

<b>COURSE CODE:</b>	VPT 402
<b>COURSE TITLE:</b>	Nervous, urinogenital, endocrine, skin and musculo-skeletal Pathology
<b>NUMBER OF UNITS:</b>	3 Units
<b>COURSE DURATION:</b>	Three hours per week

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### **COURSE DETAILS:**

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### **COURSE CONTENT:**

[3 units; 2L + 1P]

Gross and microscopic appearance of the nervous, reproductive, endocrine, skin and musculo-skeletal systems in the disease state. Pathology of associated important disease conditions including bovine spongiform encephalitis, focal symmetrical encephalomalacia, enzootic ataxia, cystic ovary, pyometra, pseudocyesis, azoturia, rickets and goiter.

### **COURSE REQUIREMENTS:**

This is a compulsory course for all DVM students and attendance of at least 75% is required to write the examination.

### **READING LIST:**

1. McGavin, M. Donald and Zachary, F. James. 2007. Pathologic basis of veterinary disease, 4<sup>th</sup> Ed. Mosby Elsevier.
2. Jubb, K. V. F., Kenedy, P. C. and Perma, M. 1985. Pathology of domestic animals. 3<sup>rd</sup> Ed. Academic Press, New York.

### **LECTURE NOTES**

## *FEMALE GENITAL PATHOLOGY*

### GENERAL

Diseases of the female genital system consist of a complex interrelationship of endocrine disturbances, infectious diseases, non-infectious diseases, immaturity and senility. These factors, individually or collectively, interfere with ovulation, fertilization and reproduction. Some of these conditions lead to sterility, others to premature expulsion of a dead or living fetus and some are reflected in disease of the offspring.

The organization of the gonads and the basic pattern of the genital tract are established early in embryogenesis. In the early undifferentiated state, each individual is anatomically bisexual and potentially able to develop phenotypic characters of the male or female (independently of the genetic sex). The early gonad consists of cortical and medullary parts. If the medulla predominates, the gonad is a testis; however, if the cortical predominates, the gonad is an ovary. The embryogenesis of the tubular genitalia is also bisexual. If the gonads are testes, the Wolffian duct persists. If the gonads are ovaries, the Mullerian duct persists.

Each structure of the female genital tract (ovaries, uterus, cervix, etc.) tends to react to disease in a characteristic manner. Since the clinical manifestation of disorders of the female genital tract parallel anatomic divisions, this lecture is divided according to anatomic structure.

### 4.3 CONGENITAL ANOMALIES OF THE FEMALE GENITAL TRACT:

#### HYPOPLASIA

Hypoplastic ovaries are normal in immature animals. However, with sexual development, they should reach the normal size and functional state of the species. When certain endocrine disturbances are present, the ovary persists in its infantile form (the condition has been studied extensively only in cattle). The primary morphologic defect in hypoplastic ovaries in cattle is a deficiency in the number of germ cells in the ovary. The degree and severity of hypoplasia may vary considerably; however, the left ovary is more frequently and severely hypoplastic than the right. In severe hypoplasia, there is an absence of follicles and luteal scars. Microscopically, the ovary is composed largely of medullary connective tissue and blood vessels. There is a deficiency of ovarian cortex.

#### FREEMARTIN

The bovine freemartin is a genetic female born co-twin with a male. Over 90% of such females have severe hypoplasia of the genital tract and are subsequently sterile. The pathogenesis of freemartinism has not been

completely elucidated. However, the condition appears to be associated with the fact that the male and female co-twin shares a common placental circulation in- utero.

Androgenic hormones produced by the testicles of the male fetus may enter the circulation and suppress the development of the female genitalia (the male gonads develop earlier than the female gonads and fusion of the allantoic circulation occurs prior to gonadal differentiation).

The morphological degree of modification of the female genitalia may vary. There may be varying degrees of modification from ovaries to testes, or the gonads may be represented by only a cord-like thickening in the anterior border of the ovarian ligament. The tubular genitalia may vary from cord-like structures to well-developed uterine horns. However, communication with the vagina is absent (no matter how well-developed the uterus may be). The vagina is hypoplastic or non-patent with a complete hymen. The vulva and vestibule are hypoplastic and the clitoris is enlarged. Also, the mammary gland fails to reach normal size. The male co-twin may be sterile or fertility may be reduced.

## OVARIES

### INFLAMMATION

The term oophoritis refers to inflammation of the ovaries. The condition is rare. When it occurs, it is usually pyogenic.

### HEMORRHAGE OF THE OVARIES

Hemorrhage of the ovaries occurs during ovulation in all species of domestic animals, but it is usually minimal and confined largely to the collapsed follicles. Intrafollicular hemorrhage occurs in calves (for unknown reasons), in follicular cysts of the bitch and occasionally in atretic follicles of cows. The most significant and severe hemorrhage occurs following manual enucleation of the corpus luteum in cattle (blood loss may be several gallons and result in death of the animal).

### CYSTIC OVARIES

Cystic ovary is the clinical term used to refer to one or more cysts in the ovaries. The condition may occur in all species, but it is most common in cattle and swine. Several kinds of cysts are recognized. These are:

#### CYSTIC GRAAFIAN FOLLICLE

This is the most common form of cystic ovarian degeneration. It arises from failure of the mature follicle to rupture. The condition is most common in heavy-producing cows and may be associated with nymphomania (nymphomania is believed to be due to the estrogenic substance present in the cyst fluid). The pathogenesis of this condition has not been completely elucidated, but there is evidence to suggest that the pituitary gonadotrophin is not released in a normal during early estrus. Thus, the mature follicle is not exposed to the ovulating and luteinizing action of the luteinizing hormone. Grossly, one or more large follicles are present on

one or both ovaries (apart from their greater size, these follicular cysts may be difficult to distinguish from normal follicles). There is an absence of corpus lutea in the ovaries. The uterus and vulva are edematous and flabby. The cervix is enlarged and produces tough gray-white mucus. Also, the clitoris may be enlarged. Microscopically, the ovum is absent and there is degeneration of the granulosa cells.

#### LUTEINIZED CYSTS

Develop when there is delayed or insufficient release of luteinizing hormone during testis. Ovulation fails to occur and the theca interna undergoes luteinization.

Grossly, there is no ovulation papilla, the luteal mass is smooth and rounded and the cyst cavity is spherical and lined by a layer of fibrous connective tissue adjacent to the zone of luteinized theca theca cells. The clinical manifestations of estrus are suppressed but the length of the estrus cycle may be normal. It appears that luteinized cysts are less severe manifestations of cystic graafian follicles and the pathogenesis is probably the same (the two conditions are assumed to be expression of different degrees of the same dysfunction). In cows the develop cystic graafin follicles, there is little or no release of the luteinizing hormone. In cows that develop luteinized cysts, the hormone is slowed and not completed for some days.

#### CYSTIC CORPUS LUTEUM

Occurs following ovulation with the formation of a cystic cavity in the center mass of the developing luteal tissues. Grossly, an ovulation papilla is present and the cystic cavity is irregularly shaped. Pathologic corpus luteum cysts usually measure from 10 to 15mm in diameter (small cystic cavities, less than 5 mm in diameter, are normal and common in cattle shortly after ovulation).

#### PARAOVARIAN CYSTS

Refer to a variety of cystic structure located adjacent to the ovaries. Such cysts may arise from mesonephric or paramesonephric ducts.

#### CYSTIC OVARIES IN HORSES:

Cystic graafian follicles comparable to that of cows and sows apparently do not occur in horses. Remember, the normal mature graafin follicle of the mare is very large (4 to 6cm) and is easily mistaken for a cystic follicle. Also, cystic granulosa cell tumor and germinal inclusion cysts can be confused with cystic follicles.

#### CYSTIC FOLLICLES IN DOGS AND CATS

Functional graafin follicular cysts can be responsible for nymphomania, vulva tumefaction, cystic endometrial hyperplasia, cystic mammary hyperplasia and genital fibroleiomyomas in dogs and cats. Functional corpora luteal cysts can cause exaggerated progesterational proliferation (cystic endometrial) of the endometrium. Thus,



the cystic corpus luteum (as well as the retained corpus luteum of pseudopregnancy) makes the uterus more susceptible to infection because of excessive progesterational activity. Pyometra is a common sequela.

#### EXTRAOVARIAN LESIONS OF CYSTIC OVARIES IN COWS:

The changes that occur in various organs and tissues of cows with cystic ovaries (cystic graafian follicles) are believed to be due to the effects of estrogen. This is especially so in those cows exhibiting signs of nymphomania. The cyst fluid contains estrogenic substances. Large amounts of estrogen are actually found in normal mature graafian follicles. However, the toxicity of estrogen depends not only on the level of circulating estrogen, but more important, on the continuity of its action, even though exciting levels are relatively minute. In established cases of follicular cysts in cattle, the following may be observed:

- \*Pituitary gland is enlarged, especially the anterior lobe. This enlargement is due chiefly to stromal hyperplasia, edema and cellular hypertrophy.

- \*Thyroid glands are enlarged and moderately hyperactive. The ultimo brachial remnant exhibits some degree of squamous metaplasia.

- \*Adrenal cortex (especially the zona fasciculate) is widened due to cellular hypertrophy. The presence of altered function is not known.

- \*Cervix is enlarged and the external os is patent.

- \*Uterus is enlarged, doughy, or atonic and the wall is edematous.

- \*Vagina and vulva are edematous. In prolonged cases, estrogen stimulation results in squamous metaplasia of the vaginal epithelium.

#### ATRETIC FOLLICLES

Atresia is a normal process for those follicles that do not attain maturity (during the cycle of uniparous animals, many follicles develop but only one is supposed to mature and ovulate. The remainders undergo atresia, degenerate and disappear).

Follicular growth is independent of pituitary gonadotrophins up to the stage of antrum formation. Thereafter, growth and maturation depend on stimulation by pituitary gonadotrophins in proper proportion and sequence. Debilitated and malnourished animals are capable of synthesizing the gonadotrophins and the ovaries are capable of responding to them. However, the defect is in the pituitary gland, which does not release its stored gonadotrophins nor does so in insufficient quantities.

#### NEOPLASMS OF THE OVARIES:

Primary ovarian tumors occur most commonly in the bitch and cow.

#### GRANULOSA CELL TUMORS

Arise from the granulosa cells of the ovarian follicle. These tumors have the potential to produce estrogen or androgen. Androgenic activity in an ovarian tumor may be result of disturbed chemistry in a primary estrogen-

producing neoplasm. Cows and mares with granulosa cell tumors may show no significant clinical signs or they may exhibit signs of nymphomania or act like males. The clinical signs are believed to be due to excessive estrogen production by the neoplasm. In the bitch, granulosa cell tumors are likely to produce cystic endometrial hyperplasia (probably associated with the production of progesterone).

Grossly, the tumor surfaces are smooth and the incised surfaces may be cystic or solid. Microscopically, there are gland-like or rosette patterns of abortive follicles (some tumors contain secretory globules resembling an ovum which is referred to as "Call-Exner"). Also, granulosa cell tumors may develop a tubular pattern similar to that of sertoli cell tumors of the testes.

#### DYSGERMINOMAS

Are rare ovarian tumors with gross and microscopic features similar to seminomas of the testes. However, no hormone is secretory and the origin of the dysgerminomas is unknown.

Surface epithelial tumor/serous cystadenomas.

#### FALLOPIAN TUBES:

Primary lesions in the fallopian tubes (oviducts) are rare. Hydrosalpinx, pyosalpinx and salpingitis may occur but these are usually secondary to diseases of the uterus or to manual manipulation of the ovaries. SALPINGITIS refers to inflammation of the oviduct. The inflamed oviducts are seldom significantly enlarged and lesions are usually not detectable with the naked eye. The exudates may be serous, catarrhal or fibrinous. Salpingitis is usually the result of extension of infection from the uterus. PYOSALPINX refers to an accumulation of purulent exudate in the oviducts. It usually follows metritis. HYDROSALPINX refers to an accumulation of fluid in the oviducts following some form of obstruction. In addition, fluid filled cysts may be present in the oviducts.

#### UTERUS:

##### INFLAMMATION OF THE UTERUS:

Inflammation of the uterus limited to the endometrium is termed endometritis. Involvement of the entire uterine wall is referred to as metritis. Inflammation of the uterine serosa is called perimetritis.

The normal non-pregnant uterus is highly resistant to infections (even specific genital organisms such as Brucella, Vibria and Trichomonas are incapable of persisting for long periods in the non-pregnant uterus). The exact mechanism of this resistance has not been completely elucidated. However, experimental evidence indicates that the uterus under the influence of estrogen (as in the case of the normal non-pregnant uterus) is highly resistant to infection. On the other hand the uterus under the influence of progesterone (as in the case of the pregnant uterus) is highly susceptible to many specific and non-specific bacterial infections.

#### ENDOMETRITIS

Endometritis refers to inflammatory processes in which the endometrium is primarily involved (actually, the reaction is limited to the endometrium only in very mild forms of infections). Almost all uterine infections begin as an endometritis, but they may progress very rapidly to involve the entire wall. There is a tendency for *Vibrio fetus* and *trichomonas fetus* to cause mild infections limited to the endometrium. In mild forms endometritis, gross lesions are absent or insignificant. Microscopically, there is a mild infiltration of inflammatory cells (the best indication of mild metritis is the presence of infiltrated plasma cells and lymphocytes in the stroma because a few neutrophils may be present in a normal uterus). A more severe form of endometritis occurs following parturition. Grossly, the uterus is enlarged, flabby and collapsed (rather than firm and contracted). The uterine lumen contains chocolate-colored, with or without a foul odor. The endometrium is reddened, swollen and edematous.

#### METRITIS

Metritis is characterized by inflammation of the entire uterine wall (including the endometrium). The term septic metritis is oftentimes used when referring to severe and often fatal inflammation of the uterus. Metritis is usually due to infections introduced at or shortly after parturition. Organisms most likely to be responsible are streptococci, staphylococci and other pus-formers. Grossly, the uterine wall is flaccid, thickened, reddened, edematous and friable (rupture may occur and result in a secondary peritonitis). There may be a scanty or abundant secretion of fetid uterine exudates via the vagina. Microscopically, inflammatory cells may be found throughout the uterine wall (in acute cases, neutrophils dominate, whereas in chronic cases, lymphocytes are prominent). Severe metritis may lead to a septicemia. However, cases may recover with proper treatment.

#### ABSCESSATION OF THE UTERUS:

Uterine abscesses occur most frequently in cows. They often result from localization of infection to one part of the uterine wall following severe metritis or localized traumatic injury.

#### PYOMETRA

Pyometra is an acute or chronic suppurative infection of the uterus with an accumulation of pus in the lumen in the presence of a "closed" cervix (the cervix may not be completely closed but there is never a heavy discharge from the uterus). The condition occurs with frequency only in the cow, bitch and cat. There are two primary mechanisms.

- (1) Mechanical obstruction to uterine discharge and
- (2) Functional obstruction to uterine discharge.

#### PYOMETRA ASSOCIATED WITH MECHANICAL OBSTRUCTION TO UTERINE DISCHARGE

This occurs following infections in which the cervix becomes indurated and stenotic, thereby preventing the expulsion of uterine exudates. This type of uterine involvement occurs most frequently in mares as a sequela to prolonged endometritis. The incriminated organisms are usually *Streptococcus zooepidemicus* or *Klebsiella pneumonia var. genitalium* which typically produce vaginitis, cervicitis and endometritis.

## PYOMETRA ASSOCIATED WITH FUNCTIONAL OBSTRUCTION TO UTERINE DISCHARGE

- \* (1) Increasing the susceptibility of the uterus to infection.
- \* (2) Maintaining functional closure of the cervix and
- \* (3) Inhibiting myometrial contractions.

Thus, infections, superimposed on the effects of retained corpus luteum and progesterone production, may lead to obstructions.

In the bitch and cat, most cases of pyometra are due to infections which are superimposed on endometrial hyperplasia and are, therefore, secondary to prolonged hormonal imbalance (production of progesterone by the retained corpus luteum). The majority of cases occur during pseudopregnancy (in pseudopregnancy, the corpus luteum is retained and secretes progesterone). Pyometra is a more acute lesion in the bitch than in cattle.

### PATHOLOGY

Grossly, the uterine horns are distended with purulent exudates (pus) and the cervix is completely or almost completely closed. Streptococcal and staphylococcal organisms produce typical purulent exudates. However, if the uterus is infected with *E. coli* or *Proteus*, the pus is thick, viscid and reddish-brown with a characteristic fetid odor. There is usually cystic hyperplasia of the endometrium. Extragenital lesions are common in dogs and cats and are due to severe intoxication and probably also to an intermittent bacteraemia. There is selective bone marrow depression which leads to anemia and severe extramedullary hematopoiesis. Leukocytosis is also observed.

In cattle, pyometra is characterized by an accumulation of pus in the lumen of the uterus (a few ounces to more than a gallon) which is thick and cream or grayish-green in color. There is no cervical seal of mucus and a small amount of pus escapes into the vagina. The uterine wall is thick and doughy or it is thin and fibrosed. Pyometria in cattle is usually asymptomatic and there are no extragenital lesions recognized.

### NOTES:

**RETAINED CORPUS LUTEUM** following ovulation, a corpus luteum develops from the ruptured follicle and, when fully mature, dominates the contour of the ovary. In the absence of fertilization and pregnancy, the corpus luteum normally degenerates shortly before the next ovulation. If pregnancy occurs and proceeds normally, the corpus luteum persists throughout the period of gestation, normal or aborted (except for the mare among the domestic species). The corpus luteum of pregnancy begins to regress shortly before parturition and subsequently undergoes rapid regression (it disappears in 7-10 days after parturition): A corpus luteum that persists beyond these physiologic ranges is considered to be retained with pathologic implications. The reason(s) for the retained corpus luteum is not definitely known. However, this may be due to a disturbed hormone imbalance resulting in excess luteinizing hormone which maintains the corpus luteum indefinitely.

## ABNORMAL ENDOMETRIAL GROWTH:

### ATROPHY OF THE ENDOMETRIUM

Endometrial atrophy may be associated with ovariectomy, hypopituitarism, a pituitary lesion or chronic inanition/wasting disease. Grossly, the endometrium is flat, thin and grayish. Microscopically, only remnants of endometrial glands remain.

### HYPERPLASIA OF THE ENDOMETRIUM:

Endometrial hyperplasia may be cystic or non-cystic/cystic hyperplasia (something referred to as “Swiss cheese” endometrium) is an extreme degree of hyperplasia. Endometrial hyperplasia is probably an exaggeration of the normal proliferative changes of the endometrium in response to ovarian hormone. In the bitch, prolonged progesterone stimulation is the major factor in the induction of endometrial hyperplasia. However, in cattle, endometrial hyperplasia is associated with prolonged estrogenic stimulation (hyperestrogenism as seen with ovarian follicular cysts and with granulosa cell tumors) in sheep, endometrial hyperplasia has been reported in animals grazing on pasture legumes with high estrogenic activity (especially *Trifolium subterraneum* and *T. pratense*). These plants may have similar effects in cattle.

Non-cystic hyperplasia of the endometrium is usually not recognizable grossly. In cystic endometrial hyperplasia, the uterine wall is soft, thick and spongy. There are numerous submucosal cysts that bulge into the lumen of the uterus. Infection superimposed on endometrial hyperplasia is common only in the dog and cat. Endometrial hyperplasia is a significant precancerous lesion in the human. This is not true in domestic animals.

### ADENOMYOSIS (endometriosis):

Adenomyosis is a condition characterized by the presence of endometrial glands and stroma between muscle fibers of the myometrium. The condition may result from congenital malformations or it may arise subsequent to hyperplasia and overgrowth of the endometrium.

### TORSION OF THE UTERUS:

Torsion of the uterus may occur in any species, but the condition is most frequently observed in pregnant cattle. Torsion of 180 degrees or more will cause dystocia and circulatory embarrassment. Minor twists of 90 degrees or less are fairly common and will apparently resolve themselves. In uniparous species (cow), the twist occurs at the level of the mesovarium, vagina or cervix with involvement of the entire uterus (these animals have a well-developed intercornual ligament which prevents very much independent movement of the uterine horns). In multiparous animals (bitch, cat), there is no intercornual ligament and the torsion may involve part of one horn or it may involve the entire uterus.

### PROLAPSE OF THE UTERUS:

Uterine prolapse occurs with some frequency in ruminants. The condition occurs most commonly after parturition in the previously gravid uterine horn. The pathologic sequelae are comparable to those of intestinal intussusception with the added factor of trauma.

#### THE PREGNANT UTERUS:

The pregnant uterus is under the influence of estrogen and is highly susceptible to a variety of disease and conditions.

#### EMBRYONAL DEATH:

A degenerate ovum, zygote or early embryo may be absorbed or expelled from the uterus. The incidence of zygotic and early embryonal death is estimated to be high (15-30%) in all species studied extensively. Early embryonal death is usually manifested by signs of infertility and irregular estrus cycles in the female (there may be no evidence of an aborted fetus or lesions in the genital tract of the female). The causes of such mortality are presumably diverse; however, chromosomal abnormalities may play an important role.

#### FETAL DEATH

A developing dead fetus may be mummified, macerated or aborted. (Abortion may be defined as the expulsion of a fetus prior to the time of expected viability. Whereas, a fetus delivered within the time of expected viability is referred to as a stillborn).

In uniparous domestic species, the dead fetus may be expelled or retained. In early pregnancy, when the main source of progesterone is the maternal ovary, death of the conceptus results in resorption, maceration or mummification. However, later in gestation, when the progesterone which maintains pregnancy is produced primarily by the fetal-placental unit, death is usually followed by expulsion of the fetus.

In multiparous species, it is most common for one or several dead fetuses to be retained with the viable ones and delivered at parturition. However, if most of the fetuses die at the same time, all are likely to be aborted (live and dead fetuses).

#### MUMMIFICATION OF THE FETUS:

A mummified fetus is one that dies and undergoes subsequent autolysis (there is an absence of infection and putrefaction does not occur). This condition occurs most commonly in multiparous species. In mummification, the fetal fluids are resorbed and the soft tissues gradually liquefy. These fluids are currently resorbed via the maternal blood and lymphatics. Eventually, the entire fetus becomes brown or black and leather-like (there is no exudates or odor). A completely mummified fetus consists of dried wrinkled skin and bones. The time required for complete mummification will depend on the size of the fetus. It takes about 6 to 8 months in the case of a 6-month-old bovine fetus. In uniparous species, the mummified fetus may be retained indefinitely.

#### MACERATION AND EMPHYSEMA OF THE FETUS

Maceration refers to a dead, retained fetus that undergoes autolysis in the presence of a uterine infection. The usual causes of fetal death and maceration are not potent gas producers. If the cervix is open and putrefactive organisms are able to invade the uterus and dead fetus, the results is fetal emphysema.

#### ADVENTITIAL PLACENTATION:

The development of intercotyledonary placentation occurs primary in ruminants. This is a mechanism of compensation for inadequate development of the placentomes (which is primarily endometrial). There are normally between 75 and 125 caruncles in the cow. However, this number may be reduced due to infection or congenital disorders. Subsequently, compensation occurs through a great increase in size of the remaining caruncles during pregnancy and by the development of a more primitive villous placentation between the placentomes. The process may involve virtually the entire intercotyledonary placenta. Abortion and hydrallantois are common complications.

#### AMNIOTIC PLAQUES:

Amniotic plaques are foci of squamous epithelium on the internal surface of the amnion. These plaques are flat and measure between 2 and 5mm in diameter. They resemble the lesions of the poxes. Amniotic plaques are more numerous on the umbilical slump. The plaques are apparently normal and they constantly occur on the bovine placenta during the middle trimester of gestation.

#### PLACENTAL CALCIFICATION:

Placental calcification refers to the presence of visible calcium deposits around small blood vessels of the placenta. These deposits appear as white streaks or spots in the amnion and allantois. They occur in many species from about the end of the first trimester to the end of the second trimester. The reason(s) for this deposition of calcium is apparently no ill effect on the fetus.

#### HYDRAMNIOS AND HYDRALLANTOIS:

Hydramnios and hydrallantois refer to an accumulation of fetal fluid in the amniotic and allantoic cavities, respectively. Normally, there is 6 to 15 liters of allantoic fluid and 3 to 6 liters of amniotic fluid in the cow with the maximum amounts begin reached around mild-gestation. The fluid is then progressively decreased until term in hydramnios and hydrallantois, the excessive fluid may reach a total volume of up to 50 gallons. The pathogenic mechanism is seldom determined; however, the excess fluid should have an obstructive origin. The fluid may accumulate slowly or rapidly and there is usually an accompanying fetal anasarca. Important sequelae are dystocia, uterine paresis, retention of the placenta and acute metritis. Also, abortion may occur.

#### SOME SPECIFIC CAUSES OF ABORTION IN ANIMALS:

##### BRUCELLOSIS

Bacteria of the genus *Brucella* are small, Gram-negative bacilli or coccobacilli which prefer an intracellular habitat. Important species include.

- (1) *Brucella abortus* which occurs chiefly in cattle,
- (2) *B. Melitensis* of sheep and goats,
- (3) *B. Ovis* of sheep,
- (4) *B. Suis* of swine and
- (5) *B. Canis* of dogs.

All of the *Brucella* organisms are capable of producing systemic infections with relapsing bacterial phases. Localization and persistence of infection may occur in many organs and tissues (especially in the genital tract and in the placenta).

*Brucella abortus* causes abortion around the 7<sup>th</sup> and 8<sup>th</sup> months of gestation. The non-pregnant uterus is not highly susceptible to infection and, following abortion, the organisms disappear from the uterus. However, the organisms have a special affinity for the pregnant placenta and endometrium. Grossly, the pregnant uterus is not remarkable. The placenta may be present. There is mild to severe necrosis of the cotyledons (they are soft and yellow). A dirty yellow sero-purulent exudate develops between the endometrium and chorion in the intercotyledonary areas. Affected areas of the intercotyledonary placenta are thickened. Opaque, tough and leather-like in appearance. There is usually edema of the placenta and umbilical cord. Microscopically, the placenta is edematous and there is an infiltration of neutrophils and mononuclear cell. In the fetus, the most important lesion is a bronchopneumonia which may be mild or severe. The abomasal contents are turbid, yellow and flaky (normal abomasal contents should be clear, translucent, thick and viscous).

*Brucella melitensis* is considered to be a major cause of abortion in sheep and goats. In most respects, the disease resembles that caused by *Brucella abortus* in cattle. However, mastitis is an early sign and the milk is watery and clotted. Organisms are excreted in the milk.

*Brucella ovis* causes placentitis and abortion in the ewe (the organism is not very pathogenic compared to the other *Brucella* spp.) Periarteritis and arteritis are distinctive features of the disease. Placental lesions are similar to those described for *Brucella abortus* in cattle. The fetus is edematous and fluid in the body cavities may contain strands of fibrin. Also a mild bronchopneumonia and a lymphadenitis may be observed in the fetus. In general, the greatest susceptibility to intrauterine is from 21 to 90 days of gestation.

*Brucella suis* is the organism of importance in swine (however, swine are susceptible to *B. abortus* as to *B. melitensis*). Transmission is primarily via coitus. The uterus lesions consist of multiple yellow-white nodules or granulomas that vary in size from 1 to 3mm in diameter. The placenta may be normal or it may be congested and edematous with many small hemorrhages. The fetuses are edematous and their stomach contents may be yellowish and slimy. In general, abortion occurs between the 2<sup>nd</sup> and 3<sup>rd</sup> months of gestation. There is also a high incidence of stillborns and weak piglets delivered at term. The placenta may or may not be retained.



*Brucella canis* has been incriminated as the cause of abortion and epididymitis in the dog. Aborted fetuses may be dead or alive at the time of expulsion. Live pups usually die within a few hours after birth. Lesions in the fetuses consist of pneumonia, endocarditis and hepatitis. Affected bitches may exhibit a sero-sanguinous vaginal discharge for 1 to 6 weeks after abortion.

#### VIBRIOSIS

Bovine genital vibriosis is venereal disease caused by *Vibrio fetus venerealis* and characterized by infertility and early embryonal death (abortion may occur in a small percentage of infected cows). Coitus is the usual means of transmission. Temporary infertility and irregularity of the estrus cycle may be the only clinical sign noted in infected cows (irregularity of the estrus cycle is believed to be due to interruption of very early pregnancy by infection). There may be a very mild endometritis, cervicitis and vaginitis. In the small percentage of cows that abort, the abortion occurs around the 5<sup>th</sup> to 7<sup>th</sup> months of gestation. Grossly, placental lesions are similar to those described for *Brucella abortus* in called, but they are less severe. Many of the cotyledons are yellow and necrotic and sero-purulent or sero-fibrinous exudates present between the endometrium and chorion. The intercotyledonary areas are thickened, edematous and leather-like. Following infection, there is no permanent maternal injury.

#### EPIZOOTIC BOVINE ABORTION:

Epizootic bovine abortion (sometimes referred to as Chlamydial abortion or "foothill" abortion) is an infectious disease caused by a strain of *Chlamydia psittaci* and characterized by abortion between the 5<sup>th</sup> and 7<sup>th</sup> months of gestation. The disease has been reported primary from the western U. S. A.

Fetal lesions consist of petechial hemorrhages which may be found throughout the subcutaneous tissue, oral cavity and in the conjunctiva. The fetal liver is enlarged and friable with a granular or roughened surface. Microscopically, there is a diffuse or focal proliferation of reticuloendothelial cells in many organs and tissue (liver, spleen, brain, lymph nodes, myocardium, etc.). Also, foci of necrosis may be found in the liver and spleen.

#### LISTERIOSIS: (Please refer to your Central system Notes)

Abortion due to *listeria monocytogenes* occurs during the last trimester of gestation in both sheep and cattle. If uterine infection develops during the early part of the last trimester, the placenta is quickly invaded by the organisms and the fetus dies as a result of septicemia. The dead fetus is expelled in approximately 5 days. By this time, autolytic changes will mask any pathologic lesions that may have been present. However, if the fetus is near term when the infection develops, dystocia occurs and there is severe metritis with septicemia of the fetus. The placental lesions consist of necrosis that involves the tips of villa. Multiple foci of necrosis may be found throughout the fetal liver.

#### EQUINE VIRAL RHINOPNEUMONITIS:

The equine herpesvirus I causes abortion in mares between the 8<sup>th</sup> and 11<sup>th</sup> months of gestation. The most consistent lesion in the fetus is severe lung edema. The fetal liver is characterized by multiple tiny foci of necrosis. Microscopically, intranuclear inclusion bodies are found in the liver, spleen and bronchial epithelial cells.

#### GENITAL TRICHOMONIASIS:

Trichomoniasis is a contagious protozoan disease of cattle characterized by sterility, pyometria and abortion. Infection in the bull remains in the preputial cavity indefinitely. Characteristically, trichomoniasis in the female results in signs of infertility and irregular estrus cycles. A few days after infections, an acute vaginitis develops with a mucoid discharge. Subsequently, the vaginitis resolves and the infection localizes in the uterus and cervix, resulting in endometritis and cervicitis. There may be a rather copious (up to a gallon) accumulation of purulent exudates in the uterus (pyometrial). There is no specific characteristic placenta or fetal lesions.

#### MYCOTIC ABORTION

Mycotic abortion occurs sporadically in cattle and it is usually caused either by *Aspergillus* or *Mucor* (*Absidia* and *Rhizopus* have also been incriminated). These agents apparently reach the uterus via the blood stream causing abortion in late gestation. There is usually necrosis of the cotyledons and thickening of the intercotyledonary areas. Fetal skin lesions may be present and they appear as irregular elevated plaques (resembling the lesions of ringworm).

