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A Study of Ovarian Abnormalities in Fowls

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A STUDY OF OVARIAN ABNORMALITIES IN FOWLS

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This is to certify that, in accordance with the requirements of South Dakota State College for the Master of Science Degree, Mr. Donald F. Petersen has presented to this committee three bound copies of an acceptable thesis, done in the major field; and has satisfactorily passed a two-hour oral examination on the thesis, the major field, Zoology, and the minor field, Chemistry.

Advisor

July 8, 1950
Date

Head of Major Department

Head of Minor Department

Rep. of Graduate Committee

Acknowledgement

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HISTORICAL

From ancient times it has been recognized that when a cow gives birth to twin calves, one a male and the other a female, the bull will develop normally whereas the cow often, though not always, will be a freemartin. That is, her reproductive organs tend toward the male type. On the other hand, if both offspring are of the same sex, they are normal. John Hunter (1) observed this condition in studies on the horse, ass, cow, and sheep but not in man, the cat, or dog.

William Harvey (2) noted in horses and cloven-footed animals which occasionally produce twins, that in some instances each twin had its own placenta, while others shared a single placenta.

Lillie (3) showed that the freemartin is zygotically a female modified by the sex hormones of the male owing to the fusion of the two chorions and the consequent interchange of blood.

In 1837 Hunter noted that the sexual characters depend largely on gonadal activity. He remarked that prior to the onset of puberty there is not much to distinguish the male from the female. The special features which distinguish the male from the female appear mostly at the time of puberty. After the reproductive period has ended the appearance of the female tends toward maleness, or more properly, the hermaphrodite.

The first demonstration of the hormonal action of the testis was made by Hunter in 1794. He reported that spur transplants from a young cock to the shank of a young hen were successful

but no spur grew. The rudimentary spur from a hen, when transplanted to a cock, grew nearly as fast and to as large a size as the natural spur of the other leg.

Berthold (4) showed that the castration effects in fowls could be alleviated by testicular grafts.

The belief that the feebleness of aged men was partially due to testicular failure led Brown-Séquard (5) in 1889, at the age of 72, to inject himself with extracts from the testes of dogs and guinea pigs. He noticed some improvement in muscular tone and increased bodily and mental vigor. However his results were not particularly striking and he thought that they were perhaps attributable to autosuggestion. Variot (6) injected male patients in the same manner as Brown-Séquard and observed similar results but likewise thought them to be the consequence of autosuggestion.

The recognition of the hormonal basis of ovarian activity began in 1905 with the work of Marshall and Jolly (7). These workers induced estrus in spayed dogs with both ovarian extracts and peritoneal implants of estrus ovaries. They also observed that the secretion which causes estrus is different from the principle produced by the corpora lutea.

The next great advance was the discovery of "oestrin" or "folliculin" by Allan and Doisy (8) in 1923. A principle isolated from the liquor folliculi of the sow's ovary induced estrus changes in the rat vagina. This work eventually led to the

synthesis and characterization of crystalline "oestrone" by Doisy, Veler and Thayer (9) in 1930, and, independently, by Butenandt (10) in 1929.

INTRODUCTION

Accurate quantitative studies of effects produced by gonadal hormones were impossible prior to 1929 because of the variations in crude extracts. With the advent of pure crystalline preparations of both estrogenic (9,10) and androgenic (11,12,13,14) substances the picture was clarified to some extent by reducing the activity of various principles to mathematical expressions.

For detailed studies of the gonadal hormones it is useful to divide them into three classes: (a) androgens, (b) estrogens, and (c) progestins. This classification is not entirely reliable in predicting the biological activity of a specific hormone within the organism because intermediates in the metabolism of a hormone may vary in activity and also because of a certain amount of overlapping in the potentialities of the various hormones.

The chief naturally occurring androgen derived from the testes is testosterone which apparently is converted in the liver to androsterone and its stereoisomer aetiocholane-3(α)-ol-17-one. The latter two are excreted in the urine. Trans-Dehydroandrosterone is formed from cortical precursors in the adrenal gland.

Graphic formulae for the chief saturated and unsaturated androgens are shown in Figure 1.

Numerous compounds closely related to testosterone and androsterone have been shown to possess androgenic activity. The members of this series differ only in the relationships of

keto- and hydroxy- groups in positions 3 and 17, the spatial relationship of the hydroxyl group, the presence--or absence-- and the location of unsaturated linkages in ring A.

The importance of these relationships has been pointed out by Butenandt (15), who has shown that by merely changing the unsaturated linkage from Δ^1 to Δ^4, the activity of androstene--3:17--dione may be altered from estrogenic to androgenic. He has further shown that hydration of the keto groups at positions 3 and 17 and the shift of the unsaturated linkage to the Δ^5 position of ring B produces a substance with both androgenic and estrogenic activities (gives a positive Allen-Doisy test and supports capon comb growth).

The substances isolated separately by Butenandt (10) and Doisy et al (9) from pregnancy urine proved to be estrone. Later estriol and estradiol were isolated from natural substances. Girard et al (16,17) characterized two other estrogenic substances, equilin and equilinin from pregnancy urine. These have proved to be relatively unimportant in physiological work.

The work of Dodds and Lawson (18) has shown that the phenanthrene nucleus is not necessary in compounds possessing estrogenic properties. Highly active substances containing two benzene rings have been prepared synthetically by Dodds and co-workers (19,20).

Formulae of the chief naturally occurring and synthetic estrogens are shown in Figure 2.

The role of progestin in birds is somewhat obscure but thought by some workers (21) to affect the interval of ovulation. The chemistry and mode of action will not be discussed here.

It should be understood that neither type of hormone, androgen or estrogen, is peculiar to either the male or to the female. Further, there is no vital antagonism between the ovary and testis (22,23,24). Hill (25) joined mature male and female rats as parabiotic twins and found that for the most part each individual retained its sexual characteristics in spite of the interchange of blood.

The production of androgen and estrogen by the gonads does not depend directly on the genetically determined sex of the host. That is, a testis transplant in a female still produces androgen and an ovarian graft in a male produces estrogen, provided the grafts are suitably located for their function. The response of a tissue to a certain hormone is then innate in the tissue and not dependent upon the sex of the host.

Danforth and Foster (26) showed that skin grafts from chicks retained the characteristics of the donor if the recipient was of the same sex, but in strains varying in color between the sexes, the graft responded appropriately to the sex of the host. The grafts then retain their capacity to react in the ordinary manner to gonadal hormones.

It should be further understood that the action of a specific hormone upon a tissue is not necessarily directly opposed

by the so-called hormone of the opposite sex. In many instances, particularly in comb reactions of fowl, both androgen and estrogen are required to support normal development and maintainence (27, 28,29). Within physiological ranges, these synergistic effects are common, and only the absence of one principle or a massive dose of the other will cause abnormal appearance or function.

Figure 3 shows the hormonal relationships controling normal growth and reproduction in the fowl.

Destruction of normal ovarian function in birds by tumor growth or disease frequently results in the acquisition of male secondary sex characters and behavior pattern.

Probably one of the most striking examples of sex reversal occurring in nature has been reported by Crew (30). A Buff Orphington hen, described as a producing bird, underwent a period of transformation during which it took on male characteristics. At the end of this transformation period the bird resembled a normal male both in appearance and behavior. The bird was found to produce viable sperm and eventually sired two chicks. Autopsy revealed that the ovary had been replaced by a tumor and two functional testes had developed.

Pell (31) examined 8 birds obtained from Crew and found them to be hens in varying stages of sex reversal. One, the specimen described by Crew, had changed completely from a fertile hen to a fertile cock. Two contained mature seminiferous tubules in the left gonad and a small testis was present in each case

on the right side. The ovary of one was largely occupied by typical immature tubules and in another the immature tubules were atrophic. The remaining three contained sex cords and small tubules of an embryonic type.

Reed and Martin (32) have reported a Rhode Island Red hen, previously described as a good layer, which developed an unusually large comb and wattles and a typical male hackle. This bird crowed and assumed the behavior pattern of a maturing cockerel. Examination revealed two well-formed testicles, a degenerate ovary and a small fatty tumor. The testes displayed typical testicular tissue although no sperm could be isolated.

These proliferations of testicular tissue following ovarian degeneration caused by neoplasms also have been reported by Mursier (33).

