TELENCEPHALIC EFFERENT PATHWAYS AND FEEDING BEHAVIOR IN THE PIGEON (COLUMBA LIVIA)

BY

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ABSTRACT

TELENCEPHALIC EFFERENT PATHWAYS AND FEEDING BEHAVIOR IN THE PIGEON (COLUMBA LIVIA)

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The present study examined the role of two extratelencephalic efferent pathways in the feeding behavior of the pigeon. The structures chosen for study, the occipitomesencephalic tract (OMT) and the septomesencephalic tract (SMT), arise in the archistriatum and hyperstriatum, respectively, and convey information to brain stem and spinal cord nuclei related to sensory systems (somatomotor and visual) involved in the exteroceptive control of feeding in this species. In addition, both tracts have been homologized with identified components of the mammalian pyramidal system.

Electrolytic lesions were placed in these tracts and their effects upon several measures of ingestive behaviors were examined. While there was no evidence for its direct involvement in drinking, OMT damage resulted in a reduced responsiveness to food which was manifested by periods of aphagia, hypophagia and decreased body weight. Furthermore, OMT birds evidenced a disruption of the consummatory response of pecking which was characterized by impairments in grasping (incomplete opening of the oral aperture) and pecking inaccuracy ("overshoots"). Finally, OMT damage

temporarily abolished operant key-pecking reinforced with food. Resumption of key-pecking was marked by significant reductions in feeding and consummatory efficiency, which, in several cases, led to extinction of the key-pecking response. Lengthening the reinforcement intervals, permitting more feeding time reinstated preoperative levels of key-pecking. However, all other behavioral measures showed persistent impairments. These findings indicate that OMT damage produces both consummatory (somatomotor) and "motivational" (responsiveness) deficits.

SMT lesions resulted in reduced food intake, but did not directly affect water intake. Unlike OMT birds, there were no decreases in responsiveness to food. Furthermore, there were no observable changes in the efficiency or accuracy of their feeding response and only transient reduction in operant responding. SMT birds, however, displayed inappropriate "sorting" behavior which suggests that the lesions may have affected visually controlled preference behavior.

OMT lesions produced feeding behavior deficits similar to those resulting from damage to central trigeminal structures previously implicated in the control of feeding behavior in the pigeon. In view of the anatomical connections between both sets of structures, these findings suggest that the OMT may be part of the pigeon's putative neural feeding system. Moreover, a comparison of the behavioral deficits produced by OMT damage with that produced by mammalian pyradotomy provide some behavioral confirmation for the anatomical homology between components of the two systems.

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Above all, I am grateful to my wife, Karol, whose love and patient understanding sustained me throughout the completion of this work, and to my daughter, Jessica, who provided delightful diversion from the many hours involved in thinking and writing. To my family I dedicate this work.

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INTRODUCTION

The diversity of food niches among extant animal species testifies to the broad spectrum of evolutionary solutions generated by a major problem of life: An organism within its natural environment must procure adequate amounts of food-stuffs without becoming part of the energy intake of another organism further along on the food chain. To insure that feeding opportunities are exploited, however, organisms must possess mechanisms responsive to the environmental matrix in which food is embedded, as well as mechanisms capable of monitoring or anticipating energy output or metabolic need. In this manner intake can be optimized within the space and time allotted for feeding.

The search for neural mechanisms controlling the various aspects of ingestive behavior has had a long and controversial past. Due in part to the anthropocentricism among behavioral workers and in part to a set of beliefs regarding cerebral phylogeny among neuroanatomical workers, the bulk of research effort on the mechanisms controlling vertebrate feeding has focused on but a few mammalian species -- most notably the laboratory rat -- while little or no systematic data has been provided on the ingestive behavior mechanisms in other vertebrate classes.

The paucity of studies utilizing avian species is somewhat surprising in view of the impetus provided to early neuro-behavioral workers by Flourens' classical demonstrations of

the behavior of decerebrate pigeons. However, the neglect of avian neurobehavioral mechanisms, in general, may have been related to the notion, at one time ubiquitously upheld by comparative neuroanatomists, that the brains of birds and mammals represent divergent directions of vertebrate evolution. Within recent years, Karten and his associates have challenged those beliefs (Cohen & Karten, 1974). In providing evidence for structural and functional homology in corresponding sensory and motor subsystems among species of birds and mammals, Karten has succeeded in establishing a neuroanatomical foundation for the systematic study of avian neurobehavioral mechanisms. As a result of these efforts, the last few years have witnessed a revival of interest among workers in the discovery of the neural bases of many classes of avian behavior.

Much of this recent work with birds has been performed on the pigeon, reflecting, in part, the utility of this species for neurobehavioral research and, in part, the tacit agreement among investigators on the pigeon's merit as a "representative" of the class Aves. Zeigler, whose work serves as an example of the renewed emphasis upon the comparative approach to the study of feeding, recently summarized those characteristics of the pigeon that make it such an excellent preparation for the experimental analysis of avian feeding behavior (Zeigler, 1974). From the behavioral standpoint, the most important factors include the pigeon's use of distinctive motor patterns in feeding and drinking and the relative ease with which its feeding

response, the peck, can be recorded and quantified in binary fashion. In addition, the pigeon's consummatory response is distinguished by a fairly high degree of stereotypy enabling a precise qualitative and quantitative analysis of the individual components of the movement pattern (Zeigler, 1976). These properties of the pigeon's consummatory activity have facilitated the characterization of behavioral deficits produced by brain lesions. In view of these characteristics and the growing body of evidence suggesting neuroanatomical homology among species of birds and mammals, Zeigler has suggested that the pigeon may serve as a useful "model system" for the neurobehavioral study of vertebrate feeding (Zeigler, 1976).

Neurobehavioral Studies of Feeding in the Pigeon

Since the early work of Rolando (1809) and Flourens (1824), it has been known that a pigeon deprived of its forebrain, while remaining fully capable of performing such complex activities as grooming, walking and even flying, ceases to initiate feeding and drinking (Akerman, Fabricius, Larsson & Steen, 1962; Brunelli, Magni, Moruzzi & Musumeci, 1972; Ferrier, 1886; Shaklee, 1921). Its swallowing reflex remains intact, so that the animal may be maintained satisfactorily for many months by hand-feeding.

A possible locus for the forebrain region specifically related to feeding was first suggested by Edinger (1908).

Noting that in many species of reptiles and birds there existed a prominent fiber pathway extending from the nucleus of the trigeminal nerve to the base of the anterior forebrain, Edinger postulated that cells within this forebrain region might subserve an "oral" sense functionally concerned with feeding. Several years later, Rogers (1922) found that aphagia could be produced by damage to restricted basal forebrain regions. More recently, a number of studies have demonstrated the effectiveness of basal forebrain stimulation in eliciting the feeding response or several of its components, such as pecking, "salivating", "chewing", tongue protrusion, and swallowing (Brown, 1973; Delius, 1971; Gentle, 1973; Putkonen, 1967; Showers, 1973).

Over the past few years, the collaborative studies of Zeigler and Karten have resulted in a more precise delimitation of those forebrain areas whose destruction is responsible for the aphagia observed in the decerebrate pigeon. Moreover, the nuclei involved have been shown to be part of a network of structures at several levels of the avian brain.

The afferent limb of this network includes central components of the avian trigeminal system. The Principal Sensory Trigeminal Nucleus (PrV) receives a topographically organized projection from the Gasserian (trigeminal) ganglion and is the origin of an ascending projection to the endbrain, the quinto-frontal tract (QFT). Arising from both dorsal and ventral subdivisions of PrV, the quinto-frontal tract undergoes partial decussation and projects bilaterally and monosynap-

tically to the basal portion of the hemisphere (Wallenberg, 1903; Woodburne, 1936; Cohen & Karten, 1974). It enters the telencephalon with the lateral forebrain bundle, passes through the paleostriatum primitivum and augmentatum to terminate in the nucleus basalis (NB) in the basolateral portion of the anterior telencephalon. On the basis of comparative anatomical data from a number of avian species, Stingelin (1961) has noted that the relative size of NB is directly proportional to the magnitude of PrV, which, in turn, reflects the relative extent of beak development in the species.

The nucleus basalis is the source of an efferent projection, the tractus fronto-archistriaticus (FAT), which terminates in a region of the caudal neostriatum overlying the dorso-lateral nucleus of the archistriatum and in the anterior subdivision of the archistriatum proper (Zeier & Karten, 1971). The archistriatum, in turn, is the origin of a major descending pathway, the occipitomesencephalic tract (OMT). Preliminary data from Zeigler's laboratory have implicated all these structures (FAT, archistriatum, and OMT) in the control of feeding (Zeigler, 1976).

The Trigeminal System and Feeding Behavior in the Pigeon

Following their identification of this group of structures, Zeigler and Karten focused their research efforts upon one component of this network, the trigeminal system. Bilateral destruction of central trigeminal nerve structures (PrV, QFT, NB) were shown to produce periods of aphagia and hypophagia

lasting from several days to several weeks, depending upon the size and placement of the lesion (Zeigler & Karten, 1973). Control lesions placed in adjacent tissue (including the lateral hypothalamus) had no significant effects. While the rate of body weight loss in the lesioned animals paralleled that of normals food deprived for an equivalent period of time, once feeding resumed, trigeminal birds did not display the compensatory overeating characteristic of the normal food-deprived pigeon. In fact, most of these birds displayed a permanent hypophagia together with a stable, but much reduced body weight. Significantly, central trigeminal lesions did not directly impair drinking behavior.

In addition to its effect on food intake, injury to afferent trigeminal structures has been shown, not surprisingly, to disrupt the neurosensory control of the consummatory response. The resulting marked reduction in feeding efficiency thereby raised the possibility that the aphagia and reduced intake of lesioned birds might be due to a disruption of sensorimotor mechanisms rather than to a reduction in the bird's responsiveness to food. In an effort to distinguish between these two alternatives, feeding responses of lesioned birds were monitored with food boxes that enabled the recording of individual pecking responses (Zeigler & Feldstein, 1971). An examination of the relation between food intake and feedometer responses together with direct observations of feeding efficiency showed the reduction in food intake following trigeminal lesions to be due both to a reduction in the efficiency of the feeding response and to a reduction in the bird's responsiveness to

food.

In a further attempt to dissociate lesion effects upon the sensorimotor and motivational process underlying feeding, an operant paradigm was employed. Although central trigeminal lesions did not disrupt key-pecking reinforced with water, they abolished food reinforced key-pecking for periods ranging from a few days to a few weeks.

Taken together, these neurobehavioral studies indicate that lesions of central trigeminal structures, while they do not affect drinking, produce a variety of feeding behavior deficits. These effects may be experimentally dissociated into "sensorimotor" or "responsiveness" deficits; however, a given lesion invariably produces both types of deficit. Thus Zeigler and Karten have concluded that the quintofrontal system (PrV, QFT, and NB) is involved in both the neurosensory and motivational control of feeding in the pigeon.

Efferent Forebrain Pathways and Feeding Behavior in the Pigeon

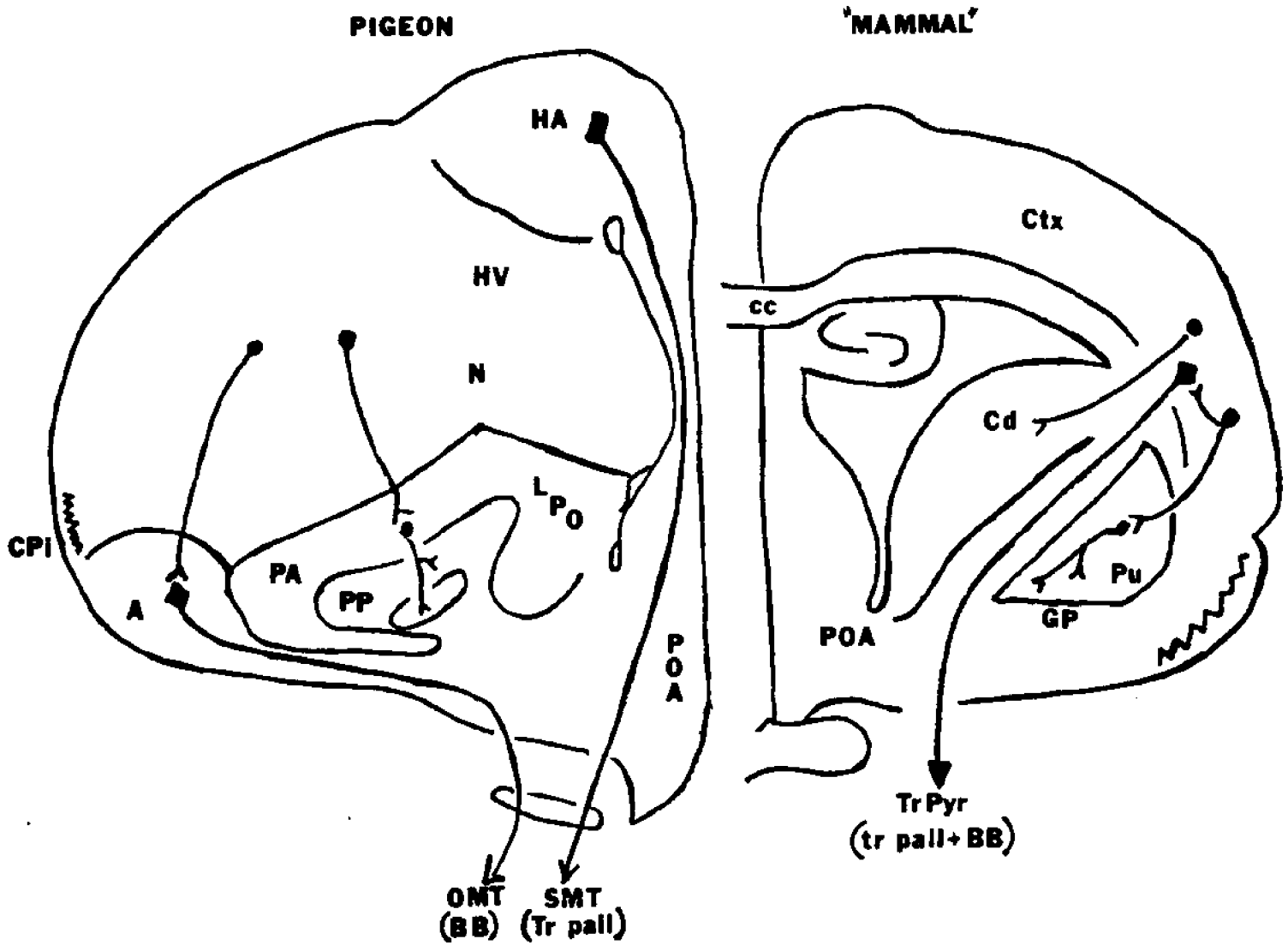
It is the aim of this thesis to examine the contribution made by two of the pigeon's major efferent forebrain pathways. The structures chosen for study, the occipitomesencephalic tract and the septomesencephalic tract, represent, respectively, output pathways from somatosensory and visual forebrain areas of the pigeon -- two afferent systems which are likely to be critically involved in the exteroceptive control of feeding in this species.

OMT and SMT: Hodological Studies

A. The occipitomesencephalic tract. As first described by Edinger in 1908, the occipitomesencephalic tract (OMT) arises from cell bodies within the archistriatum of the forebrain (Figure 1). Morphologically, the archistriatum is a relatively heterogeneous cellular mass divisible into at least four major nuclear regions, each receiving input from a wide array of forebrain areas (Zeier & Karten, 1971). The OMT originates principally from the anterior two-thirds of the archistriatum, an area which, on the basis of its cyto-morphology and connections, has been considered the avian equivalent of the mammalian somatosensorimotor cortex (Zeier & Karten, 1971). Preliminary studies from Zeigler's laboratory have implicated this region of the archistriatum in the control of feeding (Zeigler, Silver & Karten, 1969).

The OMT has a fairly extensive range of terminations, providing input to structures located at many levels of the brain. Ipsilaterally, the OMT has been traced to the thalamic spiriform complex and dorsal nuclear group, and to the mid-brain lateral reticular formation, lateral pontine nuclei and locus coeruleus. In addition, the OMT terminates ipsi-laterally in the deep layers of the optic tectum, in brain stem trigeminal nuclei, and in the gracile and cuneate nuclei of the medulla. A portion of the tract crosses in the anterior commissure and provides input to corresponding nuclei of the lower brainstem as well as to cells within the base of the

FIGURE 1


PIGEON
'MAMMAL'
HA
HV
N
CPi
A
PA
PP
LPO
POA
OMT
(BB)
SMT
(Tr pall)
Ctx
cc
Cd
Pu
GP
POA
TrPyr
(tr pall+BB)

dorsal spinal columns. Based on its origins and the nature of its terminations within the brainstem, Karten has suggested that the OMT should be considered the functional equivalent of at least a portion of the mammalian pryramidal tract (Karten & Dubbeldam, 1973). A pathway with similar origin and terminations, the Bundle of Bagley, has been described in the ungulate brain as a variant of the primate pyramidal tract (Haarsten & Verhaart, 1967; Towe, 1973).

B. The septomesencephalic tract. The septomesencephalic tract (SMT) arises from the dorsal telencephalon and contains cell bodies originating primarily from the anterior and posterior portions of the Wilst (Karten, Hodos, Nauta & Revzin, 1973). The Wulst, a bump-like structure comprising the upper layer of the hyperstriatum (Figure 1), contains third-order cells of the pigeon's thalamofugal or 'geniculostriate" visual system (posterior division) in addition to cells, which from the nature of their input from the dorsal thalamus are, considered "motoric" in function (anterior division). A more medial portion of the SMT arises from the adjacent "limbic cortex" (thought to be homologous to the mammalian hippocampal gyrus), and may contain the avian equivalent of the mammalian fornix bundles. The terminations of this latter group of fibers, however, are predominantly upon cells within the telencephalon (Karten, Hodos, Nauta & Revzin, 1973).

After its exit from the hyperstriatum, the lateral,

extratelencephalic portion of the SMT can be followed ventrally along the midline wall to the base of the forebrain where it separates into a dorsal and basal branch.

Projections of the dorsal branch, arising mainly from the posterior or visual Wulst, have been traced bilaterally to various identified nuclei of the pigeon's visual system, including the ventral geniculate, OPT complex, pretectal group, and potions of the optic tectum (Adamo, 1967; Karten, 1971; Karten, Hodos, Nauta & Revzin, 1973). The basal branch of the SMT, originating in the anterior Wulst, terminates ipsilaterally in the red nucleus, medial reticular formation, pontine nuclei, and the gracile and cuneate nuclei. Upon reaching the posterior brainstem, the remaining fibers cross in the bulbo-spinal junction and enter the spinal funiculus to terminate on cells of the spinal cord, minimally overlapping the spinal terminations of the OMT (Cohen & Karten, 1974). From the nature of its terminations, the basal branch of the SMT appears to be the other component of an avian "homologue" for the mammalian pyramidal system.

Neurobehavioral Studies: OMT and SMT

Preliminary evidence from this laboratory indicates that the integrity of the OMT may be essential for efficient consummatory activity. Following OMT lesions, pecking movements have been observed to be hesitant and frequently incomplete, and in many cases in which they were complete, the beak

failed to open sufficiently to permit complete grasping of the grain. Zeigler (1974; 1976) has noted that damage to central trigeminal structures, while it does not modify the spatio-temporal organization of the feeding response, decreases the probability of its elicitation by the appropriate visual stimuli. The relation of trigeminal afferents to the archistriatal region from which the OMT originates and the OMT's pattern of termination upon both somatosensory and visual structures within the midbrain suggests that OMT may be a substrate for somatomotor mechanisms involved in eating.

Neurobehavioral studies of SMT lesion effects should complement our studies of the OMT. Analyses of the sensory determinants of eating in the pigeon have shown the peck to be visually elicited. In addition, Boyko and Bures (1975) recently reported that in freely feeding pigeons, neurons of the Wulst were activated earlier and for a longer duration than neurons in any other telencephalic area. Furthermore, Cohen (1967; Cohen & Pitts, 1967) noted directed head movements following electrical stimulation of the hyperstriatum and a slight but persistent decrease in food intake following hyperstriatal lesions. Since lesions of the SMT would disrupt the efferent connections of a major forebrain visual area, analyses of their effects might help to identify substrates for the visuomotor mechanisms underlying the control of the pigeon's feeding behavior.

That both the OMT and SMT may participate in the sensorimotor integration underlying the pigeon's consummatory activity could prove to be of considerable heuristic value in furthering our understanding of the neural organization of similar processes in other vertebrate species. Because both pathways provide a naturally existing and surgically accessible separation of structures, which together in their entirety may be homologous with the mammalian pyramidal system, some insight might be achieved concerning the behavioral functions of the various subdivisions within the not-so-easily divisible mammalian pyramidal system. To this end, the behavioral consequences resulting from the destruction of one pathway should serve as an inherent control for deficits produced by the destruction of the other. But regardless of whether consummatory deficits can be dissociated in this manner, the identification of any neuromotor mechanism(s) controlling the pigeon's consummatory behavior would be of significant value since it would isolate a major component in the final common pathway for feeding and add to the inventory of structures already known to be part of the pigeon's neural feeding system.

Aims of the Study

One approach to the study of brain mechanisms underlying feeding behavior is the identification of the afferent and efferent structures involved and the analysis of their contribution to the control of the behavior. Previous studies from this laboratory have implicated trigeminal structures in the afferent control of feeding in the pigeon but no systematic data is available on the location of the efferent structures involved and the nature of their contribution. The present series of experiments were designed to examine the role of two efferent forebrain pathways, the occipitomesencephalic tract and the septomesencephalic tract, which preliminary evidence suggests may be components of the sensorimotor and motivational mechanisms controlling ingestive behavior in the pigeon.

Electrolytic lesions were placed in both of these tracts and their effects upon several aspects of feeding behavior were assessed using a variety of behavioral testing procedures. Experiment 1 was designed to determine the effects of such lesions upon ad libitum food and water intake and body weight. The use of photocell feedometers permitted the direct monitoring of feeding behavior and provided a measure of the pigeon's responsiveness to food in addition to and independent of its intake. Experiment 2 combined direct observational procedures with high speed cinematography to assess lesion effects upon the movement patterns constituting the consummatory response of feeding. Experiment 3 used an operant conditioning procedure

to assess lesion effects upon motivational mechanisms involved in the performance of instrumental behavior reinforced with food. The use of a photocell circuit in the food magazine permitted a direct assessment of lesion effects upon both the bird's responsiveness to the reinforcer and its feeding efficiency.

The present study should extend previous work on the neural control of feeding in the pigeon and have implications for the neurobehavioral analysis of sensorimotor and motivational mechanisms underlying vertebrate feeding behavior.

Experiment 1: Effects upon intake, weight regulation and responsiveness to food.

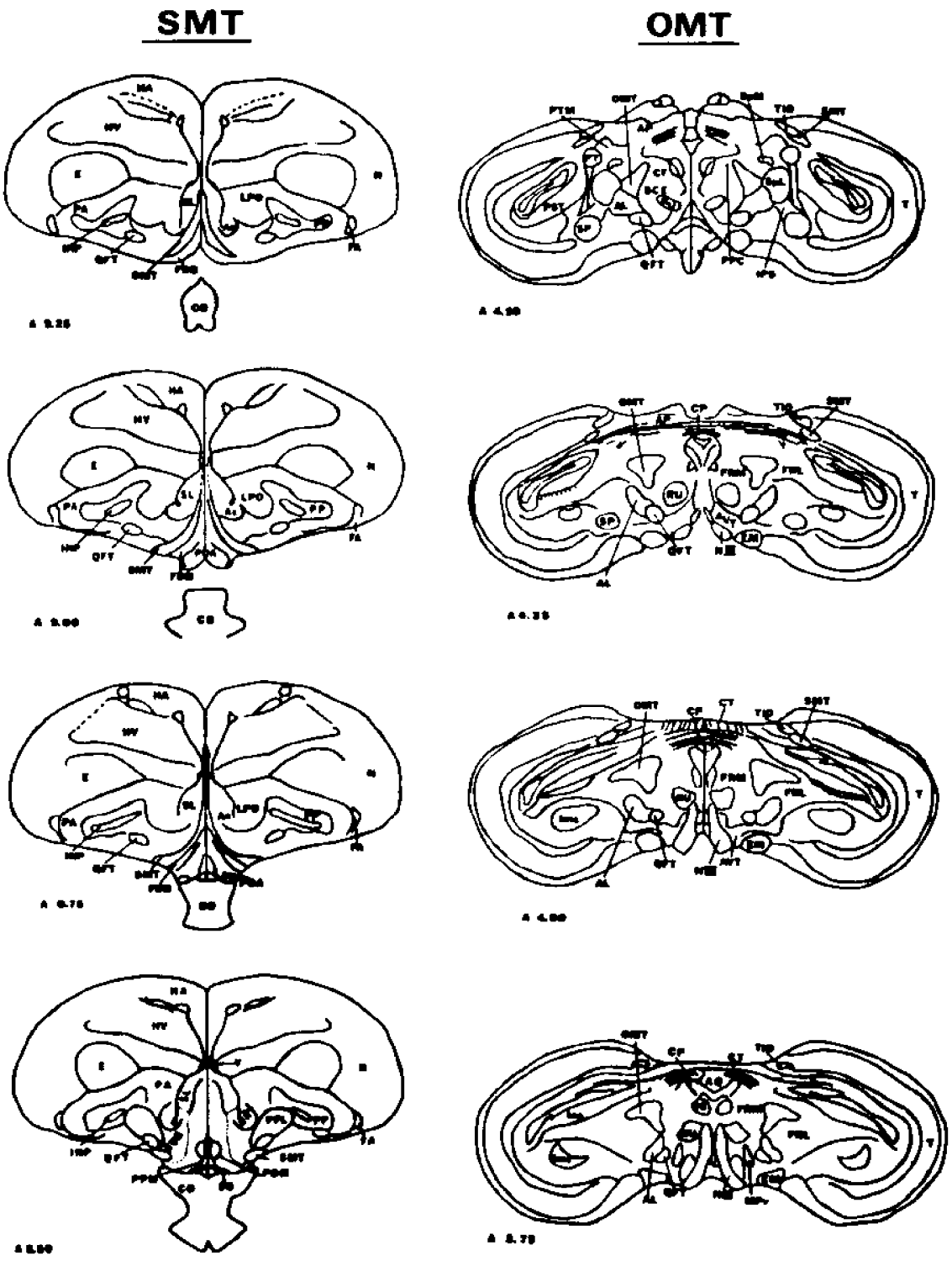
Bilateral destruction of central trigeminal structures in the pigeon produces periods of aphagia and hypophagia as well as significant and persistent losses in body weight. Following the resumption of feeding behavior in these "quinto-frontal" birds, the period of compensatory overeating seen in normal birds with comparable weight losses is either absent or considerably delayed. However, previous analyses of these deficits (Zeigler & Karten, 1973a) have shown that the effects are due primarily to reductions in the pigeon's responsiveness to food. Indeed, rates of body weight loss in aphagic quinto-frontal birds are comparable to those of normal birds food deprived for equivalent periods of time. Moreover, the quantitative relationships between food intake and body weight and the relative stability with which hypophagic birds maintain their reduced weight suggests that body weight is indeed regulated, but at a new, reduced level.

In addition to its effects upon food intake, damage to quinto-frontal structures impairs the efficiency of feeding by disrupting the mandibulation component of the consummatory response (Zeigler & Karten, 1973b). This disruption is reflected behaviorally in an increase in the number of feeding responses required to obtain a unit quantity of food. Thus the neurobehavioral analyses indicate that quinto-frontal lesions disrupt both motivational and sensorimotor mechanisms involved in the control of eating in the pigeon.

While the contribution of trigeminal afferents has been previously examined, corresponding neurobehavioral analyses are lacking for efferent structures putatively involved in the pigeon's feeding behavior. The present study was designed to examine the effects of lesions of the occipitomesencephalic (OMT) and septomesencephalic (SMT) tracts upon food and water intake, weight regulation and responsiveness to food. Preliminary data on the functional role of these structures suggest that lesions may also disrupt either somatomotor or visuomotor processes involved in the control of the consummatory response. However, although some observations on the consummatory response were made, the analysis of lesion effects upon efficiency is reserved for detailed consideration in Experiment II.

Because both OMT and SMT have extensive patterns of termination at several levels of the pigeon's brain, lesion sites were chosen so as to interrupt inputs to a maximum number of these terminal regions while minimizing damage to adjacent pathways and nuclear regions known to be involved in feeding. The site of the OMT lesion in the anterior mesencephalon represents a point of maximal isolation for this tract. At this level (Figure 2) the lesion is caudal to the termination of the hypothalamic component of OMT (HOM) but spares mesencephalic structures likely to be implicated in the control of posture or muscle tonus. The SMT lesions were placed at the base of the telencephalon (Figure 2) in order to damage both the dorsal and basal branches of the tract without impinging upon other forebrain structures known to be involved

FIG. 2



ABBREVIATIONS

A, archistriatum

Ac, nucleus accumbens

AL, ansa lenticularis

AP, area pretectalis

AQ, aqueductus sylvii

AVT, area ventralis (Tsai)

CF, campi Foreli

CO, chiasma opticum

CP, commissura posterior

CPi, cortex piriformis

CT, commissura tectalis

E, ectostriatum

EM, nucleus ectomamillaris

FA, tractus fronto-archistriatalis

FDB, fasciculus diagonalis Brocae

FPL, fasciculus prosencephali lateralis

FRL, formatio reticularis lateralis mesencephali

FRM, formatio reticularis medialis mesencephali

HA, hyperstriatum accessorium

HV, hyperstriatum ventrale

Ico, nucleus intercollicularis

Imc, nucleus isthmi, pars magnocellularis

INP, nucleus intrapeduncularis telencephali

IPS, nucleus interstitio-pretecto-subpretectalis

IS, nucleus interstitialis (Cajal)

LPO, lobus paraolfactorius

MPv, nucleus mesencephalicus profundus, pars ventralis (Jungherr)

N, neostriatum

N III, nervus oculomotorius

OMT, tractus occipito-mesencephalicus

PA, paleostriatum augmentatum

POA, nucleus preopticus anterior

POM, nucleus preopticus medialis (van Tienhoven)

PP, paleostriatum primitivum

PPC, nucleus principalis precommissuralis

PPM, nucleus preopticus paraventricularis magnocellularis

PST, tractus pretecto-subpretectalis

PT, nucleus pretectalis

PTM, nucleus pretectalis medialis

QFT, tractus quintofrontalis

RU, nucleus ruber

SCE, stratum cellulare externum

SL, nucleus septalis lateralis

SMT, tractus septomesencephalicus

SO, nucleus supraopticus (Ralph)

SP, nucleus subpretectalis

SpL, nucleus spiriformis lateralis

SpM, nucleus spiriformis medialis

T, tectum opticum

TIO, tractus isthmo-opticus

TPC, nucleus tegmenti pedunculopontinus, pars compacta

TVM, tractus vestibulo-mesencephalicus (Papez)

in the control of feeding.

