORS	PREVIOUS +VE
P1	90	1.81	310	2.19	109	53
P2	20	3.10	350	3.56	154	89
P3	30	1.97	90	1.51	45	60
P4	0	0.95	700	1.55	300	90
						MEAN 73
F1	30	0.98	330	1.38	115	64
F2	20	1.35	120	1.58	55	71
F3	40	1.02	90	1.38	31	58
F4	60	1.79	500	1.24	225	84
						MEAN 69
C1	40	3.17	110	8.55	49	57
C2	0	1.48	200	1.86	84	51
C3	30	2.69	160	1.88	52	65
C4	100	1.68	190	1.52	80	58
						MEAN 58

Table VII. **Results of Experiment 3.**

the shades of grey discrimination was too easy, so all monkeys found it easy to ignore the irrelevant colour on the lid. The trials taken to reach criterion on the two discriminations however make that explanation seem unlikely (mean trials to criterion red/green discrimination 38.3, range 0-100; mean trials to criterion greys discrimination 262.5, range 90-700) since the second task was clearly more difficult than the first.

It is suggested that the reason parietal monkeys did not cease to respond to the displaced colour cue was that the experience of prior training on the pointer task (experiment 2) had taught these monkeys the habit of orienting towards a stimulus (once they had learned of its relevance) irrespective of position, hence overriding the spatial difficulty encountered by others in parietal monkeys (e.g. Bates and Ettlinger 1960). In fact the parietal group in the present experiment made more of their errors by responding to the previously positive colour cue than did frontals or controls and it is suggested that the explanation just given in terms of previous experience could well account for this, if the following is taken into consideration.

In the discussion of the last experiment (experiment 2) it was suggested that the deficit in associating spatially discontiguous material observed by Bates and Ettlinger (1960) and Pohl (1973) after posterior parietal ablations may have been due to an impairment in the ability to integrate a complex sensory input or even more simply, an impairment of the sensory input itself. Assuming that either of these is the nature of the parietal deficit, orienting towards the stimulus on a landmark type task would remove the deficit

whereas in the present experiment, orienting towards the previously relevant colour would reduce performance in that it would force more errors to be made towards the previously positive colour cue which was found to be the case. The control animals, it may be argued, broke the colour habit more easily since they were able to perceive the whole stimulus configuration.

It will also be observed that frontal animals in the present experiment made a greater percentage of errors towards the previously positive colour cue than did normal controls. This could perhaps have been a manifestation of an impairment in response inhibition which is suggested for humans with brain damage in this area by Milner (1964) but is usually observed in monkeys with orbital prefrontal lesions rather than dorsolateral (Passingham 1972). A fixation defect could also have been present in the dorsolateral monkeys since this lesion invaded the frontal eye fields.

(d) Experiment 4. Fragmented pattern recognition.

Introduction. Experiment 4 was concerned with a different aspect of the ability to associate spatially discontiguous material; recognition of patterns presented visually in fragmented form.

Two investigations of this aspect of perception will be described. In the first (i), recognition of figures previously learned in the complete form, when subsequently presented in different degrees of fragmentation was assessed. It was predicted that parietal monkeys would be impaired on recognition of the fragmented figures because of a difficulty in associating the parts and perceiving

the input as one figure, which would not be encountered by the other animals.

In the second part of this experiment (ii), it was hypothesized that normal and frontal monkeys would learn a visual discrimination according to the shape of the pattern rather than the form in which it was presented (fragmented or continuous), whereas parietals, because of the supposed deficit in their visuo-spatial perception, would learn to discriminate in terms of "fragmentation versus complete".

The results of (i) and (ii) will be discussed together.

(i) Materials and Methods. Stimuli were presented using the two-choice apparatus shown in plate 4. The lids on this apparatus were two and a half inches wide and ten inches apart. Foodwells were one and three-eighths inches in diameter. Two screws through each lid acted as templates for black painted wooden wedges which held the stimuli secure and at 60° to the horizontal. The stimuli themselves were made with black letraset on two inches square opal Perspex pieces $\frac{1}{8}$ inch thick. These Perspex squares were glued onto the wooden wedges so the monkey could push against the actual stimulus and thereby open the lid and gain reward.

Training to a criterion of 90% (27 out of 30 consecutive trials correct) on a cross (+) and triangle ($\triangle$) discrimination was given first. Forty trials were given daily and left-right positions were varied according to the Gellermann schedule. All training was given postoperatively only. The four sessions following completion of training were equivalence testing sessions and took the following form.

Two equivalence stimulus pairs were used. Both had the same overall dimensions as the original training stimuli but the outline figures were made up with separate Letraset squares with the square: gap ratios of three:one (one stimulus pair) and one:one (the other pair). Five presentations of each pair were given in each testing session. These equivalence tests were interspersed between trials on the original discrimination task and followed 10 such trials. Altogether 10 trials were given with each member of each equivalence stimulus pair on each side. Non differential reward was given on the equivalence tests. If performance on the remaining trials fell below 80% correct, the animal was returned to training on the original discrimination until he regained criterion level (90%).

(i) Results. The results of the equivalence test sessions are given in table VIII. Errors and mean response latencies on trials with the original and equivalence stimuli are given separately. There were no significant intergroup differences.

(ii) Materials and Methods. Stimuli were made and presented as described in (i) above.

Animals were first trained to the criterion of 90% correct on a discrimination between an outline square and a fragmented (two:one) outline H shape tilted through 45°. Subsequent training to the same criterion was given with a fragmented square and a continuous outline tilted H. The positive stimulus in each case was counterbalanced so that two animals from each lesion group had the fragmented figure positive in each part and two from each

TASK / SUBJECT	△ + ERRORS (out of 120)	△ + MEAN TIME	△+ ERRORS (out of 20)	ERRORS (out of 20)	MEAN TIME
P1	9	1.42	6	10	1.51
P2	11	2.27	4	5	2.76
P3	5	1.57	2	6	2.87
P4	5	1.24	3	10	1.90
		MEAN 1.63			MEAN 2.26
F1	6	0.62	5	5	0.89
F2	12	1.09	3	8	2.70
F3	14	1.51	3	7	1.86
F4	2	1.84	5	11	2.42
		MEAN 1.27			MEAN 1.97
C1	2	1.32	2	1	1.60
C2	5	2.07	4	8	2.36
C3	18	2.95	4	13	4.21
C4	0	1.13	6	3	0.97
		MEAN 1.87			MEAN 2.29

Table VIII. Errors and mean response latencies (in seconds) on experiment 4(i). "Errors" on equivalence testing refer to responses towards the stimulus different in shape from the original positive stimulus- both stimuli on these tests were rewarded.

group had the same overall shape positive in each part.