In many instances of spontaneous sex reversal a tumor is found (34) although other causes for masculinization in hens have been reported, i.e. tuberculosis (35) and fowl typhoid (36). Greb (37) has reported the occurrence of an apparently sexless pheasant with plumage of the male type in which no remnants of gonad tissue could be found.

The present study involves three pheasants and six domestic hens which for one reason or another presented abnormalities of appearance or behavior. A normal pheasant hen collected in the fall of 1947 is included for comparison.

MATERIAL AND METHODS

Histological material from all specimens was removed and placed in suitable fixative (Zenker's, Bouin's, or F.A.A.) and sectioned at 10 microns. Harris haematoxylin and eosin were used in the preparation of sections for routine examination. Heidenhain's iron hematoxylin was employed in the cytological studies of tumor tissue.

Bird No. 1, a cock-feathered pheasant hen, was shot on November 30, 1946 in the Pierre vicinity. The plumage was typically male though somewhat lighter on the belly, and the wings and tail were somewhat shorter than those of the normal male. The bird was smaller than the usual adult male; there were no spurs; and the tarsi and head were small--typically female.

The ovary (Figure 4) was a dark granular mass 12 mm. x 6 mm. No right ovary was present. The oviduct, 4 mm. wide at the cloaca, gave evidences that eggs had been laid during the summer of 1946. One egg (yolk only) was found midway in the oviduct and securely attached to the wall. Below this point of attachment the oviduct seemed to be severed from the cloaca.

Tissue was not suitable for histological study of this specimen. Gross examination revealed the ovary to be darker in color than ordinarily found, but was not otherwise significant.

Bird No. 2, a pheasant hen, was taken at approximately the same time in the Brookings area. This bird was distinctly henny in appearance except for a spur 6 mm. long on the right leg

and a marked amount of coloration of the lore.

The ovary was an indistinct granular mass 16 mm. x 7 mm., grey in color. The oviduct was small and quite difficult to trace. Involution of the bursa of Fabricius indicated that the specimen was a young mature hen from the previous summer hatch.

Histological examination of the ovary disclosed no significant abnormalities. Several atretic follicles were visible along with a considerable number of small developing follicles (Figure 5). The supportive tissue was normal.

Bird No. 3, a normal pheasant hen, was taken in November of 1947 in the Brookings area for comparison. Both gross and histological examinations showed it to be a normal female.

The ovary was a grey granular mass 19 mm. x 11 mm. The oviduct was distinct, easily traced, and indicated that the bird had produced eggs the previous spring.

Histologically the ovary showed numerous developing follicles of small size and several large atretic follicles (Figure 6) containing granular material. The interstitial tissue was normal.

Bird No. 4, a pheasant hen, was taken alive in the area south of Cavour. It was quite thin of flesh but apparently normal. There were no spurs and the plumage was considered to be that of a normal female.

Autopsy revealed a tumorous mass 4 1/2 x 3 cm. at the site of the ovary. Other than the tumor, gross examination showed nothing significant.

Histological examination revealed a tumor tentatively identified as an hemangioma. A large part of the tumor (Figure 7) was occupied by a blood clot. The remainder of the ovarian mass consisted of normally developing follicles and supportive tissue (Figure 8).

Bird No. 5, a white Plymouth Rock in good flesh and apparently normal was examined because of failure to lay. Autopsy revealed a small tumor adjacent to the ovarian mass, and a small imperforate oviduct.

Grossly the tumor appeared as a lobular mass involving about two thirds of the ovarian tissue. Numerous small white nodules about the size of buck shot could be seen. No large follicles could be seen either in the tumor mass or the apparently normal ovarian tissue.

Microscopically the tumor was identified as an adenocarcinoma of questionable origin. Most of the mass consisted of cords of dead cells surrounding isolated areas in the periphery where mitotic figures (Figure 9) could be seen. In these areas the proliferating cells were arranged in typical cord-like structures. A connective tissue stalk joined the tumor to the ovarian mass, but no ovarian tissue could be identified within the area occupied by the tumor.

Birds No. 6 and No. 7, two non-laying hens of heavy breeds (White Rock and cross-bred) were examined to determine the cause of their failure to lay. Both had the appearance of normal hens

and are included in this study because of interesting observations on the oviducts.

No. 6 was in poor flesh and a large palpable mass was present in the abdominal cavity. At autopsy this mass proved to be a concretion of yolk material (Figure 10) situated at a point just above the isthmus of the oviduct. Some scar tissue (Figure 11) was present at the site of the concretion but there was no apparent imperforation. The ovary was normal and ovulatory as evidenced by mature follicles (Figure 12) and marked fatty infiltration of the liver due to the reabsorption of large amounts of yolk material.

No histological examination was conducted.

Bird No. 7, a cross-bred (White Rock x Rhode Island Red), appeared to be in laying condition. It was in good flesh but was not credited with laying in a trap nest record extending over a period of more than a year.

Autopsy showed the ovary to be normal and ovulatory (Figure 13). No vestigial right ovary could be seen. There was extensive fatty infiltration of the liver. The oviducts (Figure 14) were very small; the one on the left was imperforate and the right oviduct was cystic--somewhat larger than commonly seen.

Bird No. 8, a Leghorn hen was examined because of peculiar feather development and enlarged comb and wattles. The hackle, saddle, and tail feathers were of a male type.

Gross examination of the viscera showed nothing significant

The thyroid was enlarged.

Histological examination of the ovary also revealed nothing significant (Figure 15). However, the thyroid gland was goiterous, showing many follicles devoid of colloid (Figure 16). The follicle cells were enlarged and filled with vacuoles. Little, if any parathyroid involvement was seen.

Bird No. 9, a Barred Rock hatched January 17, 1950 and three months old at autopsy was selected from a pen as a young cockerel. Comb development, plumage, and general profile were all of the male type but closer examination revealed the darker barring of the plumage characteristic of the genetic female which does not possess the sex-linked dilution factor.

There was an extensive accumulation of yellowish watery fluid filling the peritoneal cavity and a large lobular tumor 3.5 x 3 x 3.1 cm. was found median and posterior to the intact mass of ovarian tissue. A striking amount of development was seen in the oviduct and the vent resembled that of a laying hen.

Histological examination of intact ovarian tissue showed it to be quite normal with many developing follicles, a few in various stages of atresia, and apparently normal interstitial structures. Sections of the tumor were somewhat confusing in that the definite glandular, cord-like structures of an adenomatous type (Figure 17) could be found only in isolated areas close to the attachment of the tumor to the ovary. Sections from other areas in the tumor more closely resembled a solid

carcinoma with a delicate connective tissue stroma.

Bird No. 10, a White Rock hen was typical of the type of reversal commonly observed in flocks. During her pullet year, egg production was fair with some fertile eggs being produced. However as a hen, only three eggs, none of which were fertile, were produced. At the time of sacrifice, the general profile was male with a large, rough, turgid comb and wattles. Plumage type was essentially "henny". There were no spurs but the voice was male in character.

Autopsy revealed a small growth directly attached to the ovarian mass by a peduncle. The growth measured 1.9 x 1.4 x 0.8 cm. and was quite vascular and dark in color. Nothing abnormal was seen in the condition of the oviduct but the ovary showed no signs of being ovulatory.

Histological examination of the growth showed it to be nonmalignant. An infectious process was evident but diagnosis was not made. The cyst (Figure 18) consisted of epithelial cells with no particular organization contained within a connective tissue capsule. Nests of leucocytes and large clotted areas could be seen throughout the cyst.

DISCUSSION

In the genetic male or female the first trace of the embryonic gonad is a thickening of the peritoneal epithelium in the angle between the mesentery and the Wolffian body. This anlage enlarges to form a ridge covered by peritoneal elements considered to be columnar germinal epithelium from which, in the next stage of development, a number of invaginations are formed; the sex cords of the first proliferation. In the female these invaginations give rise to the medullary cords which after a time abort. In the male these cords form the seminiferous tubules. The primordial germ cells migrate into the epithelium of the germinal ridge and finally into the sex cords where they appear as conspicuous rounded cells. Up to this point sex is not distinguishable but the period of differentiation then begins, and in the cords the cells steadily enlarge until these columns may be regarded as small seminiferous tubules. These tubules contain two types of cells: (a) small cells with oval nuclei arranged immediately beneath the basement membrane, and (b) large cells containing spherical nuclei which represent both primary and secondary cells derived from the previously mentioned small cells. Following this period of differentiation the period of pre-spermatogenesis appears during which Sertoli cells arise from epithelial cells, and the spermatogonia which, after repeated mitoses, give rise to the primary spermatocytes. The final stage in the development of the tubules is then spermatogenesis

in which the secondary spermatocytes are produced.