The effects of bilateral lesions of OMT and SMT were compared with the effects of unilateral lesions of these structures and with the effects produced by sham procedures and control surgical lesions.

Method

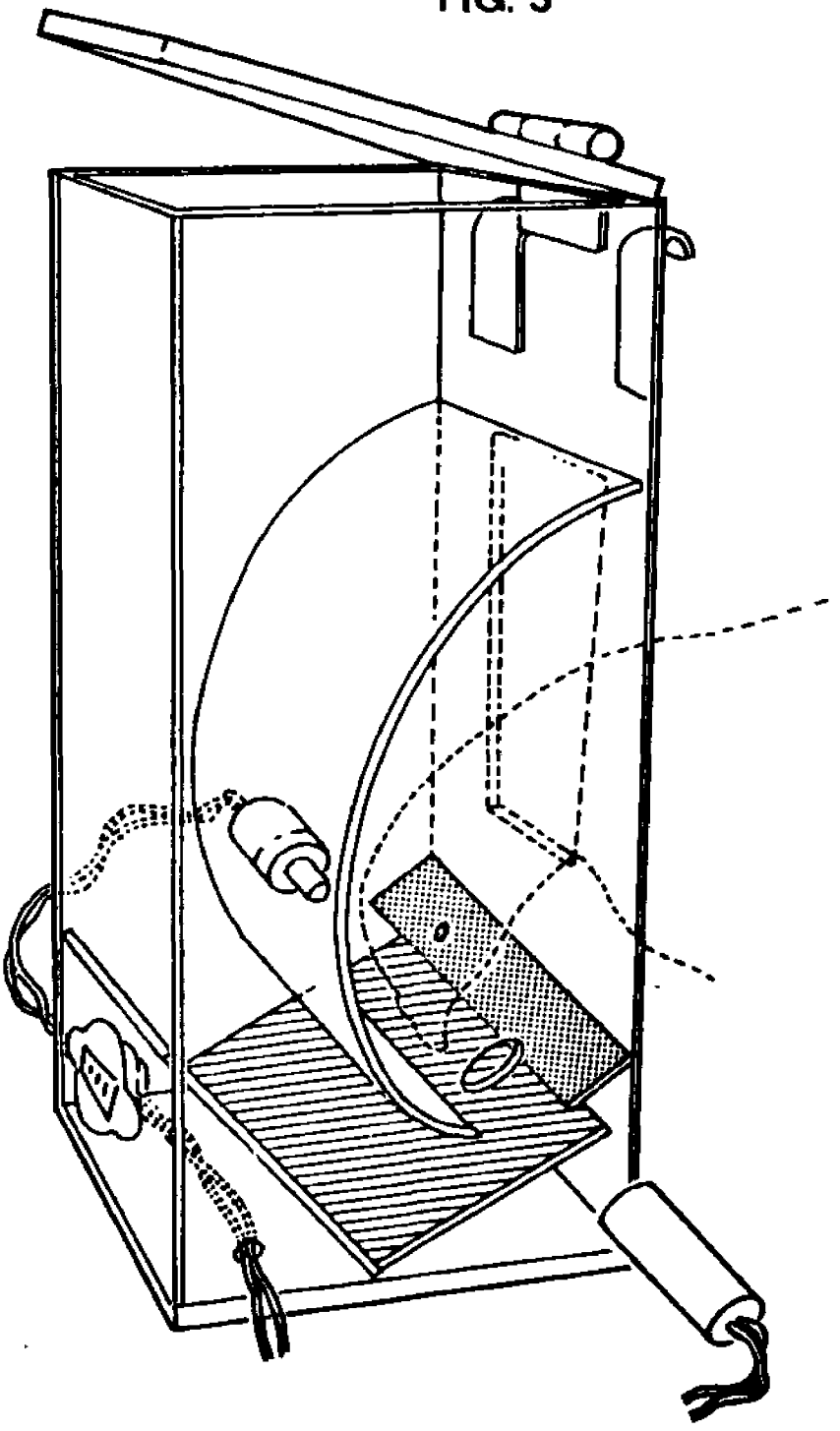
Subjects

Male and female white Carneaux pigeons (Columba livia), 3-6 year old and weighing 450-650 g, were obtained from a commercial supplier (Palmetto Pigeon Plant, Sumter, South Carolina). They were housed individually in the colony room under a 15:9 light/dark cycle (lights on: 8:00 A.M. - 11:00 P.M.) and with room temperature controlled to within a range of 70° - 80° F. (21° - 27° C). During a 2-mo. adaptation period to the laboratory, the birds were given continuous access to food and water and were left undisturbed except for routine weighing (three times weekly) and regular cage maintenance.

Apparatus

Food was provided in specially designed pigeon feedometers (Figure 3; Zeigler & Feldstein, 1971). The construction of the feedometer was such as to insure a continuous flow of fresh grain into the feeding compartment and to minimize the occurrence of grain spillage. A light source and photocell mounted 0.5 in. (1.3 cm) above the food was used to monitor feeding activity. Pecks into the food magazine interrupted a light beam, triggering a photocell detector circuit whose output was connected to a digital counter. To discourage sorting and therefore reduce occurrence of spuriously high counts, a single, though nutritionally adequate and highly preferred grain -- milo -- was used. In addition to indicating the bird's daily feeding activity, the responses recorded thus

FIG. 3



provided a direct measure of the pigeon's responsiveness to food independent of the total amount of food consumed.

Water was provided in 250-ml graduated glass drinking tubes and both food and water were available ad lib except for a short period each day when intake and body weight data were collected and routine cage maintenance performed.

Procedure

Intake and body weight data were recorded daily between 3 P.M. - 4 P.M. To simplify the recording of food intake, each feedometer was filled with enough food to bring the combined weight of the food and feedometer to 500 g. Subjects' food intake from the previous 24 hr. was then calculated as the difference between the original weight of 500 g and the new combined weight of the feedometer and food. Sufficient grain was then added to the food magazine via the feeding aperture to return the feedometer to its original weight. To insure a continuous supply of fresh grain and prevent an accumulation of grain chips and hulls in the food magazine, the birds received a completely new ration of grain three times per week.

Water intake was recorded to the nearest 2-ml demarcation on the tube and replenished anew each day. Body weight was recorded to the nearest gram by placing the bird in a restraining sleeve, and then placing the bird and sleeve on a gram balance which previously had been tared for the weight of the sleeve. Following the recording of these data, birds were allowed an

additional 15-30 minutes to recover from the effects of handling before food was returned at 4:15 P.M.

Preoperatively, data were recorded for 4-6 weeks, the mean intake and body weight of the last two weeks serving as a baseline for postoperative data comparisons. Data collection resumed 24 hr. following surgery and continued for a minimum of five weeks. To avoid the debilitating effects of dehydration, birds that were adipsic postoperatively for more than three days were encouraged to drink by gentle placement of their heads into the opening of the water tube; when necessary, they were intubated with water. Aphagic birds were maintained by hand feeding when their body weights reached 75% ad libitum. Hand-assisted feeding and drinking, however, were terminated when signs of spontaneous intake reappeared. As used in this and in the following experiments, the terms aphagia and adipsia describe, respectively, a condition in which there was no food or water intake postoperatively. Hypophagia and hypodipsia refer, respectively, to a reduction in food intake and a reduction in water intake below preoperative levels. Hyperdipsia refers to an increase in water intake above preoperative levels.

Surgery

Prior to surgery, animals were food deprived for 18-24 hr. and water deprived for 2-3 hr. to insure crop emptying and thereby reduce the risk of suffocation due to tracheal blockage by regurgitated food or water. Birds were anesthetized with Equithesin (2.0 - 2.5 cm^3/Kg) injected into the

pectoral muscles; .1 - .2 cm^3 supplementary doses were given as needed. The birds' wings were lightly, but securely, restrained with an Ace bandage, and the feathers covering the crown and external ear openings were carefully clipped. The birds were then placed upon a cloth sling between the bars of a Kopf stereotaxic instrument and their heads positioned in a pigeon headholder. An overhead 100-w lamp provided a source of heat. Following a midline scalp incision and retraction of the skin, burr holes were drilled and the brain exposed with the aid of a Zeiss operation microscope. Lesion electrodes were doubly-insulated 00-gauge stainless steel insect pins inserted halfway into the shafts of 23-gauge stainless steel hypodermic needles. An alligator clip attached to the skin flap along the incision or to an ear bar of the stereotaxic instrument served as the reference. Electrodes were inserted vertically into the brain at locations determined from the stereotaxic atlas of the pigeon brain of Karten and Hodos (1967) and lesions were produced by passing anodal direct current of 1.0 - 1.5 mA for durations of 15 - 60 sec. Occasionally, lesions were enlarged by a second penetration of the electrode a few mm distal to the first. After topical application of Xylocaine to the exposed wound, small pieces of Gelfoam were lightly placed over the exposed brain and the incision closed with silk sutures.

The data reported in the first experiment were collected from a total of 18 experimentally naive birds, each of which was assigned to one of four surgical groups. Six birds received bilateral lesions to the OMT and six to the SMT.

Three birds with unilateral OMT and SMT damage in addition to damage nearby but outside either the OMT or SMT served as operated controls. In addition, three birds which underwent all surgical procedures except for the passage of current through their lowered electrodes served as sham operates.

Histology

Following behavioral testing, the birds were sacrificed and their brains prepared for histological analysis. With the animal under deep anesthesia, perfusion through the left ventricle began with 100-200 cm^3 of .9% saline followed by at least 300 cm^3 of formol/slaine. Following decapitation, the brain was allowed to fix for several more days. The head was then placed in the stereotaxic instrument and the brain exposed and blocked transversely in the stereotaxic plane. After dehydration in a progressive ethanol series, the brain was embedded in celloiden. Serial sections at the level of the lesion were cut at 30μ on a sliding microtome and every other section stained with Luxol blue/ cresylecht violet for fibers and cells. Alternate sections from brains with lesions to the OMT or vicinity were also stained with thionin throughout the anterior-posterior extent of the lesion.

Examination of the sections for cell loss and gliosis was carried out with the aid of a microprojector and a light microscope. The locus and extent of the lesions were reconstructed on tracings of individual sections corresponding to

to the plates of the stereotaxic atlas. In addition, the extent of destruction to discrete structures at the locus of the lesion were recorded in tabular form and numbers from 0-3 were assigned to correspond to the extent of damage.

Results

Anatomical Data

Figure 4 presents a series of sections at the level of the SMT and OMT illustrating the maximum extent of the lesions in representative control and experimental birds. A quantitative summary of the histological analyses is presented in the left hand portions of Tables 1a, b and c. In all cases, the amount of damage to these structures is coded by the following symbols: 0 = none; 1 = minimal; 2 = moderate to extensive; 3 = extensive to total. Since lesion sizes tend to be continuously distributed, these categories involve considerable intergrading and are useful mainly for suggesting generalizations about the relation between the size and laterality of the lesion and its behavioral effects.

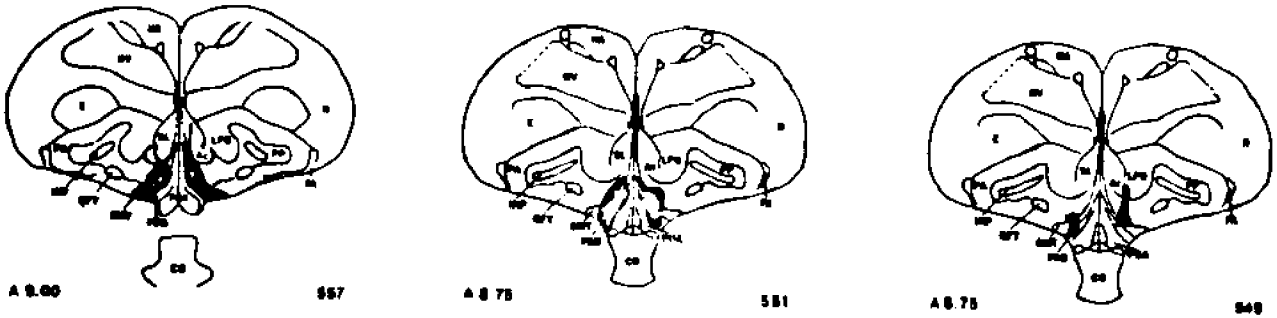
A. Control Group (Figure 4a).

1. Lesioned Controls. Three birds with subtotal, unilateral lesions to either the OMT or the SMT served as lesioned controls. Birds 550 and 523 both sustained moderate damage to the left OMT while escaping injury to the right tract. The lesions in both of these cases were asymmetrically placed resulting in varying amounts of nonsymmetrical destruction to neighboring cellular areas. In Bird 550, the lesion on the left side produced slight damage to cells within the lateral reticular formation at the diencephalo-mesencephalic boarder and, in addition, encroached slightly upon the caudal extent of nuclei within the spiriform and pretectal complexes. On the right side, damage was restricted to cells

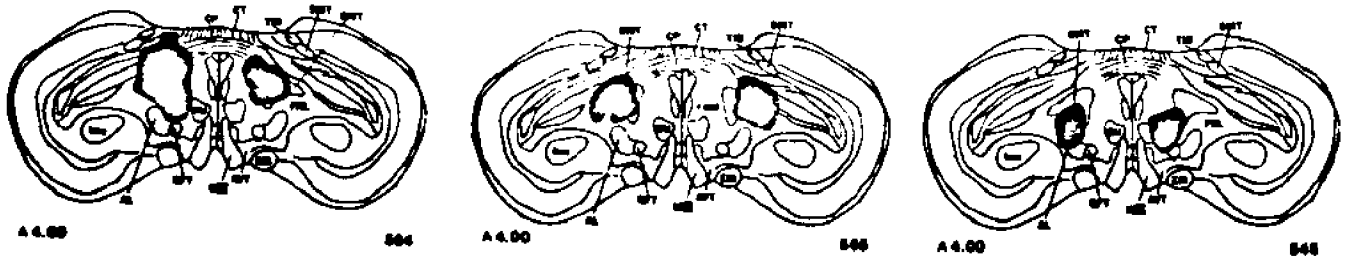
FIG. 4

A. CONTROL LESIONS

B. SMT LESIONS



C. OMT LESIONS



near the dorsomedial border of the OMT at the level of the posterior commissure. Bird 523 also sustained slight damage to the left lateral reticular area extending to the dorsal border of ansa lenticularis. On the right side, damage was restricted mainly to a small cellular area within the medial reticular formation in the vicinity of nucleus ruber. In both subjects, major pathways running through this region, including ansa lenticularis and the qunito-frontal tract, and their terminations, were spared.

Bird 560 sustained moderate unilateral destruction to the SMT within the left hemisphere. Medially, the lesion encroached upon cells within the diagonal band of Broca up to and including the lateral margin of the anterior preoptic nucleus. The right hemisphere was left intact.

2. Sham Operates. Along with the lesioned controls, data were gathered on a group of three birds which underwent all surgical procedures save for the passage of current through their lowered electrodes.

B. SMT Lesions (Figure 4b)

A total of eight birds received bilateral lesions to the SMT, but two birds were eliminated from the present analysis, one because of an ear infection and the other because its data collection was terminated prematurely.

The remaining six birds sustained lesions that were fairly uniform in size, shape and location. Lesion size ranged from 0.75 - 1.50 mm in antero-posterior extent and generally

did not exceed an area of 4 mm^2 as viewed in the coronal plane of section. Four of the six SMT birds sustained slight to moderate bilateral damage to the diagonal band of Broca and the adjacent anterior preoptic region. In three of these birds (532, 551 and 557) and lesion extended caudally up to and inclusive of the medial preoptic and anterior hypothalamic areas; the fourth bird evidenced anterior and lateral hypothalamic damage but escaped injury to diencephalic nuclei adjacent to the midline.

In the remaining two cases, Birds 549 and 554, there was a bilateral encroachment of the lesion upon the medial forebrain bundle. In Bird 554, the lesion extended rostrally to the posterior portions of the olfactory tubercle, although the amount of damage to this structure was judged to be minor. However, the lesions in both birds, spared nuclei adjacent to the third ventricle. Significantly, in all birds, there was no evidence of any damage to the nearby QFT. In addition, the lesions to SMT generally left intact major portions of the surrounding paleostriatum primitivum, lateral forebrain bundle and overlying lateral septal region.

C. OMT Lesions (Figure 4c)

Of the eleven birds receiving bilateral OMT lesions, five were excluded from this analysis. Four of these had bilateral destruction of the QFT as well as OMT, while the fifth was improperly blocked and could not be unambiguously analyzed. All of these subjects exhibited disruptions of feeding, similar to those birds reported below.

In general, the lesions of the six birds comprising the

OMT group were restricted to the area bounded medially by nucleus ruber, laterally and dorsally by the outer margins of the deep tectal layers, and ventrally by the QFT; the lesions extended from 0.75 - 1.50 mm in rostro-caudal length. In three birds, 543, 562, and 565, lesions extended anteriorly as far as the caudal borders of the pretectal region, inclusive of the spiriform complex. In two birds, 524 and 564, the lesions spread posteriorly up to the rostral margins of TVM and TPC, respectively. In four cases, lesions were symmetrically placed and damage to the OMT was accompanied by a slight to moderate degree of bilateral destruction of cells within the lateral and medial reticular areas. Two birds, 543 and 562, sustained a fairly extensive degree of bilateral damage to ansa lenticularis, while 543 additionally sustained a moderate degree of bilateral destruction to nucleus ruber. In Birds 564 and 565, the lesion extended, respectively, into the nearby overlying and lateral deep layers of the tectum and in Bird 564, interrupted fibers of the posterior and tectal commissures.

Lesions to Birds 524 and 545 were placed asymmetrically in the medio-lateral plane, and although they were partially inclusive of OMT bilaterally, little else was bilaterally damaged.

In all birds there was sparing of the overlying septo-mesencephalic and isthmo-optic tracts, the trajectory of the mesencephalic projection of the oculomotor nerve, and the quinto-frontal tract.

Behavioral Data

A. Effects Upon General Behavior

None of the birds with lesions of OMT or SMT showed gross postural impairments in standing or walking. In addition, there were no signs of muscular rigidity, tremor, flaccidity, or involuntary movements. Escape responses were brisk, and defensive behaviors, including wing-slapping and pecking, were characteristically observed within a day or two following surgery. It was not possible to directly assess the flying ability in lesioned birds because the primary feathers of the wings and tail were regularly trimmed. However, birds that were gently tossed into their cages from a distance, as well as birds placed on the floor and provoked to escape, showed well-coordinated movement patterns of the wings, legs and tail. These general effects upon behavior were noted in all birds reported here and in the birds reported in later experiments.

B. Effects Upon Food and Water Intake

The last six columns of Table 1, indicate for each subject a number of different measures of lesion effects upon intake and body weight, including the number of days of aphagia and adipsia following surgery, the extent of postoperative weight loss, the number of days required to regain preoperative weight and ad-lib and final body weight. The maximum extent of postoperative body weight loss was calculated as a percentage of mean ad libitum body weight. These data on weight loss and recovery provide a means for comparing the magnitude and

TABLE I

LESION EFFECTS UPON INTAKE AND BODY WEIGHT

BIRD #	LESION L	LESION R	SEX	DAYS OF APHAGIA	DAYS OF ADIPSIA	PER CENT WEIGHT LOSS	DAYS BELOW SURGICAL WEIGHT	AD LIB WEIGHT	FINAL WEIGHT
CONTROL GROUP									
550 (Uni OMT)	2	0	F	3	0	10	8	606	620
523 (Uni OMT)	1	0	M	1	0	5	5	620	609
560 (Uni SMT)	2	0	F	0	0	5	1	618	638
556 (SHAM)	-	-	F	1	0	4	4	626	618
558 (SHAM)	-	-	F	0	0	0	0	641	632
563 (SHAM)	-	-	M	0	0	0	2	634	644
SMT GROUP									
557	3	3	M	1	0	4	1	545	537
551	2	2	M	1	0	10	35*	638	599
554	2	1	F	1	0	11	1	542	525
567	2	1	M	0	0	11	42*	554	511
532	1	2	F	0	0	4	8	614	620
549	1	1	M	0	0	0	0	543	531
OMT GROUP									
564	3	2	F	3	2	12	28	560	525
543	2	3	M	2	1	13	42*	547	514
524	2	2	F	2	0	12	56*	557	514
565	2	2	M	4	0	12	31	629	641
562	2	2	F	4	3	14	18	586	614
545	1	1	M	0	0	17	56*	560	479

* Sacrifice prior to recovery of body weight

duration of postoperative feeding behavior deficits among the different groups of animals. Figures 5, 6, and 7, present data on food intake, water intake, and body weight plotted in 2-day blocks. Subjects whose data are presented were selected to represent a range from minimal to extensive damage in each structure.

1. Lesioned controls and sham operates. Since the overall effects upon intake and body weight did not differ markedly between surgical and lesioned controls, the data from all six animals are presented together to facilitate analysis. The results shown in Table 1 and Figure 5 indicate that while control lesions produced from 1 - 3 days of aphagia in two birds, this effect was not noticeably different from that of surgical control procedures. Disruptions of feeding in all cases were transient (mean hypophagic period = 1 day) and were followed by a period of compensatory over-eating characteristic of normal, food-deprived pigeons. The greatest effects were seen following unilateral lesions to the OMT, which in Bird 550, produced a 10 percent loss in body weight. However, in this case, as in the others, recovery of ad lib levels of intake was fairly rapid, ocurring within three days following the resumption of feeding. In no case was there a disruption of water intake, although Bird 560 evidenced an increase of water intake that could be interpreted as hyperdipsia. (Water intake in this subject is discussed below.)

2. SMT Group. Bilateral lesions of the SMT produced a

FIG. 5

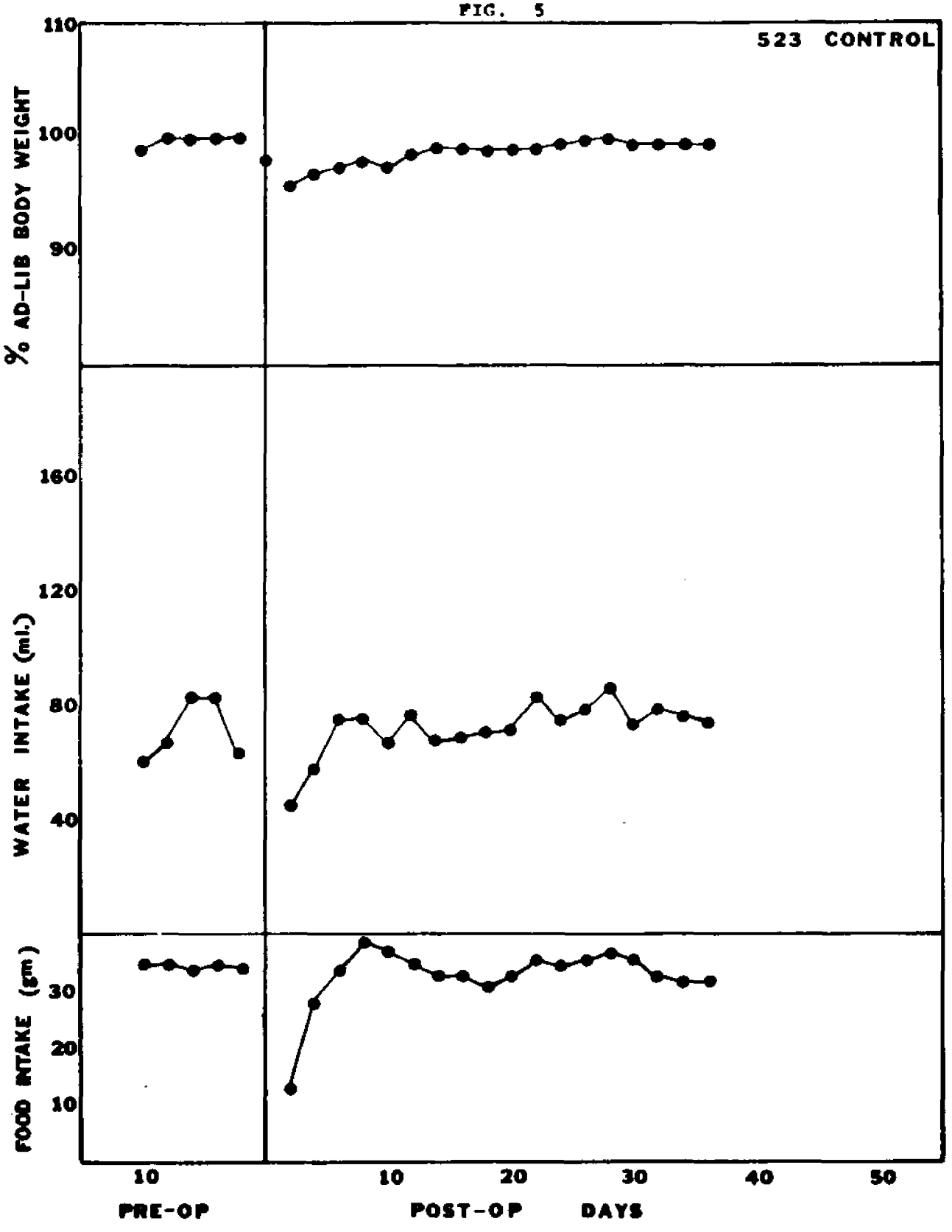

523 CONTROL
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP
DAYS

FIG. 5

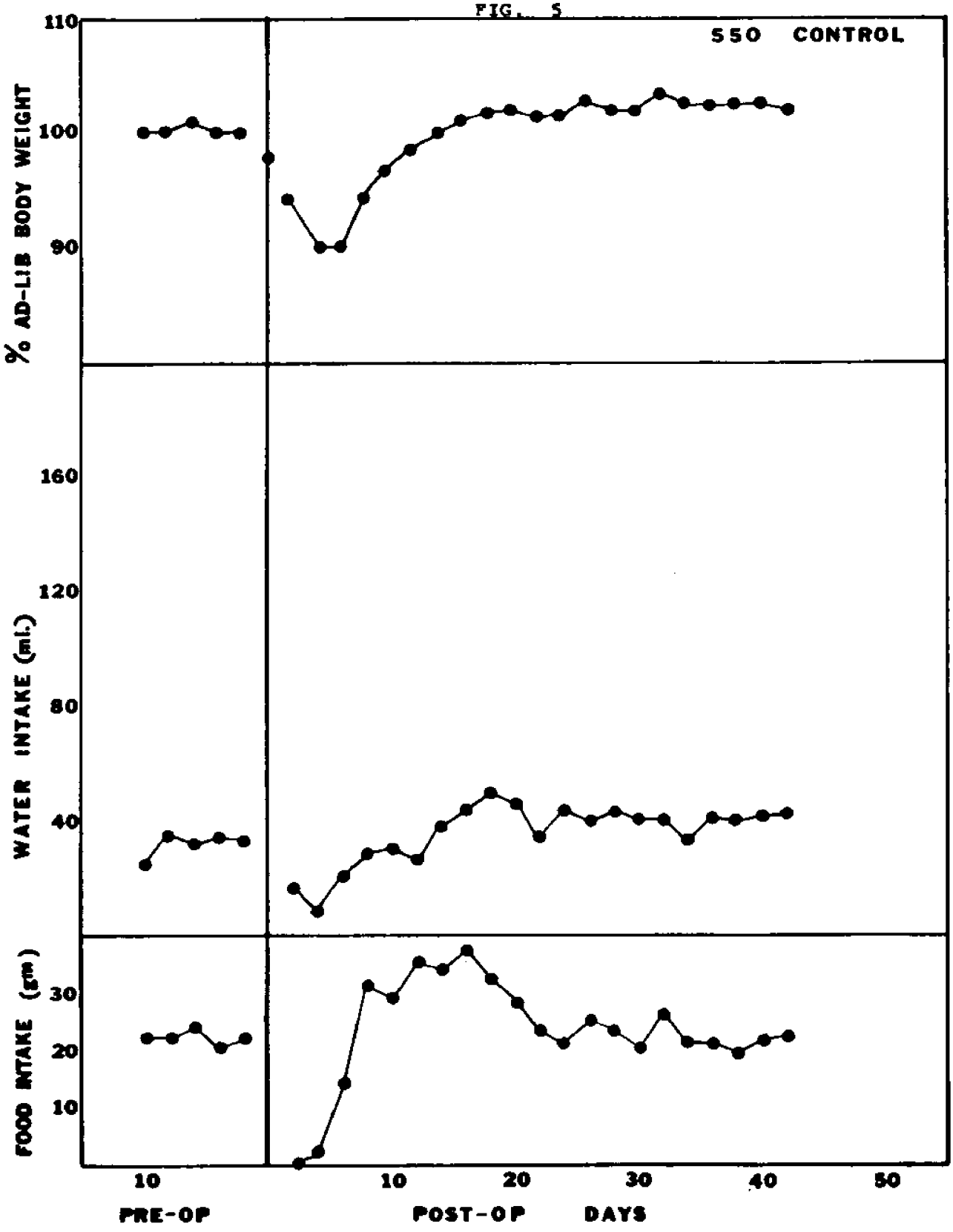

550 CONTROL
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm.)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP
DAYS

