CONGENITAL DEFECTS OF THE SKIN IN CATTLE

by

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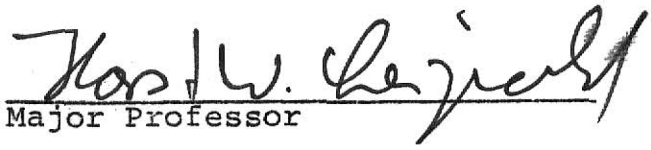
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DEDICATION

To my family, my wife Doris, and children Gustavo, Patricia and Alejandro for their understanding and continual encouragement.

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INTRODUCTION

Inherited disorders of the skin, hair, and adnexa are abnormalities present at birth. In veterinary practice, diseases of skin and its adnexa are of great economical importance and concern with animal breeders using genetic combinations and mutations as an instrument for improving their stock. Genetic recombinations such as inbreeding and crossbreeding are modern tools to obtain good animals, but results may be favorable or unfavorable. It has been established that genes are genetic units and they work as biochemical enzymes or controlling units of enzymes. Any aberration or dislocation of an enzymatic process will enhance or diminish, and also prevent a biochemical process at the molecular level.

One of the important inherited disorders of skin is hairlessness. It is rarely investigated and classification is often based on gross morphology, followed by long-standing terminology, and lacks the most elementary histological support.

Classification of skin diseases is difficult since the basic pathogenic mechanisms and pathophysiology are unknown. Discussion of each skin disorder must be based on its clinical signs and supported by laboratory studies.

This research was selected to index the pathogenesis of one skin genetic disease and the histopathological changes of other skin genetic diseases that have similar clinical signs.

The objectives of this project were to study:

1. The pathogenesis of congenital hypotrichosis in Hereford cattle;

2. The pathological changes of congenital alopecia in Hereford, Simmental, Angus, Pinzgauer, and Holstein calves; and

3. The pathological changes of alopecia and dermoid inclusion cyst in a Simmental calf.

I: REVIEW OF THE LITERATURE

Inherited disorders of skin, hairs, and adnexa are abnormalities present at birth. Embryologically, skin is developed from both ectoderm and mesoderm. The epithelial covering and its adnexa, hair follicles, sweat gland, and sebaceous gland originate from ectoderm. The connective tissue and vascular elements are contributed by mesoderm (Dellmann and Brown, 1976). This particular growth pattern of skin, from two germ layers, and its extensive and variable width, makes it an easy target for many diseases. The hair, a flexible, keratinized structure produced by hair follicle, is important in congenital diseases and it forms part of specific disorders in bovine skin such as congenital hypotrichosis and congenital alopecia (Kobori and Montagna, 1975). The hair follicle is formed by growth of ectoderm into the underlying mesoderm (Dellman and Brown, 1976). Follicles are formed in the skin of the mid-lateral region of the bovine fetal trunk between the 77th and 166th days of gestation (Lyne et al., 1959; Lyne and Heideman, 1959). Each hair follicle is associated with an apocrine gland as well as a sebaceous gland, and an arrector pili muscle. Similar studies on the normal development of bovine skin have been described by Carter and Dowling (1954). All hair follicles were homologous with the primary follicle of sheep; no secondary follicles were observed as are recognized in sheep (Hardy and Lyne, 1956); hence there was no follicle group comparable in its complexity to that in sheep and many other mammalian species (Carter and Dowling, 1954).

Barman et al. (1965) studied the biological characteristics of human scalp on the basis of sex and age, and normal values were established. The same author reported that the number of hair in the telogenic phase increases with age, hence the density of population decreases with age. Biological and physiological characteristics of scalp hair is necessary for interpreting the pathology of hairs. The pathology of hair is important to animal owners, breeders, and veterinarians, as affected animals are susceptible to environmental changes and infections, do not conform to breed standard, and lose their aesthetic value (Jayasekara, 1979). According to Lyne and Heideman (1959), there is only one type of hair follicle in cattle, similar to the primary follicle of sheep. Since all follicles were associated with sweat glands, sebaceous glands, and arrector pili muscles, no attempt was made to classify them into primary and secondary types as described in sheep and other mammals.

Hair is composed of three cylinders of cells, each one cell thick, which together constitute the inner root sheath, that arise from the peripheral portion of the undifferentiated matrix (Kral and Schwartzman, 1964; Montagna and Parakkal, 1974; Lyne and Short, 1965; Birbeck and Mercer, 1957c). The characteristic intercellular product of all three layers is trichohyaline (Birbeck and Mercer, 1957c; Happey and Johnson, 1961). Jayasekara et al. (1979) found in the Huxley's cell of the hair follicle from a Hereford calf, spheroidal microdroplets, pleomorphic material, appeared to be electron dense

trichohyaline granules. Protein is the principal constituent of the trichohyaline granules (Roger, 1959). Trichohyaline is initially formed as amorphous droplets that subsequently develops into the birefrigent form (Birbeck and Mercer, 1959). The trichohyalin droplets are stained with Altmann's acid fuchsin methyl green where they react with a brilliant fuchsinphilia (Montagna and Parakkal, 1974). The first sign of keratinization is seen at the level of the upper bulb in the Henle layer, with the formation of trichohyalin granules. Inside the Henle layer, the Huxley layer began the same process of keratinization and formed the inner layers of the hair cone (Hashimoto, 1970; Butcher, 1951).

The normal hair cycle in cattle was well described by Neurand et al. (1980). They observed the typical hair phases of anagen, catagen, and telogen. Lyne and Heideman (1959) observed that the eight stages of hair follicle development in mammalian skin as described by Hardy and Lyne (1956) were applicable to bovine skin, and added two more stages to represent the resting phases of follicle growth. Anagen, the growth phase of the hair cycle, is subdivided into five steps; anagen I, cells start to mitotic division; anagen II, growing follicle surrounding of papilla, and there is hair bulb; anagen III, bulb matrix dendritic pigment cells; anagen IV, matrix cortical medullary area produce at this point the hair, and reach the level of the sebaceous gland; and anagen V, the hair appears in the epidermis and the inner and outer root sheaths are formed. There is full differentiating of

shape, length, and form of a bovine typical hair. Catagen is recognized by three subdivisions; catagen I, as a disdifferentiation step in the hair cycle, characterized by reduction of mitotic activity. The hair is small, the inner root sheath does not produce anymore, and thickness of the hair decreases. Finally, the formation of the medulla is arrested and a club hair is formed. In catagen II, the keratogen zone of cells becomes swollen, the hair follicle is shorter than normal and looks like a ribbon of cells. In catagen III, the phase of exposure, the hair emerges, but the lower part of the bulb degenerates cells remaining from the secondary hair follicle. Telogen, the resting phase of the hair follicle, is followed by anagen (Neurand et al., 1980; Rook and Walton, 1965; Montagna and Chase, 1956; Kligman, 1959; Butcher, 1962).

Comparative studies revealed changes in human hair follicle catagen. The highlights of human catagen are involution of the bulb, epithelial strand formation, the escape of the papilla, and the signal hallmark, the enormous thickening and folding of the vitreous membrane. The precipitation of catagen is one of the most characteristic ways in which the follicles react to severe stress (Kligman, 1959). The mild dusturbance due to plucking of club hair is less traumatic than vigorous massage, and can induce activity if enough hairs are plucked, but this does not directly explain spontaneous waves or islands (Chase, 1958; Chase and Eaton, 1959-60).

The hair changes because of photoperiodicity. It is a polygenic change, and it is also a synchronized hair follicle growth, parallel to the vertebral column. Ventral areas change the hair last (Neurand et al., 1980). The inner root sheath is ultimately discharged into the pilosebaceous canal, due to chemical changes that culminate in reabsorption, dissipation or exfoliation within the pilary canal. The main function of the inner root sheath is to shape or contour the hair shaft since as it is the first structure to keratinize in the follicle (Hashimoto, 1970). Inner root sheath forms the outer boundaries through which the developing hair shaft is funneled during its formation (Montagna and Parakkal, 1974).

Bovine hypotrichosis is a congenital disease characterized by partial or complete absence of hair coat, with or without accompanying development defects (Leipold et al., 1972; Blood and Henderson, 1974). Affected animals are not in any event retained for breeding because their hair coat do not conform to breed characteristics (Jayasekara et al., 1979). The degree of hairlessness varied with individuals. The histological description of hypotrichotic skin by Becker et al. (1963) included poorly differentiated hair follicles that were shallow and rarely extended beyond the sebaceous gland. Hutt (1963) described six forms of congenital hypotrichosis in cattle. Shand and Young (1964) reported 31 hairless calves with abnormal thyroids in a Friesian herd. Eldridge and Atkeson (1953) reported streaked hairlessness

in Holstein-Friesian cattle due to a sex-linked, semidominant lethal gene.

Hypotrichosis has resulted from hypophyseal hypoplasia in Guernsey and Jersey cattle (Blood and Henderson, 1974). Shand and Young (1964) described congenital alopecia in 3 Friesian calves that had hyperplastic thyroids. Welch (1917) associated hairlessness among calves with iodine deficiency. Craft and Blizzard (1934) suggested that semihairlessness in polled Hereford herd resulted from a hereditary trait. Kidwell and Guilbert (1950) reported a similar condition and suggested that it was conditioned by a single autosomal recessive gene. Hypotrichosis in Jersey cattle as an inherited lethal condition, possibly caused by a simple, recessive single gene, was reported by Wipprecht and Horlacher (1935). Regan et al. (1935) attributed the same condition to a sublethal genetic defect. McGavin and Alexander (1961) reported a case of viable hypotrichosis in a Jersey calf.

Hypotrichosis congenita has been established as a condition caused by a recessive lethal gene in Swedish Holstein-Friesian cattle (Mohr and Wriedt, 1928). Young (1953) documented various degrees of hairlessness among grade Herefords in Australia and Jayasekara et al. (1979) describe pathological changes in congenital hypotrichosis in Hereford cattle. Holmes and Young (1954) described an alopecia condition which developed in 2 Friesian herds. Congenital alopecia has been reported in Pinzgauer cattle (Pirchner and Grunberg, 1970).

Histological and ultrastructural studies of congenital hypotrichosis of Hereford cattle, suggested that conversion of arginine-trichohyalin to citruline-trichohyalin in the hair follicle cells is impaired and results in arrest of the hair shaft development (Jayasekara et al., 1979). Other reports described a skin condition related to hypotrichosis, however, it was secondary to another skin disorder.

Several problems remain to be solved. The fact that particular changes in the congenital hypotrichosis have been solved does not mean that the problem has been solved. Jayasekara et al. (1979) suggested that degenerative changes in Huxley's layer appeared to be the key in the developmental hypotrichosis lesions. Further studies by Butcher (1951); Chase and Eaton (1956-60); and Chase (1958) reported that plucking hairs stimulated activity in the hair germ. However, additional studies related to the function of the complex pituitary-thyroid-adrenal glands and their hormones interaction are required.

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II. PATHOGENESIS OF CONGENITAL HYPOTRICHOSIS IN HEREFORD CATTLE

INTRODUCTION

Complete or partial absence of hair has been reported in most domestic animals, wildlife, and man (Becker et al., 1963; Craft and Blizzard, 1934; Huston et al., 1977, 1978; Hutt and Saunders, 1953; Hutt, 1963; Jayasekara et al., 1978; Leipold et al., 1972; Saperstein et al., 1977).

Congenital hypotrichosis in Horned and Polled Hereford cattle causes concern for the animals' health because it permits entry of bacteria or fungus to produce infection of the skin, dermatitis and dermatomycosis respectively (Leipold et al., 1972; Young, 1953; Schleger et al., 1967). In addition, the affected calves are exposed to inclement weather. This study was designed to study additional field cases of congenital hypotrichosis in Hereford cattle and to study its pathogenesis by using skin biopsies.

MATERIALS AND METHODS

Twenty-two Hereford calves with congenital hypotrichosis ranging in age from 1 day to 1 year were submitted from mid-western ranches in Kansas and Nebraska for gross and histo-pathological studies. The animals were euthanized and were subjected to a detailed, standardized necropsy. Sections of skin were taken from head, neck, shoulder, flancs, tail and legs. Tissue samples were taken from pituitary, adrenals, pancreas and thyroid; thymus, heart, lung, spleen, and liver. These tissue samples were preserved in 10 percent neutral, buffered formalin, trimmed, processed routinely,

and sectioned longitudinally and transversally 6 microns thick. Tissue sections were stained with hematoxylin and eosin (H&E), periodical acid-Schiff and diastase periodic acid-Schiff, Altmann's acid fuchsin-methyl green, Perl's Prussian blue, toluidine blue, and Masson's trichrome (Table 1).

Skin biopsies were taken surgically, at 7-day intervals from one 7 months old, female hypotrychotic Hereford, and a normal-skinned control calf, five months old normal skinned female Hereford. At the beginning of the experiment the hair was plucked over the shoulders, thorax and flanc (Plate 1).

RESULTS

GROSS FINDINGS:

Twenty-two Hereford calves affected with hypotrichosis, examined grossly, and found to have hair defects ranging from slightly affected over moderately, to severely affected (Figures 1-10). Secondary changes such as dermatomycosis, dermatitis, and sunburn were observed in calves affected with hypotrichosis. One calf had an extremely weak skin that was easily torn; another had lacerations and erosion in the skin.

Skin of affected animals was thin, pliable, and smooth in some areas, with fewer hairs per unit area. The hairs broke easily and they could be plucked easily. Scanty pelage was most evident in ventral areas, or flanks, ears, around eyes, and thorax. Some calves presented sparse

pelage on ears, around prepuce and mammary gland; axillary areas; and switch of the tail. The hair in these areas was easily epilated, thin, and undulated; in contrast, some hair from the face on very young affected animals was extremely curled.

MICROSCOPIC FINDINGS:

Field Cases: In all twenty-two cases studied, significant variations were found in the skin. The cycle of the disease was not clear in that the animals were necropsied and their skin studied under different climatological conditions. Among all lesions collected we found the classical lesion in the anagen, catagen and telogen phases (Table 2).

Anagen: Hairs in this phase showed prominent histological changes in the hair follicle, mainly in the area between the critical level of the hair follicle and the entrance of the sebaceous duct. The inner root sheath (Huxley's layer) included droplets of *spheroid*, that were semitranslucent, almost pink with dark pink borders (Figures 11-12). The Altmann's acid fuchsin methyl green reaction was strongly positive in that the pleomorphic material was stained brilliantly by fuchsinophilia (Figures 15, 16 and 18). These spherical masses (related to trichohyaline granules) were extensive in the region between the inner and outer hair sheath. Other changes seen in the anagen phase were vacuolization and necrosis in the area of Huxley's and Henle's layer; also the entire hair medulla was devoid of cells and vacuoles were present (Figures 11-14 and 17).

Catagen: In this phase hairs became smaller, and only a few microdroplets of trichohyaline were present in a ratio 1:20. Those hairs containing trichohyaline granules belonged to the anagen phase as a remnant (Figures 19 and 20). Most of the hairs in this phase had Altmann's stain negative root sheath (Figure 21). The hairs showed decreased thickening. Some club hairs were observed with moderate or negative reaction with Altmann's stain. Others showed a few trichohyaline granules belonging to the anagen phase.
Telogen: Hairs in this phase were trichohyaline negative. No eosinophilic droplets were seen in either H&E or Altmann's stains (Figures 22 and 23).

In some sections, hairs were limited to the bulb, and club hairs were trapped in follicles occluded by keratin plugs. Longitudinal and cross sections of the hairs showed moderate-to-severe changes of the inner root sheath, Huxley's layer of cells.

Biopsy case: Skin samples of this 6-month-old Hereford affected with hypotrichosis were studied for two months in winter and two months in summer to follow the hair cycle (Table 2).
Anagen: The Huxley's layer of cells became degenerative and arrested the differentiation of cells in the inner root sheath. The upper part of the hair follicle, known as a critical level, started mitotic activity to form each layer of the hair shaft. As the phase progressed, changes in the Huxley's layer became more prominent, and microdroplets of

trichohyalin were located between the sebaceous gland canal and the hair follicle. The Altmann's stain was strongly positive in that the spheroid, semitranslucent, pleomorphic material was stained brilliantly by fuchsinophilia.

Catagen: Mitotic activity was reduced and the inner root sheath produced no trichohyaline granules. However, some microdroplets were still present of the remnant anagen. Some club hairs were present with moderate to negative fuchsinophilia in the inner root sheath.

Telogen: Trichohyaline granules typically were absent in the telogen phase which is the resting part of the hair cycle.

In both, field cases and biopsy material, epidermal thickness varied and usually the epidermis was only 2 or 3 layers thick. The hair shafts were fragmented and contours of the hair follicles were abnormal. Hairs in the anagen phase showed prominent histological changes in the hair follicle, mainly in the area between the critical level of the hair follicle and the entrance of the sebaceous ducts.

In the skin section of all 22 calves, many changes were observed in the inner root sheath. Huxley's layers formed droplets that were spheroid, semitranslucent, almost pink with dark-pink borders. The Altmann's acid fuchsin methyl green reaction was strangely positive in that the pleomorphic material was stained brilliantly by fuchsinophilia. These spherical masses, related to trichohyalin granules, were extensive in the region between the inner and outer hair

sheaths. Vacuolization and necrosis were presented in the area of Huxley's and Henle's layers, the hair medulla was devoid of cells and vacuoles were present. Hairs in the catagen phase presented only a few microdroplets of trichohyalin in a ratio of 1:20. Other hairs in catagen phase were negative for trichohyaline granules. In the catagen phase hairs became smaller and the inner root sheath appeared to be negative with Altmann's stain. Some pleomorphic eosinophilic granules, however, were present as a remnant of the anagen phase. The hair showed decrease of the thickening. Some club hairs reacted moderately or negatively with Altmann's stain. Others showed a few trichohyalin granules belonging to the anagen phase. Hairs in the telogen phase were trichohyalin negative. No eosinophilic droplets were seen in either H&E or Altmann's stain.

In some sections, hairs were limited to the bulb, and club hairs were trapped in follicles occluded by keratin plugs. Longitudinal and cross sections of the hairs showed moderate to severe changes of the inner root sheath, Huxley's layer of cells.

Sweat glands were dilated. Arrector muscle pili were reduced in number and not associated with the pilious-sebaceous apparatus. Masson's trichromic stain was done to study the arrector pili muscle and their association with the hair shaft. The collagen seemed to be essentially normal microscopically.

Five calves had histological evidence of infectious dermatitis and dermatomycosis.

Histopathological studies of the endocrine glands such as thyroid, parathyroid, pituitary, adrenal, and thymus revealed no significant pathologic lesions.

DISCUSSION

Normal development of bovine skin has been reported by Carter and Dowling (1954); Lyne and Heideman (1954). Each hair follicle in cattle is associated with an appocrine gland, sebaceous gland, as well as arrector pili muscle. According to Lyne and Heideman (1959), in the bovine there is only one type of follicle. We made no attempt to classify them into primary and secondary types of those described in sheep and other mammals. Neurand et al. (1980) reviewed the normal hair cycle in the bovine, observing the three typical stages of anagen, catagen, and telogen; and subdivided of each phase.

The embryologic development of the hair follicle begins approximately with the 77th day of gestation and reaches stage 8 of the cycle in almost 126 days (Hardy and Lyne, 1956). Full complement of hair follicles is finished at about 200 days gestation. In cattle, a breed variation is observed in the stage of development of hair follicles (Goldsberry and Calhoun, 1959).

Correlation in both shape and size exists between the hair and the dermal papilla when various types of hairs are

compared (Straile, 1965). Studies by Straile (1965), indicated that the internal root sheath, Huxley's layer, acts as a depository cell in excess of those used in the follicle bulb for the differentiation of the hair. Besides, Huxley's layer and the entire inner root sheath structurally complement the shape and size portion of the hair and thus maintains the core of hardness during the hair formation.

McGavin and Alexander (1961) reported a case of viable hypotrichosis in Jersey cattle, and Wipprecht and Horlacher (1935) reported an inherited lethal condition by a recessive simple gene in Jersey cattle. Hutt (1963) described 6 forms of congenital bovine hypotrichosis. Hairless was also reported by Shand and Young (1964) with abnormal thyroid glands in calves in Friesian herd. Hutt and Saunders (1953) and Becker et al. (1963) described the hairless condition in bovine as non-lethal. Craft and Blizzard (1934) suggested that semihairlessness in a grade Polled Hereford herd resulted from a hereditary trait.

Congenital hypotrichosis was due to homozygosity of a recessive lethal gene in Swedish Holstein-Friesian cattle (Mohr and Wriedt, 1928). Young (1953) documented various degrees of hairlessness in Hereford cattle in Australia. Schleger et al. (1967) reported hypotrichosis in Jersey cattle. Blood and Henderson (1974) reported hypotrichosis as a result of hypopheseal hypoplasia in Guernsey and Jersey cattle, and Welch (1917) associated hairlessness among calves in Montana with iodine deficiency.

Histological description of congenital hypotrichosis included poorly differentiated hair follicles (Becker et al., 1963). Mohr and Wriedt (1928) established that histological criteria corresponded to an early embryological stage in normal development. This criteria appeared to be typical for description for congenital alopecia instead of congenital hypotrichosis. Hypotrichotic calves had decreased follicle density, sweat gland, reduced hair diameter, sweat gland size and skin thickness (Schleger et al., 1967). Jayasekara et al. (1979) reported degenerative changes in Huxley's layer and suggested that the changes seemed to be the key to the development of hypotrichotic lesions.

In bovine hypotrichosis as a congenital condition characterized by partial or complete absence of hair, dramatic changes occur in the inner root sheath (Huxley's layer). This condition makes the affected calves susceptible to weather condition and dermal infections. In addition, hypotrichosis animals produce a considerable economical loss and are useless for breeding purposes because their hair coat does not conform to breed characteristics and they would transmit undesirable genes.

