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Physiological and Nutritional Consequences of Brain Lesions: A Functional Atlas of the Chicken Hypothalamus

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An atlas of the chicken hypothalamus was prepared and used for stereotaxic placements of electrolytic lesions. Histological brain sections, showing the location of lesions in the hypothalamus, were prepared, and the locations of the lesions were correlated with physiological and behavioral responses. Photomicrographs of some of the electrolytic lesions that did not evoke physiological or behavioral changes are also presented.

Variations in the head of the chicken resulted in lack of correlation between stereotaxic coordinates and anticipated location of the lesion. Correlation was good, however, between the location of the lesion and physiological response. Multiple physiological responses evoked by some lesions located in approximately the same brain areas in different chickens are described. Occasionally, months after lesioning, disturbed physiological functions were restored to normal, or physiological functions appeared newly impaired.

Results suggest that correlations between lesions (as shown by histological sections) and physiological aberrations cannot be made with confidence for two to four weeks after placement of lesions, possibly because of inflammation, edema, and other reversible brain damage.

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Physiological and Nutritional Consequences of Brain Lesions: A Functional Atlas of the Chicken Hypothalamus[1, 2]

INTRODUCTION

STUDIES ON THE ROLE of the nervous system in the nutrition of the chicken have been underway in the Department of Poultry Husbandry for the past twenty years and have led to an increasing awareness of the extent to which nutrition is regulated by brain mechanisms. In the chicken, as in other vertebrates, the hypothalamus is evidently the site of many of the effector functions related to the regulation of food intake. In the hypothalamus, information on food in the external environment is integrated with information on bodily needs, and this often leads the chicken to choose food most suitable for its survival. (Pearl and Fairchild, 1921; Dove, 1935).

In planning to study nutritional processes by production of electrolytic lesions or by electrical and chemical stimulation in the hypothalamus, it was clear that use of the stereotaxic instrument would require a knowledge of chicken brain anatomy that would be useful in developing guideposts to specific locations in the brain.

Anatomy of the chicken brain has been described in varying detail by a number of investigators (Ariens Kappers, Huber, and Crosby, 1960; Crosby and Woodburne, 1940; Huber and Crosby, 1929; Kuhlenbeck, 1936, 1937, 1939; Rendahl, 1924), but knowledge of the functional significance of the brainstem has been scanty. Neuroanatomists have had difficulty in relating comparative homologies between the avian brain and that of other animals (Ariens Kappers, Huber, and Crosby, 1960). Van Tienhoven and Juhasz (1962) have published a schematic atlas of the chicken brain.

A comprehensive description of the chicken brain has been prepared posthumously from the material of Jungherr (1969).

In our studies on the role of the hypothalamus in nutritional processes, we deemed it advisable to begin mapping the hypothalamus and to produce a working atlas which could serve as a guide for this work.

It has been possible with such a functional atlas to correlate the location of lesions with specific, physiological

[1] Submitted for publication July 31, 1970.

[2] This investigation was supported in part by USPHS research grants A-1804 and NB-7585 from the National Institutes of Health, and GB-242 and GB-6578 from the National Science Foundation.

changes such as food and water intake, temperature regulation, reproductive mechanisms and behavioral changes. This communication describes a functional atlas of the diencephalon of the chicken and its use in the production of altered physiological states by production of lesions in the hypothalamus.

MATERIALS AND METHODS

Production and development of atlas of diencephalon

A series of photographs, drawings, and charts has been prepared and organized as a guide to major landmarks in the hypothalamus. Many cell groups adjacent to the hypothalamus are illustrated; not all are identified. These cell groups have served as guides to the placement of stereotaxic lesions in the forebrain.

Stereotaxic instruments used were the Horsley-Clark[3] and a modified Krieg-Johnson. Stability and fixation of the head at first depended on the use of firm earplugs extending from horizontal earbars. Later, an instrument[4] was developed in which head stability was achieved by fitting the malar bone and orbital ridge into grooves in a rigid plate (figs. 1 and 2). Rotation of the plate and head permits attaining a horizontal position of the skull surface. This is an easily reproducible position and has the advantage of making the anterior commissure-posterior commissure line nearly horizontal.

To obtain relative brain dimensions, three 1,200-gm White Leghorn chickens were anesthetized with pentobarbital. A horizontal marker wire running in an anterior-posterior direction at a measured distance from the level of earbars, and a vertical wire, at a measured distance from the midline, were placed in each chicken head. The heads were fixed with the wires in place in 10 per cent formalin.

Thionine-stained sections were made. From photographs of celloidin transverse sections 50μ thick, diagrams have been prepared indicating the principal landmarks of the forebrain on a coordinate system. Wire positions were used to determine actual dimensions of the brain.

In this atlas, the primary zero of three coordinate planes passes through the interaural line. The horizontal plane is parallel to the superior surface of the skull. The vertical zero plane is perpendicular to both the horizontal zero plane and the superior surface of the skull. The lateral zero (the midsagittal plane) lies at right angles to both horizontal and vertical planes and divides the brain into symmetrical halves.

Placement of lesions

Electrolytic lesions were placed in various sites in the diencephalon of many chickens, male and female, weighing from 1,200 to 1,500 gm. Sodium pentobarbital was injected into the wing vein for anesthesia. The head was secured firmly in the stereotaxic instrument, and the earbar zero was noted. The skull was opened with a dental drill and the midline of the brain was determined by the superior sagittal sinus. The center line of the instrument was adjusted to the dural groove. Insulated[5] steel electrodes with bared beveled points (0.2–0.3 mm) were inserted into the brain, and anodal direct current of 2 to 4 ma was applied for 10

[3] Borrowed from San Francisco Medical Center, University of California through the courtesy of Dr. John Saunders.

[4] Prepared by Mr. Karl E. Smith.

[5] No. 12 beading needles were insulated with two or three coats of Insl-X #E-33 (Insl-X Co., Inc., Ossining, N.Y.).

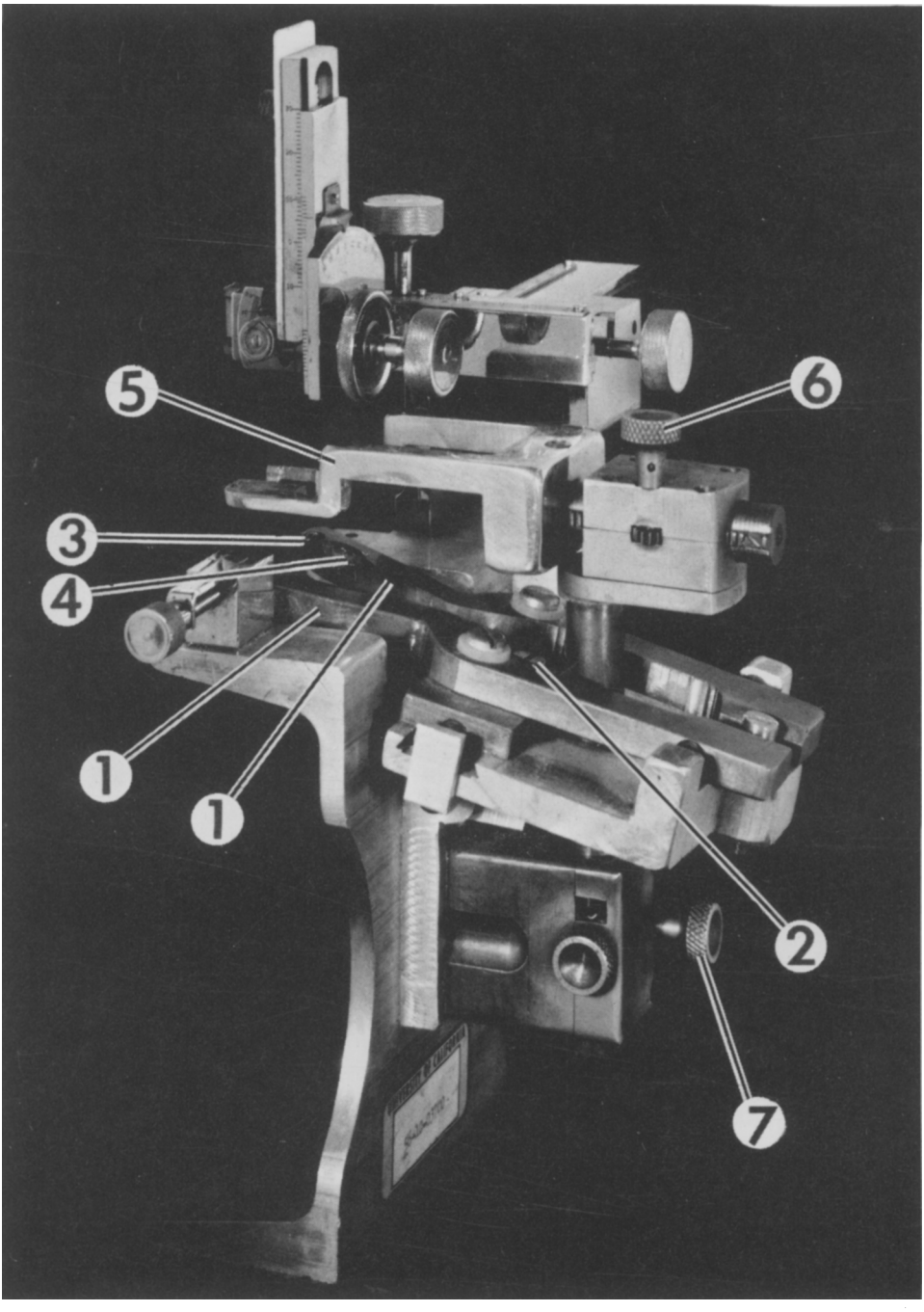


Fig. 1. Modified Stereotaxic Instrument. Horizontal position of right and left parts of rigid plate of headholder (1) can be adjusted by a screw which is not visible in this picture. Distance between right and left parts of plate can be changed by rotating visible screw (2). Malar bones and orbital ridges of chicken fit into grooves in plate (3 and 4). Retractor bar (5) is pressed onto skull of chicken, and can be moved horizontally or vertically by rotating screws (6 and 7).