Figures 19 and 20 show the development of normal testicular tissue during pre-spermatogenesis. The cyclic nature of reproduction in the pheasant displays both the active testicular tissue from late March through the breeding season and the regression of the quiescent period lasting from mid-August to the next spring.

Tumors of the reproductive organs according to Goss (38) rank second in incidence to tumors of the blood forming organs. Tumors of the ovary are most frequently adenocarcinomas.

Figure 4 shows the gross aspect of the abdominal cavity of Bird No. 1. The dark color of the ovary is definitely abnormal and may represent a tumor of the hemangiomatous type. The fact that the bird ovulated during the summer of 1946 indicates that the plumage change took place after the summer moult of 1946. Since no satisfactory material was available for histological examination nothing definite can be said regarding the state of sex reversal in this specimen.

Figure 5 shows the ovary of Bird No. 2 to be quiescent but normal--typical for the time of year when the ovary was obtained. An interesting point is the occurrence of a spur on the right leg. As previously mentioned, this was the criterion on which the examination was based. The normal spur of the female is a small plate-like disk and the incidence of an actual spur integral with the bones of the shank is extremely rare in this

sex (39).

The lore of the female varies from specimen to specimen with considerable coloration often appearing in normal birds (39).

The actual deviation from the normal in this specimen is slight and the examination offers no explanation for the occurrence of the spur.

The histological aspect of the ovary of Bird No. 4 is shown in Figure 8. Here the tumorous mass has not affected the character of the ovarian tissue in general. Developing follicles and supportive tissue are still typically female with no apparent reversal in progress.

The adenocarcinoma in Bird No. 5 was not accompanied by any enlargement of the right vestigial ovary. This compensatory mechanism is often observed in situations where normal ovarian function is impaired by malignant growth (33). Figure 9 shows an area of active proliferation. This specimen never layed due to the imperforate oviduct but possibly had at one time been ovulatory. Secondary sex characters of this specimen were typically female indicating that the tumor had not progressed far enough to impair ovarian function. However, in time this specimen would likely have taken on the altered characters accompanying progressive ovarian atrophy.

Birds 6 and 7 failed to lay eggs because of malformations of the oviducts. In Bird No. 6 scar tissue (Figure 11) present

at a point just above the isthmus indicated a previous injury which explains the massive concretion of yolk material (Figure 10) and subsequent fatty infiltration of the liver.

In Bird No. 7 the left oviduct was small and the condition appeared to be an embryonic failure of the oviduct to develop. Figure 14 shows the malformation of the oviducts. In neither case was there any evidence of sex reversal.

It is difficult to explain the occurrence of male plumage and head furnishings of Bird No. 8 on the basis of the histological examination. However, the fact that thyroid function was somewhat impaired (Figure 16) would account for changes in feather configuration according to Seyle (40). Hypofunction of the thyroid probably does not affect the ovary directly but decreased thyroid activity is accompanied by a general-adaptation-syndrome with a consequent reduction in the concentration of circulating gonadotrophins. This reduction of gonadotrophic activity eventually causes involution of secondary sex characters and subsequent atrophy of the gonads in both the male and the female. Plumage then tends toward the male type.

The condition of the ovary in Bird No. 9 is very similar to that of Bird No. 5 except that the reversal of sex characters is more pronounced. Areas of typical glandular arrangement (Figure 17) and the characteristic accumulation of ascitic fluid indicate that the malignant growth is an adenocarcinoma.

The infectious process involving the ovary of Bird No. 10

is shown in Figure 18. The growth in this case was nonmalignant and caused the arrest of ovulation. Few developing follicles and atresia of a number of follicles could be seen.

Gross and microscopic examinations have shown five pathological conditions of the ovary and one abnormality of the thyroid (goiter). In each case there has been some indication that the secondary sex characters of the specimen were altered but in no case could classic reversal be observed. It is further interesting to note that in the specimens (1,8,9, and 10) where reversal was most pronounced, significant changes in the ovarian tissue could not be observed. Parkes (41) has discussed four similar cases of apparent reversal in which no histological evidence could be found. Lipschutz (42) has also pointed out this fact in work with guinea pigs.

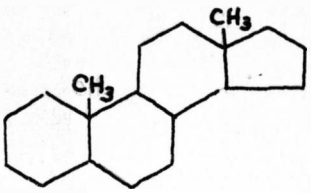
In birds of a dimorphic species the plumage of the mature female is feminized by estrogens elicited by the functional gonad. Androgen therefore would not be expected to influence the plumage of the female directly. However, in the case of the gonadectomized fowl the plumage changes from that of the normal female to a neutral or cock-like pattern. The absence of a functional gonad is then a logical explanation of male plumage in a genetic female. If only the functional left gonad of the female is destroyed the change may be transient with a reversion to plumage of the female type at a subsequent moult. This reversion may be caused by hypertrophy of elements of the vestigial

right ovary. However, absence of a functional gonad or the proliferation of medullary elements of either the functional left or vestigial right gonad may be offered as an explanation of persistence of plumage of the male type.

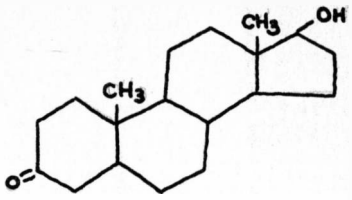
SUMMARY

Three pheasants and six domestic hens were examined to determine the causes for variation in the secondary sex characters.

1. Tumorous growths of ovarian origin were found in one of the pheasants and two of the domestic hens (Nos. 4,5 and 9). No cases of hypertrophy of the right vestigial ovary or medullary proliferation of testicular elements were found.
2. Hypofunction of the thyroid apparently caused involution of the female secondary sex characters of one of the domestic hens (No. 8) and a consequent expression of sex characters of the male type.
3. Two of the domestic hens (Nos. 6 and 7) were found to have abnormalities of the oviduct.
4. An infection of undetermined nature caused the appearance of male head furnishings and voice in one of the domestic hens (No. 10).
5. No apparent cause was found for the variations in the secondary sex characters of two of the pheasants (Nos. 1 and 2).

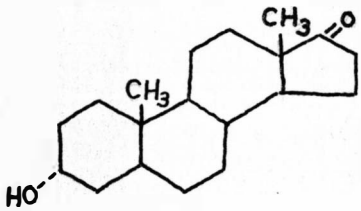


Androstane



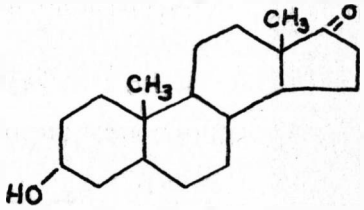
Dihydrotestosterone

(Androstane-17-trans-ol-3-one)



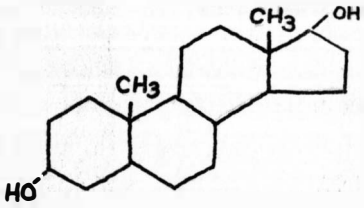
Androsterone

(Androstane-3-cis-ol-17-one)



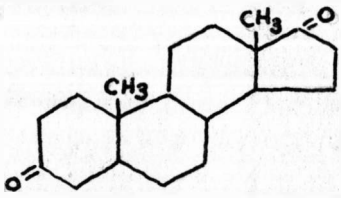
iso-Androsterone

(Androstane-3-trans-ol-17-one)



Androstanediol

(Androstane-3-cis-17-trans-diol)

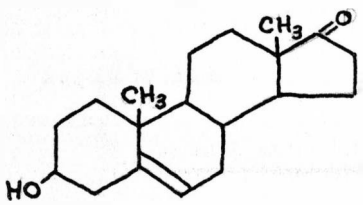


Androstanedione

(Androstane-3:17-dione)

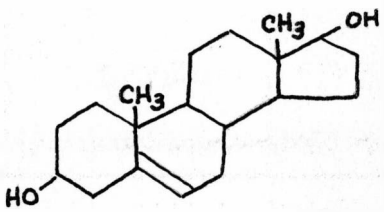
Figure 1.

Chief saturated and unsaturated androgens. (Nomenclature after suggestions by Butenandt and Ruzicka.)