(ii) Results. Trials to criterion, errors, response latencies and savings $\left(\frac{\text{errors 2nd}-\text{errors 1st}}{\text{errors 2nd}+\text{errors 1st}}\right)$ task are given in table IX with the aspect of the positive stimulus in the first task (shape or fragmentation) which was also positive in the second part of the task indicated.

The frontal group responded significantly faster on the first part of this discrimination task than did the parietals (Mann Whitney U=0, p=0.014) and the controls (U=1,p=0.029). The differences on the second part of the task did not reach significance.

Discussion. There was again a tendancy in the visual discrimination tasks just described, for frontal monkeys to have shorter response latencies than other monkeys, the difference sometimes reaching statistical significance. This was in agreement with Passingham's finding (Passingham 1972).

There were no intergroup differences in performance on equivalence tests in (i) suggesting that (contrary to prediction) parietal monkeys are not impaired on formation of a Gestalt type perception of a fragmented figure, at least not after the visual experience the parietal monkeys in the present series of experiments had had when they came to perform on this task. It remains possible that no monkeys learnt this discrimination ($+$ and $\triangle$) in terms of the overall dimensions of the stimuli and so none of them was in fact forming a Gestalt when presented with the equivalence pairs. One of the most likely alternative methods of learning this discrimination

TASK	Fragmented H and continuous square			H shape and fragmented outline square				
SUBJECT	TRIALS	ERRORS	MEAN TIME	TRIALS	ERRORS	MEAN TIME	SAVINGS	+VE ASPECT
P1	50	21	1.74	130	60	1.38	-0.48	F *
P2	10	5	3.79	20	6	4.73	-0.09	F
P3	160	68	1.90	10	7	1.48	0.81	S *
P4	10	7	2.07	80	27	1.84	-0.59	S ≠
MEAN			2.38			2.36		
F1	80	23	0.77	50	27	1.62	-0.08	F
F2	40	14	1.51	30	14	2.54	0	F
F3	200	65	1.00	90	16	1.19	0.65	S *
F4	130	42	1.10	90	34	1.26	0.11	S
MEAN			1.10			1.65		
C1	0	2	8.00	70	34	4.70	-0.84	F *
C2	0	1	2.01	30	12	1.73	-0.85	F *
C3	20	11	2.28	50	23	3.06	-0.35	S ≠
C4	120	83	1.34	100	35	2.08	0.41	S *
MEAN			3.41			2.89		

Table IX. Trials to criterion, errors, mean response latencies, savings $\frac{\text{errors 1st} - \text{errors 2nd}}{\text{errors 1st} + \text{errors 2nd}}$ part of task and consistent +ve aspect of both parts of task (fragmentation or shape). Transfer to shape is indicated * and to form (fragmentation or continuous), ≠.

is in terms of the presence or absence of the angles of the triangle. These angles remained intact in both the fragmented triangles and yet seven animals fell to the chance level of responding to the one:one fragmented stimuli, hence it was concluded that at least these monkeys were not using the angles to discriminate.

If it is assumed that the monkeys learned the original discrimination on the grounds of the overall shape of the figures then it must be concluded that all were almost equally capable of "completing" the fragmented forms of the shapes, certainly there was no parietal deficit. In (ii) clearly most of the monkeys (regardless of lesion group) learned the fragmented H/square discrimination according to the shape of the figure since this was the aspect to which they transferred when confronted with the H/fragmented square stimulus pair.

It is suggested that the stimuli in use in this experiment may not have been sufficiently large for the separation of the squares comprising the fragmented figures to have been sufficiently great for a parietal impairment in association of spatially separated input material to be demonstrated. As I have argued previously and as is suggested by Bates and Ettlinger's work (Bates and Ettlinger 1960), the parietal perceptual deficit seems to be abolished by orientation to the relevant material and I would suggest that the information available in the fragmented figures in the present experiment was sufficiently compact for orientation to the whole configuration to take place at once.

It is interesting to note that not all monkeys in (ii) showed positive savings of shape. One parietal and one unoperated control

animal had apparently learned "fragmented versus complete" and ignored the overall shape of the figures. It may therefore be that not all monkeys learn visual pattern discriminations according to the shape of the stimuli even when they have previously learned shape discriminations (experiment 3).

(e) Experiment 5. Perceptual grouping by proximity.

Introduction. This experiment, like the last, was concerned with relationships between parts of the stimulus configuration. It is a well known fact that humans group together visual stimuli which are close to each other in space. Hence AA AA is perceived as two groups of two As whereas A A A A is perceived as four As. It was predicted that normal monkeys would also treat material in this way whereas this form of Gestalt perception may be impaired after posterior parietal lesions.

It was these predictions that this experiment was designed to investigate.

Materials and Methods. Stimuli consisted of black lines (Letraset sheet 557, the second thickest lines) 32 mm long perpendicularly arranged with different spacings on two by two inch Perspex squares. The stimuli were presented as described in experiment 4 (i) using the same two-choice apparatus.

The task was presented as a successive (go-right, go-left) discrimination, left/right positions of reward being varied systematically according to the Gellermann schedule.

Training stimuli were A- two lines with an overall separation of two cm. and B- four lines equally spaced and with an overall separation of three cm.. Monkeys were trained to a criterion of 90% correct (27 out of 30 consecutive trials), two from each lesion group having each stimulus pair indicating "go right". Forty trials were given daily.

Equivalence testing was begun after criterion had been reached on the original task using four pairs of equivalence stimuli (fig. 6). All testing stimulus pairs had four lines, the outer two of which were the same distance apart as those of training pair B. The inner two lines were spaced progressively nearer to the outer ones, being eight, six, four, and two mm. from them. Thus the stimuli appeared to the human observer to be progressively more obviously grouped as two pairs of lines. Four testing sessions were given,each with 10 test trials randomly arranged within the last 30 trials and following 10 training trials. Non differential reward was given on all testing trials.

Following these equivalence test sessions, four more control sessions were carried out, two with each of two stimulus pairs, to ascertain whether the monkeys had learned the original task as two or four lines or by use of cues such as separation from the boundary or between the lines. One set of control stimuli consisted of a pair with two lines close together (one cm. apart) and a pair with four lines widely spaced (four cm.), the other set, one pair with two widely spaced lines (three cm.) and one pair with four lines close together (two cm. overall). These control sessions took the same form as the testing sessions described above.

Figure 6. Diagramatic representation of stimuli in experiment 5. (Not drawn to scale.)