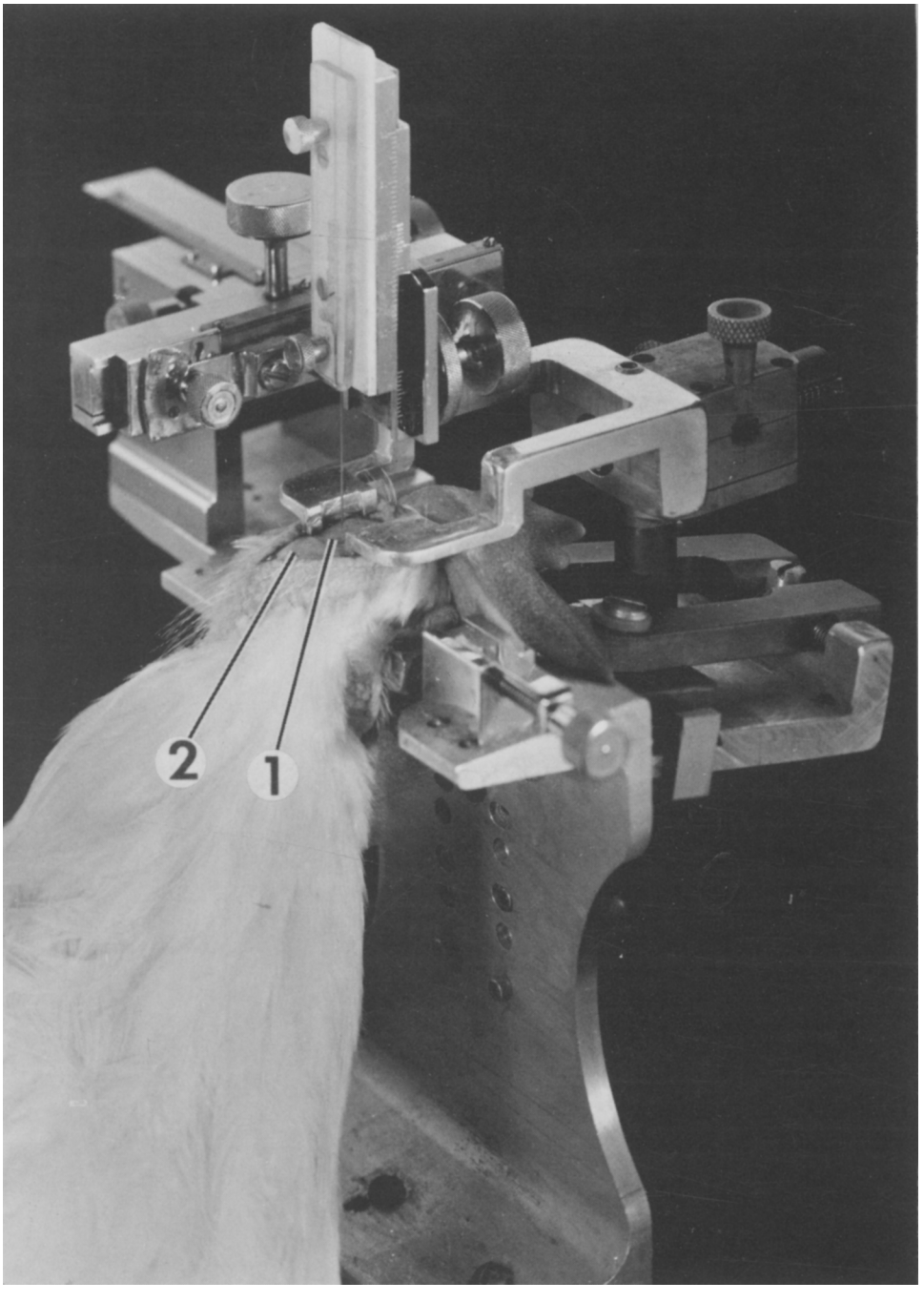


Fig. 2. Position of chicken head in stereotaxic instrument.
Note frontal and parietal bones (1 and 2) of skull.

to 20 sec. Most of the lesions were made with 2 ma for 15 sec. Bilateral symmetrical lesions were sought in most instances, although some single midline lesions have been made.

Care of chickens

The care of the chickens has been described elsewhere (Feldman *et al.*, 1957; Koike and Lepkovsky, 1967; Lepkovsky and Yasuda, 1966, 1967; Lepkovsky, Snapir, and Furuta, 1968). All surviving chickens were kept a minimum of two months and usually for a year or more. Physiological and behavioral responses were recorded. On the death of a chicken, or at autopsy, the brain was immediately fixed in 10 per cent neutralized formalin, and thionine sections (frozen or celloidin) were prepared.

RESULTS AND DISCUSSION

Figures 3 to 13 show transverse sections of the forebrain with accompanying diagrams of the sections.

Locations of lesions, and the patterns of physiological and behavioral responses

Note that for many functional changes the lesions are close to the ventromedial hypothalamic area, between 5 to 7 mm anterior to the zero point. The photographs depict only a portion of the lesions, usually at greatest diameter, and usually near the midsection of the lesion.

Aphagia (Feldman *et al.*, 1957) (figs. 14 and 15); hyperphagia accompanied by functional castration (Snapir, Nir, Furuta, and Lepkovsky, 1969) (fig. 16); hyperphagia with normal comb and testes (unpublished) (fig. 17); hyperphagia with normal comb and testes without excessive adiposity (unpublished) (fig. 18); adipsia (Lepkovsky and Yasuda, 1967) (fig. 19); polyuria or polydipsia (Koike and Lepkovsky, 1967) (fig. 20); failure to regulate body temperature (Lepkovsky, Snapir, and Furuta, 1968) (fig. 21); and permanent functionally-castrated males and females without hyperphagia (Snapir, Nir, Furuta and Lepkovsky, 1969) (fig. 22) have been produced in chickens in this laboratory by means of stereotaxic lesions. Lesions with no apparent physiological or behavioral effects are shown in figures 23 to 28.

Disturbance of egg production following hypothalamic lesions was studied by Egge and Chiasson (1963) and Ralph (1959). The effect of hypothalamic lesions on body temperature of chickens was reported by Kanematsu *et al.* (1967).

Many birds showed lesions in the anticipated loci in the hypothalamus, and gave anticipated physiological responses. The use of the same coordinates did not always result in anticipated locations of the lesions. This may have been the result of variability in the chicken head. Yasuda and Lepkovsky (1969) have described great variability in the measurements between landmarks of the chicken brain and skull. Differences in location of hypothalamic lesions using specific stereotaxic coordinates may be explained by this variability. For example, the chicken head occasionally deviated so markedly in structure that it did not fit the stereotaxic instrument. While correlation between stereotaxic coordinates and the location of the lesion leaves much to be desired, the correlation between location of the lesions and specific physiological or behavioral responses is excellent. When similar physiological changes occurred in two or more chickens, lesions were found in the same, specific areas of the hypothalamus of each bird.

KEY TO NUMBERS IN FIGS. 3 THROUGH 13

1 Entorhinal area
2 Hippocampal area
3 Lamina hyperstriatica
4 Lamina medullaris dorsalis
5 Paleostriatum
6 Lateral ventricle
7 Hyperstriatum
8 Neostriatum intermediate
9 Lateral forebrain bundle
11 Septo-mesencephalic tract
12 Ectostriatum
13 Medial preoptic N
14 Preoptic recess
15 Supraoptic N
16 Lateral borebrain bundle
17 Lateral geniculate body
18 Septal area
19 Anterior commissure
21 Suprachiasmatic N
22 Pallial commissure
23 Dorsal reticular N
24 Lateral anterior N
25 Ventral lateral N
26 Paraventricular (superior) N
27 Paraventricular (inferior) N
28 Supraoptic decussation
29 Bed nucleus of pallial commissure III Third ventricle
30 Dorsolateral N
31 N. rotundus
32 Ventral reticular N
33 Dorsal area
34 Dorsal medial anterior N
35 Paraventricular magnocellular N
36 Hypothalamic ventromedial area
37 N. subrotundus
38 Entopeduncular posterior N
41 N. internus superior
42 Superficial parvocellular N
43 Ovoid N.
44 Lateral habenular N
45 Medial habenular N
46 Dorsal mammillary
47 Principal precommissural N
48 Optic tract
50 Hypophysis (stalk)
51 Dorsomedial spiriform N
52 Ventrolateral spiriform N
53 Posterointermedius N
54 Subpretectal N
56 Ectomammillary N
58 Posterior commissure
59 Red nucleus
60 Ventromedial mammillary N
61 Optic chiasm

Figs 3–13. Transverse sections of chicken forebrain with accompanying diagrams of sections, ranging from 9.2 to 3.8 mm anterior to the interaural line. The scale of drawings is in millimeters. Fiber tracts are indicated by unbroken parallel lines. Cell group patterns are shown by interrupted lines at the approximate borders. In nomenclature we have used terms ascribed by Kuhlenbeck (1936, 1937) and by Crosby and Woodburne (1940). For explanation of numbers on drawings, see above.