FIG. 5

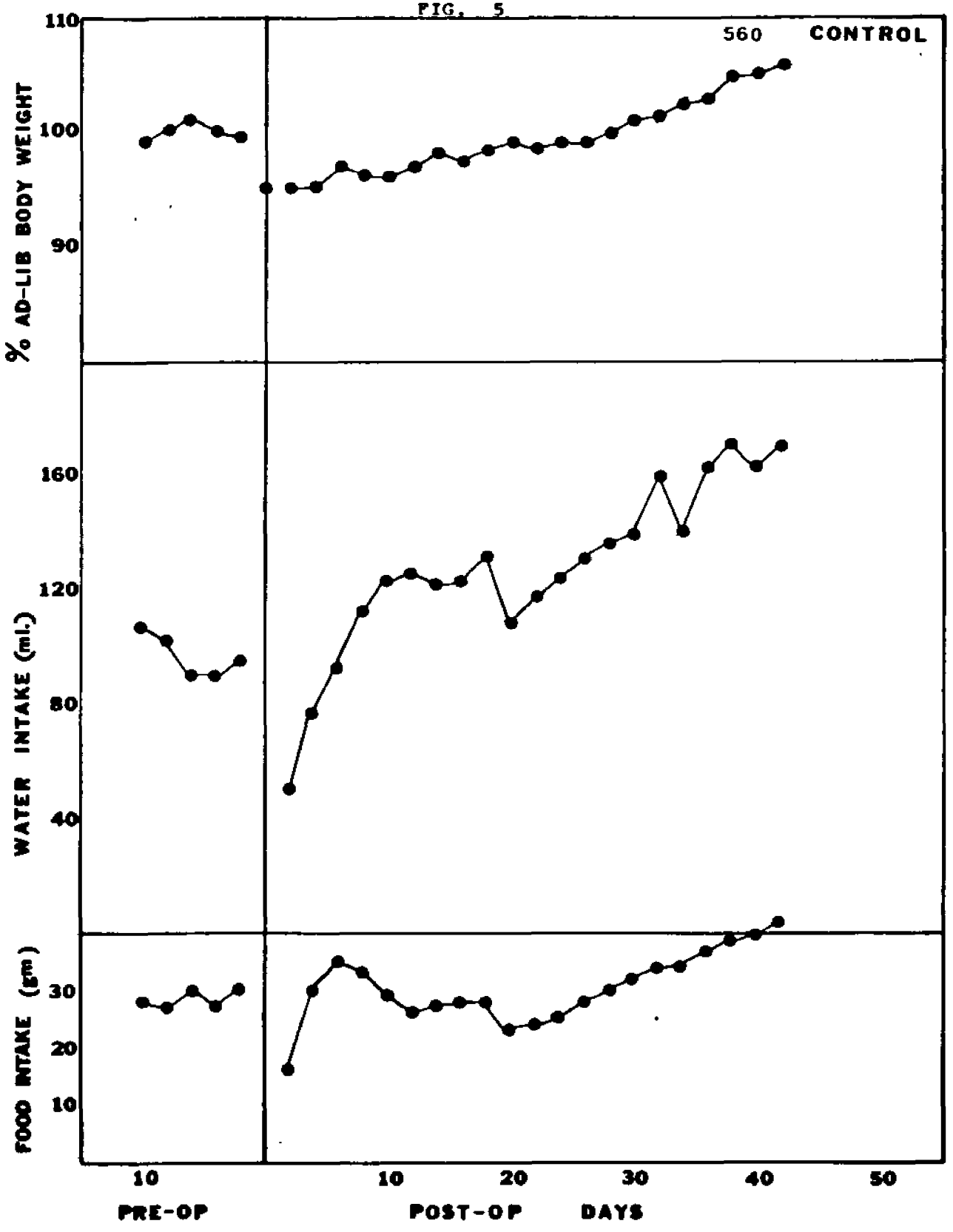
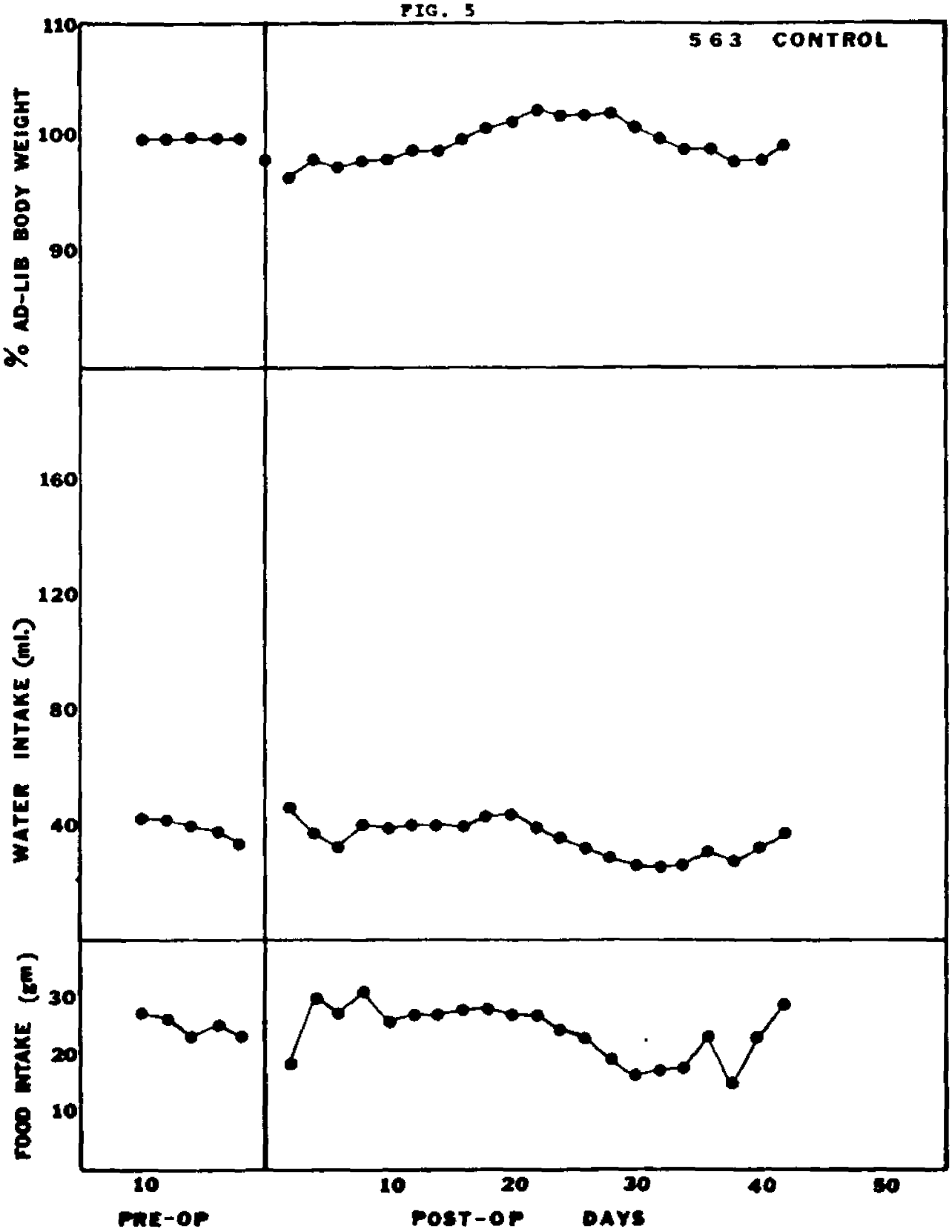

560 CONTROL
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP
DAYS

FIG. 5


563 CONTROL
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm)
30
20
10
10
20
30
40
50
PRE-OP
POST-OP
DAYS

transient (1-day) aphagia in three of six birds in this group, a result not significantly different from that of controls (Table 1). However, following surgery, all SMT birds displayed somewhat reduced levels of food intake of several weeks duration, resulting in slight, but persistent body weight losses of 2 - 6 percent (Figure 6). The single exception to this general pattern of postoperative eating was shown by Bird 532, whose daily food intake briefly returned to preoperative levels, but then slightly decreased to a level previously displayed during the first few postsurgical sessions. This was the only pigeon, however, whose body weight returned to and remained at preoperative levels. As shown in Figure 6, there was no evidence in any bird of the compensatory overeating characteristic of lesioned controls or food deprived normals.

All SMT birds resumed drinking within 24 hours following surgery. For Birds 549 and 554, water intake was slightly depressed throughout the duration of testing. In both cases, however, the observed reduction in water intake occurred simultaneously with a reduction in food intake. The remaining four pigeons in this group displayed an altogether different pattern of water intake following surgery. Similar to Bird 560, a lesioned control with unilateral SMT damage, each of these SMT birds showed a slight to extensive increase in drinking, which, once initiated, persisted until the termination of testing.

3. OMT Group. For five of the six birds in this group, fairly discrete, and for the most part, subtotal lesions of

FIG. 6

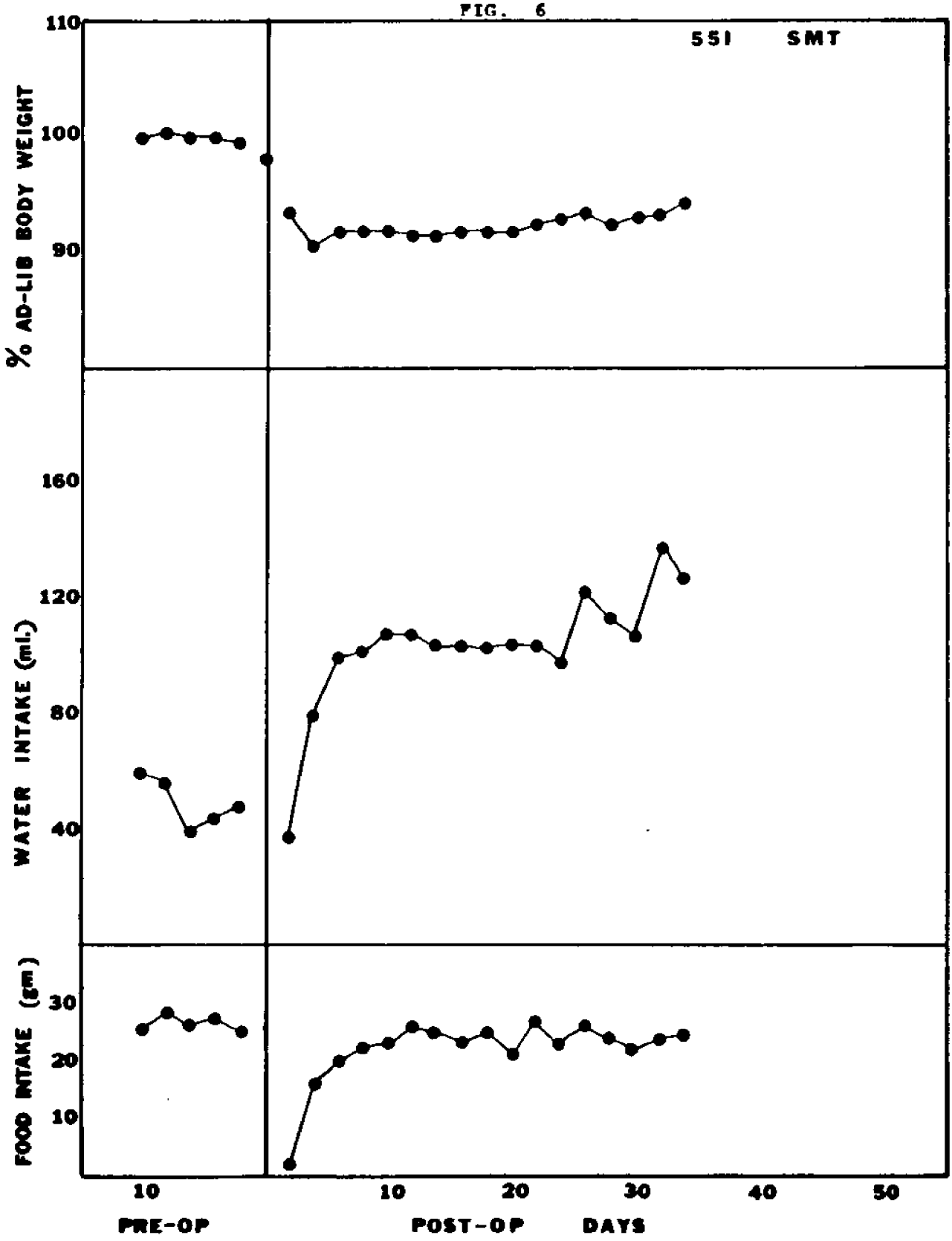

551 SMT
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP DAYS

FIG. 6

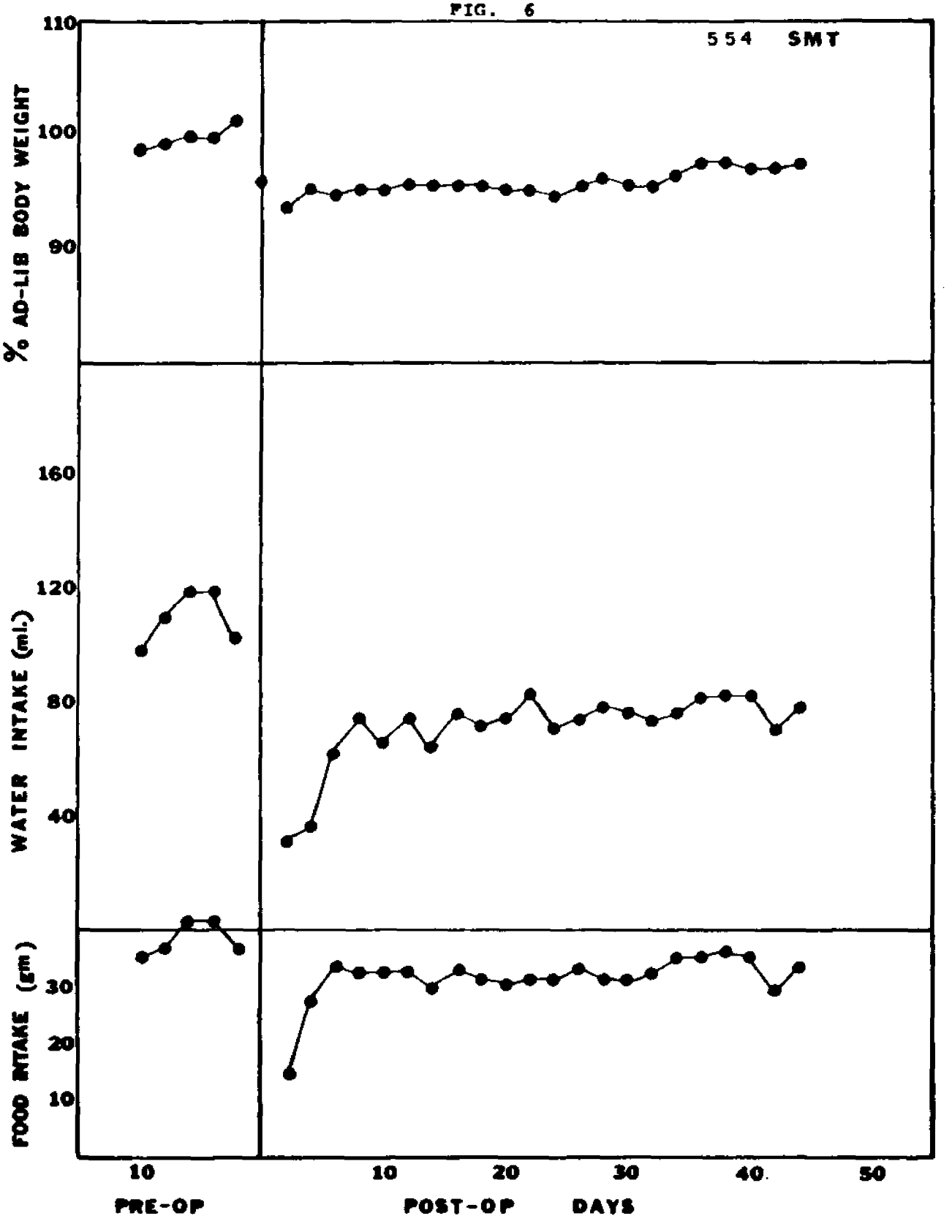

554 SMT
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP
DAYS

FIG. 6

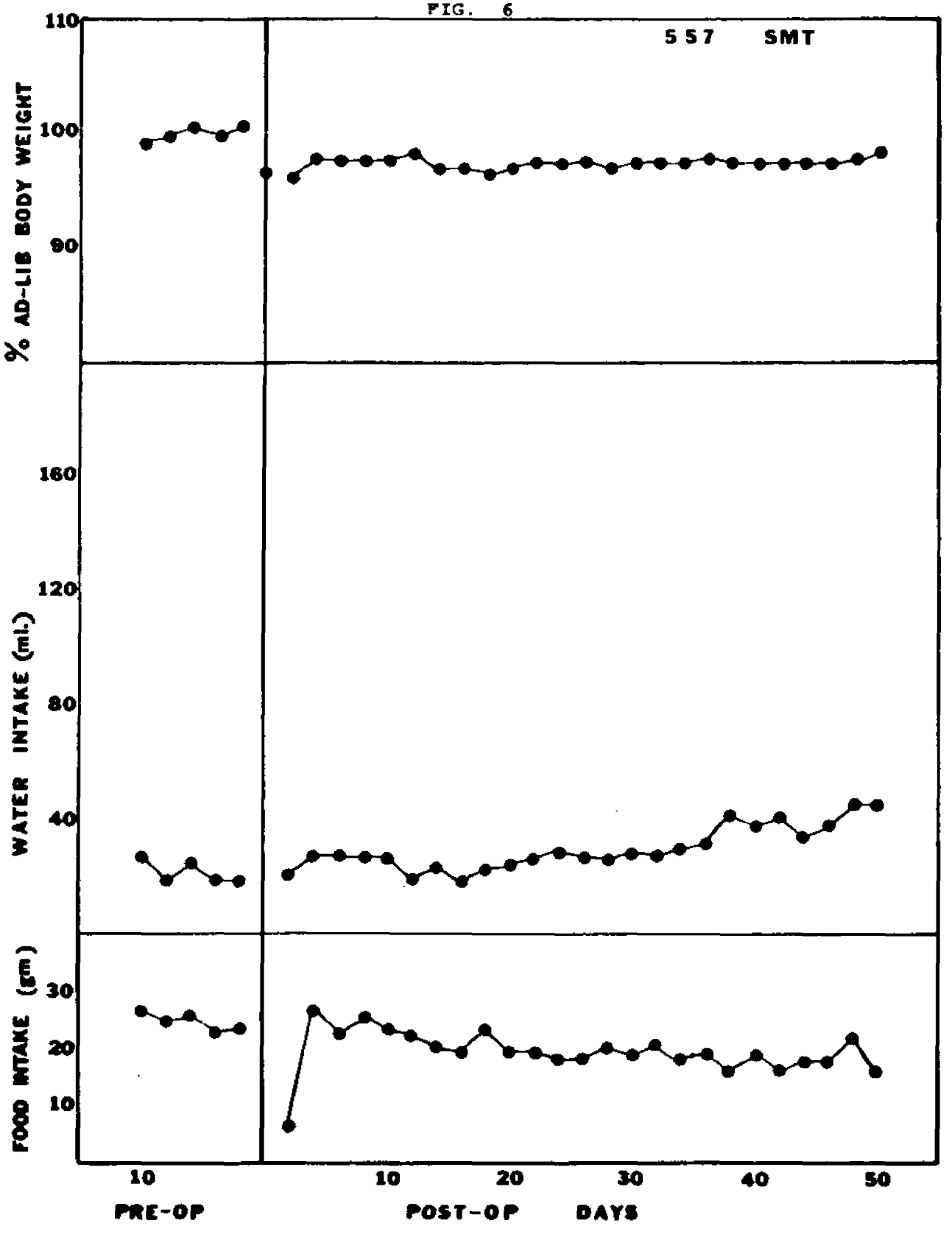
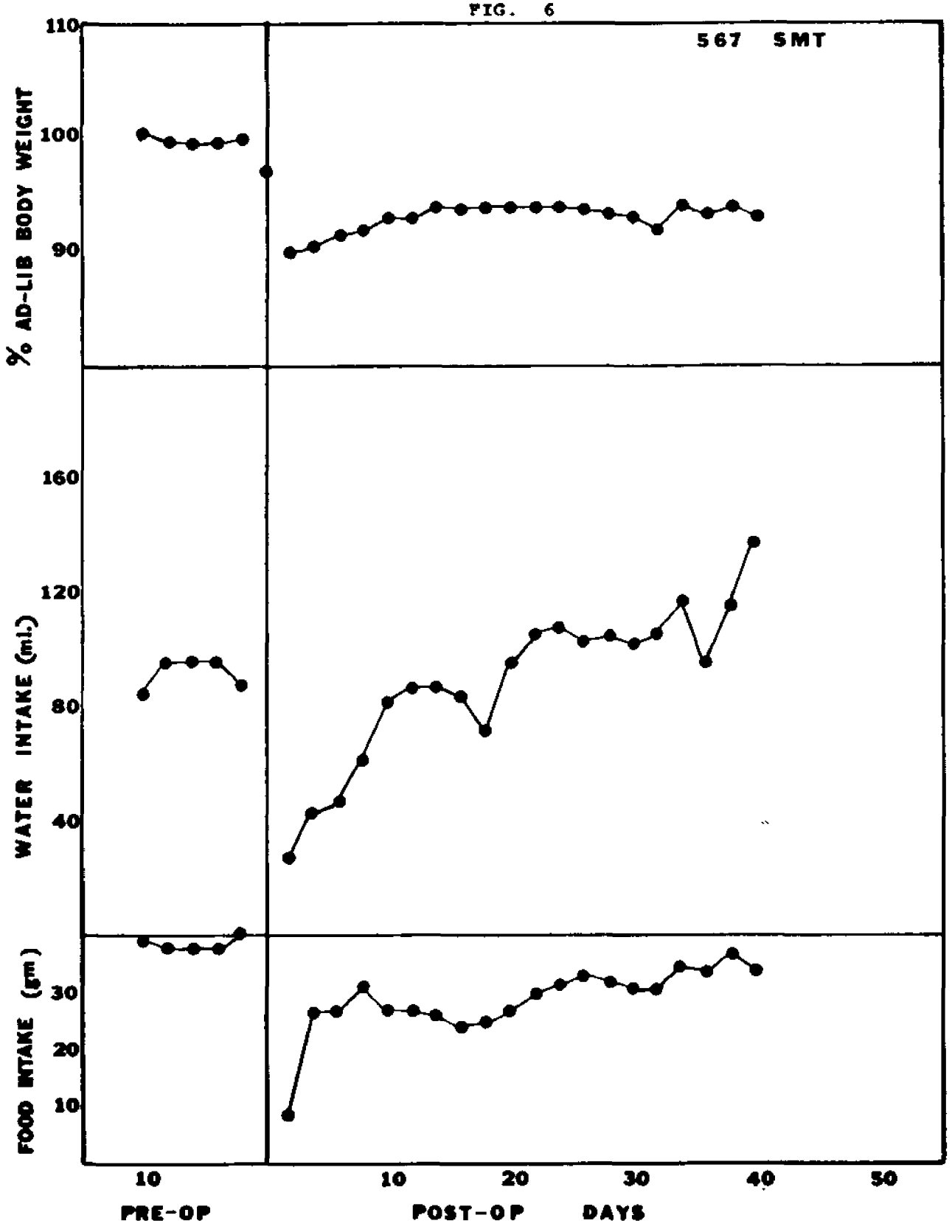

557 SMT
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm.)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP
DAYS

FIG. 6


567 SMT
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP
DAYS

OMT produced from two to four days of aphagia (Table 1 and Figure 6). In all birds, the resumption of feeding was followed by an extended period of hypophagia ranging from 4 - 24 days (Median = 9 days), resulting in body weight losses from 12 to 17 percent. Only three birds displayed the compensatory overeating and subsequent recovery of body weight characteristic of food-deprived normals. However, in two of these birds, 562 and 565, these effects were delayed for several weeks, while in the third, 564, there was a subsequent decrease in food intake to below preoperative levels which was not followed by an additional period of overeating. The remaining three pigeons displayed a gradual recovery to preoperative levels of food intake, but lacking compensatory increases in food intake, their body weights never returned to preoperative levels (Figure 7). There was no significant relation between the number of days of aphagia and the extent and persistence of hypophagia (Spearman rank-order r = -.14).

Following surgery, three OMT birds resumed drinking within 24 hours while the remainder showed only a transient adipsia of one to three days (Table 1). However, in all but a single bird, water intake was reduced for several weeks prior to returning to preoperative levels. Two birds, 543 and 545, increased their water well beyond preoperative levels, but the lesions in these birds differed in an important respect from those of the other birds in this group (see below).