Our studies confirmed the finding by Leipold et al. (1972) and Jayasekara et al. (1979). The developmental stage of hair cycle in congenital hypotrichosis begins in the anagen phase when the Huxley's layer of cells becomes degenerative and arrests the differentiation of cells in the inner root sheath. The upper part of the hair follicle,

known as a critical level, starts in mitotic activity to form each layer of the hair shaft. As the anagen phase progresses, the changes in the Huxley's layer become more prominent, and microdroplets of trichohyalin are located between the sebaceous gland canal and the hair follicle. After the catagen phase, mitotic activity is reduced, and no trichohyalin granules are produced by the inner root sheath. Some microdroplets, however, are still present in the remaining part of anagen. The new cycle of the disease restarts after the telogen phase when a new anagen begins the mitotic activity.

Some criteria have been established on the biochemistry behavior of the inner root sheath (Straile, 1965). It is reasonable to assume that the absence of genetic material in the cells of the inner root sheath could produce enzymatic imbalances or do not permit a good balanced hormone interaction from endocrine glands. Those abnormalities arrest the metabolism of the cells. In observing a hypotrichotic Hereford calf, we noted differences in the stages of the hair cycle during the summer and winter (Table 2). Further studies on the metabolic bases in endocrine glands; hormonal studies in adult animals must be done to confirm biochemical findings.

SUMMARY

Histopathological changes on the pathogenesis of 22 hypotrichotic Hereford calves revealed initial changes in

the anagen phase of the hair cycle, characterized by the presence of trichohyalin granules in Huxley's layer. The catagen phase showed remnant trichohyalin granules from the anterior phase, and the telogen phase was negative to micro-droplets of trichohyalin.

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TABLE 1: Staining Properties of Trichohyalin Granules in Huxley's Layer of Hair Follicle in Hypotrichosis of Hereford Cattle.

Stain	Reaction		Remarks
H&E	Pink	±	Acidophilic
PAS	Stains the periphery	±	
PAS-diastase	Stains the periphery	±	
Toluidine blue	Blue	-	No metachromasia
Prussian blue	Pink	-	No iron
Masson's trichrome	Brown	-	
Altmann's acid fuchsin-methyl green	Brilliant red	±	Brilliant fuchsinophilia

TABLE 2: Changes in the Huxley's Layer in Summer and in Winter Time

Hair Cycle	Summer (August and September)	Winter (December and January)	Observations
Anagen	±	+	Degenerative changes, trichohyalin droplets, vacuoles in the inner root sheath.
Catagen	-	±	Trichohyalin droplets, anagen remnant in the club hair.
Telogen	-	-	No trichohyalin granules. This phase is difficult to see in hypotrichosis. Retardation in the new anagen phase.

PLATE 1: SKIN BIOPSIES TAKEN AT SEVEN-DAYS INTERVALS

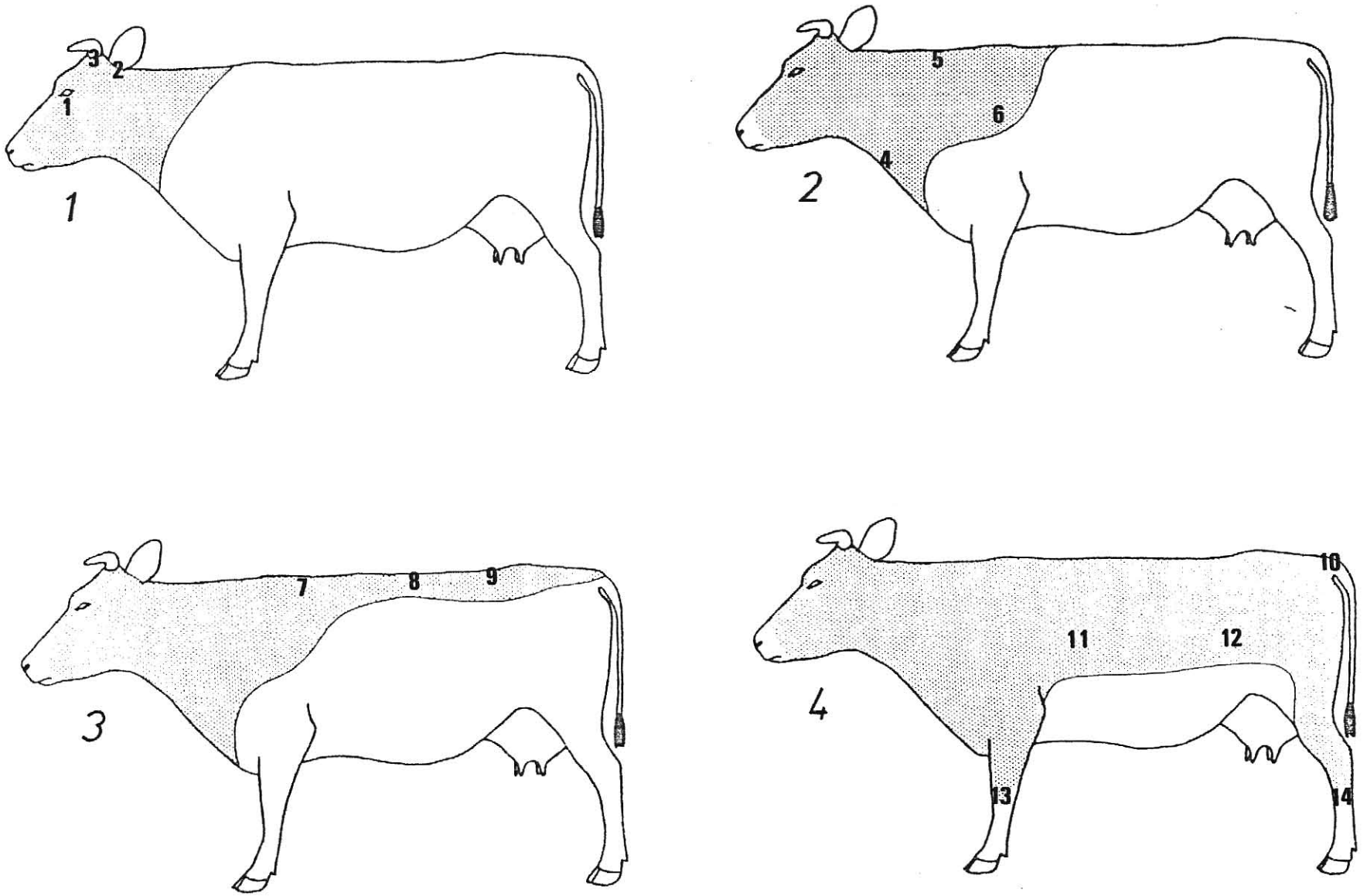


Fig. 1. Purebred polled Hereford female calf with moderate degree of hypotrichosis. Notice short hair over entire body.

Fig. 2. Purebred polled Hereford bull calf with severe degree of hypotrichosis. Notice hair changes affecting lateral and legs aspects of the body.

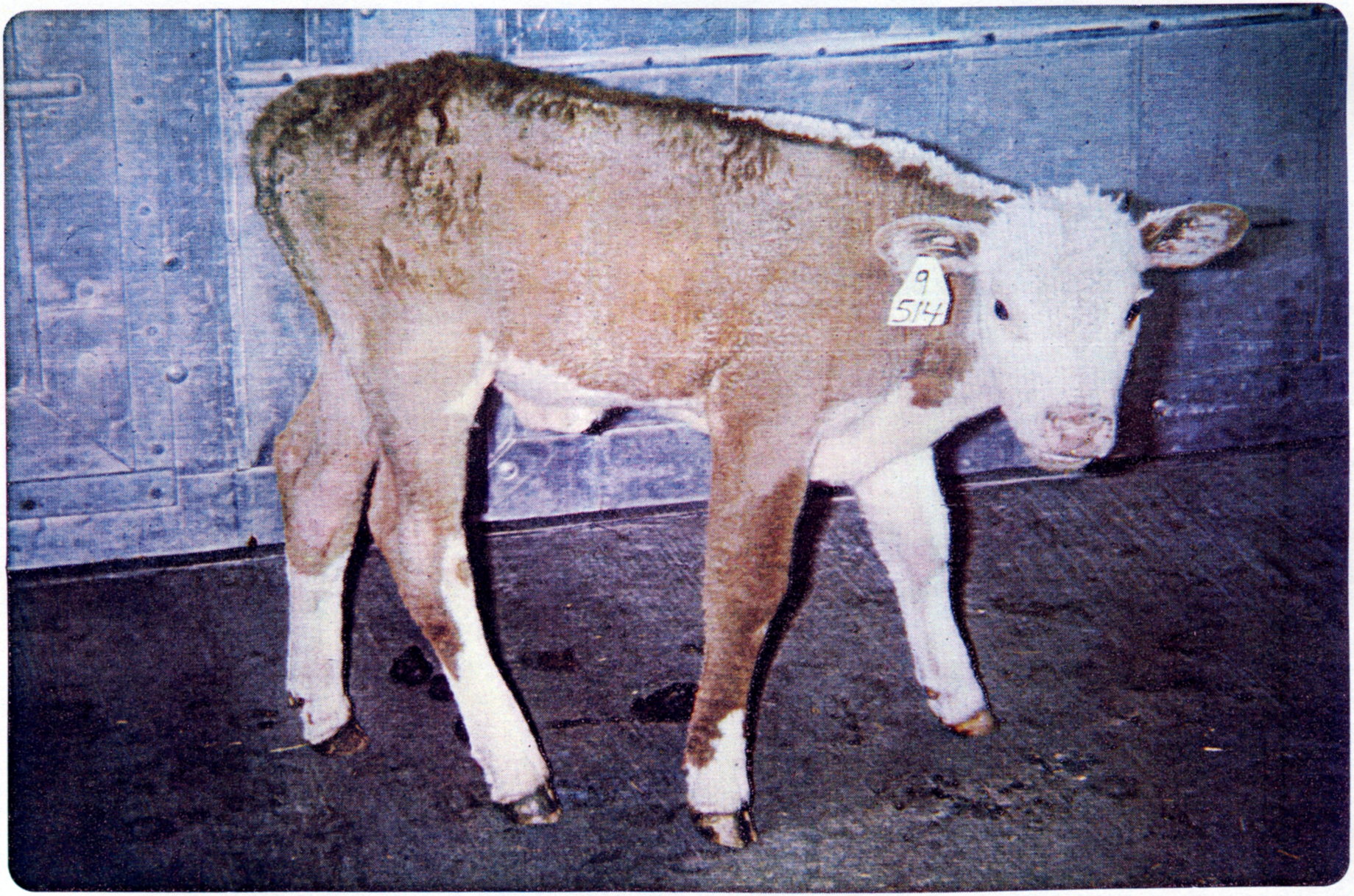

9
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Fig. 3. Purebred female Hereford calf with slight degree of hypotrichosis. Notice mild changes over the axillary and inguinal areas of the body.

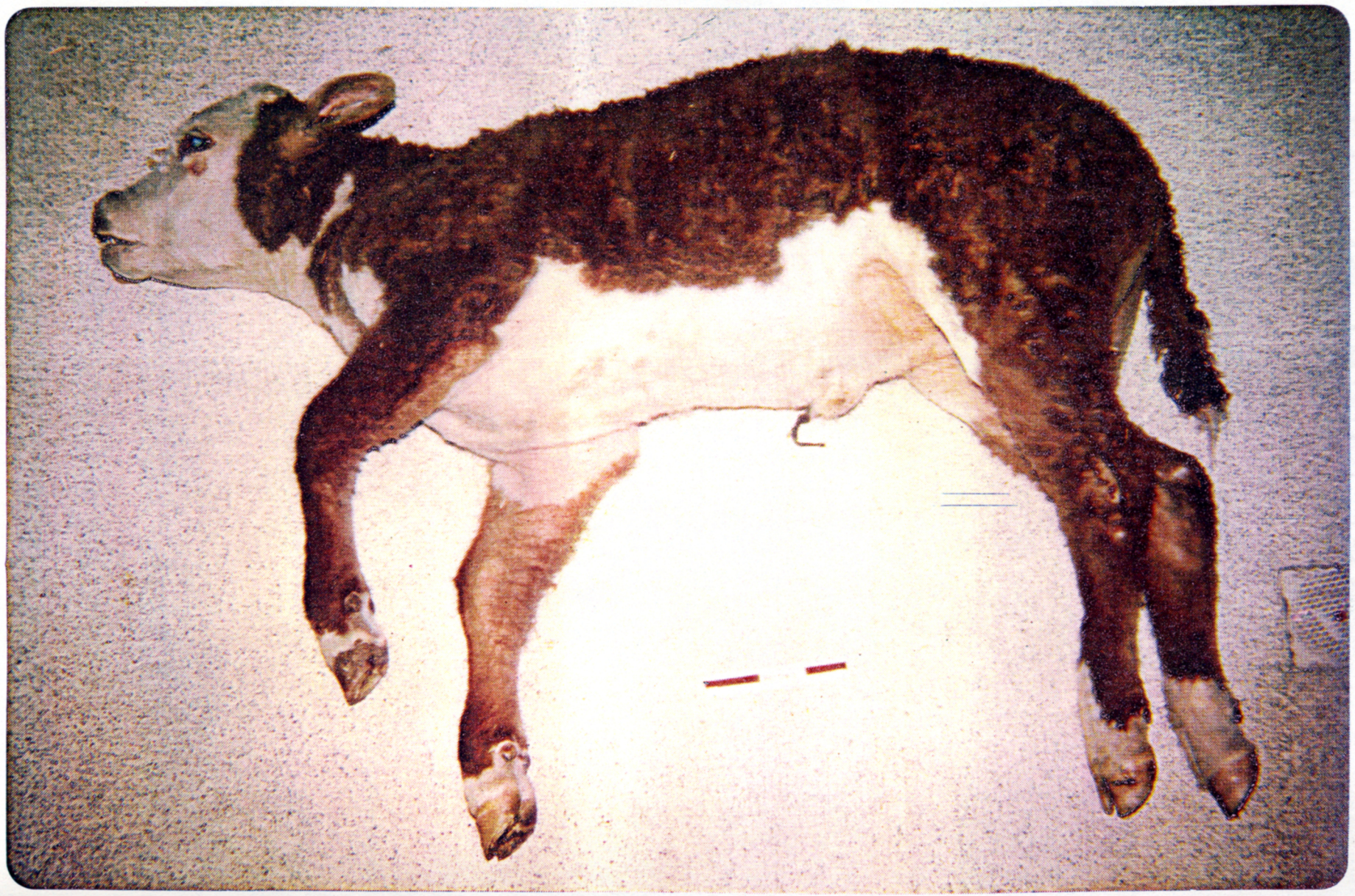


Fig. 4. Purebred female Hereford calf showing hypotrichotic changes over the head, axillary and legs.



Fig. 5. Purebred male Hereford calf affected with severe degree of hypotrichosis. Notice areas of severe dermatitis affecting carpal, Tarsal and metacarpal areas of the body.



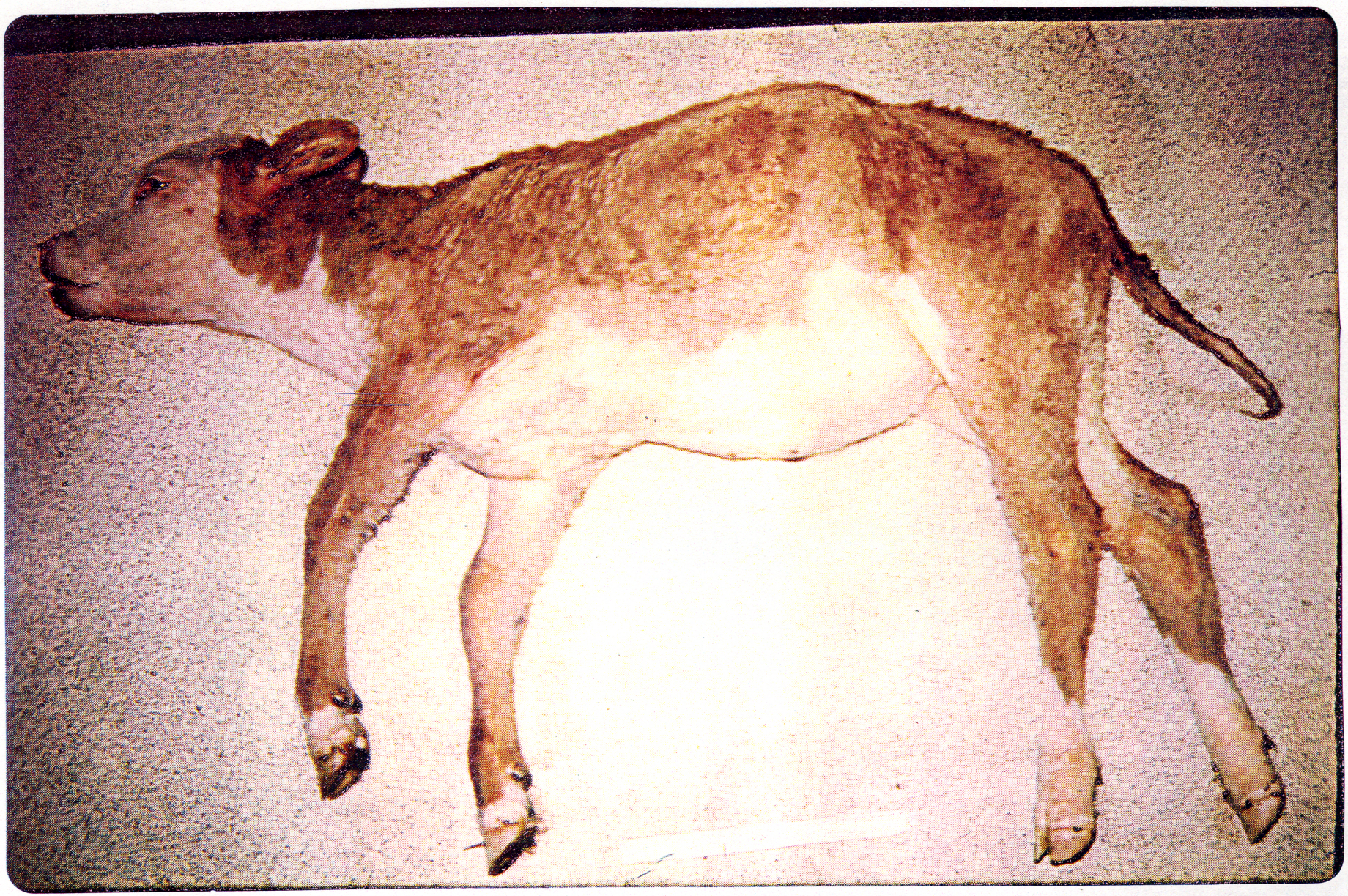


Fig. 6. Purebred female Hereford calf with severe degree of hypotrichosis over the entire ventral body area.

Fig. 7. Purebred female Hereford calf affected with severe degree of hypotrichosis. Notice almost hairless changes over the head, shoulder, legs, ventral area of the body.



Fig. 8. Purebred male Hereford calf affected with severe degree of hypotrichosis. Notice dermatomycosis over the neck and hairless areas on head, legs and ventral areas of the body.



Fig. 9. Same animal in Figure 8 showing severe degree of hypotrichosis and dermatomycosis (arrows).

