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A Comparative Study of Degenerative Tissue

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A COMPARATIVE STUDY

of

DEGENERATIVE TISSUE.

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Written by Homer Davis of Marion, S.D. in completion
of a post graduate course at the South Dakota Agri-
cultural College, 1897.

Written under the direction of

Dr. Dice McLaren,

Dept. of Biology.

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"COMPARATIVE STUDY OF DEGENERATED TISSUE"

Thesis, written by Homer Davis
for Master's degree, S.D.A.C.
1897.

Nutrition is the earliest and most constant of vital operations. So prominent is the nutritive apparatus of an animal that the animal has been likened to a moving sac, organized to convert foreign matter into its own likeness, to which the complex organs of animal life are but auxiliaries.

In growth more material is used by the body-protoplasm than is expelled. Nutrition includes excretion i.e. the removal of spent material which is usually poisonous and which if allowed to remain in the body organism would impair nutrition in its fullest sense.

The objects of nutrition are growth, repair, and propagation. The amount of matter expelled from the body must be equalled by the amount of nourishment taken into the body in order to make these objects possible, and if from any cause the nutrition is impaired, the animal tissue suffers. This impairment of nutrition may be the result of general anaemia of the body or it may be the result of an injury to the nutritive nerves; or directly an injury to the blood vessels which bring the necessary supply of blood to the part undergoing a change from its normal structure.

Disordered ^{nutrition} so far as it concerns new formations is still little known chemically; so much the more has attention been turned to histological changes. If the nutrition is incomplete, the normal form and function and form are more or less lost, and thus we have atrophy, infiltration, and degeneration.

In atrophy the tissues merely decrease in size. In the so called "fatty infiltration" the latest authorities on pathology hold that there is no real fatty infiltration, but that all fat is incorporated in making protoplasm and that all fat is a product of the protoplasm of the body

which stores it. When the particles of fat in cell degeneration become numerous they coalesce, and if the cellular structure becomes destroyed what is known as "fatty infiltration" results. The theory that calcium is an infiltration is also very well disproven by later authorities on pathology. The calcium compounds in the body are believed to have been derived by the action of a protoplasm cell. The cells select, combine, build, and tear down. A foreign substance as coal dust, etc. finds lodgment in lymph spaces and nodules. This may be correctly considered as infiltration.

In degeneration proper of the tissues, the tissues are transformed into other shapes and material. According to Hamilton "A degeneration is any process whereby a cell element or tissue undergoes such molecular changes that it can no longer maintain its functional activity, and either separates into its organic constituents or gives rise to the formation of a new product at the expense of its own substance."

Of the causes of degeneration but little can be said in a general way, the various forms depending for the most part upon different conditions. All may be said to be caused in some way by some error in nutrition. This error may be brought about in different ways.

The principal degenerations are the fatty and amyloid. Other degenerations noticed by leading authors are cloudy-swelling, mucoid, colloid, hyaline, pigmentary, and caseous. These may be independent or may be associated.

Each form will be taken up and discussed by itself.

--F A T T Y D E G E N E R A T I O N--

Fatty degeneration is a chemical change in a cell or fibre by which it becomes destroyed from the conversion of its albuminous or protied constituents into oil. Fat occurs in the normal body in considerable amount. Certain tissues of the fibrous type, like subcutaneous

and subserous structures; the marrow, etc. are always rich in fat. The fat is simply stored up in these tissues. When this process is carried on within limits, it must be considered a physiological action. When the process is carried to such an extent as to be considered pathological, it is called lipomatosis. The subject of "Fatty Infiltration" has already been sufficiently considered. True fatty degeneration must, of course, be distinguished from the so called "infiltration". The two conditions are nearly always associated.

The fat which occurs in fatty degeneration is not fat in store, but fat resulting from the disintegration of albumen in the effected cells. Fat is normally formed from the albumen of the cells, but is consumed as it is formed. This is fatty degeneration we are driven to suppose that either the disintegration of albumen is increased or that the consumption of the fat produced is impeded. Both these things may occur, but it especially to be noticed that in fatty degeneration the lost albumen is not replaced so that the reproduction of fat is associated with atrophy.

A cell undergoing fatty degeneration always shows larger or smaller oil drops in its interior. These oil drops appear under the microscope as minute black dots. Their number and size in the interior of a cell vary greatly. Take for example fatty degeneration of the muscles of the heart, and here we find many or fewer oil-drops according to the degree of degeneration. The drops however are all small and do not coalesce into larger drops.

Fatty degeneration occurs especially in muscular tissue, in epithelial cells, and in connective tissue; but it may occur in most any tissue in the body. If it effects entire cell groups or systems, it may be recognized even by the unaided eye; and the more readily as the degeneration is more advanced, the proper color of the tissue less marked, and the amount of blood present less considerable.

Colorless transparent structures like the endocardium and intima of the large blood vessels, take on a white opaque appearance. The cortical tissue of the kidney becomes grayish white or , when the fatty change is greater, it becomes opaque yellowish white; the heart muscle becomes yellowish and voluntary muscular tissue pale yellowish brown.

Fatty degeneration of some of the tissues may result in complete disintegration of the tissues, and if the mass lose water and become condensed, the fatty change passes into caseation and the tissue assumes a dull white cheesy appearance. Pus cells and those of coagulated exudations very often undergo true fatty degeneration ending in their complete destruction as cells.

Of the causes of fatty degeneration, malnutrition is the most common. It may be local or general. Tadpoles kept in distilled water without any nutrition are found to have fatty degeneration of some of their tissues. The direct cause seems to be in an alteration of the constituents of the blood, that is of the nutriment supplied to the cells. This may be due to a deficient supply of oxygen the blood becoming impoverished to such an extent that the oxygen bearing part is deficient.

The chemical cause of the change of the albumen into fat is not known, but it may be advanced as a theory that the oxygen of the albumen in the cell is taken up by the blood to supply what deficiency it can and that a material of a cheaper quality, as the oil drops which contain no oxygen to speak of, is left in the place of the albumen.

Fatty degeneration is found to occur in the most widely different organs when associated with general anaemia. The same thing comes to pass in particular organs which happen to receive too little blood either in consequence of disease in different vessels, or because the outflow of blood from them is checked and its renewal hindered. Organs like the muscles which are left unused for any reason, and so fail to

undergo an adequate amount of tissue change are very apt to become fatty,

Various poisons such as arsenic, phosphorus, and the ferments or ptomaines which produce fevers may lead to the disintegration of the albumen of the tissues and so to fatty degeneration.

-- A M Y L O I D D E G E N E R A T I O N .--

This form of degeneration seems to be rather peculiar in its characteristics. Hamilton claims that it cannot be classed under the head of degenerations and that it is simply an infiltration. Ziegler, Coats and others claim that it is a true degeneration and that the substance known as "amyloid substance" is formed directly from the cell contents of fibrous tissue which it is most likely to attack first. It is also known to pathologists as waxy or lardaceous degeneration. The small arteries are almost without exception the structures in which the new substance first appears. The muscular coat becomes thickened, translucent, and homogeneous; the fiber disappears from them, and the caliber is encroached upon. The larger arteries are usually unaffected unless the degenerative changes have undergone an advanced form. Soon the arterial capillaries undergo a similar change.