FIG. 7

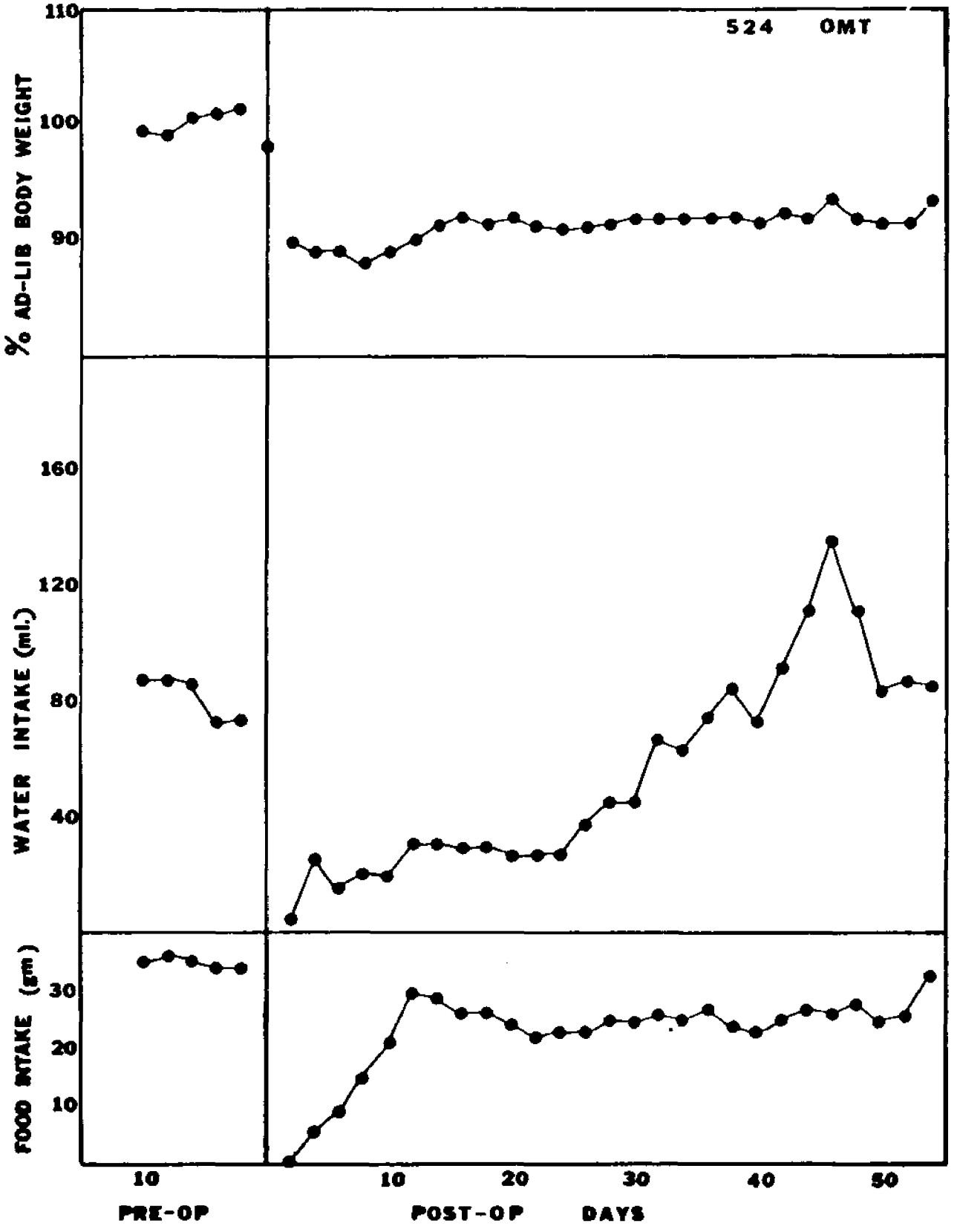

524 OMT
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP DAYS

FIG. 7

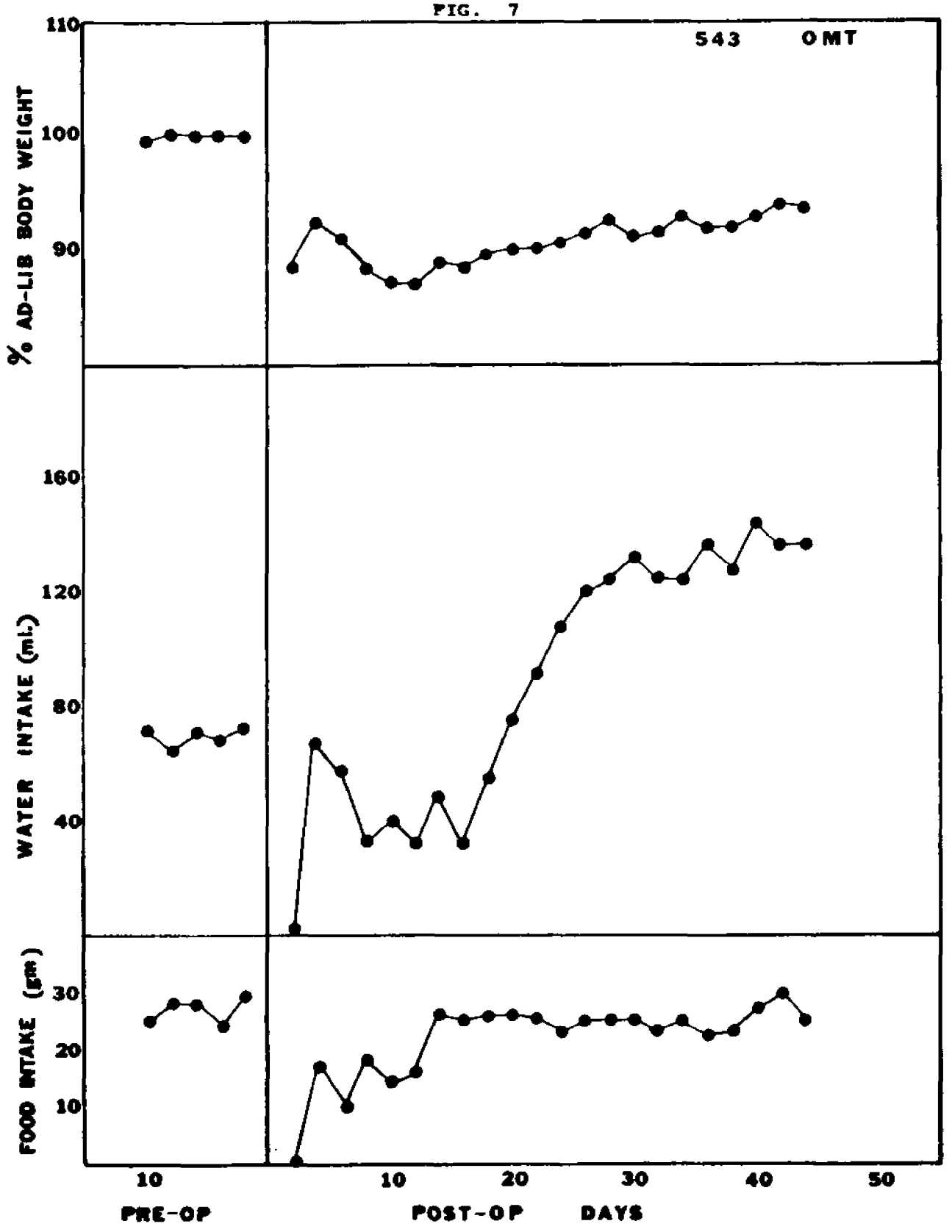

543 OMT
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP DAYS

FIG. 7

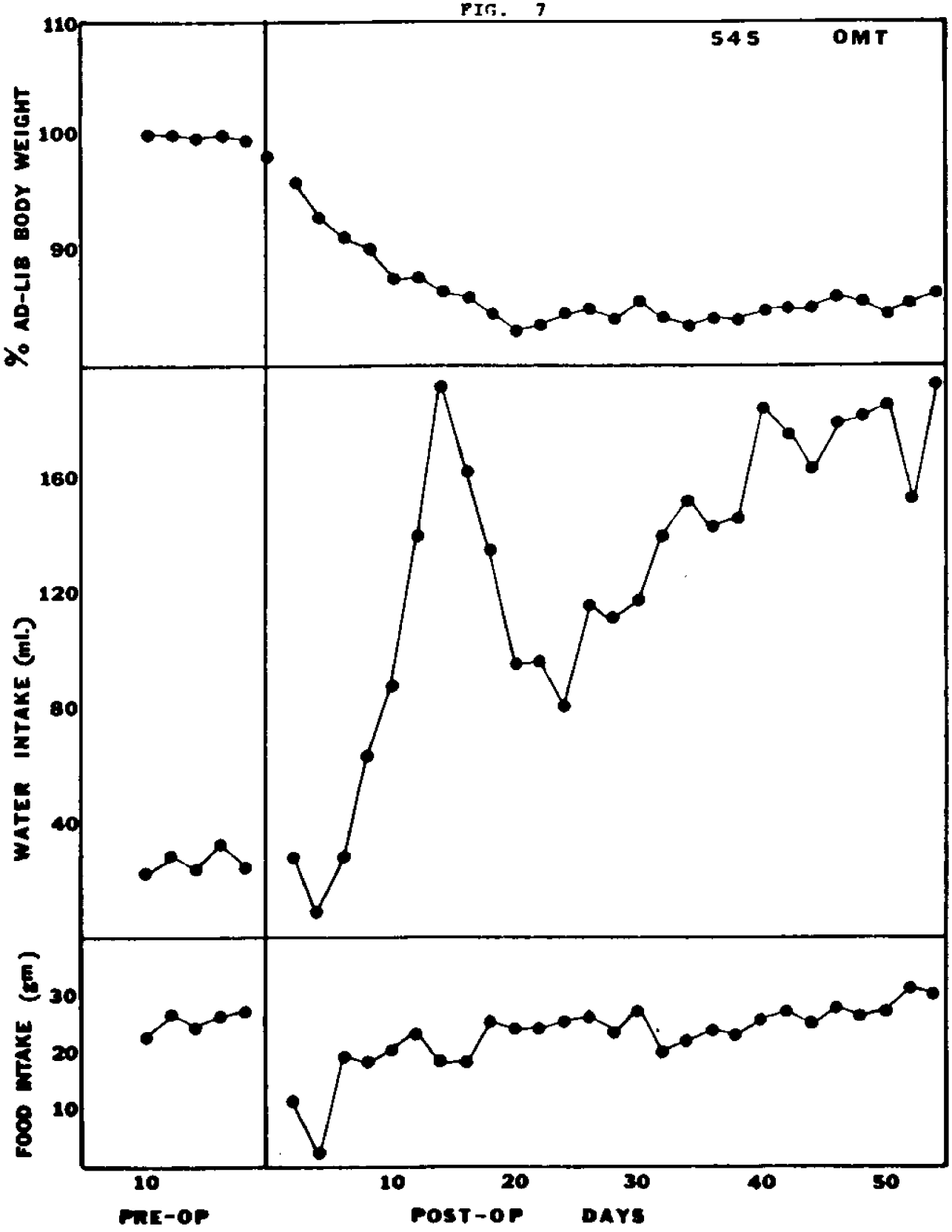
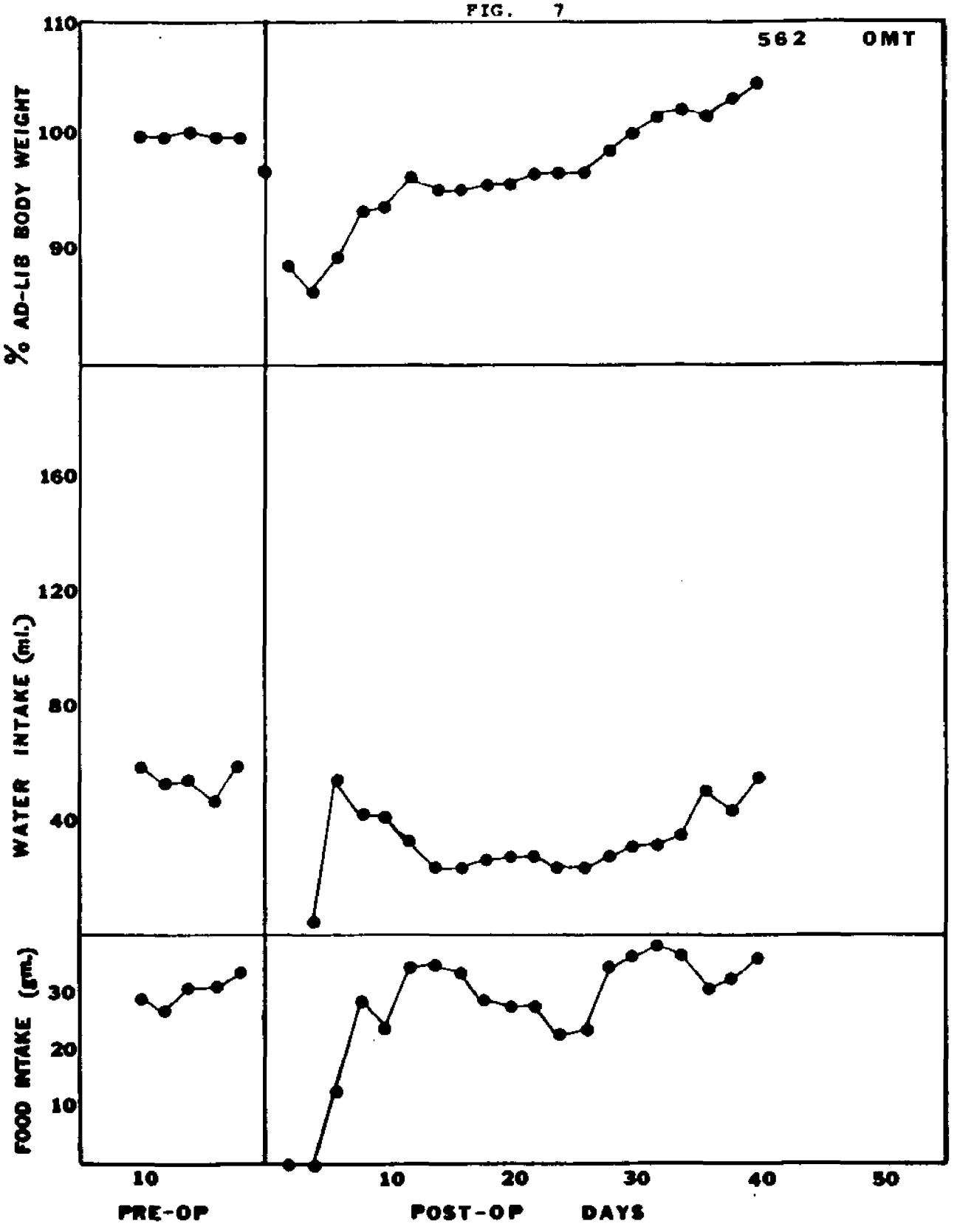

545 OMT
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm.)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP DAYS

FIG. 7


562 OMT
% AD-LIB BODY WEIGHT
110
100
90
WATER INTAKE (ml.)
160
120
80
40
FOOD INTAKE (gm.)
30
20
10
10
10
20
30
40
50
PRE-OP
POST-OP DAYS

C. Effects Upon the Interaction of Food Intake, Water Intake and Body Weight

Under ad libitum conditions, the food and water intake of normal pigeons are correlated with each other and the intake of both nutrients is a relatively constant fraction of the bird's body weight (Zeigler, Green & Siegel, 1972). This interdependence of food and water is even more apparent during periods of deprivation. Total water deprivation results in a drastic reduction in food intake, whereas food deprivation produces a reduction in water intake proportional to the size of the available ration. However, under each condition of deprivation, the proportionate intake of both nutrients to body weight is maintained (Zeigler, Green & Siegel, 1972).

1. Effects upon food intake and body weight. In the present experiment, lesions of the SMT and OMT resulted in periods of reduced food intake (hypophagia) and lowered body weight. Table 2 summarizes these data and presents pre- and postoperative relationships between food intake and body weight for both groups of birds. Preoperatively, the data are based upon the two-week period just prior to surgery; postoperatively, the data represent a two-week period characterized by a stabilized body weight. Mean intakes and body weight during these periods are shown in the first two pre- and postoperative columns in the table. The relationship between each is expressed as a ratio in the last column.

The data in Table 2 indicate that while lesions of the

TABLE 2

LESION EFFECTS UPON THE RELATION BETWEEN MEAN DAILY FOOD INTAKE (gm) AND BODY WEIGHT

	PREOPERATIVE			POSTOPERATIVE		
Bird Number	Food Intake	Body Weight	Ratio Food: Weight	Food Intake	Body Weight	Ratio Food: Weight
A. SMT GROUP						
532	33	614	0.05	22	612	0.04
549	35	543	0.06	32	516	0.06
551	26	638	0.04	24	590	0.04
554	39	542	0.07	32	519	0.06
557	24	545	0.04	20	534	0.04
567	39	554	0.07	31	519	0.06
B. OMT GROUP						
524	35	557	0.06	26	514	0.05
543	25	547	0.05	25	502	0.05
545	25	560	0.04	19	493	0.04
562	30	586	0.05	29	564	0.05
564	28	560	0.05	23	524	0.04
565	31	629	0.05	23	595	0.04

SMT and OMT may result in a reduced intake of food, the relationships between food consumption and body weight remain essentially unchanged in each bird. In this respect, lesioned birds maintain an intake level of food that is appropriate to their body weight and are thus comparable to normal pigeons maintained on reduced rations for long periods (Zeigler, Green & Siegel, 1972).

2. Effects upon water intake. Table 3 indicates the pre- and postoperative relationships between water intake and food intake and between water intake and body weight for SMT and OMT birds. These data are expressed as ratios of water intake : food intake and water intake : body weight in the last two pre- and postoperative columns in the table. Mean water intake during this period is shown in the first column. Mean food intake and body weight are identical to those shown in Table 2 as are the time periods for which these relationships have been determined.

With respect to water intake, lesioned birds fell into two groups (Table 3). One group (SMT Birds 549 and 554; OMT Birds 562, 564 and 565) showed a significantly decreased water intake that could be interpreted as a hypodipsia parallelling their hypophagia. Indeed, an examination of their water : food and water : body weight ratios indicate that, in each case, the intake of water was reduced relative to their food and body weight.

A second group of pigeons (SMT Birds 532, 551, 557 and 567; OMT Birds 524, 543 and 545) initially showed reduced drinking

TABLE 3

LESION EFFECTS UPON THE RELATION BETWEEN
MEAN DAILY WATER INTAKE (ml), FOOD INTAKE (gm)
AND BODY WEIGHT

	PREOPERATIVE			POSTOPERATIVE		
Bird Number	Water Intake	Ratio Water: Food	Ratio Water: Weight	Water Intake	Ratio Water: Food	Ratio Water: Weight
A. SMT GROUP						
532	58	1.7	0.09	73	3.3*	0.12*
549	169	4.8	0.31	128	4.0*	0.24*
551	51	2.0	0.08	94	3.9*	0.16*
554	114	2.9	0.21	77	2.4*	0.15*
557	24	1.0	0.04	31	1.6*	0.06*
567	91	2.3	0.16	106	3.4*	0.20*
B. OMT GROUP						
524	83	2.8	0.15	99	3.8*	0.19*
543	72	2.9	0.13	133	5.3*	0.26*
545	25	1.0	0.04	112	5.9*	0.23*
562	58	1.9	0.10	27	0.9*	0.05*
564	66	2.4	0.12	26	1.0*	0.05*
565	46	1.5	0.07	23	1.0*	0.04*

* $t\ (12) = 3.06$, $p < .01$: Preoperative vs. Postoperative

followed by a period of moderate to extreme increases in water intake which are reflected by significantly increased water : food and water : body weight ratios. However, the relationships between food intake and body weight remained u changed during this period (Table 2). These data suggest that while lesions may have produced significant reductions in food intake, they also resulted in a genuine hyperdipsia in these animals.

D. Effects Upon Body Weight Regulation

In normal pigeons, body weight loss is followed by a compensatory increase in food intake which is approximately proportional to the magnitude of the weight loss and which continues until the original body weight is recovered (Zeigler, Green & Siegel, 1972). In the present study, the rate of body weight loss in aphagic birds was comparable with that of normal pigeons food-deprived for equivalent periods. However, following the resumption of feeding, the time course of body weight recovery in the experimental birds was considerably extended. These data are illustrated in Table 4 which shows weekly group means and standard errors of the preoperative percentage of food intake and body weight and indicates the time course of body weight recovery following the resumption of feeding.

As shown in Table 4, body weight loss in control pigeons was followed by increases in food intake and original body weights were recovered within three weeks. However, none of the lesioned birds displayed the degree of compensatory

TABLE 4

RECOVERY OF FOOD INTAKE AND BODY WEIGHT

Weeks from Resumption of Group Feeding	1		2		3		4		5	
	Mean	S.E.	Mean	S.E.	Mean	S.E.	Mean	S.E.	Mean	S.E.
A. PER CENT AD LIB FOOD INTAKE										
CONTROL	101	6	111	24	103	11	100	4	102	10
SMT	77**	15	84*	9	78**	12	82**	11	83*	12
OMT	64**	22	80*	26	78*	18	93	16	94	13
B. PER CENT AD LIB BODY WEIGHT										
CONTROL	96	1.7	98	1.1	100	2.9	100	2.1	100	1.9
SMT	95	3.0	96	3.0	96*	3.0	96*	3.0	96*	3.0
OMT	92*	1.6	92**	4.0	91**	4.0	92**	4.8	94*	6.5

* $t(10) = 2.23$, $p < .05$ Experimental vs. Control
** $t(10) = 3.17$, $p < .01$ Experimental vs. Control

overeating shown by the controls and their body weight remained significantly below preoperative and control levels. This retardation in the recovery of body weight was most obvious from the third postoperative week from the resumption of eating at which time control birds had reached their ad libitum body weights.

E. Effects Upon Feedometer Responses

The data in Table 5 indicate the relationships between feeding responses and food intake following control surgery and lesions of the SMT and OMT, respectively. The first set of columns present the data for the week immediately preceding surgery while the following column shows the number of feeding responses made during the period of aphagia, as defined by the absence of food intake. (Numbers in parentheses indicate the number of days of aphagia.) The last two sets of columns present, respectively, the data for the first and second 7-day period following the resumption of feeding.

In normal pigeons, periods of food deprivation (and body weight loss) are followed by an increase in both intake and feeding responses which is approximately proportional to the the bird's weight loss (Megibow & Zeigler, 1968; Zeigler, Green & Lehrer, 1971; Zeigler, Green & Siegel, 1972). A similar relationship between weight loss, responsiveness and intake can be seen among the lesioned controls in the present study (Table 3a). The transient aphagia in these birds was characterized by an abolition or reduction of feeding responses. However, intake and responsiveness rapidly returned to

TABLE 5

EFFECTS OF LESIONS UPON FOOD INTAKE AND FEEDOMETER RESPONSES

	PREOPERATIVE			APHAGIC	POSTOPERATIVE					
	LAST 7 DAYS				DAYS 1-7 FEEDING			DAYS 8-14 FEEDING		
BIRD NUMBER	TOTAL FOOD INTAKE	TOTAL FEEDING RESP.	RATIO: RESP./ GRAM	TOTAL FEEDING RESP.	TOTAL FOOD INTAKE	TOTAL FEEDING RESP.	RATIO: RESP./ GRAM	TOTAL FOOD INTAKE	TOTAL FEEDING RESP.	RATIO: RESP./ GRAM
CONTROL GROUP										
523	241	14,485	60	0 (1)*	227	14,276	63	250	16,846	67
550	156	21,122	135	80 (3)*	151	12,764	91	157	29,296	186
556	173	12,105	70	0 (1)*	174	13,795	79	176	14,677	83
558	176	17,167	98	--	195	18,358	94	165	15,847	96
560	199	29,910	150	--	216	29,651	137	187	30,911	165
563	167	12,985	78	--	183	13,645	75	190	13,406	71
SMT GROUP										
532	234	13,350	57	--	172	8,369	49	207	10,478	51
549	244	11,832	48	--	214	12,233	57	197	12,496	68
551	188	15,382	82	0 (1)*	118	23,721	201	173	39,757	230
554	281	27,379	97	0 (1)*	217	15,034	69	224	28,014	125
557	170	16,380	96	0 (1)*	166	15,251	92	153	21,414	140
567	268	27,189	101	--	157	36,453	232	188	33,252	177
CUT GROUP										
524	242	13,656	56	14 (2)*	85	3,885	46	169	6,580	39
543	187	10,437	56	3 (2)*	99	3,831	37	149	4,194	28
545	178	17,065	96	--	81	4,272	53	141	6,643	47
562	219	15,599	71	0 (4)*	167	9,393	56	231	16,796	73
564	195	14,350	74	17 (3)*	188	10,819	58	199	10,422	52
565	212	34,667	164	86 (4)*	164	18,851	115	79	6,421	81

* Numbers in parentheses indicate the number of days of aphagia

preoperative levels and persisted at these levels for the duration of testing. There were no effects upon intake and responsiveness in any of the remaining control birds.

Lesions of the SMT produced only a brief aphagia in three birds, during which time feeding responses and intake were markedly reduced or abolished. In the first week of post-operatively eating, the number of feeding responses in four pigeons returned to levels equal to or greater than those observed prior to surgery, although only one pigeon (Bird 557) approached its preoperative level of intake. By the end of the second week, although the number of feedometer responses was significantly above preoperative levels, intake in most birds remained below preoperative levels. Despite their reduced food intake, feedometer : food ratios were increased for SMT birds suggesting a decrease in feeding efficiency.

These relationships for OMT birds may be seen in Table 3c. In most cases, lesions of the OMT were followed by a few days during which there was an absence of eating. During this period of aphagia, responsiveness to food, as measured by the number of feeding responses, was significantly reduced or abolished. In the first seven days following the resumption of feeding, OMT birds evidenced an increased responsiveness to food which was parallelled by an increased intake. However, both intake and responsiveness, for the most part, remained significantly below their preoperative levels. With the exception of a single pigeon, the second postoperative week was characterized

by additional increases in intake and responsiveness, although most birds continued to show decreases in both intake and feedometer responses. Feedometer : food intake ratios decreased postoperatively in OMT birds, suggesting a paradoxical increase in feeding efficiency. However, the interpretation of these ratios for both groups is far from straightforward and will be discussed at length below.

Discussion

Damage to the pigeons' SMT or OMT regions produces only a transient aphagia. However, the resumption of feeding is marked by reductions in food intake (hypophagia) resulting in persistent and significant body weight losses. Moreover, lacking the compensatory overeating characteristics of the food-deprived normal pigeon, the recovery of preoperative body weight is either absent or considerably delayed.

An examination of the relationship between food intake and body weight showed that despite their reduced intake hypophagic birds maintained their original food:body weight ratios. Thus, lesioned birds appear to utilize their available food and regulate their body weight as efficiently as before and as efficiently as normal pigeons on reduced rations (Zeigler, Green and Siegel, 1972). This suggests that their prolonged weight reduction probably stems from their reduced food intake rather than direct effects upon mechanisms controlling weight regulation.

Transient adipsias (1 - 3 days) were produced in three of the OMT birds; however, in each case the resumption of drinking always preceded the resumption of eating. Following the resumption of drinking birds in both experimental groups showed significant changes in water intake. However these effects upon water intake appear to be independant of the extent of damage to either OMT or SMT but are related to the presence of incidental damage to adjacent structures.

Based upon an analysis of the ratios of water intake to food intake and body weight, four SMT birds showed significant increases in water consumption which persisted throughout the postoperative test period. Examination of the histological material for each of these birds reveals damage to structures which have previously been implicated in the neural control of avian water intake. In all of the birds there was damage to the anterior and medial preoptic area as well as anterior hypothalamic regions. In addition, Bird 551, which showed the most abrupt and extensive increase in water intake, also sustained bilateral damage to nuclei in the periventricular region. Damage to this region in two other birds (532 and 557) was unilateral, and their hyperdipsia was less profound and delayed in its onset. Several authors have reported similarly delayed increases in water intake following lesions of these regions in the chicken (Ralph, 1959; Koike & Lepkovsky, 1967) and sparrow (Kuenzel & Helms, 1970). They have interpreted these delayed effects to the gradual depletion or blockage of transport of ADH within the neurosecretory cells of the periventricular region. It should be noted that significant increases in water intake were produced by a unilateral lesion of the anterior preoptic area in Bird 560.

Judged by their ratios of water intake to food and body weight the two remaining SMT birds showed significant persistent decreases in their water intake. Despite the fact that both animals had very different amounts of SMT damage, they exhibited similar degrees of hypodipsia suggesting that effects

upon water intake were related to damage to adjacent structures (nucleus accumbens and diagonal band of Broca).

Analysis of the water:food and water : body weight ratios indicates that OMT birds showed significant changes in water intake which were not related to their reduced food intake. Once again, these effects seem to be related to incidental damage to nearby structures rather than to OMT itself since animals with very similar degrees of OMT damage show effects upon water intake which vary with the extent of damage to these adjacent structures. In the case of hyperdipsic animals the regions involved generally extend into the medial reticular formation and are bounded medially by nucleus ruber and ventrally by the area ventralis of Tsai. Hypodipsic birds had incidental damage which extended into the lateral reticular formation.

Lesion effects upon feedometer responses differed in the two experimental groups. SMT birds, although they showed a persistent reduction in their food intake levels, they manifested a significant increase in the number of daily feedometer responses. By contrast, the reduced food intake of the OMT group was accompanied by a reduction in the number of feedometer responses. Calculation of the ratios of feedometer responses per unit of food in the two groups (Table 5) suggested that the SMT birds were less efficient after surgery while the OMT birds were more efficient. Since both groups were showing reduced food intake these paradoxical findings required clarification. Informal observations of the feeding behavior

of the two groups revealed characteristic effects upon the consummatory response which help to explain these findings.

The SMT birds made many side-to-side head movements displacing the grain to the sides of the feedometer in a manner reminiscent of the sorting behavior seen in pigeons presented with a mixture of several grains, one of which is differentially preferred (Zeigler & Feldstein, 1971). In the present test situation, such behavior is inappropriate since only a single grain is available in the feedometer. This sorting behavior was not seen preoperatively and could lead to an appearance of decreased feeding "efficiency", i.e., an increase in the number of feedometer responses without an accompanying increase in food intake.

OMT birds, on the other hand, displayed obvious difficulties in the grasping of individual grains which were frequently pecked at without being swallowed. In such cases, the pigeon characteristically keeps its head within the feedometer and does not break the photocell beam between individual pecks. This pattern of behavior has previously been seen in trigeminally deafferented birds (Miller, 1974). Such behavior could lead to the appearance of increased "efficiency", i.e., a decrease in the number of feedometer responses per unit food in birds with significant consummatory response deficits.

These observations suggest that the reduced intake and retarded body weight gain of the experimental birds is accompanied by postoperative changes in their consummatory behavior. A detailed analysis of the consummatory behavior of such birds is presented in Experiment 2.

Experiment 2: Effect of SMT and OMT lesions upon consummatory behavior.

Previous research in both the pigeon (Zeigler and Karten, 1973b; Zeigler, Miller and Levine, 1975) and the duck (Zweers and Wouterlood, 1973) has clearly indicated the significant contribution made by the trigeminal system to the neurosensory control of feeding in these species. Lesions of trigeminal structures, while they have no effect upon drinking behavior, produce a striking impairment in the bird's ability to grasp and mandibulate its food which results in a marked reduction in feeding efficiency in both these species. Because of the anatomical relationships of SMT and OMT with structure of the visual and trigeminal system respectively damage to these pathways might be expected to disrupt processes underlying the visuomotor or somatomotor control of the consummatory response of eating. Indeed, preliminary observations of lesioned birds in Experiment 1 suggested that OMT damage may have reduced the efficiency of feeding by impairing the neuromotor control of beak opening. In addition, several studies have suggested that the SMT may exert some control of brainstem structures which may be involved in head orientation (Cohen and Pitts, 1967; Boyko and Bures, 1975).

The observations on the consummatory response made during Experiment 1 were incidental to the study of food intake and weight regulation. Furthermore, any lesion effects upon feeding efficiency seen in that experiment must necessarily

be confounded by variations in the bird's responsiveness to food. Previous studies from Zeigler's laboratory (e.g. Megibow and Zeigler, 1968; Zeigler, 1976) have shown that such responsiveness varies directly with body weight loss which was uncontrolled in Experiment 1. For this reason, an attempt was made in the present experiment to study the effects of SMT and OMT lesions upon the efficiency of the pigeon's consummatory behavior under testing conditions in which responsiveness was high and relatively constant and in a situation permitting observations of both feeding and drinking. To this end, birds were tested at a fixed percentage (80%) of their ad libitum body weight. Furthermore, the relation between the stimulus properties of food and lesion effects upon feeding efficiency were explored by varying the size of the food object. Finally, a cinematographic analysis of the eating response was carried out in order to specify the consummatory response deficits with greater precision and to enable us to differentiate between consummatory deficits produced by lesions of efferent pathways and those seen in previous experiments after damage to trigeminal afferent structures.

Method

Subjects and Surgery

Ten male and six female pigeons were used. Housing and maintenance conditions were the same as those described in Experiment 1 as were the surgical and histological procedures. In the present experiment, seven birds received bilateral OMT lesions, but two were eliminated from analysis after several weeks of failing to initiate any feeding responses. (Both birds had extensive damage to the OMT and nearby structures, including the QFT.) Five birds sustained bilateral SMT lesions and four birds, served as surgical and lesion controls.

Apparatus

Birds were tested in an open-fronted observation chamber, measuring 40 x 75 x 40 cm, and painted flat white. The chamber was lit by two 20-w flourescent bulbs overhead and by two 14-w flourescent bulbs along the left wall. Grains were presented in a heavy, flat-bottomed glass bowl whose slightly concave wall measured 4 cm. The inside diameters at the top and bottom of the bowl measured 11.5 cm and 9.5 cm, respectively. A 250-ml graduated glass drinking tube was positioned on the right wall of the chamber.

Deprivation and Adaptation to the Testing Chamber

Following a 3-4 wk. period of stable body weight, a 20% weight loss was produced by gradually reducing their food ration. Throughout the experiment, they were kept at or

close to this weight in order to maintain a constant level of responsiveness to food during behavioral testing. Simultaneously with this weight loss regimen, birds were individually adapted to the observation chamber by limiting their access to food to a single, daily, 15-min. period inside the chamber. In addition, for three birds in each group, access to water was restricted to the session inside the chamber. (Pilot work had shown these procedures to quickly reduce the novelty of the testing environment, facilitate the transfer of feeding and drinking from the home cage to the testing chamber, and insure a prompt response to food and water during testing.) The remaining birds have free access to water in their home cages and in the observation chamber. Testing in the chamber began when birds reached the required body weight and the testing sequence is described below.

Procedure: Tests of Consummatory Efficiency for Food and Water

Consummatory Efficiency for Water: Drinking Test 1. At the start of each session, subjects were introduced individually into the chamber and adapted for three minutes. Birds previously deprived of water in their homes cages were now permitted access to the drinking tube in the chamber. The number of beak openings made during the act of drinking, and the amount of water ingested were then recorded. At the end of this period, a small plastic cup was placed over the opening of the tube, making the water temporarily inaccessible.

Consummatory Efficiency for Food: "Pick-up" Tests with Large and Small Grain. Following the adaptation/drinking period, birds were presented with a bowl containing 50 large grains (Peas) or 50 small grains (Milo). Latency to feed, number of pecking responses made, and number of grains consumed were then recorded. After a brief rest interval, subjects were presented with the bowl now containing 50 grains of the other size, and latency, feeding responses, and number of grains consumed were recorded as in the previous test. Testing with each grain continued until all the grains were eaten or until a ten minute interval, commencing with the introduction of grain into the chamber, had elapsed. The order of presentation of each grain was counterbalanced over sessions.

Peas and milo, the grains used in this part of the experiment, share several important physical characteristics: both are spherical or nearly spherical in shape, both are reddish-brown in color, and both are, within type, uniform in size. However, they differ markely from each other with respect to overall size (Peas; average diameter = 8 mm; Milo: average diameter = 2 mm), thus enabling an assessment of the relationship between grain size and consummatory efficiency before and after lesions.