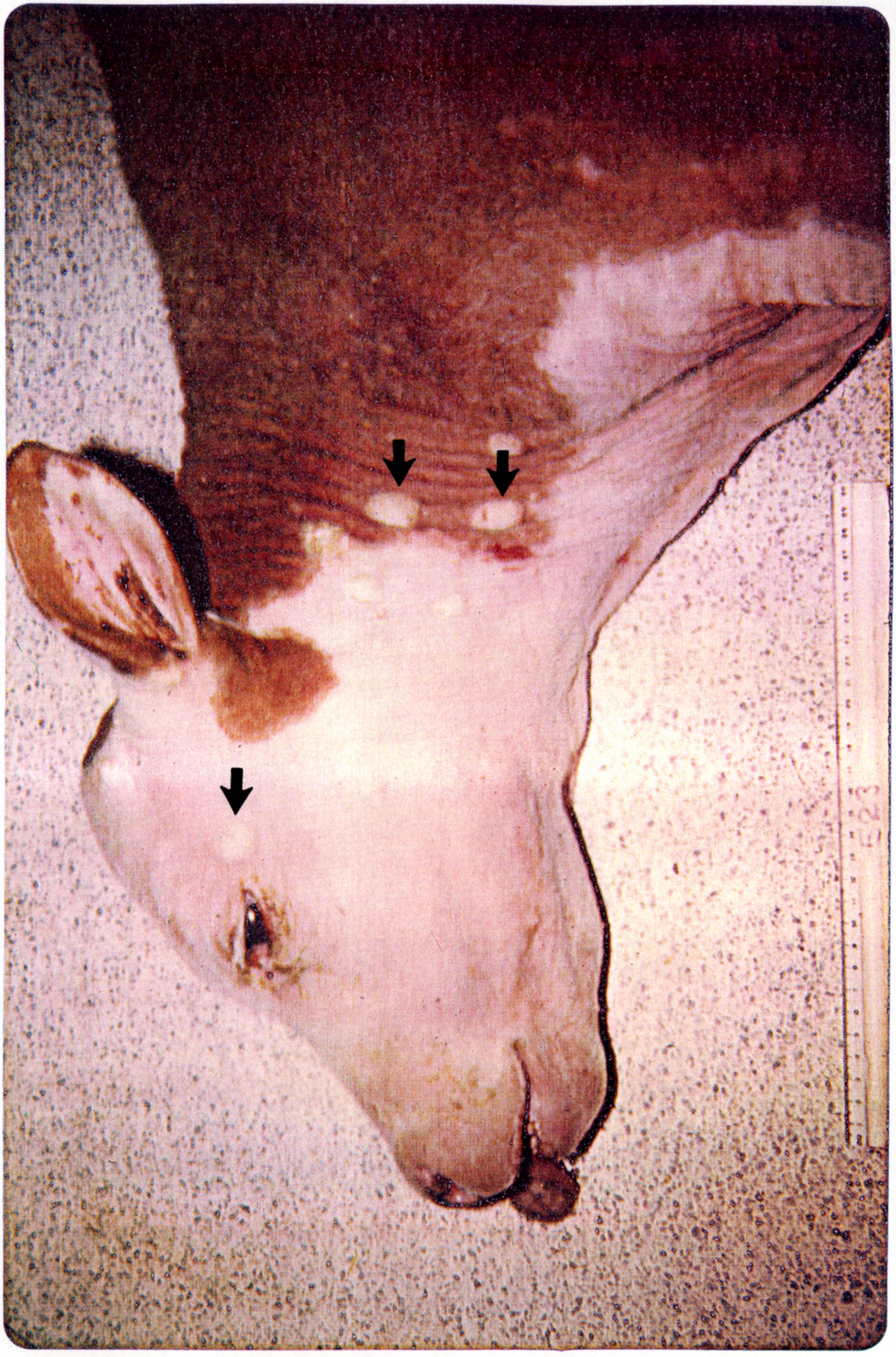
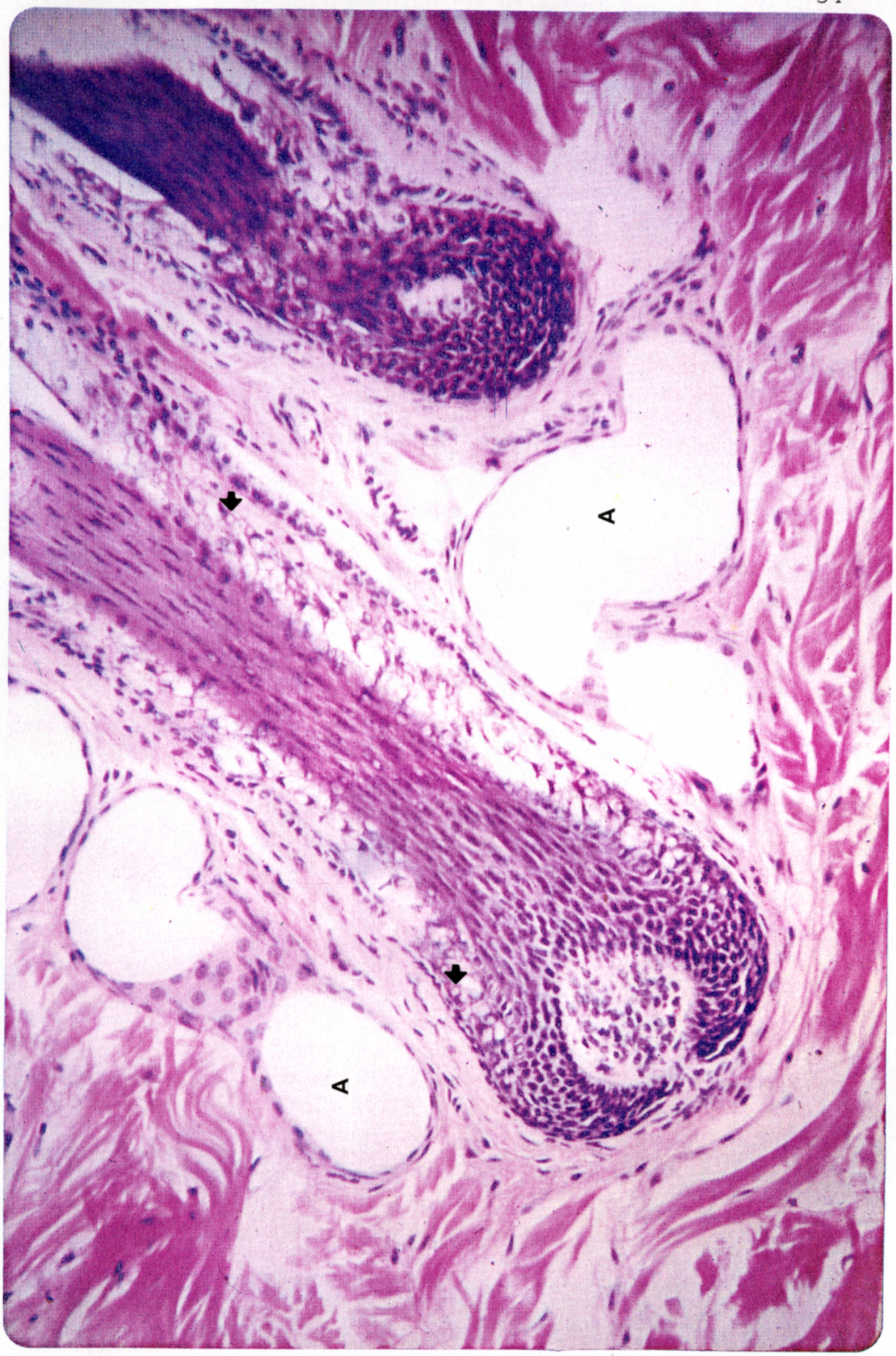


Fig. 10. Same animal in Figures 8 and 9 showing dermatomycosis.

Fig. 11. Skin section of cervical area of the calf affected with hypotrichosis. The hair in the anagen phase with trichohyalin granules in the Huxley's layer (arrows). Notice the enlargement of the sweat glands (A).
H&E X 140


A
A

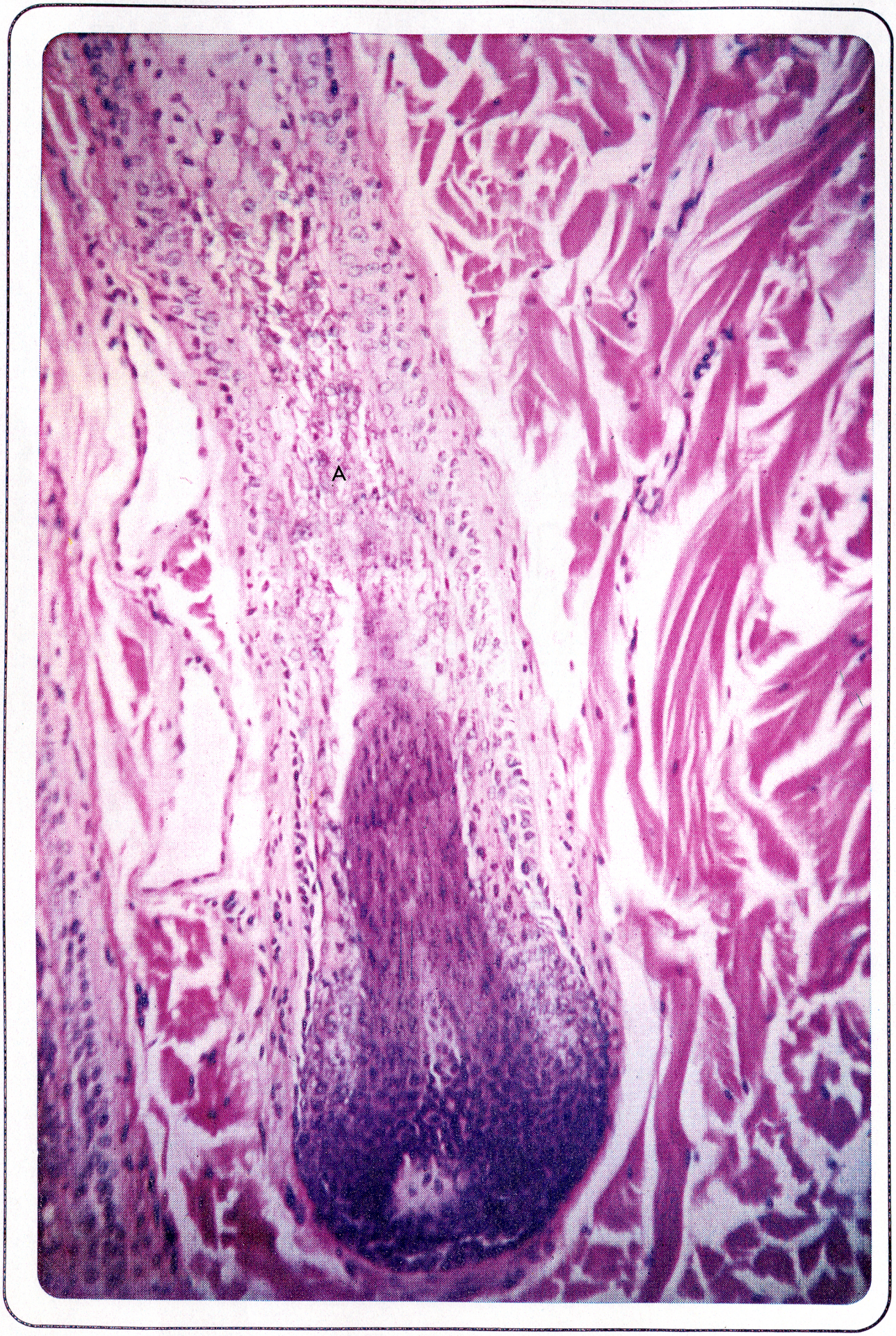

A

Fig. 12. Longitudinal section of the hair in anagen phase with abundant trichohyalin granules in the medulla (A). H&E X 140.

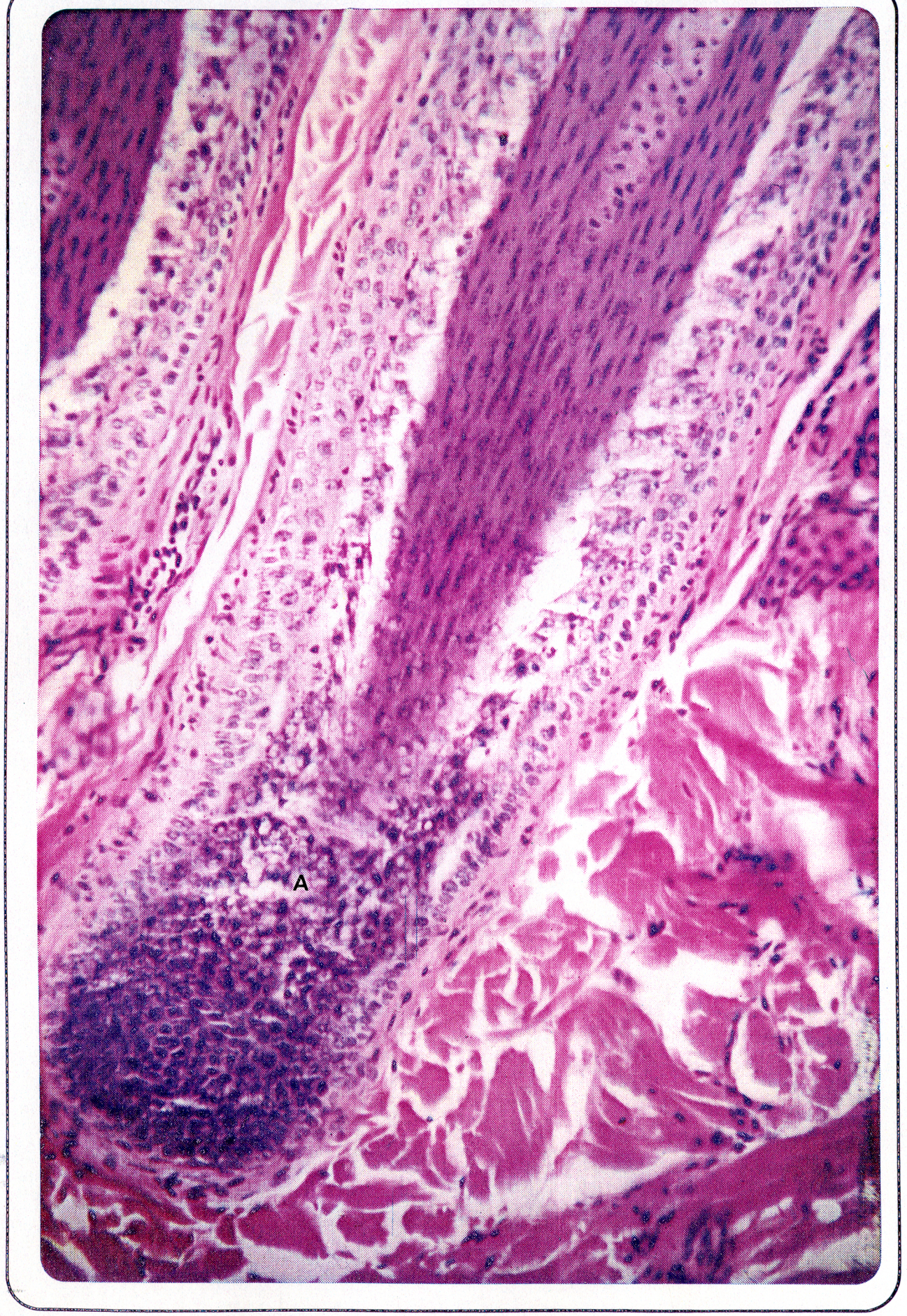

A

Fig. 13. Hair anagen, longitudinal section with trichohyalin granules in the upper limit of the hair bulb (A). H&E X 140.

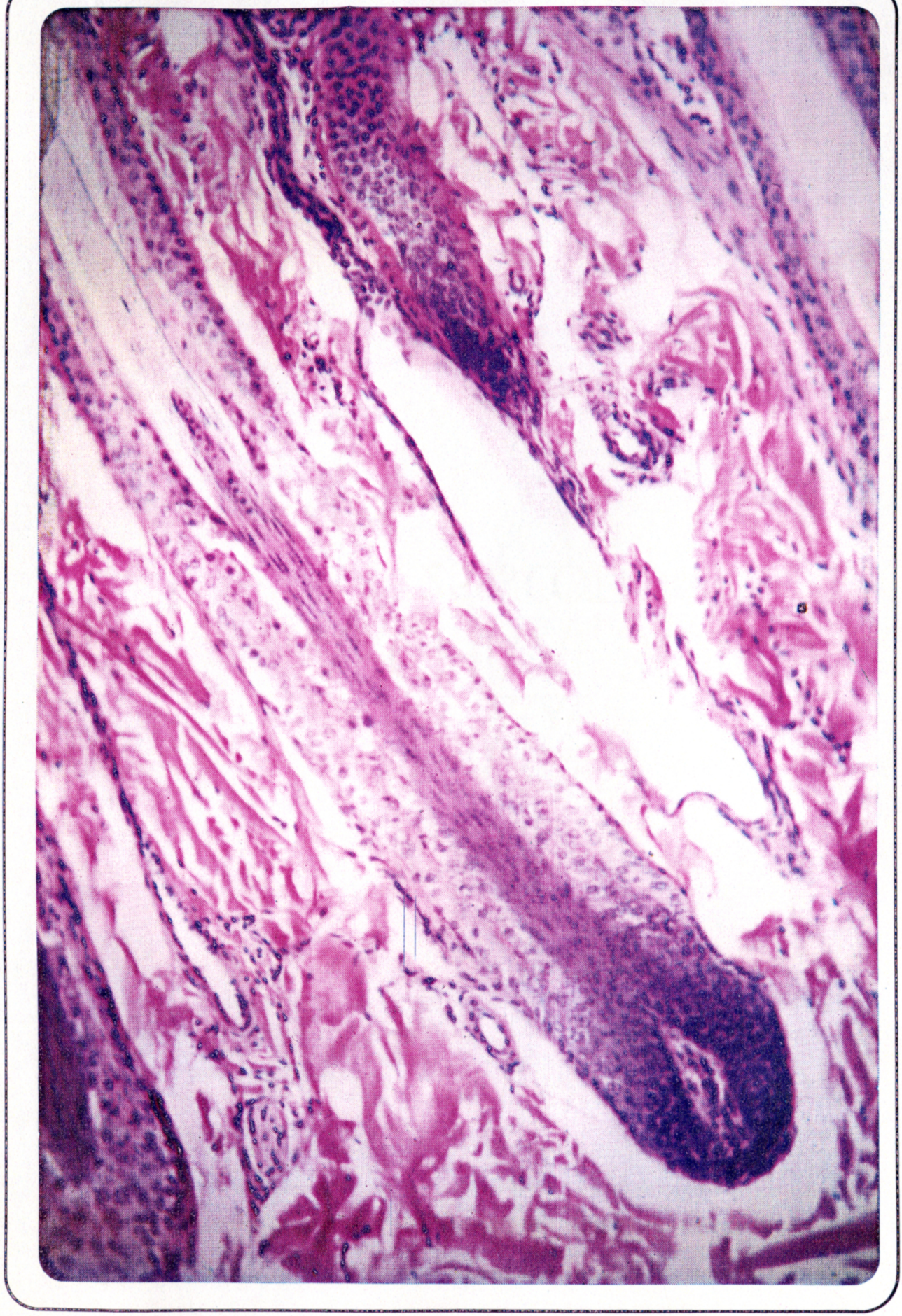


Fig. 14. Hair anagen phase longitudinal section with degenerative changes in the Huxley's layer. H&E X 140.

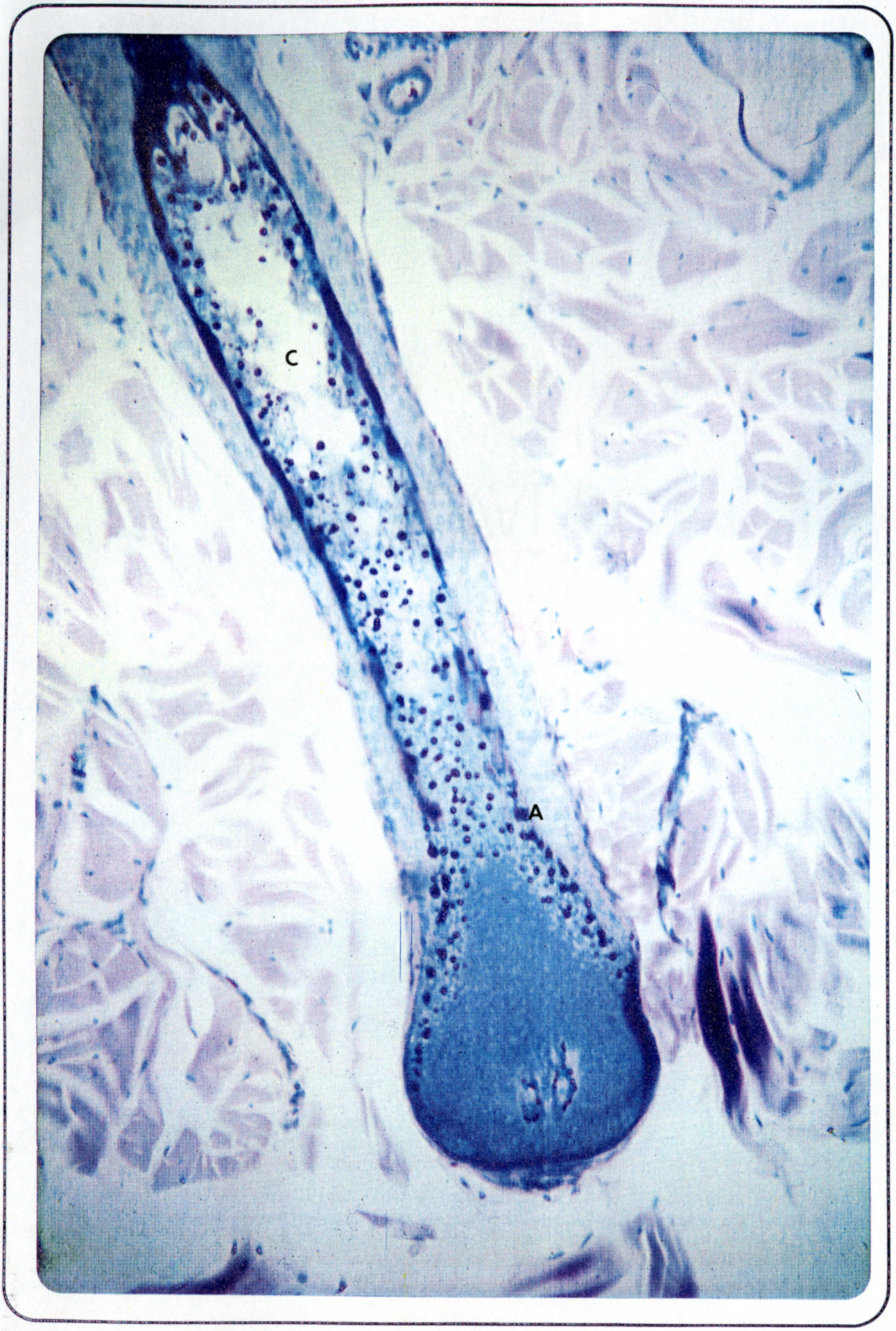

C
A

Fig. 15. Hair anagen phase with trichohyalin granules in the Huxley's layer (A); medulla (B). Notice the degenerative changes (vacuoles) in the medulla (C). Altmann's stain X 140.