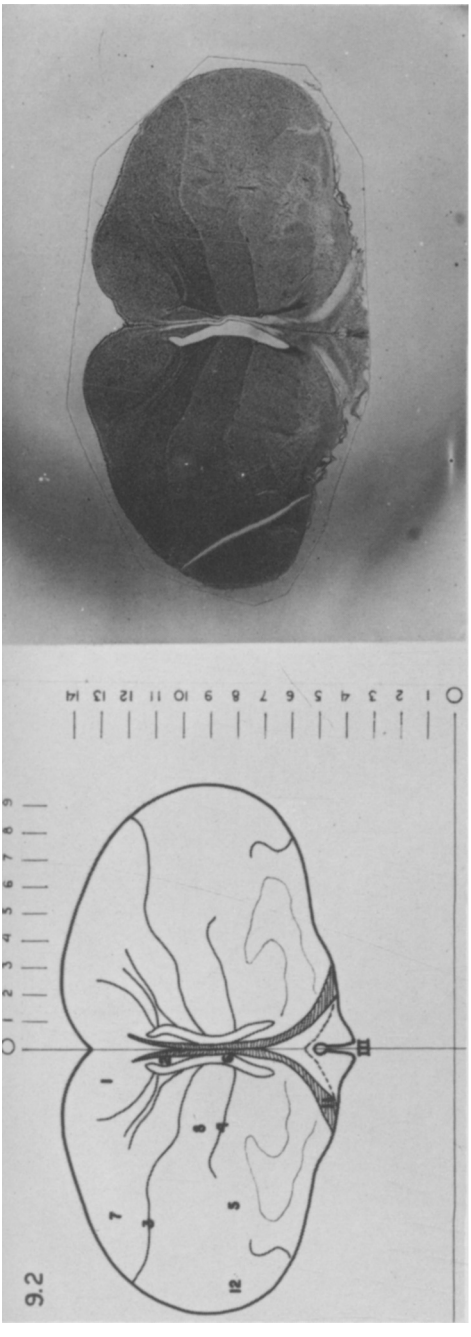


Fig. 3

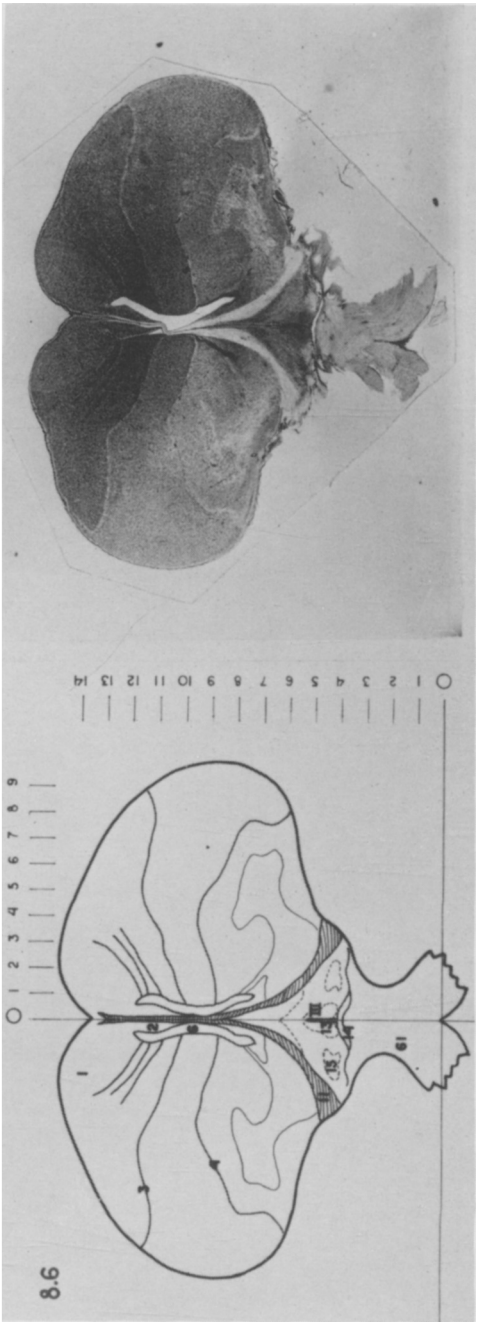


Fig. 4

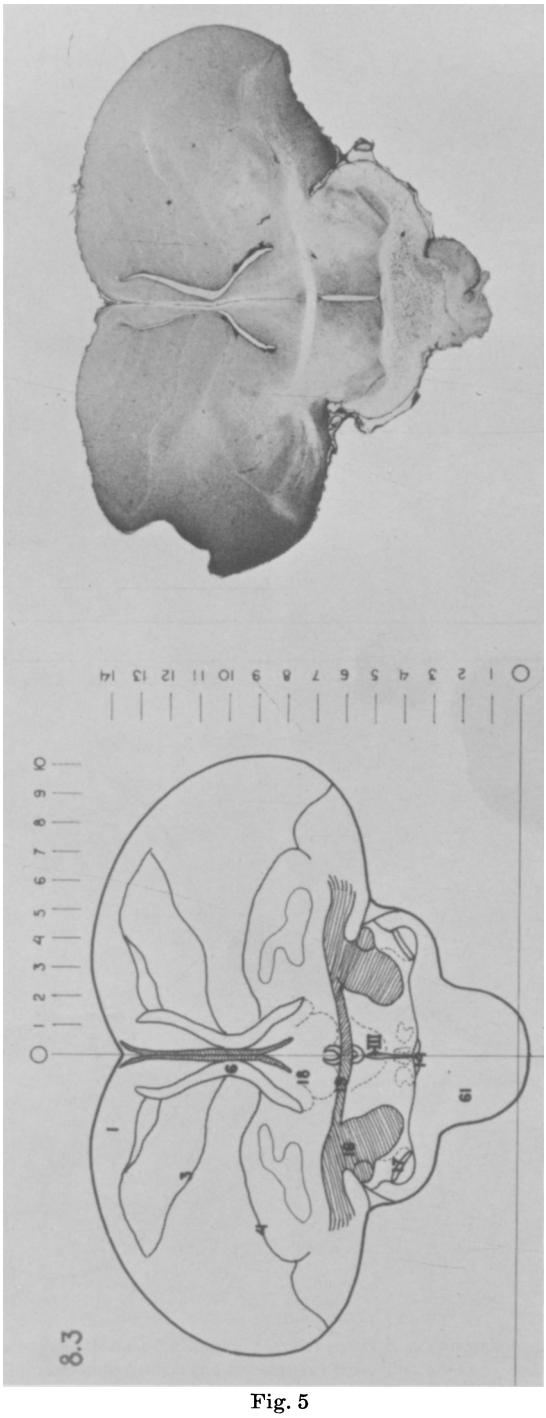


Fig. 5

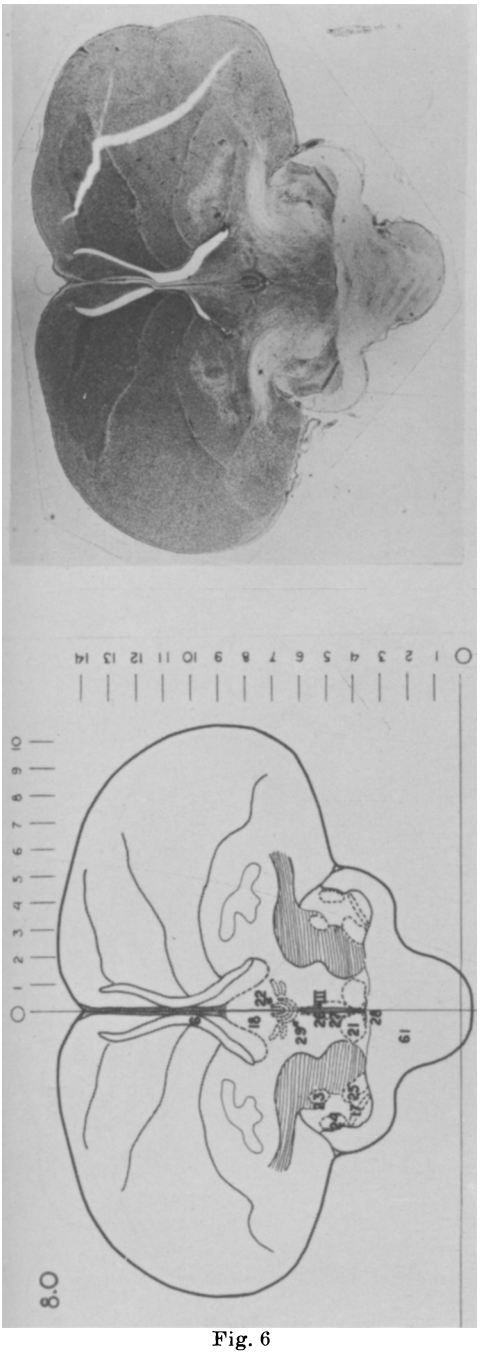


Fig. 6

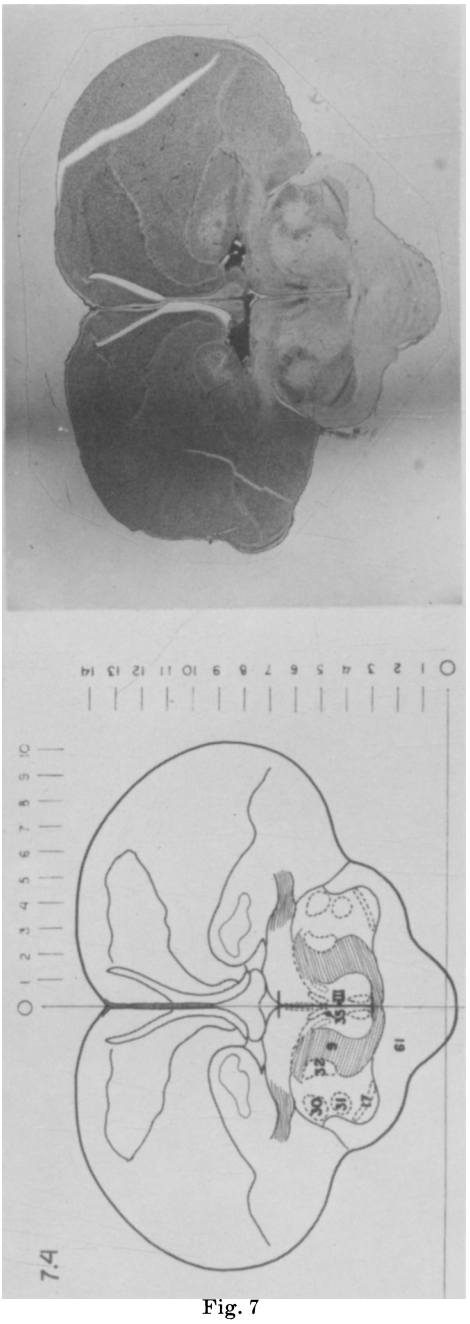


Fig. 7

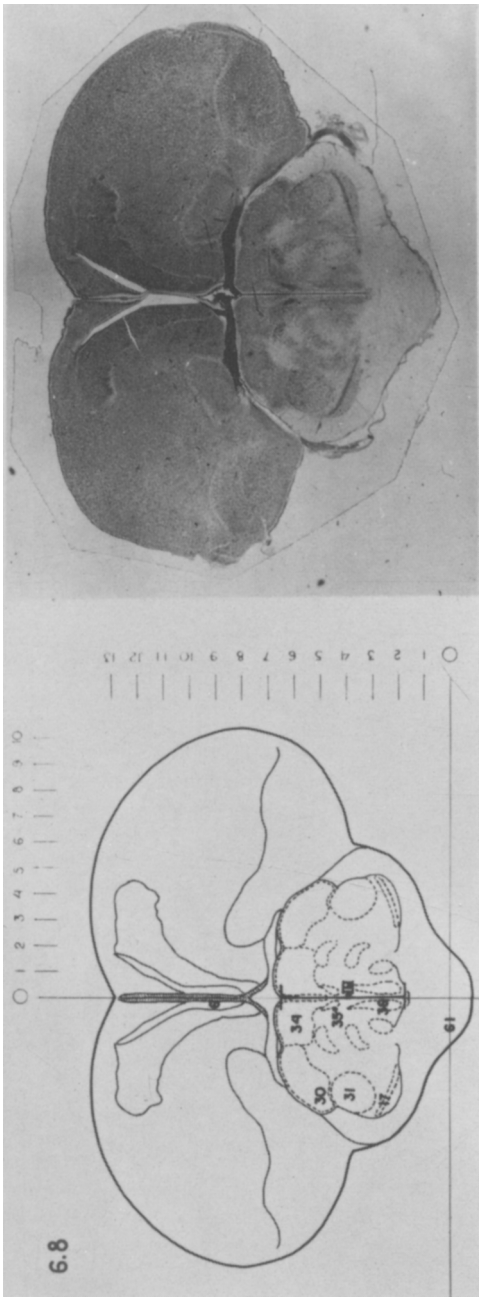


Fig. 8

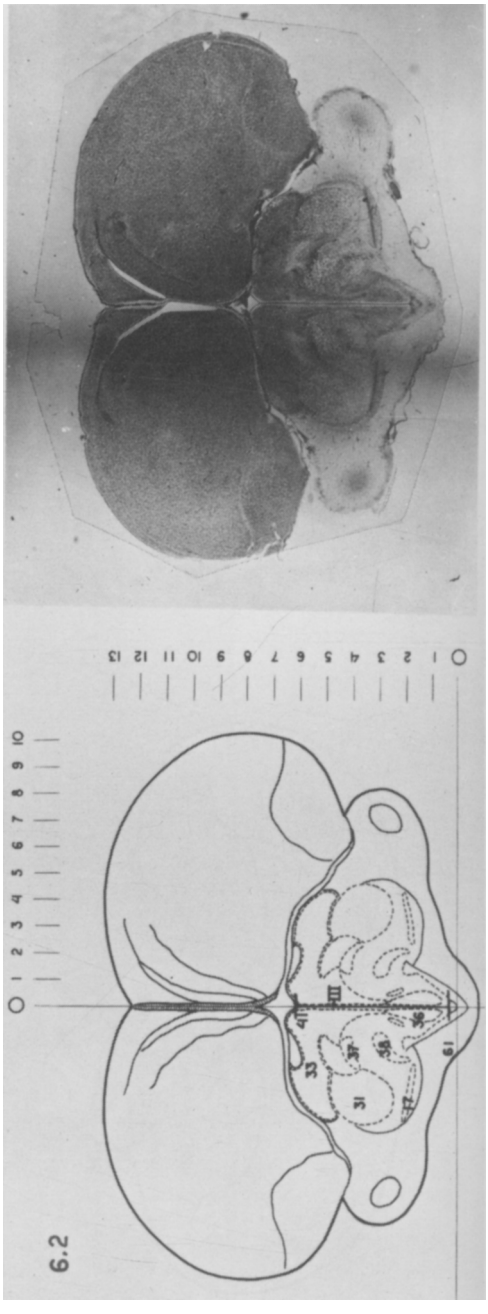


Fig. 9

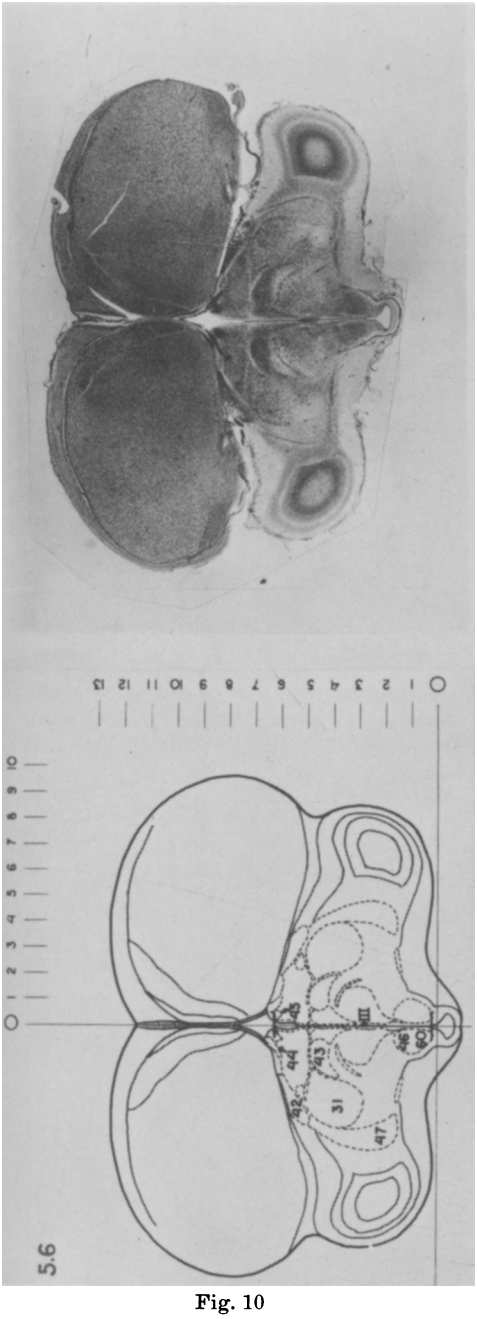


Fig. 10

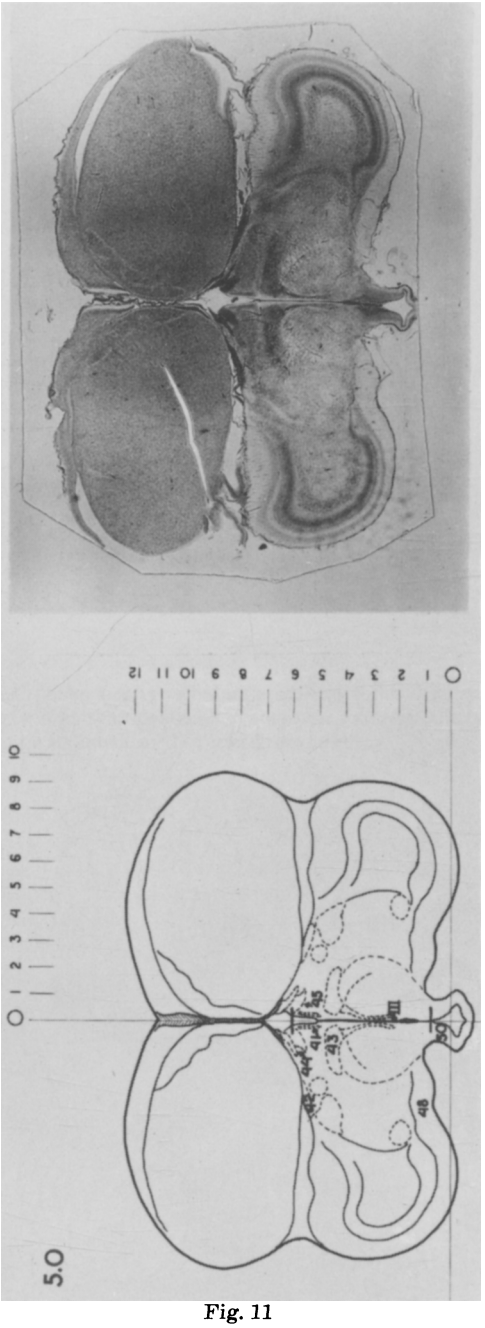


Fig. 11

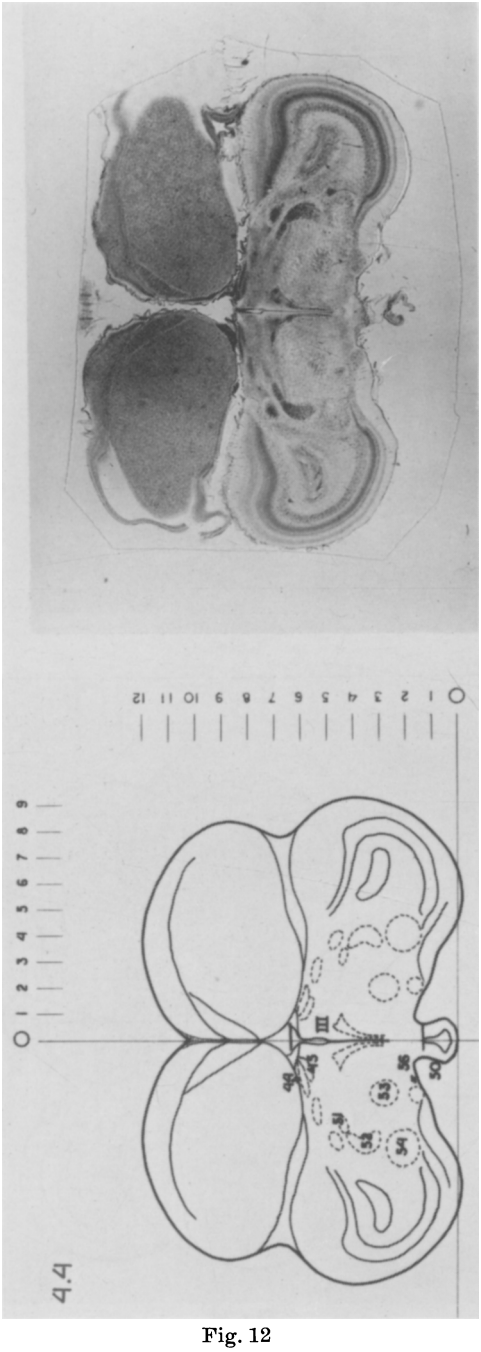


Fig. 12

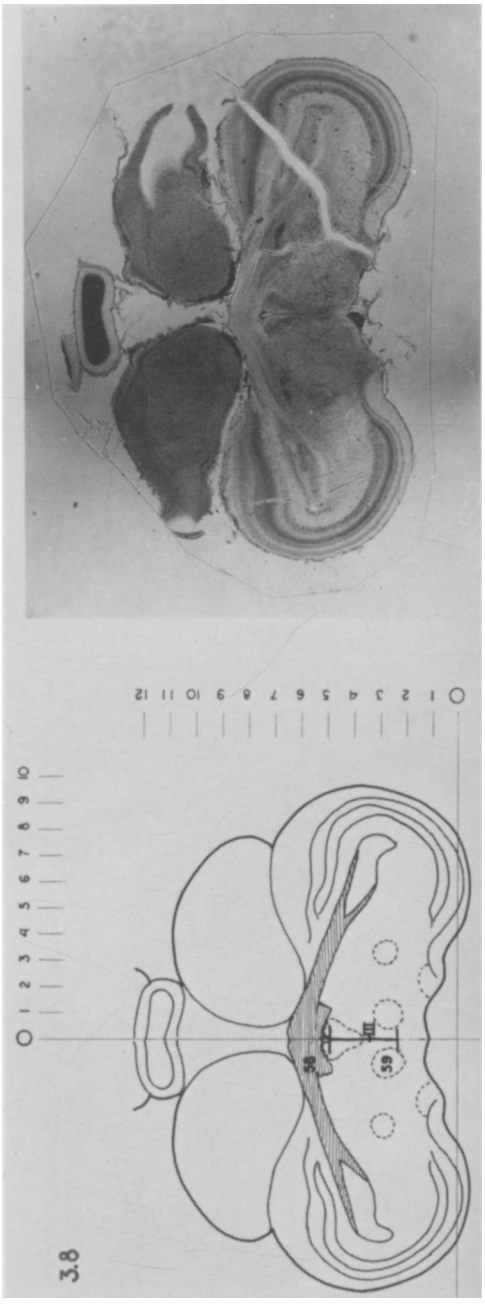


Fig. 13

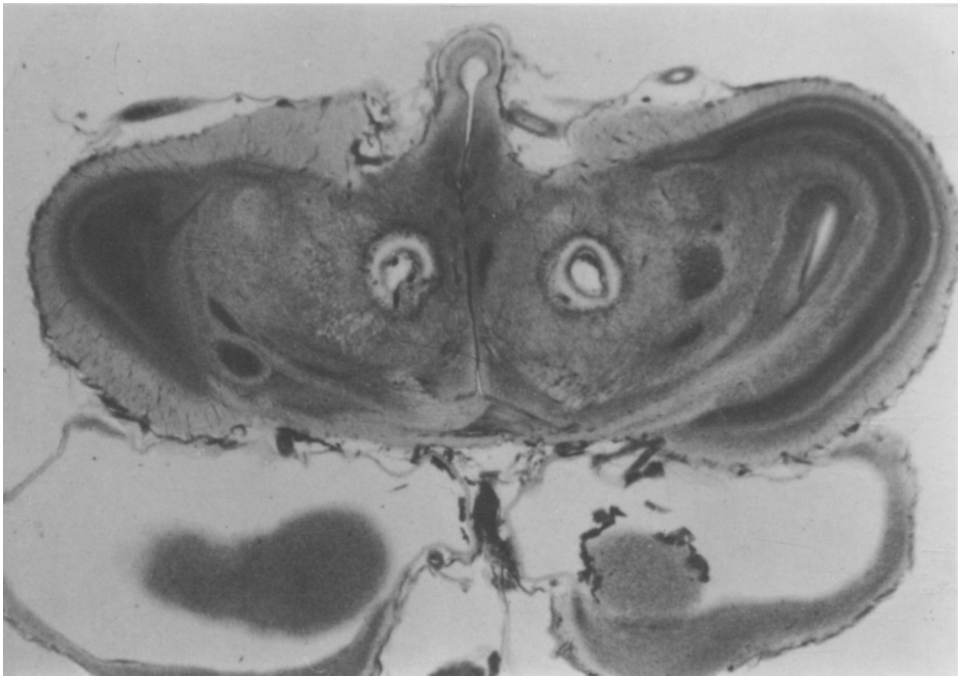


Fig. 14. Bilateral, symmetrical lesions in the posterior hypothalamus of aphagic chicken. Specimen has been cut obliquely from dorsal surface so that posterior commissure and pituitary stalk are both present in this section. Lesions range as much as 1–2 mm from midline.