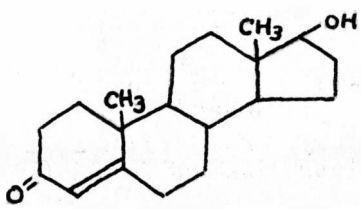
trans-Dehydroandrosterone

(Δ^5-Androstene-3-trans-ol-17-one)



trans-Androstenediol

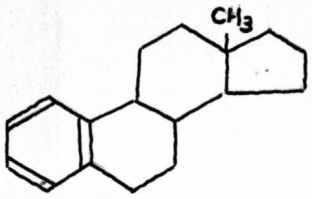
(Δ^5-Androstene-3-trans-17-trans-diol)



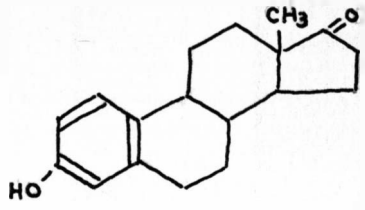
Testosterone

(Δ^4-Androstene-17-trans-ol-3-one)

Figure 1. (continued)

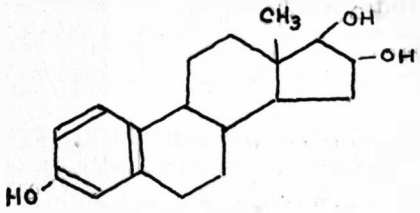


Estratriene



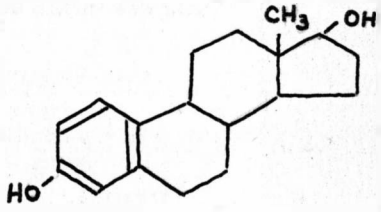
Estrone

(Estratriene-3-ol-17-one)



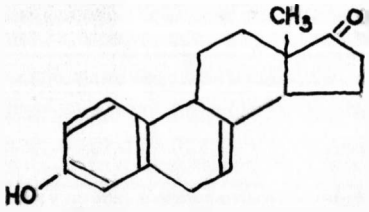
Estriol

(Estratriene-3,16,17-triol)

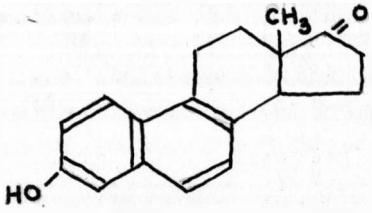


Estradiol

(Estratriene-3,17-diol)



Equilin

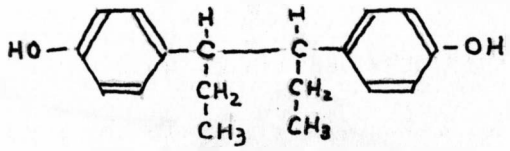


Equilinin

Figure 2.

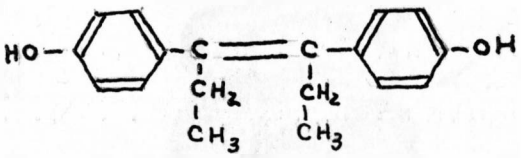
Chief naturally occurring and synthetic estrogens.

(Nomenclature after Adam et al 44.)



Hexestrol

(4,4'-Dihydroxy-7-δ-diphenyl-n-hexane)



Diethylstilbestrol

(4,4'-Dihydroxy-α,β-diethylstilbene)

Figure 2. (continued)

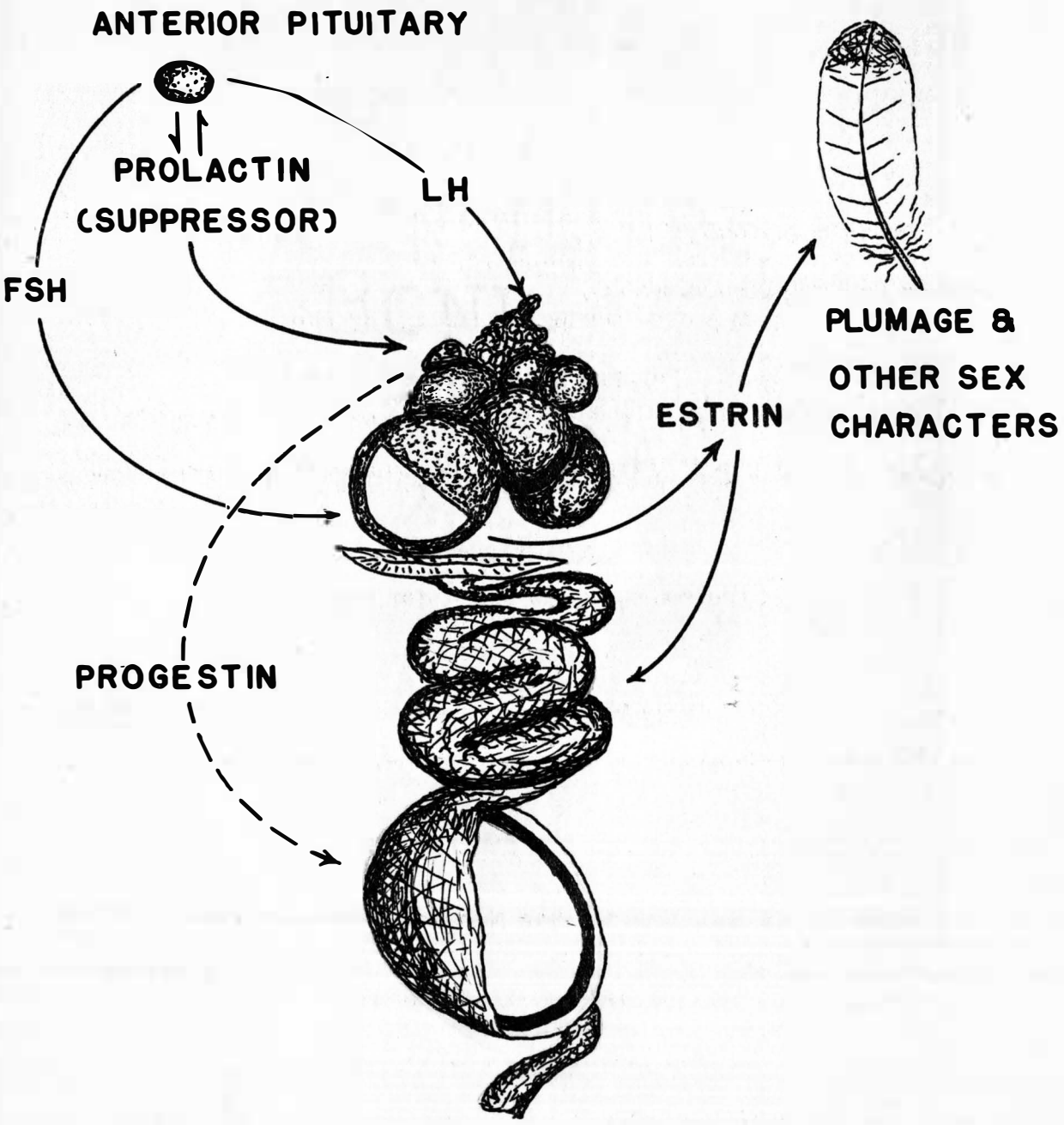


Figure 3.

Schematic diagram of hormonal control of the female secondary sex characteristics and reproduction in the fowl. (Modified after Romanoff and Romanoff 43.)

Figure 4.

Small discolored ovarian mass of specimen no. 1 shown in situ.