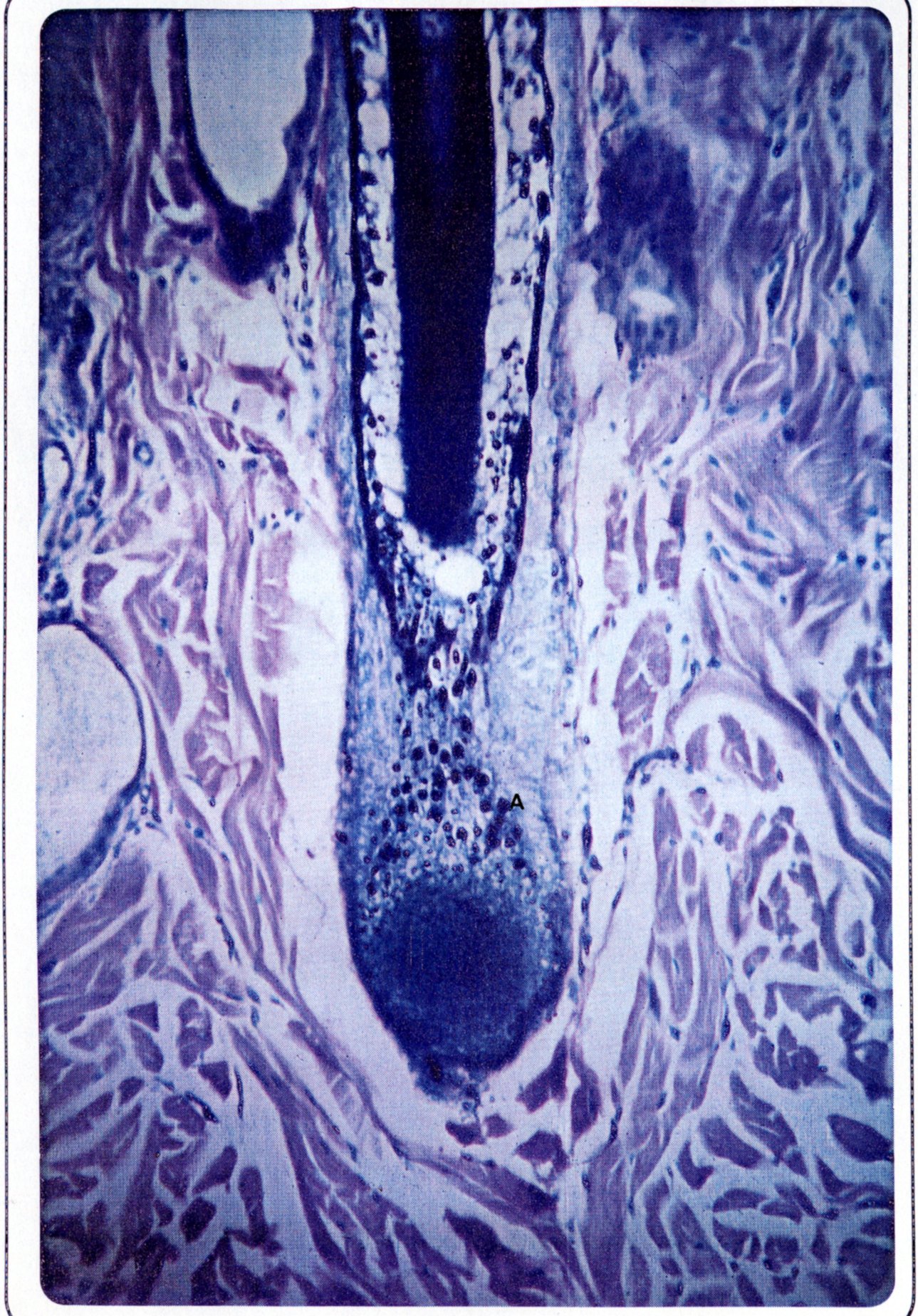

A

Fig. 16. Hair anagen phase longitudinal section showing severe changes in the Huxley's layer with trichohyalin granules production (A). Notice vacuolization of the cortex of the hair shaft. Altmann's stain X 140.

Fig. 17. Cross section of a hair shaft of a calf affected with hypotrichosis. Notice abnormal trichohyalin granules, semitranslucent with dark pink border in Huxley's layer (arrows). H&E X 140.

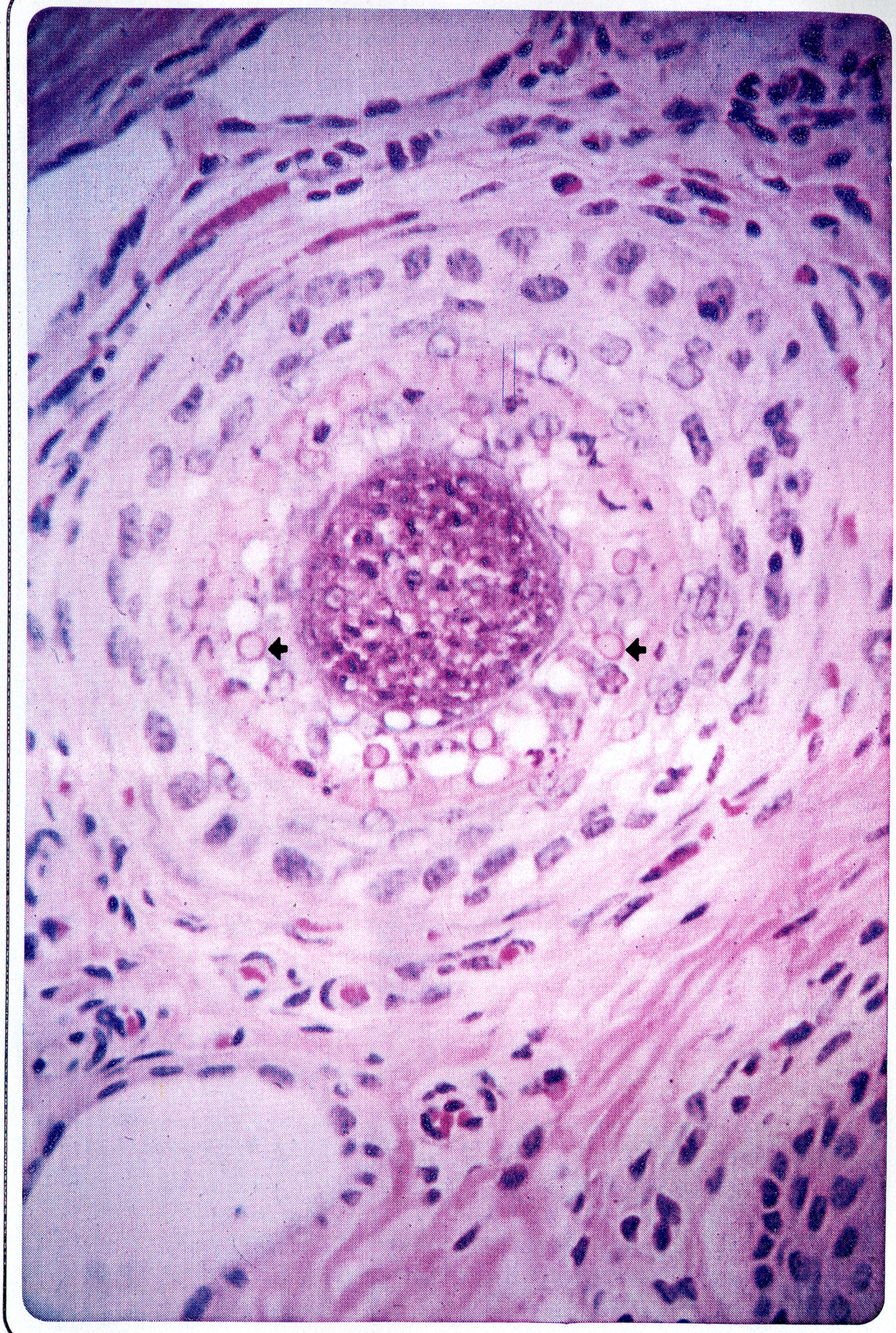


Fig. 18. Cross section of a hair showing the spheroidal microdroplets of trichohyalin granules in Huxley's layer. Notice the dilatation of the sweat glands (A). Altmann's stain X 1400.


A
A

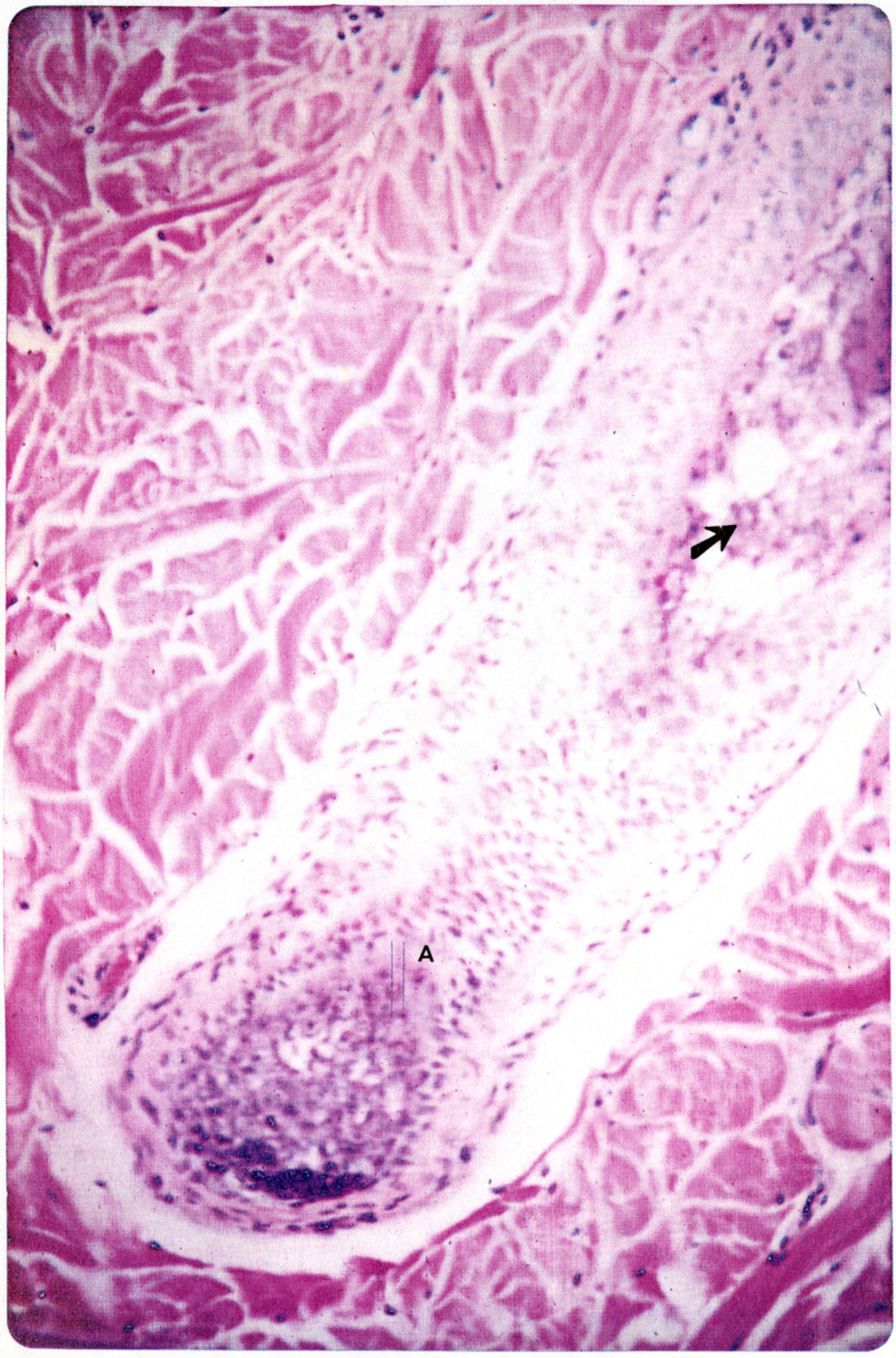
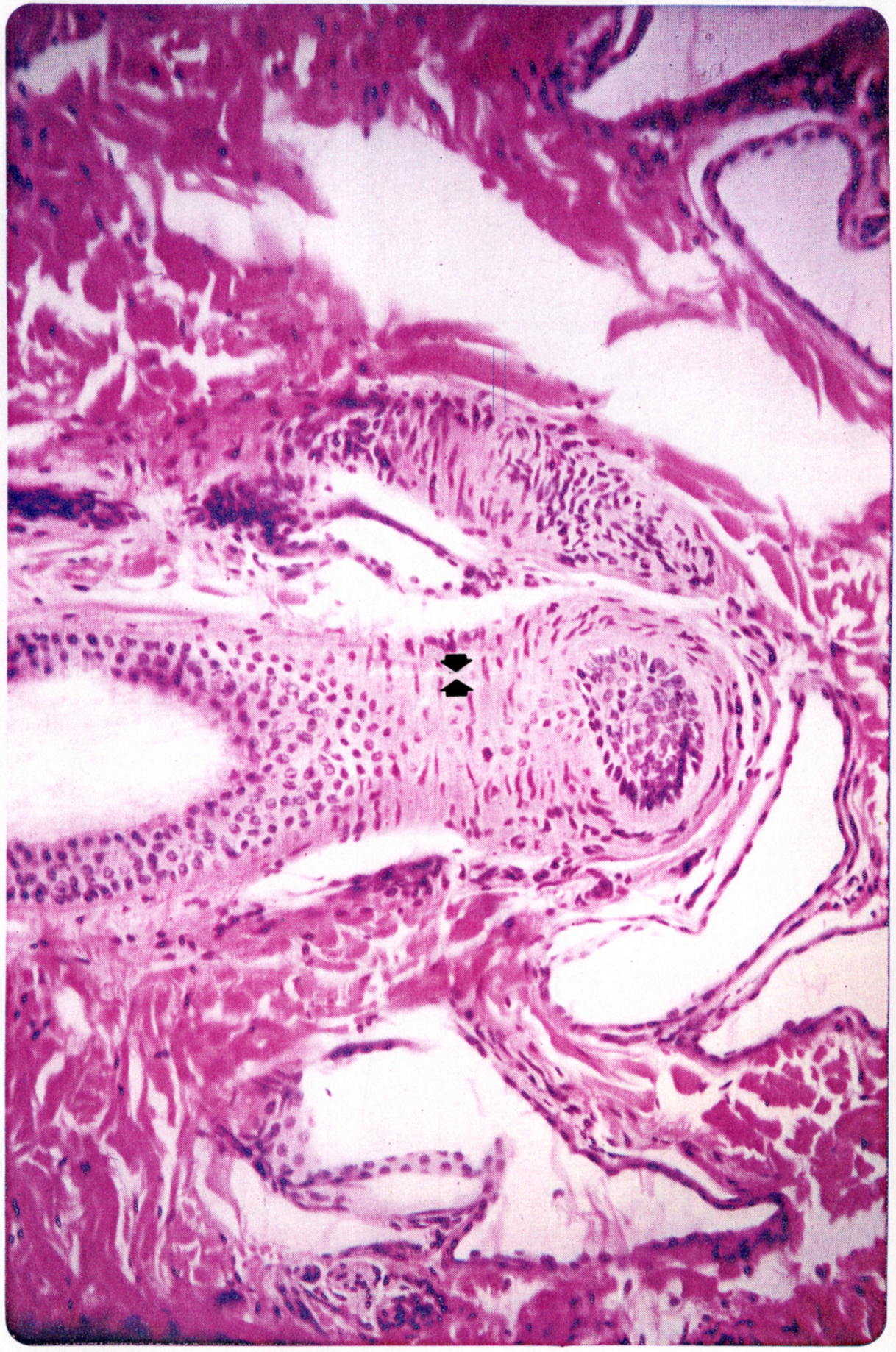

A

Fig. 19. Catagen phase of a hair showing the epithelial portion of the bulb undergoing resorption (A) and some trichohyalin granules in the club hair (arrow). H&E X 140.

Fig. 20. Hair follicle in catagen. The glassy membrane (between arrows) is thickened. The hair bulb is undergoing resorption. In the upper area is the well-formed club hair. H&E X 140.



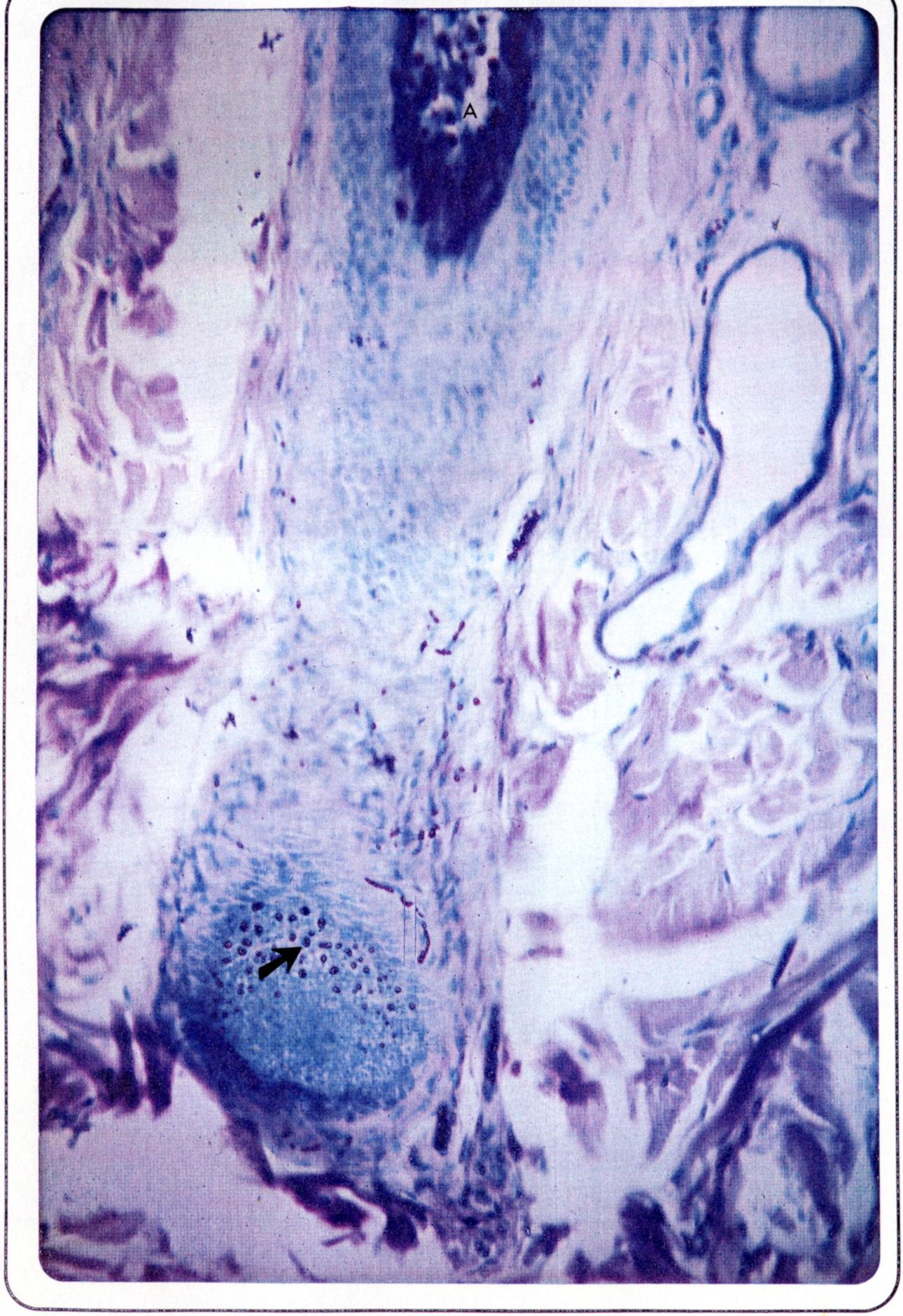

A

Fig. 21. Hair follicle in catagen. The bulb has some trichohyalin granules (arrow) as a remnant of the anagen phase. The club hair (A) also present trichohyalin granules from the anagen phase. Altmann's stain X 140.

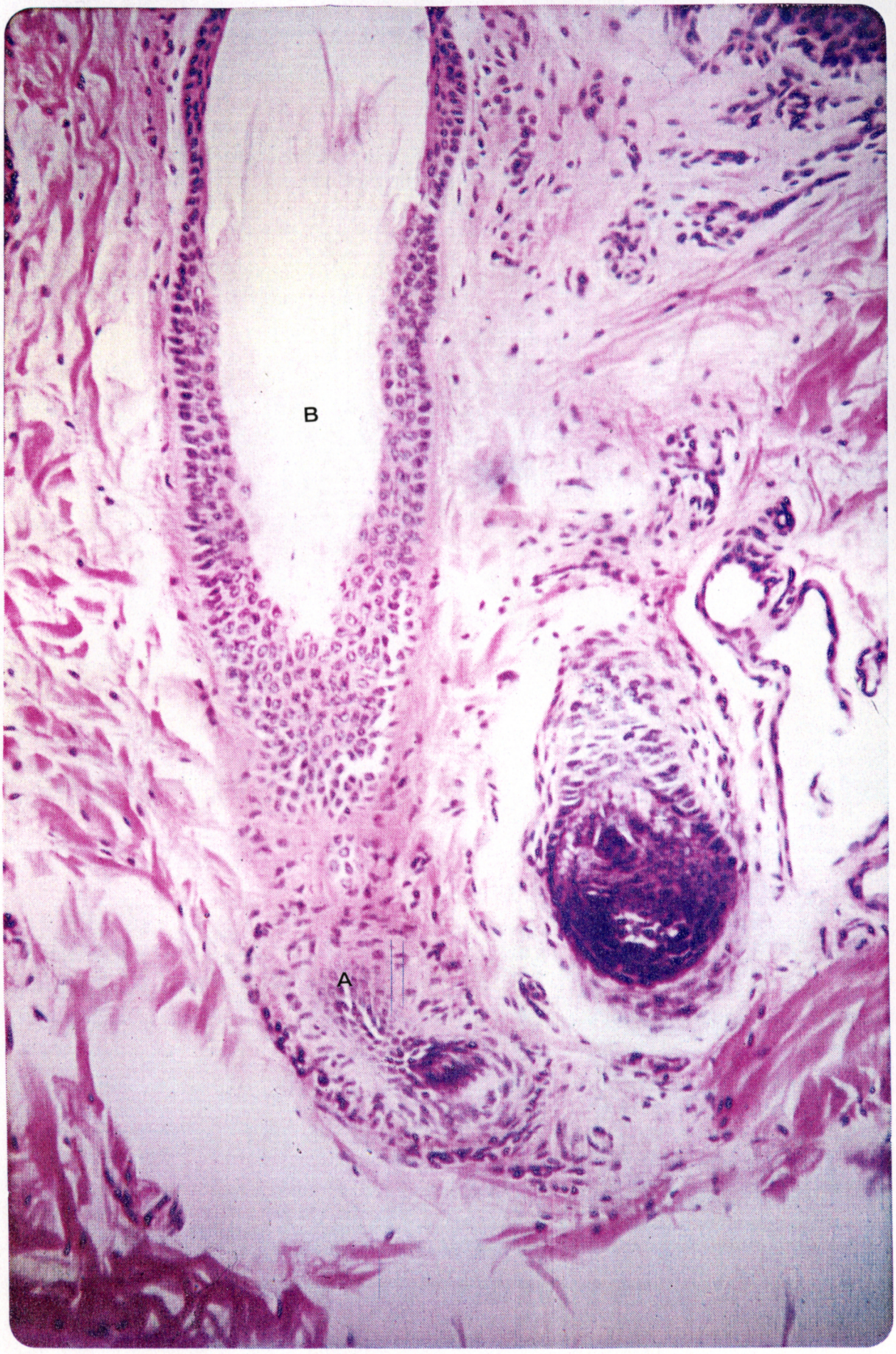

B
A

Fig. 22. Hair follicle in telogen phase. Notice there is no relationship between the old bulb (A) and the club hair (B). A new germ is starting a new hair phase (between arrows). H&E X 140.