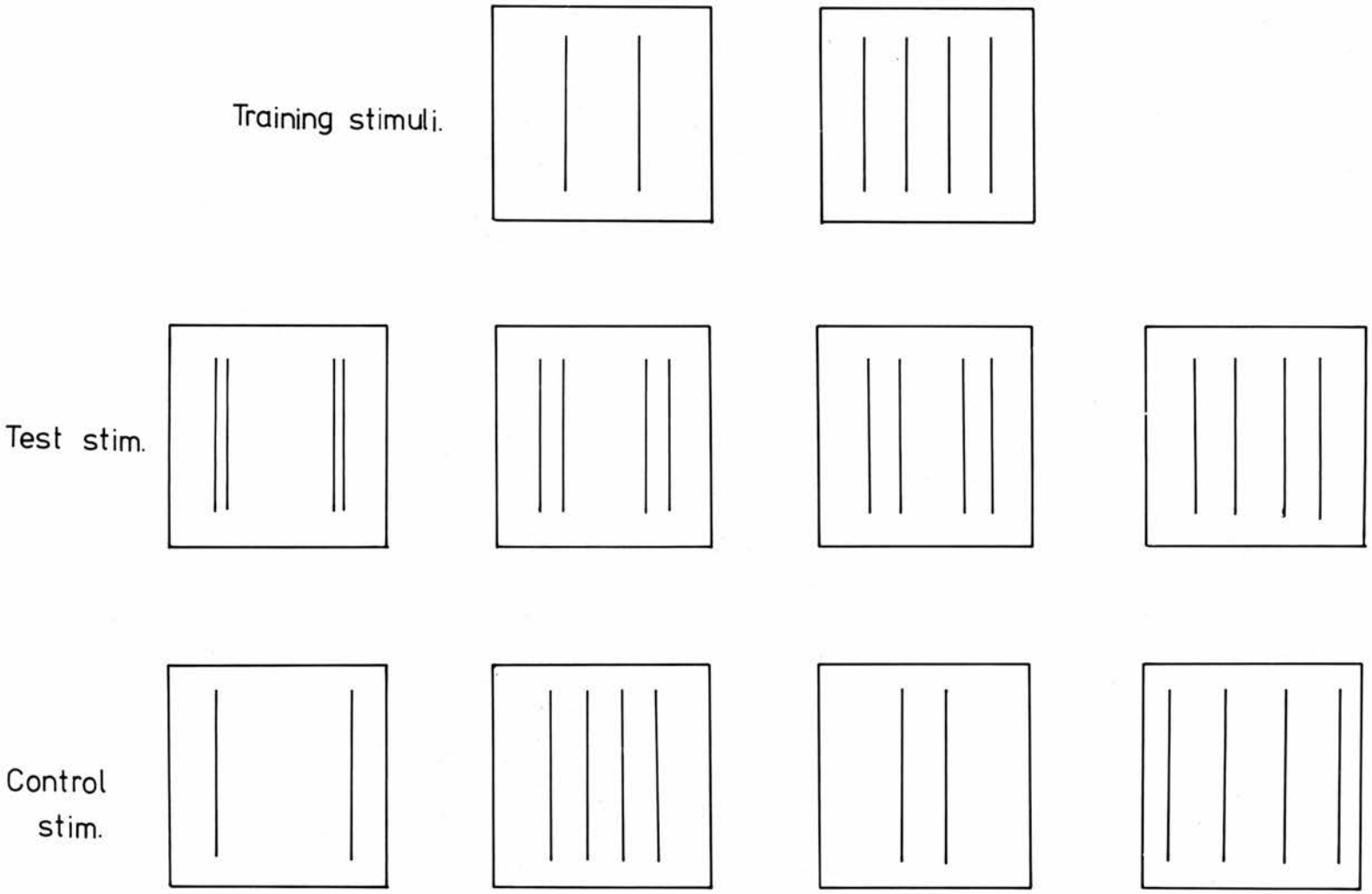


Fig 6

If in any of the testing sessions, performance on the original discrimination fell below the 80% level, the animal was returned to training until he regained the original criterion of 90% over 30 trials.

Results. Results of response to equivalence stimuli are given in table X. Clearly there were no consistent intergroup differences. Five animals treated the material as human subjects would be expected to do, only three deviated markedly from this state and only one of these (F1) appeared to treat all the test stimuli as four separate lines.

Results of the control tests are given in table XI. Only two monkeys showed evidence of having the original discrimination as two versus four lines by responding this way consistently on both control tests.

Response latencies on training trials during the test sessions and on test trials are given in table XII. Frontal animals did not have shorter response latencies than other monkeys on this discrimination.

Discussion. The eight monkeys which responded to the equivalence test stimuli as humans would be expected to do- that is treating two pairs of lines as two lines when grouped close together and as four when further apart appeared initially to be subject to the same proximity grouping laws as guide human perception. The control tests were included to ascertain whether in fact these animals were responding to the two- and four-ness of the stimuli or not.

The results of this experiment were interesting in that they

SUBJECT	‖ ‖	// //	| | | |	\ \ //
P1	9	5	2	1
P2	8	6	2	1
P3	6	7	7	5
P4	5	3	0	2
F1	2	3	2	3
F2	4	7	5	1
F3	7	4	4	3
F4	6	3	4	1
C1	10	6	3	2
C2	10	8	3	0
C3	1	5	3	2
C4	5	2	4	8

Table X. Number of trials out of 10 on which stimuli indicated were treated as two lines in experiment 5.

SUBJECT	STIMULI			
	\ \ (separate)	\\\\ (close)	\\ (close)	\ \\\ (separate)
P1	10	5	9	4
P2	10	7	8	10
P3	10	8	10	6
P4	7	10	6	10
F1	7	9	3	10
F2	4	8	10	8
F3	1	8	9	6
F4	9	7	8	10
C1	3	9	8	9
C2	8	10	6	10
C3	5	6	8	6
C4	8	10	6	10

Table XI. "Correct" responses out of 10 trials to stimuli indicated for control tests in experiment 5.

SUBJECT	TRAINING	TEST TRIALS
P1	2.27	2.18
P2	1.43	1.39
P3	1.75	1.93
P4	1.56	1.81
MEAN	1.75	1.83
F1	0.97	0.96
F2	2.81	4.30
F3	1.78	1.81
F4	1.17	1.27
MEAN	1.68	2.09
C1	1.50	1.79
C2	1.57	1.56
C3	3.42	4.40
C4	1.55	1.86
MEAN	2.01	2.40

Table XII. Mean response latencies for test trials and training trials during equivalence testing sessions in experiment 5.

demonstrated individual variation in the aspect of a stimulus extracted by animals for solution of a discrimination task. These results therefore act as a caution against hasty interpretation of the mechanisms involved in visual discrimination learning. This experiment demonstrated that not all animals extract and use the same information from given stimuli.