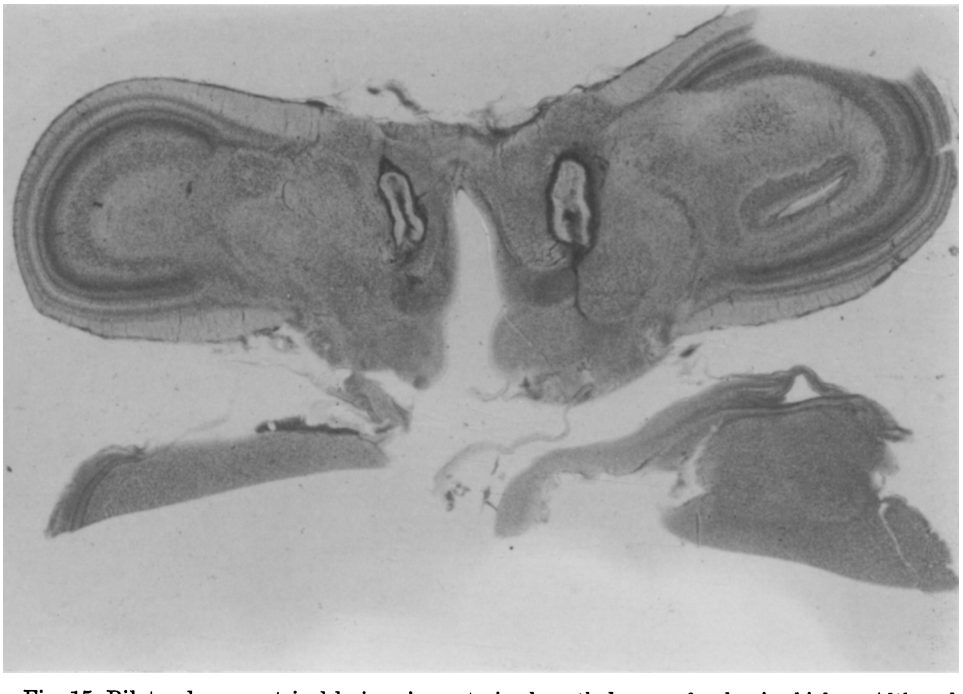


Fig. 15. Bilateral, symmetrical lesions in posterior hypothalamus of aphagic chicken. Although lesions appear to be posterior to hypophysial stalk, the dorsal structure of the brain above the lesions shows both ovoid N., indicating that each lesion extends into area of the stalk—the section being cut in a postero-ventral direction. In addition to being aphagic, this chicken did not regulate its temperature.

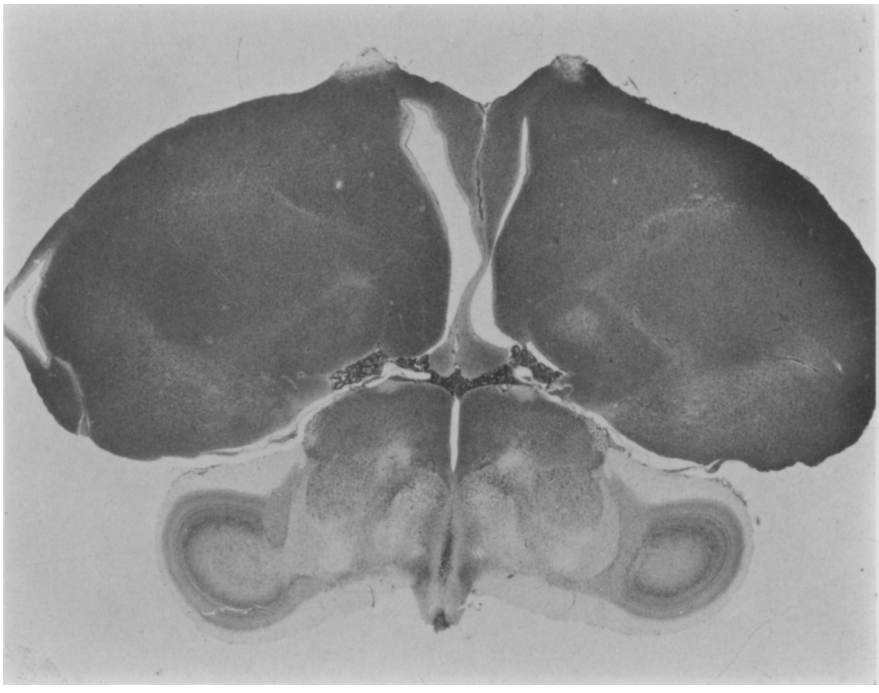


Fig. 16. Brain section of a functionally castrated hyperphagic chicken. Lesion is confluent, close to the midline (within 0.3 mm), and in addition to involving the ventromedial area on each side, extends caudally to encroach