For each test session with each grain, two different measures of feeding efficiency were calculated: the percentage of total grains consumed and the ratio of pecking responses to grains consumed. Data from previous work in this laboratory indicate that under these testing conditions normal birds consume

greater than 90% of the available grains and exhibit ratios of about 1.5 pecks per grain (Zeigler and Karten, 1973b; Zeigler, Miller & Levine, 1975). The highly stable nature of these measures over time provides a reliable baseline against which to evaluate deficits in the consummatory response of eating.

Consummatory Efficiency for Water: Drinking Test 2. Following the pick-up tests, the birds on the water deprivation schedule were permitted access to the drinking tube for an additional three minutes. Data were recorded as described previously for the first test with water. An index of each subject's drinking efficiency for each session was determined by combining the data from both daily tests and then calculating the ratio of beak openings per milliliter of water ingested.

Swallowing Test. Following testing in the observation chamber, subjects were placed in a plastic pigeon sleeve. Their bills were opened gently, a single pea was placed on the back of the tongue with forceps, and the bills were released. After five sec., the occurrence or non-occurrence of swallowing was noted. A series of 10 such swallowing trials concluded the test session for each bird.

Testing was conducted 3-6 days per week for at least two weeks preoperatively and for 3-5 weeks postoperatively, each bird receiving a minimum of 10 pre- and postoperative test sessions. Testing generally resumed 48-72 hr following surgery and continued until the bird's performance appeared stable over a two-week period. On all non-testing days, subjects were

placed in the chamber for a short period during which they were permitted free access to water and were presented with a quantity of grain comparable to that received during testing.

Prior to surgery, subjects always consumed enough food to maintain their body weights at 80% ad libitum; supplementary rations were therefore unnecessary. For several days following surgery, some birds were aphagic or otherwise unable to obtain adequate amounts of grain during testing. Force-feeding was therefore required to maintain their body weight at the 80% level. Immediately following surgery, water was returned and left on the home cages of the water deprived birds and the birds' drinking tests resumed only when attempts at feeding returned. Several OMT birds were adipsic for a short period after surgery and were either intubated or, if possible, encouraged to drink by gently immersing their beaks into the water.

During the postoperative tests of consummatory efficiency, protocols were kept of simple, observable behavioral patterns (e.g., postural adjustments, walking, preening, vocalization, and fear and escape responses), together with records of difficulties or abnormalities in consummatory behavior. In addition, several birds in each group underwent a series of neurological tests to determine the viability of a number of reflexive responses.

Cinematographic Procedures: A day or two following the final preoperative test of consummatory efficiency, birds were placed in the observation chamber and presented with small

heaps of either peas or milo. The grain was placed so as to permit an unobstructed view of the bird's head and beak during feeding. In addition, the grains were spaced a few millimeters apart to minimize ambiguity as to grain selected. Birds were then photographed twice, once with large grain and once with small grain. Filming was done at speeds of 100 frames/sec. using a HyCam 16 mm motion picture camera (Redlake Corporation, Santa Clara, California) and Kodak Tri-X film. Following surgery, the birds were photographed again when their feeding behavior appeared stable. Photographic data were analyzed using a L-W Photo-Optical Data Analyzer (L-W Photo, Incorporated, Van Nuys, California).

Results

Anatomical Data

The first two columns of Table 6 provide numerical estimates of the extent of damage to the OMT or SMT in each of the birds used in Experiment 2. As in the previous experiment, the amount of damage is coded by the following symbols: 0 = none; 1 = minimal; 2 = moderate to extensive; 3 = extensive to total. Figures 8a, b, and c present frontal sections through the brains of several representative pigeons to illustrate the maximum extent of the lesions.

A. Control Group

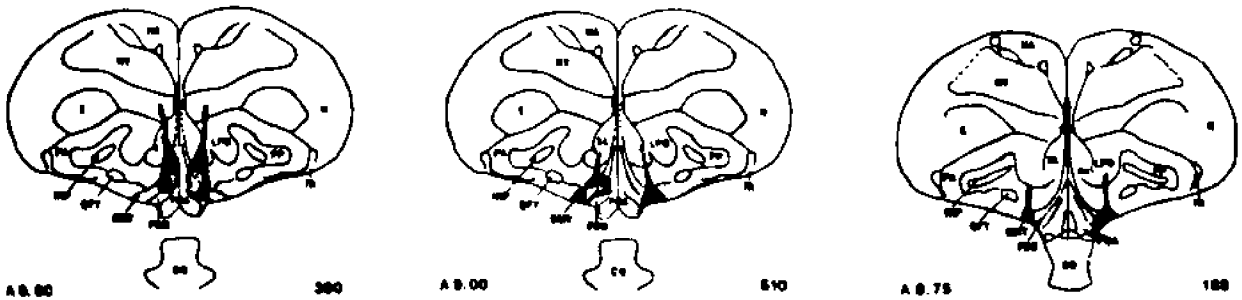
1. Lesioned Controls (Figure 8a). Two pigeons with bilateral destruction in the vicinity of either the OMT or SMT served as lesion controls. Bird 440 (OMT control) sustained fairly extensive damage to structures within the pretectal area, just dorsal to the OMT. In addition, the lesion on both sides interrupted fibers within the posterior and tectal commissures and impinged upon the isthmo-optic tract. OMT damage was restricted to a small region along the dorsomedial margin of the tract on the left side. The lesion in Bird 339 (SMT control) resulted in bilateral damage to the diagonal band of Broca and an adjacent anterior preoptic area. Posteriorly, the lesion extended along the ventral border of the medial forebrain bundle up to the anterior hypothalamus.

SMT involvement was limited to the destruction of a small portion of the right tract near the ventral telencephalic surface of the right hemisphere.

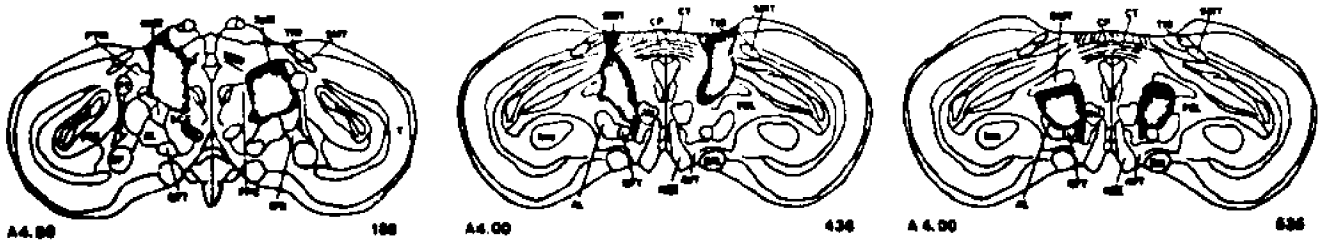
FIG. 8

A. CONTROL LESIONS

B. SMT LESIONS



C. OMT LESIONS



2. Sham Operates. In the remaining two control birds the electrodes were aimed at structures overlying either the OMT or SMT, but no current was passed.

B. SMT Lesions (Figure 8b)

SMT lesions generally produced more extensive damage to that pathway than was the case in the first experiment. In most birds the lesion also impinged medially upon nearby structures including the diagonal band of Broca and anterior preoptic region, and dorsally upon the nucleus accumbens. In some cases the lesion spread dorsally as far as the lateral septal nucleus. In Birds 390, 510, and 534, the lesion extended posteriorly into the anterior hypothalamus and in Bird 534 it encroached upon nuclei adjacent to the periventricular wall. In Bird 189, the lesion was placed more posteriorly than in the other birds resulting in moderate damage ventrolateralis within the thalamus and to the lateral margins of the optic tract. In addition, Bird 189 evidenced slight damage along the medial border of the quintofrontal tract on the right side. The QFT was spared entirely in all the other birds in this group.

C. OMT Lesions (Figure 8c)

The extent of lesions in all five birds in this group was generally comparable to those observed in Experiment 1. In each case, the lesion extended from the level of the pretectum up to and inclusive of the anterior margin of the tectal commissure (AP 4.75 - 3.75; Karten & Hodos, 1967). In all but a

single pigeon, OMT damage was accompanied by a slight to moderate degree of unilateral destruction to either the right (Birds 188 and 337) or left (Birds 321 and 436) lateral spiriform nucleus. In Birds 188 and 436, the lesion spread further laterally to include cells within the right or left nucleus pretectalis. Most birds evidenced some damage to both ansa lenticularis and nucleus ruber, but in all cases the damage was unilateral, and in all but one bird (535), it was judged to be minor. There was no evidence of damage to the quintofrontal tract in any of the OMT birds whose data is reported below.

Behavioral Data

A. General Observations

All birds showed a rapid recovery from surgery with no sign of impairment in posture, preening, and locomotion. While it was not possible to directly assess the effects of surgery upon flying behavior (wing and tail primaries were clipped), wing and tail movements appeared normal in birds placed on the floor when an escape response was elicited.

A variety of reflexes were also examined. Corneal, pupillary, grasp and righting responses were all demonstrable. Tactile and vestibular placing reactions also appeared intact, although visual placing often appeared sluggish in some birds with lesions of OMT. Head nystagmus (resulting from horizontal movement of the bird past the vertical bars of the colony room cages) and object avoidance (head withdrawl from an approaching

hand) were also unaffected.

Following lesions of the OMT, several birds were tame enough to perch calmly on an outstretched hand and several birds would even take grain from an open palm. In the anticipation of food, these birds often displayed a gentle "nibbling" behavior directed at the fingers that was reminiscent of the "begging" response of courting birds or hungry squabs. In addition, several OMT birds were often observed to calmly walk directly over the edge of the chamber floor and fall out onto the table below.

B. Effects Upon Consummatory Efficiency for Food: "Pick-up" Tests with Large (Peas) and Small (Milo) Grain

Tables 6 and 7 present for each grain the data for the "pick-up" tests of individual birds in each of the three groups. The number of observations for each bird is based upon tests conducted during the last 10 preoperative sessions and the first 10 postoperative sessions from the resumption of feeding. For several birds in the OMT group, the postoperative data represent a conservative estimate of the magnitude of the consummatory deficit, since, in each case, sessions in which pecking occurred without consumption of grain were omitted from the analysis of efficiency. In all groups, the extent of post-operative aphagia is shown as the number of days and sessions marked by an absence of feeding.

The tables include two different measures of feeding efficiency: the percentage of total grains consumed and the ratio of feeding responses to grains consumed. Efficiency

ratios were obtained by dividing the total number of observed pecks by the total number of grains eaten in the 10-day period. The mean number of feeding responses per session is also indicated to provide a measure of the birds' responsiveness to food. Preoperative data (N = 14) for both grains indicate that under the testing conditions of this experiment, birds consume about 97% (Peas, 95%; Milo, 99%) of the available grains, that the ratio of feeding responses to grains consumed is about 1.4 (Peas: Mean = 1.38, S.E. = .16; Milo: Mean = 1.42, S.E. = .18) and that performance is highly stable over time.

1. Consummatory efficiency with large grain (Peas). Since the overall effects upon consummatory efficiency in the pick-up tests do not differ significantly among control birds and birds with lesions of the SMT, the data for both groups are discussed together. As shown in Table 6, controls and SMT's resumed pecking and eating peas in the first postoperative session. There were no observable changes in responsiveness to food, as measured either by the median latency to feed (Preoperative: 1 sec; Postoperative: 1 sec.) or by the mean number of feeding responses made in each session. Most importantly, there were no demonstrable changes in consummatory efficiency as measured either by the percentage of grains eaten or by the calculated ratio of pecking responses to grains consumed. The data thus indicate that, irrespective of lesion size, bilateral destruction of the SMT has no significant effect upon either level of responsiveness or consummatory efficiency for large grains. In addition, the effect of SMT

TABLE 6

EFFECTS OF OMT AND SMT LESIONS UPON THE EFFICIENCY OF FEEDING BEHAVIOR: PEAS

BIRD NUMBER	LESION L	LESION R	Preoperative NO. OBS.	Preoperative RESPONSES/ SESSION	Preoperative PER CENT TOTAL GRAINS OBTAINED	Preoperative RATIO: PECKS/ GRAIN	Postoperative: Absence of Pecks DAYS	Postoperative: Absence of Pecks SESSIONS	Postoperative: Pecking NO. OBS.	Postoperative: Pecking RESPONSE/ SESSION	Postoperative: Pecking PER CENT TOTAL GRAINS OBTAINED	Postoperative: Pecking RATIO: PECKS/ GRAIN
CONTROL GROUP												
440	1	0	9	63	99	1.3	0	0	8	84	100	1.7
339	0	1	6	64	100	1.3	0	0	6	77	99	1.5
519 (SHAM)	-	-	8	85	100	1.7	0	0	9	93	99	1.9
521 (SHAM)	-	-	8	65	95	1.4	0	0	8	67	100	1.3
SMT GROUP												
390	3	3	6	59	100	1.2	0	0	8	64	100	1.3
509	3	2	6	65	100	1.3	0	0	7	60	91	1.3
534	2	3	6	45	71	1.3	0	0	6	45	70	1.3
510	2	2	6	55	100	1.1	0	0	7	58	100	1.2
189	1	1	8	65	99	1.3	0	0	9	72	100	1.4
OMT GROUP												
188	3	3	9	82	100	1.6	1[a]	1	9	496	44	23.6
436	3	2	9	45	67	1.4	14[b]	11	7	235	35	13.5
321	3	2	8	76	99	1.5	2[c]	2	6	642	94	13.7
337	2	2	6	65	100	1.3	0[d]	0	5	461	55	16.7
535	2	2	7	66	99	1.3	0	0	6	300	99	6.1

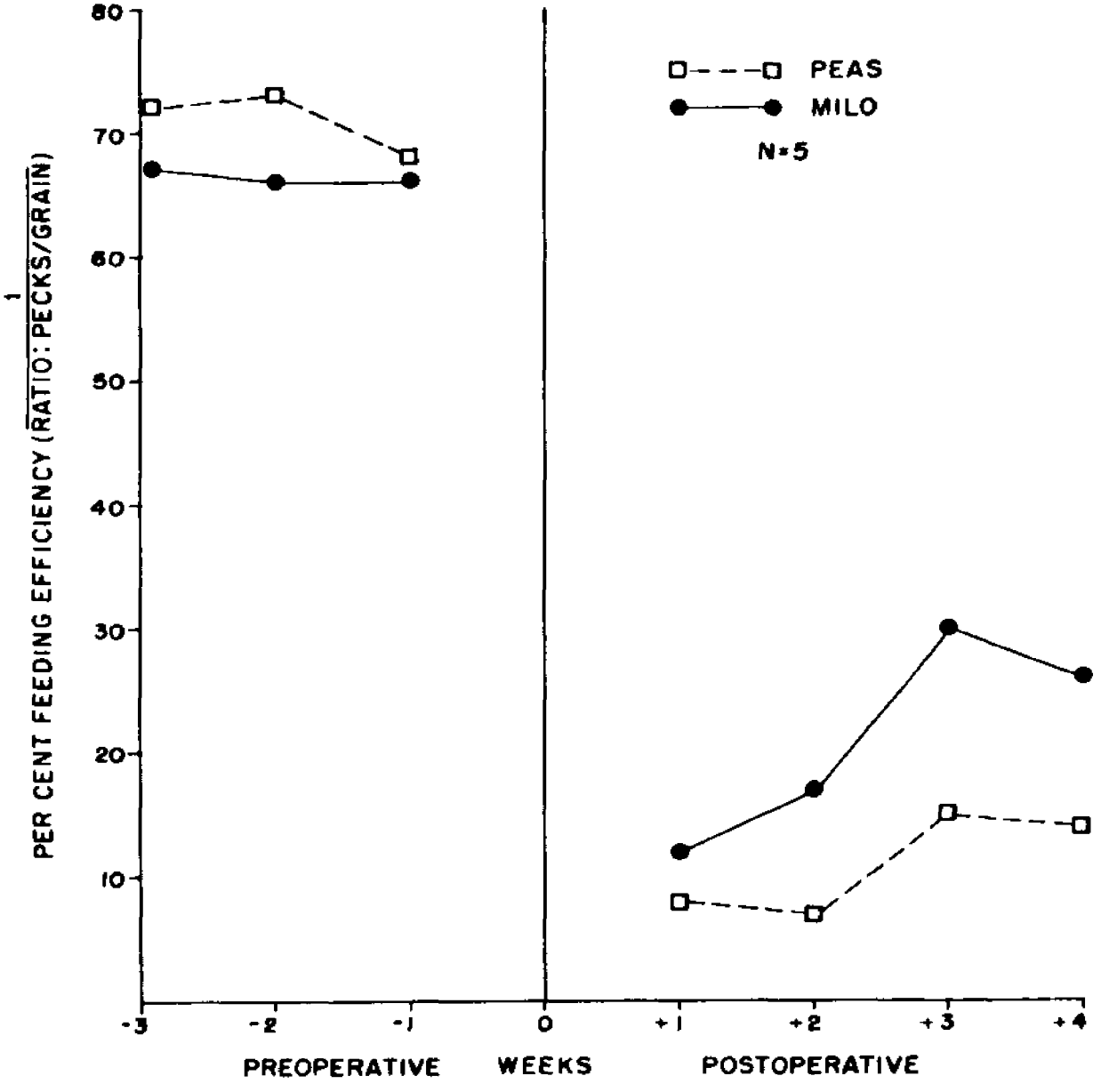
a - 1 day, 49 pecks, 0 grains
b - 5 days, 93 pecks, 0 grains
c - 4 days, 133 pecks, 0 grains
d - 1 day, 104 pecks, 0 grains

surgery on each of these measures is comparable with the of control procedures.

By contrast, birds with bilateral OMT lesions exhibited a significant reduction in feeding efficiency as measured by the percentage of grains consumed or by the ratio of feeding responses per grain, or both. Although this conclusion is based on the data of five birds, there is a generally consistent relationship between lesion size and the magnitude of the consummatory deficit (Spearman rank order $r = .58$). In several birds, the lesions resulted in a period of aphagia characterized by a complete absence of responding. During this period, birds spent a considerable time near the food or circling the food bowl, often stopping to place their heads over the food as though about to initiate pecking. The resumption of feeding was marked, in all but a single bird, by one or more sessions during which pecking occurred without the successful consumption of grain (Table 6). In these sessions, feeding responses often appeared hesitant, and in many cases, incomplete. In addition, many attempts at feeding were accompanied by a failure of the beak to open wide enough to permit grasping of the grain between the bill tips. Some birds also appeared to direct their pecking responses several millimeters beyond the grain, either missing the grain entirely or striking the grain with the underside of the lower bill. Many of these deficits appeared to persist throughout the duration of testing.

The persistence of these consummatory deficits with large grains is indicated in Figure 9. Consummatory efficiency is

FIG. 9


80
70
60
50
40
30
20
10
PER CENT FEEDING EFFICIENCY ($\frac{1}{\text{RATIO: PECKS/GRAIN}}$)
PEAS
MILO
N=5
-3
-2
-1
0
+1
+2
+3
+4
PREOPERATIVE
WEEKS
POSTOPERATIVE

expressed in this figure as the reciprocal of the ratio of pecks per grain, that is, the percentage of pecks resulting in the successful consumption of the grain. Pre- and post-operative data points represent the mean weekly consummatory efficiencies of all five birds in the OMT group. As shown in Figure 9, consummatory efficiency improved slightly over the first four postoperative weeks following the resumption of feeding. However, the final ratios still represent a significant reduction in efficiency from preoperative levels.

While OMT birds evidenced a postoperative reduction in the ability to consume large grain, once feeding resumed their responsiveness to food, judged either by median feeding latencies (Preoperative: 1 sec; Postoperative: 1 sec) or by numbers of feeding responses, was at or above preoperative levels. Indeed, in each case the mean number of feeding responses over the first 10 sessions from the resumption of eating exceeded the preoperative mean (Table 6). The observed increase in the number of feeding responses probably reflects the extra responses made to compensate for the reduction in grains obtained by the lesion. Thus, in birds reduced to 80% of their ad lib body weight, high levels of responsiveness are not incompatible with reduced feeding efficiency, at least in short-term tests of feeding. In the absence of a significant reduction in the postoperative responsiveness to food following the resumption of eating, the reduction in the consumption of large grain must be attributable to the reduction of efficiency in the birds' feeding behavior.

2. Consummatory efficiency with small grain (Milo). Table 7 summarizes the data of the pick-up tests with milo for individual birds in each of the three groups. Control and SMT pigeons resumed eating milo on the first postoperative session. There were no significant postoperative changes in median feeding latency (Preoperative: 1 sec; Postoperative: 2 sec; Median test, χ^2 (1) = 1.28, $p > .10$, NS), responses per session, percent grains obtained, or in the ratio of pecks per grain. In general, the postoperative feeding behavior of both control and SMT groups was indistinguishable from its preoperative performance.

By contrast, pigeons with lesions of the OMT displayed a pattern of postoperative behavior similar to that observed in their tests with large grain. In three birds, the lesions produced periods of aphagia (non-responding) ranging from one to fifteen days. In all but a single case, the resumption of feeding was characterized by one or more days in which the birds failed to obtain any grain (Table 7). However, following the resumption of feeding, responsiveness to milo returned to preoperative levels, as measured either by the median feeding latency (Preoperative: 2 sec; Postoperative, 4 sec; Median test, χ^2 (1) = .45, $p > .10$, NS) or by the number of observed feeding responses (Table 7). As in tests with peas, all birds evidenced a significant reduction in consummatory efficiency, judged either by their efficiency ratios or by their percentages of total grains consumed. The relationship between the extent of OMT damage and the consummatory deficit with milo was identical to that found with peas (Spearman rank

TABLE 7

EFFECTS OF OMT AND SMT LESIONS UPON THE EFFICIENCY OF FEEDING BEHAVIOR: MILO

BIRD NUMBER	PREOPERATIVE NO. OBS.	PREOPERATIVE RESP./ SESSION	PREOPERATIVE PER CENT TOTAL GRAINS OBTAINED	PREOPERATIVE RATIO: PECKS/ GRAINS	POSTOPERATIVE: ABSENCE OF PECKS DAYS	POSTOPERATIVE: ABSENCE OF PECKS SESSIONS	POSTOPERATIVE: PECKING NO. OBS.	POSTOPERATIVE: PECKING RESP./ SESSION	POSTOPERATIVE: PECKING PER CENT TOTAL GRAINS OBTAINED	POSTOPERATIVE: PECKING RATIO: PECKS/ GRAINS
CONTROL GROUP										
449	9	76	99	1.5	0	0	8	77	92	1.7
339	6	68	99	1.4	0	0	6	72	91	1.6
519	8	76	100	1.5	0	0	9	80	100	1.6
521	8	75	100	1.5	0	0	8	79	99	1.6
SMT GROUP										
390	6	62	99	1.3	0	0	8	75	100	1.5
509	6	69	99	1.4	0	0	7	68	100	1.4
534	6	67	100	1.3	0	0	6	70	100	1.4
510	6	60	100	1.2	0	0	7	57	96	1.2
189	8	65	99	1.3	0	0	9	68	98	1.4
OMT GROUP										
188	9	94	99	1.9	1[a]	1	9	308	47	13.1
436	9	75	96	1.6	15[b]	12	7	155	69	4.5
321	8	70	99	1.4	5[c]	5	6	141	99	2.8
337	6	62	100	1.2	0[d]	0	5	232	47	9.9
535	7	67	99	1.4	0	0	6	206	98	4.2

[a] 2 days, 51 pecks, 0 grains
[b] 1 day, 14 pecks, 0 grains
[c] 1 day, 3 pecks, 0 grains
[d] 1 day, 59 pecks, 0 grains

order $\underline{r}$ = .58).

The persistence of consummatory inefficiency with milo for birds with lesions of the OMT is shown in Figure 9. While the performance of the group improved over the duration of testing, efficiency remained significantly below pre-operative levels even after several weeks following the resumption of feeding. However, over the entire postoperative period, the percentage of pecks resulting in successful eating was consistently greater for milo than for peas. This may also be seen in the individual efficiency ratios of OMT birds in Tables 6 and 7. The smaller size of the grain may have played an important role in this behavioral difference, especially in view of the noticeable difficulties encountered by several OMT birds in opening their bills to permit grasping of the larger grain. A more detailed examination of the consummatory deficits with both peas and milo is presented below.

C. Effects Upon the Efficiency of Drinking

Six control and SMT pigeons began drinking within 24 hours following surgery, while the remaining birds each began within 48 hours. Among the OMT's only Bird 321 immediately resumed drinking. Birds 188, 337, and 535 each experienced three days adipsia, while Bird 436 evidenced 20 days of adipsia -- each required intubation or hand assistance in drinking.

The results of lesions or control procedures upon the efficiency of drinking is shown in Table 8 for three birds in each group. Efficiency is expressed as the ratio of beak openings per milliliter of water ingested. To insure uniformity of the response, data were analyzed only for bouts of drinking in which the bird's bills were immersed deeply into the water and which were 10 ml or more in volume. Thus, the last two pre- and postoperative columns in Table 8 indicate the totals upon which the efficiency data are based, rather than an estimate of the bird's responsiveness to water.

Thus, drinking efficiency was unimpaired by SMT lesions or control procedures. Moreover, in contrast with its disruptive effect upon feeding, bilateral destruction of the OMT also produced no impairment in the efficiency of the drinking response (Table 8).

D. Effects of Lesions Upon Swallowing

In all birds within each of the three groups, the mean probability of swallowing a grain placed at the back of the mouth did not differ in the pre- and postoperative conditions (Preoperative probability = 100%; Postoperative probability = 100%).

TABLE 8

EFFECT OF OMT AND SMT LESIONS UPON EFFICIENCY OF DRINKING

	Preoperative			Postoperative		
Bird Number	No. Sessions	ML. Drunk	Efficiency*	No. Sessions	ML. Drunk	Efficiency*
CONTROL GROUP						
339	6	162	4.3	6	252	3.0
519	8	208	3.0	8	296	4.4
521	7	175	3.5	5	195	3.7
SMT GROUP						
509	6	120	3.0	6	204	2.6
510	6	144	2.6	6	240	1.8
534	6	114	5.3	6	162	2.6
OMT GROUP						
321	8	184	2.4	5	155	1.3
337	6	144	1.7	4	60	1.6
535	7	150	5.9	4	76	4.0

* Ratio of beak openings per milliliter of water ingested

E. Effects of OMT Lesions Upon the Consummatory Feeding Response: Cinematographic Analysis of Eating

Observations of the eating behavior of OMT birds in the pick-up tests suggested that consummatory deficits were due to reductions in the extent of mouth opening or impairments in pecking accuracy, or both. High- speed photography was employed to specify more precisely the nature of the deficits produced by OMT lesions upon the consummatory feeding response.

1. Eating in the pigeon: Qualitative description. In the normal pigeon eating may be divided into a cycle of four distinct response sequences: Pecking, grasping, mandibulation, and swallowing (Zeigler, 1976). The response cycle begins with pecking, which consists of a downward movement of the head with beak initially closed and the eye fully open. As the head approaches the grain, the beak gradually open and the eyelids and nictitating membrane close so that the eye is shut just before contact is made with the substrate or grain. Just prior to the grasp, the width of the beak is at its maximum extent, and this extent varies with the size of the grain (Figure 10). The width of the oral aperture at this point is generally 2 - 3 millimeters wider than the width of the grain. Contact with the grain or the substrate then terminates the downward movement of the head and elicits grasping. During this act, the lower beak shuts while the upper beak remains stationary, in effect serving as a barrier against additional forward progression of the lower beak and grain. With the grain now firmly seized fore and-aft by the

bill the head is sharply withdrawn and the eye begins to open. If the grain is large, such as a pea, the beak may reopen and the upward movement of the head momentarily halted. The head may then be quickly brought forward and the grain re-grasped in mid-air. The momentum generated by the quick upward thrust of the head also serves to propel the grain from the bill tips deeper into the buccal cavity. Mandibulation occurs as the head resumes its upward movement. The tongue is now brought under the grain and the grain is moved along the palate to the back of the throat by one or more "peristatic" movements of the tongue. Swallowing at this point terminates the feeding response cycle.

2. Effects of OMT lesions. The cinematographic analysis of the pigeon's feeding behavior following lesions of the OMT confirmed the existence of two types of consummatory deficit -- one involving incomplete opening of the oral aperture and the other involving inaccurate placements of the head and beak with respect to the grain. The percentages of each type of impairment for both peas and milo are presented for individual birds in Table 9 . Because of the relatively small number of unsuccessful grasping attempts characteristic of intact pigeons, the preoperative data are combined for all five birds.