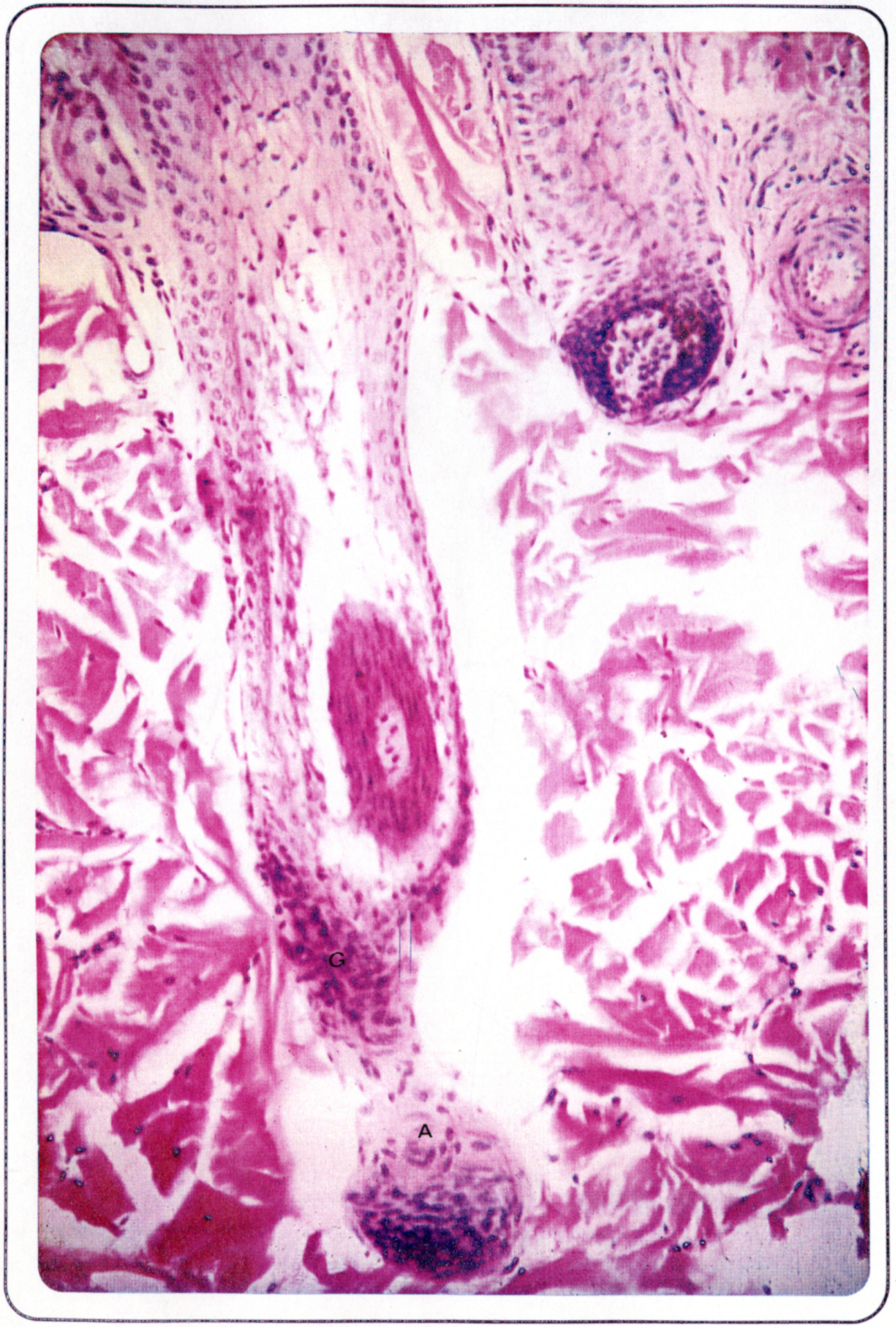

G
A

Fig. 23. Hair follicle in telogen phase. Notice the old resorbed bulb (A) and the new germ (G). H&E X 140.

III. CONGENITAL ALOPECIA IN CALVES

INTRODUCTION

Congenital absence of hair has been reported in all domestic animals. The degree and severity of hairlessness was variable depending on the breed (Jubb and Kennedy, 1970). Hairlessness has been described in different animals as a lethal problem (Hutt and Saunders, 1953). It has been described as viable genetic condition in two Guernsey calves (Becker et al., 1963). These calves presented a remarkable deficiency of hairs and the sweat glands were cystic. Hutt (1963) described six forms of congenital hypotrichosis in cattle, and Bianchi (1953) reported hair and hoofs dysplasia in newborn calves. Hairlessness at birth is one of the economical features among breeders and its frequency varies according to breed. Within breeds, factors such as function or structure affected, level of nutrition, environment, age of parents, management, and others; modify the total number of congenital defects (Leipold, 1978). One case of combined signs of prolonged gestation and hairlessness has been reported by Rasbech (1950). Maijala and Viljo (1964) studied the occurrence of hairless calves associated with prolonged gestation, a recessive lethal gene was incriminated.

Hypotrichosis and hyperkeratosis have been described in Pinzgauer cattle as a genetic problem caused by homozygosity of a recessive autosomal gene (Pirchner and Grunberg, 1970). Derlogea (1963) reported two cases of hairlessness in Pinzgauer cattle and compared them with those described as

semilethal alopecia by Hutt and Saunders. The eyelids and the tail were bare of hair.

This present report describes five cases of congenital alopecia encountered in Simmental, Hereford, Angus, Pinzgauer, and Holstein cattle.

HISTORY AND GROSS PATHOLOGICAL CHANGES

Case I: Simmental female calf, 4 days old, was presented with smooth skin. Close examination revealed a short fine coat with small tufts of longer well developed hair on the legs, flanks and back. The ears and the base of the tail had only a few well developed hairs. The muzzle and eyes had tactile hairs (Figure 1). The thyroid gland was enlarged.

Case II: Hereford female calf, one week old, had almost complete absence of hair. The hairlessness was located mainly in the axilla, inguinal area, head, and legs. The trunk had many isolated areas of fragile hairs of varying length. Scarcity of coarse hairs occurred in the nasal region, vulvar ventral commissure, and base of the ears. Eyelashes were also present (Figure 3). Internal examination revealed enlargement of the thyroid gland, internal hydrocephalus, interauricular communication, and enlargement of both adrenal glands. Mild dermatitis was present.

Case III: Angus male calf, two days old, presented with severe hairlessness; tufts of hair were present mainly on

the trunk, dorsal and ventral areas. The skin was smooth, dry and some vellus were seen on the tip of the tail and tailend, base of ears, and around the muzzle (Figure 5). The teeth were erupted.

Case IV: Pinzgauer male calf, five days old, presented with complete absence of hair, primarily affecting the face and ears, neck, trunk, and legs. Only random tufts of vellus were present on the end of the tail and posterior area of the hind right leg. The skin looked thick like "sandpaper" different from the rest of cases where the blood vessels were easily seen. The abdomen and flanks in a very close observation showed a very fine vellus resembling lanugo hair (baby hair). Severe serious dermatitis in both right and left flanks was seen (Figure 7).

Case V: Holstein female calf, seven weeks old, affected with a slight degree of hairlessness. The skin was thickened. Gross examination revealed absence of hair on the face and base of ears, axilla and inguinal area (Figure 9). There were no teeth erupted (anodontia) (Figure 10). Dermatomycosis on the left shoulder was present.

HISTOPATHOLOGICAL FINDINGS

The histological cross and longitudinal sections through the skin revealed sparsely distributed hair follicles (Figures 6 and 11), the sheaths of which were much enlarged and distorted resembling small lodgement. The hair roots

were coiling and sometimes erupted beneath the epidermis (Figure 8). Epidermis in all cases was hyperkeratotic. The hair papillae were misshapen, atrophic, and the hair follicles were clogged by plugs of keratinous material (Figures 2, 4, and 6). There was a total disturbance in the orientation of the hair follicles in the skin from the Pinzgauer calf (Figure 8). The Angus, Hereford, and Holstein calves skin revealed hypoplasia of the hair follicle (Figures 6, 4, and 11). Sections of skin from flanks, dorsal, cervical, and lumbar areas revealed thin epidermis with a moderate degree of hyperkeratosis and parakeratosis. Some hairs were plugged with laminated keratin and debris and many had not erupted. There were no mature hair follicles in most of skin sections studied, especially in the Simmental calf (Figure 2). Other areas had nests of cells resembling vestigeal embryonic hair germ (Figure 6). The sweat glands were dilated and sometimes cystic, and had flattened epithelium (Figures 2 and 6). Masson's trichrome revealed no arrector pili muscles in the pilosebaceous apparatus.

DISCUSSION

The available breeding histories of congenital alopecia were indicative of a simple recessive autosomal gene (Pirchner and Gruneberg, 1970; Schleger et al., 1967; Leipold et al., 1979; McGavin and Alexander, 1960; Kidwell and Guilbert, 1950). Hair development in the bovine fetus is well established between the 77th and 166th days of

gestation (Hubbert, 1974; Lyne and Heideman, 1959). Goldsberry and Calhoun (1959) reported the comparative histology of the skin of Hereford and Aberdeen Angus cattle, and found significant differences between two breeds.

The histological description of hypotrichotic skin included poorly differentiated hair follicles and rarely extended beyond the sebaceous gland (Becker et al., 1963). Furthermore, observations by Mohr and Wriedt (1928) included marked delay in development of hair follicles and their appendages, and histological criteria corresponded to an early stage in normal development. In our cases, similar findings were observed that were typical of congenital alopecia and not congenital hypotrichosis.

Sections of skin studied by light microscope from hairless areas lacked well developed hair follicles. The follicles were arrested and occluded due to horny laminated keratin masses, and in some cases, the skin resembled an embryogenic hair germ. According to reports by Leipold et al. (1972) and Pirchner and Grunberg (1970) affected animals with this condition and other kinds of abnormal epitheliogenesis, succumbed to septicemia. Occlusion of the hair canal retarded hair development and prevented hair emergence. Rudimentary hair is typical of this disease. The hyperkeratotic change involved the hair roots and sebaceous glands as noted by Pirchner and Grunberg (1970) in Pinzgauer cattle. The sweat gland in all cases was dilated and the arrector pili muscle was absent. Our findings in the Pinzgauer calf

confirmed the lesions described by Pirchner and Grunberg (1970). Denis et al. (1975) summarized their report of congenital hypotrichosis as a late skin development; in the first months of life it is of a fetal type.

Microscopical observation on hairs taken from one of the semihairless calves revealed that the hairs have a tendency to be flattened and spiraled, and the hair follicles were prematurely developed, and no hairs were formed in the premature follicles (Craft and Blizzard, 1934).

Streaked hairlessness in Holstein-Friesian cattle were reported by Eldridge and Atkeson (1953). Holmes and Young (1954) reported two cases of symmetrical alopecia in cattle and described similar lesions that were of congenital alopecia.

SUMMARY

Five cases of congenital alopecia were encountered in Simmental, Hereford, Angus, Pinzgauer, and Holstein calves. Affected calves were one day to seven weeks old, and the disease affected both males and females. The calves had dry, smooth skinned skin. One calf had severe dermatitis and the others descamative dermatitis. Genealogical data of all cases were indicative of a simple autosomal recessive type of genetic disorder.

Histopathological changes were atrophy of hair papillae, arrested hair growth, and disorientation of growth of some hair and hyperkeratinosis.

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Fig. 1. Purebred female Simmental calf, severely affected with congenital alopecia. Notice thinness of the skin.



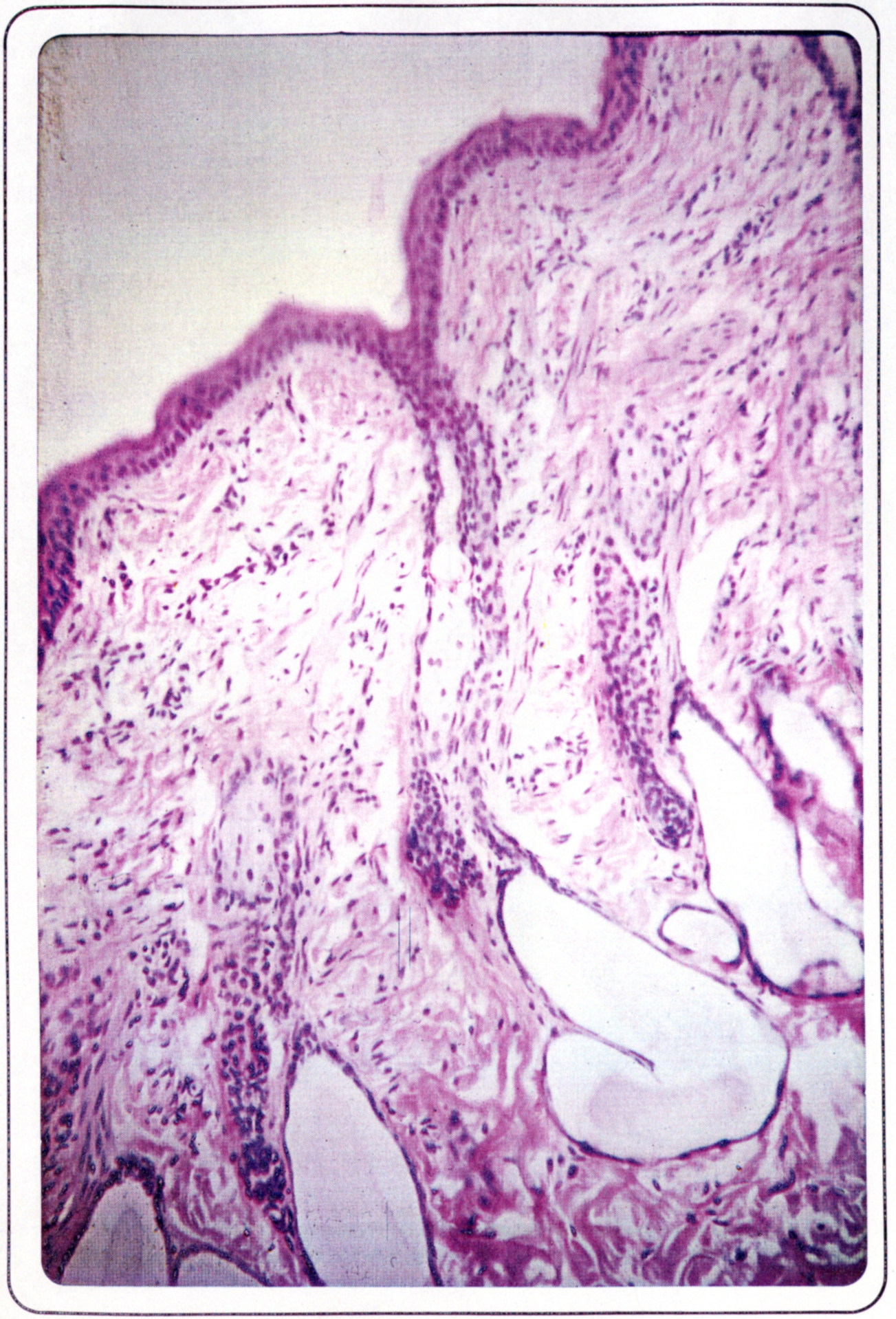


Fig. 2. Skin section of the calf affected with congenital alopecia. Notice the shallow, poorly differentiated hair follicles and dilated sweat glands. H&E X 108.

Fig. 3. Purebred female Hereford calf with moderate degree of congenital alopecia. Notice areas of severe dermatitis affecting the joints (arrows).

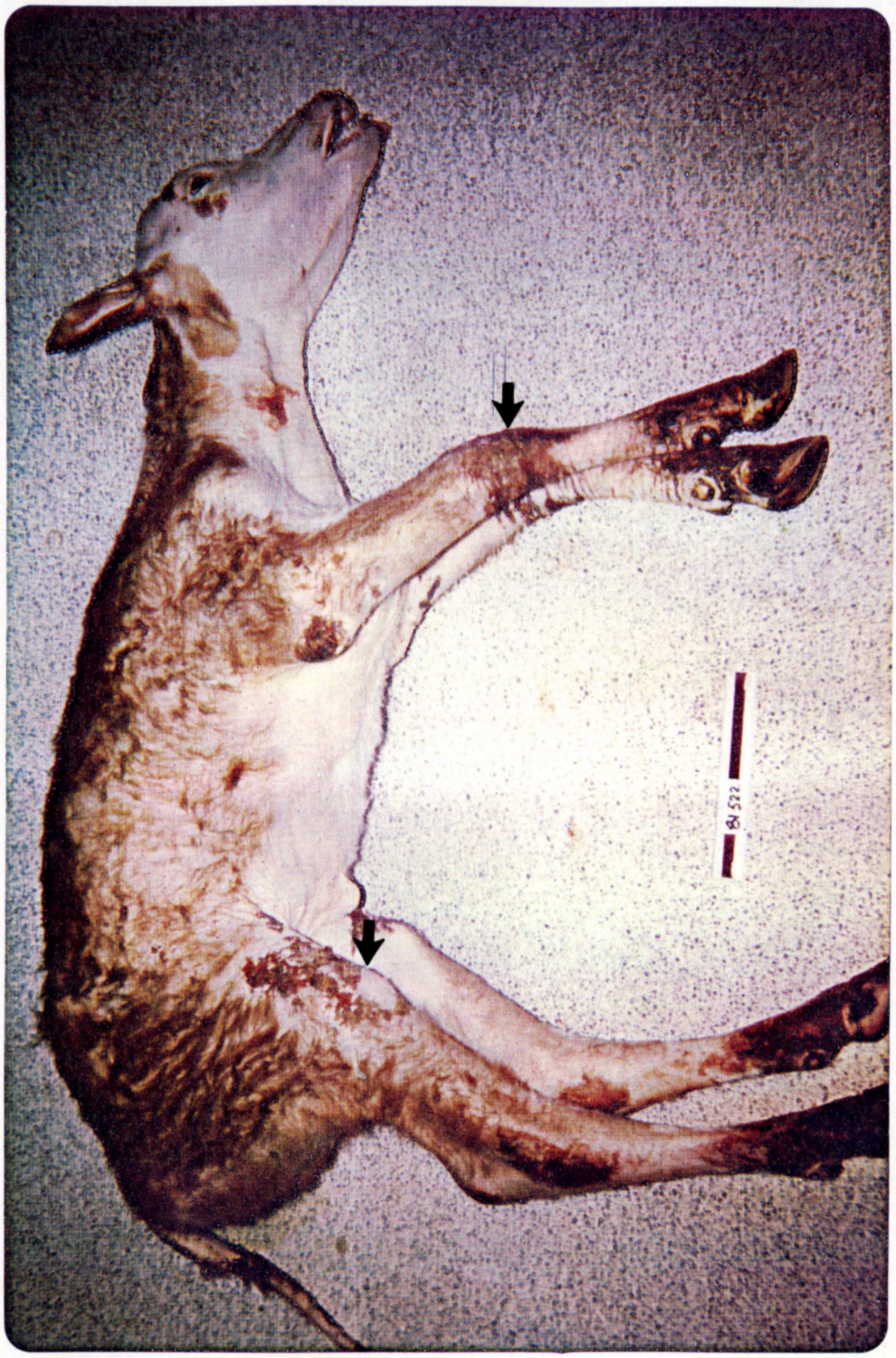

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Fig. 4. Skin section of the calf with congenital alopecia. The pilosebaceous apparatus is totally atrophic. H&E X 140.