#### CERVIX INFLAMMATION

Inflammation of the cervix (cervicitis) is usually an extension of uterine or vaginal infections. Actually, cervicitis is seldom a separate disease entity. Most inflammations are superficial. In general, the mucous-secreting epithelium of the cervix provides a good defense against bacterial invasion.

#### VAGINA AND VULVA: INFLAMMATION

Inflammation of the vagina and vulva is a frequent sequela of parturition in which the mucous membranes are bruised or lacerated. Other causes of vaginitis and vulvitis include malicious injury foreign bodies, etc. The inflamed vagina is painful, reddened and edematous; necrosis and a fetid discharge are common.

#### INFECTIOUS PUSTULAR VULVOVAGINITIS OF CATTLE

Is caused by the same virus as that which causes infectious bovine rhinotracheitis. In general, the respiratory and vaginal infections behave as separate and distinct disease entities (however, the syndromes may occur together in individual animals).

Infectious vulvovaginitis is an acute contagious disease characterized by inflammation, necrosis and pustule formation on the mucosa of the vulva and vagina (occasionally, similar lesions may occur on the skin of the penis and prepuce). The syndrome is frequently transmitted by coitus, but it can also be transmitted by other mechanical means. Initially, there is hyperemia of the vaginal and vulval mucosa and focal hemorrhages in lymphoid follicles of the submucosa. Subsequently, pock-like lesions (2 to 3 mm in diameter) which are slightly elevated, pale, soft and friable, replace the hemorrhage over the lymphoid follicles. The affected epithelium becomes necrotic, ulcerates or erodes. Microscopically, intranuclear inclusion bodies may be observed in epithelial cells early in the disease. The disease usually subsides in 10-12 days.

#### MAMMARY GLAND

##### INFLAMMATION OF THE MAMMARY GLAND:

The term mastitis refers to inflammation of the mammary glands. The condition is greatest economic importance in dairy cattle; however, all species may be affected.

##### BOVINE MASTITIS:

The causes of mastitis in cattle are diverse with more than 50 species of bacteria and more than 20 species of fungi being incriminated. Factors that predispose to mastitis are poor milking hygiene, faulty milking machines, trauma, etc. The bacterial pathogens most commonly recovered from mastitis (in approximately decreasing order of frequency) are *Staphylococcus aureus*, *Streptococcus agalactiae*, other streptococci, coliform organisms, *Corynebacterium pyogenes* and *Pseudomonas aeruginosa*.

The morphological patterns of mastitis are usually divided into several types based largely on severity and duration of the disease.

##### ACUTE CATARRHAL MASTITIS

Is characterized by a mild systemic response of short duration. Individual quarters are affected. Secretions may be blood stained and contain flakes of inspissated pus. Grossly, affected lobules are pale, firm and project above the cut surface. Flakes of purulent exudates are present in the cisterns and lactiferous ducts. Edema may be mild or pronounced. Microscopically, the alveoli within lobules are filled with neutrophils.

##### ACUTE HEMORRHAGIC MASTITIS

Is characterized by marked swelling of the affected quarter and by a severe systemic response which is often fatal. The small amounts of secretory that can be obtained consist of sero-sanguinous or fibrinous exudates. Grossly, the tissue is intensely hyperemic and hemorrhagic with areas of necrosis. Gangrene may occur.

##### CHRONIC MASTITIS

Is usually a sequela of acute mastitis. However, even in the chronic stages, foci of active (acute) inflammation may be present. There is no systemic involvement. Grossly, the affected tissue is tough and smaller than normal (due to proliferation of fibrous connective tissue and glandular atrophy).

### SUPPURATIVE MASTITIS

Usually associated with *Corynebacterium pyogenes* or *Pseudomonas aeruginosa* infection. Multiple abscesses are present.

### GRANULOMATOUS MASTITIS

Is the response to chronic inflammation by a variety of organisms (*Mycobacterium*, *Nocardia*, *Cryptococcus*, etc.).

### STREPTOCOCCAL MASTITIS

*Streptococcus agalactiae* mastitis is one of the more important forms of bovine mastitis (presently, the condition can be controlled via therapy).

The organisms depend on mammary tissue for survival and the mammary gland of the cow and goat is the natural and sole habitat. The organisms enter the mammary gland via the teat canal and reside in the milk or on the surface of the milk channels (they may persist without causing an inflammatory process). Under certain conditions (altered virulence, etc.), the epithelial cells lining the teat channels are briefly invaded and acute inflammation may follow. However, inflammation of mammary tissue is due primarily to the diffusion of toxic products. Once an inflammatory process is initiated, the infiltrating neutrophils will rapidly destroy the invading organisms (this destruction of organisms may at times sterilize the udder or decrease the numbers so that bacterial count taken during a crisis may be negative).

Grossly, more than one quarter is usually involved but not uniformly. Most of the changes take place in the distal portion of the gland around the cisterns and larger ducts. In the acute stages, the glandular tissue is swollen and turgid and lobulations are distinct on the cut surfaces. The affected mammary tissue is grayish (whereas normal lactating tissue is more or less white in color). In the chronic stages, fibrosis becomes prominent. Microscopically, the acute stage is characterized by an exudative process in which there is severe edema and an extensive infiltration of neutrophils into interlobular tissue and acini. The acinar epithelial cells become vacuolated and they desquamate. This acute exudative phase gives rise to pathologic fibrosis and/or involution. Penicillin is highly effective against *Streptococcus agalactiae*.

Other *Streptococci* that may cause mastitis include *Streptococci dysgalactiae*, *Streptococci uberis*, *Streptococci zooepidermicus*; *Streptococci pyogenes*, *Streptococci fecalis* and Lancefield groups of G and L *Streptococci*.

### STAPHYLOCOCCAL MASTITIS:

*Staphylococcus aureus* is considered to be the most important cause of mastitis in most dairy areas. The organism is ubiquitous and can colonize the skin as well as the udder. Antibiotic treatment is not always successful for certain isolates. *Staphylococcus aureus* is capable of causing peracute, acute, subacute, chronic, gangrenous and subclinical types of mastitis. The acute form of the disease usually occurs shortly after parturition and tends to produce gangrene of the

affected quarters with high mortality. Grossly, the affected tissues are swollen, tense, hot, firm and painful. Milk secretion is reduced. Gangrenous tissues become blue and eventually black in color.

The acute non-gangrenous forms of the disease are characterized by lesions similar to those described for Streptococcal mastitis. However, the Staphylococcal organisms have the ability more deeply into the interacinar tissue and establish as persistent foci of infection (a granulomatous type reaction may be provoked).

#### COLIFORM MASTITIS:

Coliform mastitis is usually caused by *Escherichia coli* and *Aerobacter*. Under certain condition, Coliform organisms are capable of multiplying very rapidly, producing large pool of potential endotoxin. The resulting toxemia is responsible for the local and systemic sign of mastitis. The body temperature ranges from 103 to 108 degrees F. Milk secretion ceases and there is anorexia, depression, dehydration and rapid weight loss. The infection is usually limited to one quarter and the course is rather short (a unique feature coliform mastitis is that the udder tissue usually returns to normal following an acute infection). If for some reason the course is prolonged, extensive necrosis occurs. Eventually, fibrosis is a prominent feature. Remember, gangrene is not produced by coliform bacilli alone.

#### CORYNEBACTERIUM PYOGENES MASTITIS

*Corynebacterium pyogenes* is commonly associated with suppurative mastitis in cattle and swine. The condition may involve lactating and dry cows. There is evidence that *C. pyogenes* is actually a secondary invader in most cases of mastitis. The disease is characterized by the presence of multiple abscesses.

#### NEOPLASMS OF THE MAMMARY GLAND

Mammary neoplasms occur frequently and are often fatal in the dog (less frequent in the cat). The hard worked bovine udder is quite susceptible to inflammation, but neoplasms are rare. Also, mammary tumors are seldom observed in other domestic species.

In the dog, mammary tumors may consist of epithelial and/or connective tissue components. Mammary tumors represent approximately 25% of all neoplasms in female dogs, about 50% of these tumors are benign mixed mammary tumors and about 25% are adenocarcinomas. The reminders are adenomas, duct papillomas, myoepitheliomas and malignant mixed mammary tumors.

#### MIXED MAMMARY TUMORS

Contain neoplastic cells of both glandular (epithelial) and stromal (connective tissue) origin. Also, there may be a myxomatous portion represented by a proliferation of myoepithelial cells (myoepithelial cells play a role in the production of basement membrane material and cartilage). Frequently, the connective tissue elements will undergo metaplasia with the formation of cartilage and bone (thus, some mixed tumors may consist primarily of

cartilage and bone). In addition, the glandular and stromal elements may assume the neoplastic characteristics of anaplasia, hyperchromatism, hypercellularity, etc.

The canine mixed mammary tumors are related to the endocrine status of the individual. Ovariohysterectomy has a negative effect on tumor formation. Bitches neutered before their first estrus have only 0.5% of the mammary cancer risk of the intact female.

#### ADENOCARCINOMAS OF THE MAMMARY GLAND

These are similar to those that occur in other organs and tissues. These tumors are characterized by a proliferation of neoplastic epithelial cells with the formation of acini or duct-like structures.

#### FIBROADENOMAS

Occur with some frequency in the mammary tissue of aged rats (this is the most frequent benign tumor of the human female breast). These tumors are composed of both connective tissue and glandular elements. Grossly, these tumors are firm, grayish-white, with grayish-yellow specks. Microscopically, there is a loose connective tissue network with pleomorphic glandular and cystic spaces.

# SKIN PATHOLOGY

BY

DR O. L. AJAYI

## Introduction

This consists of the skin and its associated structures like adnexa (sweat and sebaceous glands), nails, scales, feathers and hair.

The skin is the single largest organ in the body and represents a physical barrier to the animal to protect them from environmental hazards.

It is the visible and accessible organ in the body; however it is by far the most overlooked both clinically and at PM examination.

## Important Functions of the Skin

- Prevents fluid loss and heat loss against external physical and chemical agents.
- It conserves critical substances particularly H<sub>2</sub>O
- It synthesizes vitamin D
- It excretes excess water, urea etc.
- It stores carbohydrates and fats
- It also filters harmful ultraviolet rays from the sun
- It is an organ of immunocompetence like dendritic cells which present antigens to the immune system

Though the skin is flexible, soft in some areas and elastic, it is

vulnerable to damage by chemical or physical or biological insults.

It is divided into 3 layers:

- a. Subcutis or hypodermis
- b. Dermis
- c. Epidermis

## COMMON TERMINOLOGIES USED IN SKIN PATHOLOGY

- Macroscopic Terms
- Macule: a spotted area of <1.0cm in size characterized by its flatness and usually distinguished from the

normal surrounding skin by its colouration which may be reddish. When it is >1cm it is called patches.

- Papule: this is an elevated solid area of 5mm or less in diameter.
- Nodule: an elevated solid area greater than 5mm in diameter.
- Plaque: this is an elevated flat topped area usually greater than 5mm in diameter.
- Vesicle: this is a fluid-filled area of 5mm or less in diameter.
- Bullae: this is a fluid-filled raised area greater than 5mm in diameter. It is also known as a large blister.
- Blister: a common term used to describe a vesicle and pustules
- Pustule: this is a discrete pus-filled raised area.
- Wheal: this is an itchy transient elevated area with variable blanching which could be reddish or bluish or erythema formed as a result of dermal edema especially in light skins or hairless areas.
- Excoriation: this is a traumatic lesion characterized by the breakage of the epidermis causing raw or linear or jagged area. It is caused either by barbed wire or rough substances or from scalpel blade cut.

### Microscopic Terms

(A) Hyperkeratosis: this is an increase in the thickness of the stratum corneum. It may be absolute, which is more common, or relative which occurs due to the thinned underlying epidermis. This can be of 2 forms:

- a. Orthokeratotic hyperkeratosis: this is an increase in the anucleated stratum corneum layer.
- b. Parakeratotic hyperkeratosis: increase in the nucleated S. corneum layer.

Both are common findings in any chronic dermatosis. They simply imply altered epidermopoiesis, whether inflammatory, hormonal, neoplastic or developmental in origin.

The presence of diffuse parakeratotic hyperkeratosis is most consistent with ectoparasitism, Zinc responsive dermatoses, some vitamin A-responsive dermatoses, thalotoxicosis, dermatophilosis and dermatophytosis.

Diffuse orthokeratotic hyperkeratosis suggests endocrinopathies, nutritional deficiencies, secondary seborrheas and developmental abnormalities ( Ichthyosis, hypotrichosis, colour mutant alopecia).

(B) Hypokeratosis: this is an decrease in the thickness of the s. corneum. It is much less common than hyperkeratosis and reflects an exceptionally rapid epidermal turnover time and/or decreased cohesion between cells of the s.corneum. This may be found in seborrheic and other exfoliative skin disorders. It may also be



produced by excessive surgical preparation of the biopsy site or by friction and maceration in intertriginous areas.

### Microscopic Terms contd.

- Dyskeratosis: this is a premature and faulty keratinization of individual cells of s.corneum. The cells are characterized by swollen and diffusely eosinophilic cytoplasm and condensed dark staining nuclei. The condition is seen in pemphigus complex, lichenoid dermatoses, papilloma, squamous cell carcinoma, keratoacanthoma and warty dyskeratoma.
- Hyperplasia: this is an increased thickness of the non-cornified epidermis due to an increased number of epidermal cells. The term acanthosis is often used interchangeably with hyperplasia. However, acanthosis specifically indicates an increased thickness of the stratum spinosum and is usually due to hyperplasia and occasionally to hypertrophy of cells of the c. spinosum. This condition is often accompanied by rete ridge formation in which “pegs” of epidermis appear to project downward into the underlying dermis.
- Hypoplasia: is decreased thickness of the non-cornified epidermis due to a decreased number of cells. Atrophy is decreased thickness of the non-cornified epidermis due to a decreased size of cells. An early sign of epidermal hypoplasia or atrophy is the loss of the rete ridges in areas of the skin where they are normally present.

### Microscopic Terms contd.

- Acantholysis: loss of intracellular connections resulting in intraepidermal clefts, vesicle and bullae. Acantholysis may be caused by severe spongiosis, ballooning degeneration, proteolytic enzymes released by neutrophils or eosinophils in inflammatory process, developmental defects as in bovine familial acantholysis and neoplastic transformation as in squamous cell carcinoma, actinic keratosis and warty dyskeratoma. Marked acantholysis is seen with the autoimmune pemphigus complex.
- Exocytosis: is the migration of inflammatory cells and /or erythrocytes through the intercellular spaces of the epidermis. Exocytosis of inflammatory cells is an indication of any inflammatory dermatoses.
- Erosion: this is a discontinuation of the skin exhibited as an incomplete loss of the epidermis with the basement membrane intact. This should be differentiated from epitheliogenesis imperfecta which is a developmental defect.
- Ulceration: discontinuity of the epidermis with loss of basement membrane, portions of the dermis and subcutaneous part. It may be seen as a raw, bleeding surface often contaminated and may contain granulation tissue, pus or scab.
- Hyperpigmentation(Hypermelanosis): this is the presence of excess melanin deposited in the epidermis. It may be focal or diffuse or confined to the basal layer. It is seen in chronic inflammatory and neoplastic dermatoses.
- Hypomelanosis: presence of decreased melanin in an area otherwise normally high pigmented e.g. iris. It may be associated with congenital or idiopathic defects in melanization.

Spongiosis

Acantholysis

Macules

Vesicle/ pustule

Pustules

Crusts

Lichenification

Nodules

Wheals

Orthokeratotic hyperkeratosis

Parakeratotic hyperkeratosis

Epidermal dysplasia

Epidermal spongiosis

#### Dermal Defects

- Dermal collagen hyalinization: this is the degenerative change of dermal collagen in which there is confluence, increased eosinophilia.
- Dermal fibroplasias: characterized by increase in the formation of and development of fibrous tissue element in the dermis. It is often synchronously found along with granulation tissue in which there is accentuation of the vascular element within the tissue.
- Fibrosis of the skin: this is the latter of fibroplasias and is characterized by increased fibroblasts and collagen as well as decreased vasculature of the dermis. Sclerosis or scar tissue formation is usually the end point of dermal fibrosis.
- Papillomatosis: this is the projection of the dermal papillae above the skin surface. It is seen in chronic inflammation and neoplastic dermatoses. It is associated with epidermal hyperplasia. There are 2 types:

- a. villus type- these are dermal papillae covered by one or two layers of epidermal cells projecting into a vesicle or bullae.
- b. fistulous type – this is devoid of cellular covering and also projection into a vesicle or bullae.
- 

### DEGENERATIVE CONDITIONS OF THE SKIN

- These include

1. Hydropic vacuolar or ballooning degeneration seen in viral diseases especially and in conditions of irritation by exogenous substances.
2. Vesiculation and bullae formation.
3. Abnormal depositions e.g. mineral, amyloid, carbohydrates etc.

Hydropic degeneration

### CONGENITAL ANOMALIES

- 1. Epitheliogenesis imperfecta: this is congenital inherited, discontinuation of the squamous epithelium of the skin and oral mucosa membrane. It occurs occasionally in calves and piglet and extremely rare in dogs.
- 2. Congenital Ichthyosis: this is the presence of cutaneous lesions resembling fish scales. This skin is composed of large horny plates separated by deep fissures. It is seen in calves and dogs.
- 3. Congenital alopecia: this is hairlessness of varying degrees at birth. It is often related to iodine deficiency. It may be partial to complete absence of hair. Seen in all domestic animals.
- 4. Vegetative dermatoses: this is an inherited disease of land race breed of pigs. It is characterized by hairless, papillomatous plaques on the skin and giant cell pneumonia with fetalization of alveolar wall.

### VIRAL DISEASES OF THE SKIN

- Pox infection in domestic animals
- Aetiology
- The poxviridae share group-specific nucleoprotein antigen. The genera include
- a. Orthopoxvirus e.g. Cowpox, Vaccinia, Rabbit pox, Monkey pox, Buffalo pox and Camel pox.
- b. Capripoxvirus e.g. Sheep pox, goat pox, lumpy skin disease pox, etc.
- c. Avipoxvirus e.g. fowl pox, pigeon pox etc.
- d. Leporipoxvirus e.g. Myxoma rabbit, hare and squirrel fibromas.
- e. Suipoxvirus e.g. swinepox.
- f. Parapoxvirus e.g. Contagious Pustular Dermatitis, Papular Stomatitis, pseudopox.
- g. Unclassified – the molluscum contagiosum agents and horse pox virus.

- Mode of Transmission is usually via respiratory or cutaneous route.
- Pathogenesis- pox virus induces lesions by a variety of mechanisms:
  - a. they cause degenerative changes typically hydropic degeneration in the epithelial cells. This is induced by viral replication and leads to vesicular lesions characteristic of many poxvirus infections
  - b. Others induce degenerative lesions in the dermal or sub mucosal tissues as a result of ischaemia secondary to vascular damage due to viral multiplication in endothelial cells.

Pox infections contd.

- Pathology Gross
- Pox lesions have a typical developmental sequence. They commence as erythematous macules, become papular and then vesicular. The vesicles develop into pustules with depressed centres and raised erythematous border. This is the so called pock lesion.
- The pustules rupture and a crust forms on the surface. The crust may eventually form scab or ulcers.
- Histopathology
- - This consists of typically epidermal cytoplasmic swelling, Hydropic degeneration and vesiculation usually first affecting the cells of the stratum spinosum.
- - ruptured cells give rise to multiloculated vesicles and these may coalesce to form bullae.
- - the intercellular edema makes the epidermis spongy and causes accentuation of the intercellular bridges.
- - dermal lesions include: edema, congestion of vesicles, perivascular mononuclear infiltration and variable numbers of neutrophils.
- - micro abscesses may be found in the epidermis as neutrophils migrate into the vesicles.
- - variable necrosis of adnexa structures especially of the hair roots and sebaceous gland, may be observed.
- - inflammatory crust which may contain ortho or parakeratotic cells colonized by surface bacteria may be seen on the ulcerated, eroded or mucosal surface.
- - there is intracytoplasmic inclusion bodies in degeneration keratinocytes.
- - evidence of mitosis may be found in the germinal layer.

### LUMPY SKIN DISEASE

- This is an acute viral disease of cattle and buffalo caused by a member of the Capri poxvirus ( also known as Neethling virus) closely related to the sheep pox and goat pox virus. LSD is confined to the African continent and Madagascar.
- The disease is characterized by the eruption of multiple well-circumscribed skin nodules accompanied by fever, ventral edema and generalized lymphadenopathy.
- Transmission – It is transmitted mechanically by a variety of biting insects.

Lumpy skin contd.

- Gross Pathology

- A history of fever, profuse salivation, oculonasal discharges, ventral oedema and swelling of superficial lymph nodes usually accompanying the acute disease, while fever is not noticed in the mild form. The most obvious gross lesion is the presence on the skin of firm, circumscribed flat-topped nodules 0.5 – 5cm in diameter all over the body, including the entire skin, skin of the scrotum, perineum, udder, vulva, glans penis, eyelids and conjunctiva. The nodules in the latter areas are usually flatter.
- - In pigmented areas, the nodules are surrounded by a zone of intense hyperemia.
- - Some of the nodules may coalesce and form a grayish creamy colour on cut section.
- - It usually affects the full width of the cutis extending into the subcutis.
- - the nodules may undergo necrosis and sequestration and their removal reveals a rim of dermal granulation tissue.

#### Lumpy skin contd.

- - secondary bacteria infection of necrotic ulcers may lead to lymphagitis and lymphadenitis.
- Extra cutaneous lesions may include ulcers in the upper respiratory and upper alimentary tracts, aspiration pneumonia, stenosis of the anterior portion of the trachea. Occasional nodules in parenchymatous organs such as kidneys, lungs and testes.
- Histopathology
- Vasculitis is the central lesion due to damage to endothelial cells.
- Other lesions include lymphadenitis and lymphagitis, marked dermal oedema with dermo-epidermal separation and infarction.
- Typical hydropic changes associated with pox virus infection is characteristic of the epidermis.
- Intracytoplasmic, eosinophilic homogenous, occasionally granular inclusion bodies are found in endothelial cells, pericytes, keratinocytes. Macrophages and fibroblasts in the dermis in the acute stage but becomes predominantly mononuclear (Macrophages and lymphocytes) in the later stages.
- Affected lymph nodes are oedematous and hyperplastic.

#### Lumpy skin contd.

- Differential Diagnosis
- - Dermatophilosis and Demodecosis complex. However, LSD is not associated with severe scab formation, hair loss as in these 2 conditions.
- - Malignant Catarrhal Fever: due to vasculitis

#### CONTAGIOUS ECTHYMA

- This is mostly observed in sheep and goats. This disease is also known as 'sore mouth' infectious Labial Dermatitis or Orf. The aetiology of the condition is the pox virus.
- Pathogenesis : As in pox infection.
- Pathology

- Grossly
- There is formation of erythema and small flat superficial pustules. A moist exudation soon appears and dries to form a scab. The scabs are thick wart-like brown crusts, somewhat grayish on the surface.
- The labial lesions occur mainly on the outer surface near the commissures, but they also occur on the margins and may spread to the lips and muzzle.
- The buccal lesions are raised, reddened or grayish foci surrounded by a zone of acute hyperemia; but scab scarcely forms. The scabs are firmly attached and if forcibly removed, leave a raw spongy base which bleeds easily.

orf

CONTAGIOUS ECTHYMA contd.

- Histopathology
- Degenerative changes develop in the more superficial nucleated cells of the epidermis. These cells in the granular and outer malpighian layers swell, the cytoplasm becomes vacuolated and clear and the nucleus shrinks.
- Intracytoplasmic inclusion bodies are observed and the dermis may be heavily infiltrated when there is secondary bacterial infection.
- The scab is made up of coagulated exudates, cellular debris and bacteria colonies.
- Differential Diagnosis

### BACTERIA DISEASES OF THE SKIN

- The normal bacteria flora of the skin are divided into two groups; the permanent resident population which forms stable colonies on the stratum corneum, and the more diverse unstable transient population. The ability to produce antibiotics and lipases that split sebum into fatty acids, toxic to non-resident bacteria helps the resident flora keep its competitive advantage. However, once this skin barrier is damaged, bacteria from the transient flora and occasionally the resident flora may assume pathogenicity.
- In dogs and cats, the resident population are:
  - Staphylococcus spp.
  - Micrococcus spp.
  - Corynebacterium spp.
  - Streptococcus spp.
  - Acanobacter spp.
- In equines, it is the same as dogs and cats with others like Bacillus spp., Flavobacterium spp., Lactobacillus spp. and Nocardia spp.
- In cattle, sheep, goats and pigs: they are all coagulase negative Staphylococci such as S. colinii, S. saprophyticus, S. sciuri and S. xylosus

## PYODERMA

- This is the bacterial infection of the skin. It may be primary or secondary and superficial or deep. Primary pyoderma develops in otherwise healthy skin i.e. with no apparent underlying cause and is induced by one species of organism and shows a characteristic disease pattern.
- Secondary pyoderma on the other hand occurs in diseased skin and is often caused by more than one bacteria specie and has variable clinical appearances. Conditions that may lead to secondary pyoderma include: allergic dermatitis, ectoparasitism and seborrheic conditions. Secondary pyoderma are more common in dogs than in other domestic animals.
- Examples of primary pyoderma include:
  - Canine and equine folliculitis and furunculosis caused by *S. aureus*.
  - Ulcerative dermatitis in sheep – *S.aureus*
  - Exudative dermatitis in pigs - *S. aureus*
  - Dermatophylosis in cattle – *Dermatophilus congolense*

## PYODERMA contd.

- Example of secondary pyoderma include
  - Fleece rot in sheep associated with *Pseudomonas aeruginosa* and *Proteus* spp.
  - Folliculitis and ulcerative lymphagitis in horses associated with *Corynebacterium pseudotuberculosis*.
  - Cat-fight abscess associated with *Bacteroides* spp., *P. multocida*, *Fusobacterium* spp. and *Peptostreptococcus anaerobius*.
  - Pyogranulomatous deep dermal or subcutaneous dermatitis have been associated with organisms such as *Fusobacterium necrohorus*, *Borrelia swilla*, *Nocardia* spp., *Actinomyces* spp., *Actinobacillus* spp., *Mycobacterium* spp., and *S. aureus*.

## Specific Bacteria Diseases

- EXUDATIVE EPIDERMITIS OF SWINE
  - This is an acute, rapidly progressive, often fatal superficial pyoderma of suckling or early-weaned piglets 5 – 35 days of age. It is caused by *Staphylococcus hyicus*. It is also known as seborrhea oleosa, impetigo, contagious suis and greasy pig disease.
  - Pathology
  - Grossly
  - the condition is characterized by sharply delineated red or yellow patches on the skin which may become greasy and easily removable focal erosion of the skin of the ear, eyes, snout, lips, legs and abdomen, leaving highly erythematous underlying dermis.

- Older lesions are malodorous and consist of dry exudates, deep cracks and fissures on the blackish-brown crusts.
- Some animals may show suppurative lymphadenitis, ulcerative glossitis and stomatitis.

#### Specific bacteria Diseases contd

- Histopathology
- There is exudative superficial folliculitis, intraepithelial pustular dermatitis, ortho- and parakeratosis, neutrophilic microabscesses in the epidermis and superficial dermis, lakes of serous effusions and numerous gram negative cocci.
- Cells of the outer spinous layer may show hydropic degeneration along with generalized spongiosis.
- Neutrophilic exocytosis is very prominent.
- The dermis is oedematous and contains numerous neutrophils especially around the blood vessels

#### Specific bacteria Diseases contd

- DERMATOPHILOSIS
- This is a disease of cattle and other species caused by a gram positive organism *Dermatophilus congolensis*. The synonym of the condition are; Kirchi(hausa), Cutaneous Streptothricosis and Mycotic dermatitis.
- The disease is associated with constant wetting as in rainy season and prior to injury to the skin caused by biting insects, ticks and thorns. The zoospores (branching filaments) are the infective form of the *Dermatophilus congolensis*.
- Pathogenesis
- The respiratory efflux of low concentration of carbon dioxide from the skin attracts the motile zoospores to defective protective barrier of the skin surface. Zoospores germinate to produce hyphae which penetrate into the living epidermis and spreads in all directions. Penetration causes acute inflammatory reaction.

#### Specific bacteria Diseases contd

- Pathology
- In cattle the gross lesions include presence of papules evident only on palpation of the skin.
- Presence of serous exudates around groups of hair shaft which may become matted and stand erect.
- These papules may coalesce to produce irregular mosaic plaques which may affect most of the skin
- There are raised, rough, circular, thick lamellated grey brown scale crusts that are penetrated by tufts of hair.
- In very severe lesions, the hairs may be buried in thick plaques of scale crusts.



- When individual crusts are forcefully detached, the hair is also epilated and undersurface of the scale crust is typically concave and most with little or no pus.
- Underlying epidermis is usually moist and erythematous and in older lesions, small raised granulation tissue may be present.

## Dermatophilosis

### Specific bacteria Diseases contd

- Histopathology
- the lesions are characterized by neutrophilic exocytosis, microabscesses in the epidermis.
- Superficial dermal oedema and accumulation of large number of NQ in the dermis around the external root sheath of the hair follicle.
- Branching filaments and zoospores of the organism are found in the epidermis and rarely in the dermis.
- Epidermal lesions may also be accompanied by ortho- and parakeratosis and premature keratinization of the stratum spinosum.
- Differential Diagnosis
- Warble fly infection, lumpy skin disease or warts. Contagious ecthyma and ulcerative dermatosis in sheep. Dermatophytosis and immune mediated scalding disease of horses.

### Specific bacteria Diseases contd

- FOOT ROT
- This is a specific contagious disease of sheep caused by *Dichelobacter nodusus*, *Fusobacterium necrophorum*. This occurs by the initial damage by other agents and cold, wet condition of the feet. It is a superficial inflammation of the integument of the foot which spreads between the horny and spinous layers causing separation. It affects the claws of the hoof and usually more than one foot.

## FUNGAL OR MYCOTIC DISEASES OF THE SKIN (Dermatophytosis or Ringworm)

- This is a superficial infection of the keratinized layer of the skin and its appendages caused by a group of taxonomically related fungi known as Dermatophytes. Dermatophytes belong to 3 genera:
  - Microsporium
  - Trichophyton
  - Epidermophyton
- Most animal spp belong to the first two genera while *E. floccosum* is the cause of *Tinea pedis* (athletes foot) in humans. Dermatophytes of various animals at the end of the condition.
- Pathogenesis:

- The dermatophytes do not invade living tissue but remain confined to the keratinized layers which they attack by proteolytic enzymes having keratinolytic activity. These include keratinase collagenase and elastase.

#### Ringworm contd.

- Pathology
- Gross
- The gross lesions are variable but their character is similar. It consists of different degrees of erythema , follicular papules, scaling, crusting and alopecia.
- Histopathology
- Having degree of hyperplasia which may range from moderate to severe, rete ridge prominence and ortho and parakeratotic hyperkeratosis are observed.
- Neutrophilic exocytosis into the fungus-laden cornified layer leading to subcornea and intracornea microabscesses.
- There is mild lymphocytic perivascular infiltration in the superficial dermis and around adnexa.