The liver, spleen, and kidney are the three organs most commonly effected. In the liver there is no doubt of the degenerative process extending beyond the fibrous tissue into the hepatic cell substance. In the kidney it ^{is} confined almost exclusively to the fibrous ^{tissue} of the framework of the organ and the blood vessels. In the spleen it does not confine itself to the vessels and connective tissue.

It is found also in the stomach, intestine, and heart and rarely in the oesophagus, muscles of the tongue, lymphatic glands, lung tissue, skin, and very rarely in the supra-renal capsules.

The amyloid structures are greatly increased in weight and bulk

and thus the whole of the effected organ is greatly increased in size. The liver, spleen, and kidney are often greatly enlarged and present a waxy or lardaceous appearance which has given rise to the names waxy and lardaceous disease, often applied to amyloid degeneration.

In amyloid degeneration the tissues are permanently involved, for the amyloid substance being but slightly soluble does not disappear when once deposited. In appearance it resembles starch, but upon chemical analysis it is found to be almost identical with albumen.

In regard to the cause of amyloid degeneration it may be said that it is due to some alteration in the blood, probably an impoverishment of it in albumen. It is not an independent disease but comes on in certain cachetic states due to chronic tuberculosis, syphilis, diseases of the bone involving prolonged suppuration, chronic dysentery, chronic albuminaria, etc. Authors generally agree that it is due to an alteration in the condition of the blood, and that the albumen received enters into combination with the protoplasm in such a way as to produce this peculiar amyloid substance.

--)(C L O U D Y S W E L L I N G)(--

This is so named because of the appearance of the tissues when in this condition. The cells become enlarged and thus enlarge the tissues giving them at the same time a dark cloudy appearance. It generally effects the secreting and excreting epithelia and also the muscular fibers of organs that are in a state called parenchymatous inflammation. It especially effects the kidneys, but may effect the heart and liver where it is found quite often. When the cell or fiber takes on this form of degeneration it swells and becomes granular. Its outline is indistinct and its substance dusky. These granules are very small so that the cell presents a fine dotted appearance. The granules hide

the nucleus of the cell , and if it be muscular tissue that is effected in this way, the striae disappear. These granules are said to be precipitated albumen because they give a chemical reaction like that of albumen. The tissues when effected with cloudy swelling contain an abnormally small quantity of blood. However , before the cloudy swelling appears there is generally a congestion of the organs effected. Take for example cloudy swelling following an attack of acute parenchymatous nephritis. This is generally caused by exposure of some kind: being out in a cold rain, lying on the cold damp ground, etc. The outward parts of the body are deprived of their usual supply of blood and consequently the internal organs are highly congested. In this way the circulation of the kidney is considerably impeded and hence the function seriously interfered with, the organ itself becoming swollen^{ed} red, and soft. The cells lining the tubules having the very important function of excreting the urea are not sufficiently nourished to maintain their normal condition and so they become swollen and granular. This is called cloudy swelling. These cells are exfoliated and replaced by new cells which are in turn effected in the same way and consequently thrown off/. The process may penetrate the tissue of the organ, it may assume a chronic form, or it may take on a variety of different attitudes.

Besides effecting the kidneys, liver, and heart, it some times effects voluntary muscular tissue and other tissues. It is especially liable to follow an attack of scarlet fever, in this case nearly always effecting the epithelium of the tubules of the kidneys. It may also follow an attack of typhoid fever, smallpox, erysipelas, diphtheria, septicaemia, etc/ effecting the kidneys, liver, or heart. It is said by a few writers to be caused by poisoning by carbonic, phosphoric, and arsenic acids.

Cloudy swelling may and is liable to undergo fatty degeneration of its albuminous granules, thus making it more complex and unfavorable in its prognosis.

--MUCOID AND COLLOID DEGENERATION--

These two forms of degeneration present a close resemblance in general appearances. They are often associated so that it is difficult to tell the one from the other. They are both formed from the healthy tissue albumen and are albuminates. That found in mucoid degeneration has a definite chemical principle called mucin. The one found in colloid degeneration has no well known chemical relations and the substance is simply called colloid material. They can thus be told and separated from each other by chemical reagents. The mucoid substance has more of a trembling jelly-like aspect than the colloid. Both substances are found in the foetus at an early period of life and are in all of the subcutaneous areolar tissues. The vitreous humor of the eye is of a mucoid nature.

Mucoid degeneration is mainly a degeneration of connective tissue tumors and may effect any of this group, such as fibrous, sarcomatous, cartilaginous, or fatty. It is said to be commonest in the sarcomata and generally is found in those of a subcutaneous origin. It is a theory that the cells in this form of degeneration may undergo the same changes as do the goblet cells of the mucous membranes in their secretions, only the one measure is a physiological action and the other pathological. It may effect any tissue where connective tissue predominates. The ground work becomes effected first and then the whole mass becomes homogeneous. If examined with the microscope it appears perfectly transparent and homogeneous.

Colloid degeneration is especially liable to effect epithelia lining ducts or vesicles whose outlet are naturally closed or have been closed by disease. It is always to be found in the thyroid gland and so sometimes gives rise to the trouble known as goitre, although it is not always the cause. All cancerous tumors are subject to it, especially those of the gastro-intestinal tract. Colloid degeneration may occur in the tubes of the kidneys in various diseases. When examined microscopically it is seen to be a homogeneous mass presenting perhaps a slight stringy appearance thus differing from mucoid degeneration.

The cause of these two forms of degeneration is not exactly understood, but is supposed to be due to some form of altered nutrition.

--PIGMENTARY DEGENERATION--

Concerning this form of degeneration but little need be said. It is not a very common form of degeneration and not very important, effecting chiefly connective tissue growths, such as sarcomata and fibrous tumors, and only these when they are located in parts or near parts naturally pigmented.

It is said that in intermittent fever the spleen often becomes effected with a pigmentary substance derived from haemoglobin and the disease is called melanosis. These pigment particles may be carried to the other parts of the body and there lodged.

--HYALINE DEGENERATION--

Ziegler gives this form of degeneration and says that it resembles very closely amyloid degeneration in every way except that it is found in the adventitious tissue of the arteries.

--C A S E A T I O N--

Hamilton gives this form of degeneration and gives as the definition of it, "A dry fatty degeneration in which the albuminous and oily constituents of the tissue become converted into a substance like cheese in appearance and somewhat allied to it in chemical composition."

The tissue fallen into this state is of a yellow color, has no blood vessels, and examined under the microscope presents a fine granular appearance. A caseated nodule or part is hard, well defined, dry, and compressed. It is dead tissue and its final course is to break down into granular matter and very fine oil globules. This form of degeneration is due chiefly to the presence of microorganisms or to a chemical change. A nodule may have a calcareous substance deposited about it and thus become permanently incysted. The most common form of this degeneration is the tubercle bacillus. The gummata caused by the syphilitic bacillus sometimes undergoes a cheesy degeneration.

PREPARATION OF SPECIMENS.

A few words in general as to the preparation of the specimens presented in and with this thesis will probably be in order.