The data in Table 9 are based on 538 responses recorded on approximately 1500 feet of film. A few pecks were eliminated from the data of each bird because the angle of the animal's head with respect to the camera obscured the beak and grain and prevented analysis. Thus, for each bird, the number of

TABLE 9

OMT LESIONS: CONSUMMATORY RESPONSE DEFICITS

A. ANALYSIS OF DEFICIT TYPES

	Preoperative		Postoperative									
Bird No.	Mean Combined N=5		188		321		337		436		535	
Grain	Peas	Milo	Peas	Milo	Peas	Milo	Peas	Milo	Peas	Milo	Peas	Milo
	N=52	N=51	N=51	N=63	N=82	N=31	N=50	N=55	N=29	N=14	N=35	N=25
Orientation % Deficit	81	96	84	100	62	97	92	100	100	100	74	92
Aperture % Deficit	17	4	73	3	95	16	52	2	34	0	71	8

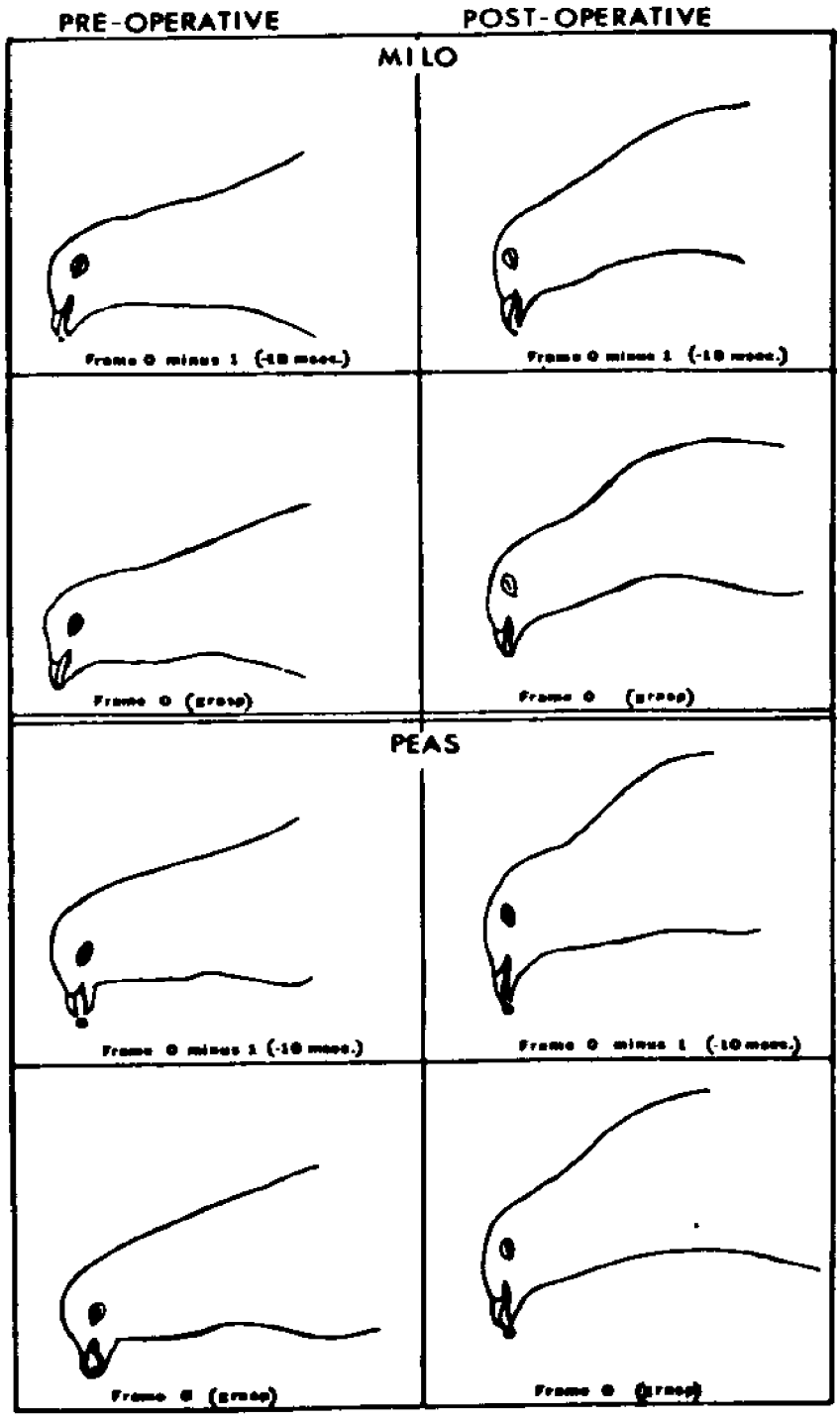
responses upon which the results are based represents those pecks which permitted analysis, rather than an indication of the bird's efficiency or responsiveness to food. However, the range of pre-and postoperative efficiency ratios were comparable to those observed in the tests of consummatory efficiency (Peas: Preoperative, 1.1 - 1.9; Postoperative, 2.7 - 20.0; Milo: Preoperative, 1.1 - 1.7; Postoperative, 1.8 - 11.5).

Deficits related to the size of the oral aperture. As shown in Table 9 , prior to surgery, only a relatively small percentage of unsuccessful grasping attempts were due to insufficient opening of the oral aperture. Following lesions of the OMT, the percentage of aperture-related deficits for milo remained near or only slightly above preoperative levels. However, in tests with peas, OMT destruction resulted in a significant increase in this particular deficit. The restriction of this deficit to feeding responses directed at the large grain suggest that OMT damage may have impaired processes underlying the neuromotor control of beak opening.

Figure 10, which presents schematics of individual movie frames of four separate pecks, illustrates the relationship between grain size and aperture adjustment in a single, representative bird. Each frame represents a temporal interval of 10 msec and shows the activity just prior to grasp or contact, and the actual grasp or contact itself. An examination of these figures indicates that, preoperatively, the width of the oral aperture just prior to grasping is adjusted according to the size of the grain, and this is

FIG. 10

BIRD 321 OMT



accomplished as the head is moving and the eye practically closed. Following lesions of the OMT, beak adjustment is successful only with the smaller grain. Pecks at the larger grain are generally characterized by an inability of the beak to open sufficiently, even though, as in this case, there may be no deviation of the head and beak orientation with respect to the grain.

Quantitative data illustrating this consummatory deficit with large grain are presented for Birds 188 and 535 in Table 10 . The data in Table 10 were obtained by measuring the distance between the open bill tips utilizing the ruled grid directly behind the bird's head. Measurements were limited to pecks during which the head was judged to be perpendicular to the camera axis. The column labelled "Frame 0" represents the single frame during which the grain was grasped, or in the case of an unsuccessful peck, the frame in which the bill tips contacted the substrate or grain. "Frame 0 minus 1" signifies the 10 msec period just prior to grasping or contact at which point the bills were maximally opened. Inspection of the data in Table 10 confirms that lesions of the OMT may impair the degree to which the oral aperture may open prior to and during actual contact with the grain.

Deficits relating to impaired head orientation. The data in Table 9, indicate that a large percentage of unsuccessful grasping attempts observed preoperatively were characterized by inaccurate spatial placements of the head and beak with respect to the grain. Postoperatively, these percentages remained nearly identical for individual birds.

TABLE 10

OMT LESIONS: CONSUMMATORY RESPONSE DEFICITS

B. EFFECTS UPON BEAK WIDTH (IN MILLIMETERS)

	Preoperative			Postoperative		
Bird No.	No. Resp.	Frame -1	0	No. Resp.	Frame -1	0
188	27	1.00	.80	21	.48	.26
535	29	.93	.80	29	.74	.60

However, grain size appeared to be a much less important determining factor in the occurrence of orientation-related deficits than in those involving the oral aperture, although the percentages of such orientation deficits were generally observed to be slightly higher for milo than for peas. In the normal pigeon, the major portion of inaccurate responses were directed to either the left or right side of the grain; few inaccurate pecks were directed elsewhere. However, observations of feeding behavior provided by high-speed cinematography revealed that lesions of the OMT resulted in a preponderance of pecks several millimeters beyond the grain, implying that such lesions may have disrupted processes underlying the visuomotor control of the consummatory response.

These relationships are shown in Table 11 , which indicates the nature of the head and beak orientation for inaccurate pecks before and after lesions of the OMT. For each bird, movie frames of spatially inaccurate feeding responses were individually examined, and based upon the position of the head and beak relative to the grain, the orientation of each recorded in one of the categories in the left-hand column of the table. The categories "too far right" - "too far left" and "overshoot" - "undershoot" specify inaccurate responses along an imaginary set of coordinate axes intersecting at the midpoint of the grain. The remaining categories specify responses within any one of the quadrants formed by axes intersection (See Figure 11). The total number of responses analyzed for each bird is shown under the appropriate grain. Numbers in the body of the table represent

TABLE 11

OMT LESIONS: CONSUMMATORY RESPONSE DEFICITS

C. ORIENTATION DEFICITS

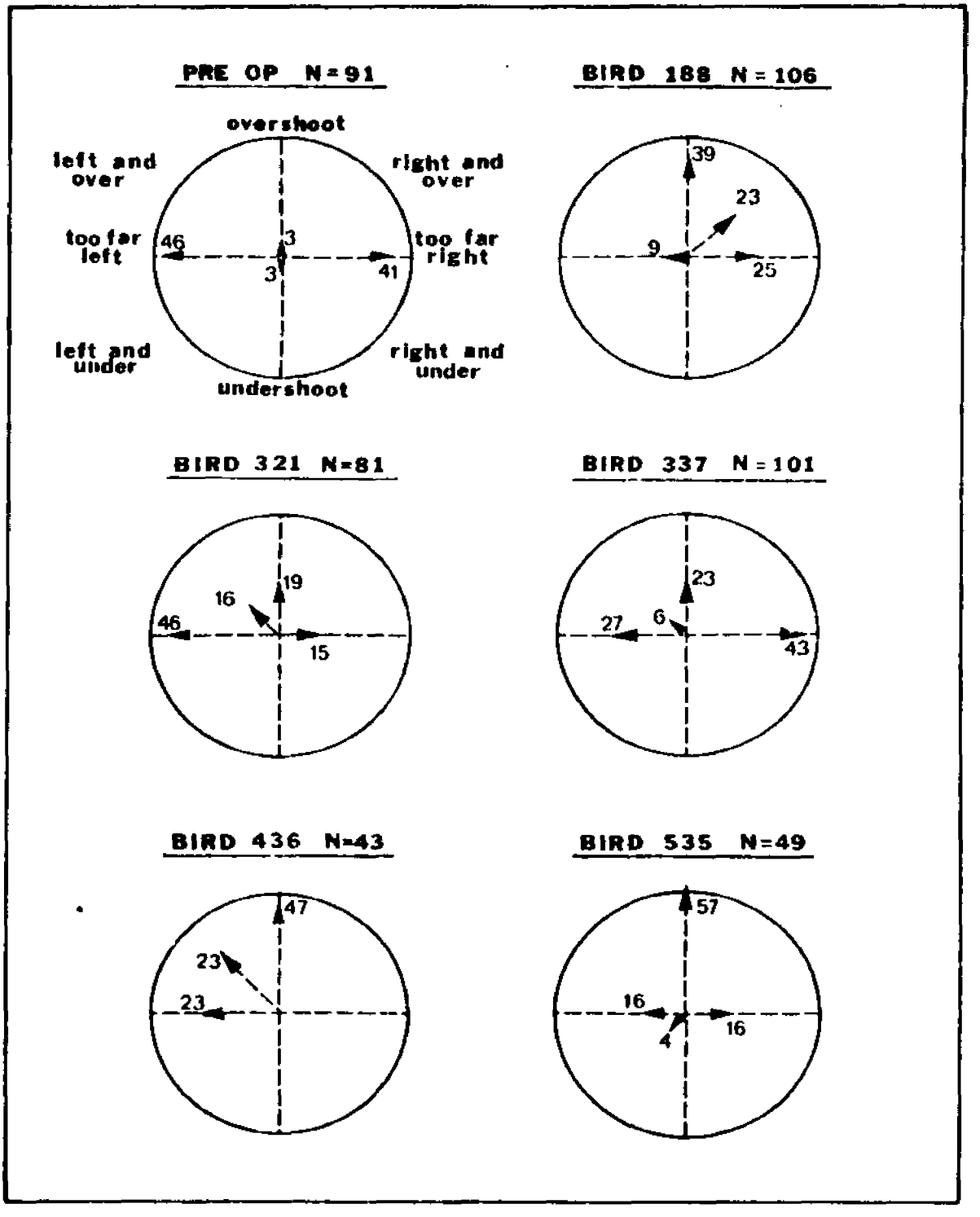
	Preoperative		Postoperative									
Bird No.	Combined N=5		188		321		337		436		535	
Grain/ Orientation Deficit	Peas N=42	Milo N=49	Peas N=43	Milo N=63	Peas N=51	Milo N=30	Peas N=46	Milo N=55	Peas N=29	Milo N=14	Peas N=26	Milo N=23
Too far right %	33	47	21	27	16	13	46	40	0	7	15	17
Too far left %	48	45	7	11	45	47	20	33	21	29	27	9
Overshoot %	5	2	47	33	22	13	26	20	45	50	58	57
Undershoot %	2	4	2	0	4	0	0	0	0	0	0	4
Right & over %	5	0	23	22	2	0	4	0	3	0	0	0
Right & under %	0	2	0	2	0	3	0	0	3	0	0	4
Left & over %	5	0	0	2	12	23	4	7	28	14	0	4
Left & under %	2	0	0	3	0	0	0	0	0	0	0	4

percentages of responses within each orientation category. Preoperative percentages were calculated from the combined data of all five animals.

An examination of the data in Table 11 indicates that for individual birds grain size had little or no effect upon the percentage of inaccurate responses within a particular orientation category. As previously noted, normal birds tended to distribute their inaccurate pecks fairly evenly to the left and right sides of the grain. Following surgery, however, several birds evidenced an orientation "bias", so that inaccurate pecks on either side of the grain were directed predominantly to one side or the other (Right: Birds 188 and 337; Left: Birds 321 and 436). In addition, each bird displayed a significant increase in the percentage of their responses within the "overshoot" category or within the combined "right and overshoot" and "left and overshoot" categories.

Figure 11 illustrates these data as a series of polar diagrams for peas and milo combined. Each diagram represents a schematic, "bird's-eye" view of the "pecking field" as seen from above. The position of the grain is arbitrarily designated at the point of axes intersection (origin). Pecks on either side of the grain are represented by "vectors" drawn along the abscissa; overshoots and undershoots are represented by vectors along the ordinate. Vectors representing the combined spatial orientation deficits are drawn at 45° angles to the principal axes. The length of each vector indicates the relative magnitude of a particular orientation deficit and the numbers beside each show the actual percentage of

FIG. 11


PRE OP N=91
overshoot
left and over
right and over
too far left
too far right
left and under
right and under
undershoot
46
3
3
41
BIRD 188 N=106
39
23
9
25
BIRD 321 N=81
16
19
46
15
BIRD 337 N=101
23
27
6
43
BIRD 436 N=43
47
23
23
BIRD 535 N=49
57
16
4
16

responses within that category. For clarity of presentation, vectors representing orientation categories of less than 3% of the total response have been omitted.

It is evident from Figure 11 that subjects in the "OMT" group exhibit a large percentage of feeding responses which are directed beyond the locus of the grain. These overshoots may also combine with orientation deficits either to the right or left side of the grain to produce a compound orientation deficit. There is a relationship between the magnitude of the overshoot deficit and the extent of OMT destruction (Spearman rank-order r = .60), but no such correlation between OMT damage and right-left inaccuracies. The relation between lesion size and locus and the nature and extent of these visuomotor deficits is discussed below.

Discussion

An examination of the feedometer data in Experiment 1 and informal observations of feeding suggested that both OMT and SMT lesions produce changes in consummatory behavior. Lesions of SMT resulted in a mode of feeding behavior which could be classified as "sorting" even though only a single grain (milo) was presented for consumption. The eating of OMT birds was characterized by impaired grasping. Unfortunately, the measurement of feeding efficiency in Experiment 1 may have been confounded by uncontrolled variations in the bird's responsiveness to food. Thus, in the present experiment, an attempt was made to examine the effects of SMT and OMT damage upon the pigeon's feeding efficiency under conditions of constant responsiveness. In addition, we examined the effects of such lesions upon the consummatory behaviors of drinking and swallowing.

The results of the present experiment indicate that lesions of the SMT (and adjacent structures) have no significant effects upon any of the measures of consummatory performance. Responsiveness to food remained at preoperative levels in SMT birds and their feeding efficiency, drinking efficiency and swallowing were unimpaired. The "sorting" behavior seen in SMT birds in Experiment 1 was absent during efficiency tests when small amounts (50 grains) of milo was presented for consumption. But "sorting" was clearly evident in most SMT birds when large heaps (10 grams or more) were presented as a supplementary food

ration. During these sessions, birds made many of the sorting responses characteristic of ad libitum SMT birds in the first experiment, and on several occasions, refused this grain altogether. These behaviors were never observed during the presentation of peas, the larger of the two grains. Since milo has generally been found to be a preferred grain in previous studies from this laboratory (Moon, 1975), this change in preference is worthy of comment.

Birds with bilateral OMT lesions showed no impairment in either swallowing or drinking. However, the consummatory response of eating was severely disrupted and these effects were more profound when testing was conducted with the larger to the two grains (peas). Moreover, these feeding deficiencies persisted throughout the duration of testing with evidence of only slight improvement.

Observations of the eating behavior of OMT birds in tests of consummatory efficiency suggested that feeding deficits were of two types -- "somatomotor" deficits involving incomplete opening of the oral aperture and "orientation" deficits involving inaccurate pecking. Analysis of film taken at high speeds confirmed the existence of both types of deficit. Somatomotor deficits involving the oral aperture were most obvious during feeding attempts with large grains. Under these conditions, OMT birds failed to open their beaks wide enough to permit successful grasping of the large grain. Orientation deficits during pecking, however, appeared to be independent of grain size. Such deficits were of two types -- one involving an

"overshooting" of the grain and the other involving inaccurate left-right placements of the beak with respect to the grain. While there was a relationship between the extent of OMT damage and the degree of the overshoot deficit, there was no such relationship between OMT damage and left-right orientation deficits. However, a correlation between such orientation deficits and damage to lateral spiriform and pretectal nuclei was evident. Birds with unilateral lesions of these nuclei were characterized by orientation deficits which correspond to the laterality of the lesion. In one bird (Bird 535) with bilateral damage to these nuclei, the deficit was approximately equal on both sides. In all cases, the magnitude of the orientation deficit was related to the extent of the damage.

The results of this experiment indicate, that while lesions of SMT do not produce deficits in the somatomotor control or orientation of pecking, such lesions appear to affect the bird's responses to food in a manner suggesting a change in its visually controlled preference behavior. Lesions of the OMT, on the other hand, disrupt both somatomotor (impaired beak opening) and visuomotor (overshooting) processes underlying the consummatory response of feeding.

Experiment 3: Effects of OMT and SMT lesions upon food reinforced operant behavior.

Under conditions of food deprivation, pigeons show an increased responsiveness to food which may be reflected in some parameter of feeding behavior itself, or in the probability that the animal will engage in previously learned instrumental behaviors reinforced with food. The observed relation between deprivation and its behavioral consequences is assumed to represent the operation of motivational variables (Bolles, 1975). In view of the widespread use of the operant paradigm to measure "hunger" in studies relating brain mechanisms to feeding behavior, we examined the effects of OMT and SMT lesions upon key-pecking responses reinforced with food.

Previous studies from this laboratory have shown that lesions of quintofrontal structures (PrV, QFT, and NB) abolish food-reinforced key-pecking (VI-60 sec.) for periods ranging from a few days to a few weeks postoperatively (Zeigler & Karten, 1973b). During this time, birds do not make pecking responses at food during probe trials when the food hopper is presented, although they will often accept a supplementary food ration offered either in the operant chamber or in the home cage. Key-pecking and feeding responses return to preoperative levels fairly abruptly, although feeding efficiency, as measured by the ratio of feeding responses to food intake, is significantly reduced (Zeigler & Karten, 1973b). By contrast, destruction of peripheral trigeminal structures (trigeminal deafferentation) produces no immediate impairment

of operant performance, although there is a significant reduction in responses to food in the reinforcement interval. The consequent absence of reinforcement, in turn, eventually leads to a gradual decline in key-pecking similar to that seen during extinction in normal pigeons (Zeigler, 1975).

Interestingly, Zeier (1971) has reported deficits in both operant and consummatory performance following destruction of the somatosensory portion of the archistriatum, the region from which OMT originates. However, the deficits that Zeier reports in key-pecking are of a different nature from those observed in quinto-frontal birds. Following anterior archi striatal lesions, his animals were observed to increase their rate of operant responding on a VI-60 second schedule, while decreasing their rate on a DRL schedule. An opposite effect is observed after lesions to the limbic portion of the archi-striatum. Since he suggests that "optimal" performance on both schedules involves some degree of response inhibition requiring a low rate of key-pecking, he interprets the performance of his birds on the VI schedule as an impairment in "operant efficiency" (Zeier, 1971). In addition, he suggests that these effects may be due to a "motor disinhibition" and that the OMT may be the revelant structure involved. His data on VI performance appear contrary to the bulk of behavioral data from this laboratory which suggests that lesions to structures projecting upon the archistriatum (e.g., NB) impair VI performance.

The present experiment was designed to examine the effects of lesions of OMT and SMT upon food reinforced operant conditioning using a paradigm which permits the dissociation of lesion effects upon both operant and consummatory behaviors.

Method

Subjects

A total of 18 pigeons served as subjects. The birds, both male and female, ranged from 3 - 7 yrs. in age and from 500 - 700g in weight at the start of the experiment. Housing, maintenance, and laboratory adaptation procedures were identical to those described in Experiment 1.

Experimental histories. Seven of the pigeons used in this study had previous experimental experience. Pigeons 323, 368, 417 and 418 were used, several months earlier, in a study designed to examine the effects of short-term weight loss on food intake. Each had undergone 5-6 consecutive days of food deprivation resulting in a temporary body weight loss of 10 per cent. When permitted to resume ad libitum feeding, however, they quickly regained their lost weight. Pigeon 513 experienced a greater weight loss, approaching 30 per cent of its ad libitum weight, in an experiment designed to study the effects of food deprivation upon grain-type preferences. When permitted to refeed, this bird also rapidly regained its previously lost weight. Pigeons 512 and 536 also participated in an earlier grain preference study; however, these birds were supplied with a variety of grains and never experienced weight loss. For 6 - 9 months prior to their inclusion in the present study, all seven pigeons were at full weight and living undisturbed under ad libitum conditions in the colony room. The 11 remaining birds were experimentally naive.

Apparatus

The pigeons were trained and tested in a conventional two-key operant chamber, but only the left key was used. The response key was made of transluscent plexiglass and was located behind a 3 cm aperture approximately 25 cm above the wire mesh chamber floor. The key was transilluminated by a 6-w red lamp and required a minimum force of about 20g for its operation. A 6-w clear lamp mounted on the chamber ceiling provided continuous ambient illumination during testing.

Key-pecking responses made during reinforcement availability operated a standard solenoid-controlled feeder which was located behind a 5 cm X 6 cm opening in the center of the intelligence panel, 5.5 cm above the floor. During reinforcement, the key light was extinguished and the feeder was illuminated by a red lamp which also served as the light source for a photocell in a photodetector circuit. Both the lamp and the photocell were mounted on opposite sides of the food magazine enabling feeding responses to be automatically monitored (see Zeigler & Feldstein, 1971, for a complete description of the photodetector circuitry).

Conventional reed-relay programming and recording equipment was located behind two floor-to-ceiling wooden partitions approximately 3 m from the chamber. Equipment utilizing electro-mechanical relays (e.g., cumulative recorders) was located inside a sound attenuated cabinet in an adjacent room. Attenuation of extraneous noise was accomplished by housing the operant chamber within a second, sound-insulated outer chamber and by supplying both chambers continuously with white noise.

During testing, the occurrence of key-pecking and the number of reinforcements obtained were recorded on separate counters. Feeding responses monitored by the magazine photocell and lamp were recorded on a third counter. Prior to testing each bird, a measured amount (100 g) of fresh grain was placed inside the food hopper. Following testing, spillage of grain due to the operation of the solenoid or to the bird's inefficiency was collected and replaced in the hopper. The amount of food consumed was then calculated to the nearest .5-g by subtracting the amount of food remaining in the hopper from the amount originally placed inside. With the above arrangements it was therefore possible to record for each subject in each test session the number of key-pecking responses, the number of reinforcements delivered, the number of feeding responses made and the amount of food consumed. In addition, calculation of the ratio of feeding responses per gram of food consumed provided a measure of the bird's feeding efficiency. Furthermore, individual cumulative records for both key-pecking and feeding responses were kept for the last preoperative week and for the duration of the postoperative testing.

Procedure

Pigeons were subjected to a 23 hr. food deprivation schedule while undergoing daily adaptation/feeding sessions in the operant chamber. During this period, the birds were gradually reduced to 80 per cent of their free-feeding weight by limiting their food ration and were trained to feed exclusively from the

food hopper within the chamber. As the pigeons reached their target weight they were hand-shaped to peck at the response key and then gradually shifted from a CRF to a VI-60 sec schedule of reinforcement. Reinforcement consisted of 8-sec access to a three-grain mixture (50% milo, 40% vetch, 10% hempseed).

Testing was conducted in 30 min. sessions, at approximately the same hour daily, 5 - 7 days per week, and continued pre-operatively until stable baseline data were available. (The criterion for stability of performance was arbitrarily designated as the absence of increasing or decreasing trends in key-pecking and feeding behavior for 10 consecutive sessions as determined by visual inspection of the data.) The 30 min. daily session yielded, on the average, 30 reinforcements or 240 seconds (8 sec/R X 30 R's) of feeding time per bird. These conditions provided most normal birds with the opportunity to eat between 10 and 20 g of food and thereby receive their entire daily ration during a test session. Supplementary feedings, when they occurred, were administered either in the chamber itself (e.g., as a test of responsiveness to food or as the normal ration on non-testing days) or in the home cage at least one hour following the termination of testing. During periods of aphagia or adipsia, hand-assisted feeding or drinking was administered to maintain the bird's body weight at the 80% level.

Following preoperative training, the birds were matched for body weight and operant performance and then surgery was performed. One group of pigeons (N=8) received bilateral lesions in the OMT while a second group (N=6) received bilateral SMT lesions. Birds

with lesions in the vicinity of the OMT or SMT but with one or both tracts of either structure spared, served as a lesioned control group (N=2). A surgical control group comprised of 2 birds underwent operative procedures duplicating those of the lesioned birds except for the passage of current through the electrode.

Postoperatively, testing resumed following a 48 - 72 hr. period of recovery and continued for a minimum of four weeks, with most birds receiving at least 21 postoperative sessions. From time to time, the birds were observed in the darkened testing room through a glass window in the outer chamber.

In four birds, the duration of the reinforcement interval was manipulated to assess the possibility that failure to obtain reinforcement was due to an increased latency of response to presentation of the food hopper. For this purpose, the reinforcement interval was increased from 8 - 15 sec. for a series of 8 - 10 sessions. On any postoperative session during which key-pecking failed to occur or during which birds failed to eat during food presentation, three probe reinforcement trials were administered at random intervals to determine whether the birds were responsive to the grain reinforcement. Birds were kept in the operant chamber for the entire 30 min. regardless of the non-occurrence of key-pecking and/or feeding.

Results

Anatomical Data

Coronal sections illustrating maximal lesion extent in representative control and experimental pigeons are shown in Figure 12 and the anatomical data for all birds are summarized in Table 12 . The extent of damage is coded as before: 0 = none; 1 = minimal; 2 = moderate to extensive; 3 = extensive to total.

A. Control Group

1. Lesioned Controls (Figure 12c). Two birds with slight unilateral damage to either the SMT or OMT served as lesioned controls. Bird 323 (OMT control) sustained slight damage to the OMT on the left side, while on the right, damage was limited to a restricted portion of area pretectalis overlying the tract. Bird 514 (SMT control) evidenced slight damage to SMT on the left side. In addition, the lesion encroached upon cells within the diagonal band of Broca and the anterior preoptic nucleus. On the right side, the damage was largely confined to nucleus accumbens and the diagonal band of Broca.

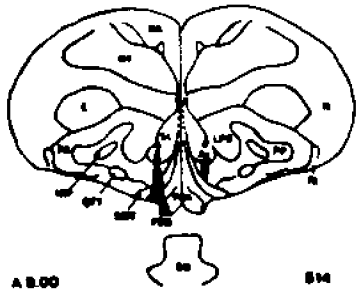
2. Sham Controls. In addition to the lesioned controls, two birds which underwent all surgical procedures, except for current passage through their electrodes, served as sham controls.

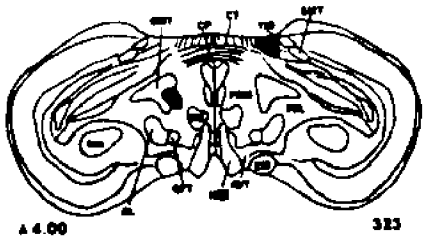
B. SMT Lesions (Figure 12b)

Six birds received lesions of the SMT ranging in size

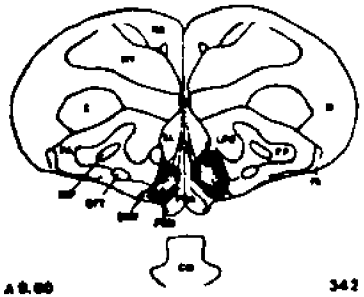
FIG. 12

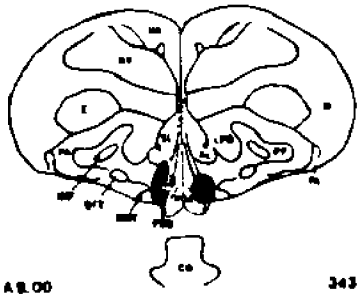
A. CONTROL LESIONS

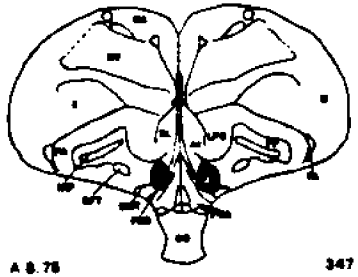




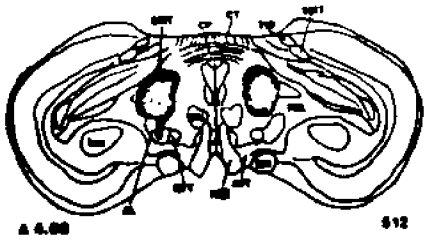
B. SMT LESIONS

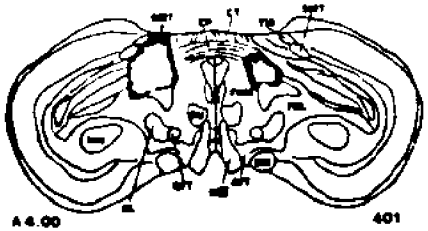


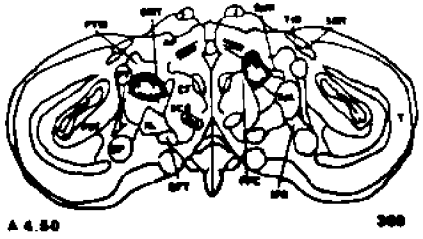




C. OMT LESIONS







from slight to extensive. In addition, all birds sustained varying degrees of damage to nearby structures, including the nucleus accumbens, the diagonal band of Broca, and the medial forebrain bundle. In three birds (342, 343, 513) the lesion extended medially, impinging upon cells within the anterior preoptic area. In four birds (335, 345, 347, 513) the lesion extended caudally as far as the anterior hypothalamus, although in Bird 513, hypothalamic damage was unilateral. In addition, the lesion in Bird 513 was placed asymmetrically and encroached upon the paraventricular nuclei on the left side. In one bird (347), the lesion extended dorsally invading cells within the lateral septal area. There was no evidence of damage to the QFT in any of the birds in this group.