Only one animal (P2) which treated the equivalence stimuli in the present experiment as predicted from knowledge of human perception also responded fairly consistently "correctly" (falling to 70% on one stimulus pair) to the control stimuli. It may be concluded reasonably confidently that this monkey had learned the original discrimination as "two versus four" and was perceptually grouping stimuli on testing trials according to proximity.

One animal (F1) treated all equivalence stimuli as equivalent to the training stimuli with four lines, which they were if he was responding to total blackness of the stimuli. He showed no grouping. The control test scores of this monkey indicated that he may have been responding on the basis of the proximity of the two central lines since all stimuli except two distal lines were treated as equivalent to the original stimulus with four lines (the centre two of which were closer than were the two lines of the other training stimulus pair).

Two other monkeys (F3 and C1) also made unexpected interpretations of one set of control stimuli- the widely spaced two lines. These were treated as equivalent to the four equally spaced lines of the original training stimulus. Both pairs of stimuli had the same overall separation (three cm.) and it is suggested that this was evidence of the original discriminations having been learnt in terms

of the distance of the outermost part of the stimulus from the border of the Perspex square.

All except two monkeys came down on some aspect of the control stimuli. All except the three already mentioned went to chance responding to one or more of the control configurations. This indicated the rather disconcerting fact that most monkeys had been responding to some information contained in the training stimuli which was not present in the control stimuli, and not the supposed information of the presence of two or four lines.

This demonstration of the idiosyncratic extraction and use of information from relatively simple visual stimuli by some monkeys must serve as a caution in the assumptions made about animal discrimination learning.

(f) Summary of results of experiments 1-5.

(i) An impairment was found in stylus maze performance following posterior parietal lesions. Control experiments failed to eliminate conclusively the possibility that this impairment was due to a deficit in sensory motor coordination.

(ii) An initial postoperative impairment on a pointer task with stimulus-response discontiguity was found following dorsolateral prefrontal cortical lesions but not following parietal lesions. The effect of stimulus-response separation distance and the interaction of this factor with condition of task presentation (with or without a raisin on the pointer) were significant. The pattern

of errors by parietal monkeys was somewhat different from the others but not significantly so (text fig. 4).

(iii) There were no group differences in ignoring a laterally displaced irrelevant cue.

(iv) Frontal animals generally had shorter response latencies on two-choice visual discrimination tasks than did other monkeys.

(v) There were no differences in recognition and completion of fragmented stimuli between the lesion groups.

(vi) Individual monkeys were shown to use different facets of the same visual stimuli when learning to make a discrimination between two and four vertical lines on a square background.

(g) Experiment 6. An investigation of the effects of cortical lesions in the posterior nonsensory area of the rat on perception of complete and fragmentary horizontal and vertical lines.

Introduction. In 1938 Krechevsky published an account of the perceptual principle of proximity as he had found it applicable to rats. The principle belongs to Gestalt psychology and includes the clause that "when the field contains a number of equal parts, those among them which are in greater proximity will be organized into a higher unit". (Koffka 1936)

Krechevsky found that in a situation where there was a need for a rat to make a particular type of perceptual organization, the principle of proximity was adhered to, the rat selecting continuous lines in preference to fragmented ones of the same orientation even when the fragmented ones had previously indicated reward.

This phenomenon and the associated one in which a rat selects a usually negative fragmented stimulus orientation in favour of continuous lines of the same orientation have proved to be reliable and are known as the "anomolous transfer effect". This effect has been further investigated by Dodwell and coworkers (Dodwell 1970a and b, Dodwell, Litner and Niemi 1970) and by McGonigle and Jones (1973).

Krechevsky interpreted his finding as indicating Gestalt forces of cohesion of "autochthonous" origin. Dodwell however, after ascertaining that the effect was a genuine perceptual one and not one of preference or novelty of stimuli, wrote that (1970a and b) it could be explained in terms of pattern coding. This model assumes that horizontal and vertical components of a visual array are summed separately by the organism, hence the discrimination is made on the basis of the "amount" of horizontality or verticality. This pattern coding system according to Dodwell generates the continuum horizontal, rows, columns, vertical since,for example, horizontal lines have more horizontal component than do rows of squares, hence acting as a super-stimulus. McGonigle and Jones were critical of Dodwell's explanation since they found that dot arrays presented in isolation were not organized by rats as they were when presented in pairs,which should not be so if pattern coding were the explanation of the proximity effect. Hof (1966) also put forward evidence that

the Dodwell type of discrimination of orientation of lines was not active in the rabbit. The mechanism underlying the application of the proximity principle in rats is thus under discussion.

It was proposed that there may be some mechanism active in the formation of Gestalt perceptions of this type in the rat which could be destroyed by cortical lesions in areas other than the striate area. This experiment was devised in an attempt to answer this question. The prestriate area was selected as the area for primary investigation since its anatomical connections resemble those of the posterior parietal cortex of the monkey (chapter I part 4), damage to which was assessed in terms of effects on proximity and closure in perception (experiments 4 and 5).

Materials and Methods. Three hooded rats from the Rowett Research Institute Aberdeen took part in a preliminary experiment to determine the exact location of a prestriate lesion (according to degeneration of thalamic nuclei). Bilateral cortical lesions were made in these rats by aspiration (Meyer and Meyer part II 1971). The positions of these lesions were varied in the antero-posterior plane in order to determine the lesion which was nearest to the striate area without causing damage to it (as assessed by damage to the lateral geniculate nucleus). Three weeks after the surgical intervention, these rats were sacrificed by intraperitoneal injection of Nembutal and the brains were fixed by perfusion with formalin. Frozen sections through the thalamus were cut 40µm thick, stained with toluidine blue and mounted in Euparal.

Twenty male rats of the same strain took part in the main

experiment. The rats were gradually introduced to a food deprivation schedule before training was begun. Food was given for about one hour after the training session each day during training or testing. Water was available ad lib. throughout. The rats were introduced to a modified Lashley Jumping Stand (Lashley 1938). A Y-shaped jumping platform three inches wide and with a separation of nine inches between the branches of the Y was supported four feet from the ground. A ledge four inches by ten inches in front of each stimulus prevented the rats from falling when an incorrect response was made, a net as described by Lashley was not used. The jumping stand was not roofed over and the side walls extended one foot 11 inches from the front wall holding the stimulus apertures. This front wall was two feet wide and contained two six inch apertures separated by six inches. The entire apparatus was painted with Dulux dark grey undercoat. Illumination was provided by a fluorescent tube on the ceiling of the testing room above the centre of the jumping stand. White noise was "piped" to the room. Stimuli were made with black Letraset on nine inch squares of hardboard painted the same matt grey as the rest of the apparatus. These stimuli could be locked in place with turnbuckles. The training stimulus arrays were made from ¼ inch squares arranged in two 12 by six matrices with horizontal:vertical centre to centre ratios of two:one , hence forming one stimulus array with six rows of 12 squares each and one with six columns of 12 squares each. Testing stimuli were six horizontal or vertical lines of the same width and separation as the training stimulus fragmentary lines , and one blank (plain grey) stimulus door.