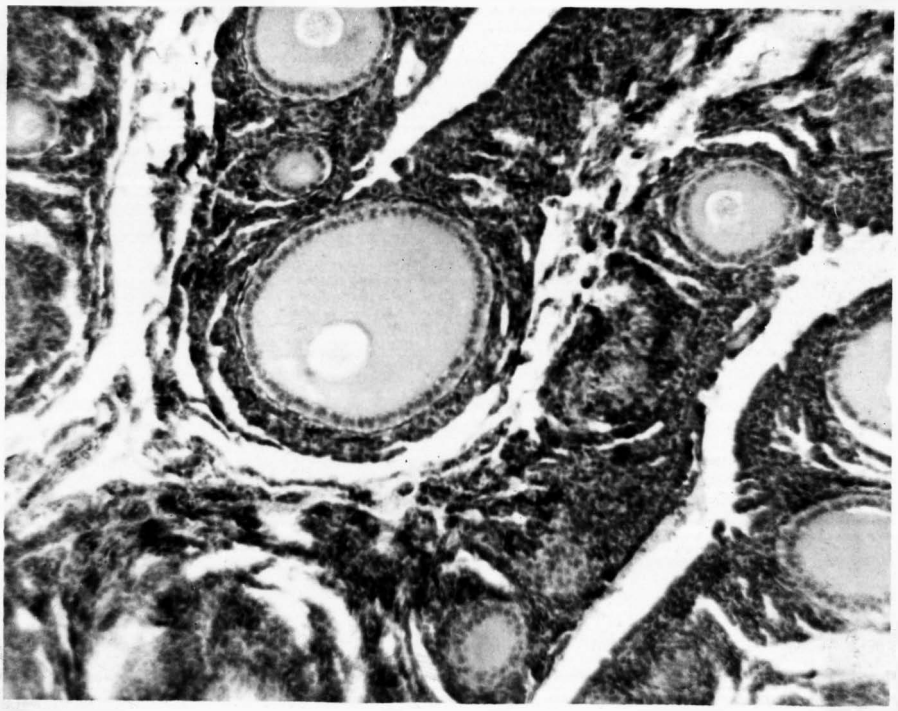


Figure 5.

Ovary of specimen no. 2 showing normally developing follicles and interstitium. Hematoxylin and eosin. X 100.

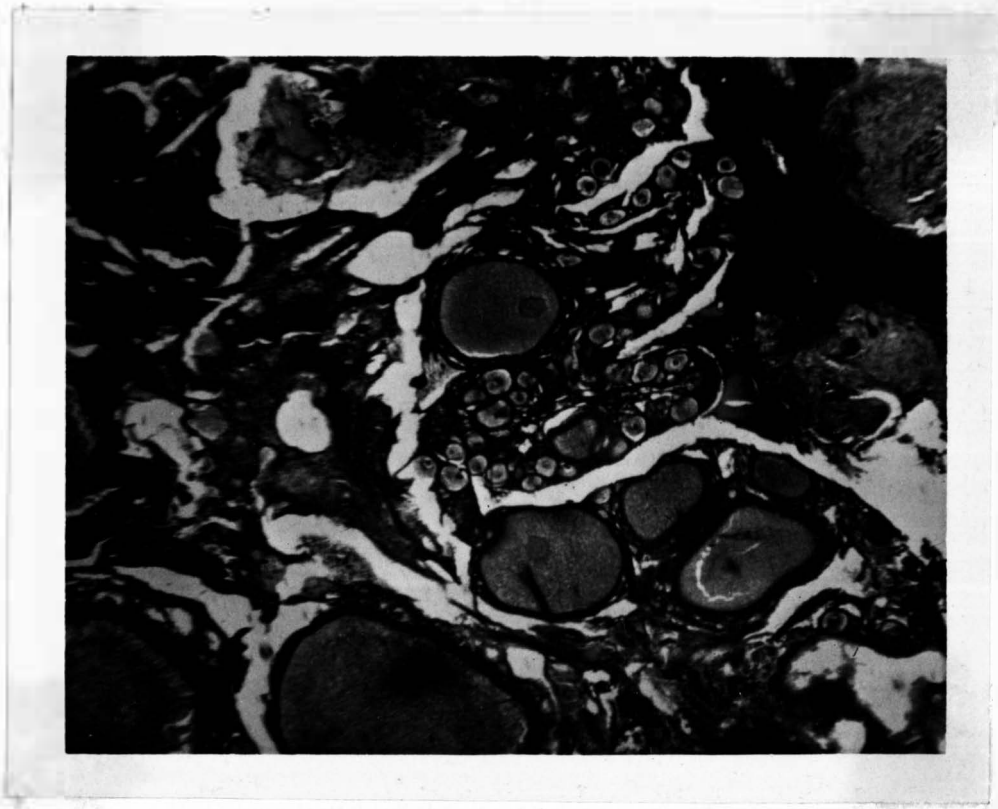


Figure 6.

Normal ovary from specimen no. 3 showing various stages of follicle development. Hematoxylin and eosin. X 42.

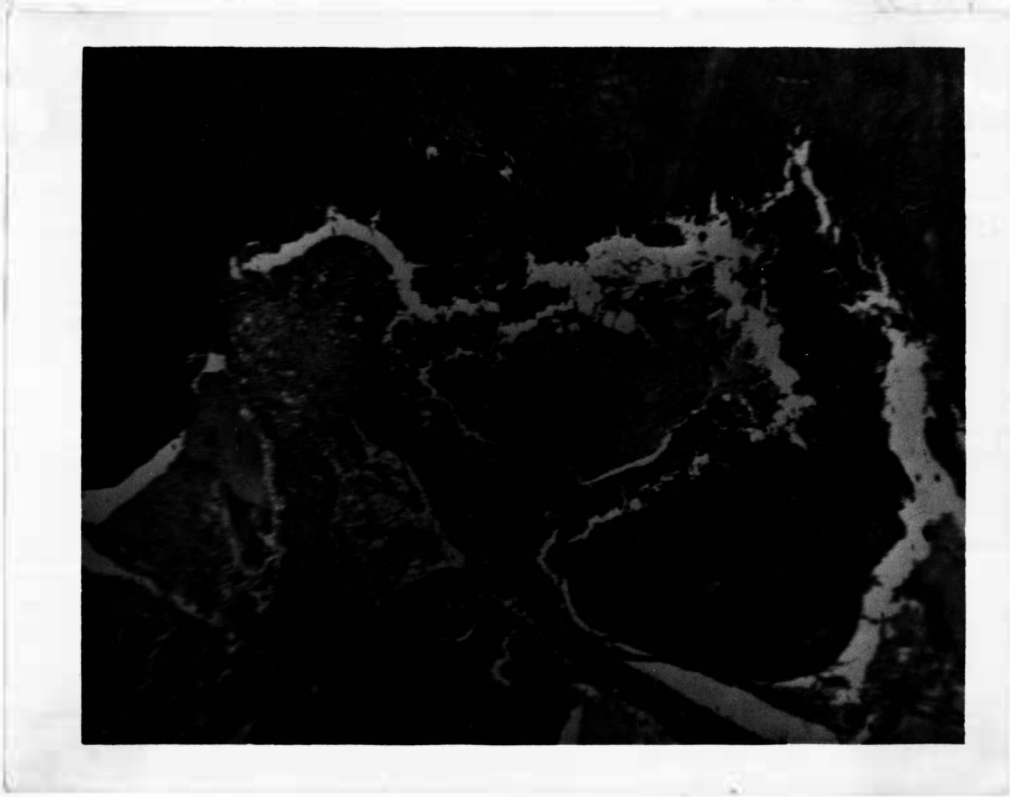


Figure 7.

Hemangioma of the ovary from specimen no. 4 showing large clotted area. Hematoxylin and eosin. X 42.

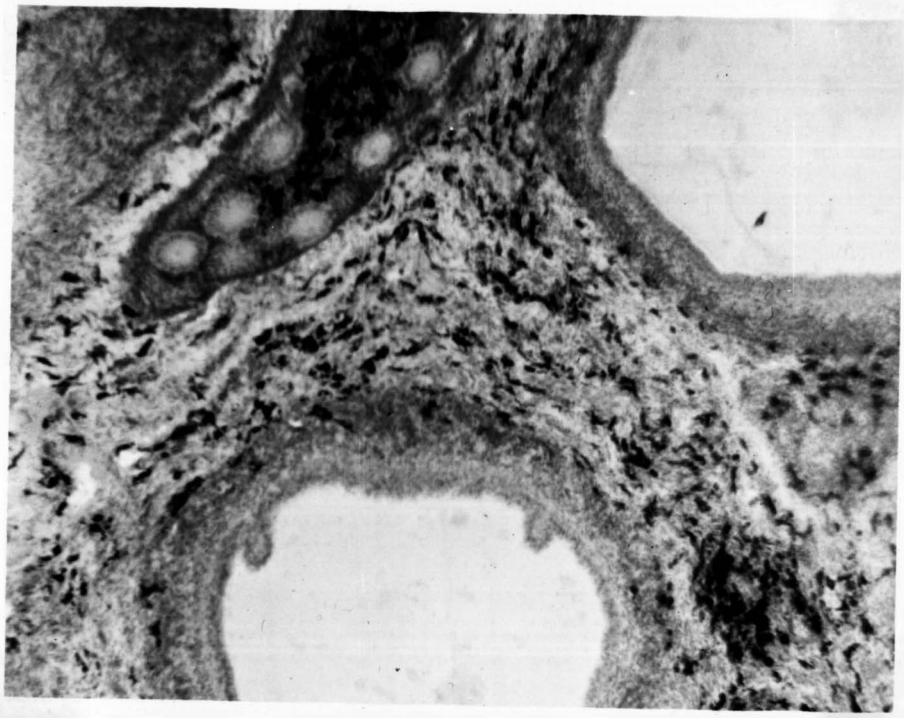


Figure 8.