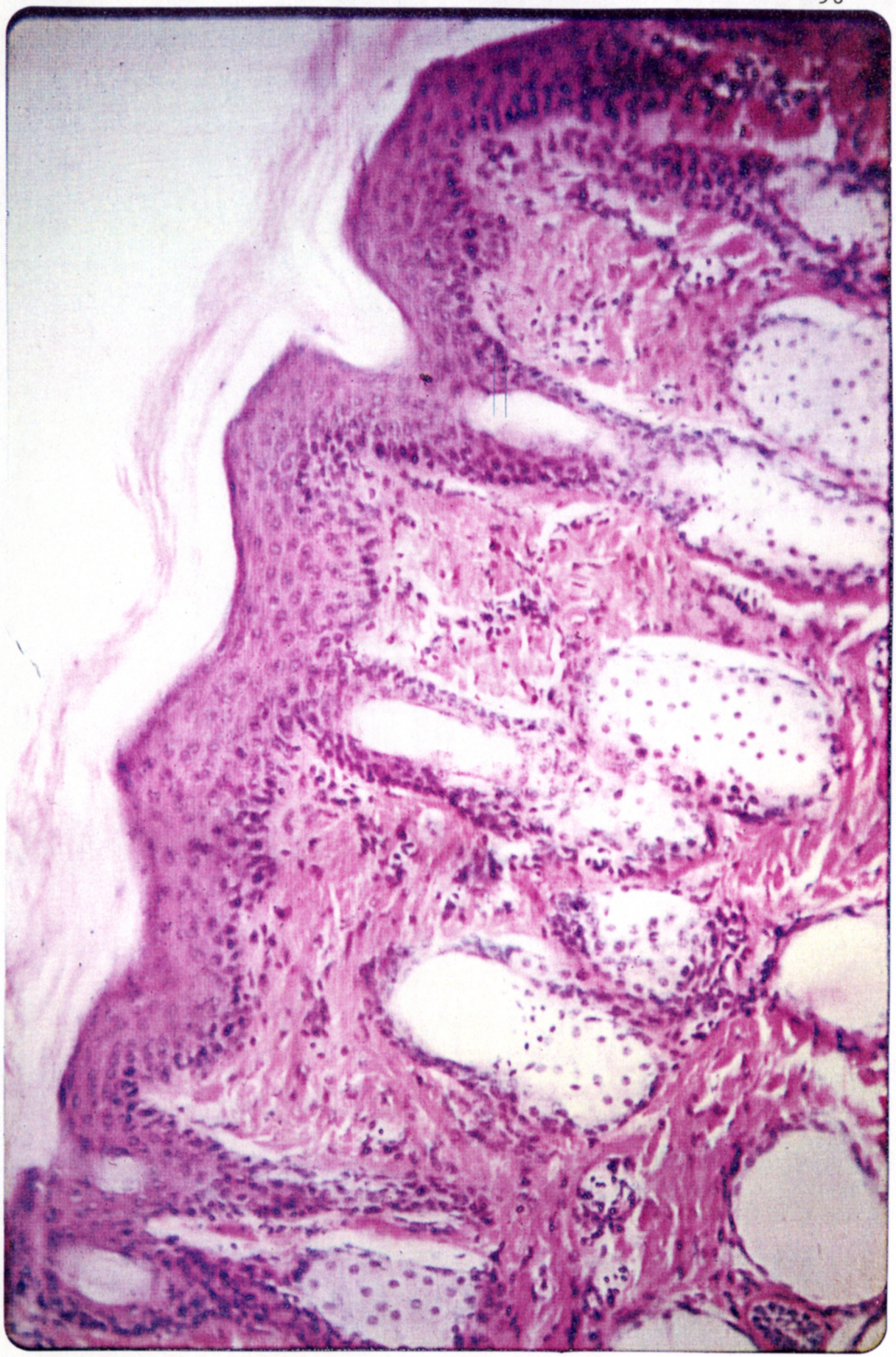


Fig. 5. Purebreed male Angus calf with severe degree of congenital alopecia. The black spot areas present tufts of hairs.

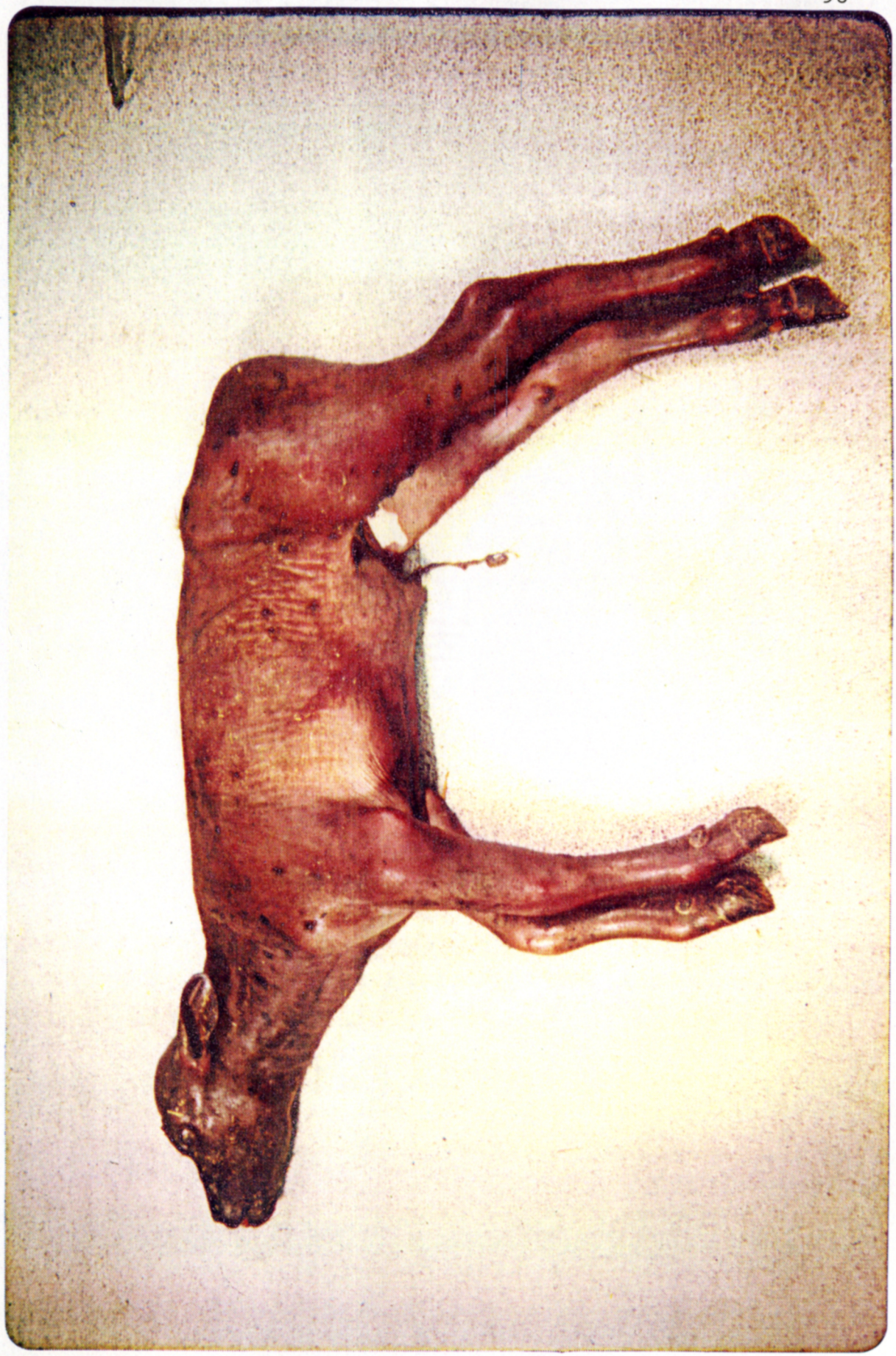


Fig. 6. Skin section from the Angus calf affected with congenital alopecia. Note that epidermal layer is two or three layers thick, undevelopment of the hair follicle, and dilatation of the sweat gland. H&E X 140.

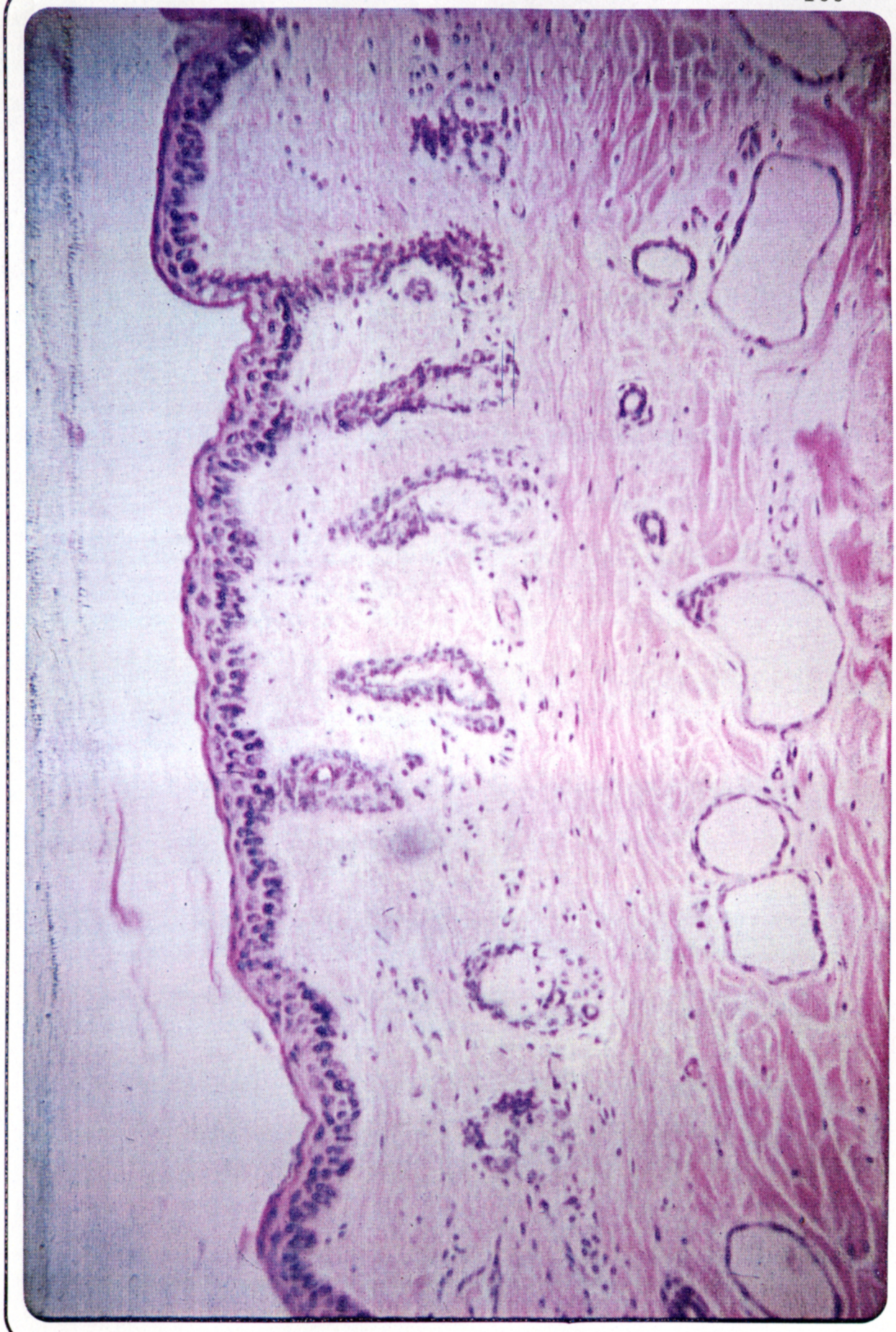
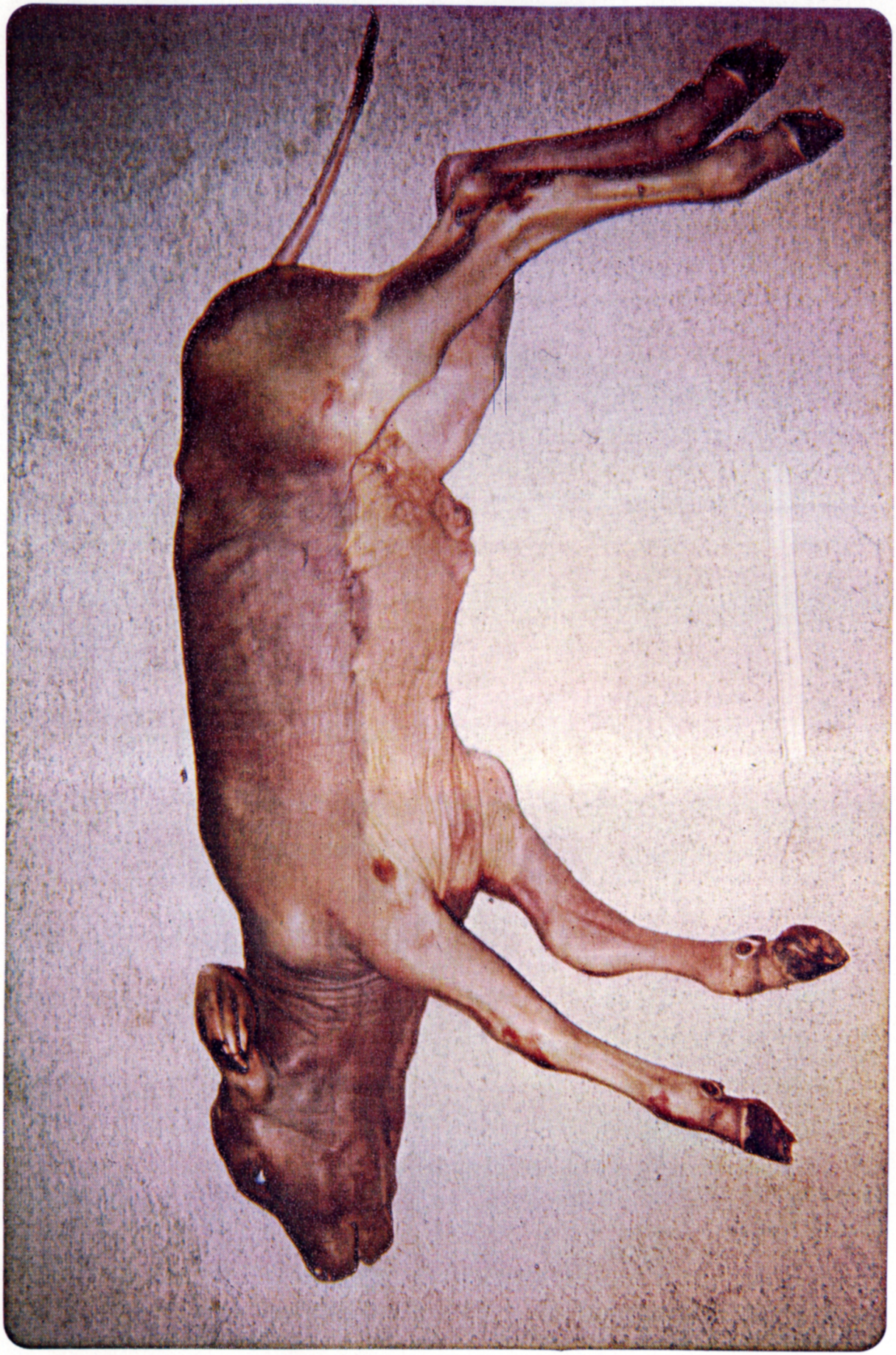


Fig. 7. Purebred male Pinzgauer calf with generalized congenital alopecia.



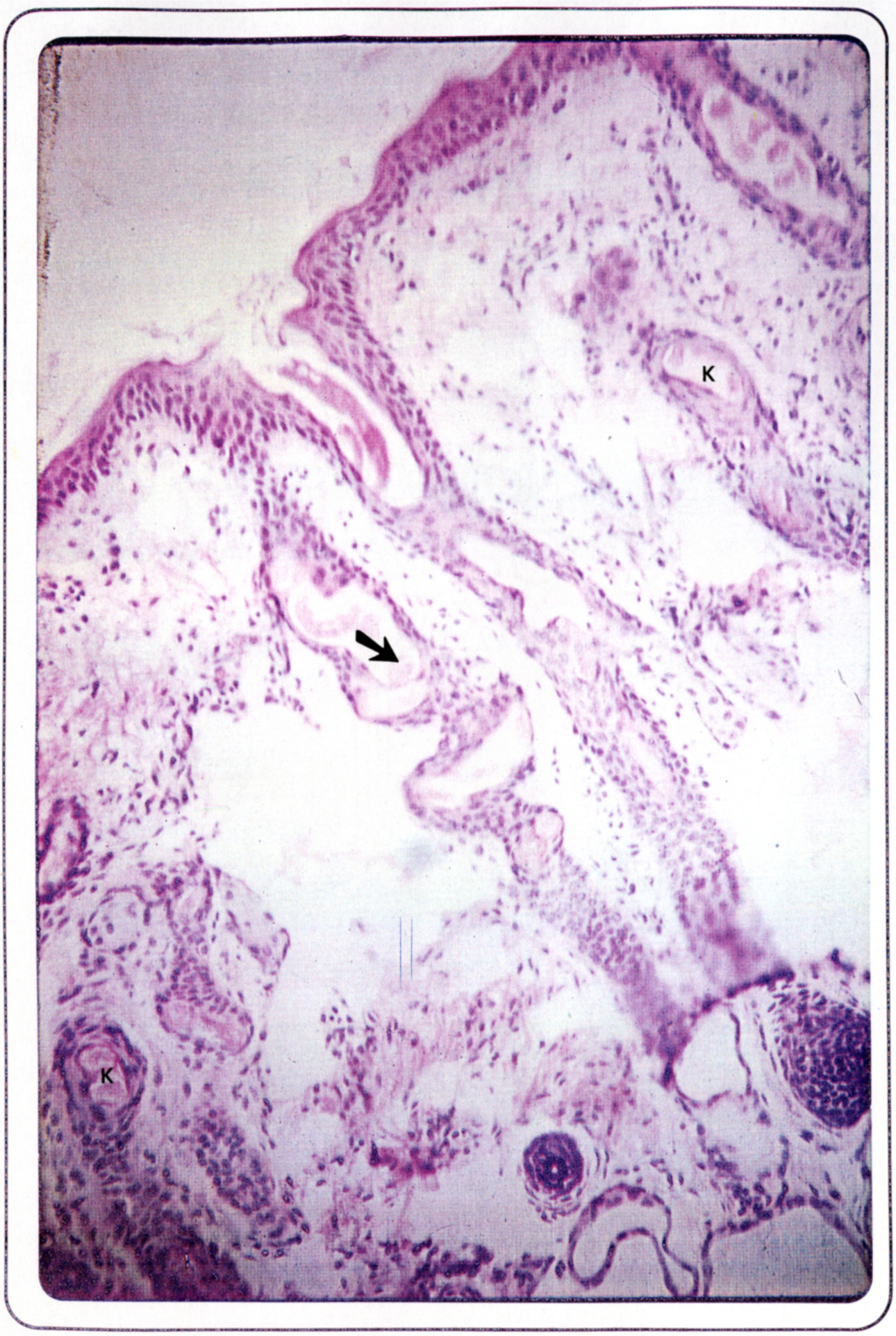

K
K

Fig. 8. Skin section from Pinzgauer calf affected with congenital alopecia. The hair shafts are coiled (arrows) and hair follicles are plugged by keratinous material (K). H&E X 108.

Fig. 9. Purebred female Holstein calf affected with a slight degree of congenital alopecia.

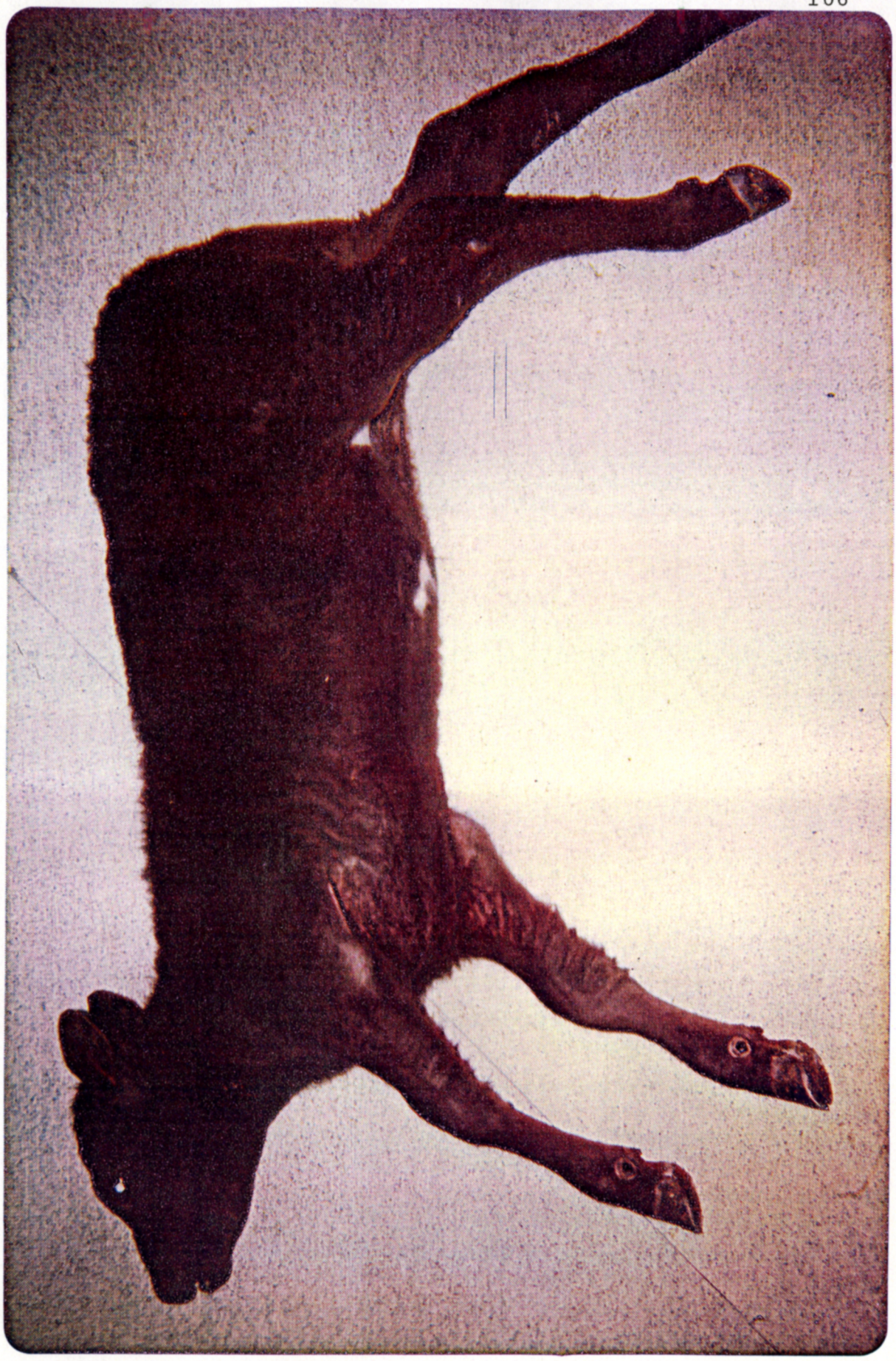


Fig. 10. Mandible of the Holstein calf affected with congenital alopecia. Notice the absence of incisors, anodontia (arrows).