Some of my specimens were already cut for the slides when I procured them. In fact it would almost have been impossible to properly prepare several of them without the use of the microtome. Some of them were taken fresh and were hardened in a one per-cent alcoholic solution of corrosive sublimate, twenty parts of solution to one of tissue. After they had been left in this solution for from 24 to 72 hours, they were transferred to commercial alcohol until needed for use. Some of these specimens were too soft and spongy to obtain suitable specimens by ordinary cutting, so they were imbedded in parafin in the following manner: Upon taking them from the commercial alcohol solution they were cut in long square blocks of the required size for suitable specimens. They were then transferred to benzol until all air bubbles had been expelled when they were put in the imbedding parafin. A mold of ~~parafin~~ writing paper was used and the parafin heated simply to the melting point. They were quickly cooled and were ready for the knife.

Various staining preparations were used. Some stains bringing out features in certain tissues that other stains would not, but as it is not under the scope of this thesis to enter into a discussion of the virtues of the different stains, I will simply mention the various stains used. They were Hematoxylin (DeLafield's), Borax-carmin, Gentian-violet, Eosine, Fuchsin, Methyl-blue, and R.&J. Beck's double stain. Some of these stains were used but a little.

A very nice method used by myself after some experience with the common way was to fix the specimen directly on the slide after it was cut, by the use of the following carbolized solution of egg albumen:- Beat up the white of one fresh egg in eight ounces of a two per-cent aqueous solution of carbolic acid; then filter until a clear, pearly

white solution results. A small bottle should be filled for convenient use at the work-table, and the remainder bottled, well stoppered, and put away for future use.

With a camel's hair pencil place a drop of the fixing mixture on the center of the slide and place the specimen on this drop of fluid. A piece of heavy white filter paper is placed on the section and over this a piece of oiled smooth card-board is pressed to squeeze out the excess of fluid and smoothly fix the specimen to the slide. After the removal of the papers the slide is wiped and exposed to the air for say five or ten minutes to firmly fix the specimen to the slide. If the tissues of the specimen are filled with parafin, the slide is placed in spirits of turpentine for from one to ten hours to dissolve the parafin. After removal from the turpentine the specimen is thoroughly washed in benzol and wiped close to the section. Then the slide is put into commercial alcohol No.1 and within the next five or ten minutes transferred to alcohol No.2, where it may remain until needed for the staining process. Several specimens may be carried through this process together. In the use of some of the weaker stains, the specimen may be put in water just before it is placed in the stain. It will then more readily take up the stain. However this is not always advisable.

{ It is most convenient to have all of these various liquids in bottles
{ having large enough mouths to admit the slide with out trouble.

The stain may either be applied with a small dropper or the slide may be put in the wide mouthed bottle containing the stain. As to the time the specimen should be exposed to the stain, there is no rule to be governed by except practice and common sense. The time varies with the nature of the tissue and the stain from a few seconds to one or several minutes. This all has to be learned by experience. After staining, the specimen is to be decolorized until the stain in the main body of the tissue does not interfere with the examination of the specimen. If

a weak stain has been used, the specimen is decolorized with commercial alcohol, then put in absolute alcohol for a few moments, and then cleared up with benzol^{oil of cloves} or xylol -I prefer the latter, and mounted in Canada Balsam. If a strong stain has been used, as borax-carmin, it is better and quicker decolorized with an acid solution of 70% alcohol with water made as follows:-To each ounce of 70% alcohol add from five drops to one-half dram of C.P. HCl . If the acid solution is used in decolorizing the specimen must be thoroughly washed in distilled water to get rid of the acid and then put into commercial alcohol for a few moments and from this into absolute alcohol and cleared up as before mentioned and mounted.

The acid decolorizing solution gave good satisfaction with the strong stains, but cannot be used with satisfaction with the weak stains

The same can be said with reference to decolorizing with either distilled water, commercial alcohol, or the acidulated solution of alcohol as was said about the use of the stain viz: There is no rule to be governed by except practice and common sense. The time varies with the stain and the nature of the tissue from a few seconds to one or several minutes.

If the method of fixing the specimen directly to the slide is not used, the specimen may be lifted from one fluid to another during the process of staining with a lifter made of filter-paper. When the specimen is ready to be placed on the slide, -at the time of using the absolute alcohol or the clearing fluid, one end of the slide may be placed in the fluid and the specimen put on the slide and straightened while in the fluid. The specimen is then slid along the slide to its place in the center.

--IV.--

Staining in Borax-carmine

- (1) Stain from five to fifteen minutes.
- (2) Wash for a half to one minute in an alcoholic HCl acid solution.
- (3) Completely remove the acid by washing in water.
- (4) Alcohol, oil or xylol, Canada balsam.

Staining in Gentian Violet.

- (1) Stain for three to five minutes.
- (2) Wash in alcohol until the sections acquire a light blue color.
- (3) Absolute alcohol, xylol, Canada balsam.

Staining in Haematoxylin.

- (1) Stain for one to three minutes.
- (2) Wash in plenty of distilled water.
- (3) Leave sections in distilled water for from twelve to twenty-four (hours.
- (4) Alcohol, absolute alcohol, oil or xylol, Canada balsam.

Staining in Eosine.

- (1) Stain for a few minutes until the sections become pink.
- (2) Wash in water.
- (3) Alcohol, alcohol, xylol, Canada balsam.

Staining in Fuchsine.

- (1) Stain for a few minutes.
- (2) Wash in water.
- (3) Alcohol, alcohol, xylol, Canada balsam.

Staining in Methyl-blue.

- (1) Stain for two to ten minutes.
- (2) Wash in water.
- (3) Alcohol, alcohol, xylol, Canada balsam.

Double Staining in Haematoxylin and Eosine.

- (1) Stain in haematoxylin for from one to three minutes.
- (2) Wash in plenty of distilled water.
- (3) Leave sections in distilled water for from twelve to twenty-four
(hours.
- (4) Alcohol.
- (5) Place section in eosine solution of alcohol for one to five minutes
made as follows:- A few drops of alcoholic solution of eosine is
added to one dram of absolute alcohol.
- (6) Wash in absolute alcohol.
- (7) Oil of cloves or xylol, Canada balsam.

Fatty Degeneration.

Fatty degeneration is bestv studied in fresh tissues, either by
teasing or fresh tissues.

Tests.

- (1) The fat does not disappear when treated in acetic acid.
- (2) It also resists the action of dilute caustic potash and
(soda.
- (3) It is dissolved by chloroform or ether.

Alcohol used too strong dissolves the fat gradually. For keeping
any length of time these specimens should be hardened in Muller's
solution and osmic acid. I have used alcohol with good results as a
hardening fluid, being careful to not have it too strong (about 70 %).

Amyloid Degeneration.

The tissues containing this form of degeneration may be hardened
in either alcohol or Muller's solution.

Test for amyloid substance as follows: The section is placed in
sherry-colored iodine solution made by diluting (iodine 1 part,
potassium iodide 2 parts, water 100 parts) with water until it assumes

the sherry color. The section is stained in this for three minutes, washed in distilled water, and mounted in glycerine. The parts that have undergone an amyloid change acquire a dark brown color, while the rest of the tissue becomes yellow.