C. OMT Lesions (Figure 12c)

Of the 12 birds receiving bilateral OMT lesions, four sustained bilateral QFT damage and were dropped from this analysis. All of these birds exhibited disruptions in key-pecking and feeding that were more severe than in the birds reported below.

In general, for the six birds whose data are shown in Table 12, bilateral OMT lesions were accompanied by damage to adjacent structures including the lateral and medial reticular formations. In addition, the lesion in several birds extended rostrolaterally to include cells within the spiriform and pretectal complexes on one (Birds 512 and 375) or both (Birds 429 and 368) sides. Caudally, the lesions in five of the six birds impinged upon overlying deep tectal structures

and in three birds (375, 401, 512), interrupted the posterior commissure or the tectal commissure, or both. In Bird 368, the lesion spread anteriorly as far as the posterior dorsal thalamic group, and in Bird 432, the lesion extended posteriorly as far as locus coeruleus. Only two birds (368 and 512) sustained any damage to ansa lenticularis, but in both cases it was unilateral and judged to be minor. Significantly, there was complete sparing of the QFT in all of these birds.

Two birds, 417 and 511, whose data are not included in Table 12 but are discussed below, also evidenced bilateral damage of the OMT. In Bird 417, the lesion spread medially on the left side to the lateral margin of nucleus ruber and destroyed cells within the medial reticular formation. In addition, the lesion spread ventrally, slightly damaging QFT and nucleus ectomammilaris. On the right side, damage was restricted mainly to the OMT.

The lesion in Bird 511 was more laterally placed and impinged upon cells within the spiriform and pretectal complexes in addition to the OMT. There was also moderate bilateral destruction of ansa lenticularis. On the right side the lesion spread ventrally to include the dorsal portion of the QFT. In both 417 and 511, the unilateral damage to the QFT was judged to be slight.

Behavioral Data

Table 12 summarizes the data on the effects of control, SMT and OMT lesions upon several measures of performance in the operant conditioning situation. The preoperative data reflect performance for the last five sessions immediately prior to surgery. The postoperative data are divided into three periods. Phase 1 includes the period from the first postoperative day to the resumption of key-pecking and indicates the number of days and sessions without any key-pecks. Phase 2 includes data of all sessions immediately following the resumption of key-pecking in which operant responding is below preoperative levels. Numbers within the brackets specify the number of sessions below preoperative levels. Phase 3 presents performance data for the first five sessions in which key-pecking approximates its preoperative level.

In addition to data on operant performance (mean number of key-pecks and mean reinforcements), several other measures are shown in Table 12 . Responsiveness to food was assessed by the ratio of feedometer responses : reinforcements (FDM./REINF.). This ratio was calculated by dividing the total number of feeding responses by the total number of reinforcements. Food intake per reinforcement is indicated by the FOOD/REINF. ratio, obtained by dividing total food intake (in grams) by total reinforcements. Feeding efficiency is shown in the column labelled FDM./FOOD and was calculated by dividing the total number of feeding responses by the total amount of food consumed from

TABLE 12

EFFECTS OF OMT AND OMT LESIONS UPON OPERANT CONDITIONING OF KEY PECKING RESPONSE

Bird No.	Sex	Lesion L	Lesion R	PREOPERATIVE Mean Key-Pecks	Mean Reinf.	Fdm. Reinf.	Food Fdm. Reinf.	Food Fdm. Food	POSTOPERATIVE PHASE 1 Days	PHASE 1 No. Ses.	PHASE 2 Mean Key-Pecks	PHASE 2 Mean Reinf.	PHASE 2 Fdm. Reinf.	PHASE 2 Food Fdm. Reinf.	PHASE 2 Fdm. Food	PHASE 3 Mean Key-Pecks	PHASE 3 Mean Reinf.	PHASE 3 Fdm. Reinf.	PHASE 3 Food Fdm. Reinf.	PHASE 3 Food Fdm. Food
CONTROL GROUP																				
323	M	1	0	1325	30	14	.4	32.2	--	--	--	--	--	--	--	1442	30	11	.3	34.9
514	F	1	0	1191	30	12	.6	20.0	--	--	--	--	--	--	--	1037	27	10	.6	16.6
418	F	SHAM		1441	30	12	.5	25.3	--	--	--	--	--	--	--	1576	30	11	.5	23.6
536	F	SHAM		1875	31	16	.7	24.0	--	--	--	--	--	--	--	1807	31	16	.7	23.7
SHAM GROUP																				
345	M	3	2	867	30	16	.5	29.8	--	--	--	--	--	--	--	991	30	14	.4	31.8
342	F	2	3	1243	30	17	.5	35.4	--	--	795(3)*	23	11	.4	28.0	1332	30	14	.5	30.1
343	M	2	2	1559	30	10	.3	30.0	--	--	668(2)*	16	7	.2	35.7	1707	30	8	.2	35.1
513	M	2	1	1551	30	6	.2	27.5	--	--	--	--	--	--	--	2230	29	6	.2	32.6
347	M	1	1	775	30	13	.4	32.3	--	--	572(2)*	26	6	.2	37.0	1191	30	12	.3	47.1
335	F	1	1	949	30	14	.6	24.1	6	5	472(3)*	9	11	.5	23.2	1500	30	12	.5	23.5
OMT GROUP																				
512	F	3	2	1454	30	17	.7	23.7	7	5	573(8)*	18	4	.1	57.6	1171	28	3	.1	53.1
432	F	2	2	583	30	16	.5	32.9	6	4	--	--	--	--	--	775	30	10	.2	54.1
401	M	2	1	746	28	11	.3	37.5	10	8	25(2)*	1	0	0	0	702	26	3	negl.	141.7
375	M	2	1	1214	30	11	.3	31.9	3	2	770(7)*	26	5	negl.	152.0	1353	30	5	.1	62.3
429	M	2	1	493	30	11	.4	31.0	2	2	210(3)*	19	7	negl.	262.0	579	30	6	.1	81.3
368	F	1	1	1533	30	10	.5	20.6	--	--	--	--	--	--	--	1719	31	4	.1	42.9

* Number of days below preoperative level

the magazine hopper. All these ratios reflect performance that is independent of the level of key-pecking and the number of reinforcements obtained. Thus, they permit us to assess changes in responsiveness, feeding, and consummatory efficiency irrespective of any changes in the level of operant behavior.

1. Control Group (Table 12). Control lesions and sham surgery had no significant effects upon any of the performance measures. Within two or three sessions, the behavior of these birds was indistinguishable from that seen prior to surgery. The slight decrease observed in the FDM./REINF. ratio (responsiveness) was not statistically significant, $t(6) = .97$, NS.

2. SMT Group (Table 12).

Phase 1. Following surgery only a single SMT bird displayed an absence of key-pecking. However, from the second postoperative session, Bird 335 readily ate food placed in its home cage and occasionally responded to food during probe reinforcement trials.

Phase 2. Following the resumption of key-pecking, this bird and three others in the group showed a marked, but transient reduction in the number of operant responses (Mean reduction in key-pecking: 42%; Range 36% - 58%). During this period, fewer reinforcements were received. An examination of individual cumulative records suggested that each of the birds were capable of emitting operant responses at

rates approximating preoperative levels. However, during these sessions, there was a significant change in the temporal distribution of their key-pecks characterized by long pauses in responding. Birds 343 and 347 initiated key-pecking and obtained reinforcement at the start of the session. However, both birds ceased pecking before its termination. Bird 335 showed a similar pattern of behavior, but this bird often resumed pecking near the end of the session. Bird 342 also ceased pecking prior to the end of the session, but this bird displayed a long "warm-up" period as well.

In addition to decreases in instrumental performance, each of these pigeons evidenced significant reductions in responsiveness to the food reinforcement as measured by the ratio of feedometer responses: reinforcements [Preoperative vs. Phase 2: t (6) = 2.71, $p < .05$]. However, there were no significant changes in any of the birds with respect to intake or consummatory efficiency. It should be noted that all these effects were transient (2 - 3 sessions).

Phase 3. Following their immediate postoperative decreases in operant performance, all four pigeons, in addition to two birds who never evidenced any postoperative decrement, showed increases in operant responding ranging from 7% - 59% (Mean increase = 31%) above preoperative levels. Moreover, an examination of individual records of the last five postoperative sessions indicated that these increases persisted until the termination of testing. Birds 513, 347, and 535, who showed the greatest increases in key-pecking during the

initial five sessions of Phase 3 (44%, 54%, and 59%, respectively) also showed the greatest increases at the end of testing (30%,48%, and 79%). The remaining three birds evidenced more moderate increases (Mean increase = 12%). Apart from their increased level of key-pecking, the performance of all six birds with respect to responsiveness, food intake and consummatory efficiency was indistinguishable from that seen preoperatively and from the performance of the controls.

3. OMT Group (Table 12).

Phase 1. In contrast to SMT and control lesions, damage to the OMT abolished key-pecking in five of six birds for periods ranging from 2 - 9 days (Mean = 4.4 days). In addition, there was a fairly consistent relationship between lesion size and the number of days characterized by an absence of operant responding (Spearman Rank-Order r = .81). During the first few sessions, birds made no attempt to eat from the food magazine on probe reinforcement trials during which food was made available for the standard 8-sec. reinforcement interval. Nor would they attempt to eat a supplementary ration offered in either the operant chamber or in the home cage. However, a day or two prior to the reappearance of key-pecking, each bird resumed feeding or attempting to feed in each of these situations. Observations of individual birds in the operant chamber responding to probe reinforcement trials revealed that while there were no observed difficulties in finding the hopper or getting their heads through the magazine opening, birds approached the hopper toward the end of the

reinforcement interval. In addition, pecks into the hopper were often hesitant and incomplete. Observations of feeding behavior in the home cage confirmed the existence of the consummatory difficulties.

Phase 2. Following the resumption of operant responding four OMT birds evidenced several days of reduced key-pecking (Mean reduction = 63%; Range = 37% - 97%) in addition to decreases in the number of obtained reinforcements. An examination of local response rates within the cumulative records of individual birds indicated that the birds were capable of emitting bursts of responses at rates characteristic of those seen preoperatively. However, the records also revealed changes in the temporal patterning of responding similar to those seen in SMT birds. In the first session or two, most birds displayed a lengthy warm-up period, with responding commencing near the end of the session. Over the next several days, birds gradually began pecking at the beginning of the session but evidenced frequent pausing throughout.

In addition to the changes in instrumental behavior, lesions of the OMT resulted in a significant reduction in responsiveness to the food reinforcement as well as a reduced level of intake. Moreover, OMT birds displayed a striking increase in their feedometer response : food intake ratios, suggesting a reduction in feeding efficiency similar to those seen for OMT birds in tests of consummatory efficiency in Experiment 2.

Phase 3. During Phase 3, neither the mean number of key-pecks nor the mean number of reinforcements appeared to differ significantly from their preoperative values. However, responsiveness to the grain reinforcement and the intake of grain continued to be significantly reduced below preoperative levels and below the performance of the SMT birds and the controls. While there was some improvement in consummatory efficiency in several of the birds, feeding efficiency remained considerably impaired. All of these effects persisted until the termination of testing.

Analysis of the Deficit in OMT Birds

Apart from an initial impairment in operant responding, the most obvious effects of OMT lesions were reductions both in the number and the efficiency of the pigeon's feeding response. These effects could easily account for the reduction in the pigeon's intake of food. However, in several birds, high levels of operant responding persisted although food reinforcement was drastically reduced. In other birds (401, 417, and 511), high levels of operant responding accompanied by diminished intake were followed by a reduction in key-pecking reminiscent of the effects of extinction procedures. Observations of each of these birds in the test chamber indicated that during reinforcement availability, the approach toward the magazine was delayed until the end of the reinforcement interval. Thus, decreases in key-pecking may have been an indirect consequence of the primary responsiveness and consummatory deficits.

The long-term effects of OMT lesions upon several performance measures may be seen in the data of four representative pigeons in Figures 13 to 16 . Bird 512 (Figure 13) resumed preoperative levels of key-pecking only after several weeks characterized by an absence or reduced level of responding. However, the return of preoperative performance was mediated without any significant improvements in responsiveness, intake or efficiency. Following the resumption of pecking, Birds 401 and 511 (Figures 14 and 15) showed several sessions of key-pecking in which response rates were comparable with those observed preoperatively, although in both birds feedometer responses were greatly reduced and food intake was minimal. These conditions eventually resulted in a decline in key-pecking seen in normal birds under an extinction schedule. At this point the reinforcement interval was increased from 8 to 15 seconds to compensate for the delayed reaction to the hopper during reinforcement availability. As seen in Figure 14 and 15, feedometer responses increased dramatically, but because of persistent consummatory deficits, intake remained greatly diminished. Significantly, the opportunity to feed was enough to restore and maintain operant performance at high levels. Reinstating the standard 8-sec. reinforcement interval clearly resulted in a decrement in performance. An interesting parallel may be seen in the data of Bird 417 (Figure 16). After several weeks marked by the absence of responding, Bird 417 resumed pecking and was immediately tested for several sessions using the 15-sec. reinforcement interval. As shown in Figure 16 key-pecking quickly resumed to preoperative levels while res-

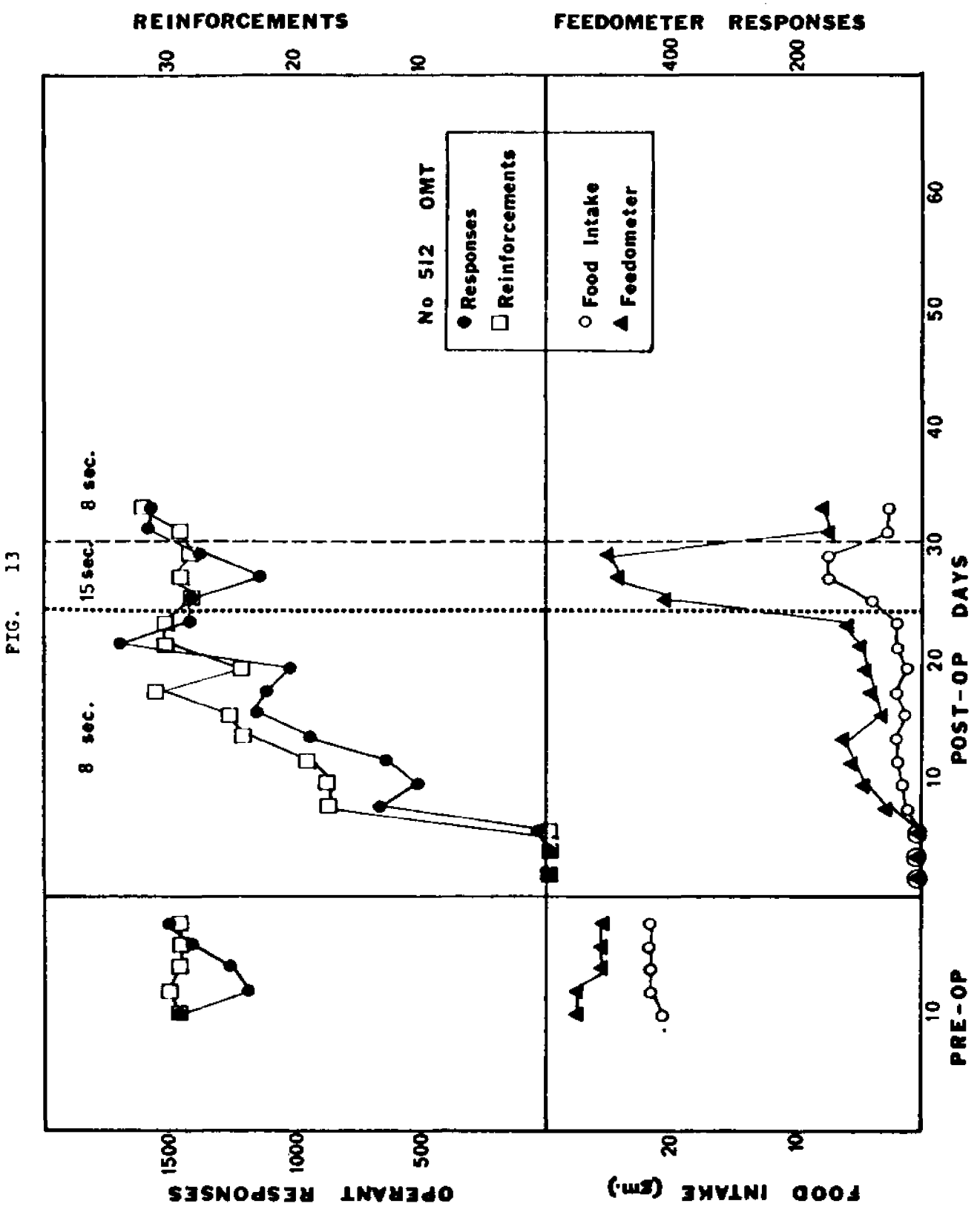
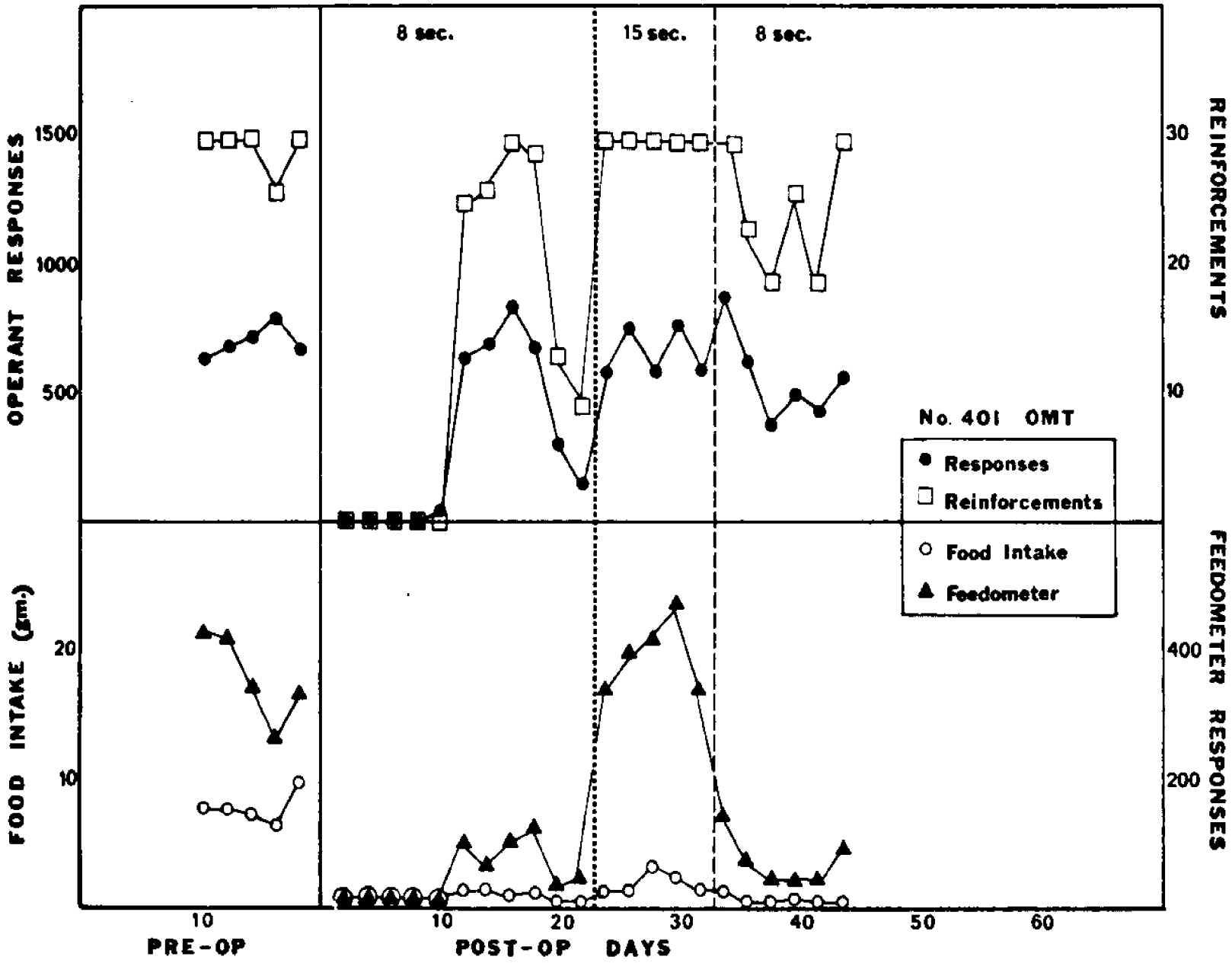

REINFORCEMENTS
FEEDOMETER RESPONSES
No 512 OMT
Responses
Reinforcements
Food Intake
Feedometer
8 sec.
15 sec.
8 sec.
PRE-OP
POST-OP DAYS
OPERANT RESPONSES
FOOD INTAKE (gm.)

FIG. 13

FIG. 14


8 sec.
15 sec.
8 sec.
OPERANT RESPONSES
1500
1000
500
REINFORCEMENTS
30
20
10
No. 401 OMT
Responses
Reinforcements
Food Intake
Feedometer
FOOD INTAKE (gm.)
20
10
FEEDOMETER RESPONSES
400
200
10
PRE-OP
10
20
30
40
50
60
POST-OP DAYS

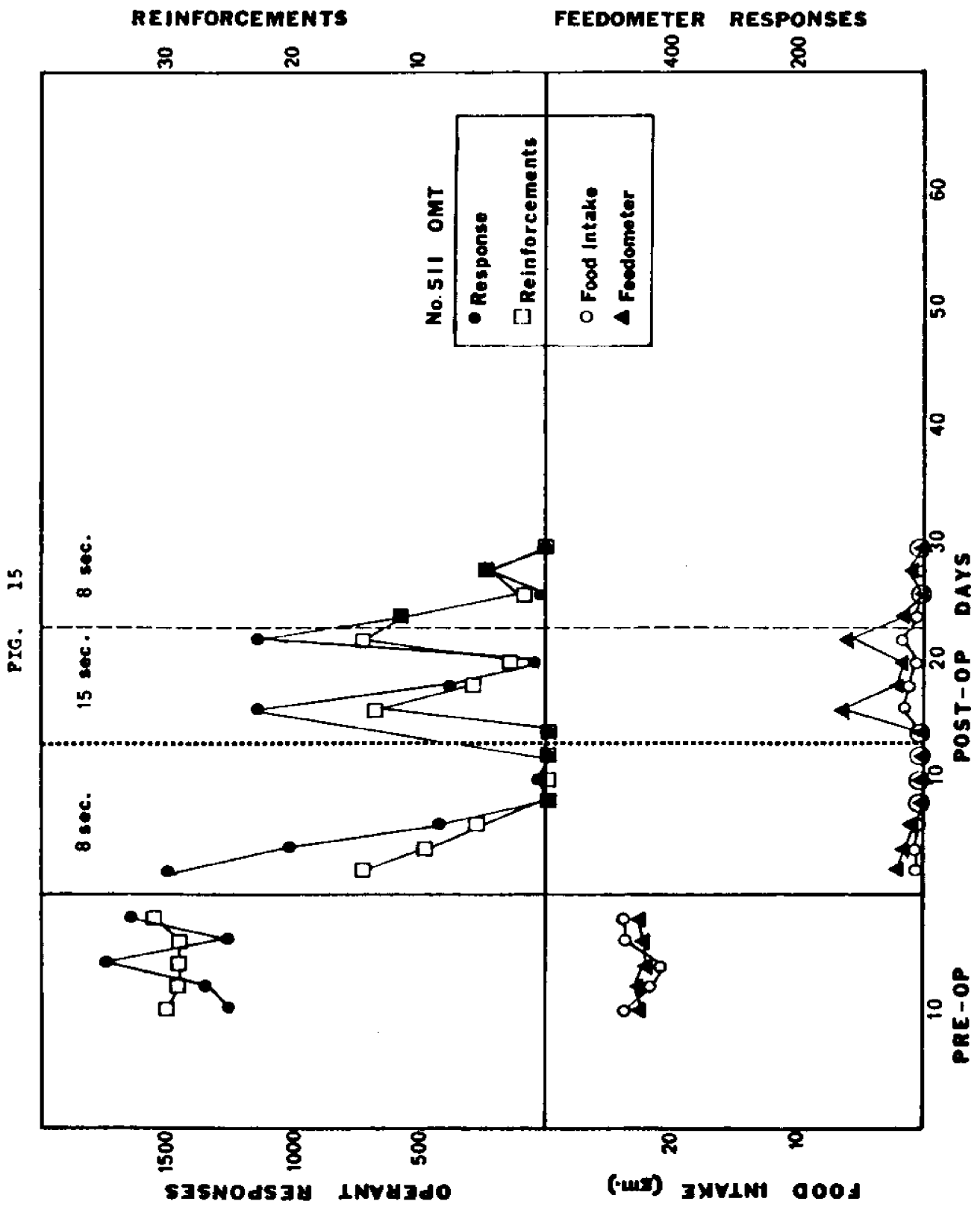
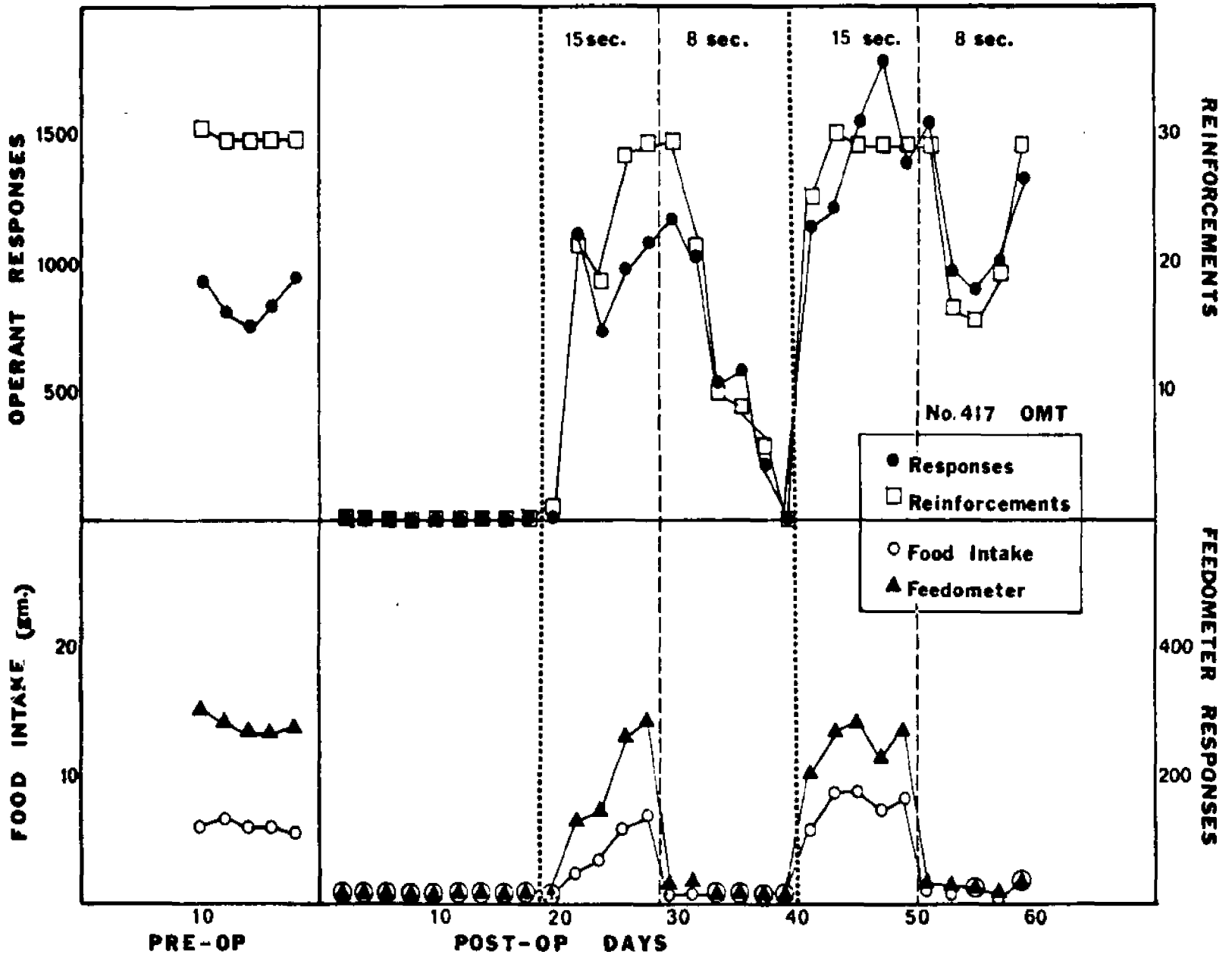

REINFORCEMENTS
FEEDOMETER RESPONSES
No. 511 OMT
Response
Reinforcements
Food Intake
Feedometer
8 sec.
15 sec.
8 sec.
PRE-OP
POST-OP DAYS
OPERANT RESPONSES
FOOD INTAKE (gm.)

FIG. 15

FIG. 16


15sec.
8 sec.
15 sec.
8 sec.
OPERANT RESPONSES
1500
1000
500
REINFORCEMENTS
30
20
10
No. 417 OMT
Responses
Reinforcements
Food Intake
Feedometer
FOOD INTAKE (gm.)
20
10
FEEDOMETER RESPONSES
400
200
10
10
20
30
40
50
60
PRE-OP
POST-OP DAYS

ponsiveness (feedometer responses), intake and consummatory efficiency improved steadily. At this point, substituting an 8-sec. reinforcement interval for the 15-sec. interval significantly reduced the number of feeding responses, subsequently resulting in decreased operant performance. However, the resumption of testing using the 15-sec. reinforcement interval once again reinstated the original level of performance. A final series of sessions with the 8-sec. interval resulted in a rather variable performance, although in the final three sessions, high levels of key-pecking were maintained despite marked reductions in intake. It should also be noted that for birds displaying high postoperative rates of key-pecking under the standard 8-sec. reinforcement interval, increasing the duration of the interval to 15-sec. had no further effects upon the rate of operant responding (see the data for Bird 512 in Figure 13).

Discussion

Damage to the SMT results in a transient change in the intra-session distribution of key-pecking. In addition, while there is no effect upon efficiency of the feeding response, there is a significant decrease in the number of feeding responses made during the reinforcement interval.