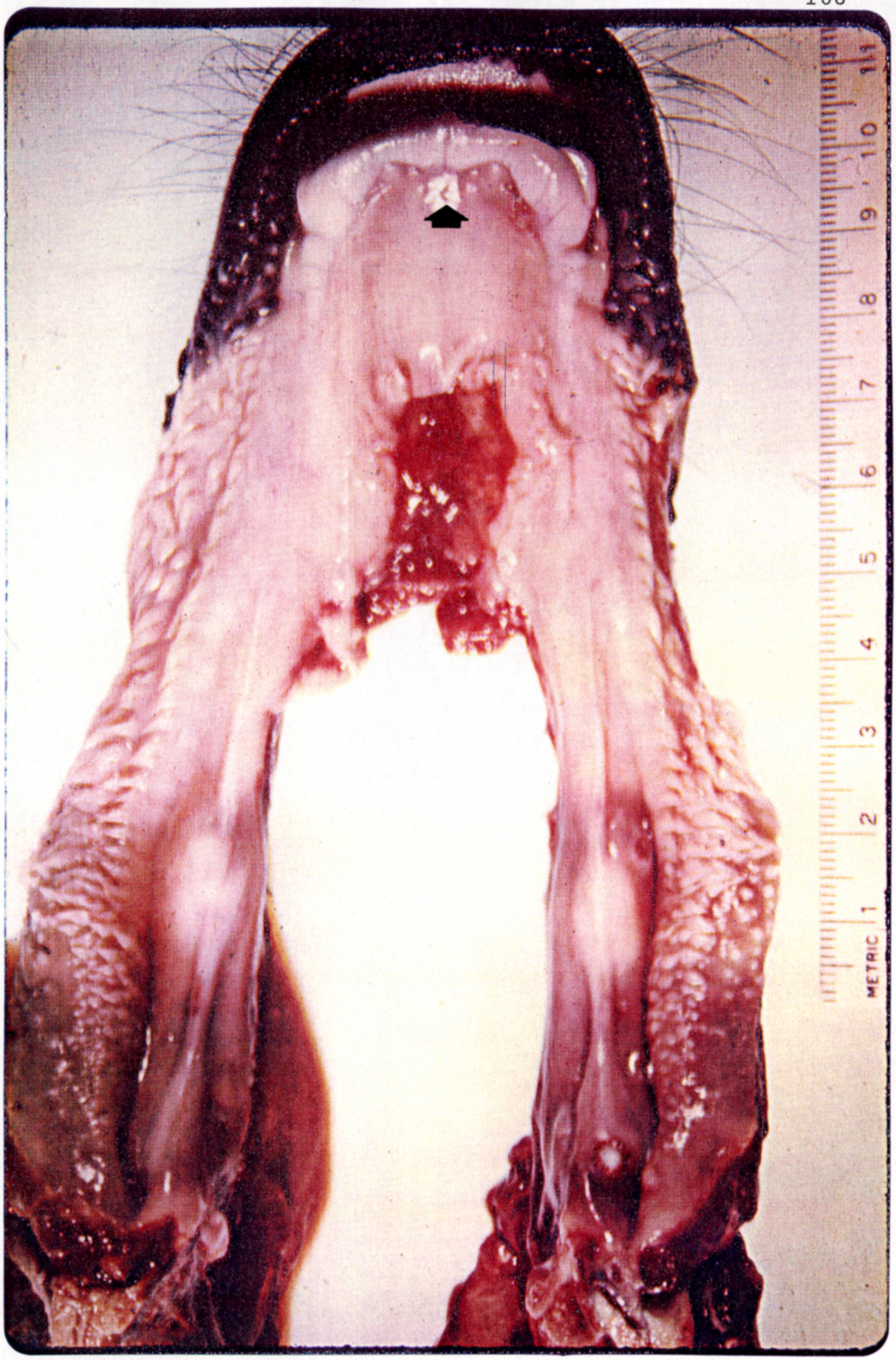
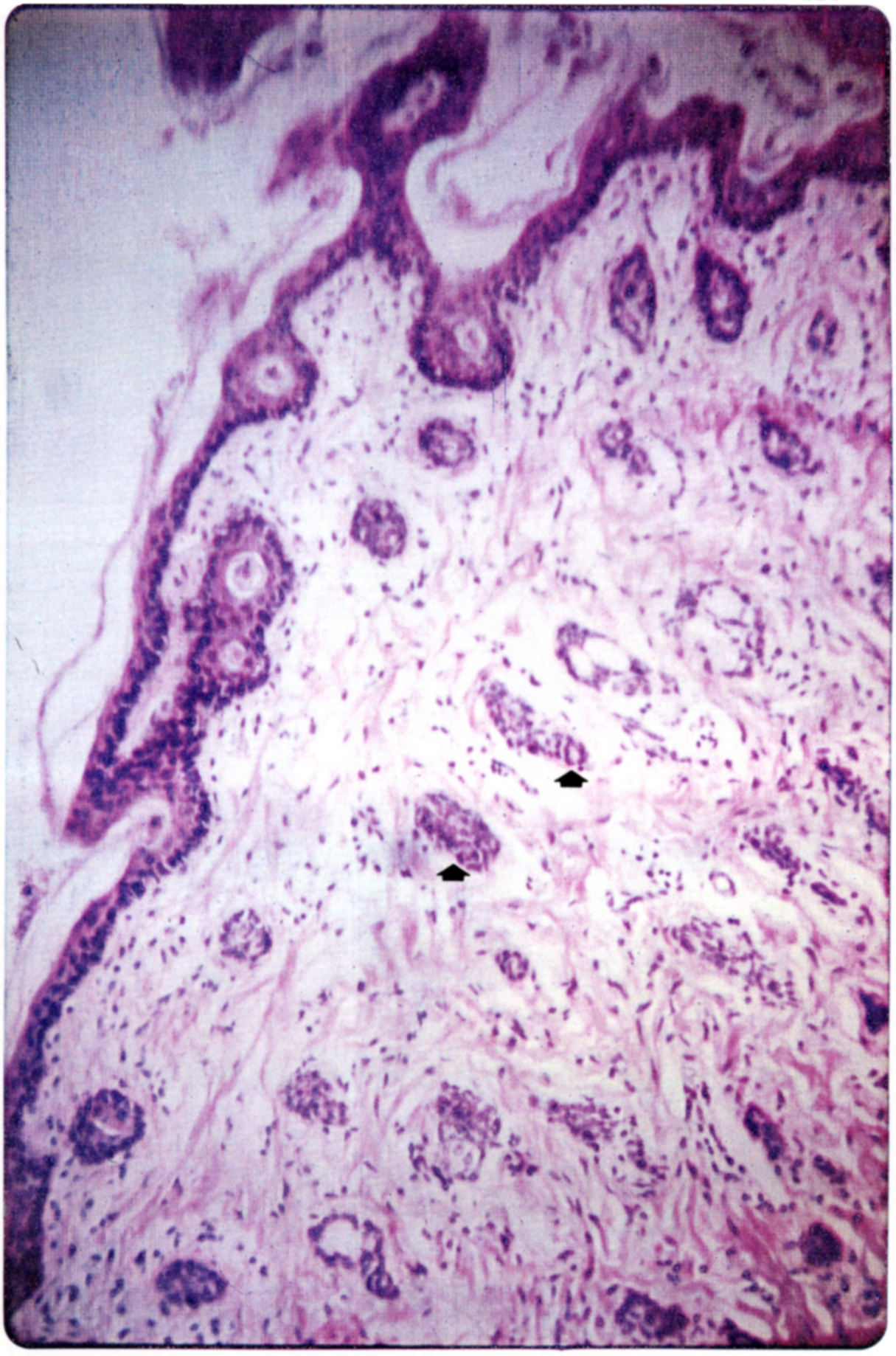

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Fig. 11. Skin section from Holstein calf affected with congenital alopecia. Notice the complete atrophy of the hair follicle (arrows). H&E X 108.



IV. ACANTHOLYSIS AND DERMOID INCLUSION CYSTS IN A SIMMENTAL CALF

INTRODUCTION

Although a relative large number of diseases in man and animals occur in the skin as a genetic condition (Leipold, 1979; Hutt, 1963), there is no conclusive evidence that lack in the development of the stratum spinosum is primarily involved in many of these conditions. Two diseases have been studied in human with primary defect in the desmosomes; Keratosis Follicularis, also called Darier's disease and Familiar Benign Pemphigus or Hailey and Hailey (Level, 1975). It is believed by Charles (1961) that the occurrence of suprabasal acantholysis in human, represents a genetic defect.

Congenital defects have caused economical loses since the breed programs have been checked by genetist-pathologist. This kind of disease increases perinatal calf mortality, decreases maternal productivity, and decreases value of relatives of affected calves (Leipold, 1981).

Veterinary dermatology is a relative young discipline, its contribution as a whole has been of a moderate order. There is much variation in dermatology terminology, mainly in congenital skin disorders. Epitheliogenesis imperfecta, hypotrichosis, congenital alopecia, icthiosis and others, have assigned to a single autosomal recessive gene as hereditary defects, and have been reported in cattle, horses, pigs, and dogs (Rook, 1965; Leipold, 1979; Hutt, 1963; Blood et al., 1974).

The classification of the skin diseases presents some difficulties since the basic pathogenic mechanism and

pathophysiology are not known in the majority of these disorders. It is necessary to classify them according to the presenting signs.

The present paper describes the gross as well as microscopic changes of the skin of an unknown congenital skin disease in cattle.

CASE REPORT

A one week old three quarter Simmental female calf weighing 70 lbs (31.8 kgs) was brought to our attention because of irregular alopecia randomly distributed in the body. Euthanasia was elected because the calf was in poor condition.

GROSS PATHOLOGICAL CHANGES

The skin presented large irregular patches of alopecia on head, neck, shoulder, hip and legs. The hairless areas were scaly and dry. The tail had a little hair. Raw denuded skin areas were present. Some areas of the skin were fissured and bleeding. Blood samples were submitted to the Diagnostic Laboratory at Kansas State University to confirm virus involvement with negative results for IBR, BVD, PI-3, A-3, and A-5.

HISTOPATHOLOGICAL CHANGES

There was separation of the prickle cell layer (acantholysis) resulting in the formation of corps ronds

and grains. Suprabasal acantholysis led to formation of clefts or lacunae which sometimes were filled with inflammatory cells as a secondary change. Irregular proliferation into the lacunae lined by basal cells were present. The stratum corneum was thick (hyperkeratosis), however, other areas had complete absence of corneal cells. These changes were modified by secondary invasion of suppurative bacterias.

Numerous dermoid cysts were present in the subcutis. The cysts varied in size and shape. Most of the present hair follicles projected into the lumen of the cysts. The dermis of the cysts had sebaceous gland and some sweat glands; which tried to form pilosebaceous apparatuses.

The change characterized as corp rond occurred in the stratum of Malpighii, particularly affecting the granular and corny layer. However, acantholytic reaction occurred in the stratum spinosus. Grains were found in the horny layer as acantholytic cells within the suprabasal lacunae, corps ronds possess a central large, round, homogenous, basophilic masses, which were surrounded by a clear halo. The central basophilic masses consisted of dyskeratotic material. This change resembled parakeratotis. Keratin plugs were often founded filling the hair canal. Vesicle formation was sometimes associated with the acantholytic changes in the epidermis, and filled with pus. The Altmann's stain revealed a weak to negative reaction in the dyskeratotic material meaning at least an abnormal keratin production.

DISCUSSION

On the basis of the light microscopic finding presented here one may assume that this disease in a Simmental calf involves a defect in the normal development of the desmosomes and this defect is associated with formation of dermoid cyst generalized in the body. Since the blood samples submitted to confirm virus involvement were negative for IBR, BVD, PI-3, A-3, and A-5; one assumes that this condition is not related with virus diseases. All of the samples of skin investigated under light microscope showed the same type of anomaly of the epidermal layer and the presence of dermoid cyst. The disease seemed to be a generalized defect in the development of the epidermal cells. The pathological characteristics of the disease are very similar to those described in human by Lever (1975) as a Keratosis Follicularis or Darier's disease. The occurrence of suprabasal acantholysis represents a genetic defect as noted by Charles (1961) who believed that the primary defect lies in the desmosomes which fail to form contact layers. Caulfield et al. (1963), Pierard et al. (1968) agreed that separation of tonofilaments from their attachment at the desmosomes is the first change to occur, and that is followed by loss of desmosomes. The grains formation described in the histopathological changes are identical in shape as those described in Darier's disease in human. Their characteristics were strong positive fuchsinophilia with Altmann's stain. However, others remained Fuchsin negative. It seems to have a concordance

with the finding of Dowes (1968) who studied the histology and histochemistry of the Darier's disease and found that the center of corps ronds is Feulgen positive at first, but in the higher layers of the epidermis became Feulgen negative.

Another aspect of the lesions found in our case is related with dermoid cyst. Dermoid cysts are referred in veterinary pathology as a cyst wall to be made up of flattened squamous epithelium surrounded by a dense collagenous capsule, skin adnexa are present in association with the cyst wall (Smith et al., 1972). In our case the condition is very similar to those described by Smith and coworkers (1972).

These two disorders found together in the skin, could be one of genetic origin. Further careful monitoring of offspring of the bull that sired this calf is indicated and breeding trial might be indicated if further cases should occur. Biochemical and ultrastructural studies must be conducted to establish whether the disease in cattle represents the same genetic condition as described by Level (1975) in man.

On the basis of severe clinical picture and clinical course in the calf, it appears likely that this disease could represent a more severe or more complete metabolic defect than congenital alopecia with dermoid cysts and acantholysis.

SUMMARY

A possible hereditary disease seen in one week old three quarter Simmental female calf is described. The skin showed two kinds of disturbances; acantholysis with round corps formation, and dermoid cysts. In the light microscopic it was seen that the stratum spinosum cells do not have desmosomes and skin adnexa was associated with the dermoid cyst. It is concluded that this disease in cattle involved a defect in the normal development of desmosomes and dermoid cyst formation.

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Fig. 1. Three quarter female Simmental calf affected with severe hemorrhagic dermatitis (D) and hairless areas (H).


H
D

Fig. 2. Same calf of Figure 1 with severe dermatitis, and hairless areas.



Fig. 3. Posterior legs showing denuded areas and hemorrhagic dermatitis.

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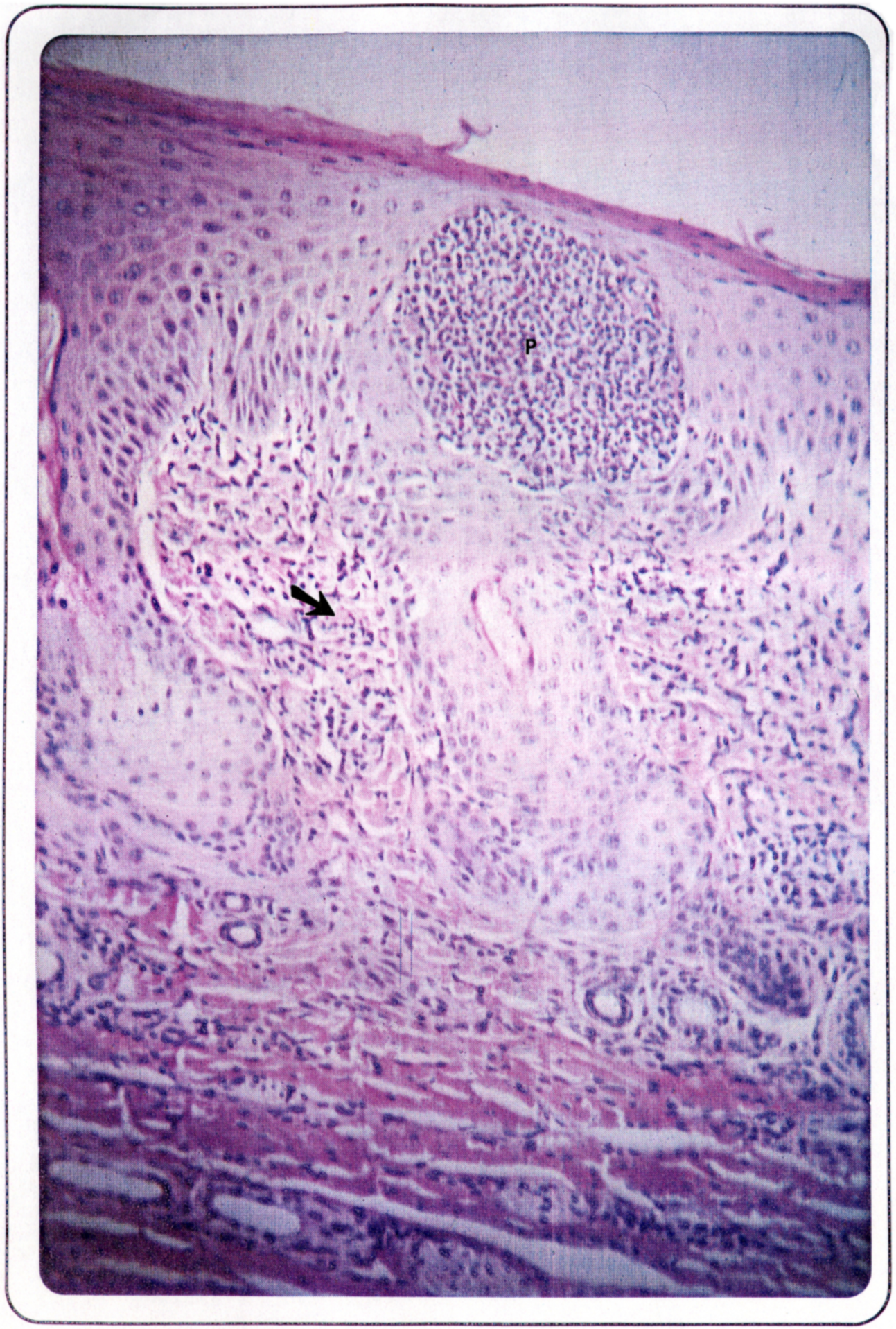
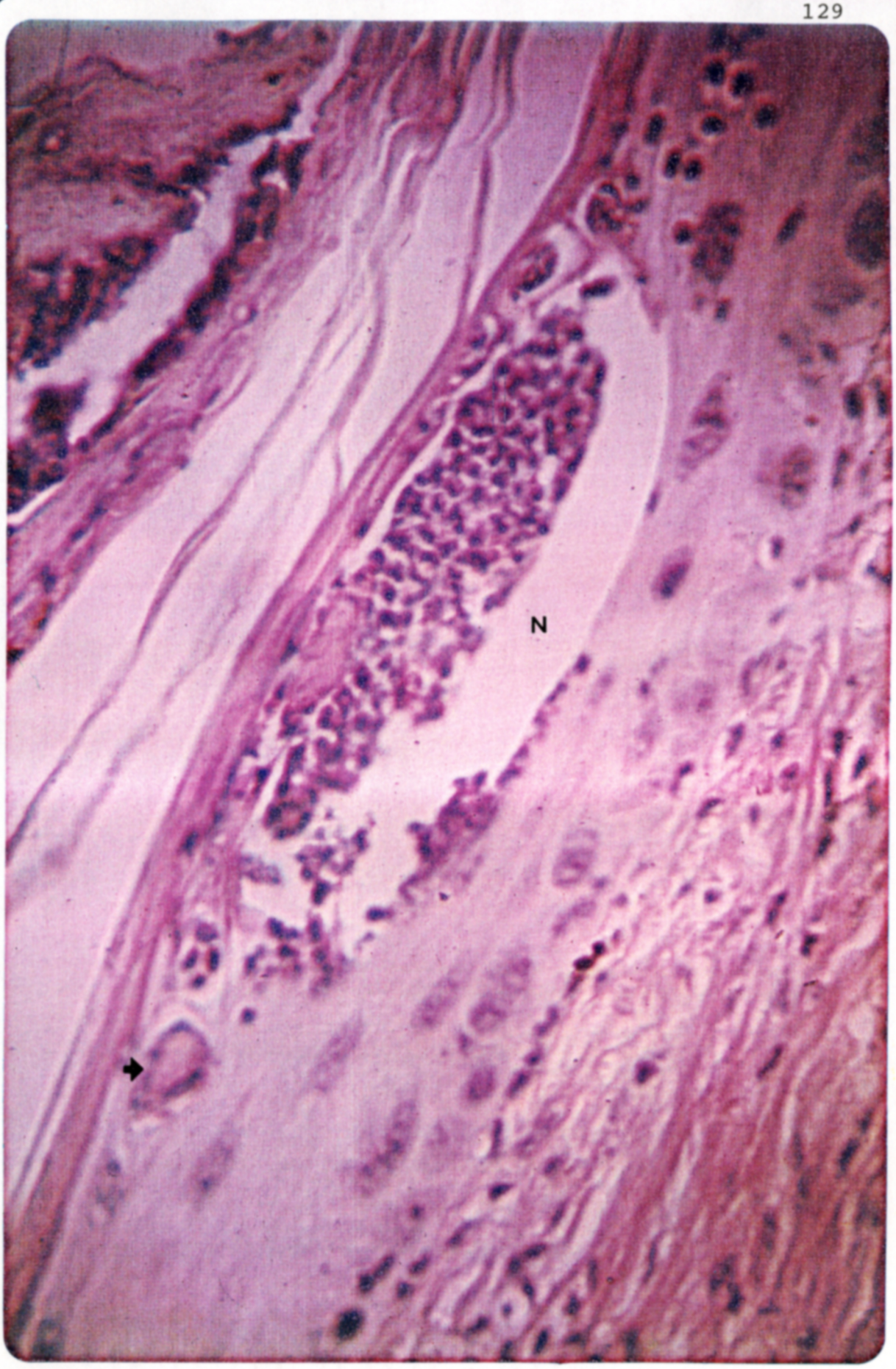

P

Fig. 4. Section of the skin from Simmental calf affected with acantholysis. Notice the subcorneal pustule (P) and inflammatory changes in the dermis (arrows). H&E X 140.