on the mammillary area and stalk and sometimes ventrocaudally into the apparent arcuate nucleus region. These chickens, in addition to increasing their food intake and depositing large amounts of fat, showed gonadal atrophy and comb atrophy.

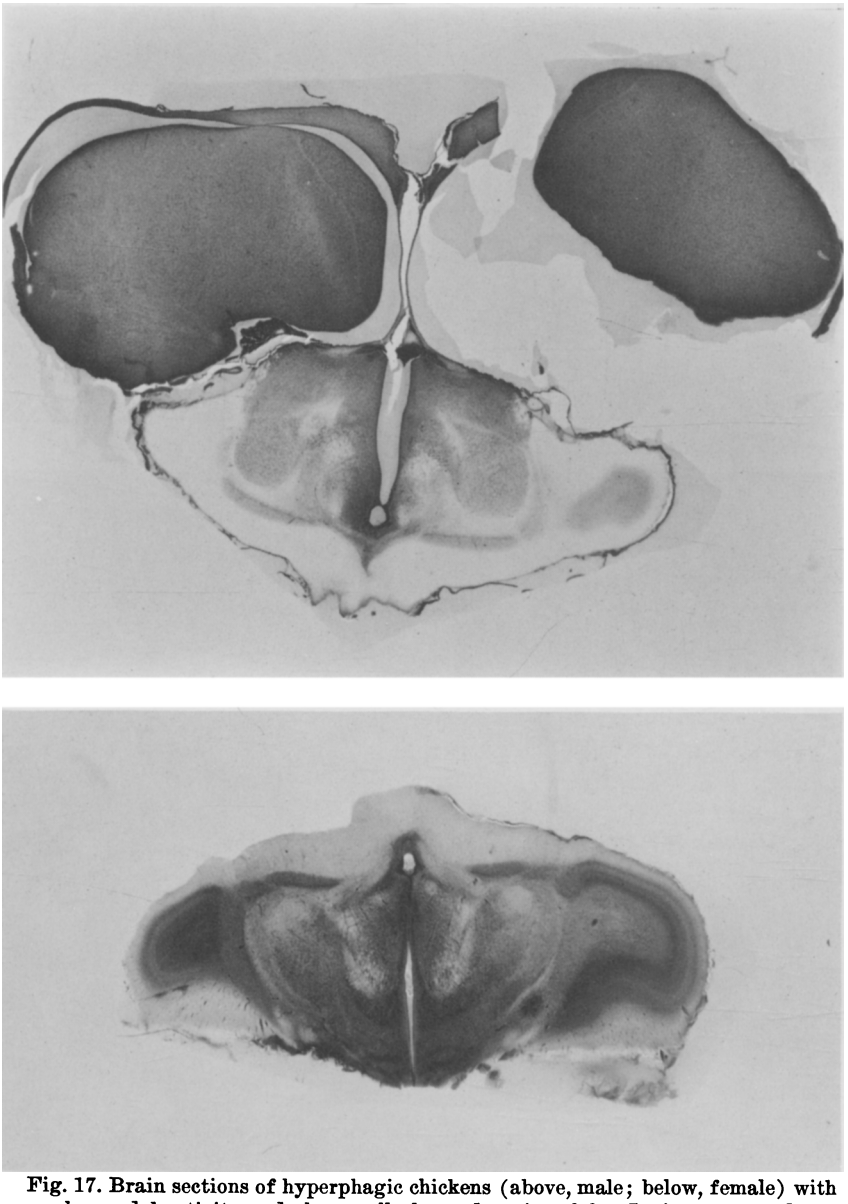


Fig. 17. Brain sections of hyperphagic chickens (above, male; below, female) with normal gonadal activity and abnormally large deposits of fat. Lesions are confluent in the midline, and are located at junction of dorsal and ventral halves of ventromedial region. Note that these lesions are located approximately 2 mm above the point of the inverted pyramid made by the ventromedial area, and include most of the ventromedial nuclei area on each side. Lesions do not extend posteriorly to the area above the stalk.