#### Ringworm contd.

- The dermatophytes of various species of animals include:
- Trichophyton equinum which causes 'girth itch' in horses
- Microspora equinum also in horses.
- T. mentagrophyte in cattle.
- T. verrucosum in cattle.
- M. canis
- M. gypsum
- T. metagrophyte

#### PROTOZOA INFECTION OF THE SKIN

- Cutaneous globidiosis or Besnoidiosis
- Leishmaniasis
- BESNOITIOSIS (Cutaneous Globidiosis)
- The aetiological agent is Besnoitia besnoiti. This is a disease of cattle, goats, horses and wild animals. The gobicious organism affects the dermis of the skin and also the sclera, eyelid vein and intestines. It has two host life cycle including the cat. Most infections are subclinical but globidiosis may cause infertility in bulls.
- Pathology
- Gross

- The parasite appears in the skin as thickened, rugged, partially hairless area on the legs, thighs and scrotum. Cystic forms are also common in the intima of veins. As the parasite becomes encysted, the oedema of connective tissues diminishes, but the skin remains thickened, scurfy or crustous and fissured.

BESNOITIOSIS contd.

- Histopathology
- There is epidermal hyperplasia, congestion and oedema of the corium and leukocyte infiltration mostly macrophages which play host to the parasite.
- The cyst consists of a thick wall of homogenous, lamellated, eosinophilic substance lined on the inner surface by a thin ring of cytoplasm which contains two or more nuclei.
- Differential Diagnosis

#### HELMINTH DISEASES OF THE SKIN

- CUTANEOUS HABRONEMIASIS
- This condition is caused by Habronema . It is transmitted by stable fly or horsefly which is attracted to the site by pre-existing wounds. It affects mainly the eyelid, conjunctiva, prepuce and glans penis. The organism causes production of ulcerative, granulomatous or hard nodules with pruritis.
- Histologically, the larvae of the organism are present in the yellowish-white necrotic foci and the inflammatory cells are mostly eosinophils and few giant cells. Marked fibrous tissue reaction is observed especially in the genital lesions.

- Filiarisis
- Stephanofiliariosis

#### ARTHROPOD DISEASES OF THE SKIN

- These include fly, flea, lice, tick and mite infestation.
- MITE INFESTATION (MANGE)
- These are a group of cutaneous diseases in domestic animals caused by different species of arthropod ectoparasites – mites. The various conditions or the various species cause are known by different names and in relation to the anatomic sites of the various species of animals affected

Mange contd.

- Sarcoptic Mange
- This is caused by Sarcoptes scabiei (Acarina Sarcoptidae). It is the most important ectoparasite disease in swine. It also causes disease in cattle and goats but not as important as psoroptic mange in these species.

- Pathology
- Gross
- The primary parasite related lesion of sarcoptic mange are erythematous macules or papules that develop a local scale crust in reaction to burrowing mites. In animals with massive mite infestation (especially in malnourished animals), the lesions are characterized by alopecia, marked lichenification, accumulation of scale crust and fissuring.
- The lesions of allergic Sarcoptic mange largely result from self-trauma induced by pruritus. In the early stages, these include erythematous papules, excoriation, haemorrhagic crusts and patchy alopecia. Chronic lesions include marked alopecia, scaling and lichenification

Mange contd.

- Histopathology
- The epidermal lesions include moderate to large number of adult mites in hollows lined by parakeratotic stratum corneum, thick scale crusts of both ortho- and parakeratotic hyperkeratosis, prominent rete pegs, serum lakes, neutrophilic debris and numerous refractile *Sarcoptes* ova.
- Dermal lesions include vascular congestion, neutrophilic exocytosis, perivascular mononuclear cellular infiltrations, endothelial swelling, oedema, fibrosis which depends of the stage and extent.

Mange contd.

- Notoedric Mange
- This is caused by *Notoedris cati*, a predominant parasite of cats and rabbits but infestation in dogs can occur. The infestation is highly contagious with transmission chiefly by direct contact. The life cycle is similar to that of *Sarcoptes scabiei*. The major clinical sign is pruritus and lesions which are similar to those of sarcoptic mange are produced on the skin of the head and ears but may extend to neck, paws or become generalized.

Mange contd.

- Psoroptic Mange
- This is a parasitic disease of sheep, cattle, horse and goats. They are more host specific than the sarcoptic mites.
- *Psoroptes ovis* – both cattle and sheep
- *P. cuniculi* - infects ears of rabbits, horses, goats and sheep
- *P. nataliensis* - body mites of cattle
- *P. equi* - body mites of horses
- Psoroptic mange is a serious skin disease of cattle and sheep. The economic importance in sheep and cattle results from a marked decrease in weight gain, reduced milk production, reduced fleece weight and

quality and occasionally death and costs due to prevention and eradication. Psoroptic mange is characterized by intense pruritus, dermatitis which is restricted to the epidermis as psoroptes mites do not burrow the epidermis.

- “Sheep scab”, as the condition in sheep is called, affects the ears, infraorbital tissue, inguinal and perineal folds and the base of the horns

Mange contd.

- Pathology
- Gross
- Lesions are papules approximately 0.5cm in diameter, covered with yellow serous crusts that may matt the fleece. Many of these papules may coalesce to form diffuse lesions over most of the body surface.
- In goats, *P.cuniculi* is known as “ear canker” because of its predilection for the external auditory meatus. Mites induce severe head shaking and severe encrusted lesions on the inner surface of the ear.

Mange contd.

- Otodectic Mange
- *Otodectes cyanotis* is an obligate parasite of the external skin surface of dogs and cats especially the external ear canal, even though they may be found at several body sites. The major lesion is otitis externa, however focal erythematous alopecia or excoriated lesions occur on the face, feet, neck and head

Mange contd.

- Demodectic Mange
- Demodex mites are normal inhabitants of the hair follicles or sebaceous gland. Different species of demodex mites are found in different animal. Examples include:
- *Demodex canis* – dogs
- *Demodex bovis* – cattle
- *Demodex phylloides* – swine
- *Demodex glanensis* – cattle
- *Demodex folliculorum* - man
- Transmission is by a direct contact from the dam to her offspring during nursing in the neonatal period. The most severe expression of demodectic mange is seen in the dog as generalized dermatitis which is occasionally fatal. Demodex mites in cattle, sheep and goats are rarely pathogenic. The disease in dogs takes two clinical forms:
- The localized or squamous demodecticosis- this occurs in young dogs 3-10 months of age and is usually self-limiting. Grossly the lesions are single or multiple, well circumscribed erythematous, scaly and alopecic patches usually affecting the head around the lips and eyes or on the extremities.
- The generalized or pustular form is characterized by patchy to diffuse alopecia, erythema scaling and

crusting. Secondary bacteria infection is usually common. The lesions are seen on the face, forelimbs and feet and are often pruritic. Peripheral lymphadenitis is about 50percent in affected dogs.

#### Demodecosis

#### Mange contd.

- Histologically

- The localized form is characterized predominantly by lymphocytes, plasmacytic perifolliculitis, marked follicular hyperkeratosis. Presence of large number of mites in the upper third of the hair follicles.
- In the generalized form, secondary bacteria infections are usually common and may lead to suppurative dermatitis. The keratin and other irritant substances stimulate a granulomatous reaction, chiefly epitheloid macrophages, but a few multinucleated giant cells may be present.
- This pyogranulomatous furunculosis of demodectic mange differs from most other types of furunculosis in that eosinophils are rare or absent in the reaction.
- Epidermal lesions include hyperplasia, ortho- and parakeratosis, variable spongiosis, neutrophilic exocytosis, ulcerative and inflammatory crusting. In chronic cases there is dermal fibrosis, often with obliteration of adnexa.

#### PHOTOSENSITIZATION DERMATITIS

- It is recognized as an entity in farm animals especially in sheep and cattle and the clinical signs appear a few hours after exposure to strong sunlight. These include:
  - burning or itching sensation
  - erythema and inflammatory oedema
  - in cattle it affects mainly hair and unpigmented teat of udder.
  - In sheep it affects the head and ear
  - The lesion may heal in a week but may eventually become infected or gangrenous
- Pathogenesis
- Photosensitization results from action of sunlight upon certain fluorescent pigments which have accumulated in tissue over time. Three distinct conditions can give rise to this type of accumulation.

#### PHOTOSENSITIZATION contd.

1. Congenital porphyria photosensitization. This is a metabolic defect in the synthesis of haeme pigment protoporphyrin resulting in the release of free Uroporphyrin III and Coproporphyrin III which accumulates in the tissue and serum. This condition occurs spontaneously in cattle and swine. When it accumulates and the sun acts on it, it becomes fluorescent and inflamed.
2. Hepetotoxic photosensitization. This is caused by phyllocrythrin, a degradation product of chlorophyll which is excreted in the bile. Obstruction to bile secretion as in hepatitis can give rise to unusual level of phyllocrythrin and when the excretion capacity of the liver is deranged, the phyllocrythrin accumulates in the

blood and sensitized the animal to light.

- Ingestion of certain plants such as *Lantana camara* can also cause photosensitization.
- 3. The primary photosensitization. This is due to ingestion of exogenous photodynamic agents such as plants, lush green stage or rapidly growing plants like *Hypericum perforatum* (St. John's wort). Hypericin obtained from *H. perforatum* is such a photosensitization agent which becomes inflamed when exposed to sunlight

### AUTOIMMUNE SKIN DISEASES OR PEMPHIGUS

- This occurs in cats, dogs and man and is characterized by formation of blisters or vesicles on the skin or mucus membrane as a result of auto antibodies directed against intercellular cement in the skin. These antibodies can be demonstrated by immunofluorescent stain. The binding of the auto antibodies to intercellular cement leads to acantholysis i.e. separation of epidermal cells from one another, cleft formation, vesicles and bullae.

PEMPHIGUS contd.

- Types of Pemphigus
- 1. Pemphigus vulgaris: this is the most severe form which occurs in the mucocutaneous junction of the nose and lip. The prognosis is poor. The acantholysis is in the suprabasal region.
- 2. Pemphigus vegetant: this is similar to vulgaris but is accompanied by papillomatous proliferations on healing.
- 3. Pemphigus foliaceous: this occurs in the supracorneal region.
- There is acantholysis and vesicle formation. The prognosis is good.
- 4. Pemphigus erythematous: this is milder than pemphigus foliaceous.
- 5. Bullus pemphigus: subepidermal vesicles occur because the auto antibodies are directed against the basement membrane of the skin and mucus membranes.

### NEOPLASTIC DISEASES OF THE SKIN

- The skin is the most common site for neoplasia in dogs, horses, cattle and cats. Neoplasm can arise from all structures in the skin. The tumour can be primary or secondary.
- In general, ectodermal neoplasms (from epidermis & adnexa) are benign and many types of mesodermal tumours (from fibrous tissue, muscle, fat, blood vessels) in contrast are histologically malignant and regularly exhibit locally infiltrative growth and occasionally will metastasize.

NEOPLASTIC DISEASES contd.

- Papillomatosis (Warts)
- This is usually observed in cattle, horses, dogs and goats. The predilection sites of this condition on the

skin of cattle are the head, neck and shoulder. In horses it is chiefly on the muzzle and lips. In dogs it is in the oral mucosa.

- Pathology
- Gross
- The tumors are small, rounded, finely dimpled elevations or the abnormally cauliflower-like appearance. Hair is absent from the lesion. The surface is grayish, rough and horny.

NEOPLASTIC DISEASES contd.

- Histopathology
- The dermal papillae are drawn out into long, thin strands and they are covered by thickened epidermis which matures in the regular sequence from basal to superficial layers. The proliferation of epithelium is greatest over the top of the papillae and here keratinization is incomplete.
- There is breakdown of intercellular bridges at the stratum granulosum layer and their cytoplasm becomes vacuolated or homogenous and acidophilic. There may be presence of inclusion bodies in these cells.

NEOPLASTIC DISEASES contd.

- Read about : Squamous Cell Carcinoma
- Melanosis
- Basal Cell and Adnexal Tumor
- Fibrosarcomas