Fig. 5. Skin section of acantholysis. Notice the separation of individual cells (arrow) and neutrophyls filling the subcorneal vesicle (N). H&E X 200.


N

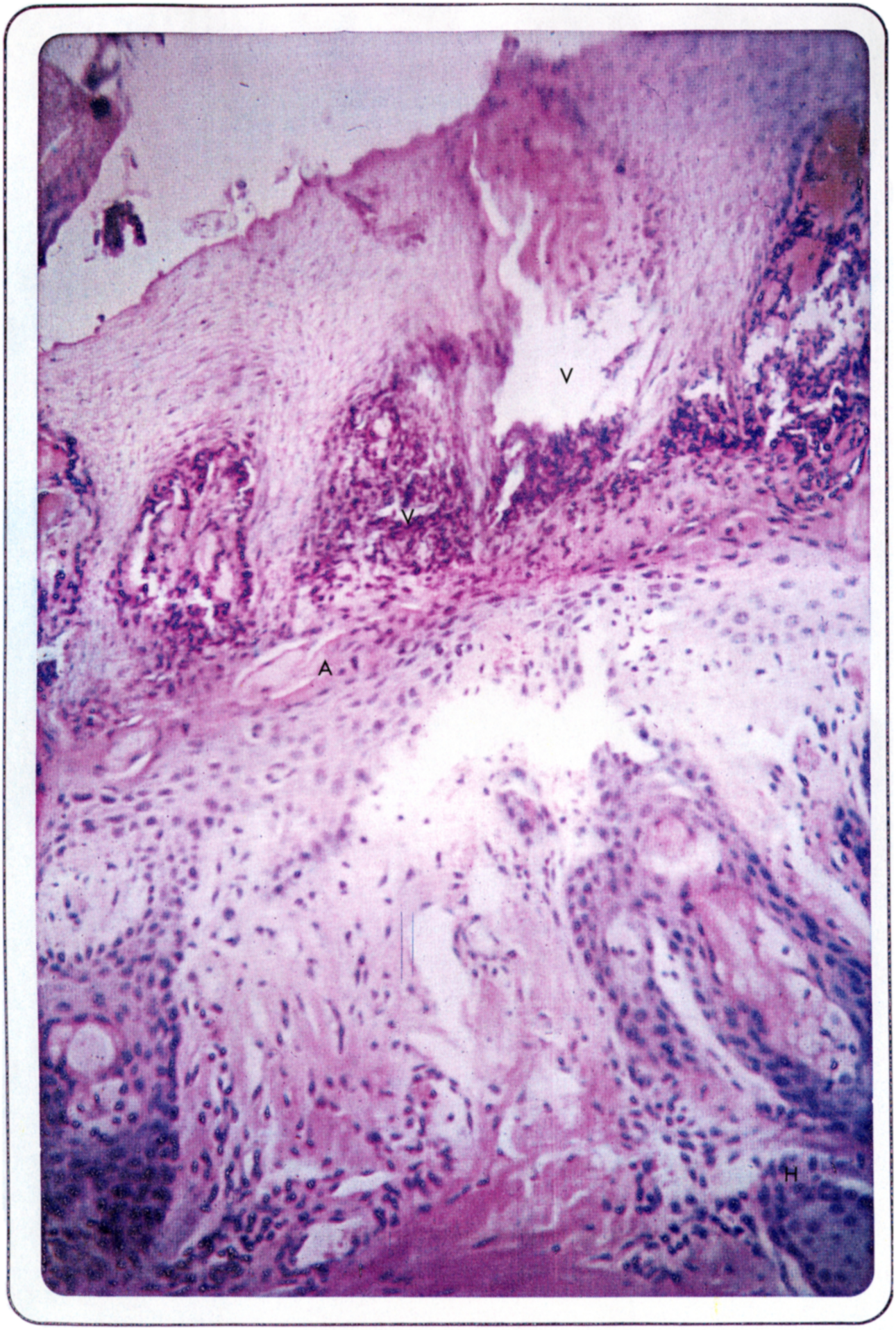

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Fig. 6. Skin section showing atrophy of the hair follice (H), acantholysis (A), and vesicles filled of inflammatory cells (V). H&E X 104.

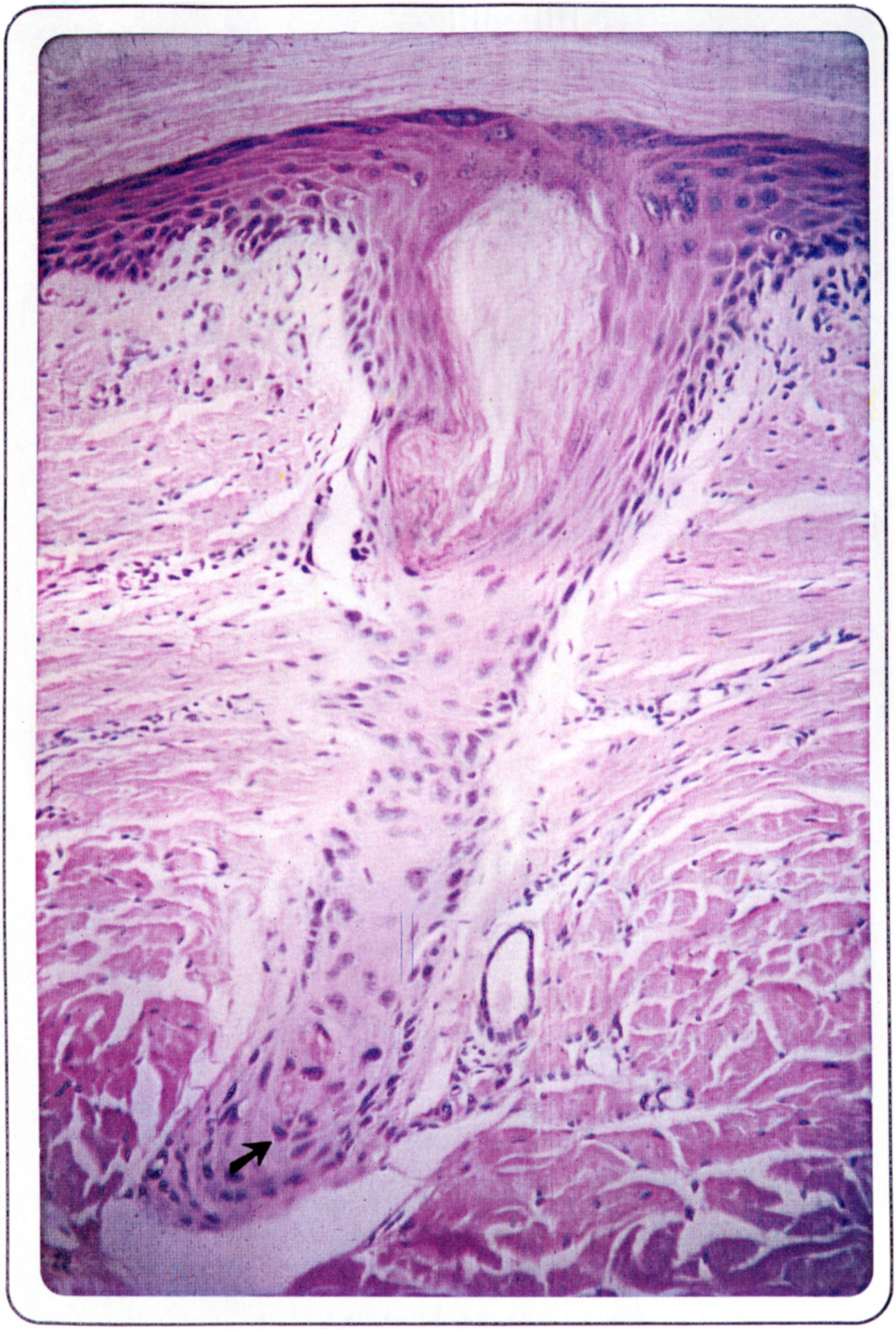


Fig. 7. Skin. Elongated rete pege showing acantholytic change. Notice the basophilia of the cells (arrows). H&E X 140.

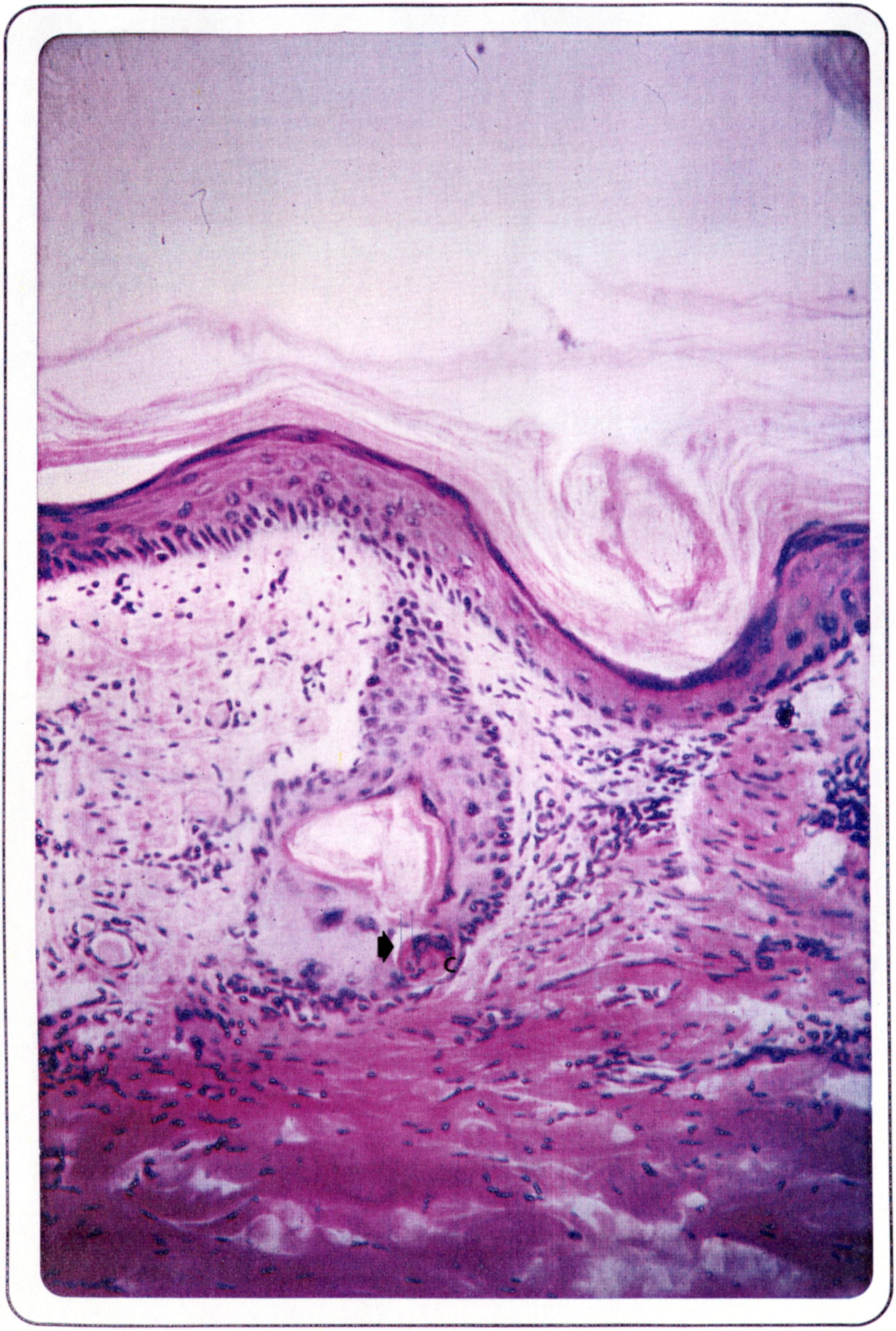


Fig. 8. Skin. Notice a corp rond (C) homogeneous, dyskeratotic mass surrounded by a clear halo (arrow). H&E X 140.

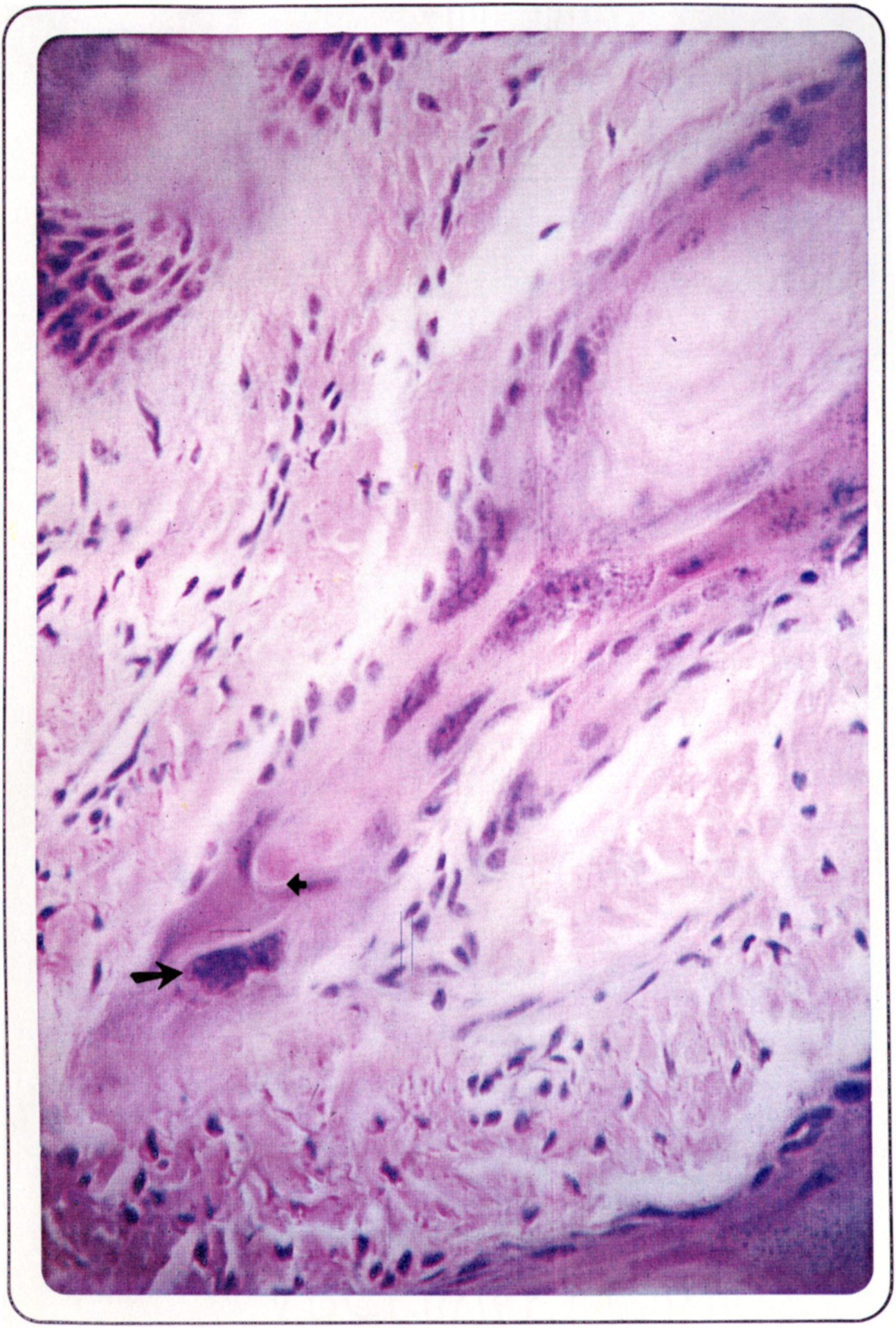
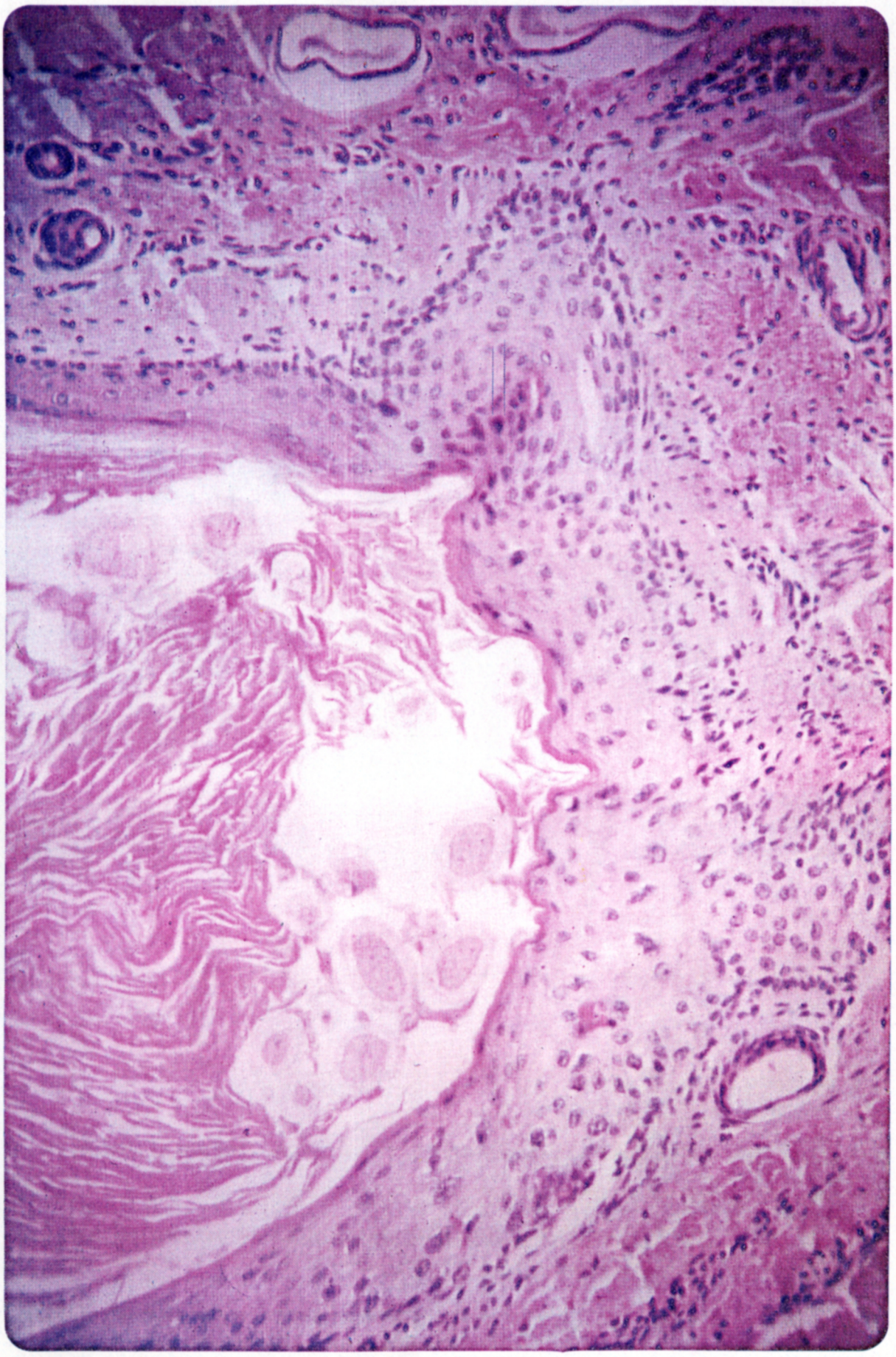


Fig. 9. Skin, high magnification of Figure 8. Notice a basophilic corp rond (arrow) and acantholysis (head arrow). H&E X 180.

Fig. 10. Skin section. Dermoid inclusion cyst. Notice the keratinized material and cross section of hairs shafts in the lumen of the cyst. H&E X 200.



CONGENITAL DEFECTS OF THE SKIN IN CATTLE

by

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D.V.M., Universidad del Zulia, Venezuela, 1969

AN ABSTRACT OF A THESIS

submitted in partial fulfillment of the

requirements for the degree

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1982

Congenital defects of the skin are disorders of structure in their intrinsic embryogenic development present at birth. They are identified as clinical, aesthetic, and economically important diseases in cattle. Pathogenesis and microscopic changes were conducted on congenital hypotrichosis, congenital alopecia, and alopecia, acantholysis and dermoid inclusion cyst in cattle.

The pathogenesis on congenital hypotrichosis of Hereford calves suggested that there was a metabolic abnormality in the transformation of citruline-trichohyaline to arginine trichohyalin, and an environment influence by the climate conditions. The biochemical abnormalities in Huxley's layer resulted in arrest of hair shaft development. This condition had a tendency to be perpetuated in families with a history of genetic involvement.

Observations on congenital alopecia presented evidence that this disease affected at least five different breeds; Hereford, Simmental, Angus, Pinzgauer, and Holstein. Histological data revealed that hair follicle development was arrested in early development and the skin revealed early normal development.

Histopathological studies in a Simmental calf suggested a defect in the normal development of the desmosomes that was associated with generalized dermoid cyst formation.

It is concluded that further studies into the pathophysiology and histopathology of skin are required and that

the mode of inheritance of congenital skin disorders in cattle have an important role in the breeding programs.