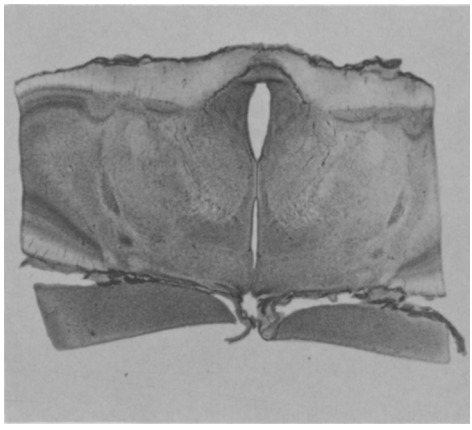


Fig. 18. Brain section of hyperphagic chicken in which increased food intake was noted without abnormal fat deposition. Testes markedly enlarged. Lesions are high in the ventromedial area, on the midline.

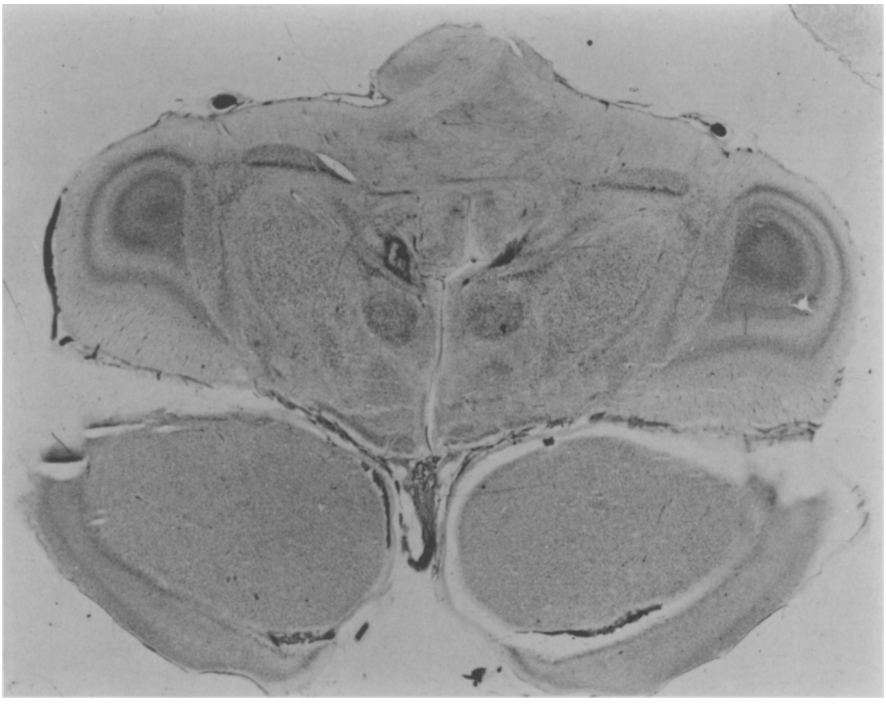
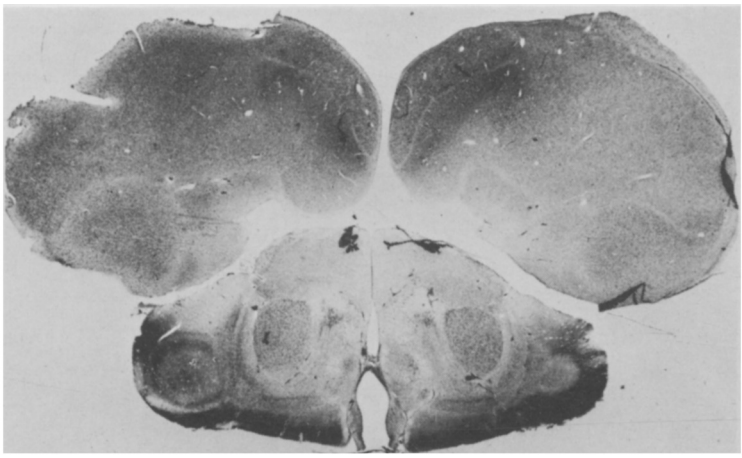


Fig. 19. Sections of two adipsic chicken brains. Above, lesions are bilateral about 1 mm from the mid-line in dorso-lateral hypothalamus, slightly anterior to ventromedial area. Note that, as obliquely sectioned, ventral part of brain is more rostral than the dorsal. Page 621, upper photo, the lesions are in the dorsal hypothalamus anterior to the stalk and about 0.5 mm from the midline.



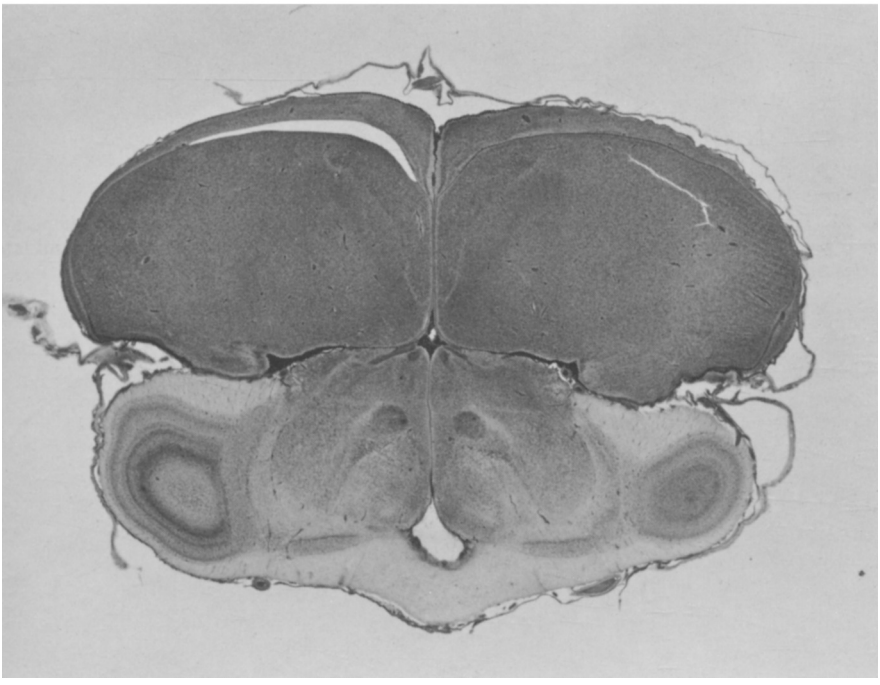


Fig. 20. Lower photo only, brain section of a chicken with polyuria and polydipsia. Note that, as cut, the ventral part of the brain lies slightly more rostral than the dorsal. Symmetrical lesions are slightly rostral to ventromedial area, as noted in fig. 7, and are slightly more lateral than in hyperphagic birds. They extend 0.5 mm from midline on each side. There is associated dilatation of the third ventricle.

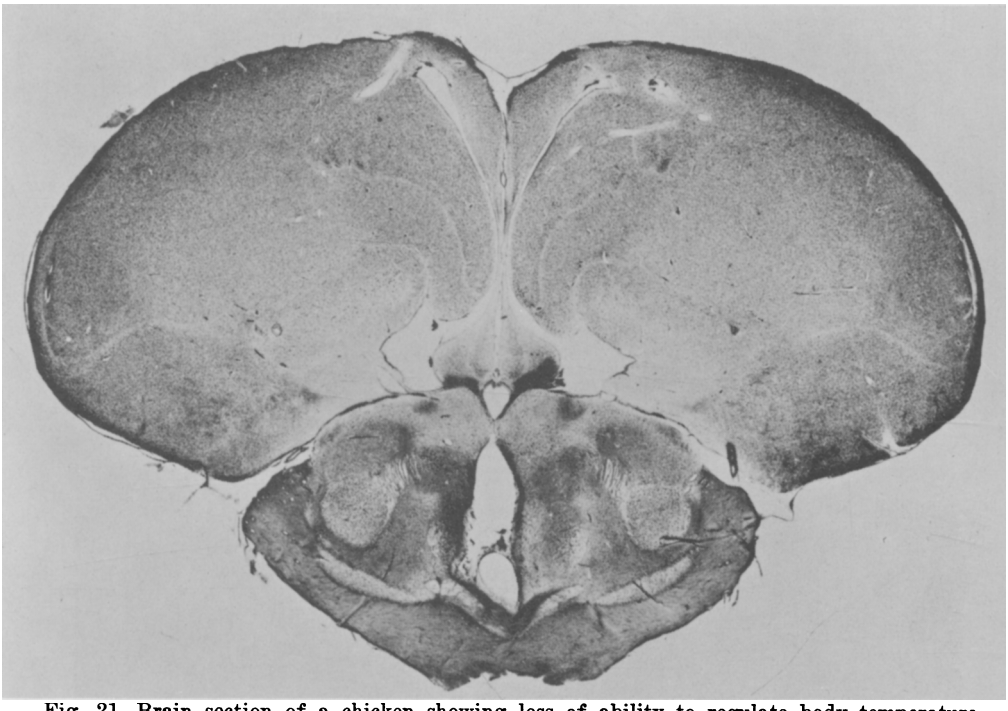


Fig. 21. Brain section of a chicken showing loss of ability to regulate body temperature. Bilateral lesions are in anterior hypothalamus, dorsal to the ventromedial area. Third and lateral ventricles are dilated.

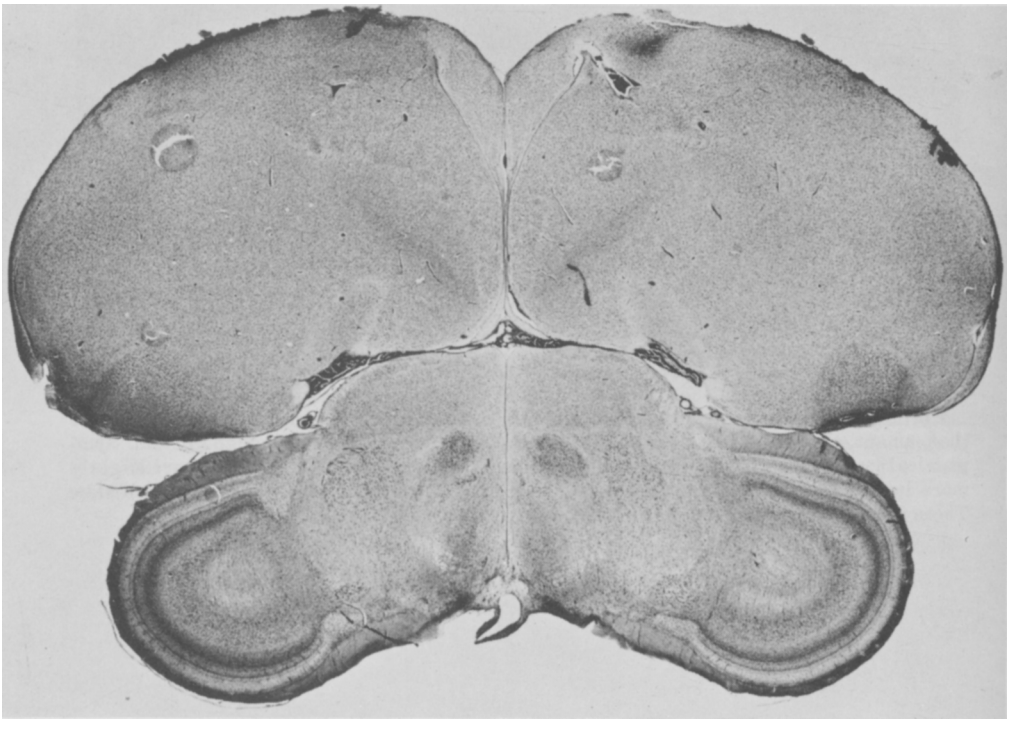


Fig. 22. Brain of chicken which became functionally castrate. Lesions are bilateral, close to midline, in area of the stalk, and do not involve ventromedial nuclei. These lesions are comparable to the stalk section in their physiological effect.