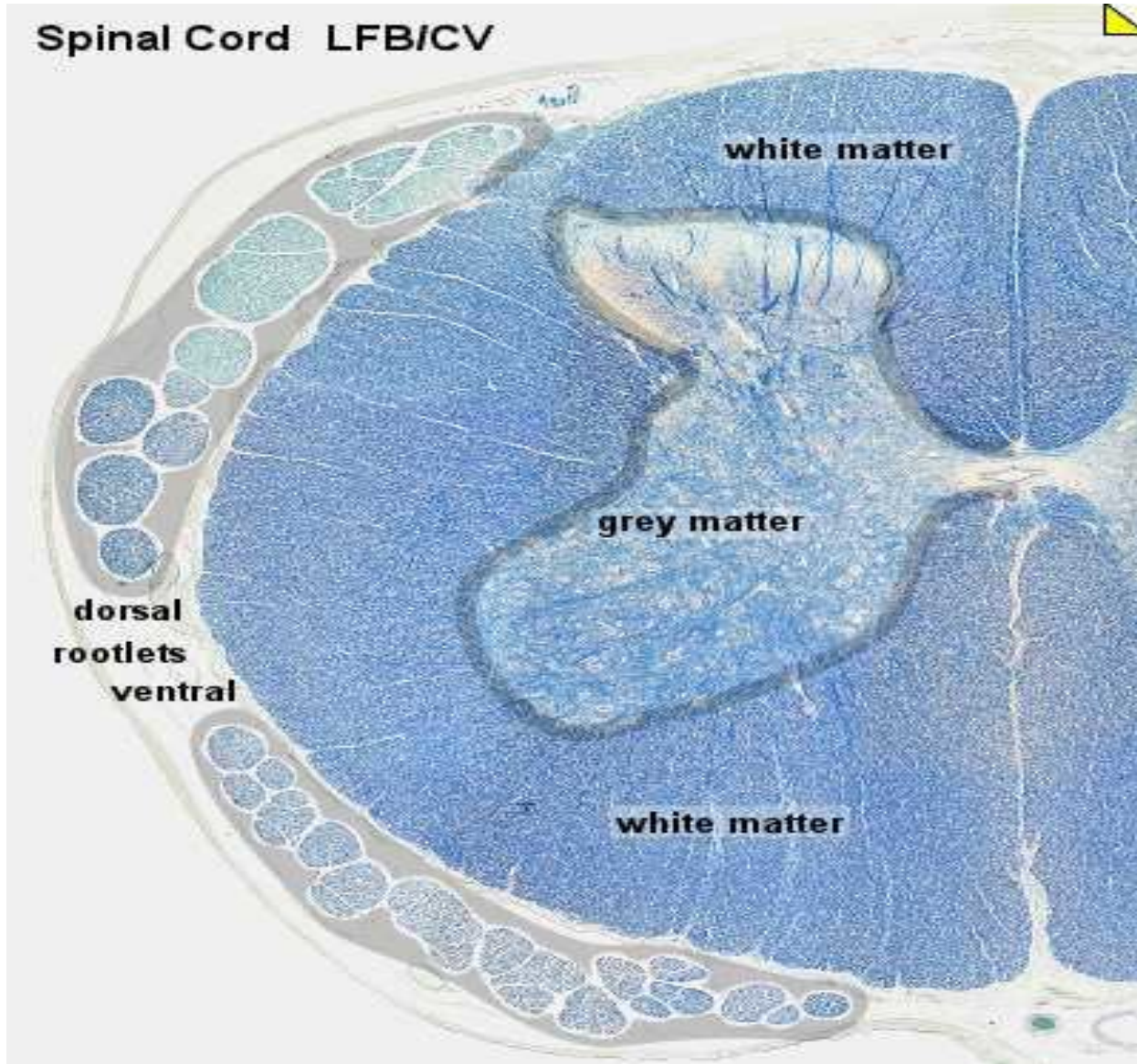


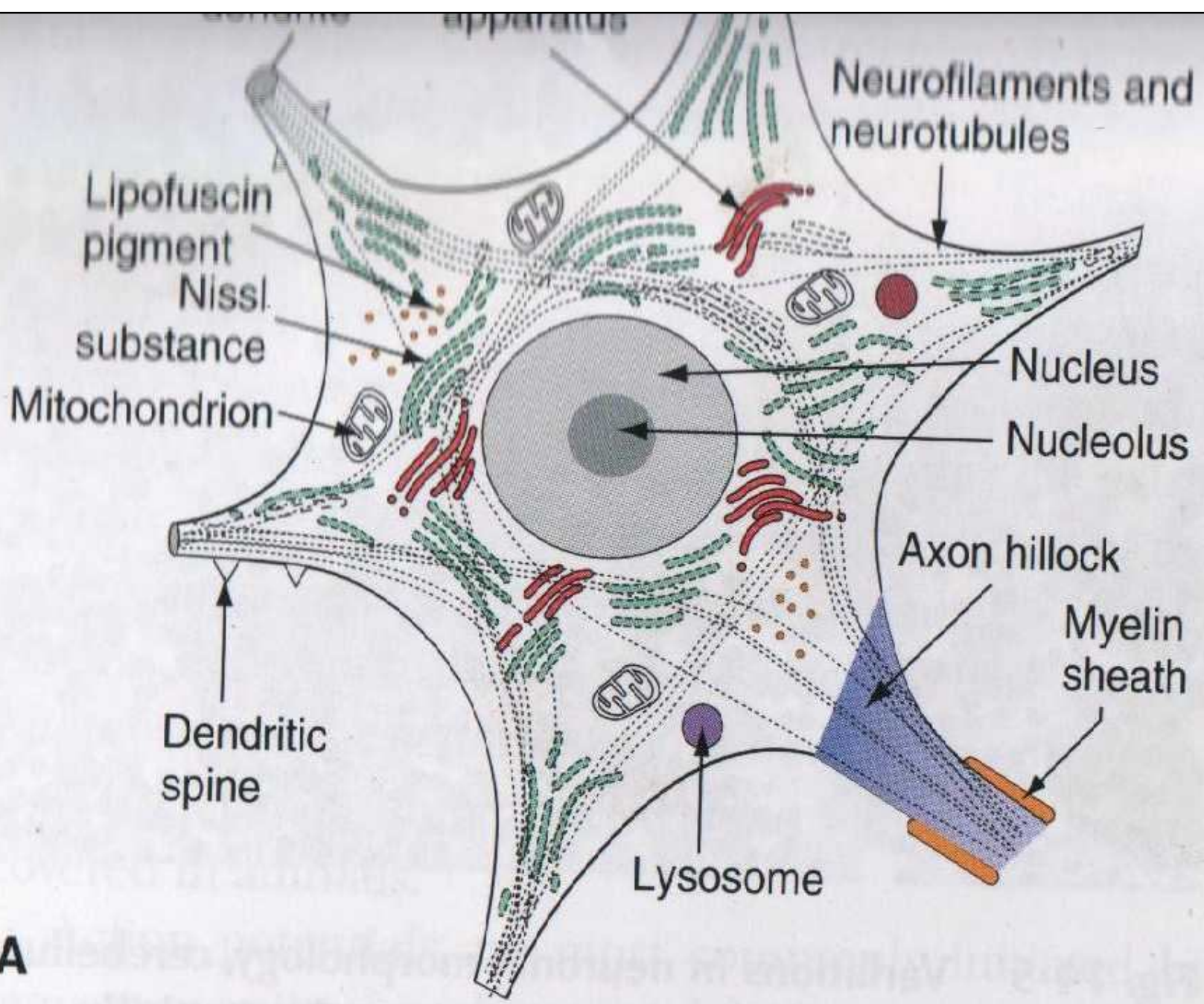
Nervous System pathology

By Prof O. B. Kasali

Spinal Cord

Spinal Cord LFB/ICV

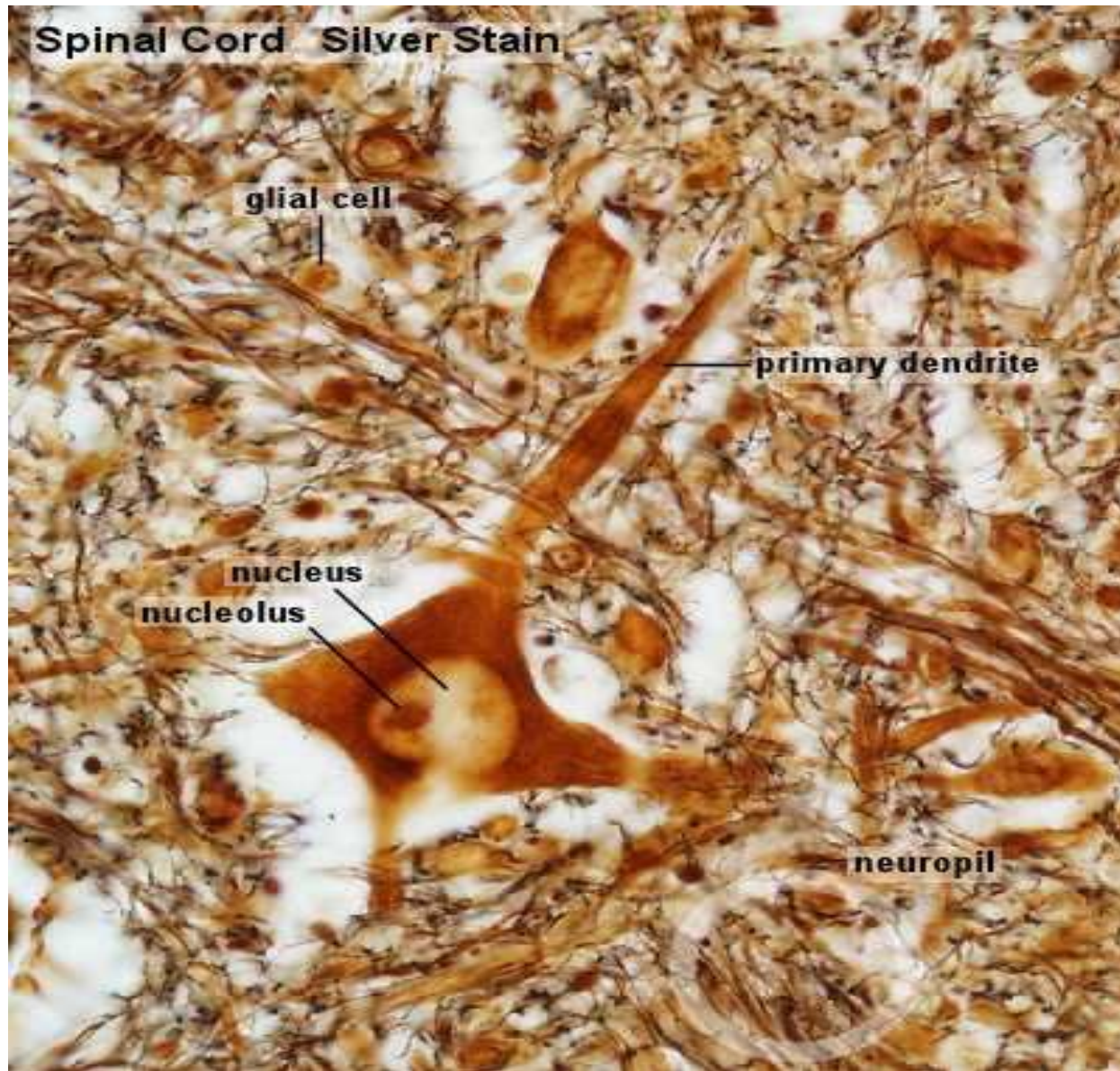




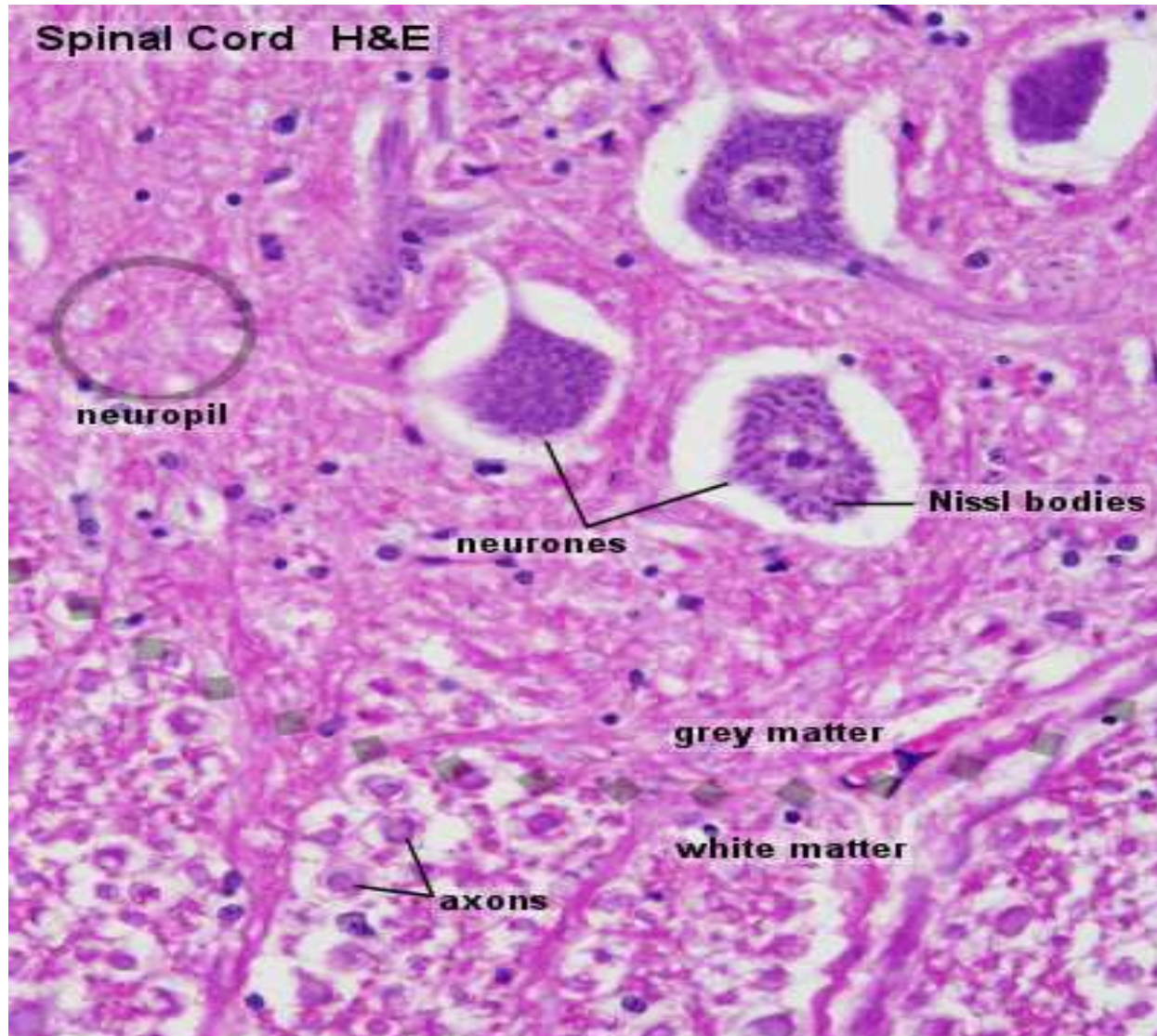
**A**



Neuron in section. Silver stain

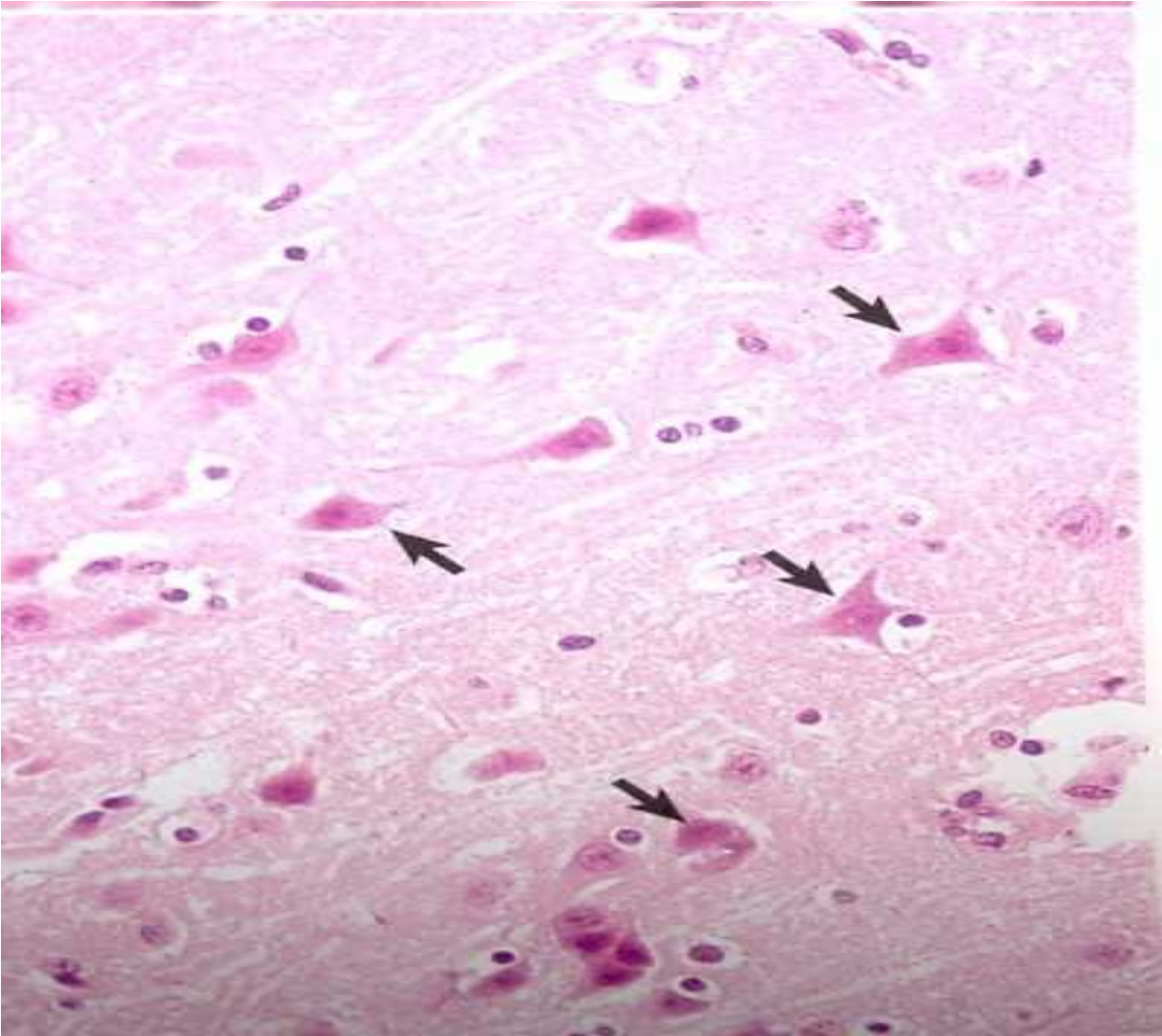


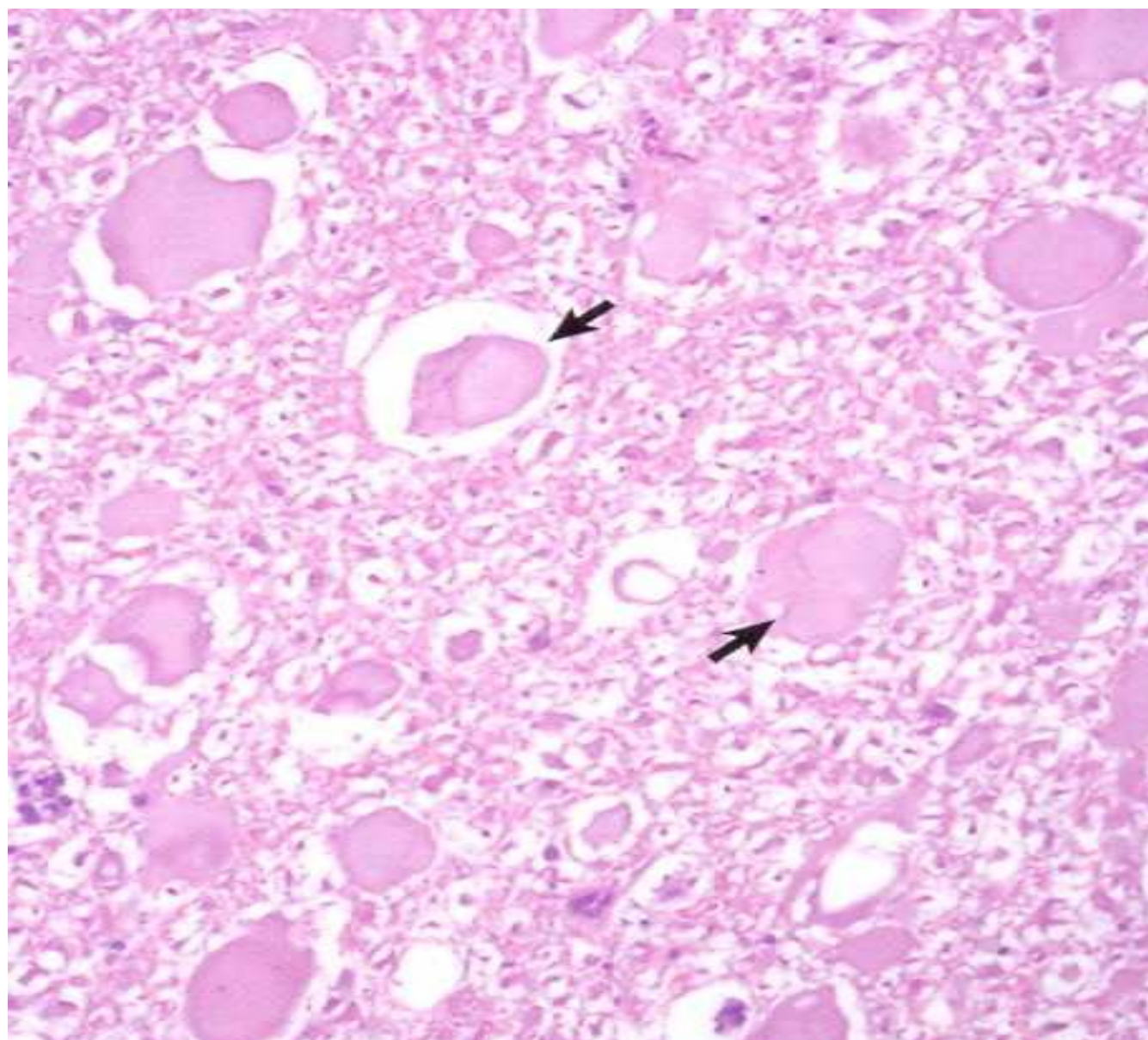
## Neurons





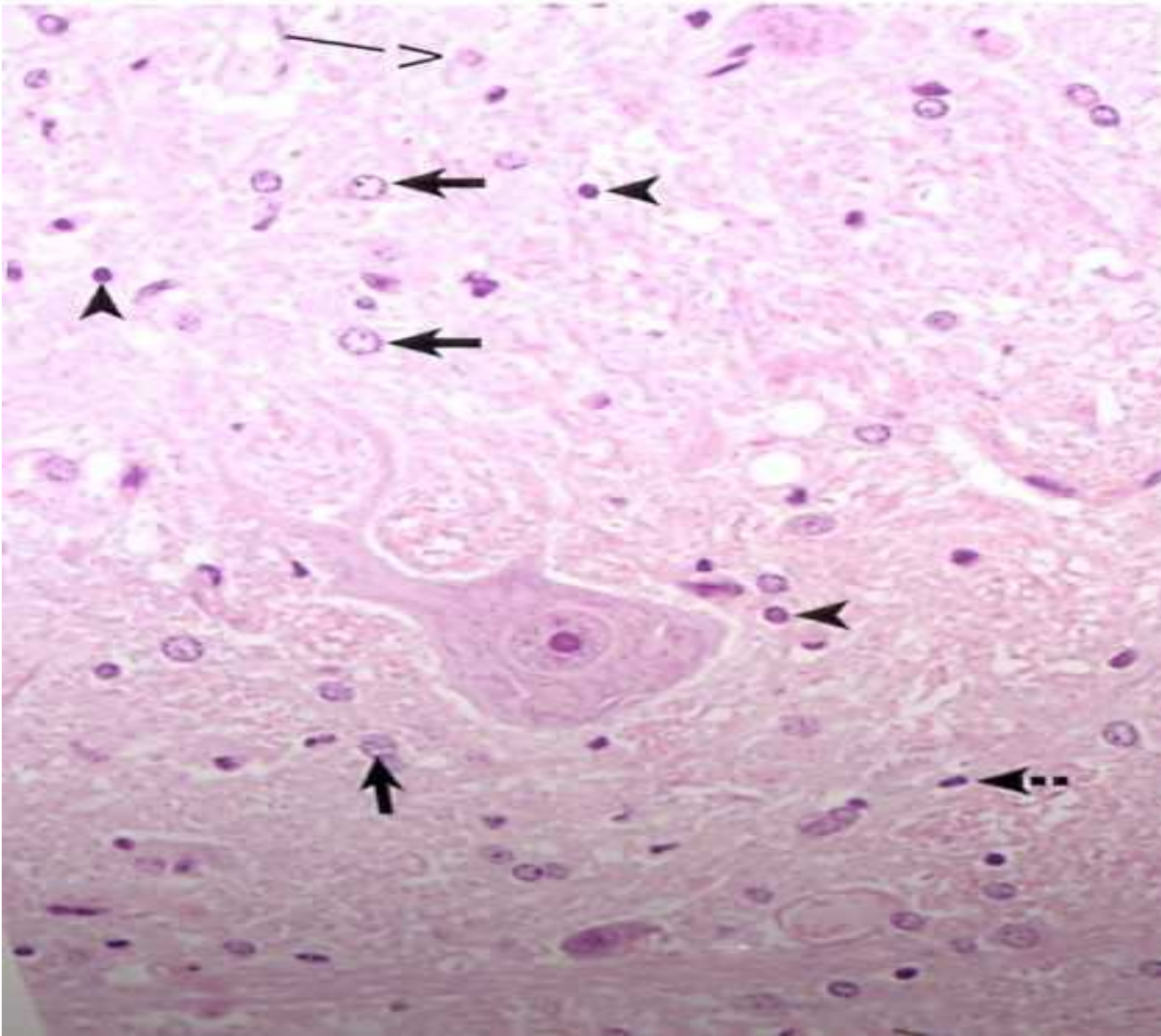
Neuronal degeneration and necrosis





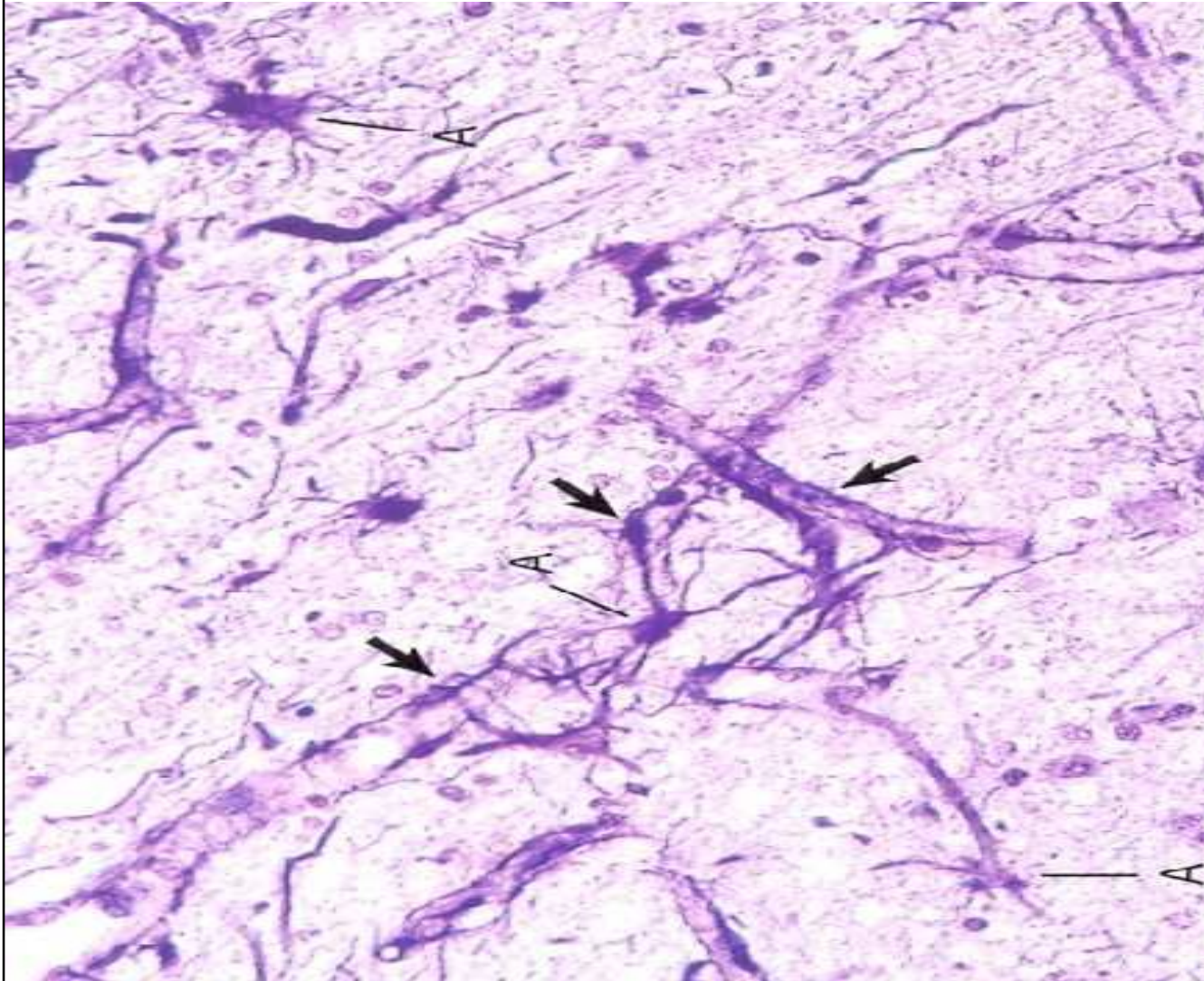


Glia cells (arrow)

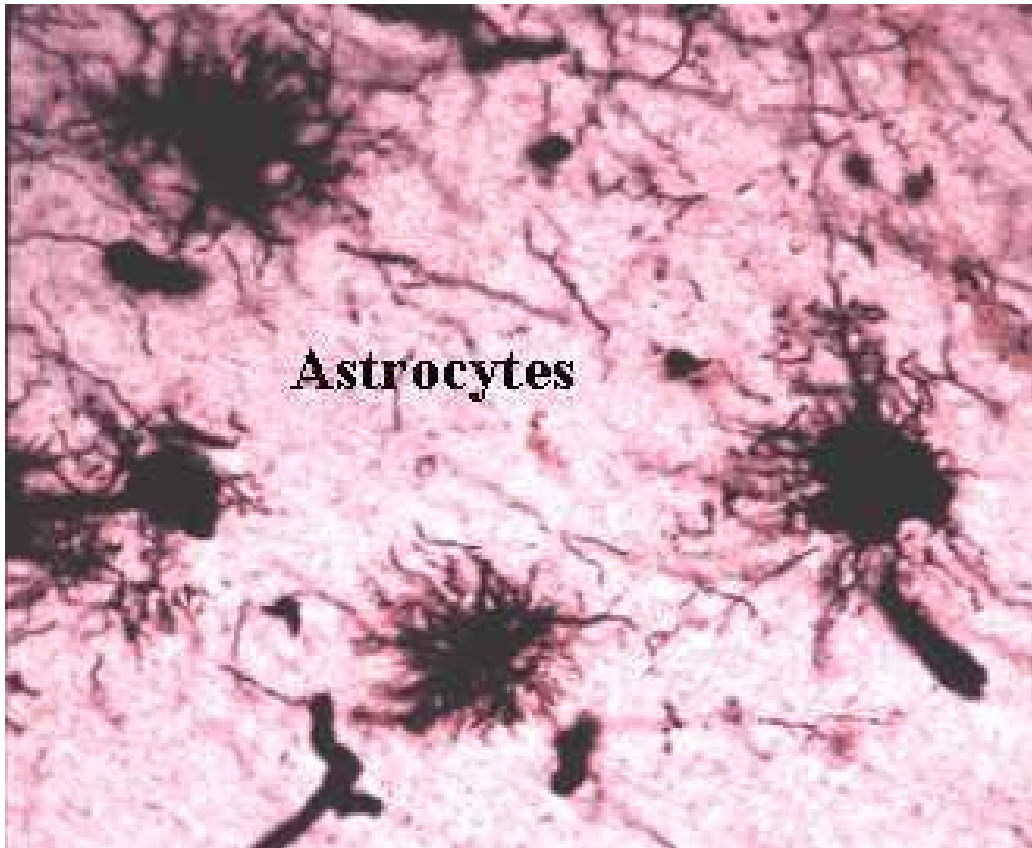




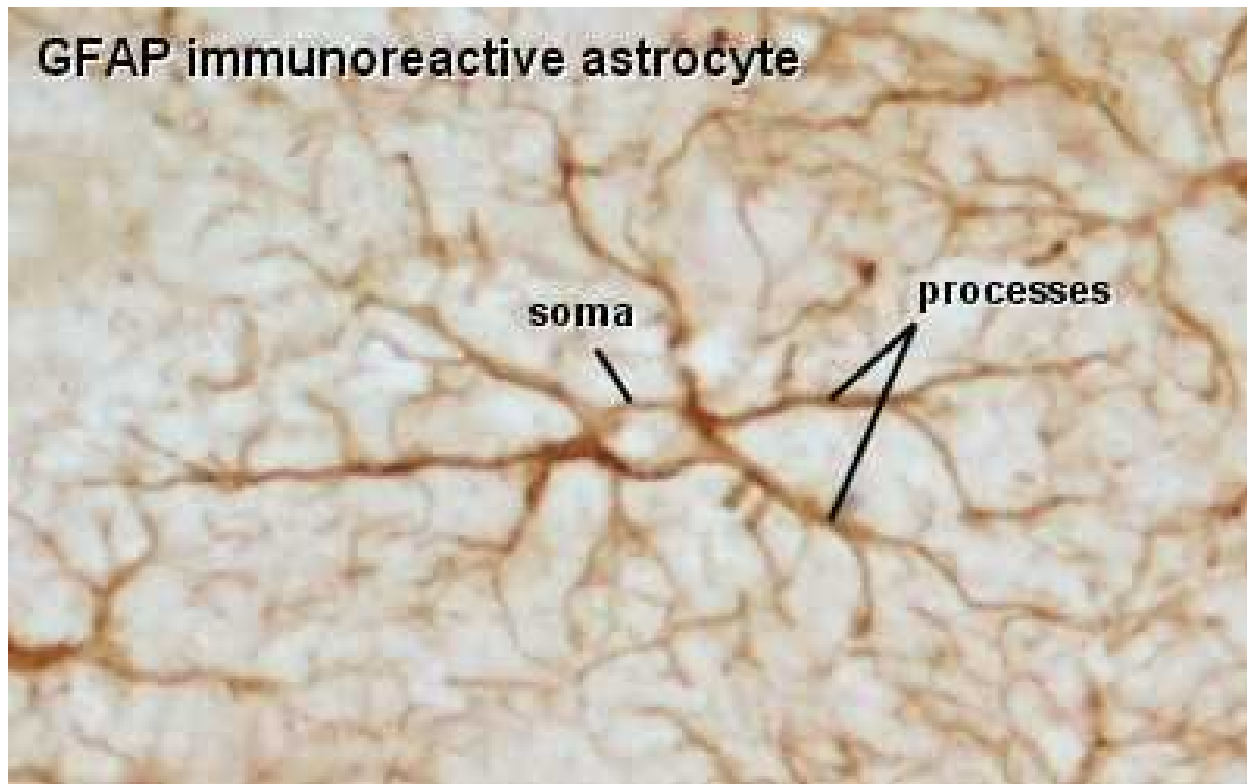
Astrocytes (special stain)