Preoperative training on the rows/columns discrimination was given to a criterion of 18 out of 20 consecutive correct. Fellows series was used to determine the left-right sequence of presentation (Fellows 1967). A partial correction procedure was used- an animal being allowed three errors on any trial before having the correct response forced upon him (Bitterman 1966). Twenty trials were given daily and each rat was normally tested on about two days each week since it was impossible to test all rats each day. This was not considered important, since the experiment was not concerned with acquistion scores. A jumping distance of 10 to 13 inches was achieved by all rats by the conclusion of preoperative testing. Reward for correct response was three to four wheat grains. The incorrect door was locked.

Following acquisition of this rows/columns habit, rats were put on ad lib. food and assigned one to each of the following groups for surgery:

(i) Group P. Those rats receiving bilateral lesions in the parieto-occipital region which had been formerly ascertained not to encroach to any significant extent on the geniculo striate system. This lesion was estimated to include area 7 and large parts of areas 18, 18a and 39 as described by Krieg (1942). The intended lesion superimposed on Krieg's brain map is shown in text fig.7.

(ii) Group pSt. Animals in this group received small bilateral partial striate lesions to control for any effects of any damage to the striate region which may have occurred in group P. The intended lesion is shown in text fig. 9.

Figure 7. Cortical areas of the rat redrawn from Krieg (1946) showing position of intended posterior lesion on dorsal and lateral views.

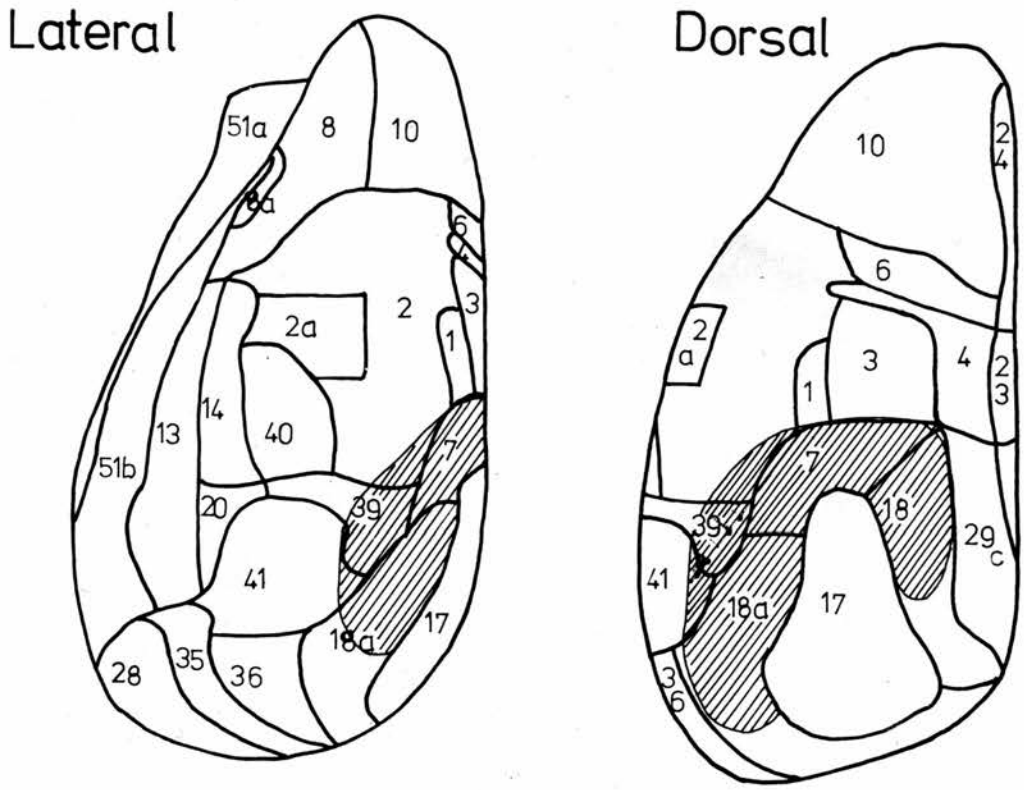

Lateral
51a
8
10
8a
6
2
3
2a
1
14
13
40
51b
20
39
41
18a
17
28
35
36
Dorsal
10
2
4
6
2
a
3
4
2
3
1
7
39
18
29
c
41
18a
17
3
6

Fig 7

Figure 8. Position of intended frontal lesion (striped) superimposed on Krieg's cortical map for the rat.

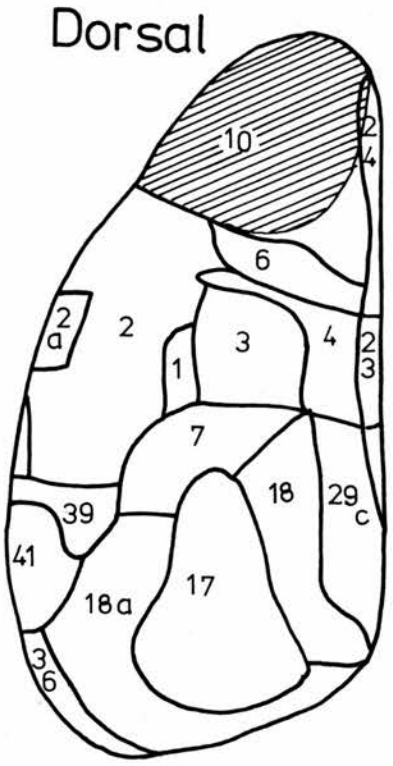


Fig 8

Figure 9. Position of the intended partial striate lesion (striped) on Krieg's cortical map for the rat.

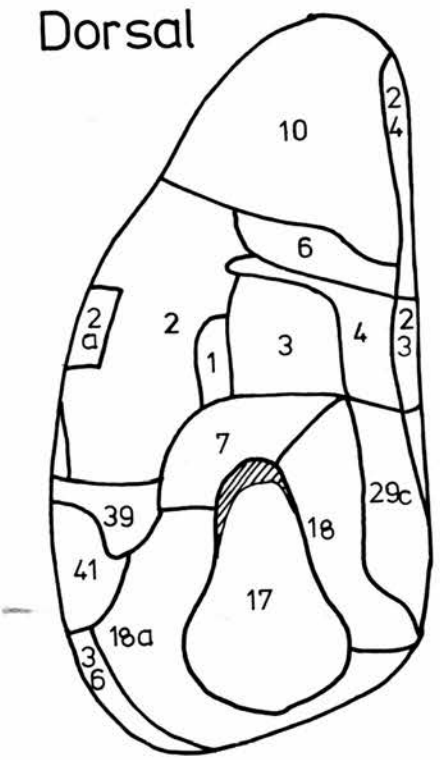


Fig 9

(iii) Group F. This group received fairly large bilateral frontal cortical lesions, the intended site being shown in text fig.8. This lesioned group was included as a control for equipotentiality of function of the cortex in perception of the type investigated.