For permanent mounting double stain with haematoxylin and eosine, or gentian violet and eosine. Other special stains are used for this degeneration but I did not try them.

Pigment.

Pigmentary substances may be found in tissue without the help of stain, but may be better studied by staining with haematoxylin.

Cloudy Swelling.

To distinguish cloudy swelling from fatty changes, acetic acid is added. The granules due to cloudy swelling vanish, while, on the other hand, they are unaffected by fat solvents such as absolute alcohol, ether, Chloroform, etc.

Mucoid Degeneration.

Mucoid degeneration is said to stain well in tissues with all analine basic dyes, but particularly well with methylene-blue.

Colloid Degeneration.

The colloid substance in tissues is not injured by hardening in alcohol or Muller's fluid. The haematoxylin and eosine double stain gives good results.

Hyaline Degeneration.

This ~~Change~~ is best shown by the double stain of haematoxylin and eosine. In sections which have been only singly stained, however, the homogeneous, glassy condition of tissues which have undergone hyaline degeneration is generally sufficiently visible.

Calcareous Degeneration.

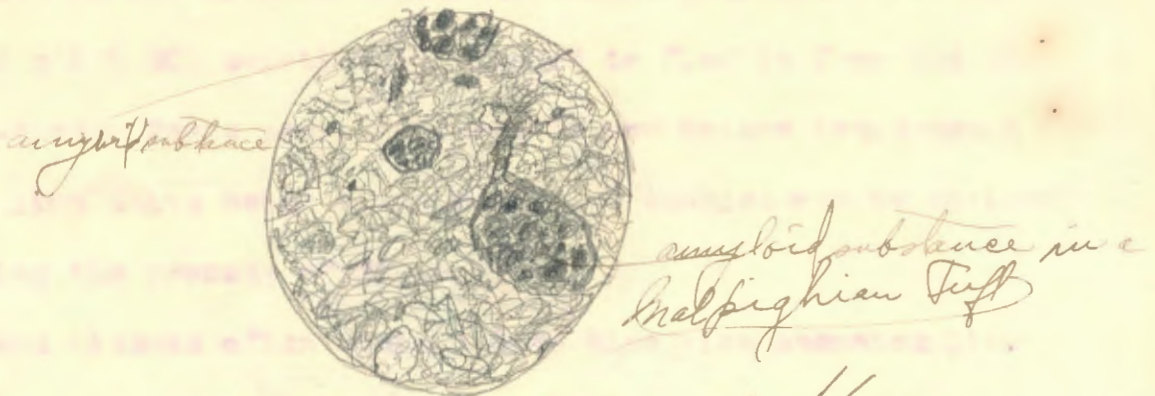
Lime salts in the tissues appear as bright granules which readily dissolve when a 5 % HCl solution is allowed to flow in from the edge of the cover-glass. Parts previously opaque now become transparent owing to the lime salts being dissolved. Gas bubbles may be noticed escaping during the process of dissolution.

Calcareous tissues often stain a deep blue with haematoxylin.

Pigmentary Degeneration.

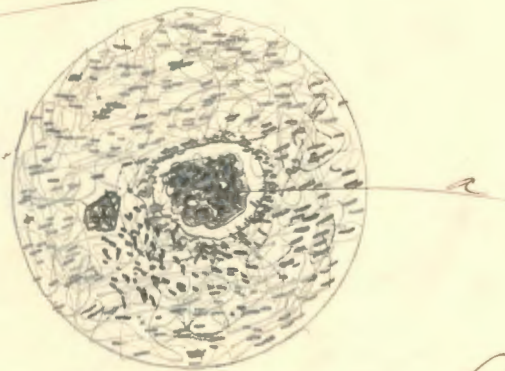
Several methods are outlined for testing and examining pigmentary deposits and degenerations, but they are too complex for simple laboratory practice and as I did not put them to practice, I will not enter into a discussion of them here.

Plate I

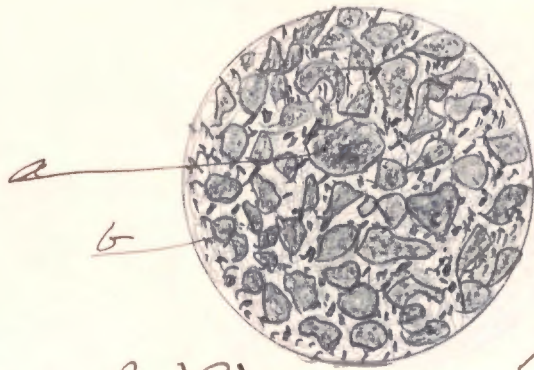


Amyloid degeneration of Kidney showing amyloid substance in Malpighian tuft and probably the small artery leading to it.

Plate II



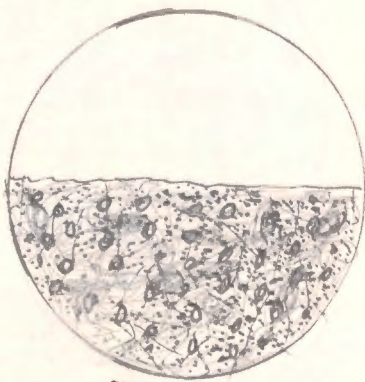
Amyloid degeneration of Kidney showing amyloid substance in Malpighian tuft - a and stained with a nuclear stain (hematoxylin) to show that the cells are not all obliterated.



X 400

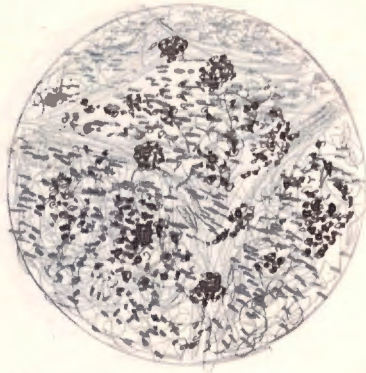
Gumma of Liver, Central part and surrounding zone.

a = central zone showing fatty degeneration.
 b = zone of cells in fibrous matrix.



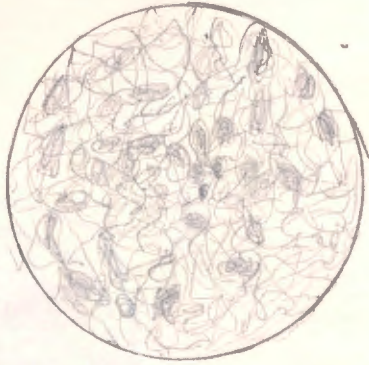
X 500,

Fatty Degeneration of Liver,
 caused by Phosphorus poisoning
 Shows cells of liver substance (intralobular)
 permeated with minute oil globules.



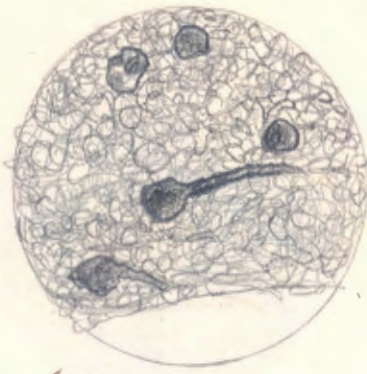
x 400

Amyloid degeneration of spleen (diffuse)
stained with methyl blue.