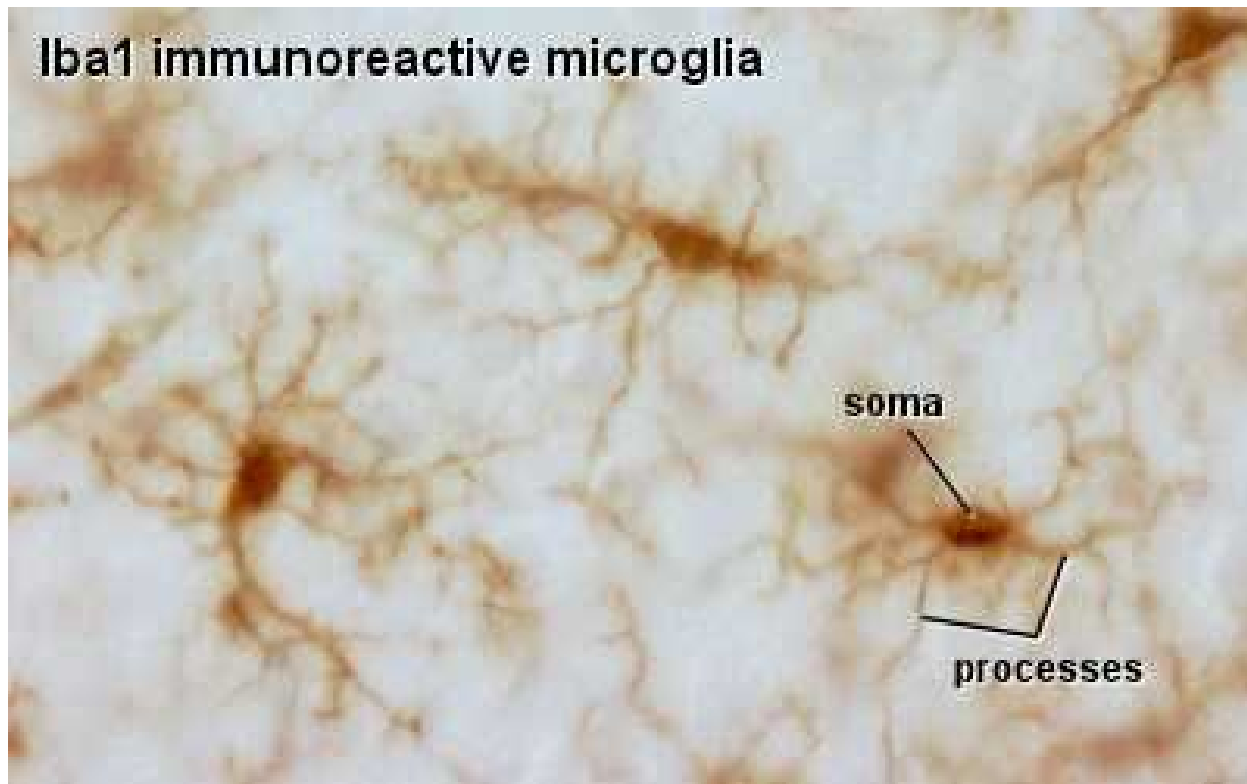
*Astrocytes*



**GFAP immunoreactive astrocyte**



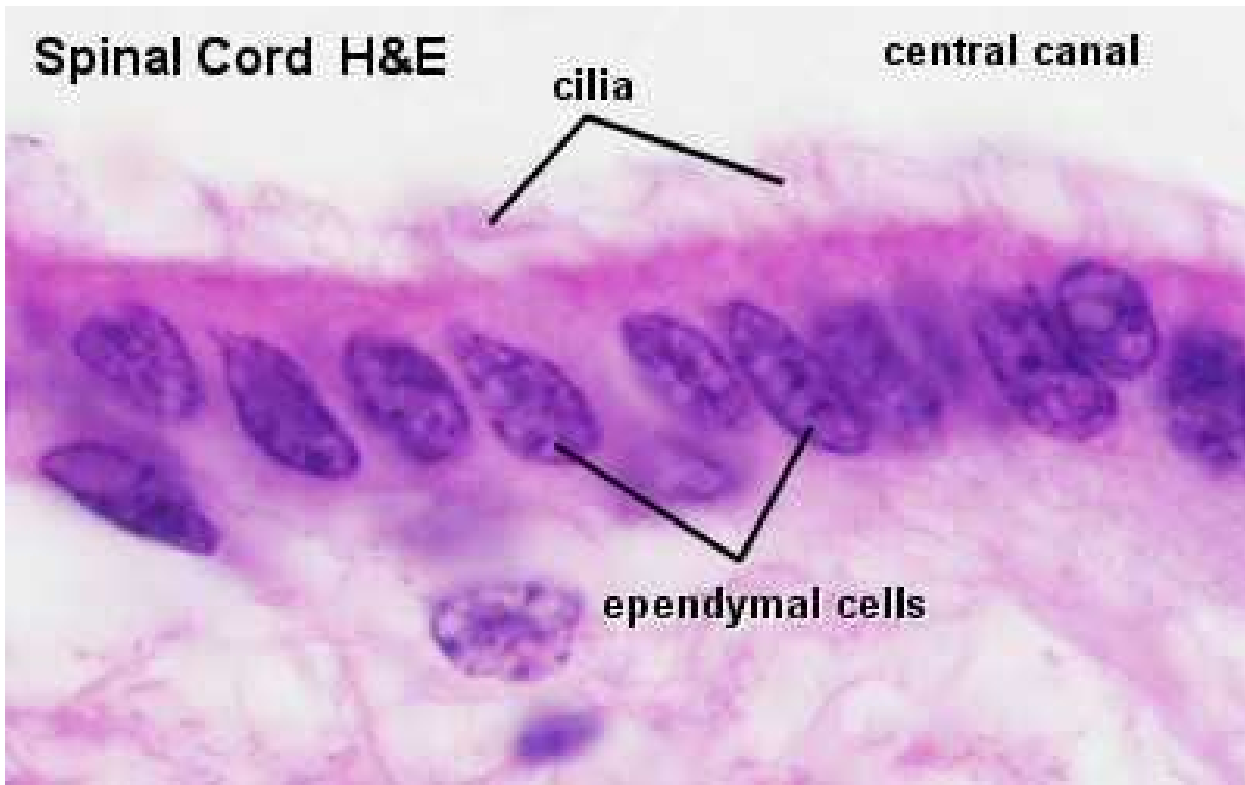
**Iba1 immunoreactive microglia**



**Spinal Cord H&E**

**central canal**

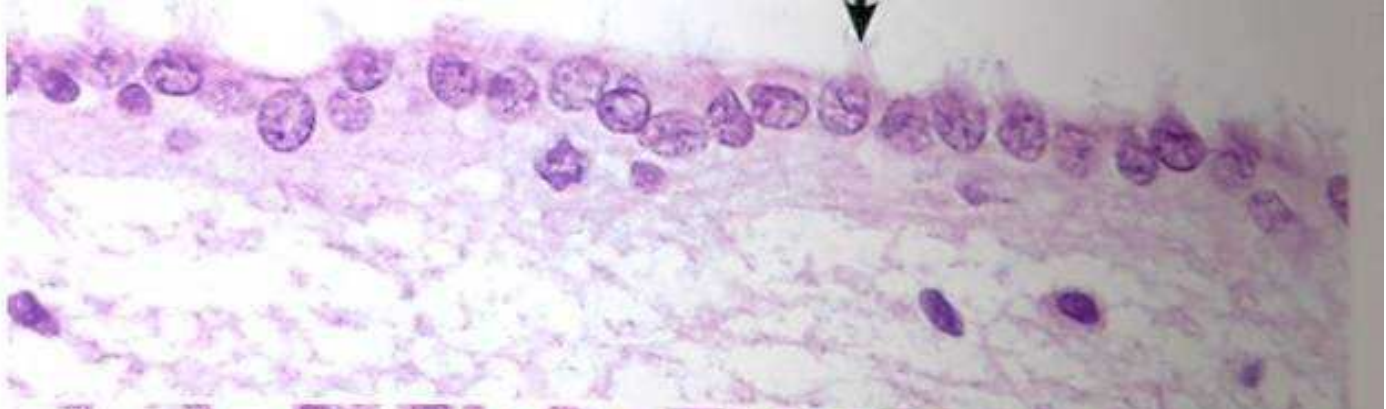
**cilia**



**ependymal cells**



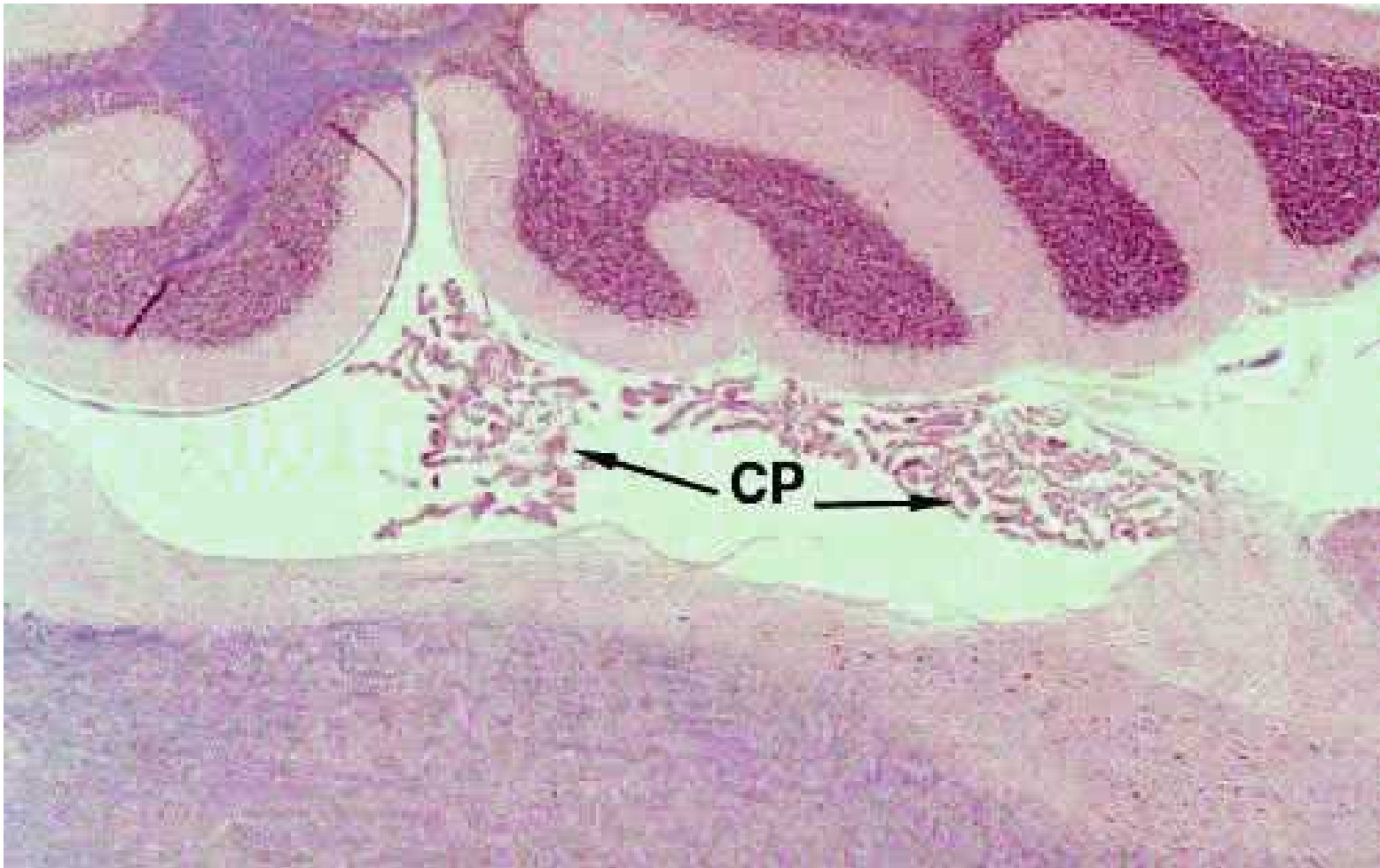
**A**

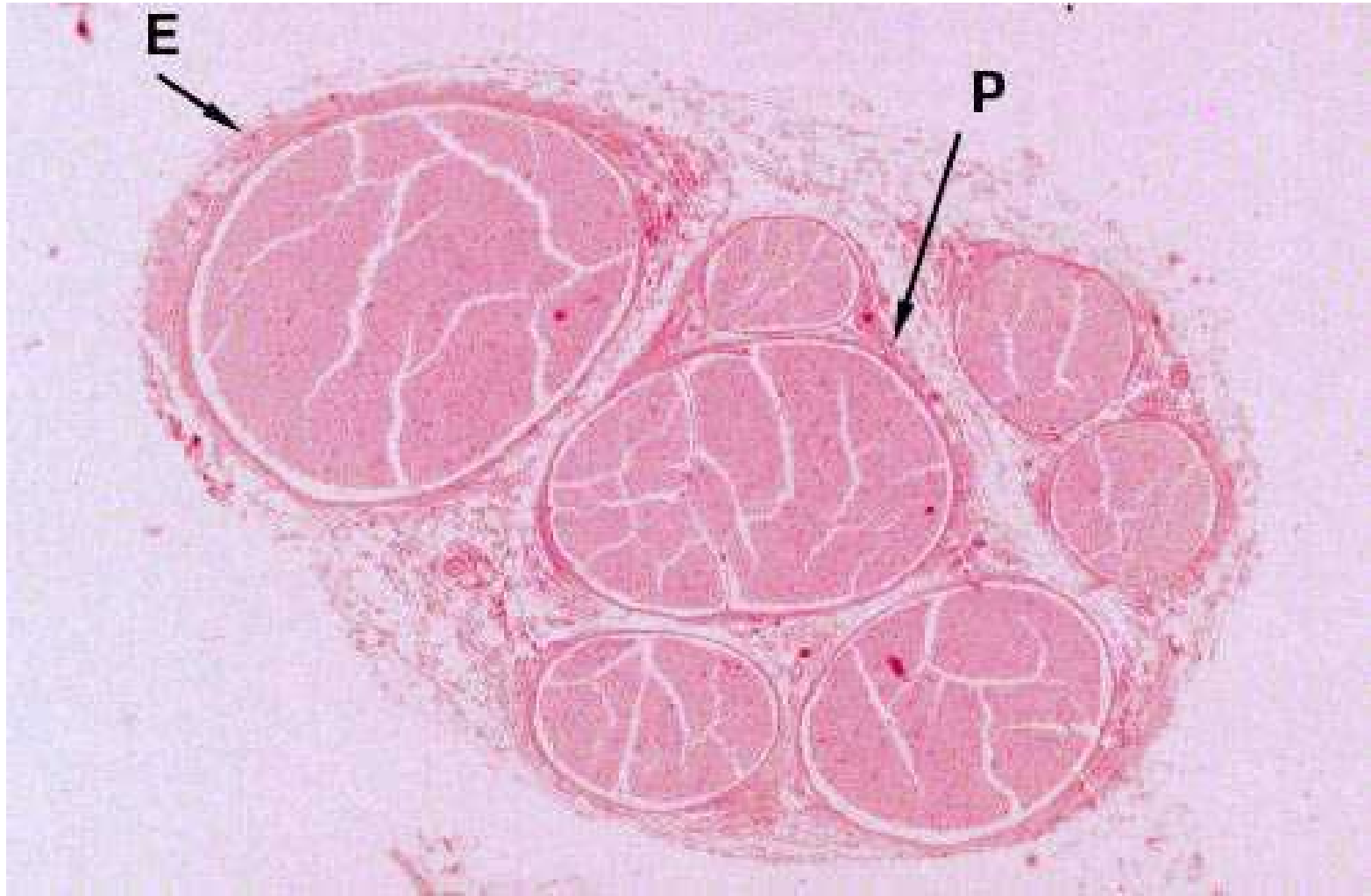


**B**



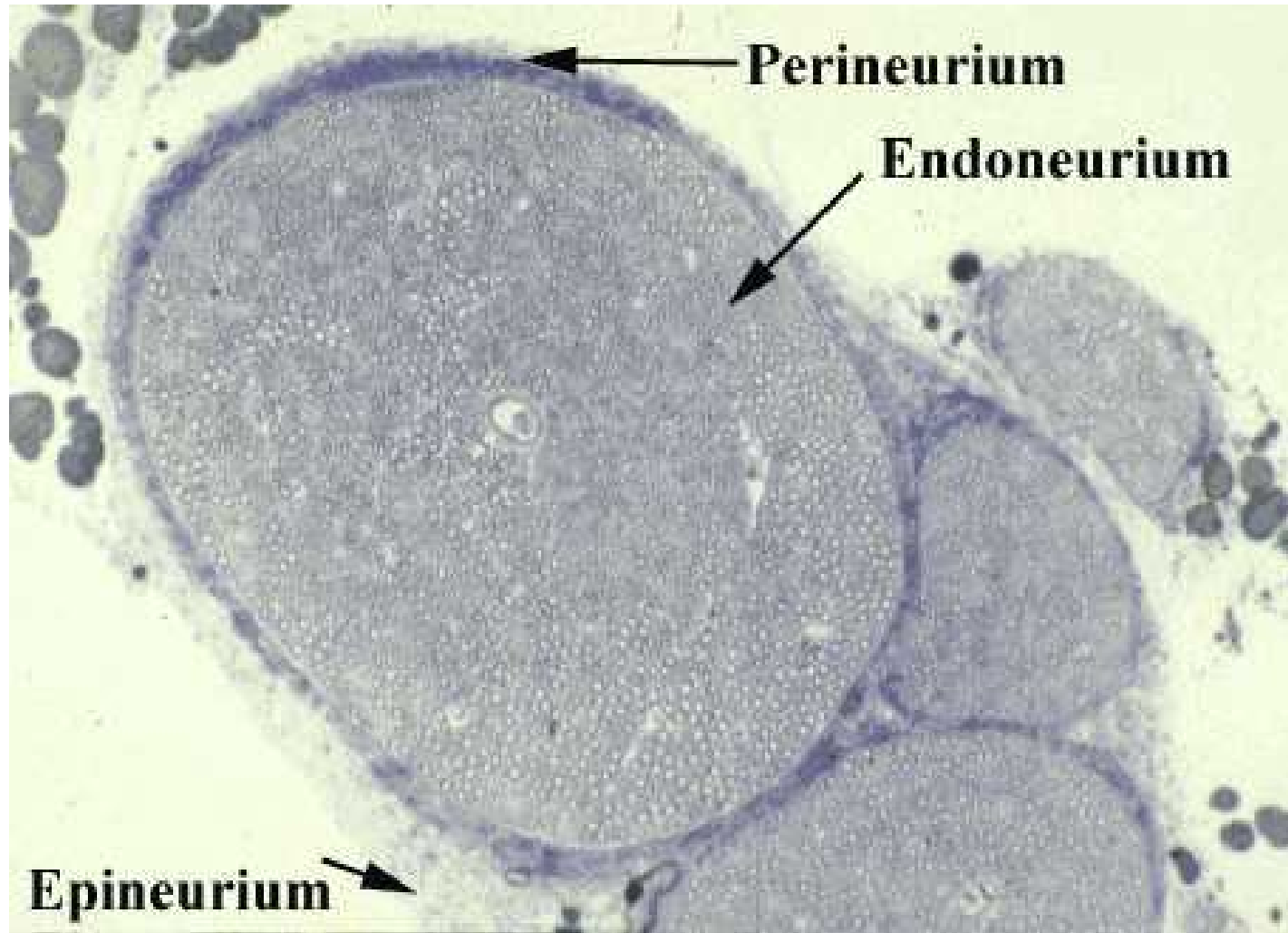
Choroid plexus



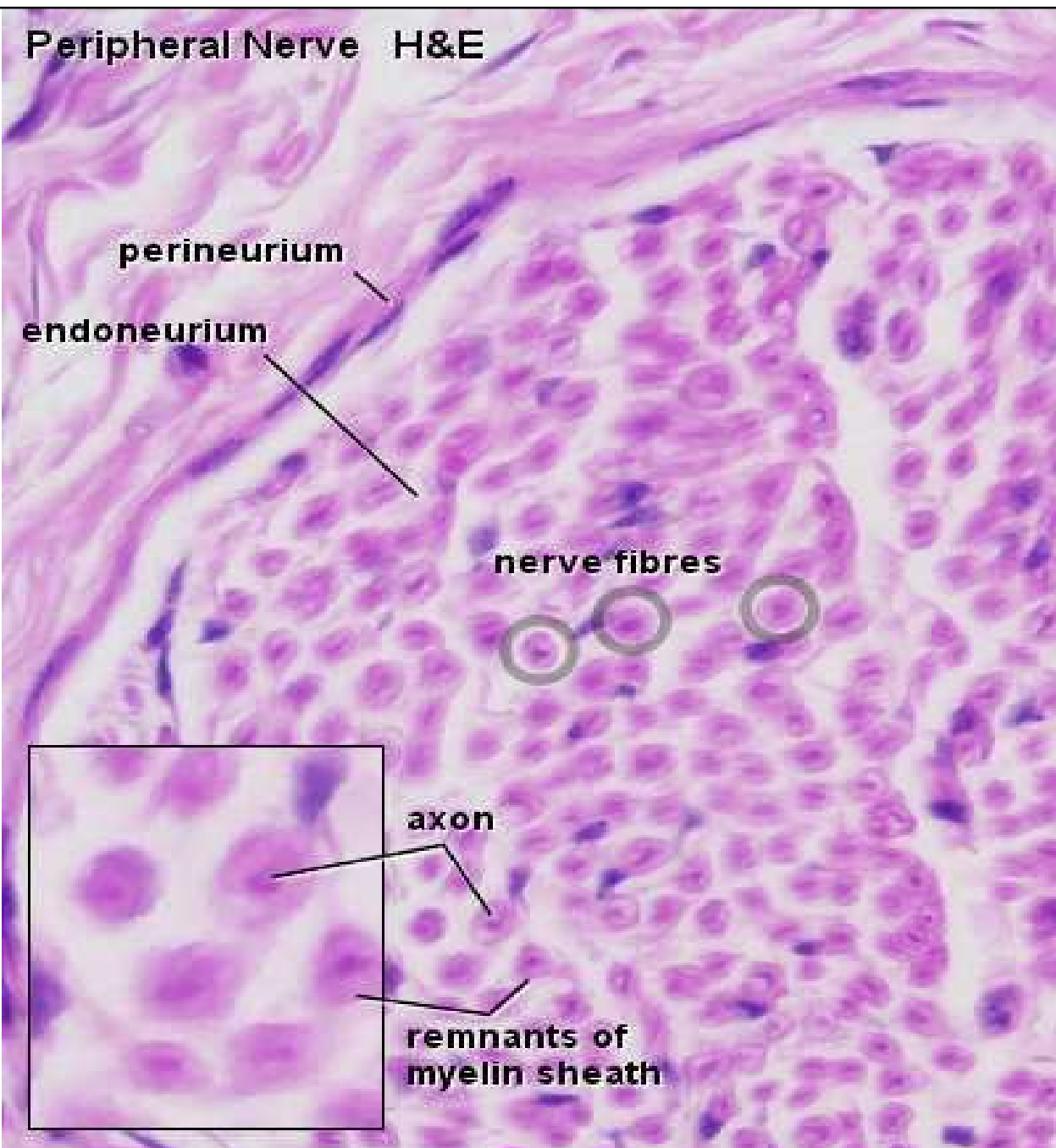




Peripheral nerves



**Peripheral Nerve H&E**



**perineurium**

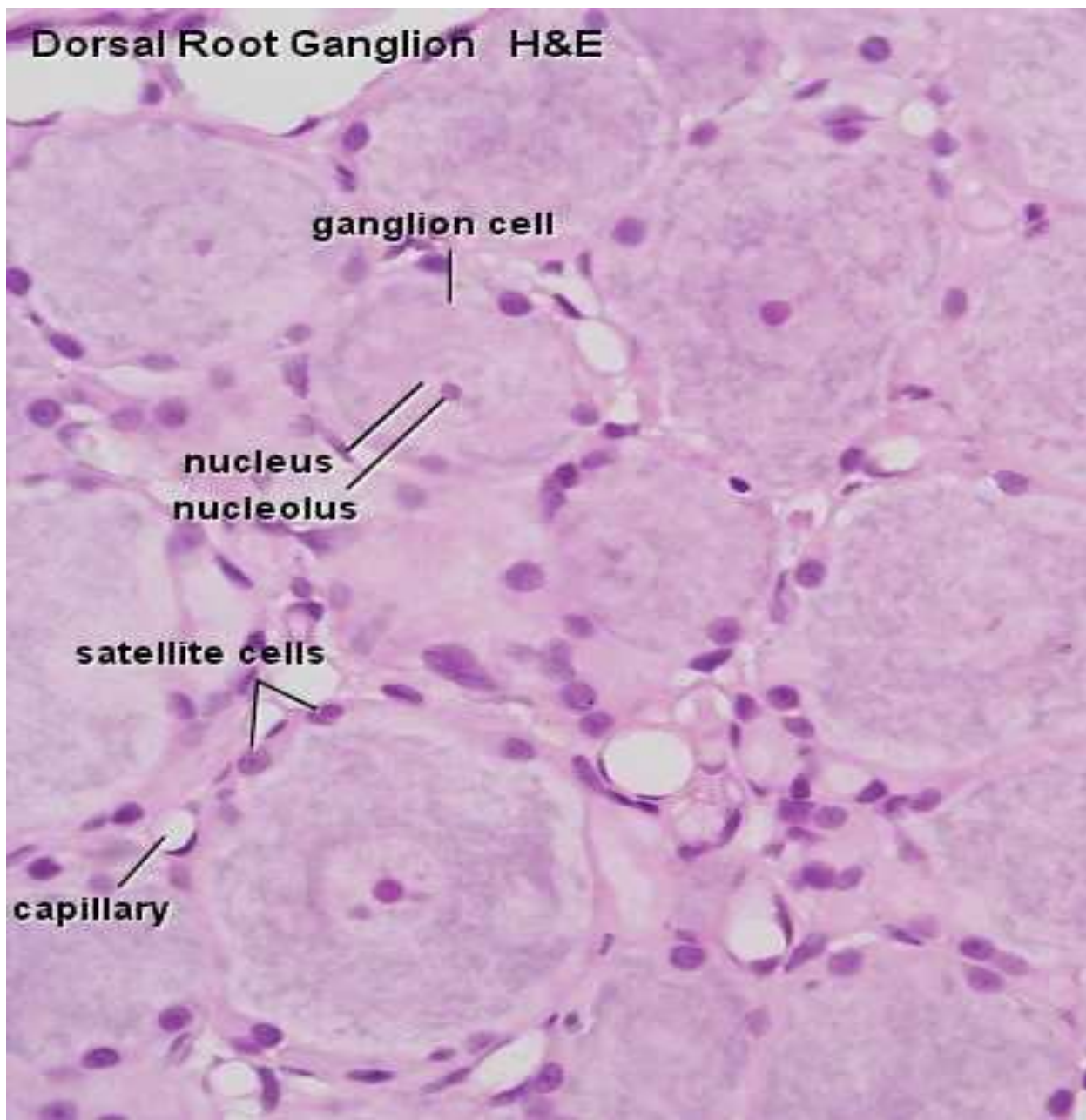
**endoneurium**

**nerve fibres**

**axon**

**remnants of myelin sheath**

**Dorsal Root Ganglion H&E**



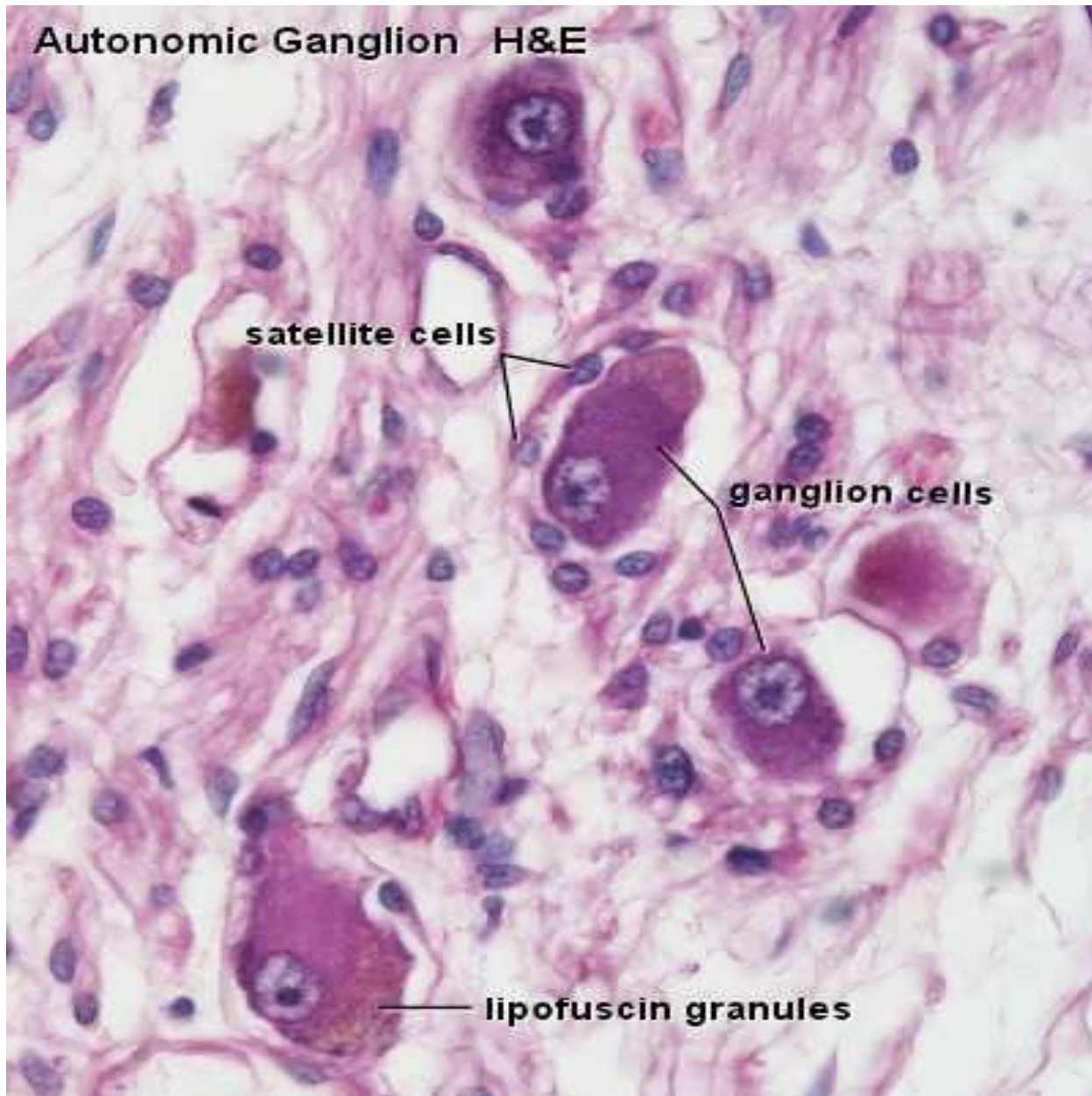
**ganglion cell**

**nucleus**  
**nucleolus**

**satellite cells**

**capillary**

**Autonomic Ganglion H&E**

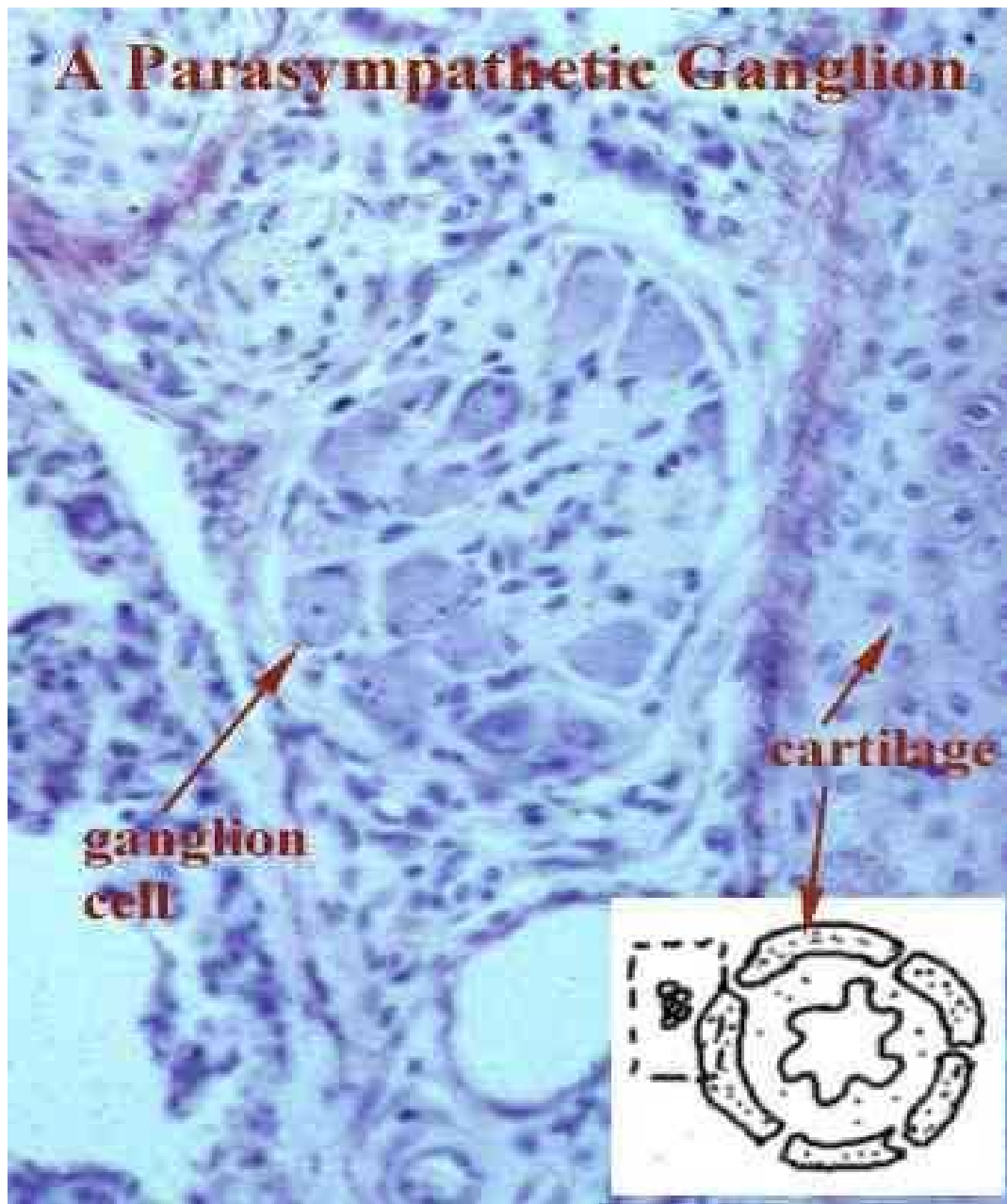


**satellite cells**

**ganglion cells**

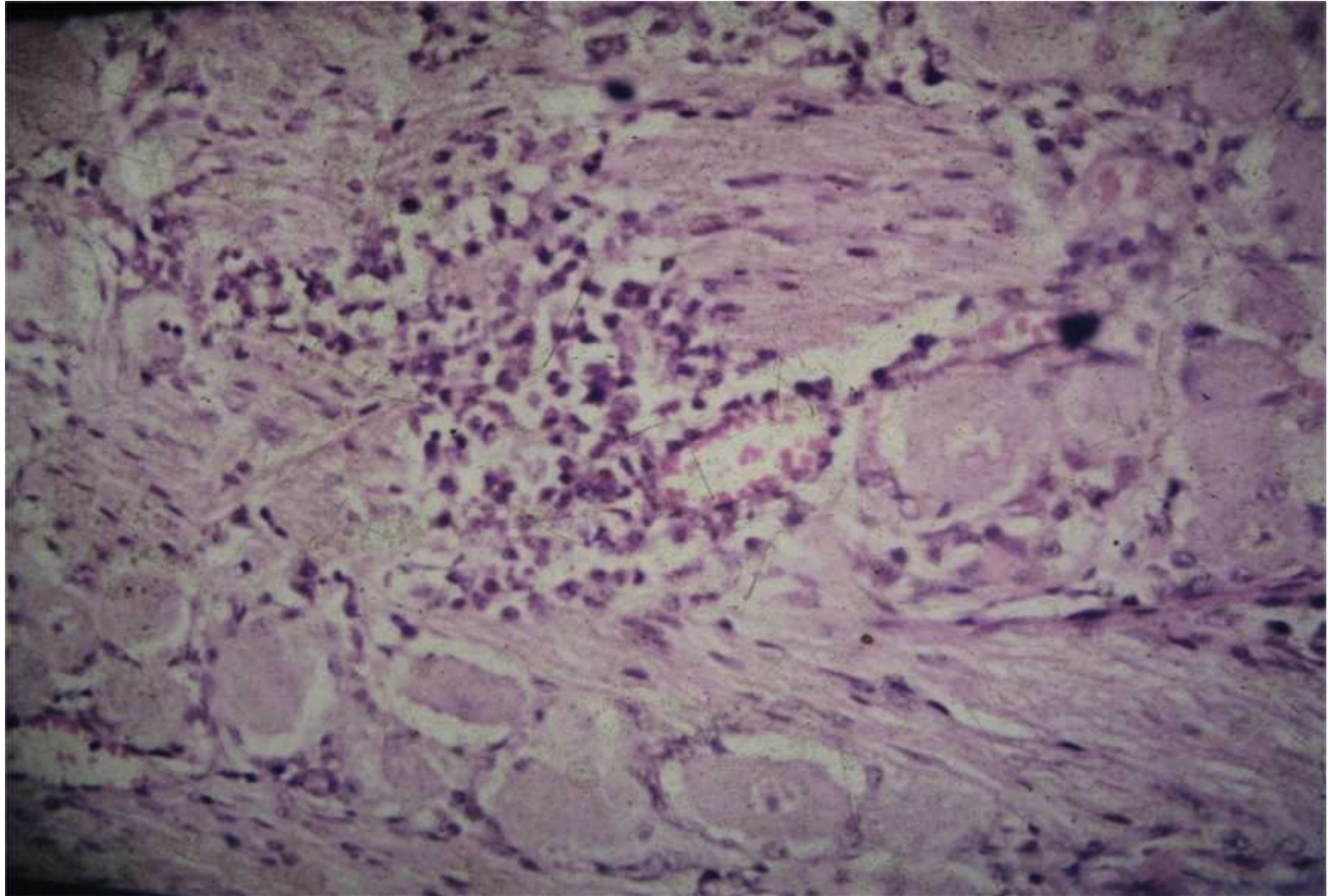
**lipofuscin granules**

# A Parasympathetic Ganglion

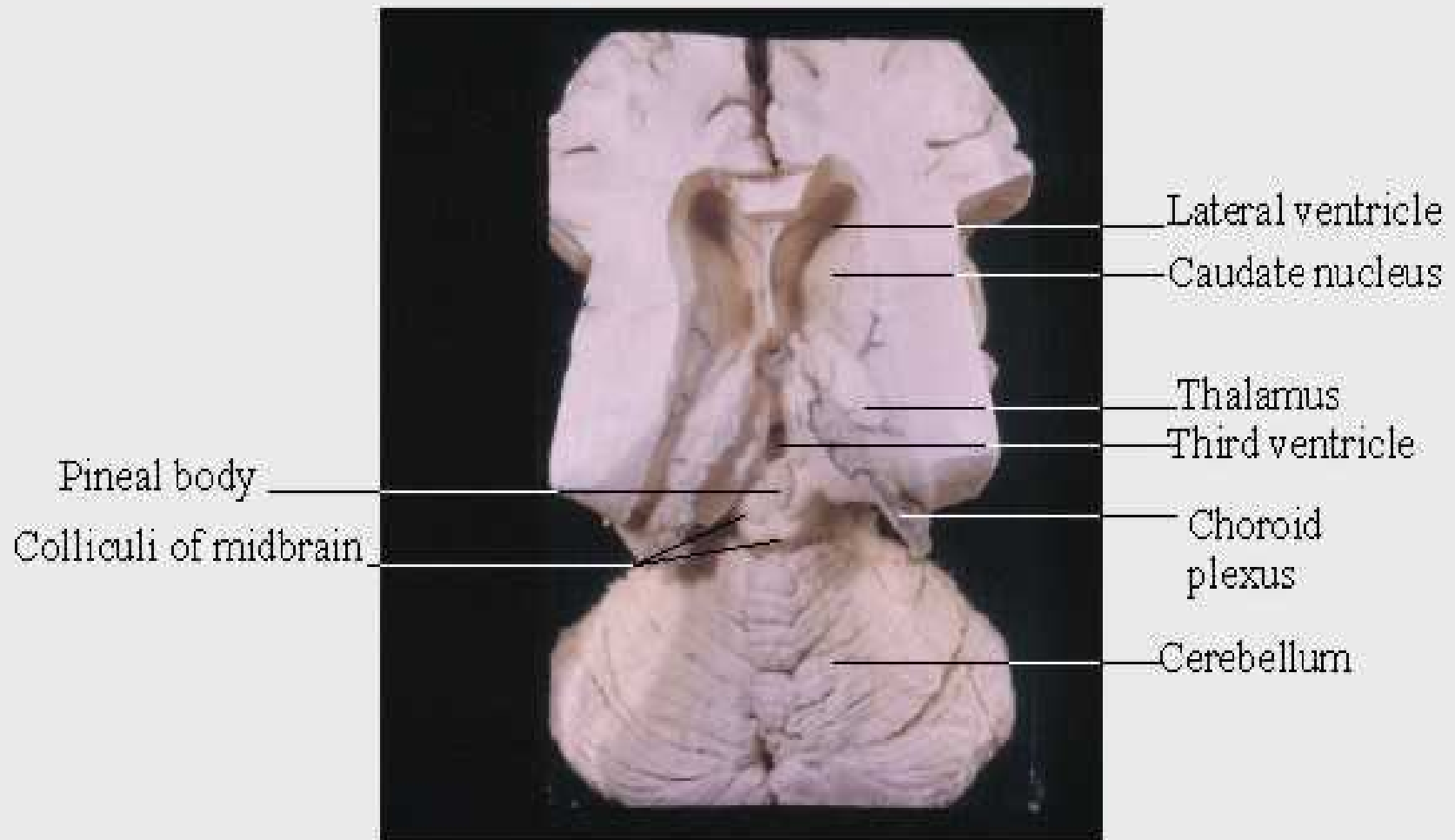


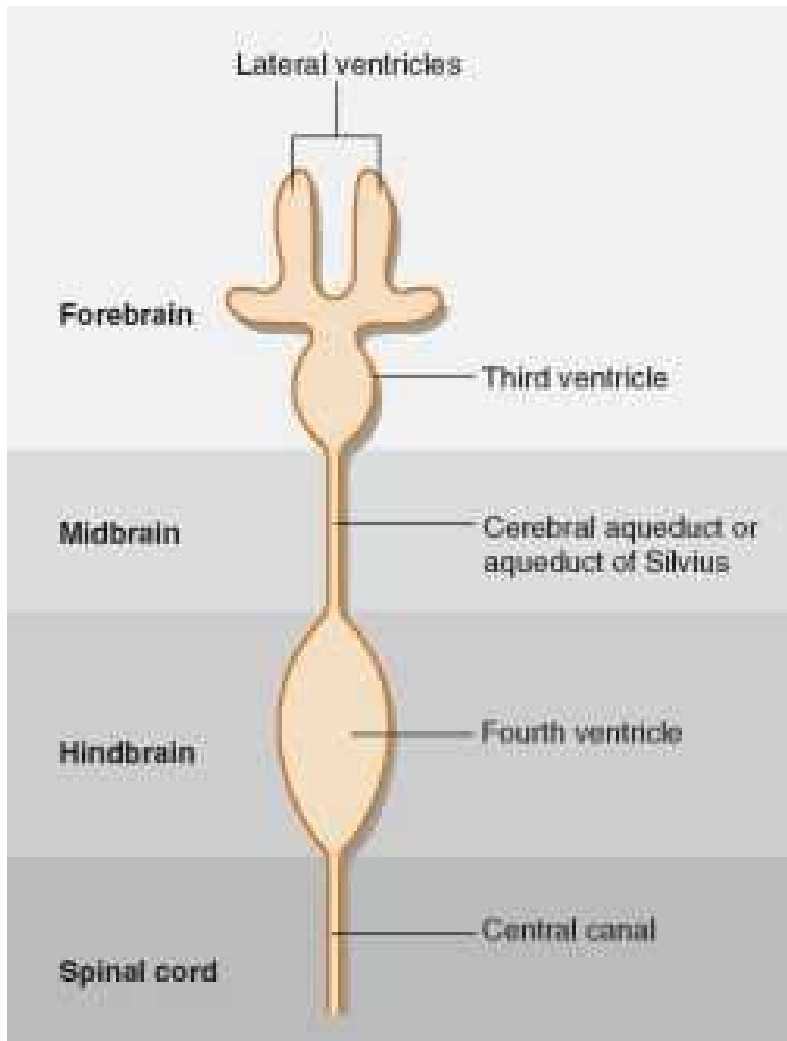


ganglioneuritis

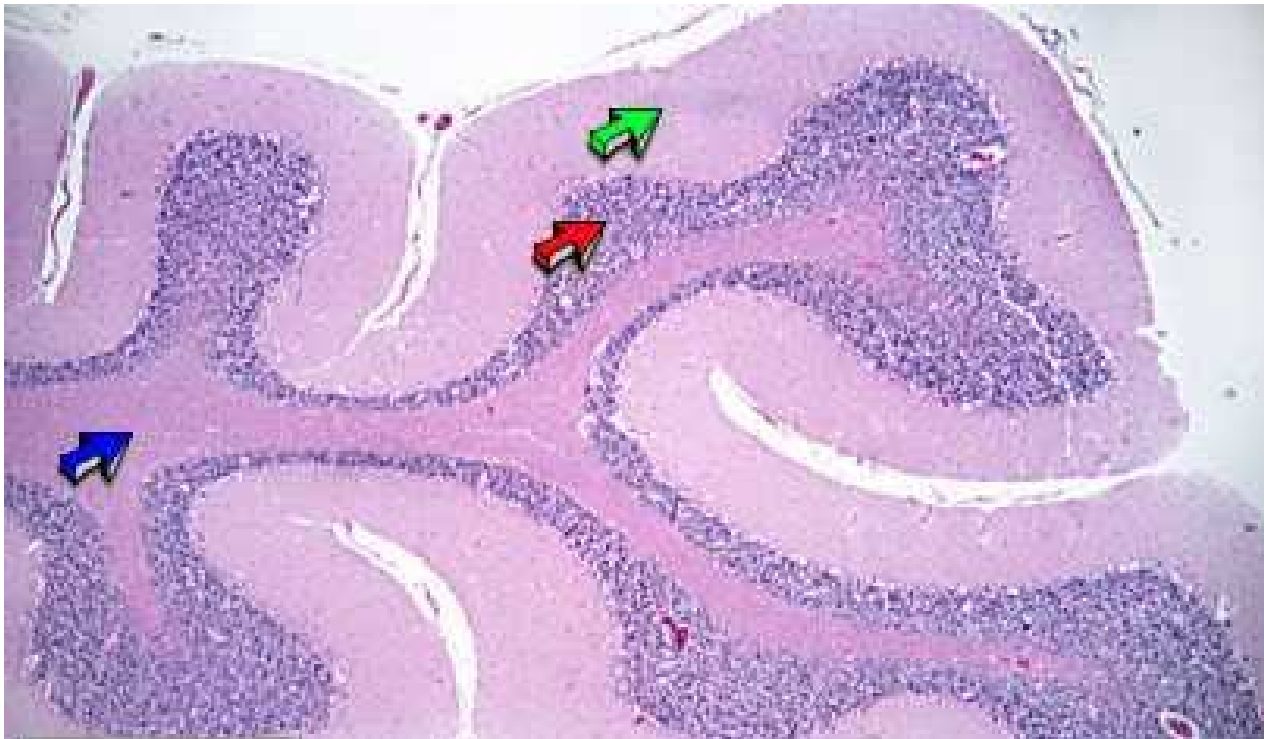


## Ventricles of the Brain









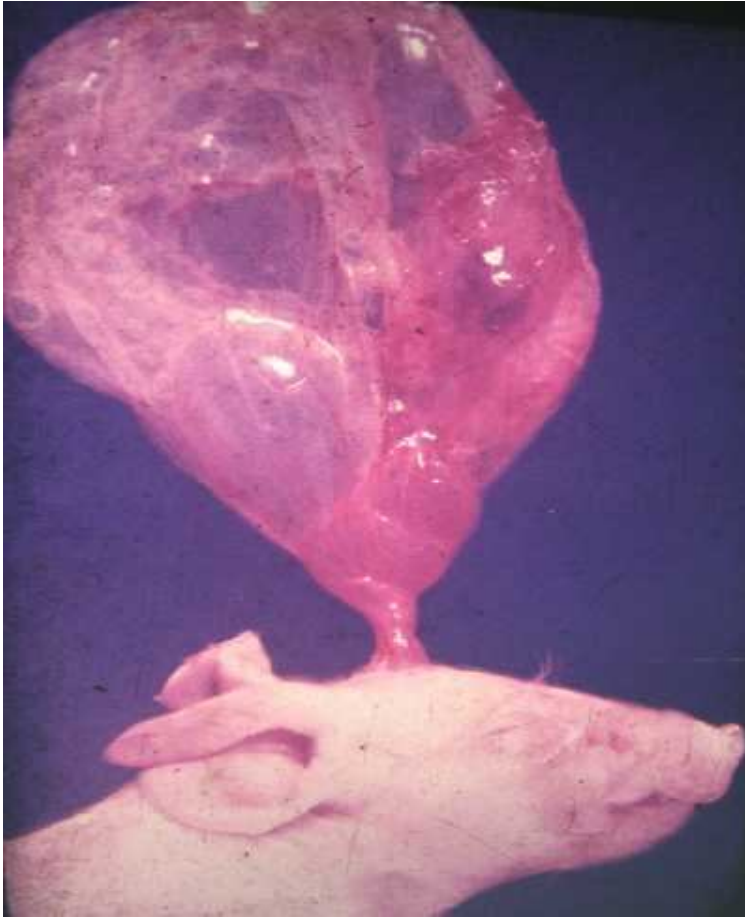
**Slide 8 Cerebellum**

**H&E Slide. At low power, the cerebellum is distinguished by its three visible layers. The inner layer (blue arrow) is white matter on the inside compared to its "outer" spinal cord position.**