Ovary of specimen no. 4 showing normally developing follicles. Hematoxylin and eosin. X 100.

Figure 9.

Adenocarcinoma from specimen no. 5 showing mitotic figures of an actively proliferating area. Heidenhain's Iron Hematoxylin. X 450.

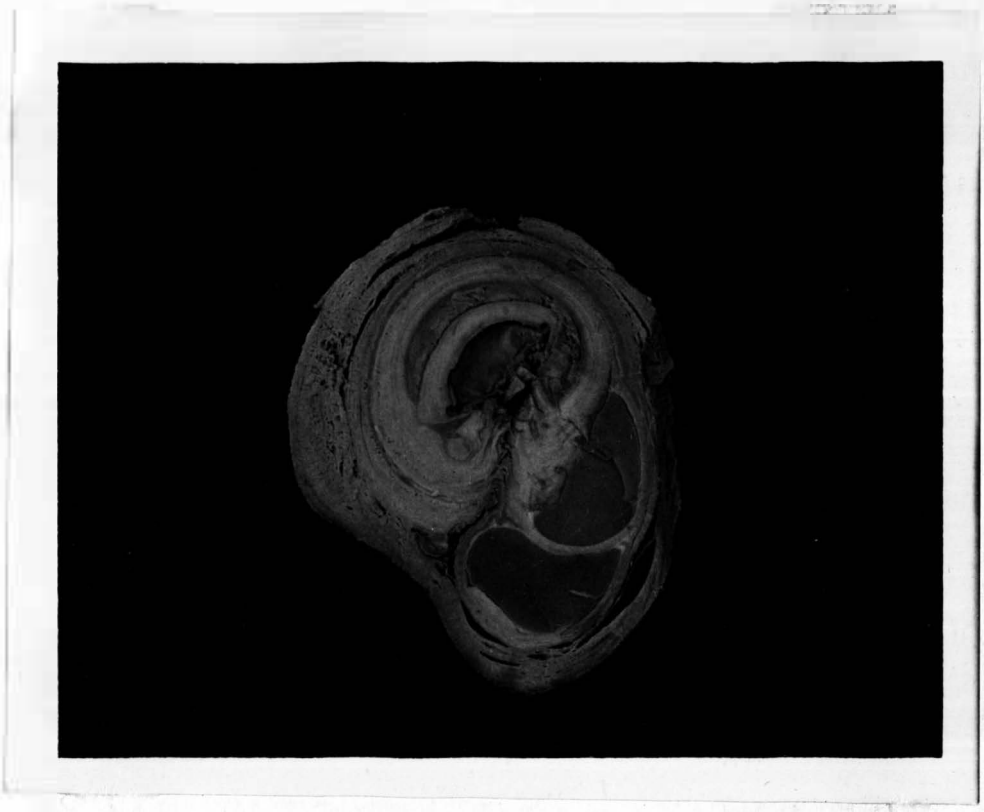


Figure 10.

Cross section of yolk concretion removed from the oviduct of specimen no. 7.

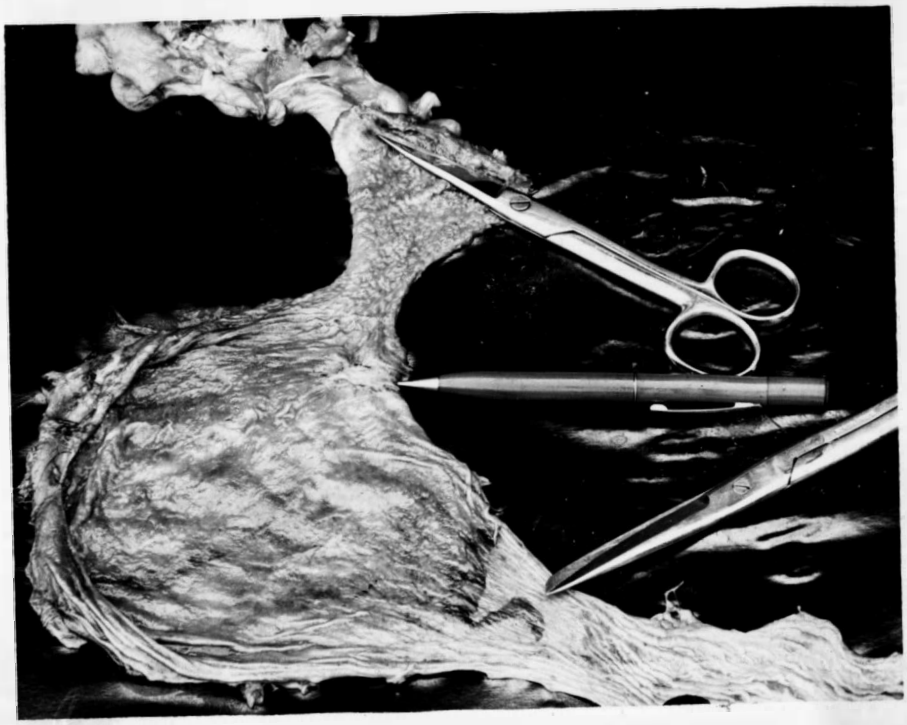


Figure 11.

Dissected oviduct of specimen no. 7. Note scar tissue at points indicated and the distention of the oviduct at the site of the concretion.

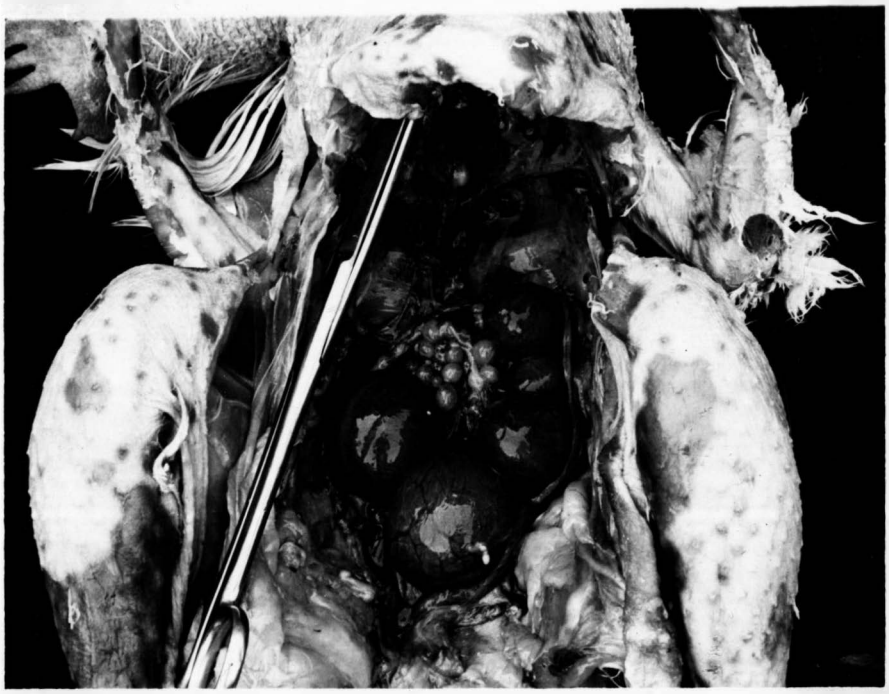


Figure 12.

Ovulatory ovary of specimen no. 6 shown in situ.