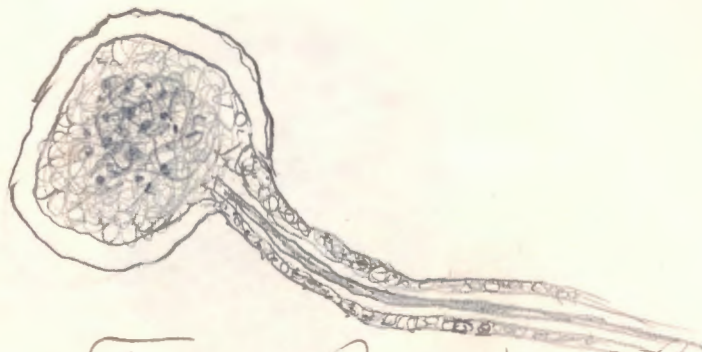


x 400

Amyloid degeneration of liver

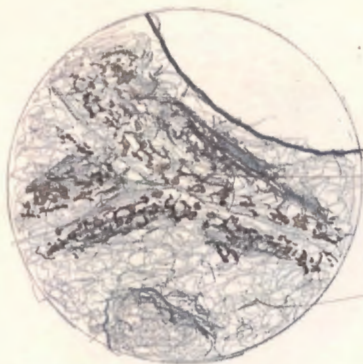


Amyloid Kidney x 100
 Showing degeneration of Malpighian Tuft,
 and thickeningⁱⁿ of the walls of the small
 artery.



X 400

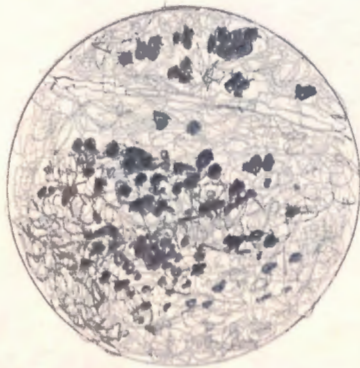
Malpighian Tuft of Amyloid Kidney showing
 the thickening of the cells of the circular muscular
 coat of the small artery also the
 thickening of the cells of the longitudinal muscular
 coat and the enlargement of the cells of the
 intima.



a low power

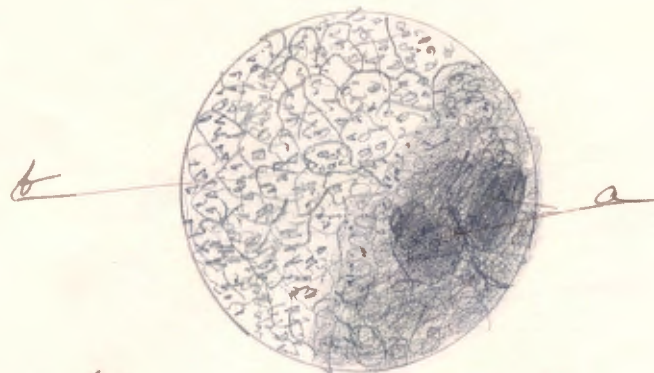
Amyloid spleen (diffuse)

a = normal splenic tissue
b = amyloid substance

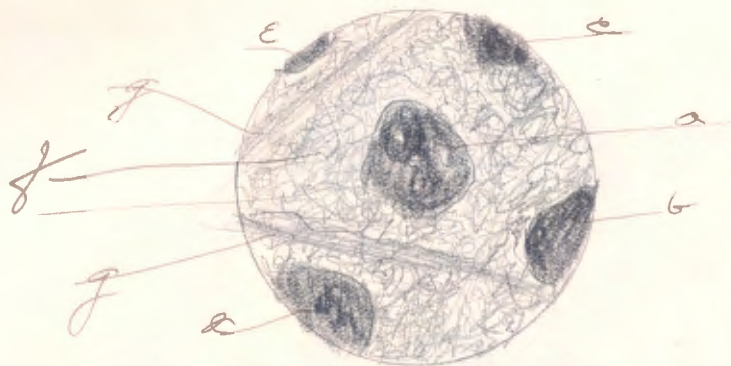


Amyloid spleen (diffuse) x 300

showing normal splenic tissue
with amyloid substance.

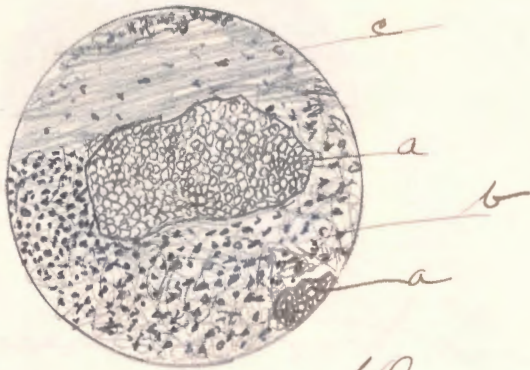


Amyloid Spleen (Sago) x 300
a = part of an altered Malpighian corpuscle.
b = normal spleen tissue

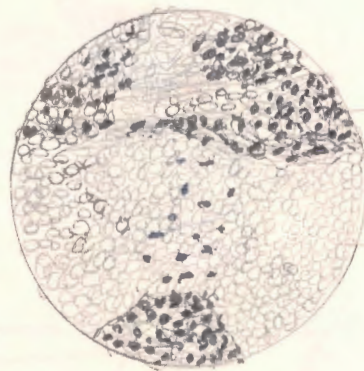


Amyloid Spleen (Sago) x 100

a, b, c, d, e = Altered Malpighian corpuscles,
 f = normal spleen tissue
 g = fibrous tissue or trabeculae



Amyloid Degeneration of Spleen (Sago) x 400
 Amyloid substance = a
 Normal splenic tissue = b (showing nuclei)
 Fibrous tissue = c

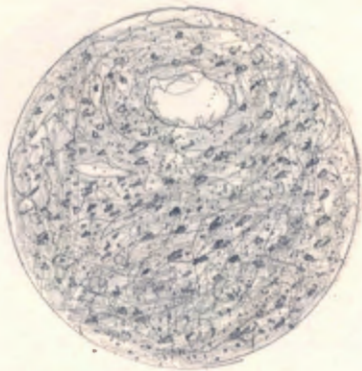


splenic tissue

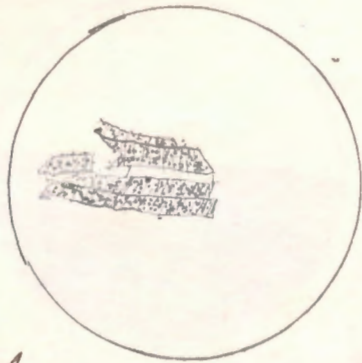
amyloid substance

Amyloid Deg. of Spleen (Sago) x 400

This specimen seems to be mixed somewhat with diffuse amyloid degeneration as the above plate shows.