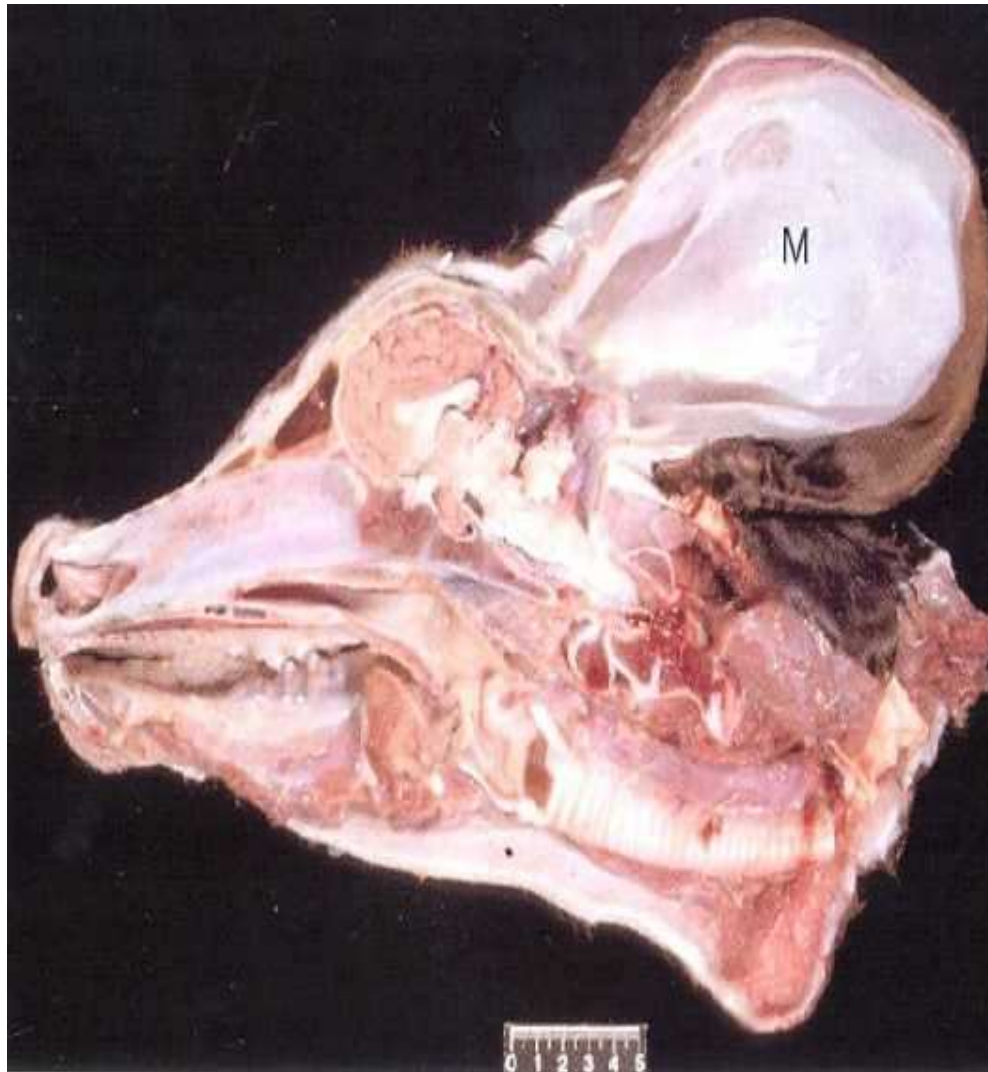
**The outer layers are gray matter: granular layer (red arrow), molecular layer (green arrow), and Purkinje cell layer between those two layers.**

**Bar = 1 mm**

**meningocoele**



meningocele



Spina bifida occulta



hydromyelia

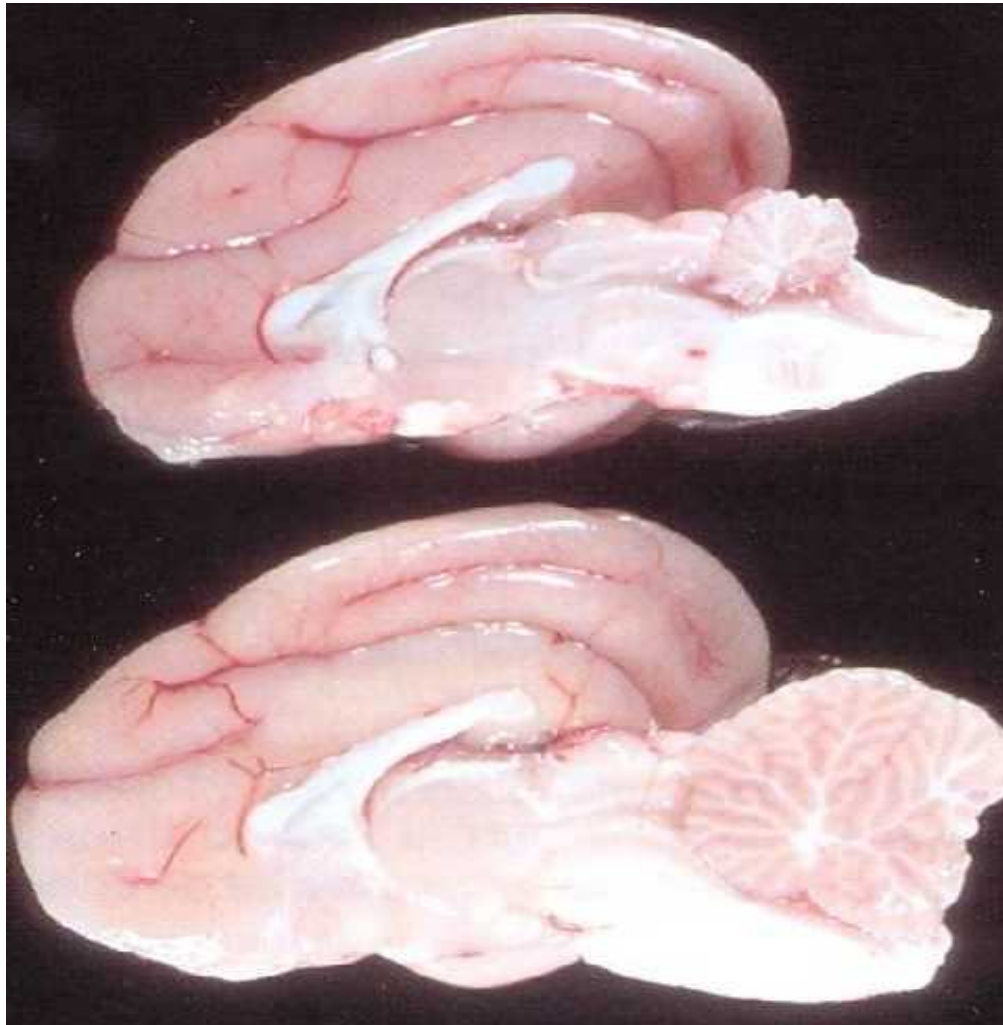


lyssencephaly





## Cerebellar hypoplasia



## Cerebellar hypoplasia

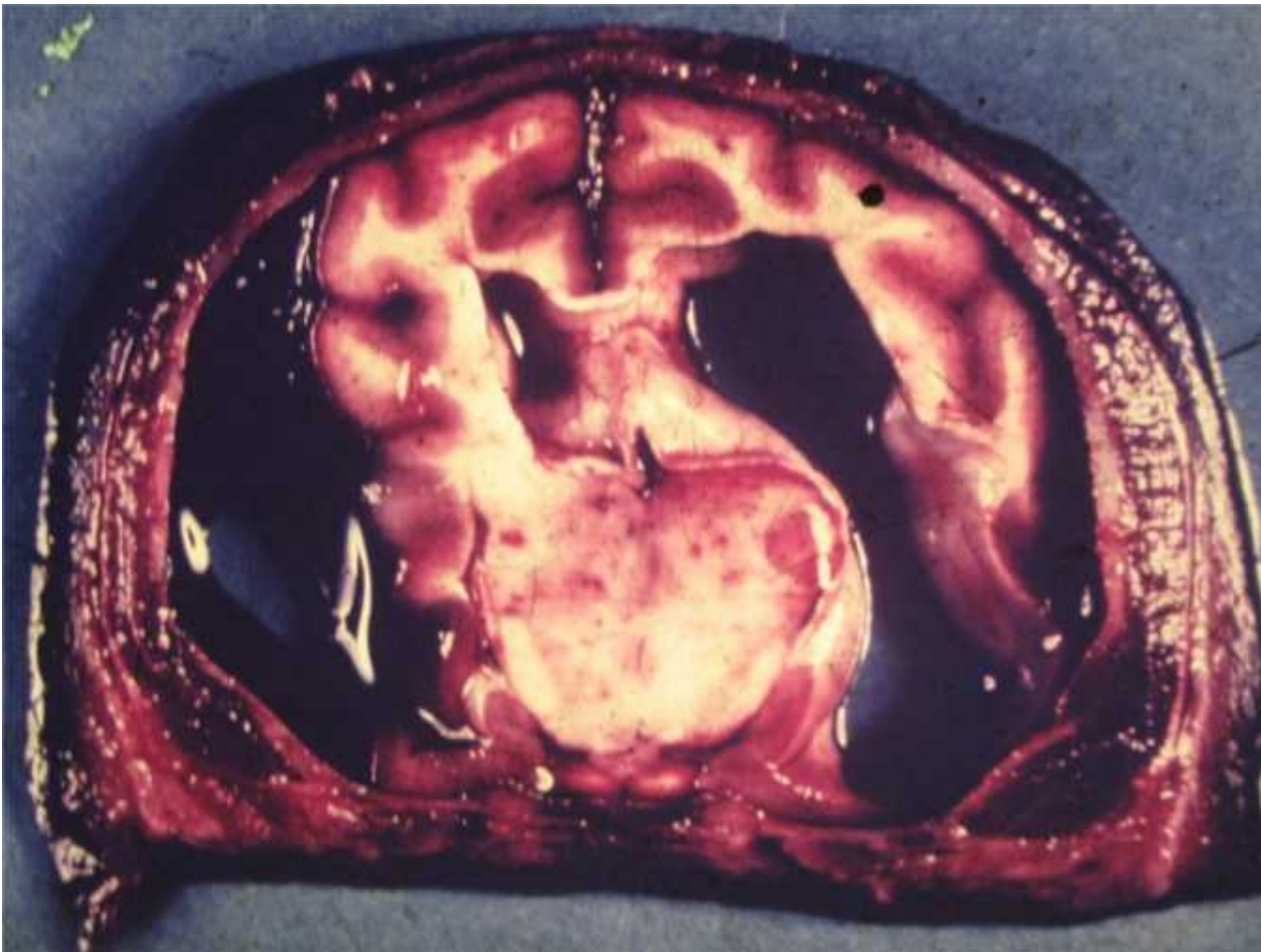


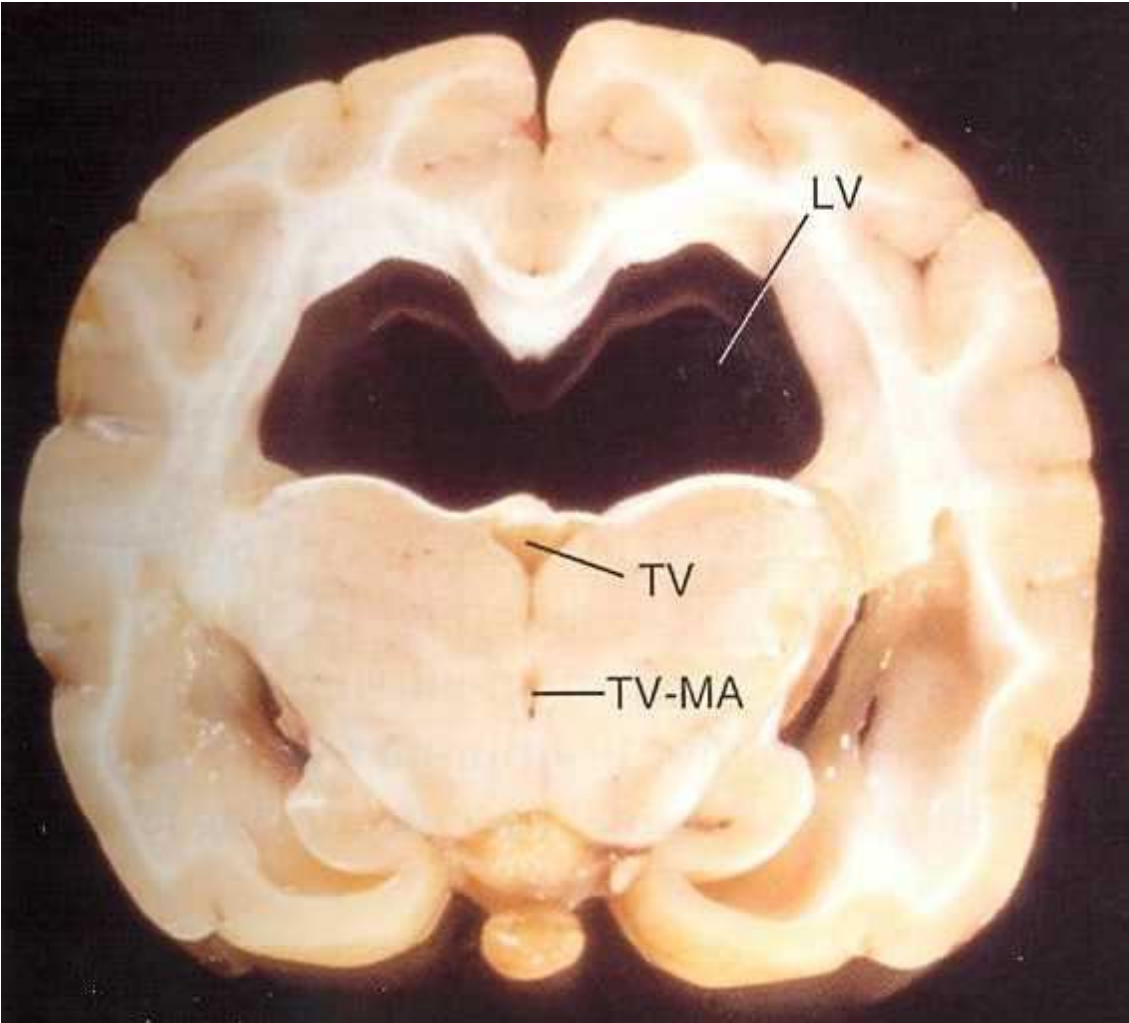


**hydrocephalus**



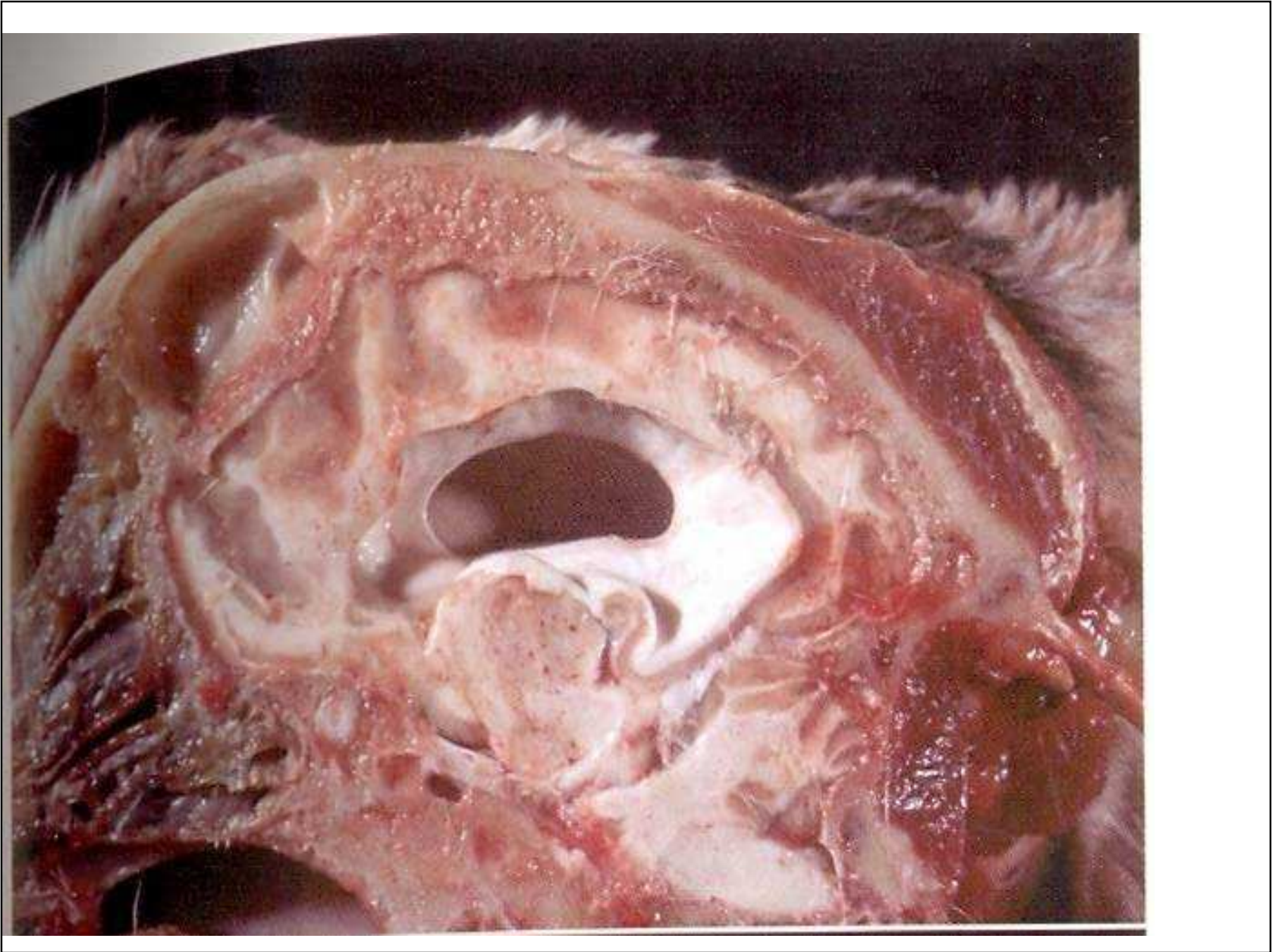
hydrocephalus



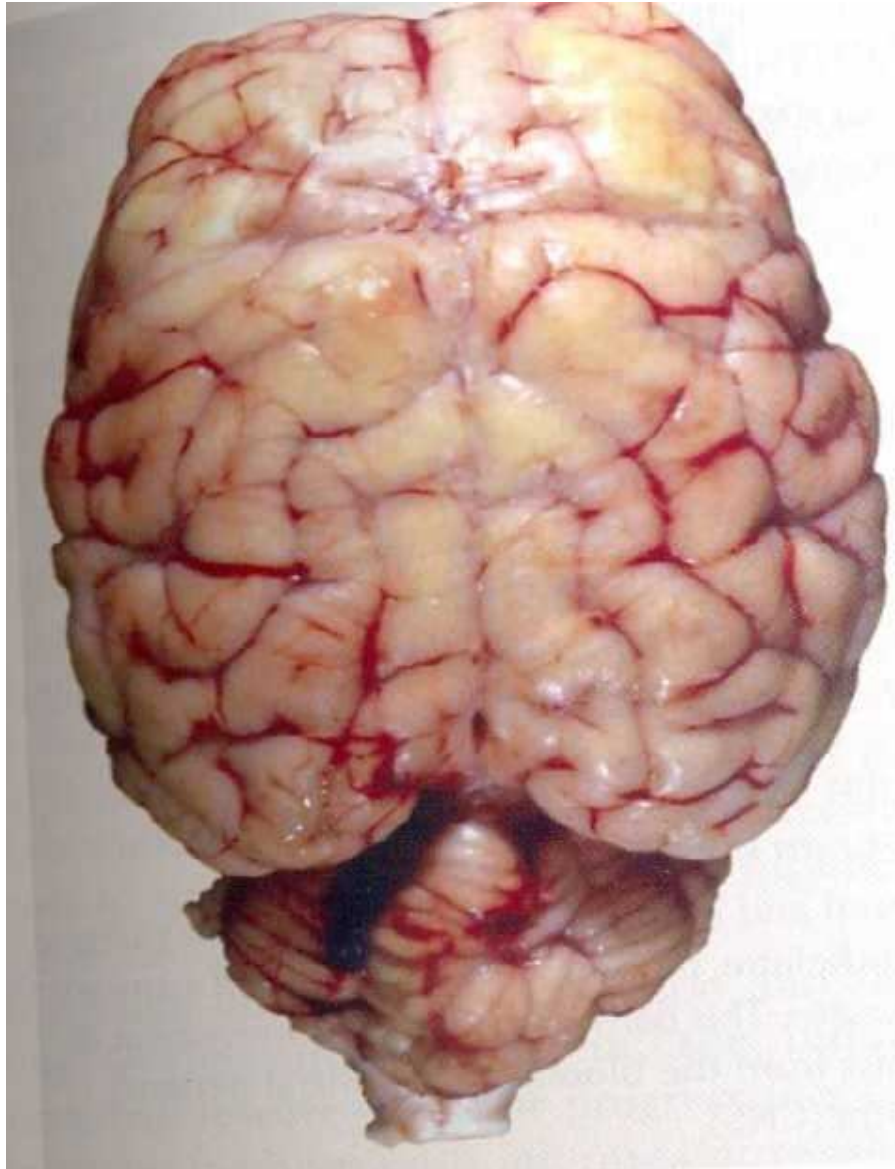








Cerebral edema





Cerebral abscess

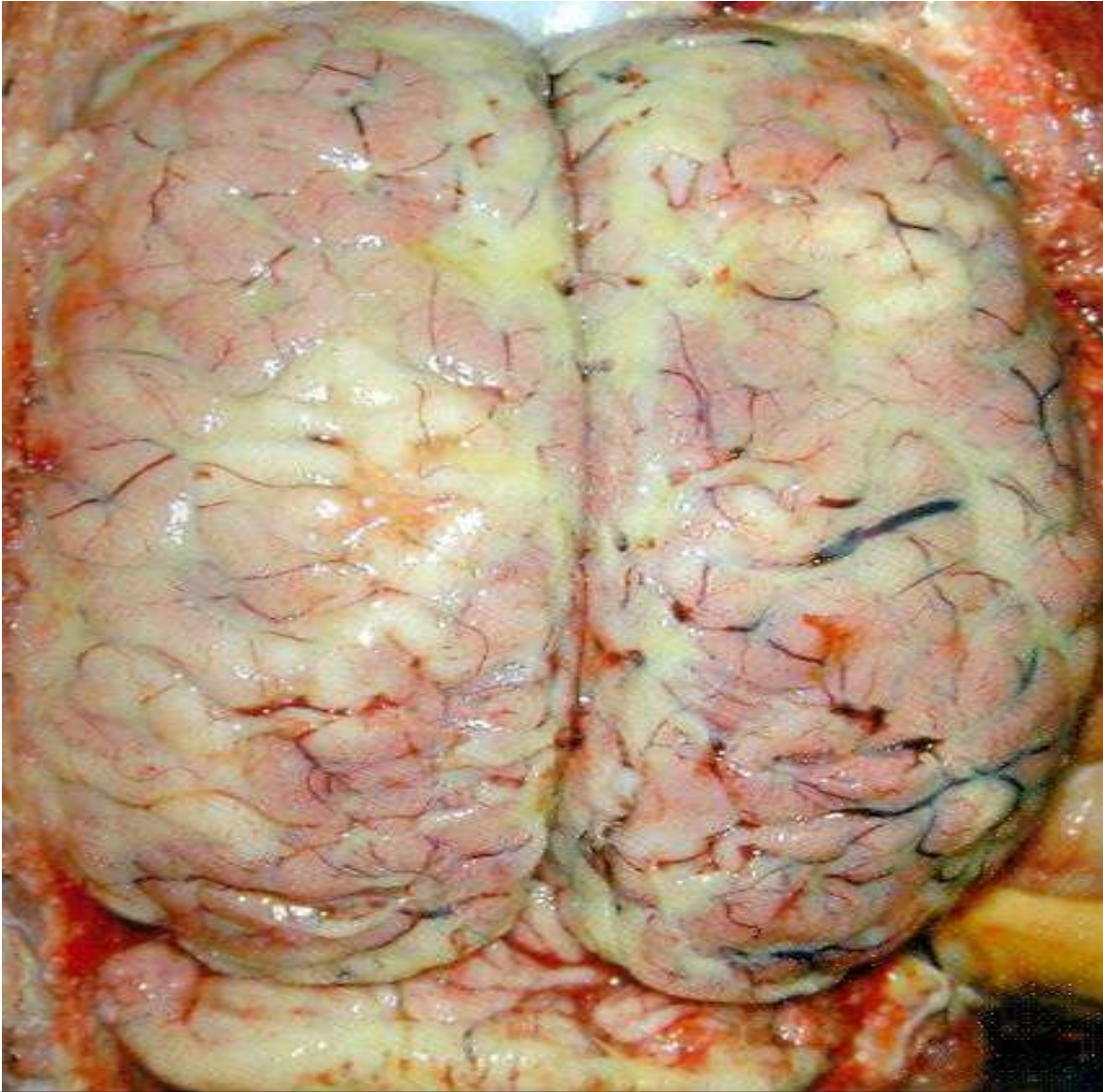




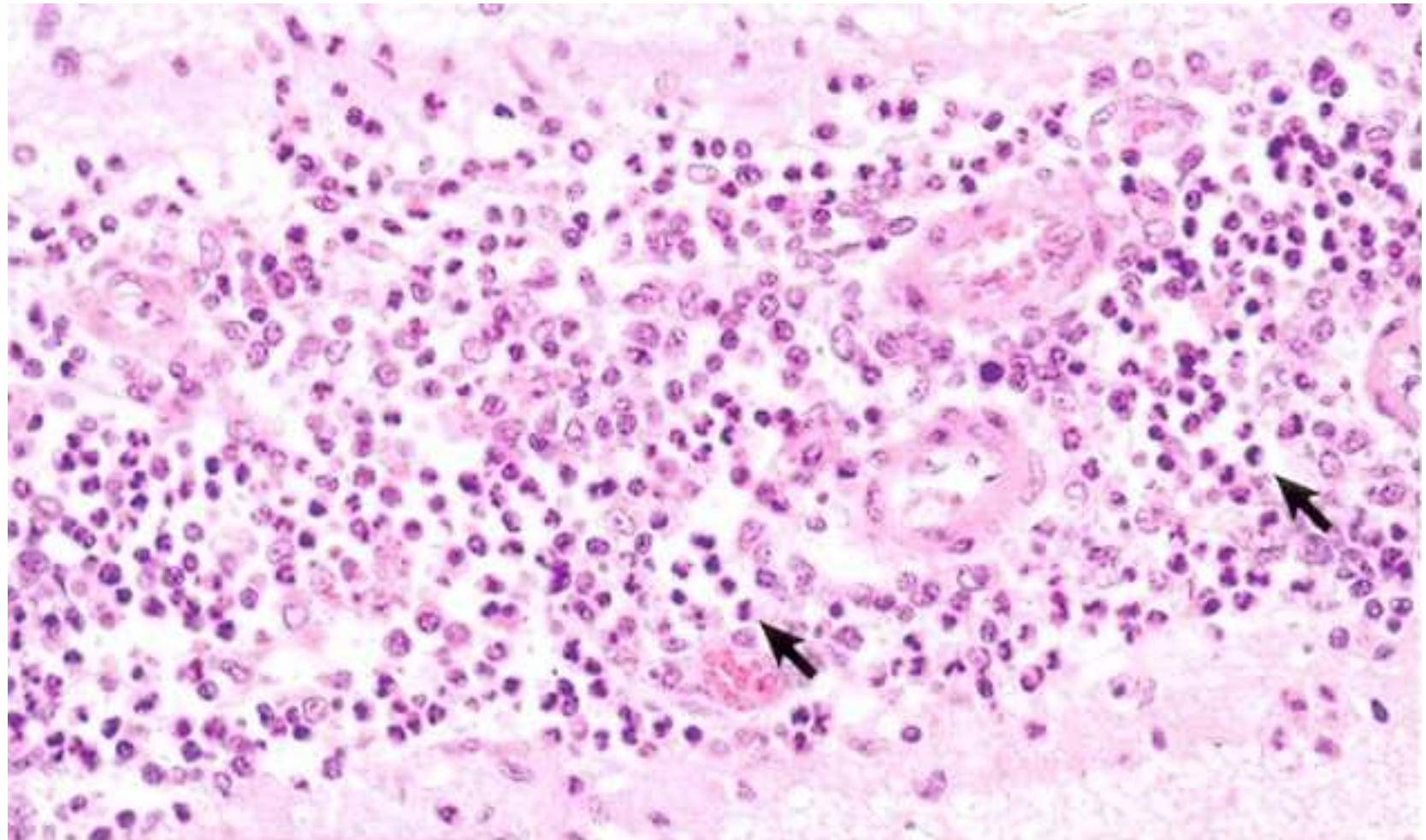
Cerebral abscess



Meningitis (purulent)