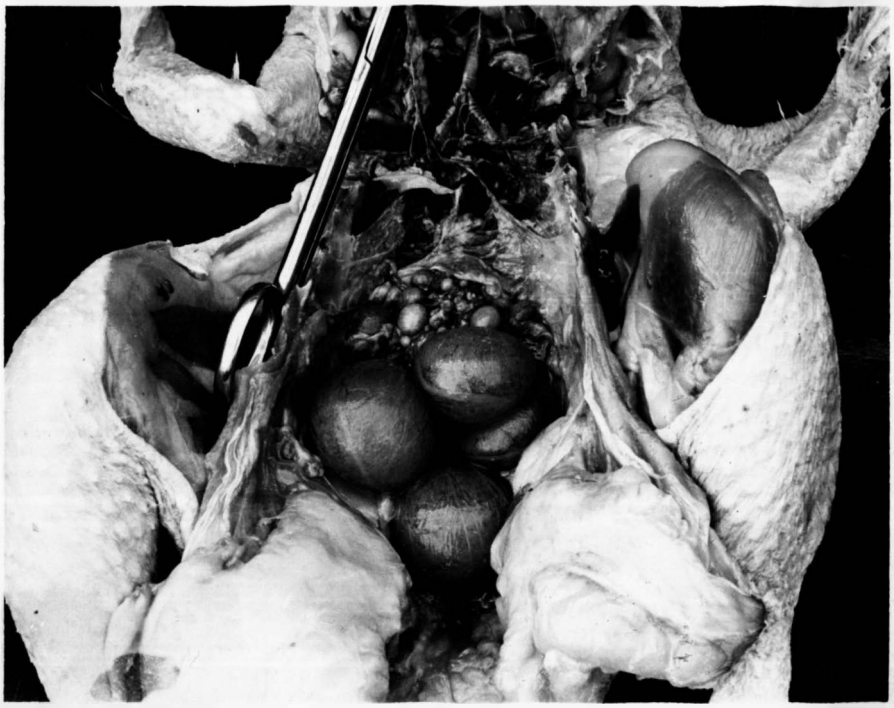


Figure 13.

Ovulatory ovary of specimen no. 7 shown in situ.

Figure 14.

Undeveloped left (indicated) and cystic right oviducts of specimen no. 6.

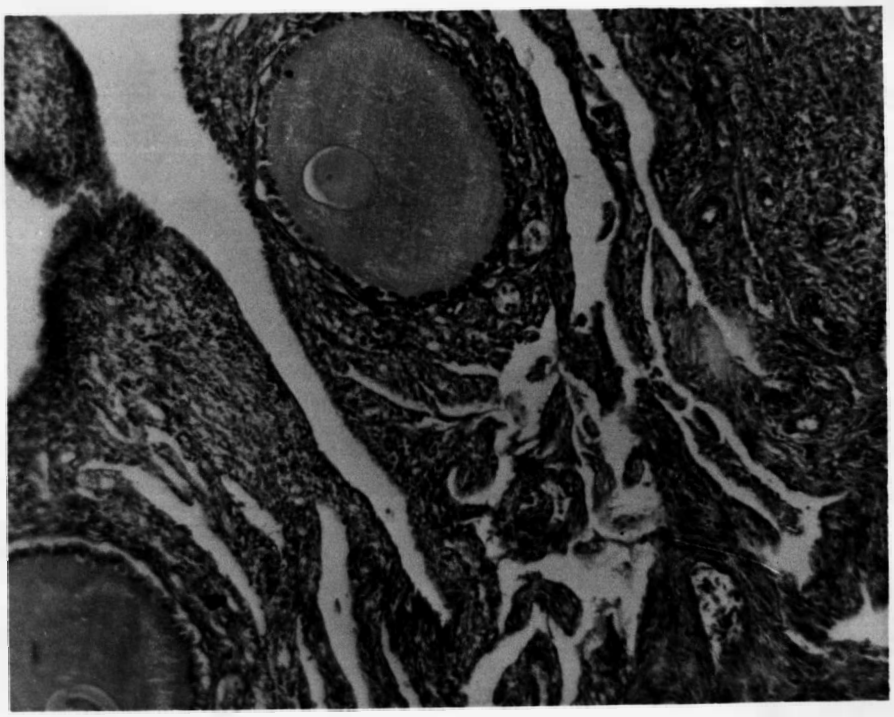


Figure 15.

Ovary of specimen no. 8 showing developing follicles and normal interstitium. Hematoxylin and eosin. X 100.

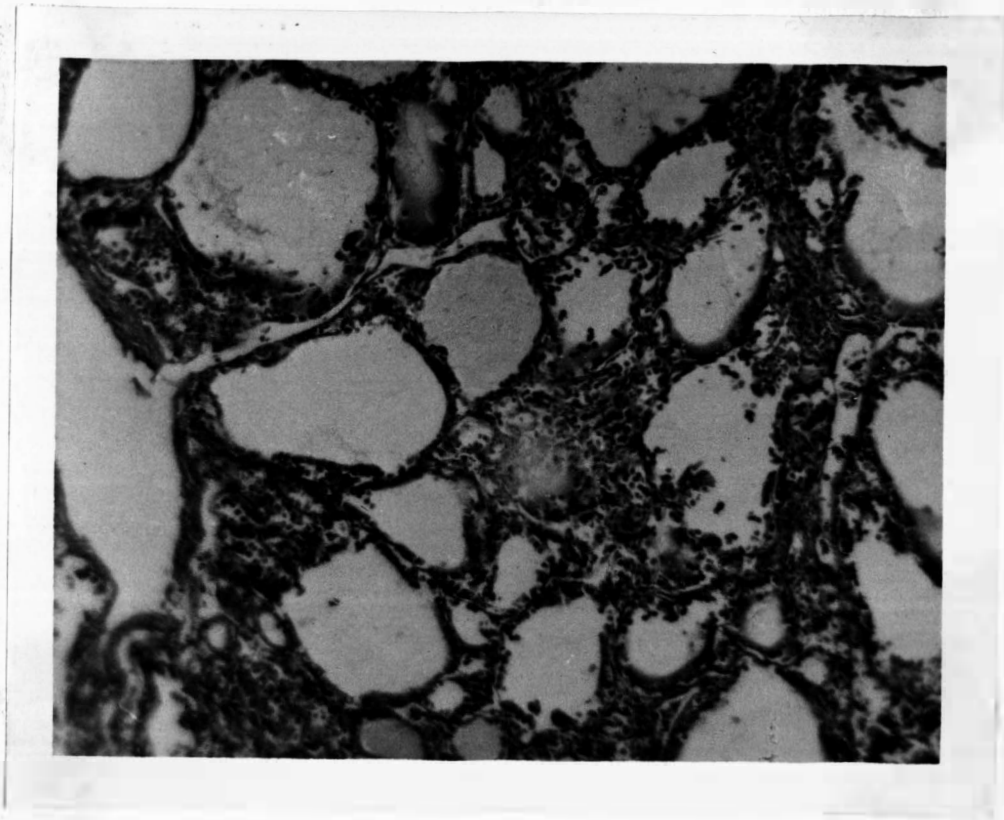


Figure 15.

Thyroid goiter of specimen no. 8 showing hyperplasia and absence of colloid. Hematoxylin and eosin. X 100.

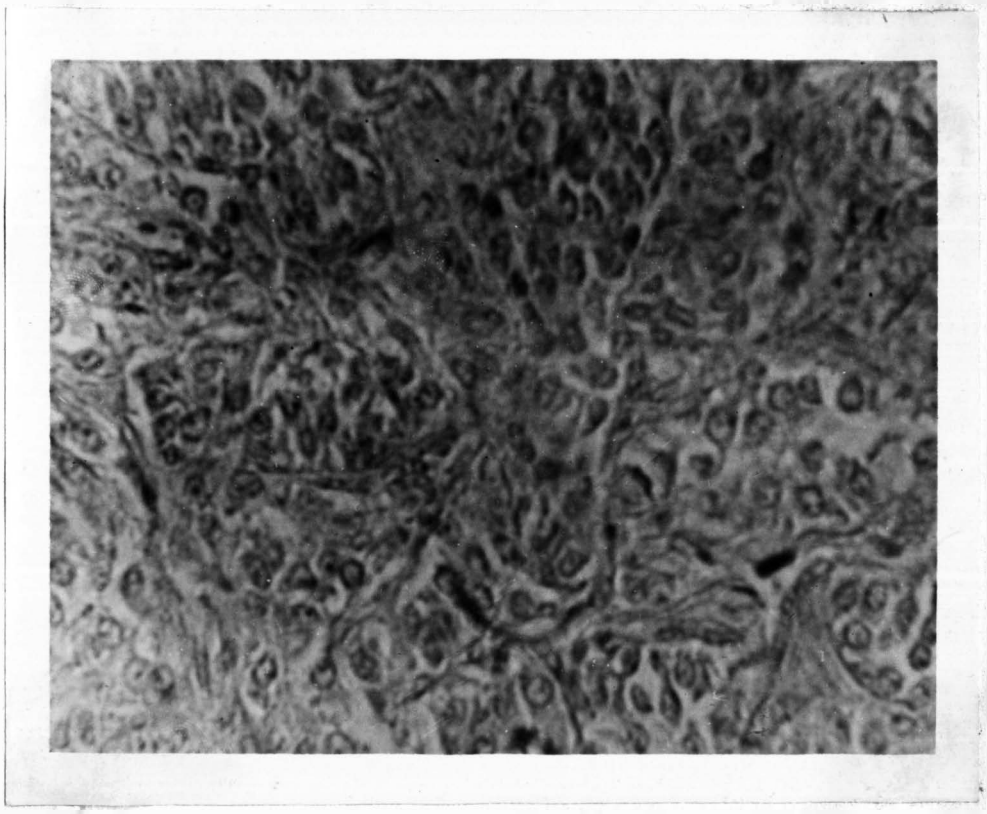


Figure 17.