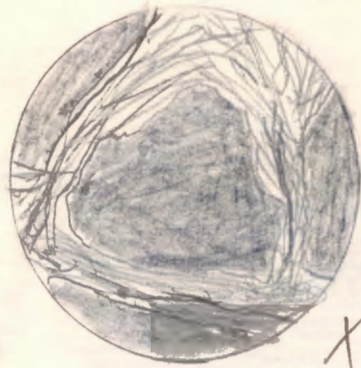
Fatty Degeneration of Liver



X400

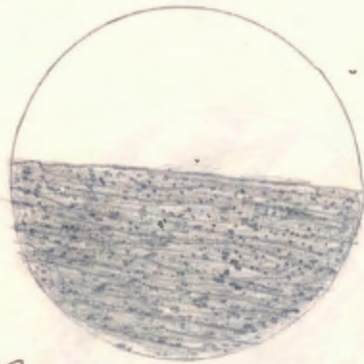
Fatty Heart

Showing a few cells of
the muscular tissue, containing the
oil globules,



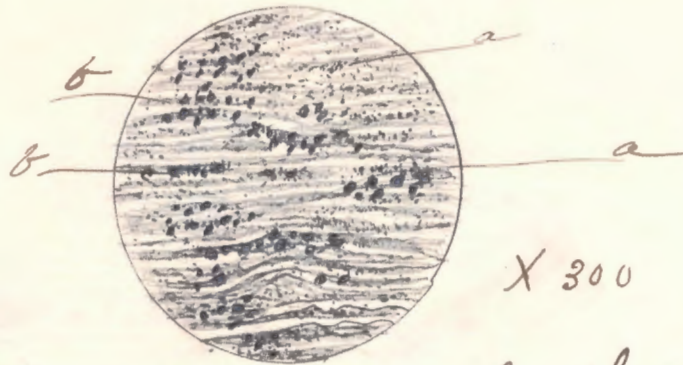
X 100

Gumma of Liver as seen by low power
showing two of the three zones
Fat cells in intercalary zone can hardly be seen

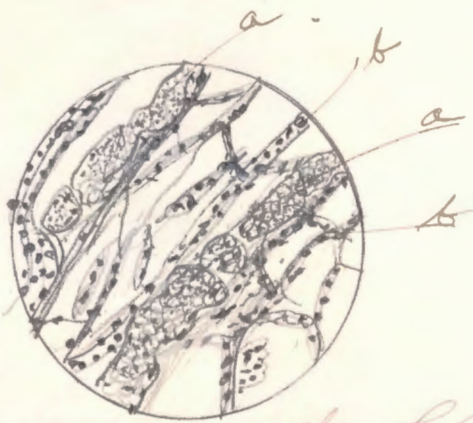


X 400

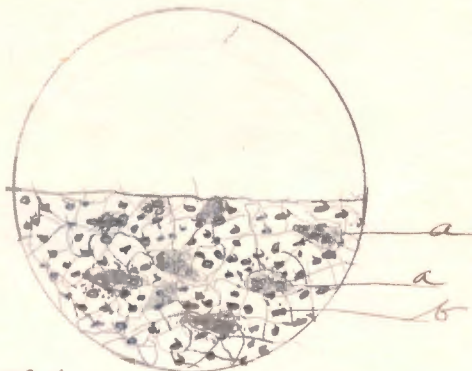
Gumma of Liver, intercalary zone
showing fatty degeneration.



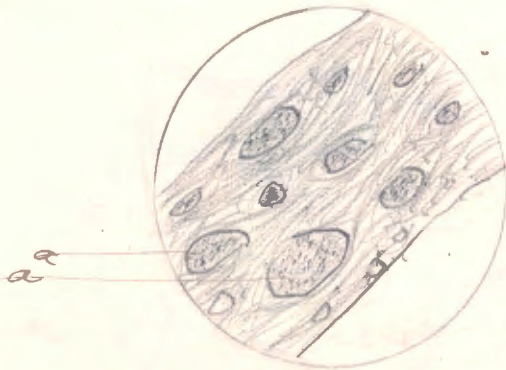
Fatty Degeneration of the Kidney
 Epithelium of the ~~cortex~~ medullary part
 a, a oil globules.
 b, b. nuclei



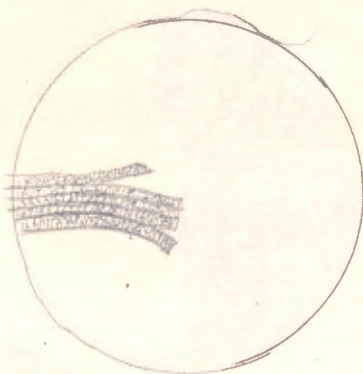
Fatty Degeneration of the Kidney
 a, a - collection of cells containing fatty degeneration.
 b, b, fibrous part, containing no fat.
Cortex of Kidney



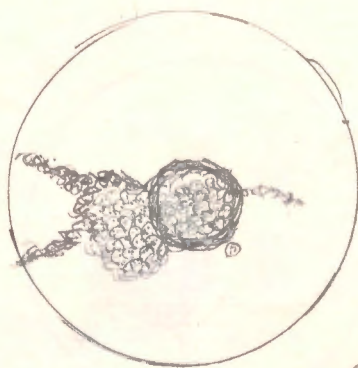
Fatty Infiltration Liver
 aa collections of fat globules, each of these globules showing adjacent cells with fatty degeneration
 bb apparently healthy liver tissue.



Tuberculosis of Epididymus showing tubercles with cells showing fatty degeneration
 a, a tubercles.



Fatty Degeneration Heart

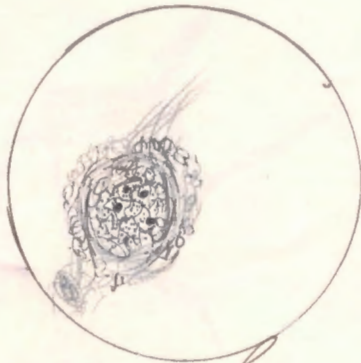


Ischemia of Lung Tissue
showing fatty degeneration of cells



X 400

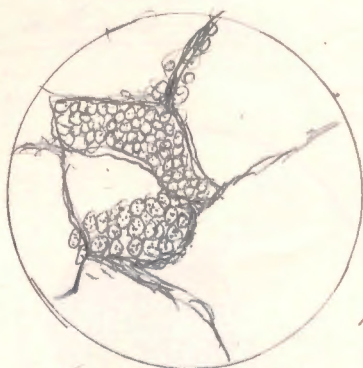
Tubercle of Liver showing
fatty degeneration



Tuberculosis of Knee Joint
showing tubercle with fatty degeneration
of giant cells.