(iv) Group S. A control group of sham operates.

Surgery was performed under Nembutal anaesthesia according to the methods described by Meyer and Meyer (1971). Surgery was carried out by Dr. M.A.Goodale and Dr. A.D.Milner with some assistance from me.

One of every four rats to reach criterion on the preoperative task was assigned to each group. The one of the four which had taken the fewest acquisition trials to reach criterion was assigned to the P group, the next to the pSt group, then the F group, then S, so as to reduce apparent post-operative defects of P compared with S rats. There were five rats in each group and as far as possible they were split evenly according to whether rows or columns had been the positive stimulus.

Seven days after surgery rats were returned to food deprivation and three days later to behavioural testing. Rats were retrained to the same criterion of 18 out of 20 on their original task. Jumping distance was reduced to seven inches on initial postoperative testing to reduce falls and was increased gradually to nine inches at which distance all testing sessions were performed. Postoperative testing (20 trials daily) was carried out every day on each rat. The day following reacquisition of the rows/columns habit, equivalence testing was begun. The following six equivalence test stimulus pairs

were presented to each rat:

(i) The original positive stimulus and stripes of the same orientation.

(ii) The original negative stimulus and stripes of the same orientation as it.

(iii) Continuous stripes of the same orientation as the original positive stimulus and blank door.

(iv) Continuous stripes of the same orientation as the originally negative stimulus and blank.

(v) The original positive (rewarded) stimulus and blank.

(vi) The original negative stimulus and blank.

The order of presentation of these stimulus pairs was counterbalanced according to a six by six Latin square design.

Each equivalence test session was preceded by five trials on the original training task. If more than one error was made, the session was converted to a training session until the criterion of nine out of 10 consecutive correct trials was reached. Otherwise the equivalence testing session continued with five test trials randomly arranged among the next 15 trials. Two sessions on each equivalence test pair, that is 60 test trials altogether over 12 testing sessions were given. If more than three errors were made on all the training trials in a testing session (that is if performance fell below the 80% level), the session was abandoned and the animal retrained to the criterion of nine out of 10.

Results. Informal observation revealed some degree of abnormal placing and neglect of hind limbs in all the P group of rats.

Pre and postoperative acquisition scores on the original task are given in table XIII and results of the equivalence testing in table XIV.

Comparisons of results between members of each equivalence stimulus pair for each individual subject were significant (Binomial test) where indicated. Comparison of responses as predicted to test pairs iii and iv combined, with v and vi combined yielded no significant difference (Wilcoxon matched pairs signed ranks test). Analysis of variance overall revealed no difference between the four surgical groups ($F<1$). The effect of the different stimulus pairs was significant beyond the 0.01 level ($F=6.52$, df=5 and 80).

Discussion. Table XIV clearly shows that the anomolous transfer effect as described by Krechevsky and Dodwell did not take place in general. In fact only five rats showed the positive anomolous transfer effect to a significant degree, and three showed the negative effect.

Regardless of the surgical intervention in the animals of the present study, there was one important way in which the experimental design differed from all the previous demonstrations of the anomolous transfer effect. That is that the rats in the present experiment were trained (to the same criterion, on the same discrimination apparatus and using the same stimulus dimensions as were used by , for example, Dodwell 1970b),then were left without training and on ad lib. diet for a minimum period of two and a half weeks before being retrained to criterion on the same task and then undergoing equivalence testing. Hence there was some degree of overtraining

SUBJECT	TRIALS TO CRITERION PRE-OP.	POST-OP.	+VE STIMULUS
P1	183	118	COLUMNS
P2	221	118	"
pSt1	220	237	"
pSt3	285	336	"
pSt5	460	85	"
F1	221	349	"
F4	263	228	"
S1	246	329	"
S2	254	112	"
S5	589	82	"
P3	164	228	ROWS
P4	207	51	"
P5	421	136	"
pSt2	247	125	"
pSt4	218	156	"
F2	252	21	"
F3	309	97	"
F5	550	35	"
S3	328	0	"
S4	334	162	"

Table XIII. Pre and postoperative trials to criterion on columns/rows discrimination task.

Table XIV. Results of equivalence testing on rats in experiment 6. Scores given refer to response out of 10 to the first member of the pair of equivalence stimuli mentioned.

	EQUIVALENCE TEST STIMULUS PAIR.					
	Continuous stripes in same orientation as +ve stim. and +ve stim.	Continuous stripes in same orientation as -ve stim. and -ve stim.	Continuous stripes in same orientation as +ve stim. and Blank.	Continuous stripes in same orientation as -ve stim. and Blank.	Original +ve stim. and Blank.	Original -ve stim. and Blank.
SUBJECT	(i)	(ii)	(iii)	(iv)	(v)	(vi)
P1	8	3	7	0*	6	1*
2	7	1*	6	1*	6	3
3	10*	3	7	6	7	2
4	10*	6	5	2	2	1*
5	7	7	8	3	5	4
pSt1	0/	4	8	5	10*	8
2	6	5	9*	1*	7	0*
3	4	3	6	5	9*	3
4	7	5	5	5	6	2
5	9*	7	7	4	5	1*
F1	9*	7	8	3	4	3
2	8	8	5	5	7	2
3	2	2	10*	4	10*	10/
4	10*	8	6	2	5	1*
5	3	4	5	5	1	2
S1	8	8	4	3	2	0*
2	4	1*	7	5	6	8
3	6	1*	10*	5	9*	6
4	5	2	10*	2	9*	8
5	4	5	4	6	5	5

*Significant in the predicted direction beyond the 0.01 level.
/Significant in the opposite direction to that predicted beyond the 0.001 level.

after the animals originally reached criterion on the training task. It is known that overlearning leads to inflexibility (Gilhousen 1931) and this is discussed with regard to the Sutherland-Mackintosh 2-stage theory of discrimination learning and other theories by Fellows (1968 chapter 8). If overtraining in the present study was responsible for the failure to demonstrate anomolous transfer, it would be predicted that the transfer results would instead show evidence of absolute responding, that is responding always to the training positive stimulus and avoiding the training negative stimulus regardless of the other member of the simultaneously presented stimulus pair.