Adenocarcinoma from specimen no. 9 showing mitotic figures and glandular arrangement of cells. Hematoxylin and eosin. X 450.

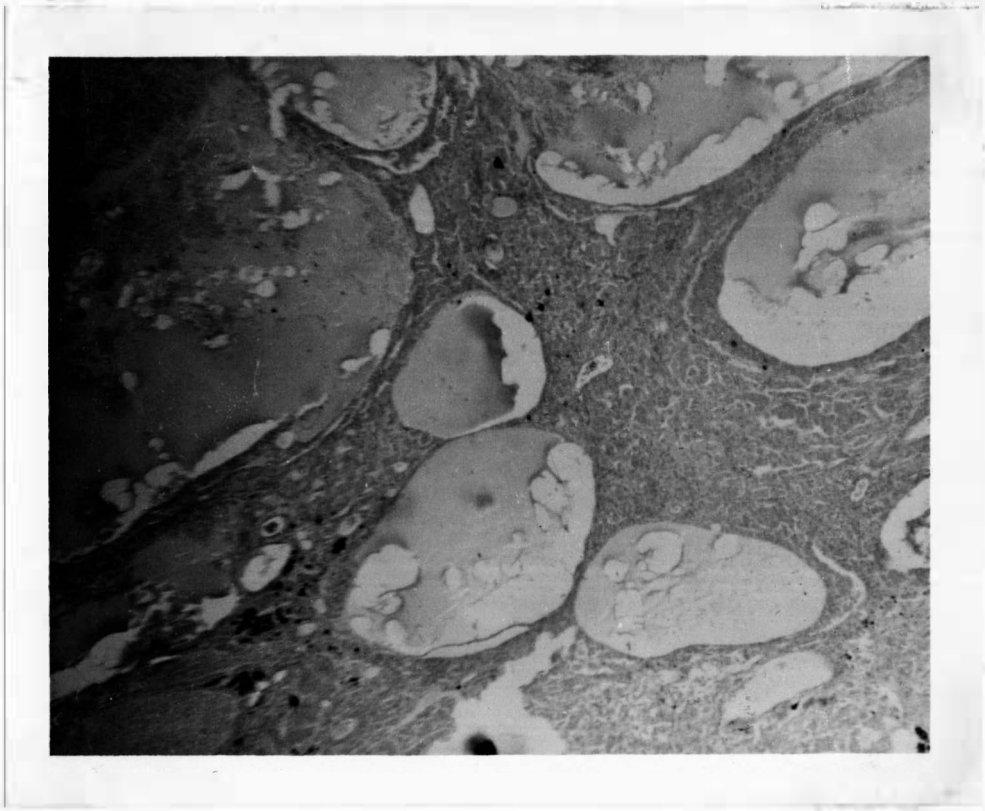


Figure 18.

Ovarian cyst from specimen no. 10 showing epithelial cells and leucocyte nests. Hematoxylin and eosin. X 100.

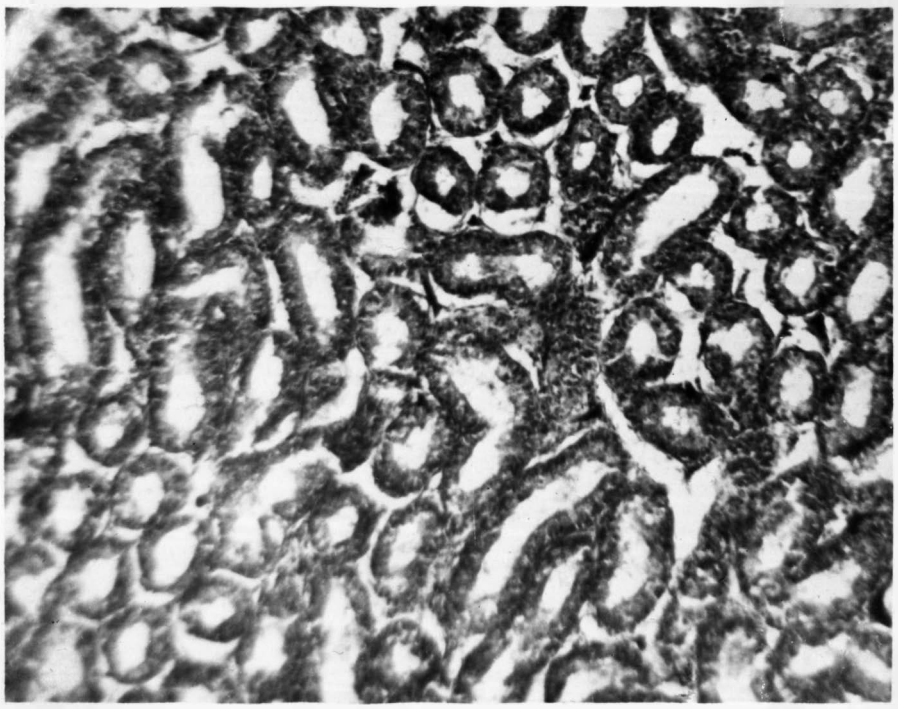


Figure 19.

Inactive testis. Hematoxylin and eosin. X 100.

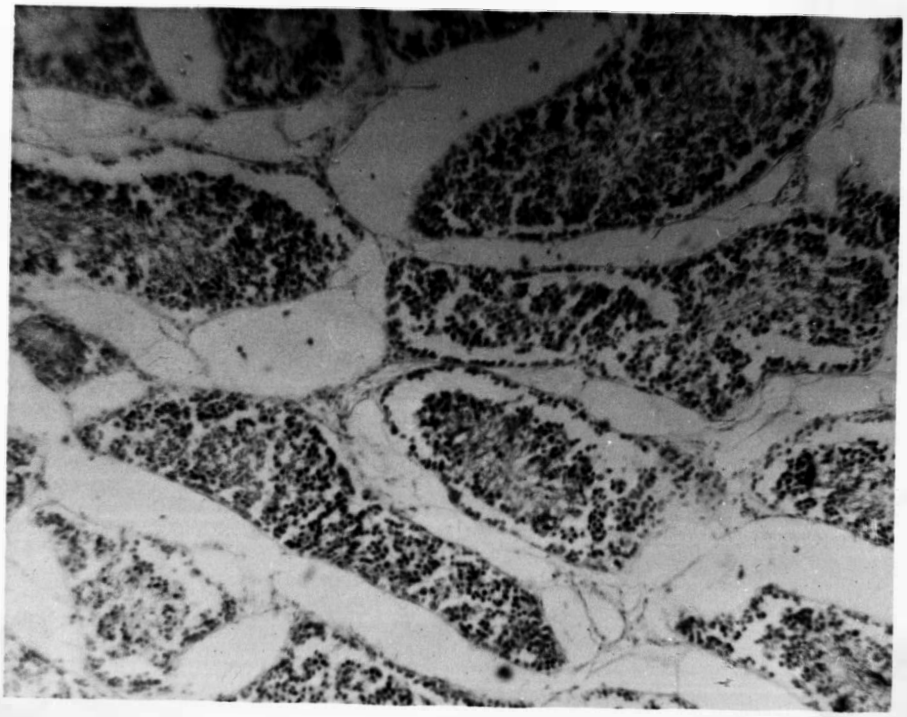


Figure 20.

Testis showing active spermatogenesis. Heidenhain's Iron Hematoxylin. X 100.

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