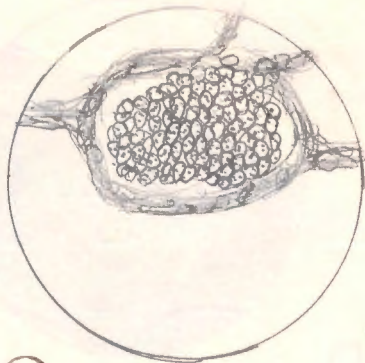


Tuberculosis Granular showing
Fatty degeneration of cells in
tubercles.



x 300

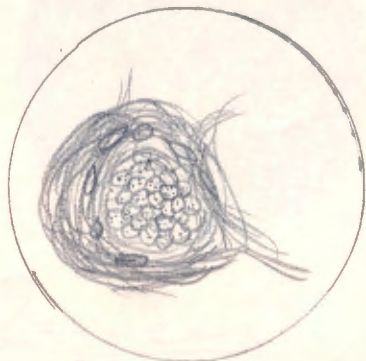
Catarrhal Pneumonia showing
a large accumulation of
cellular elements within one
of the pulmonary alveoli which
have undergone extensive
fatty degeneration.



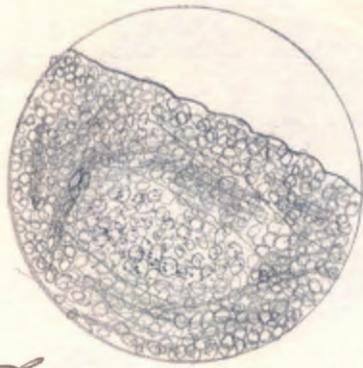
x. 400

Croupous Pneumonia

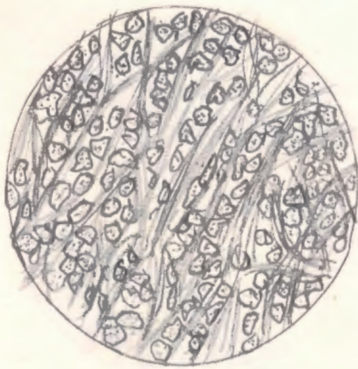
Showing the accumulation of cellular elements within one of the pulmonary alveoli in stage of gray hepatization, which have undergone extensive fatty degeneration.



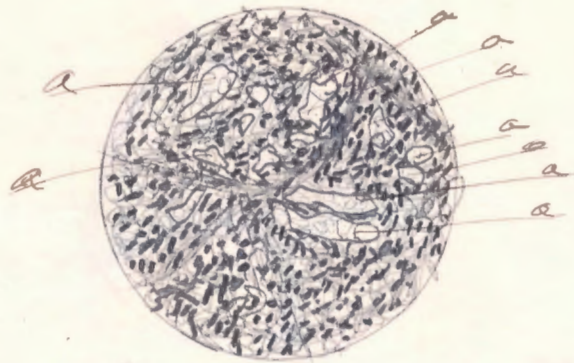
Epithelioma of Lower Lip showing cells that have undergone fatty degeneration.



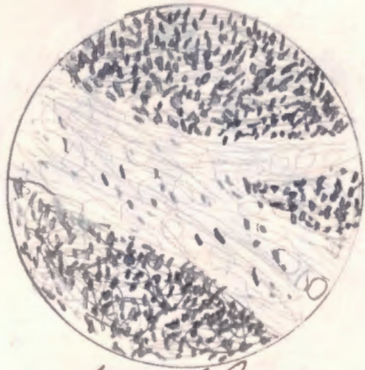
Chronic Pyaemia of Kidney,
showing a zone of cells that have
undergone fatty degeneration.



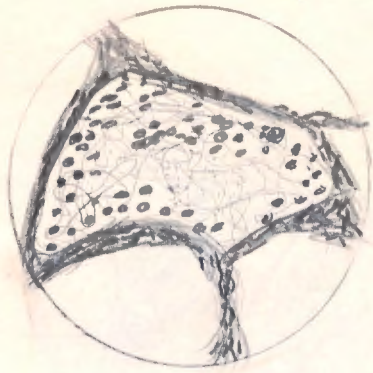
Scirrhus Cancer, showing
fatty degeneration of cells of alveoli



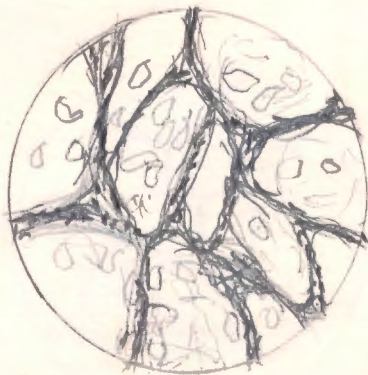
Carcinoma of Lung showing
Colloid material,
a colloid cells and groups,



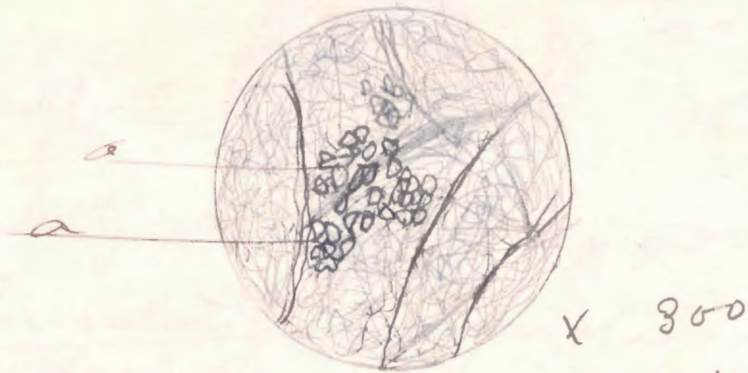
Carcinoma of Stomach
Showing Colloid Regeneration



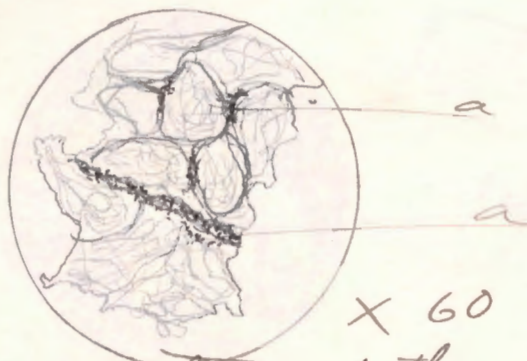
Colloid Regeneration of the Thyroid.
Body showing a vesicle filled
with colloid material.



Colloid Carcinoma Ovary
showing vesicles within which is
contained the colloid material.



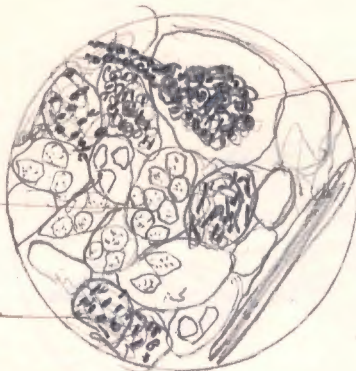
Hyaline degeneration of the Thyroid Body
aa-hyaline substance