If the first two columns of table XIV are considered (the results of an attempt to replicate the positive and negative anomolous transfer effects respectively), it is seen that responding to these stimulus pairs was never in the direction of absolute responding over the two columns for any rat. It is however of interest to note that the rats in the present study showed much more tendancy to respond to the training positive stimulus and to avoid the training negative stimulus when they were paired with a blank than did the rats in McGonigle and Jones's study using the same stimulus pairs. In fact in the present study the orienting of the rows and columns stimuli in isolation was as good as the orienting of the horizontal and vertical lines (there was no significant difference between "hits" on the two kinds of stimuli when each was paired with the blank). This does suggest that at least some of these rats showed a tendancy towards absolute responding, since in McGonigle and Jones's experiment, it was shown that rats could not organize rows

and columns arrays without the appropriate comparison stimuli. Hence it remains possible that it was this tendancy which disrupted performance on the anomolous transfer trials (columns i and ii table XIV).

Warren and McGonigle (1969) wrote a cautionary note on interpretation of equivalence test results based on their work with cats. They found cats given non-differential reward on equivalence test trials (the method used in the present experiment) made significantly more appropriate responses than those to which differential reward was given on generalization tasks and that "negative results on equivalence tests with non-differential reinforcement cannot be construed as unequivocal evidence of perceptual incapacity in animal subjects". It is nevertheless necessary to explain why the present experiment failed to replicate the results of earlier studies.

While there was some indication of a tendancy towards rigidity of response to the training stimuli as discussed above, if the results of individual rats (table XIV) are examined, it may be noted that some rats formed strong preferences for one form of stimulus configuration (continuous or fragmented lines or blank). It is particularly interesting that all animals in the frontal lesioned group responded fairly rigidly to either the continuous or fragmented lines in columns i and ii (table XIV), that is, regardless of orientation. Whether or not any weight should be put on this finding in view of the unexpected variability of all these results, is doubtful but it is tempting to draw a likeness between this response tendancy and the well known finding of perseveration in the higher mammals after frontal cortical ablation (e.g. Mishkin

1964).

The most obvious retrospective criticism of this experiment is that there was no group run on the same design as were the previous demonstrations of anomolous transfer, that is progressing straight from the attainment of criterion on the training task to equivalence testing. The reason for the exclusion of such a group was that the anomolous transfer effect was reported to be very reliable (Dodwell 1970b) and previous reports of failure to demonstrate it have not been made. It was thus considered highly unlikely that the difference in experimental design used here would disrupt this effect in sham operated rats, and so in the interests of economy, an unoperated group which passed directly from original training to equivalence testing was not included. The effect of overtraining cannot therefore be evaluated.

If the fifth group discussed above had been included and had failed to show the classical phenomena, a strain difference could have been suggested (since training methods were the same). Arguments based on acuity differences between **different** rat strains could have been raised (Lashley 1930). From the starting point of the Lashley Jumping stand with the Y shaped jumping platform as used in the present experiment and by McGonigle and Jones, vision of the individual squares or the smaller gaps used to make up the row and column arrays was only just possible for hooded rats according to the minimum visual angles given by Lashley (about 25 feet of arc) and not at all for albino rats, although at the actual jumping distance (about 12 inches) where the response is made, the visual angle would be $1^{\circ}12'$ (easily seen by hooded rats but near threshold

level for albino rats). It would thus be predicted from Lashley's figures for minimum visual angles that albino and hooded rats, when confronted with the dot arrays used would "see" them differently.

Dodwell raises the question of receptive field sizes (Dodwell 1970b) with respect to his receptive field coding explanation of anomolous transfer, his point being that two small squares constituting the array must be sufficiently close to activate the same cortical receptive field for his explanation in terms of Hubel and Wiesel's findings in the cat (1962) and monkey (1968) to hold. Dodwell's experiments used hooded rats whose acuity should be sufficiently adequate to separate the individual squares of the dot arrays. According to Lashley's figures however, the visual acuity of the albino rat is sufficiently poor for the closest edges of two small squares along a row or column to be seen as one, hence the whole row would appear as an approximation to a continuous line. If this is so, the "anomolous transfer" demonstrated in albino rats on a jumping stand (McGonigle and Jones) was not so "anomolous" after all since actual continuous lines understandably appeared as better representations of fragmented lines which (because of poor acuity) appeared as continuous lines in a certain orientation. If this were the case, it would be expected that the dot patterns could be organized almost as efficiently as are the horizontal and vertical line displays which they were not in McGonigle and Jone's experiment, so if visual acuity in these rats is as poor as Lashley thought, there must be some other explanation of this. It does remain possible that strain differences in visual acuity with the strain used in the present experiment perhaps having particularly good

acuity (which could explain the rather high acquisition scores since the rats would actually be learning in terms of dot organization rather than approximation to lines) are responsible for the discrepancy in experimental findings. Further experimentation is necessary before this possible explanation of the failure in the present experiment to demonstrate the anomolous transfer effect is rejected.

CHAPTER III. DISCUSSION

1. The results of the present studies in relation to previous studies.

The only significant impairment following posterior parietal lesions to be demonstrated in the series of experiments just described was on a stylus maze task. The possibility that this was caused by a loss of motor coordination was not eliminated. Posterior parietal lesioned monkeys were not found to be impaired on tasks with stimulus and reward/response site spatially discontiguous contrary to predictions from other studies and from human brain lesioned material. It is suggested that this was due to the method of training on the first S-R separation task, the pointer task, and hence to the way in which the monkeys learned to respond to material. The finding of Bates and Ettlinger's (1960) that the parietal deficit on tasks involving S-R separation could be overcome, apparently by learning to orient to the relevant (stimulus) material could be applicable to the present experiments and it is suggested that the basis of the posterior parietal impairment in monkeys as observed by for example Pohl (1970 and 1973) is a defect in orientation or selective attention which can be overcome by suitable training methods.

If it should prove to be the case that the posterior parietal deficit in monkeys is one of selective attention, this would be evidence of an apparently fundamental difference between the functions of this area in monkeys and of the homologous area in man. How fundamental this difference is depends on the outcome of the attempts

at present under way to redefine the "agnosias" described after brain damage in man. Animal experiments are however showing at least a considerable quantitative difference between man and other animals in the effects of ablation of "association" areas. Unless it should be disclosed, when the brains of the monkeys which took part in the present series of experiments are examined histologically, that the lesions made were small and incomplete, there is also evidence that effects on S-R separation tasks are not so reliable as are the effects on spatial perceptual behaviour of humans.

It must always be borne in mind when comparing the effects of brain damage in man and animals that the human brain is damaged to an unknown extent by a penetrating missile or a pathological entity (for which a surgical excision may have been made) whereas in animals tissue is removed from a relatively controllable area of a normal brain and the extent of at least gross damage can be assessed before final interpretation of results is made.