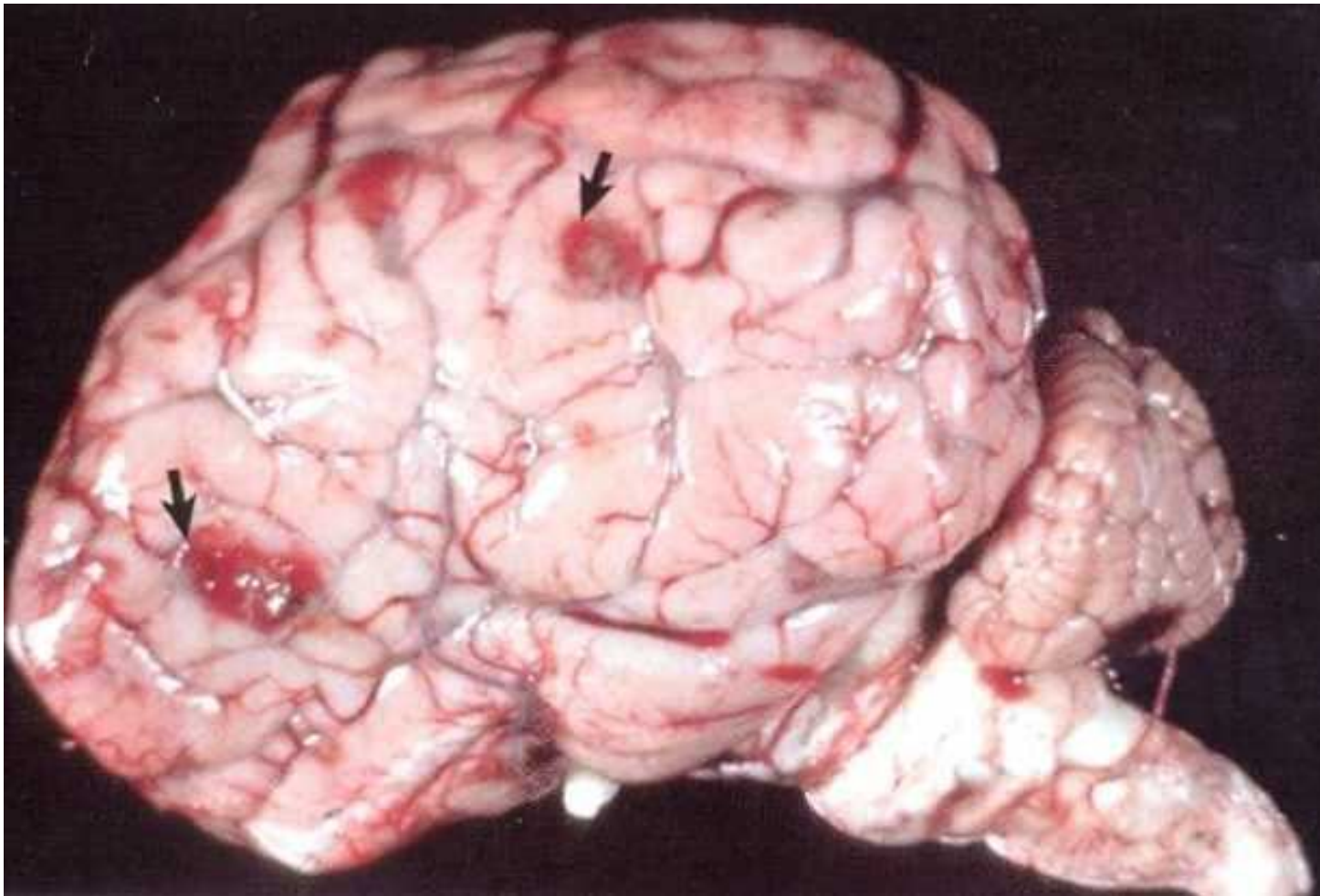




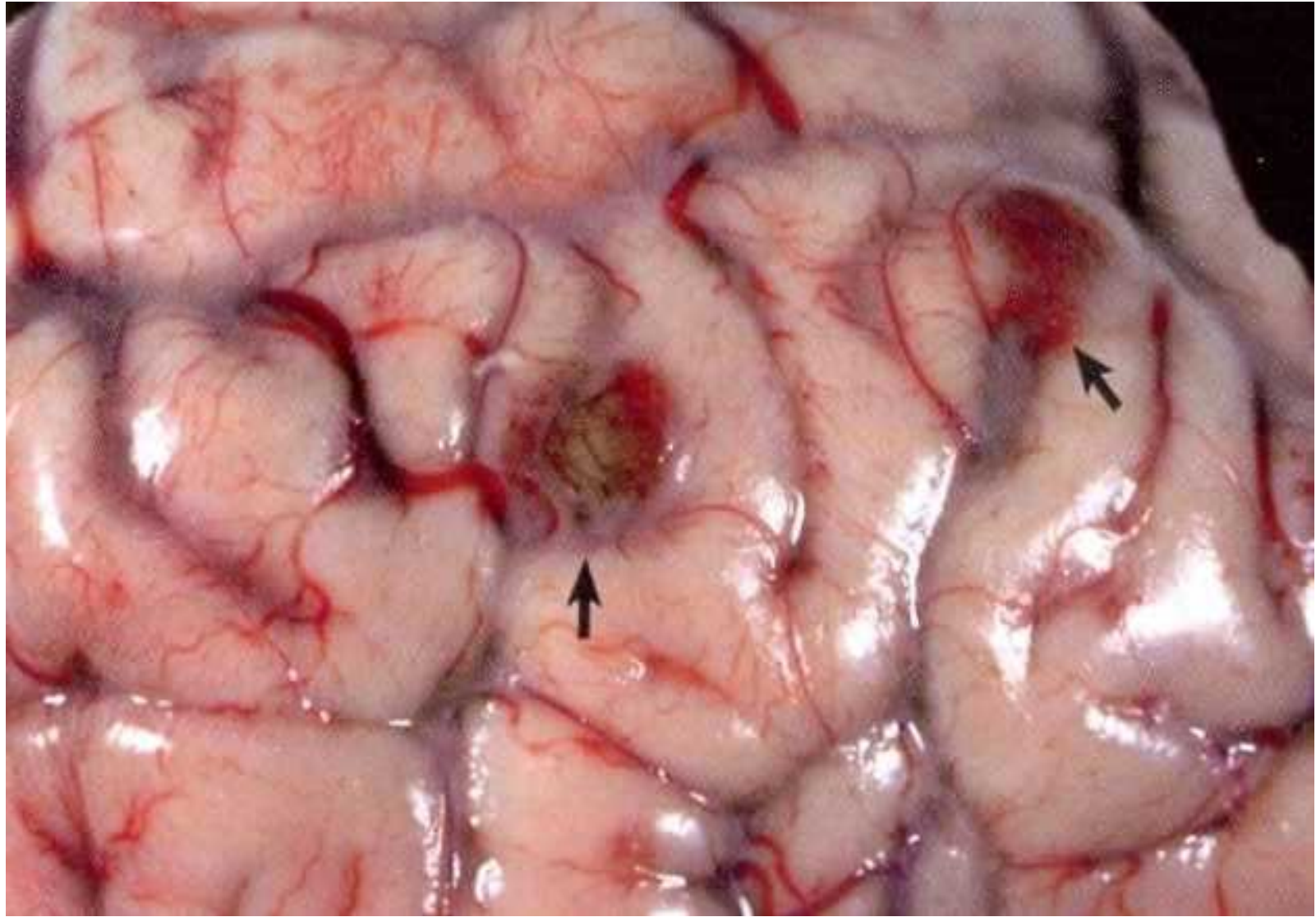


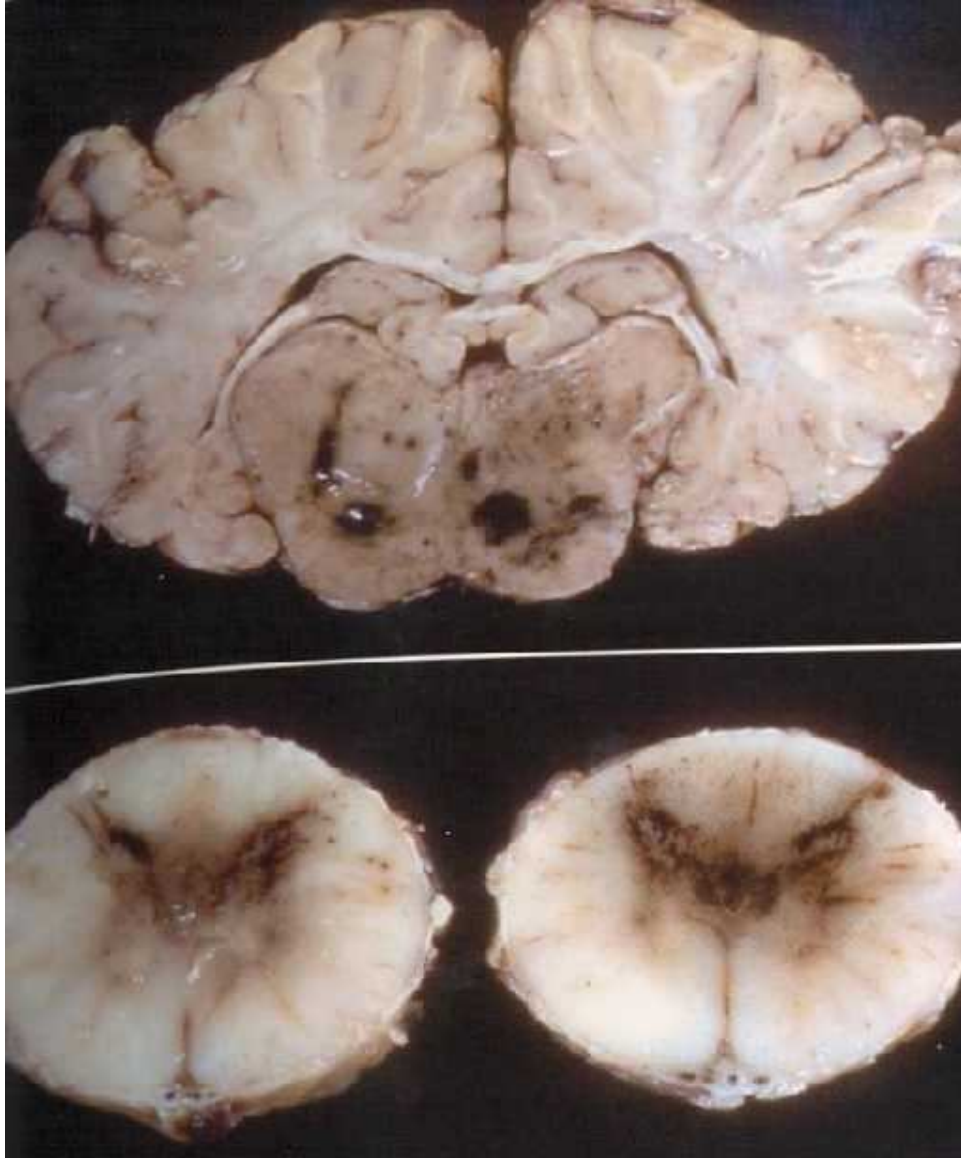
Cerebral infarcts

(TEME)