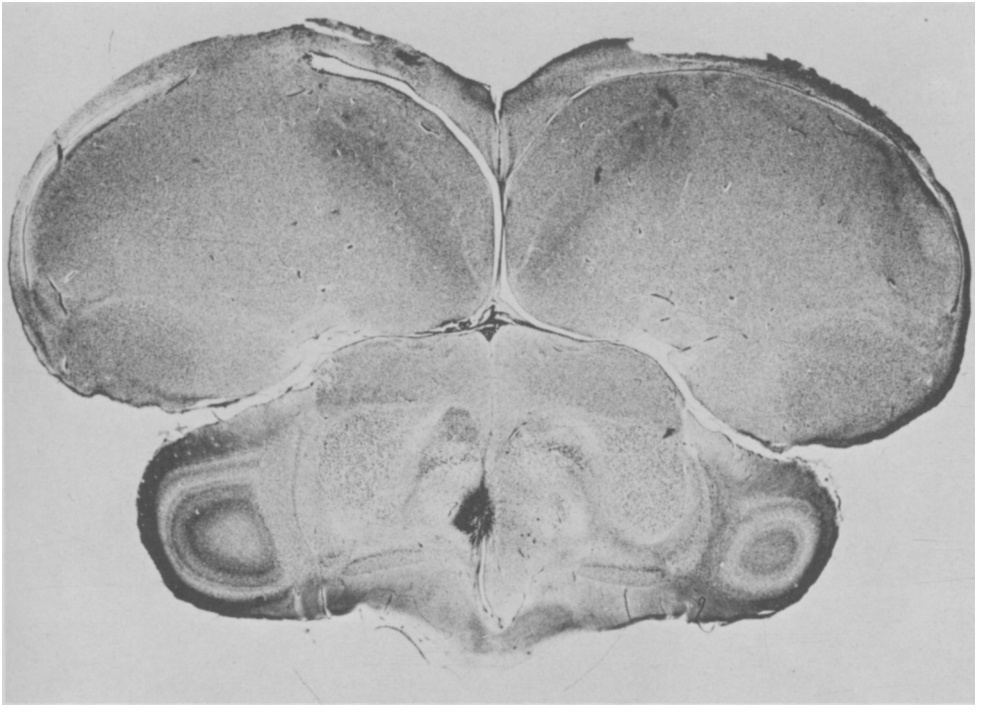
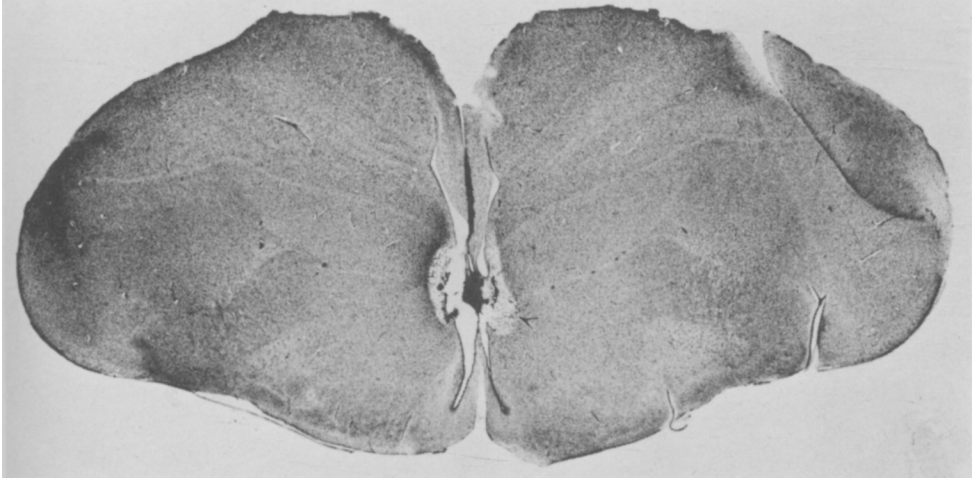


Fig. 23. Above only, brain lesions destroying dorsal part of ventromedial area. No physiological effect was noted from lesions in this chicken.



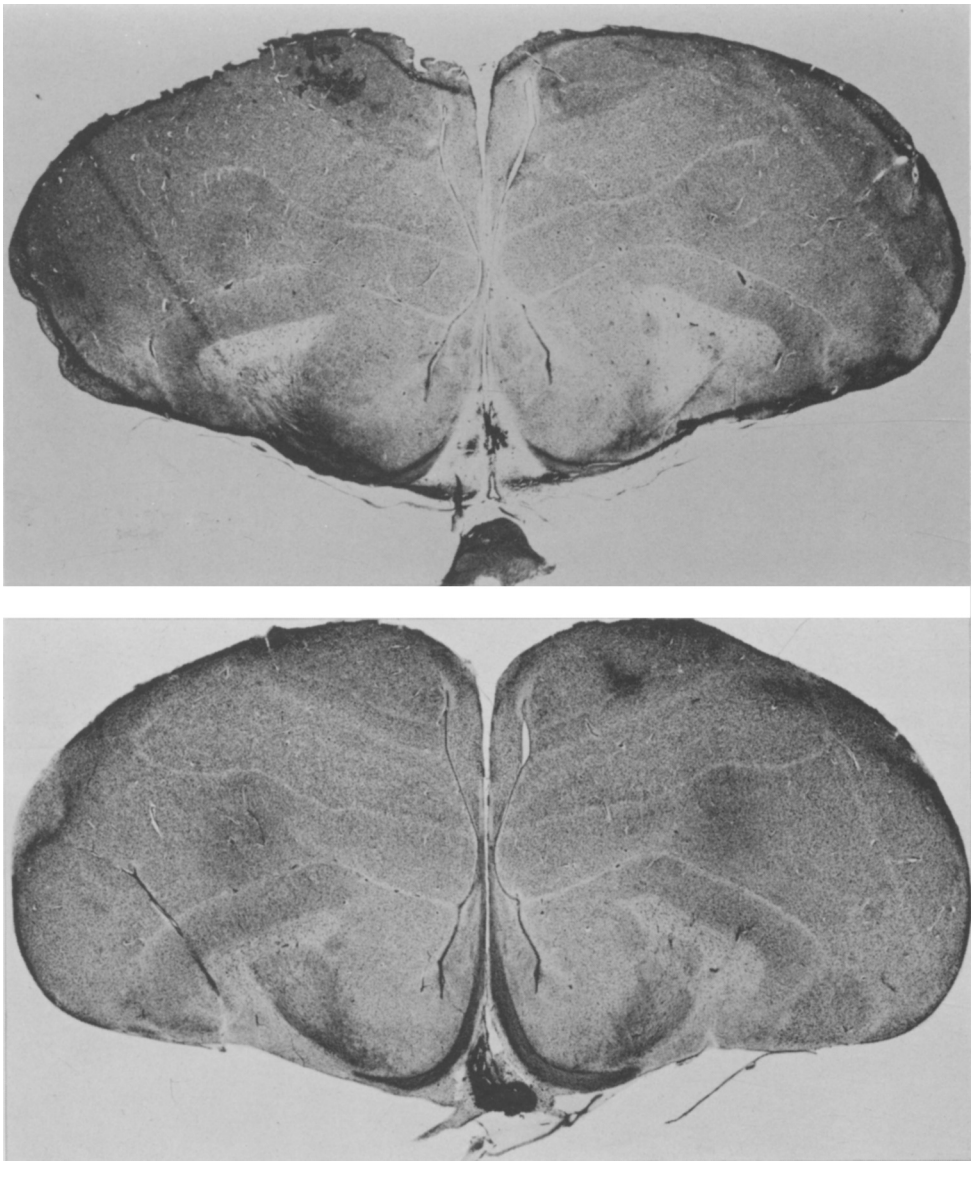


Fig. 24. (See also bottom photo, page 623.) Brain lesions in area of septo-mesencephalic tract. These 3 chickens lived normally for 2 months.

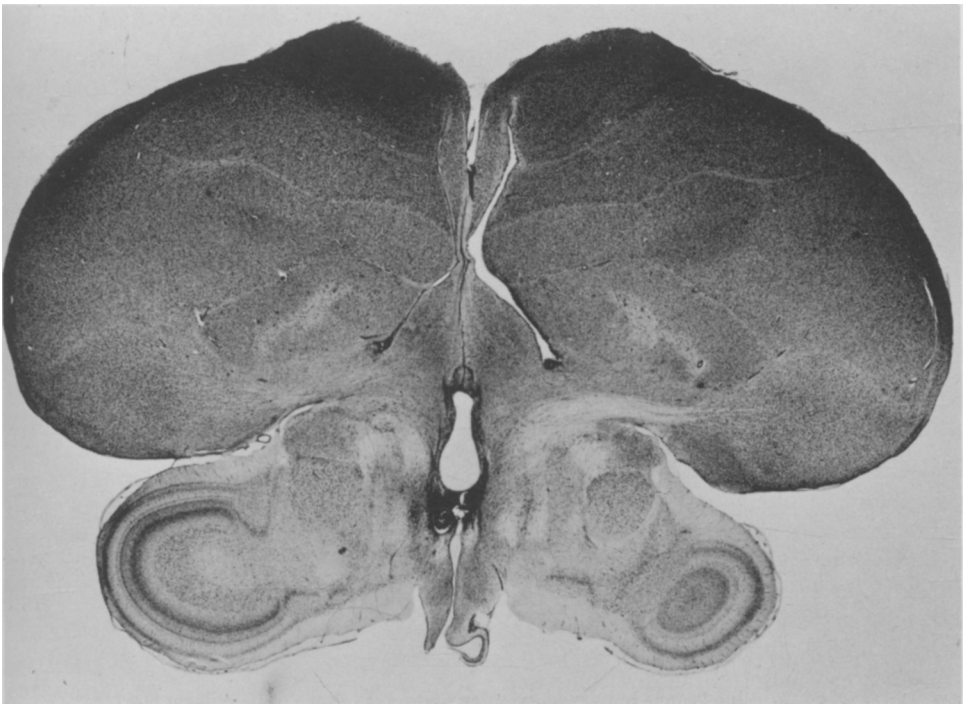


Fig. 25. Midline lesions in plane of stalk area of brain, with dilatation of third ventricle. Lateral lesions at this level produce aphagia, but 5 chickens with lesions as above showed no effect as a result of lesioning.