Hyaline degeneration of the Thyroid Body
aa hyaline substance

degenerated cells
or almost

apparently healthy ones

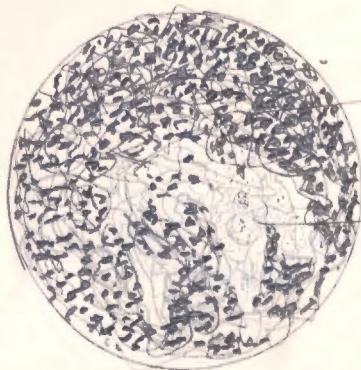


glomerulus with
small artery

x 400

Pyonephritic Degeneration of Kidney
Cloudy Swelling

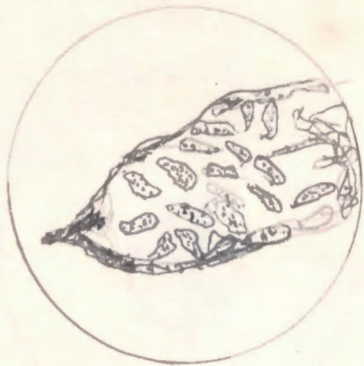
Some cells having almost passed into the
fatty degenerative stage.



healthy tissue

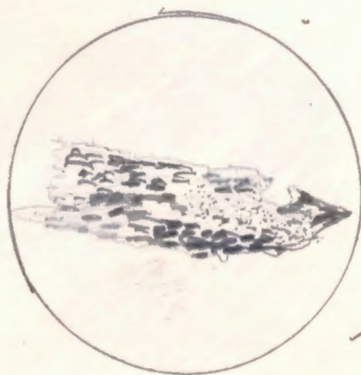
degenerated cells

Pyonephritic Degeneration of Kidney
Cloudy Swelling



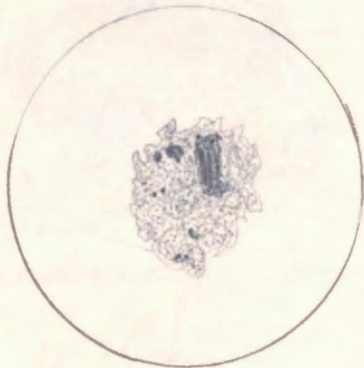
X 500

Pigmentary degeneration of Eye
showing pigment in cells of vitreous humor



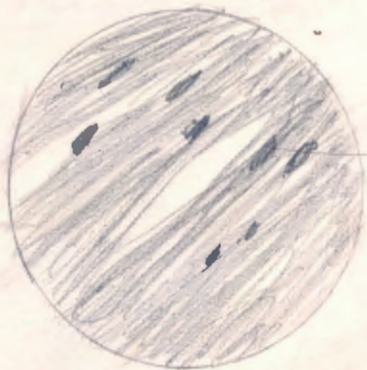
X 100

Pigmentary degeneration of Eye
showing pigment gathered in bunches of
cells



Tuberculosis of Lung

Calcareous showing lime deposit also
fatty metamorphosis of cells.

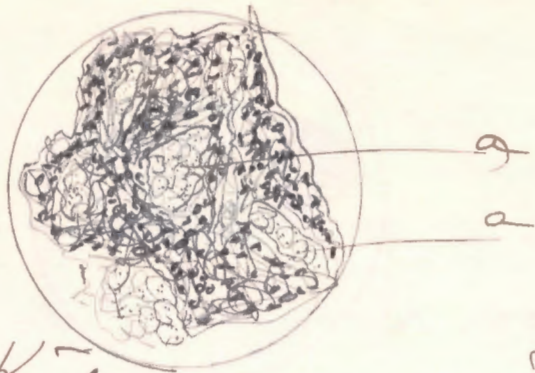


calculi

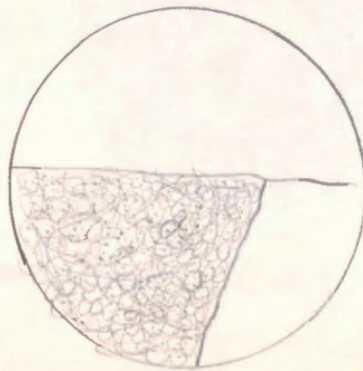
x 300

Calcareous fibroma.

Tumor from which the above
specimen was obtained contained
some calculi as large as a pea

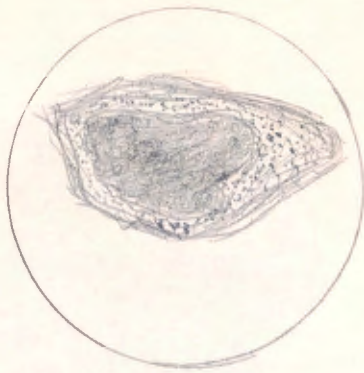


Surgical Kidney associated with
 suppurative nephritis, showing a
 general clogging of the tissues and some
 degenerative changes in local cells,
 aa degeneration of some cells showing
 fat debris.



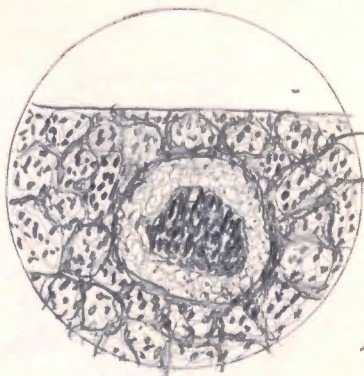
x 500

Casated center of a tubercle showing
 degenerated cells.



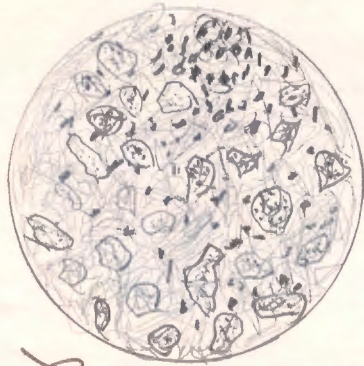
X 100

Tubercle which has undergone cheesy metamorphosis of its center.

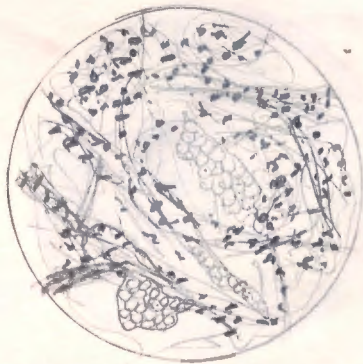


X 400

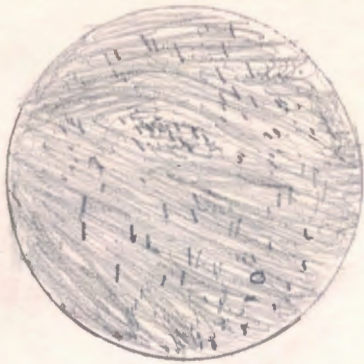
Acute glomerular nephritis showing glomerulus and cells surrounding it. The cells surrounding it showing degeneration.



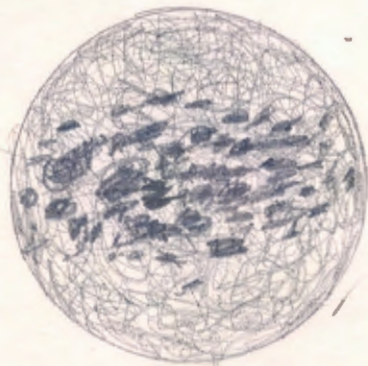
Liver showing deposit
of pigment and some cells that
have undergone fatty degeneration.



Kidney showing
some cloudy swelling cells and some
cells that have undergone fatty degeneration.



Calcareous Fibroma



Amyloid Spleen