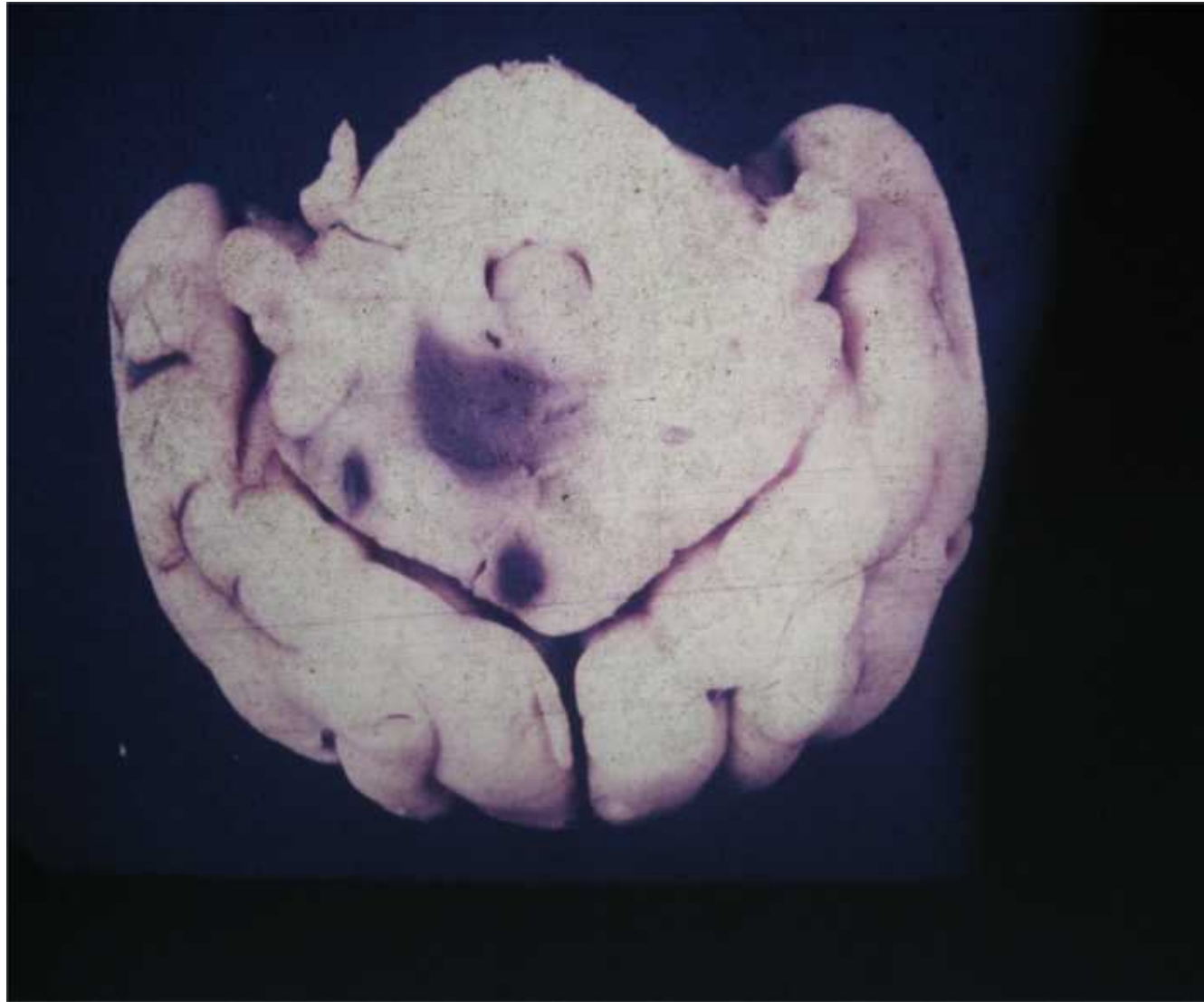


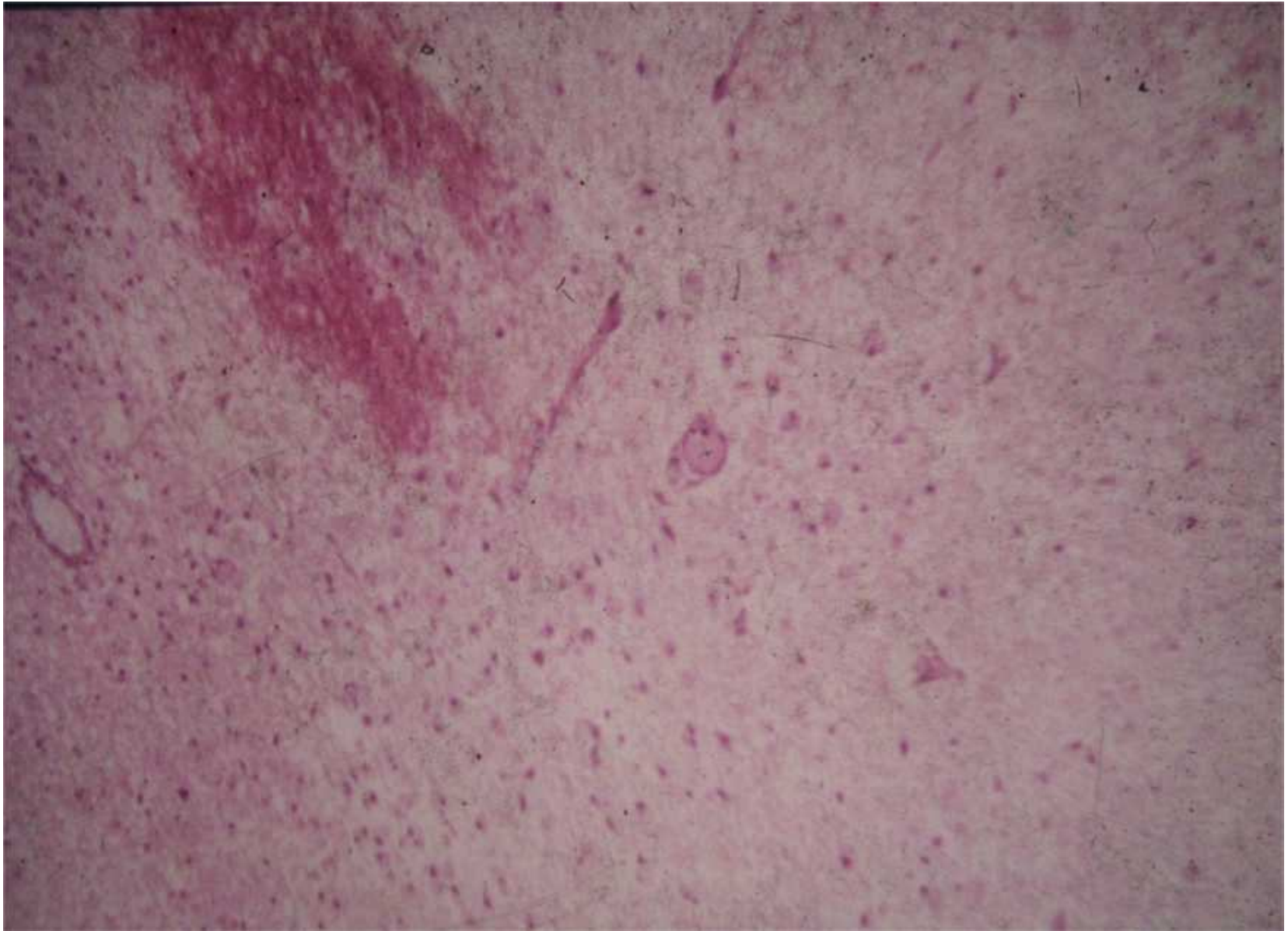


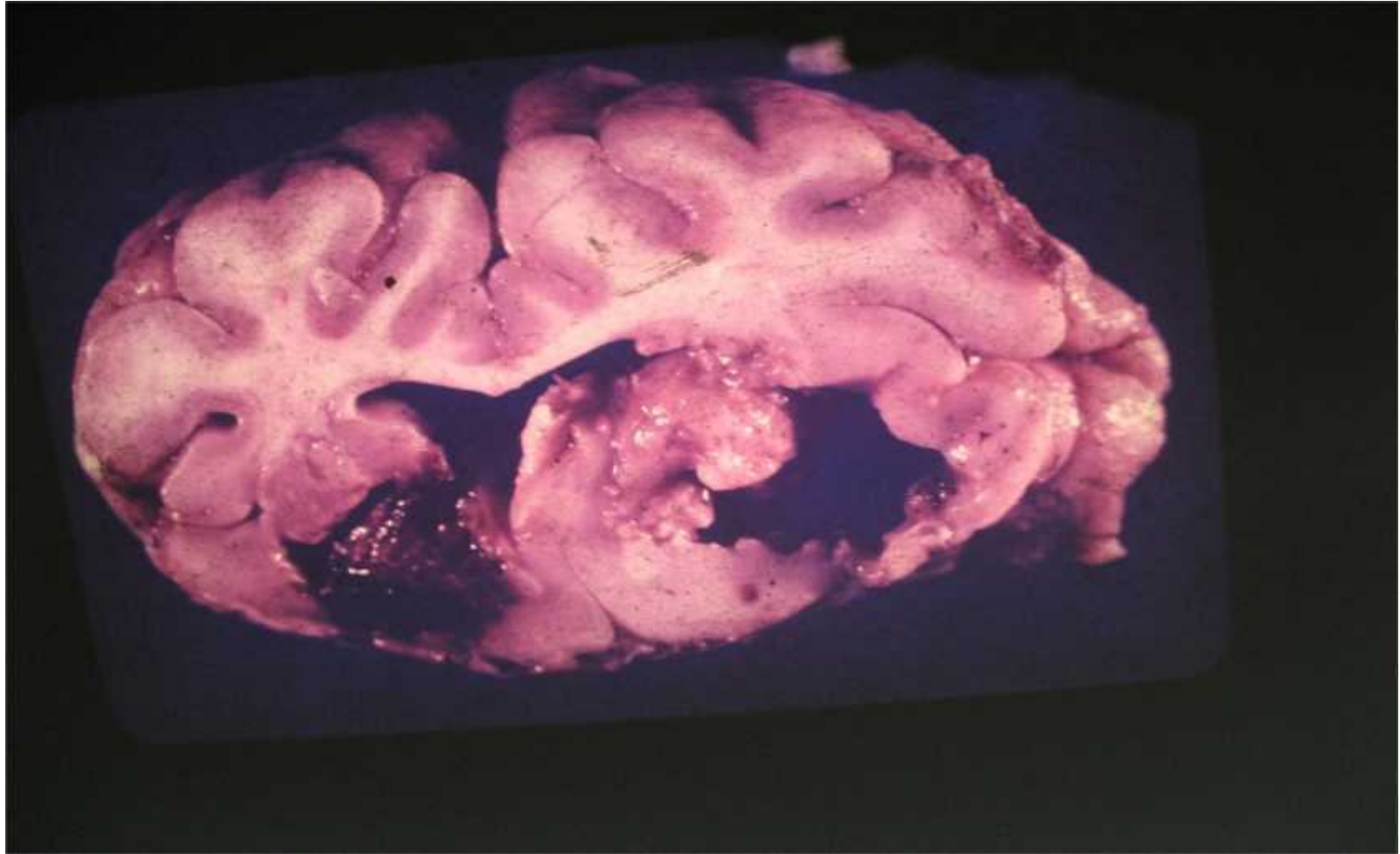












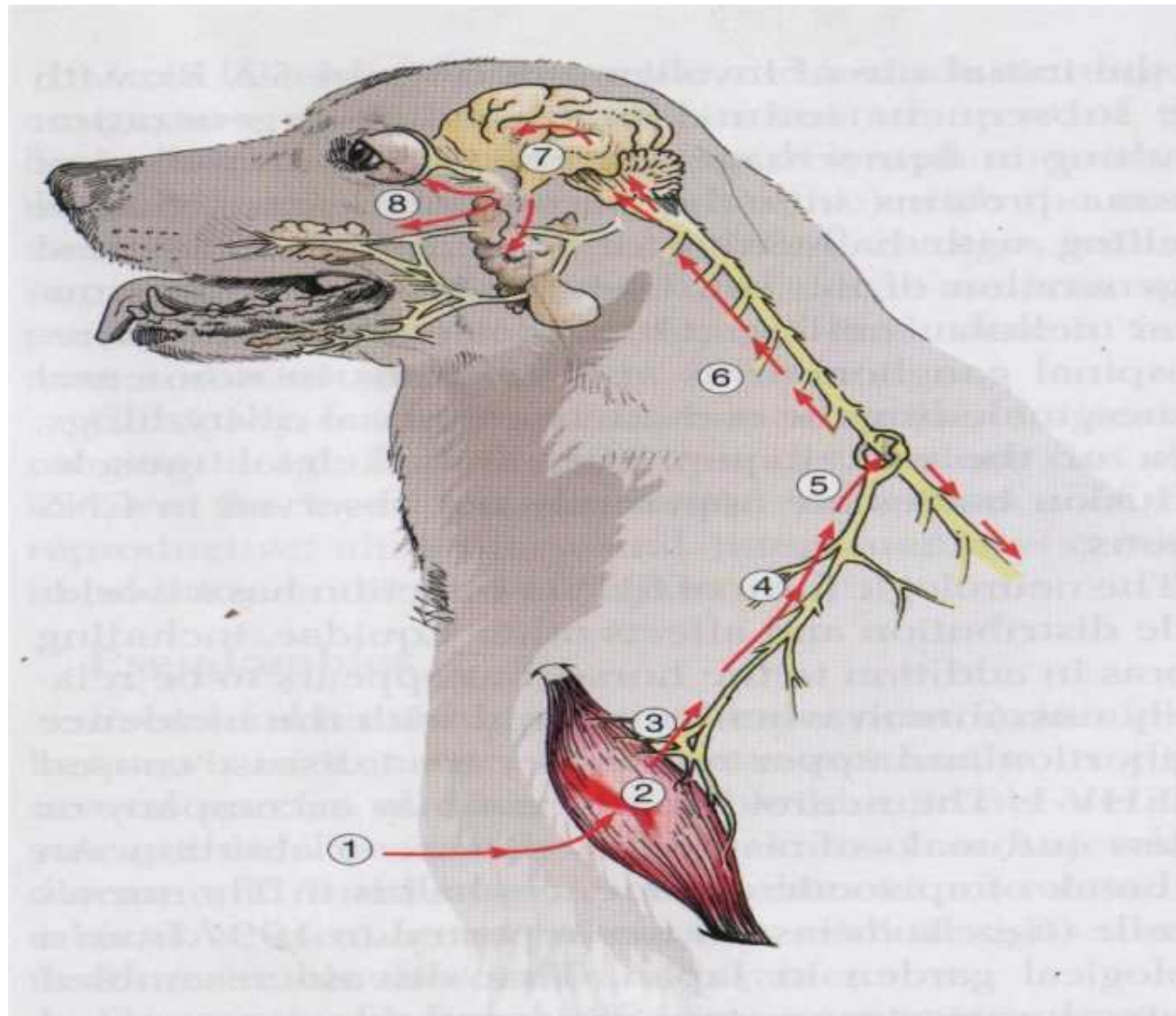


Cytoplasmic vacuolation

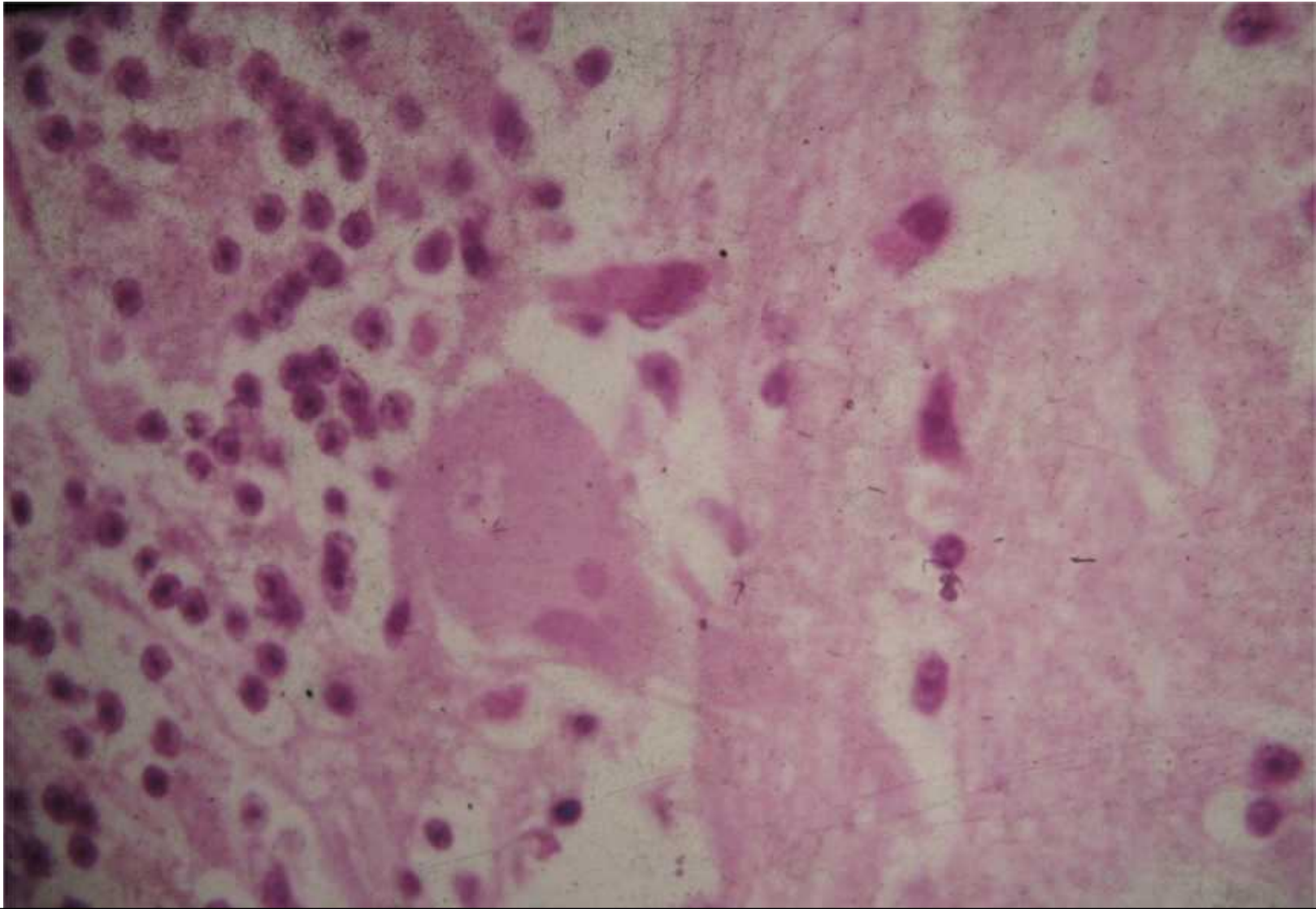




## Pathogenesis of Rabies

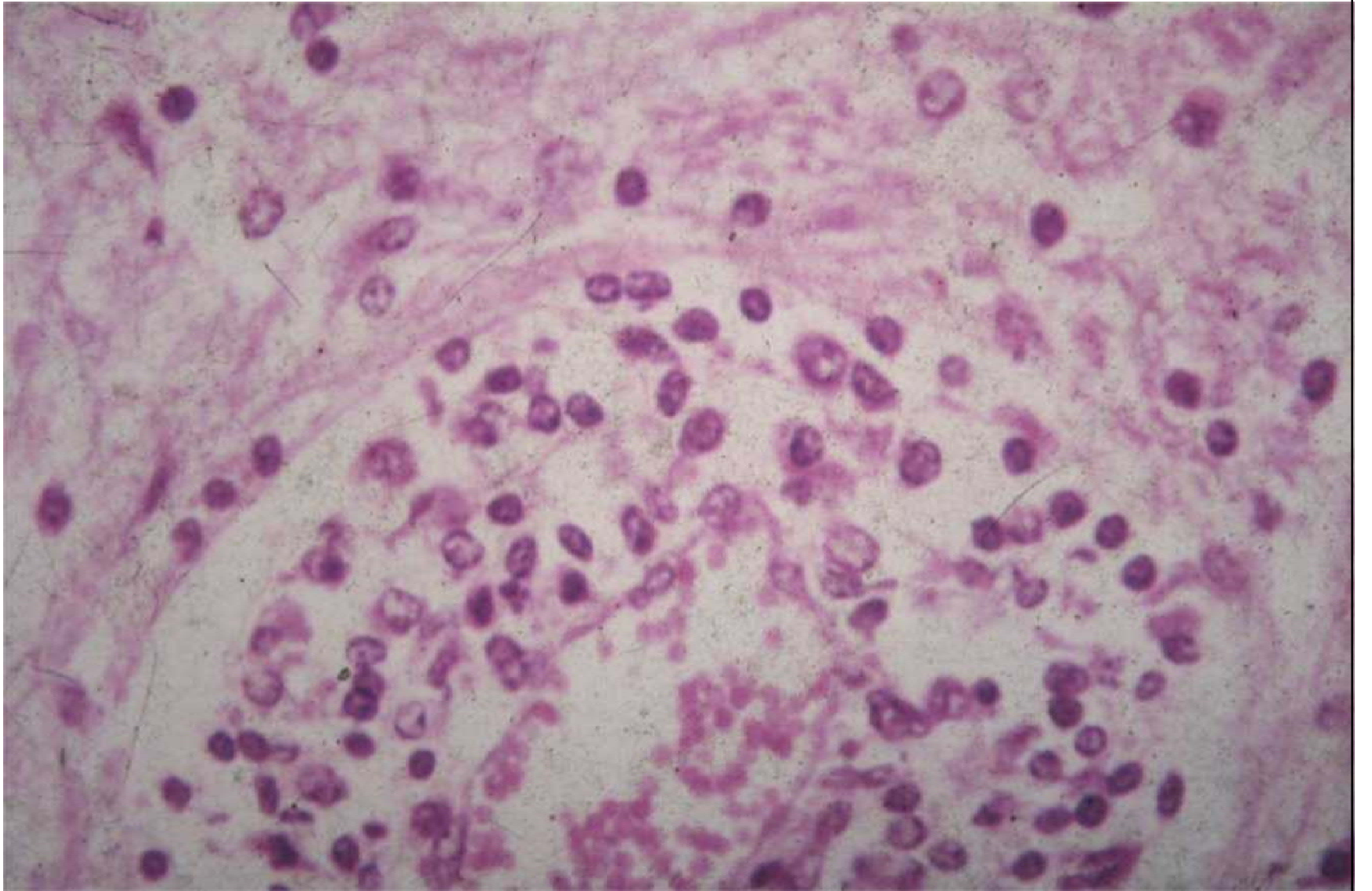


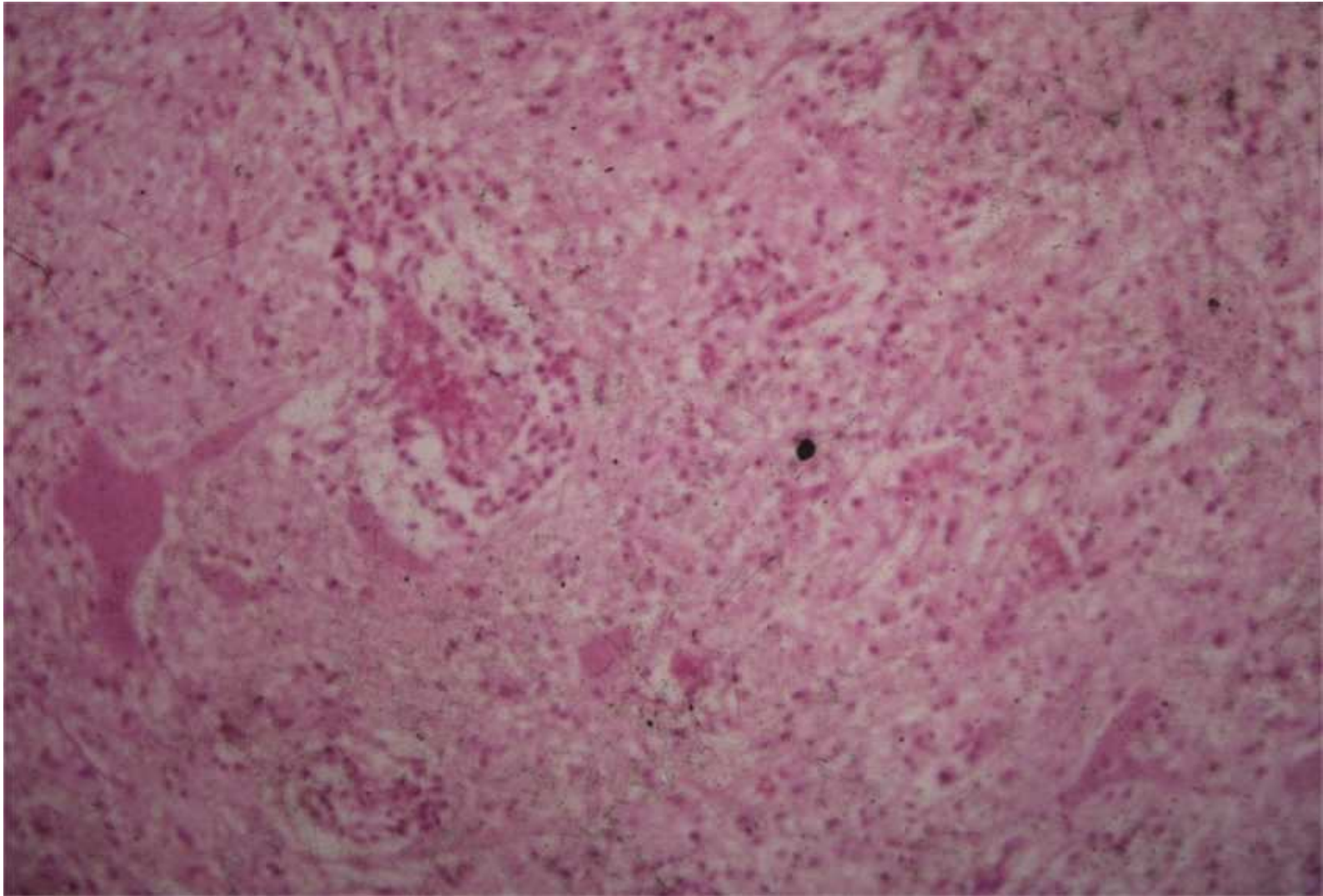
Negri bodies



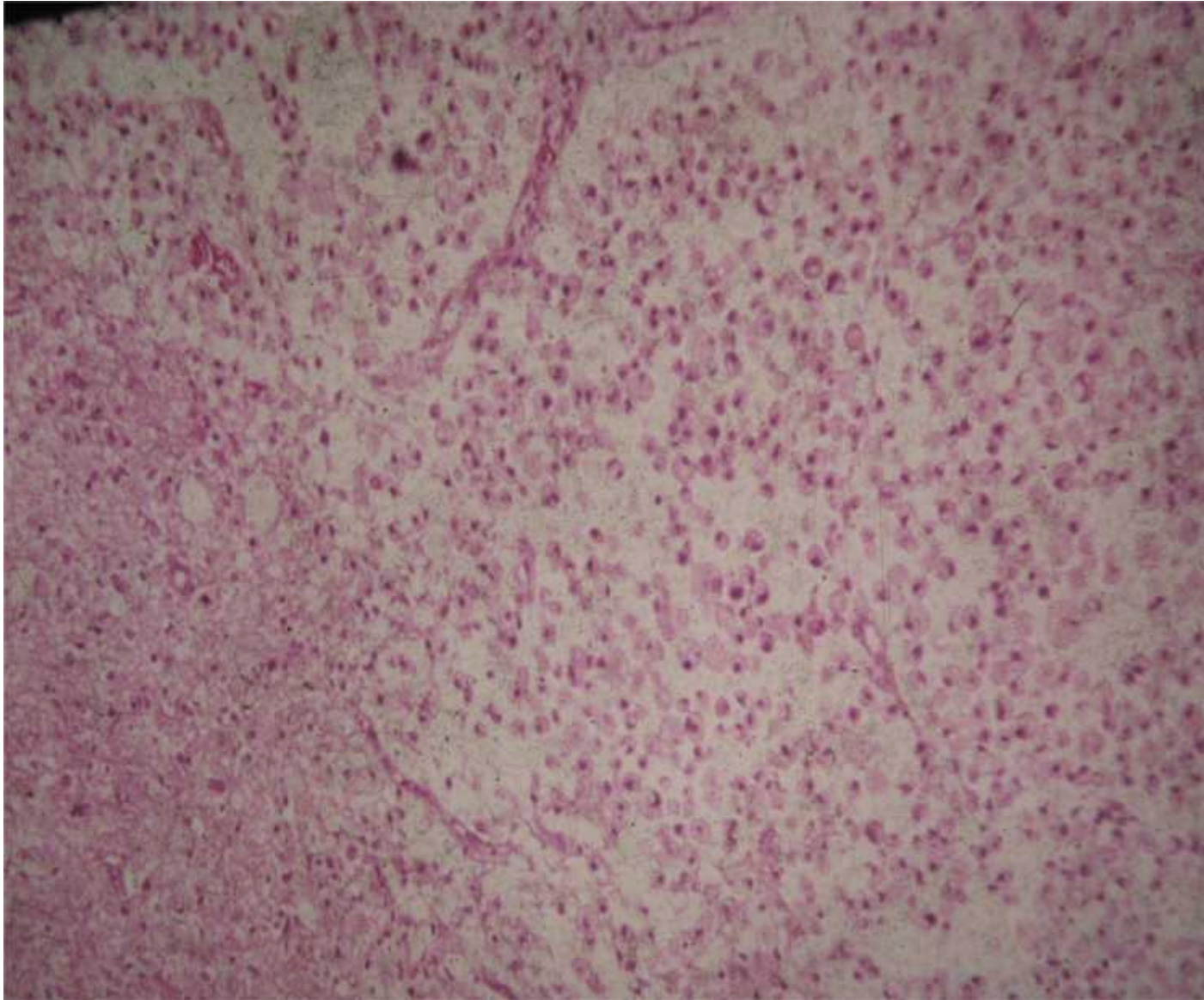


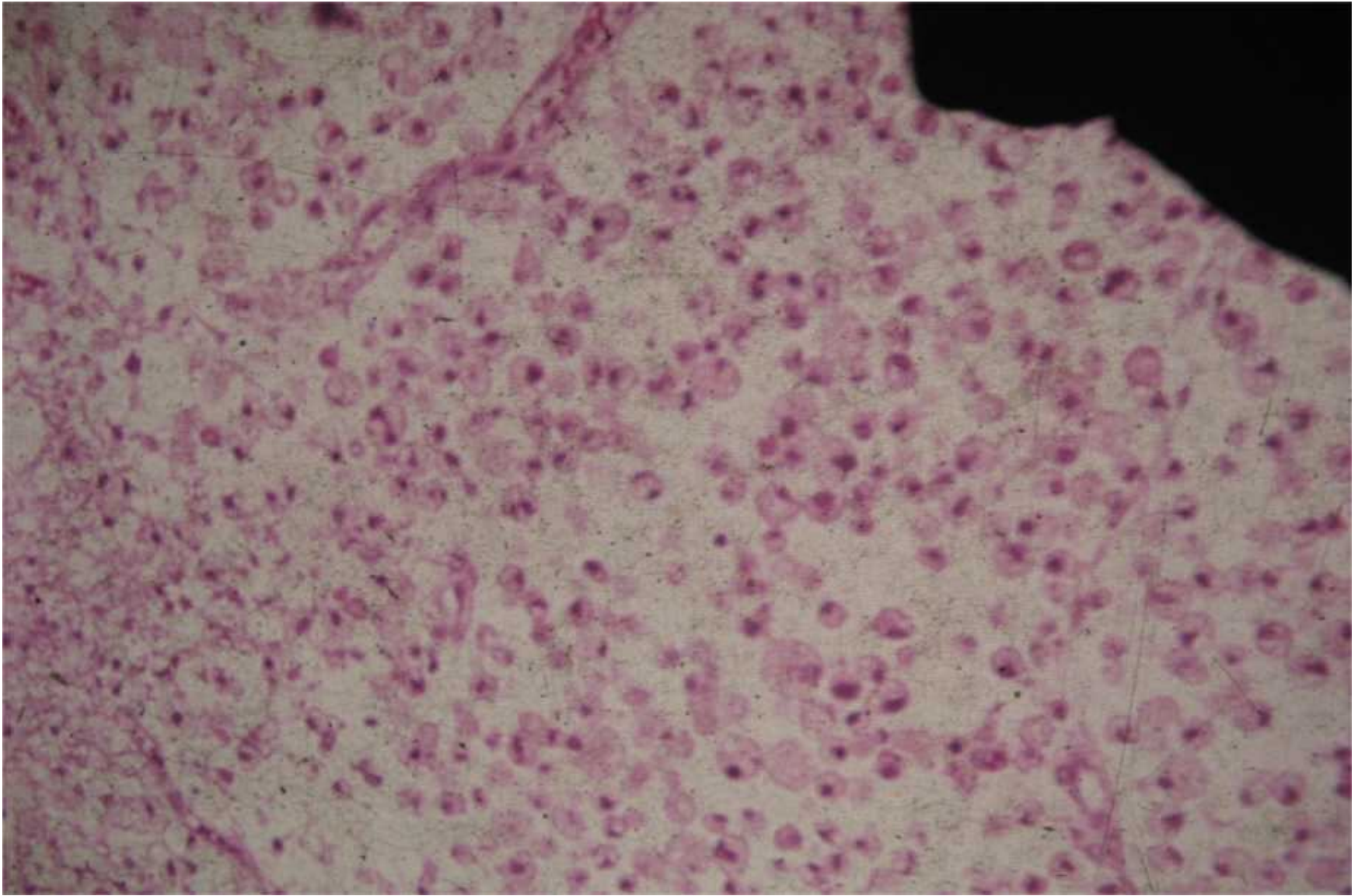




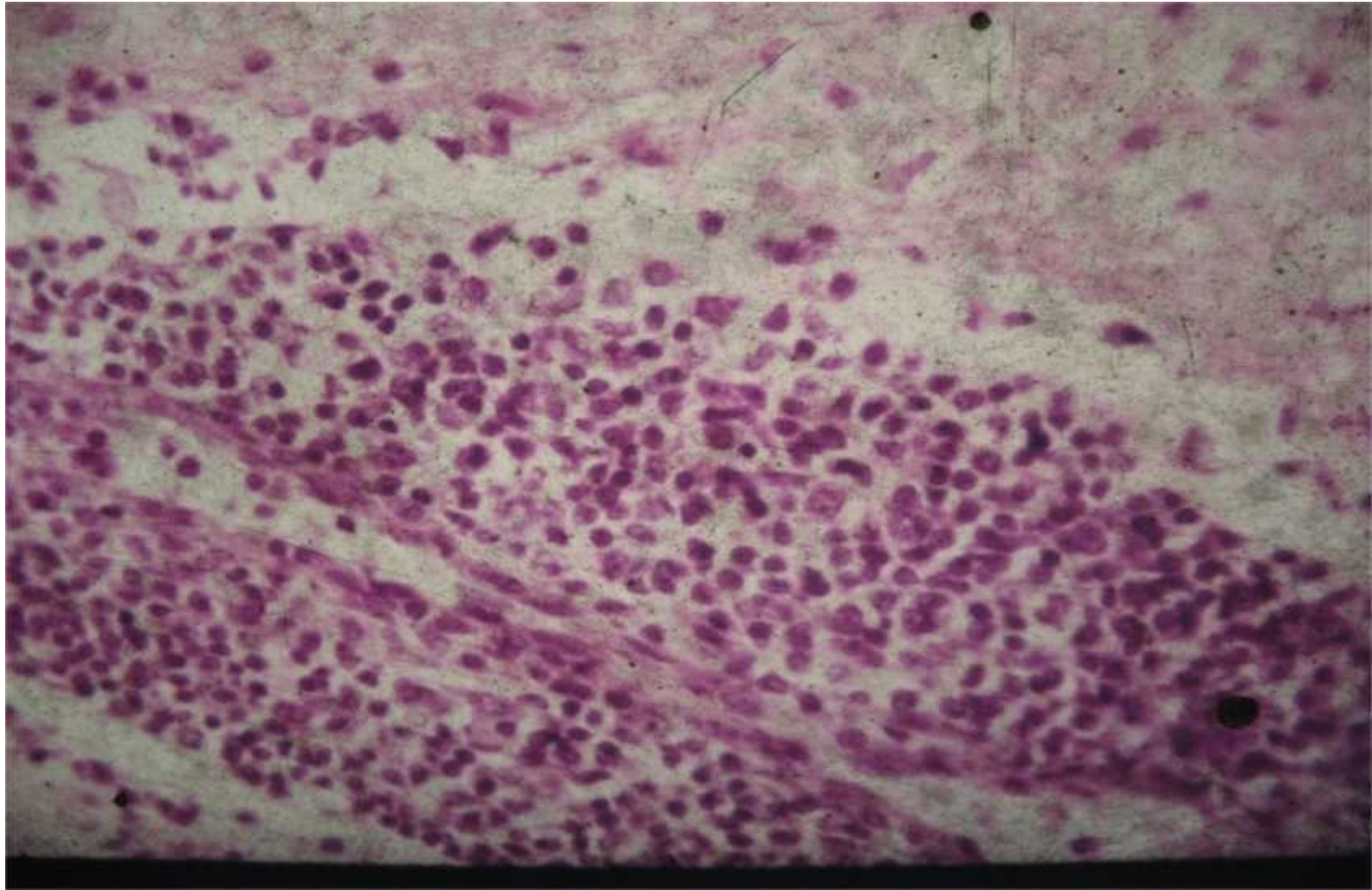




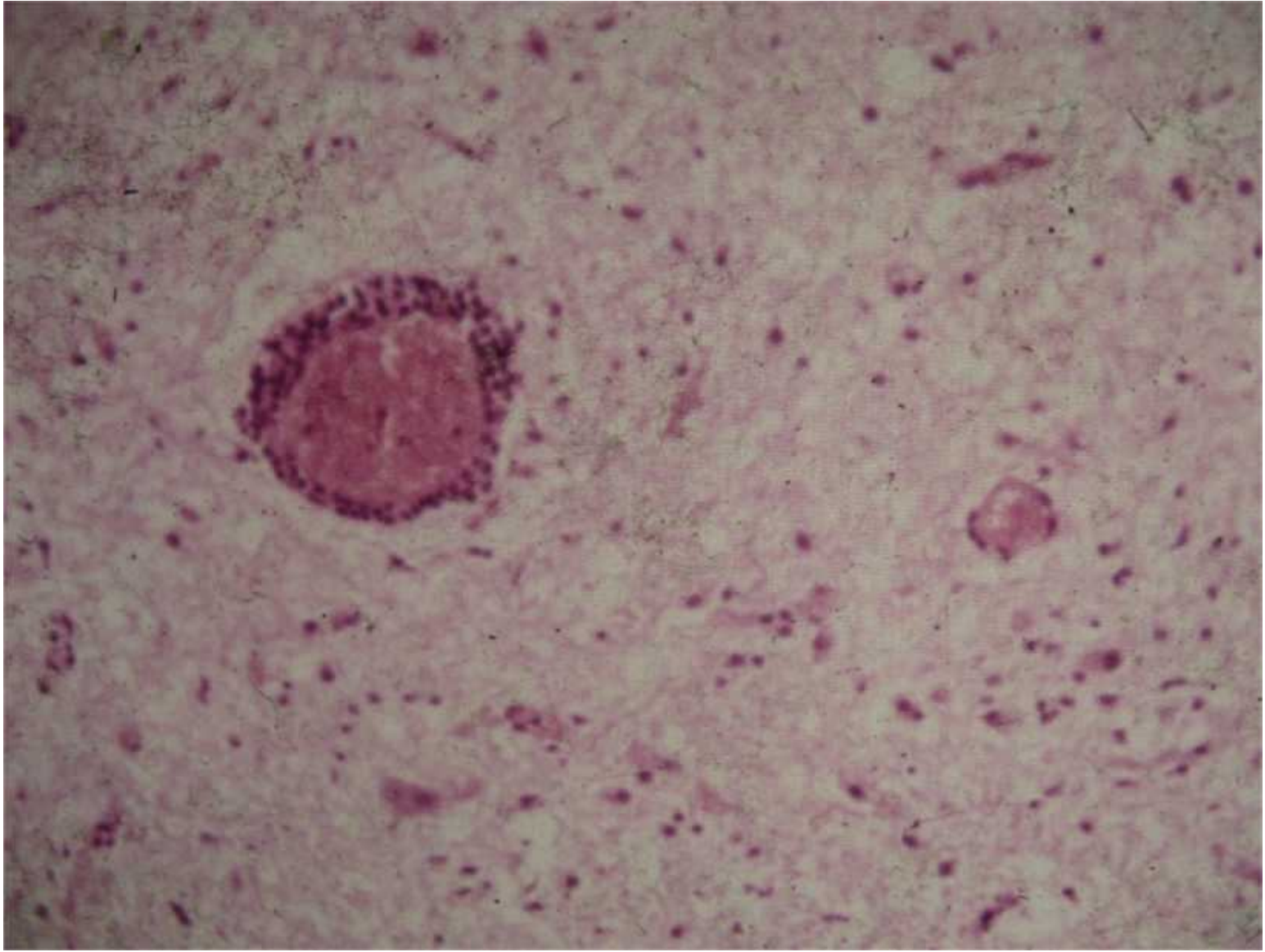


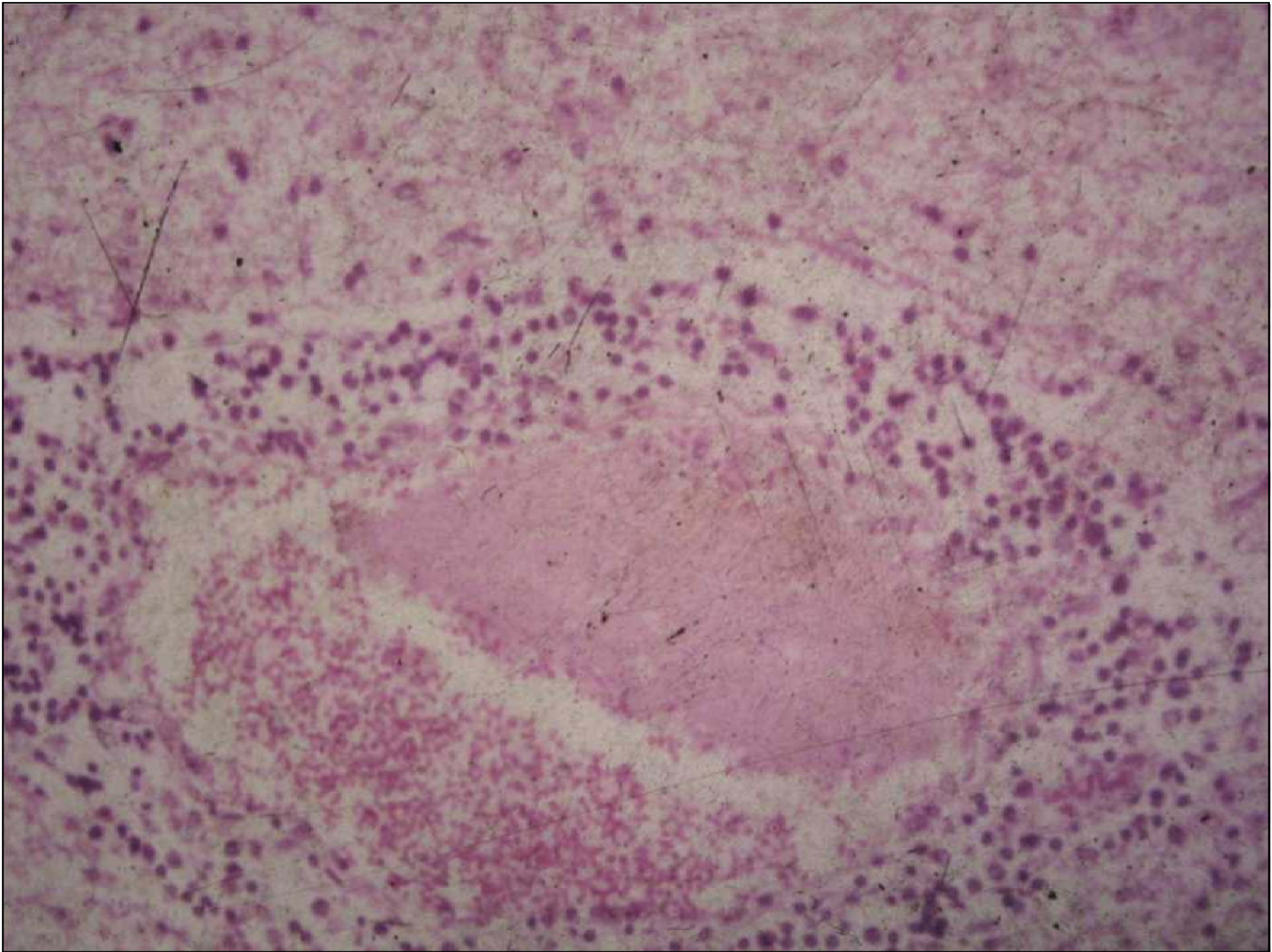




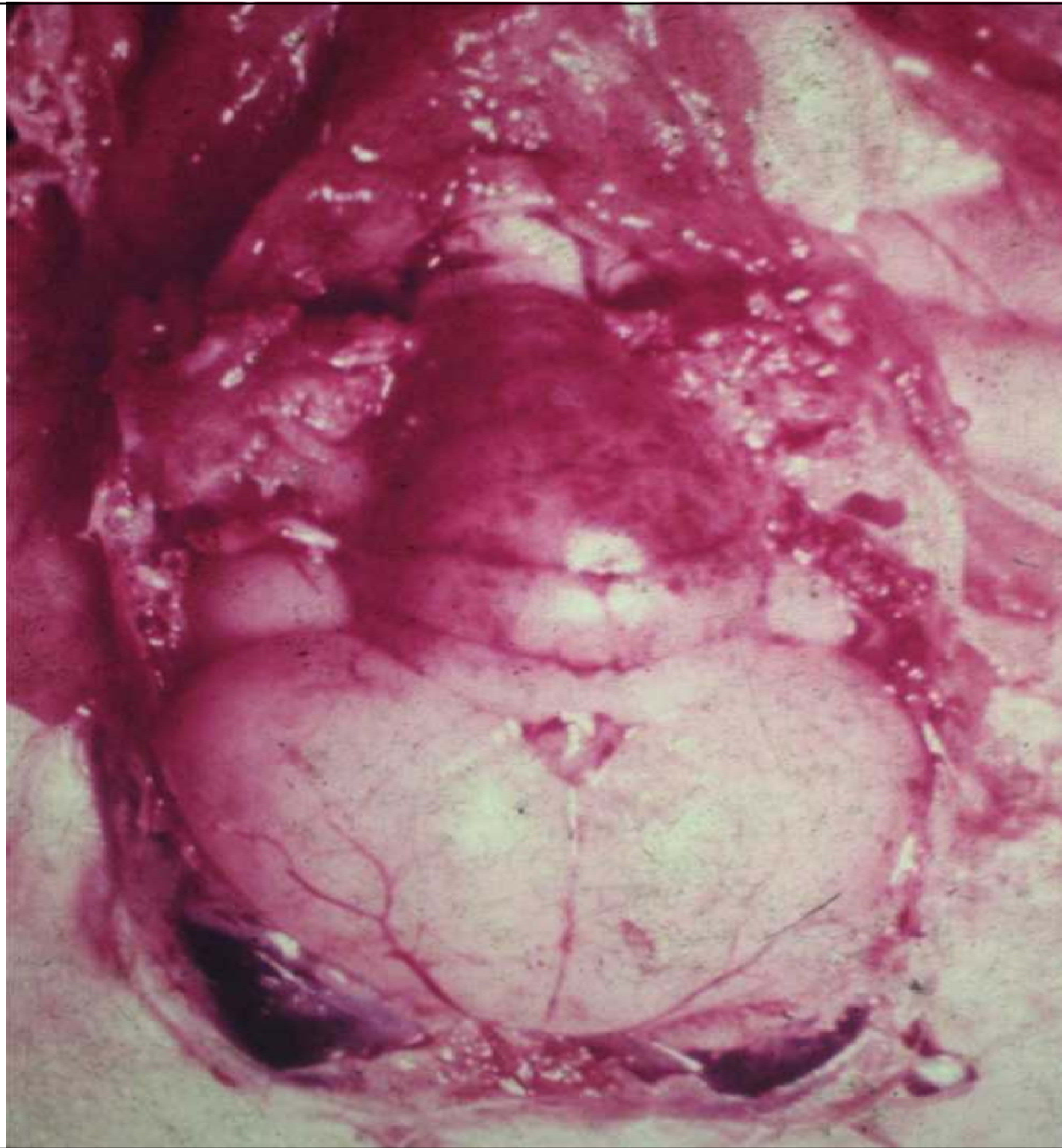












Babes nodules