Following these transient effects upon key-pecking and feeding, SMT birds display significant increases in their rate of key-pecking which persist throughout the duration of testing. However, there is no apparent relationship between the magnitude of the increase in key-pecking and the extent of damage to the SMT. In fact, there is a strong negative correlation between these two variables (Spearman Rank-order $r = -.82$) suggesting that damage to nearby structures may mediate the observed increases in operant responding. Indeed, an examination of the relationship between the degree of incidental damage to adjacent septal structures (nucleus accumbens, diagonal band of Broca, and lateral septal nucleus) and the degree of increased key-pecking reveals a positive correlation (Spearman Rank-order $r = .71$). It should be noted that the increased rate of key-pecking was not accompanied by an increased rate of eating during the reinforcement interval.

By contrast, OMT damage is followed by a period of variable length (2 - 9 days) during which both key-pecking and feeding (whether in the operant chamber or in the home cage) always

preceeds the resumption of key-pecking. In the first few sessions following the return of key-pecking there is a significant reduction in the number of operant responses and significant reductions in feedometer responses, food intake, and consummatory efficiency. Key-pecking then resturns to preoperative levels and remains at these levels for the duration of testing, but there are no significant improvements in any of the other measures of performance.

Observations of individual OMT birds during this time reveal that OMT damage increases the latency to feed during the reinforcement interval, leading, in some cases, to extinction of the key-pecking response. These changes in "reaction time" following the onset of reinforcement availability (key light off) would account for the reduced numbers of feeding responses seen in these birds. Lengthening the reinforcement interval reinstates operant performance at preoperative levels by permitting more feeding time. However, the return to preoperative levels of key-pecking is accomplished without further improvements in consummatory efficiency so that actual food intake remains significantly reduced. Thus, despite greatly decreased amounts of food reinforcement, high rates of key-pecking persist. These data suggest that the underlying mechanisms controlling feeding efficiency, responsiveness to food and operant responding may be potentially separable.

General Discussion

The present study was designed to examine the contribution made by two extratelencephalic efferent pathways to the feeding behavior of the pigeon. The structures chosen for study, the occipitomesencephalic tract and the septemesencephalic tract, arise in the archistriatum and hyperstriatum, respectively, and convey information to brain stem and spinal cord nuclei related to the sensory systems involved in the exteroceptive control of feeding in this species. Preliminary data on the functional role of these structures suggested that they might provide a substrate for the somatomotor and visuomotor control of the consummatory response of eating. Therefore, electrolytic lesions were placed in these tracts and their effects upon several measures of ingestive behavior were examined.

While there was no evidence for its direct involvement in drinking, OMT damage was followed by a significant reduction in responsiveness to food. This reduced responsiveness was manifested in periods of aphagia and hypophagia and in a marked retardation in the recovery of lost body weight. An examination of the relation between food intake and body weight indicated that OMT birds utilized their available food as efficiently as normals and that the retardation in body weight recovery did not reflect lesion effects upon digestive or metabolic processes. Thus, the prolonged reduction in body weight appeared to be a direct consequence of the decreased responsiveness to both food and weight loss rather than a direct lesion-induced effect upon weight regulation.

Cinematographic analyses revealed that OMT damage, in addition to its effects upon food intake, produced disruptions in the consummatory response of eating. One of these deficits, impaired grasping, was strikingly evident on feeding attempts with large grains (peas) and is in agreement with Zeier's (1971) observations of grasping difficulties following anterior archistriatal lesions. In addition, these findings are consistent with previous studies reporting bill opening following electrical stimulation of either the somatomotor portion of the archistriatum or the OMT (Phillips, 1964; Phillips & Youngren, 1971; Putkonen, 1967). A second observed consummatory impairment involved inaccurate pecking characterized by an increased frequency of "overshoots". Thus, OMT damage may have also disrupted processes underlying the visuomotor control of the peck, perhaps by interrupting a critical source of telencephalic input to tectal structures involved in the pecking response. In an earlier study, Bremer, Dow and Moruzzi (1939) reported directed head movements following caudal neostriatal stimulation, and more recently Showers (1973) described integrated movements of the beak and head following stimulation of the intermediate neostriatum. Interestingly, both Zeier and Karten (1971) and Showers (1973) have described a prominent pathway from these regions to the anterior, somatomotor portion of the archistriatum, which, in turn, is the source of the OMT. While the exact nature of the information provided by these structures to the OMT awaits further analysis, these data provide a neuroanatomical

framework within which to explore this problem.

In addition to impairing feeding directly, OMT damage also abolished key-pecking behavior reinforced with food. The resumption of key-pecking was followed by a transient period during which operant responding, feeding, and consummatory efficiency were significantly reduced. Preoperative levels of key-pecking eventually returned, but there were no subsequent improvements in any other behavioral measure. Observations of individual birds within the operant chamber revealed increased feeding latencies during the reinforcement interval, which, in several cases, led to an extinction of the key-pecking response. Lengthening the reinforcement interval, permitting more feeding time, reinstated operant performance at preoperative levels. However, in the absence of significant improvements in consummatory efficiency, food intake remained considerably reduced.

These data indicate the nature of the deficits produced by damage to the OMT. While these deficits could be experimentally dissociated into "consummatory" (neuromotor) or "motivational" (responsiveness) impairments, OMT damage invariably produced both.

Lesions of the SMT also produce persistent reductions in food intake without directly affecting water intake. Lacking the compensatory overeating characteristic of food-deprived normal pigeons, body weight in SMT pigeons also remained reduced. As in the case of OMT birds, there were no changes in the relation between food intake and body weight in SMT

birds, suggesting that these birds regulated their body weight in a manner comparable to normal pigeons maintained on reduced rations. However, unlike OMT birds, the reduced levels of food intake among SMT pigeons were not correlated with decreases in responsiveness to food. Furthermore, there were no observable changes among SMT birds in the efficiency or accuracy of their eating response and only transient reductions in operant responding. The lack of such behavioral changes makes their feeding behavior deficit rather difficult to characterize.

SMT birds displayed inappropriate "sorting" behavior in the presence of large amounts of milo. Such "sorting" produced increased numbers of feedometer responses without concommitant increases in food intake. Such findings have previously been interpreted as reflecting a lesion-produced consummatory inefficiency but such an interpretation is clearly inappropriate in the case of SMT birds. Our findings suggest the need for caution in using feedometer responses as an index of responsiveness. Futhermore, they call forth Miller's (1967) admonition that neurobehavioral studies of hunger should invoke an analysis of treatment effects upon a variety of behavioral measures.

While there have been no previous behavioral studies on the functional role of the SMT, there have been several studies dealing with behavioral changes following destruction of various subdivisions of the hyperstriatum (reviewed by Macphail, 1975), the source of input to the SMT. Most of

these studies have employed a discrimination learning paradigm, utilizing food-deprived birds in an operant conditioning situation. Unfortunately, these studies shed little light on the nature of the feeding deficits produced by lesions of the SMT in pigeons tested under ad libitum conditions. Cohen (1967), however, has reported incidental observations on reductions in ad libitum food intake without consummatory impairments following hyperstriatal damage, a finding which is consistent with the results of the present study.

The neurobehavioral mechansim(s) mediating the reduced intake of SMT birds remains obscure. However, on the basis of the present study, we may tentatively conclude that damage to the SMT affects the pigeon's responsiveness to food in a manner suggesting a change in its visually controlled preference behavior.

Relationship Between Feeding Deficits Produced by OMT Damage and Damage to Central Trigeminal ("Quinto-frontal") Structures

Previous lesion studies in the pigeon have focused primarily on afferent pathways and nuclei involved in the control of feeding. The deficits produced by lesions to one such group of structures, the "quinto-frontal" system, has been analyzed in detail by Zeigler and Karten (1973a, b). These structures appear to be among those responsible for the aphagia in the decerebrate bird. Considering the anatomical relationship between quinto-frontal structures (PrV, QFT, NB) and the OMT (see Introduction), we might anticipate similarities in the feeding behavior deficits produced by lesions to either set of structures.

In both qunito-frontal and OMT birds there is an immediate postoperative period of aphagia characterized by a marked reduction or absence of feeding responses. This is followed by a period of hypophagia (anorexia) during which intake is reduced from its preoperative level. During this time, birds in the two groups are significantly less responsive to both food and body weight loss. Although given unlimited access to grain, they do not display the compensatory overeating typical of normal birds who have sustained comparable weight losses. Furthermore, the reduction of food intake is prolonged, so that the recovery of body weight is either absent or considerably delayed. However, the quantitative relations between food intake and body weight in these hypophagic birds and the relative stability with which their reduced weights

are maintained suggests that they are capable of regulating their weight, but at a new, lower level.

In addition to these effects upon food intake, damage to the OMT and to quinto-frontal structures produces a consummatory deficit which is reflected by an increase in the number of feeding responses required to obtain a unit quantity of grain. Analyses of electrophysiological (Zeigler & Witkovsky, 1968; Witkovsky, Zeigler & Silver, 1973) and cinematographic (Zeigler & Karten, 1973) data suggest that the reduced feeding efficiency seen in quinto-frontal birds results from a disruption of tactile and proprioceptive information normally involved in the control of mandibulation -- one of the movement patterns constituting the consummatory response of feeding in the pigeon. By contrast, OMT damage results in reduced consummatory efficiency by disrupting both somatomotor (grasping) and visuomotor ("overshooting") processes involved in the control of eating. Interestingly, with respect to consummatory efficiency, there is an inverse relationship between lesion locus and grain size: trigeminal birds show less consummatory impairment with large grain (Zeigler, Miller & Levine, 1975) while OMT birds perform better with small grain.

Quinto-frontal damage abolishes food-reinforced key-pecking on a VI-60 sec. schedule for varying periods depending upon the bilateral extent of the lesion (Zeigler & Karten, 1973b). During this time, birds do not make pecking responses into the food hopper on probe reinforcement trials

when the hopper is presented automatically for the standard reinforcement interval. In all cases, the reappearance of both key-pecking and feeding occurs both simultaneously and abruptly. However, lesioned birds displayed significant consummatory deficits resulting in a marked reduction of food intake.

The effects of OMT lesions upon operant responding are similar to those seen after quinto-frontal damage. Following OMT destruction, both key-pecking and feeding are abolished for varying periods depending upon the bilateral extent of the lesion. In all cases, the resumption of feeding always precedes the resumption of key-pecking. However, the return to preoperative levels of key-pecking are accompanied by delays in responding during reinforcement presentation, resulting in significant reductions in both feeding responses and food intake. In addition, consummatory efficiency is disrupted in a manner similar to that seen in freely feeding OMT birds. In several birds, these deficits lead to an extinction of key-pecking, which may be reinstated by lengthening the reinforcement interval to provide more feeding time. However, the resumption of key-pecking is not accompanied by further improvements in feeding efficiency so that actual food intake remains significantly reduced. As noted previously, these data suggest that the underlying mechanisms controlling feeding efficiency, responsiveness to food and operant responding may be potentially separable.

The reduced responsiveness to food observed in OMT birds

suggests that destruction of this pathway disrupts processes underlying the "motivational" control of feeding. However, in view of the homology between the OMT and a component of the mammalian pyramidal system (bundle of Bagley), we must distinguish between process specific to feeding from generalized deficits in somatomotor control. Thus, one could argue that interrupting the OMT might conceivably disrupt feeding by eliminating critical sources of input to the motor nuclei or to the effectors controlling the consummatory response of pecking. According to this view, the reduced feeding in lesioned birds might represent reduced phasic muscular activity from processes akin to muscular fatigue, caused perhaps by impaired muscular activity. Several lines of evidence bearing directly on this problem render this interpretation unlikely.

First, there were no indications in lesioned birds of any kind of motor abnormality. Second, assuming the existence of muscular fatigue as a causal factor controlling responsiveness to food, it is difficult to reconcile the specificity of its effectiveness upon eating to the exclusion of drinking, in which there was no evidence of a lesion-induced reduction in responsiveness. Both consummatory acts presumably share much of the same peripheral effector apparatus. In addition, behaviors other than feeding requiring frequent use of the beak, such as feather preening and operant responding return to preoperative levels following surgery. Finally, in short term tests of eating (Experiment 2), lesioned birds deprived to 80% of their ad libitum body weight increase their output of feeding responses beyond preoperative levels, an

observation that is inconsistent with the hypothesis that decreased responsiveness is due to muscular fatigue.

It is clear from all of these studies that damage to quinto-frontal structures and the OMT disrupt the consummatory response of eating, reduce responsiveness to food and impair food-reinforced instrumental behavior. Although the different types of behavior may be experimentally dissociated, they appear to be mediated by common neural substrates. As Zeigler (1976) has noted, the similarities in the deficits produced by lesions of different structures may be accounted for by the fact that all these structures are anatomically and functionally connected. Thus, deficits observed following destruction of one of the structures may reflect both direct damage to that structure and indirect effects upon other structures of this putative neural "feeding system".

Relationship to the Mammalian Pyramidal System

Based on their telencephalic origin and pattern of termination within the brainstem and spinal cord, Karten, (Karten & Dubbeldam, 1973; Cohen & Karten, 1974) has argued that the OMT and SMT may be homologous to components of the mammalian pyramidal system. However, it has become increasingly evident from recent morphological and electrophysiological work that the components of the mammalian pyramidal system may be neither anatomically nor functionally homogeneous (Wiesendanger, 1973). Neurobehavioral studies exploiting the existing anatomical separation characterizing the avian "pyramidal system" could provide potential insight into the

functional roles of the various mammalian pyramidal subdivisions. Thus, a comparison of the deficits produced by OMT or SMT damage with the effects of damage to the mammalian pyramidal system could prove to be of considerable heuristic value.

On the basis of his thorough review of the literature, Towe (1973) has noted that the presumed function of the mammalian pyramidal system "has become thoroughly entangled with ideas about anatomical organization" (p. 71) and that "textbook accounts of pyramidal tract section have been exaggerated out of all proportion to their magnitude" (p. 71). On the basis of his review, he concludes that the so-called "paralysis of voluntary movement", which for years was believed to characterize the pyramidotomized animal, results typically from lesions which include both pyramidal and adjacent lemniscal structures. Damage restricted solely to the pyramidal tract fails to produce such impairments. Furthermore, the morphology and connections of the tract appear to be species-specific, so that damage to the tract might be expected to produce corresponding species-specific behavioral deficits (Towe, 1973).

In more recent studies, damage has been restricted to the pyramids by lesions placed at the base of the medulla, for it is at this locus that the tract is most accessible in mammalian species. Pyramidotomized monkeys typically show no impairment in either initiating or sustaining directed movement; however, finely graded movements, such as those

involving the forelimb digits, may no longer be carried out smoothly (Lawrence & Kuypers, 1968). Wiesendanger (Hepp-Reymond & Wiesendanger, 1972), has also shown that unilateral pyramidal tract lesions in the monkey affect the latency with which a conditioned gripping response may be performed. Furthermore, Laursen (1972) has noted that, in the monkey, unilateral pyramidal tract section reduces response rate and increases the duration of inter-reinforcement pausing in a conditioned level-pressing response. None of the animals in any of these studies showed signs of muscular weakness or fatigue. Hepp-Reymond and Wiesendanger (1972) analyzed electromyograms of the affected musculature in these animals and suggested that the impaired performance was due to a delay in the build-up of muscular force necessary to reach the threshold of movement. Thus, appropriate conditioned movements were performed more slowly because of a delay in the execution of the response, rather than a delay in its initiation.

In cats, pyramidal section at the bulbar level has little effect upon normal activity (Laursen & Wiesendanger, 1967; Edwards & Flynn, 1972). In addition, Laursen and Wiesendanger observed no impairment in either the performance or in the rate of an instrumentally conditioned lever pressing response for food reinforcement. However, these authors reported a significant reduction in response rate ("reaction time") when operated cats had to make a visual brightness discrimination which required them to depress a lever situated below the brighter of two visual stimuli. Interestingly, Bucy (Bucy,

Ladpli & Ehrlich, 1966) reported a significant deficit of rapid visuomotor reactions (catching food from a turntable) following unilateral or bilateral pedunculotomy in monkeys. Wiesendanger (1969) has concluded from these data that the pyramidal tract may serve as a substrate for rapid reactions to external signals rather than for the performance of the movement as such.

A similar interpretation could be offered for the data of Edwards and Flynn (1972) who observed the "striking" behavior of cats. Following unilateral pyramidal tract damage, the locomotory behavior involved in "stalking" prey (a moving rat) remained completely intact. However, electrically induced striking behavior was impaired on the side contralateral to the stimulation. This impairment was manifested by an increased latency to strike and by a disruption of the spatial characteristics of the response. Similarly, Castro (1972) reported that rats with unilateral pyramidal tract lesions suffered an impairment in the "grasping" response when presented a (stationary) small pellet of lab chow. While his rats showed no obvious impairment in digit flexion, they frequently dropped the pellet or missed the pellet when using the affected forepaw. Significantly, these misses were due to impairments in the orientation of the grasping response that were characterized by slight over- and underextensions of the paw, and occasionally, by an incomplete extension of the forelimb at a point above the location of the stationary pellet.

All these studies involving various mammalian species have

been concerned primarily with the neuromotor control of the distal musculature, and more specifically with the hand or forepaw. It is important to recognize that, in avian species, the beak subserves many of the functions of the mammalian forepaw, including those related to feeding, grooming (preening), "signalling", and fighting, to name a few. Thus, it is striking that many of the deficits characteristic of the mammalian pyramidal preparation is also observed in pigeons with damage to the OMT. These observations provide some behavioral confirmation for Karten's proposed homology between these two structures. Furthermore, on the basis of these findings we might speculate that lesions restricted to the mammalian equivalent of the OMT (the bundle of Bagley in the ungulate brain) may produce many of the deficits observed following bulbar pyramidotomy.

The lack of observable neuromotor deficits following lesions of the SMT was somewhat unexpected in view of the "awkwardness" of head movements reported by Cohen (1967) and the "clumsiness"of movement reported by Tuge and Shima (1959) following hyperstriatal damage in the pigeon. However, in both these studies, the lesions were quite large, and included hyperstriatal areas other than those of SMT origin. A recent study by Sprague (Palmer, Rosenquist & Sprague, 1972) may also provide an alternative interpretation. Damage to a corticotectal pathway in the cat, originating in the cat's visual "striate" cortex, severely disrupted tracking of moving objects. According to the anatomical work of Karten (Karten,

Hodos, Nauta & Revzin, 1973), the posterior Wulst in the pigeon, a major source of efferent fibers within the SMT, may be homologous with area 17 in the mammalian brain. Considering that the grain eaten by the pigeon is typically stationary, our feeding tests may not have permitted the manifestation of a "tracking" deficit analagous to that seen in the cat.

Moreover, it should be recalled that SMT consists of two components: a basal branch distributing primarily to brainstem structures, and a dorsal branch which synapses on structures in the ascending visual pathways. What appear to be changes in the "valence" of certain seeds, may reflect damage to the latter component. In anv case, the present findings suggest further hypotheses for neurobehavioral studies.

Bibliography

Adamo, N. J. Connections of efferent fibers from hyperstriatal areas in chicken, raven and African Lovebird. Journal of Comparative Neurology, 1967, 131, 337-356.

Akerman, B., Fabricius, E., Larsson, B., & Steen, L. Observations on pigeons with prethalamic radiolesions in the nervous pathways from the telencephalon. Acta Physiologica Scandinavica, 1962, 56, 286-298.

Bolles, R.C. Theory of Motivation (2nd Ed.). New York: Harper & Row, 1975.

Boyko, V. P., & Bures, J. Electrophysiological correlates of pecking. Physiologia Bohemoslovaia, 1975, 24, 117-125.

Bremer, F., Dow, F. S., & Moruzzi, G. Physiological analysis of the general cortex in reptiles and birds. Journal of Neurophysiology, 1939, 2, 473-487.

Brown, J. L. Behavior elicited by electrical stimulation of the brain of the Stellar's jay. Condor, 1973, 75, 1-16.

Brunelli, M., Magni, F., Moruzzi, G., & Musumeci, D. Brain stem influences on waking and sleep behaviors in the pigeon. Archives italiennes de Biologie, 1972, 110, 285, 321.

Bucy, P.C., Ladpli, R., & Ehrlich, A. Destruction of the pryamidal tract in the monkey. The effects of bilateral section of the cerebral peduncles. Journal of Neurosurgery, 1966, 25, 1-23.

Castro, A. J. Motor performance in rats. The effects of pyramidal tract section. Brain Research, 1972, 44, 313-323.

Cohen, D.H. The hyperstriatal region of the avian forebrain: A lesion study of possible functions, including its role in cardiac and respiratory conditioning. Journal of Comparative Neurology, 1967, 131, 559-570.

Cohen, D. H., & Pitts, L.H. The hyperstriatal region of the avian forebrain: Somatic and autonomic responses to electrical stimulation. Journal of Comparative Neurology, 1967, 131, 323-336.

Cohen, D. H. & Karten, H.J. The structural organization of the avian brain: An overview. In: I.J. Goodman & M. W. Schein (Eds.), Birds, Brain and Behavior. New York: Academic Press, 1974.

Delius, J.D. Foraging behavior patterns of Herring Gulls elicited by forebrain stimulation. Experientia, 1971, 27, 1287-1289.

Edinger, L. Vorlesungen über den Bau der Nervosen Centralorgane des Menschen und der Thiere. Leipzig: F.C.W. Vogel, 1908.

Edinger, L. The relations of comparative anatomy to comparative psychology. Journal of Comparative Neurology, 1908, 18, 437-457.

Edwards, S. B., & Flynn, J.P. Corticospinal control of striking in centrally elicited attack behavior. Brain Research, 1972, 41, 51-65.

Ferrier, D. The functions of the brain (2nd Ed.). New York: G.P. Putnam's Sons, 1886

Flourens, P. Recherches expérimentales sur les propriétés et les fonctions du système nervaux dans les animaux

vertebres. Paris: Balliere, 1824.

Gentle, M. J. Diencephalic stimulation and mouth movements in the chicken. British Poultry Science, 1973, 14, 167-171.

Haarsten, A. B., & Verhaart, W. J. C. Cortical projections to brainstem and spinal cord in the goat by way of pyramidal tract and bundle of Bagley. Journal of Comparative Neurology, 1967, 129, 189-202.

Hepp-Reymond, M-C., & Wiesendanger, M. Unilateral pyramidotomy in monkeys: Effect on force and speed of a precision grip. Brain Research, 1972, 36, 117-131.

Karten, H. J. Efferent projections of the Wulst of the owl. Anatomical Record, 1971, 169, 353.

Karten, H. J., & Dubbeldam, J. L. The organization and projections of the paleostriatal complex in the pigeon (Columba livia). Journal of Comparative Neurology, 1973 148, 61-90.

Karten, H. J., & Hodos, W. A stereotaxic atlas of the brain of the pigeon (Columba livia). Baltimore: Johns Hopkins Press, 1967.

Karten, H. J., Hodos, W., Nauta, W. J. H., & Revzin, A. M. Neural connections of the "visual Wulst" of the avian telencephalon. Experimental studies in the pigeon (Columba livia) and owl (Speotyto cunicularia). Journal of Comparative Neurology, 1973, 150, 253-278.

Koike, T., & Lepkovsky, S. Hypothalamic lesions producing polyuria in chickens. General and Comparative Endocrinology, 1967, 8, 397-402.

Kuenzel, W. J., & Helms, C. W. Hyperphagia, polydipsia, and other effects of hypothalamic lesions in the white-throated spearrow, Zonotrichia albicollos. Condor, 1970, 72, 66-75.

Laursen, A. M. Static and phasic muscle activity of monkeys with pyramidal lesions. Brain Research, 1972, 40, 125-126.

Laursen, A. M. & Wiesendanger, M. The effects of pyramidal lesions on response latency in cats. Brain Research, 1967, 5, 207-220.

Lawrence, D. G. & Kuypers, H. G. J. M. The functional organization of the motor system in the monkey. I. The effects of bilateral pyramidal lesions. Brain, 1968, 91, 1-14.

Macphail, E. M. Arousal and orientation functions of the avian telencephalon. In: P. Wright, P. G. Caryl, & D. M. Vowles (Eds.), Neural and endocrine aspects of behavior in birds. Amsterdam: Elsevier, 1975.

Megibow, M., & Zeigler, H. P. Readiness to eat in the pigeon. Psychonomic Science, 1968, 12, 17-18.

Miller, M. G. Trigeminal deafferentation and feeding behavior patterns in the pigeon (Columba livia). Unpublished doctoral dissertation, City University of New York, 1974.

Miller, N. E. Behavioral and physiological techniques. In: C.E. Code (Ed.), The Handbook of Physiology, Sect. 6: The Alimentary Canal, Vol. 1: Control of food and water intake. Wash., D. C.: American Physiology Society, 1967.

Rolando, L. Saggio sulla la vera struttura del cerevello e sopra la funzioni del sistema nervosa. Sassari, 1809.

Shaklee, A. O. The relative heights of the eating and drinking archs in the pigeon's brain, and brain evolution. American Journal of Physiology, 1921, 55, 65-83.

Showers, M. J. C. The interrelations of the striatum with subcortical areas through the lateral forebrain bundle birds. Journal für Hirnforschung, 1973, 13, 427-436.

Stingelin, W. Grossenunterscheide des sensiblen Trigeminuskern bein verscheidenen Vogeln. Revue Suisse Zoologie, 1961, 68, 247-251.

Towe, A. L. Motorcortex and the pyramidal system. In: J. D. Maser (Ed.), Efferent organization and the integration of behavior. New York: Academic Press, 1973.

Tuge, H., & Shima, I. Defensive conditioned reflex after destruction of the forebrain in pigeons. Journal of Comparative Neurology, 1959, 111, 427-446.

Wallenberg, A. Der Ursprung des Tractus isthmo-striatus (oder bulbostriatus) der Taube. Neurologisches Zentralblatt, 1903, 22, 98-101.

Wiesendanger, M. The pyramidal tract. Recent investigations on its morphology and function. Ergebnisse der Physiologie, Biologischen Chemie und experimentellen Pharmakologie, 1969, 61, 72-136.

Wiesendanger, M. Some aspects of pyramidal tract functions in primates. In: J. E. Desmedt (Ed.), New Developments in Electromyography and Clinical Neurophysiology, (Vol. 3), Basel: S. Karger, 1973.

Moon, R. D. Food preferences in the pigeon (Columba livia). Unpublished doctoral dissertation, City University of New York, 1975.

Palmer, L. A., Rosenquist, A. C., & Sprague, J. M. Cortico-tectal systems in the cat: Their structure and function. In: T. Frigyesi, E. Rinvik, & M. D. Yahr (Eds.), Cortcothalamic Projections and Sensorimotor Activities, New York: Raven Press, 1972.

Phillips, R. E. Wildness in the Mallard duck: Effects of brain lesions and stimulation on "escape behavior" and reproduction. Journal of Comparative Neurology, 1964, 116, 139-155.

Phillips, R.E., & Youngren, O. M. Brain stimulation and species-typical behavior: Activities evoked by electrical stimulation of the brains of chickens (Gallus gallus). Animal Behaviour, 1971, 19, 757-779.

Putkonen, P. T. S. Electrical stimulation of the avian brain. Annales Academiae Scientiarum Fennicae, 1964 (Series A, V. Medica, No. 130).

Ralph, C.L. Polydipsia in the hen following lesions in the supra-optic hypothalamus. American Journal of Physiology, 1959, 198, 528-530.

Rogers, F.T. Studies of the brainstem VI. An experimental study of the corpus striatum of the pigeon as related to various instinctive types of behavior. Journal of Comparative Neurology, 1922, 35, 21-60.

Witkovsky, P., Zeigler, H. P., & Silver, R. The nucleus basalis of the pigeon: A single-unit analysis. Journal of Comparative Neurology, 1973, 147, 119-128.

Woodburne, R. T. A phylogenetic consideration of primary and secondary centers and connections of the trigeminal complex in a series of vertebrates. Journal of Comparative Neurology, 1936, 65, 403-501.

Zeier, H. Archistriatal lesions and response inhibition in the pigeon. Brain Research, 1971, 31, 327-339.

Zeier, H., & Karten, H. J. The archistriatum of the pigeon: Organization of afferent and efferent connections. Brain Research, 1971, 31, 313-326.

Zeigler, H. P. Feeding behavior in the pigeon: A neurobehavioral analysis. In: I. Goodman, & M. Schein (Eds.), Birds, Brain and Behavior. New York: Academic Press, 1974.

Zeigler, H. P. Dissociation of operant and consummatory responses by trigeminal deafferentation in the pigeon. Physiology and Behavior, 1975, 14, 871-874.

Zeigler, H. P. Feeding behavior of the pigeon. In: J. S. Rosenblatt, R. A. Hinde, E. S. Shaw, & C. Beer (Eds.), Advances in the Study of Behavior (Vol. 7), New York: Academic Press, 1976.

Zeigler, H. P., & Feldstein, S. A feedometer for the pigeon. Journal of the Experimental Analysis of Behavior, 1971, 16, 181-187.

Zeigler, H. P., Green, H. L., & Siegel, J. Food and water intake and weight regulation in the pigeon. Physiology and Behavior, 1972, 8, 127-134.

Zeigler, H. P., & Karten, H. J. Brain mechanisms and feeding behavior in the pigeon (Columba livia). I. Quinto-Frontal structures. Journal of Comparative Neurology, 1973, 152, 59-82.

Zeigler, H. P., & Karten, H. J. Brain mechanisms and feeding behavior in the pigeon (Columba livia). II. Analysis of feeding behavior deficits after lesions of Quinto-Frontal structures. Journal of Comparative Neurology, 1973, 152, 83-102.

Zeigler, H. P., Miller, M., & Levine, R.R. Trigeminal nerve and eating in the pigeon (Columba livia): Neurosensory control of the consummatory response. Journal of Comparative and Physiological Psychology, 1975, 89, 845-858.

Zeigler, H. P., Silver, R., & Karten, H. J. Archistriate lesions and feeding behavior in the pigeon. Paper delivered at meeting of Eastern Psychological Association, Philadelphia, April, 1969.

Zeigler, H.P., & Witkovsky, P. The main sensory trigeminal nucleus in the pigeon: A single-unit analysis. Journal of Comparative Neurology, 1968, 134, 255-264.

Zweers, G., & Wouterlood, F. Functional anatomy of the feeding apparatus of the mallard (Anas platyrhynchos L.). Paper presented at the Third European Anatomical Congress, Manchester, England, 1973.