A positive finding of the experiments with monkeys was that following dorsolateral frontal cortical lesions, monkeys' response latencies on visual discrimination tasks were generally reduced. The existence of some confusion between the effects of dorsolateral and orbital frontal lesions in man and monkey has already been mentioned. There seem to be parallels between the effects of dorsolateral frontal lesions in man and of orbital frontal lesions in monkey, in each case the ability to suppress responses apparently being affected. The reduced response latencies of the dorsolateral frontal monkeys in the present experiments compared with unoperated controls may also have been due to an impairment in response

inhibition. This explanation is given further support by the observation that the frontal monkeys on the pointer task (experiment 2) made more errors on initial postoperative trials than did controls. This finding seems to tally with Milner's (Milner 1964) suggestion that humans with dorsolateral frontal brain damage have reduced interest in avoiding errors.

2. Some speculations and suggestions.

In outlining the anatomical connections relevant to "association" cortex, the connections between the pulvinar nucleus and the superior colliculus in some species were mentioned (e.g. Altman and Carpenter 1961 in the cat). The pulvinar, it was also stressed in chapter I part 2, is the main subcortical connection with the posterior association cortex. There is increasing evidence that the superior colliculus is important in eye movement control and in perception of real movement (Schiller and Koerner 1971) apart from being (along with the nucleus lateralis posterior- forerunner of the pulvinar in higher mammals) an important relay of visual input in the more primitive forms.

Some effects of ablation of superior colliculus on "higher" functions in animals have been recorded as the following examples show. Porter and Rioch (1962) found an impairment in visual recognition in the contralateral half field after deep unilateral collicular lesions (in the stratum profundum) in three chimpanzees. Barnes, Smith and Latto (1970) found superior collicular lesions impaired orientation to brightness but <u>not</u> to form in the rat.

Schneider (1969) abolished orienting to visual and auditory stimuli by undercutting the superior colliculi in hamsters and, also in 1969, Anderson and Symmes found rate of movement discrimination impaired in monkeys following colliculectomy. Sprague (1966) cast some light on the function of the subcortical connections of the posterior association (occipitotemporal) cortex by identifying, by collicular and cortical lesions, an interaction of the two areas. Humphrey however in 1968 did not find the properties of superior colliculus altered by neocortical ablation, but he was recording in the superficial layers of superior colliculus and was primarily concerned with visual sensory information of a simple kind.

The behavioural findings and the anatomical connections suggest that the functions of the superior colliculus and the parieto-temporo-preoccipital cortex (which Blum, Chow and Pribram 1950 suggested acts as a complex) may be linked. Barlow (1970), between his elaborate analogies of disorders caused by parietal damage in man and function of modern navigational systems, suggested that difficulties in visual- occulomotor integration may be the basic nature of the parietal impairment.

Humphrey (1968) attributed superior collicular function in rats and monkeys to visual attention which it is suggested may be the mechanism impaired by posterior parietal ablation in monkeys. It is suggested that visual orientation may be subserved by an interaction between posterior association cortex and superior colliculi in an intact animal.

A complex cortical/subcortical interaction in control of perceptual mechanisms of the type under discussion presents problems as far as experimental investigations are concerned. Electrical

recordings from the relevant brain areas would give indications of activity during certain types of behaviour but to identify the way in which such areas may be interacting to form perceptions of say spatial relationships would probably not be possible at present in so complicated a system. Lesions studies could give indications (as they already have to a small extent) of the possible existence of such a system as the one suggested but the results of such studies are very difficult to evaluate and these could not lead to definitive answers. Positive results may be precipitated by unknown damage caused by surgery and negative results may be because of insufficient damage or because the area ablated, although involved in a given function in an intact animal, is not essential for the behaviour measured.

To continue the criticism of lesion experiments, the experiment with rats as subjects described in chapter II was of a typical simple type. The effects of lesions in an area thought to be involved in formation of a particular type of perception on a well documented behavioural phenomenon were investigated. The failure of the lesioned animals to deviate from controls in their behaviour on the given task does not indicate anything about the physiological mechanism underlying this behaviour. Only if it could be shown that material is perceived one way before surgical intervention and is reliably perceived another way afterwards, is there evidence that the intervention has altered its perception, but still the way in which it has altered it cannot be known.

Pure physiology is a long way from an understanding of the perceptual mechanisms of such complex systems as mammalian brain.

Lesion experiments on this organ as attempts to gain understanding of these complex mechanisms are of doubtful value at present on account of the impossibility of satisfactoy interpretation. It is hoped that there will soom come a time when today's experiments can be interpreted in terms of the physiological mechanisms of psychological function.

SUMMARY

A review of the clinical effects of damage in the posterior parietal area of the brain was given followed by a comparative anatomical and psychological survey of the problem of definition of association cortex.

Five experiments with 12 Macaca arctoides as subjects were described. Four of the monkeys were given bilateral posterior parietal cortical lesions and 4 bilateral dorsolateral frontal cortical lesions. The remaining 4 monkeys served as unoperated controls. The 5 experiments were designed to measure performance on aspects of visual perception of spatial relationships. All tasks were presented in a Wisconsin General Testing Apparatus. It was predicted from human and other animal studies that parietal monkeys would be impaired on tasks of this type.

Parietal monkeys were impaired only on a stylus maze task and control tests failed to eliminate the possibility that this may have been due to sensory motor incoordination rather than a higher order perceptual defect.

The failure of the present study to demonstrate parietal impairment on stimulus-response separation tasks or tasks involving completion of fragmented patterns was attributed to the initial training given and it was suggested that all monkeys had learned to orient to a relevant stimulus regardless of position.

On a task involving grouping of lines by proximity in two groups of two, it was demonstrated that results can be misleading and individual animals use different cues to learn the same discrimination.

Dorsolateral frontal monkeys were shown to have generally shorter response

latencies on two choice visual discrimination problems. It was suggested that this was due to a disturbance of response inhibition.

An attempt was made to replicate the anomolous transfer effect in rats and to affect the organization of dot arrays presented visually, by lesions in prestriate cortex. The anomolous transfer effect was shown to be less reliable than has been hitherto reported and the possibility that slight overtraining or strain differences may affect it was raised.

Attention was drawn to the anatomical connections of superior colliculus and pulvinar nucleus and some behavioural consequences of superior collicular ablation were mentioned. The possibility that the superior colliculus and the posterior parietal cortex may be jointly involved in spatial perception was put forward. It was suggested that the deficit described by some following posterior parietal ablation in monkeys may be due to selective attention loss.

The question of the usefulness of lesion experiments to psychology at the present stage of physiological knowledge was raised.

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