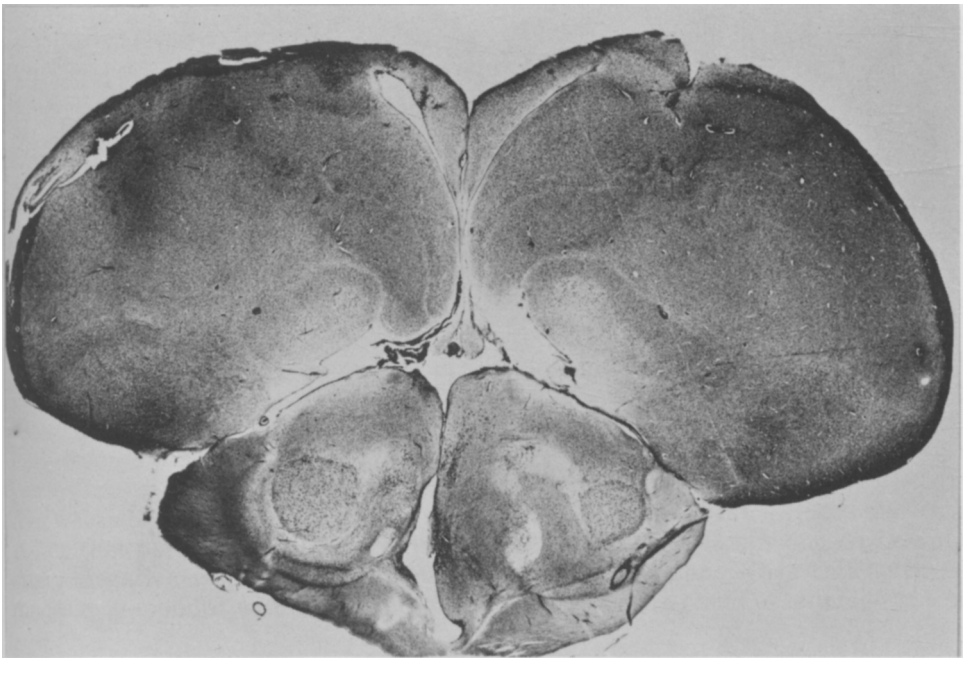


Fig. 26. Brain lesions in anterior hypothalamic area, which are too high to produce hyperphagia and too anterior to interfere with regulation of body temperature.

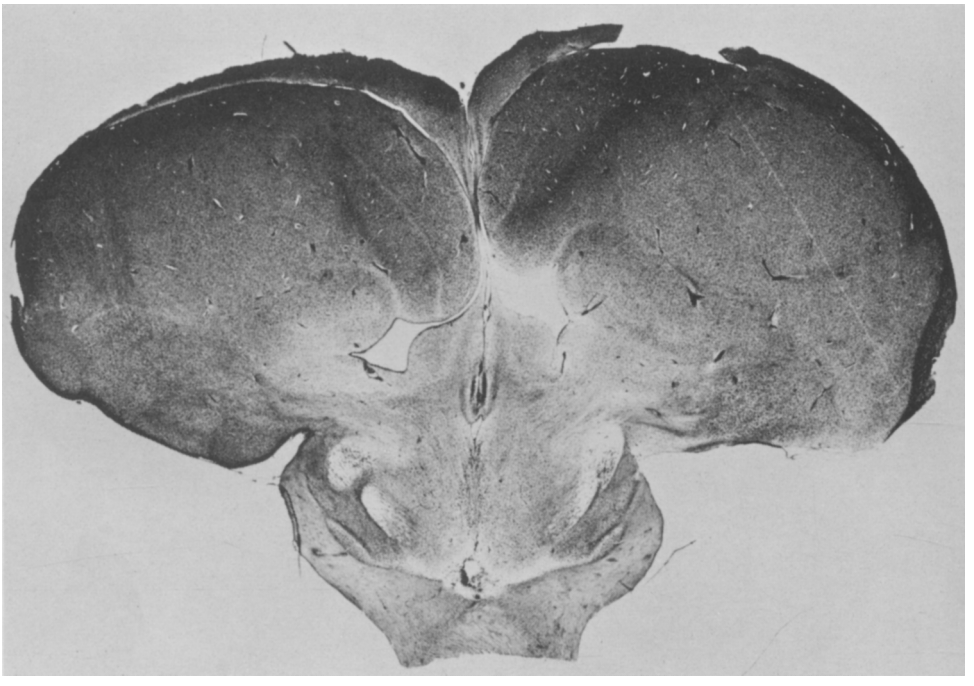


Fig. 27. Midline lesions in paraventricular plane just above optic chiasm but too far anterior to ventromedial area to cause hyperphagia.

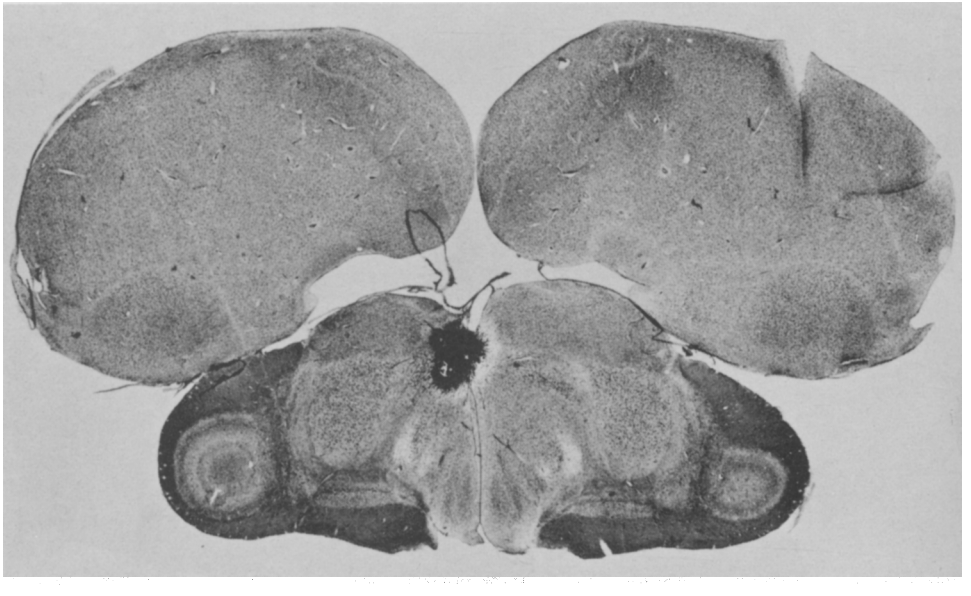


Fig. 28. Brain lesions on midline, without observable effect.

Multiple physiological responses

Frequently, lesions evoked multiple responses. Some physiological responses were transitory, some permanent.

1. Sixteen lesioned chickens showed hyperphagia, polyuria, and functional castration. Only polyuria was transitory, lasting 5, 7, 25, 30, 30 and 60 days, respectively, in six of the 16. Six other chickens showed hyperphagia and polyuria. Again, polyuria was transitory, lasting approximately one month. Apparently, polyuria associated with either hyperphagia or functional castration is transitory. In 23 polyuric chickens without other complications, polyuria was permanent (three to four months).

2. Twenty-four chickens did not eat for six days after lesioning. After they had been tube-fed for six days, 16 started to eat. The rest were permanently aphagic.

3. Out of 15 chickens that showed impairment in regulating body temperatures at 70°F, seven were aphagic for one to two weeks and one for eight weeks.

4. Six chickens were adipsic. Of these, four showed transitory impairment in temperature regulation. Two showed both permanent adipsia and impaired temperature regulation, one for over three years.

5. While functional castration was common (regression of combs and atrophy of testes), 12 males showed transitory regression of combs for about six to nine weeks, indicating functional castration. During that period, abdominal massage and "milking" procedure failed to evoke libido or the discharge of semen. Spontaneous recovery occurred; combs became red, and grew for four to five weeks to approximately normal size. After that period, the cocks with previously negative responses to abdominal massage responded with libido and semen discharge. At autopsy, the chickens had normal-sized testes.

Multiple responses such as hyperphagia and functional castration, adipsia, obesity, and impaired temperature regulation emphasize the complexity of hypothalamic lesioning (Fisher and Coury, 1962; Morgane, 1967). Stevenson (1967) pointed out that manipulation of any area of the hypothalamus involves thousands of cell bodies and fibers in the lesioning process.

Possible causes for transitory physiological responses to lesioning

In the first 10 to 14 days after lesioning, physiological changes noted may give no information as to the possible permanent effect of the lesions. Some of the physiological responses may be the result of edema, inflammation, and disturbances in circulation which accompany electrocoagulation of brain tissues. Such disturbances may subside in the course of a week or two, after which residual physiological responses may be attributed to effects of the lesion. In many cases, recovery of normal functions of reversible damage was delayed for longer periods of time, in some cases as long as two months (Rowland, 1966).

Food intake and body composition

The table shows averages of body weight gain, food intake, and efficiency in the utilization of the food in various categories of hypothalamic-lesioned hyperphagic White Leghorn cockerels four weeks after the operation.

Although lesions, even when properly placed, will occasionally have no effect on the rooster's body fat, the usual reaction is increased body weight generally due to the production and accumulation of extra fat—which, in turn, tends to modify in varying degrees the body composition. When this extra fat does occur as a result of lesioning, it con-

tinues to increase and body composition of the cock can be controlled by the time the rooster is permitted to live. Hyperphagia also continues, as well as increased efficiency in the utilization of food.

AVERAGES (±S.E.) OF BODY WEIGHT GAINS, FOOD INTAKE, AND EFFICIENCY IN THE UTILIZATION OF THE FOOD IN VARIOUS CATEGORIES OF HYPOTHALAMIC-LESIONED HYPERPHAGIC WHITE LEGHORN COCKERELS,—CALCULATED FOR A PERIOD OF FOUR WEEKS AFTER LESIONING (n=15)

Groups of cockerels	Initial body weight	Body wt. gain	Abdominal fat	Comb wt.	Testes wt.	Food intake	Food eaten per body gain
	gm	*gm*	*gm*	*gm*	*gm*	*gm*	*gm*
Hypoth.-lesioned: obese, atrophied comb and testes...	1380±20	641±18	124±8	2.9±0.2*	0.5±0.07*	3973±53	6.5±0.1
Hypoth.-lesioned: obese, normal comb and testes...	1370±18	582±16	72±10	101±6	13±1	3524±44	6.2±0.1
Hypoth.-lesioned: not obese, normal comb and testes...	1390±18	597±16	10±2*	113±5	13±1	3636±50	6.3±0.1
Normal controls.....	1375±22	400±10*	4±0.3*	114±7	14±1	2750±40*	7.3±0.1*

* Significant at the 1 per cent level.

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