Prognosis:- The prognosis of this disease is somewhat indefinite. As to the immediate life of the patient you can say that the disease is slow but progressive and they may go on from 3 to 15 years or more. There may be increasing regidity and deformity with more or less tremor. There may be much regidity and but little tremor or considerable tremor and little regidity untill late. At times there is monoplegia,hemiplegia perhaps debility. They finally die of toxic exaustion or some intercurrent infection.

Diagnosis:- It is not always hard to make although not always easy

Differentiate - 1st. Multiple sclerosis. Here you have the intentional tremor,upon active motion,while in paralysis agitans it is present during res or during voluntary motion. In multiple sclerosis the knee reflex is exagorated 2nd.and nystagmus.Lavid speech increasing irritability and reduction of the mental capacity of the patient

2nd. Post homiplegis tremor must sometimes be considered in the differentiation.

At times thrombosis leaves behind a hemiplegia and it may be so improbed untill you can't notice it in the movements or gate and then the hemiplegic tremor results. On careful examination regidity,exagerated reflexes on one side and evidences of organic lesion.

3rd. Senile tremor. Here you take into consideration the age of the patient,the stopping weak posture,tremor of the head and neck which is a rare place for the tremor to occur in paralysis agitans. Other evidences of senili

4th. General paresis. It is much more liable to occur earlier in life although it may occur late in life though not usually. There may be the mask like expression on the face like in paralysis agitans. There may be a tremor but is faster and finer. The knee reflexes are exagerated,abolishod or unequal. The axagerqted character of tye mental concepts in contradistinction to the clear mind in paralysis agitans.

Early in the disease the differentiation may be hard to make but later on it becomes quite easy to make. (insert,"on it becomes")

5th.Wry neck. It will be only necessary to consider this tortio collis or retro collis occuring in pralysis agitans. Most of these patients will give a rheumatic history.

Treatment:- It is directed to rendering the patient comfortable, heed him well,give him rest in bed,plenty sleep,stop the motor agitation. The best results are obtained by putting the patient to bed. In the milder cases you can let them out in the afternoon,into the fresh air. There must be perfect freedom from worry,care and anxiety. You can order baths which are warm but not to warm. The wamm baths will produce a relaxing influence on the rigid muscles,and a soothing influece upon the patient. Massage prevents to a considerable extent the contractures,tremor,regidity and pains in the muscles;it also improves the neutrition of the muscles. Apply the massag every day or every other day. Use galvanic electricity to aleviate the pains;use a current of 5 - 15 milliampers for 10 - 15 minuits a day. When there is much tremor,excitement,etc. give hyocine hydromate 1/150 gr. or you can give hyocine hydrobromate 1/200 combined with morphine 1/8 gr.. Codine may give relief. Some patients respond to Na and K bromate in 10 - 30 gr. doses. Veronal,trional or sulphonal may give results. As a stimulant to the digestion you can give strychnine,dilute acidum nitro muriaticum,quinine 1/2 - 2 grs.gives very good results in some cases. The amoniated tincture of valerian 1 - 2 drms once or twice a day may be given. Tr of gelsemium will lessen the agitation. Gland extracts have been used for this condition but their action is not uniform enough to warrent good results. Ext.of parathyroid 1/2 gr, pituitary body Ext. 20 - 30 grs. have been tried;also thyroid extract By the above treatment you can keep the patient in pretty fair condition treating the various symptoms as they arise.

MULTIPLE NEURITIS

This condition has also been called poly neuritis.

Motor or motor sensory or paralytic form:-

This the form of the disease which is present in 90% of the cases of multiple neuritis. Then comes the sensory form,sensory neuritis,which is assotiated with ataxia and paralysis without motor paralysis.

This disease is seen in alcoholics and especially in those who have diabetis. There is a toxemia which causes the results. You have a toxic neuritis which results in ataxia.

Beriberi:- It is a tropical disease. It is not very frequent here. It is endemis and infective.

Acute pornicious variety,viz. motor and sensory combined.

Etiology:- The etiology of this disease is marked and can be easily recognised. Alcoholism is at the bottom of 65 - 75% of the cases. Dr. Graves thinks the percent is higher than the one given. As the next most frequent cause dphtheria is given but it produces it only to a limeted extent. Led phosphorous and the infections as typhoid may produce it. Tis type is assotiated with paralysis and atrophy. It usually occurs in early life but may occur up to 45 years of age rarely laterbthan this. Men are more often affect ted with the disease than women because of the more frequent exposures although if women and men are equally exposed,women get the disease more often than men due to thexx greater susceptibility of their nervous system to those lesions. Epidemic infections as cerebro spinal meningitis,play a causal role. Other contributing causes are,cold,excesses of any kind,starvation, neuritis,tbc,at times leprosy,rheumatism,diabetis and even malaria has been blamed although Dr.Graves does not think that maleria is causative. To free drinking of tee predisposes to the disease also drugs as,chloral hydrate, chloral,Hg.,trional,etc..

Symptoms:- Usually there are certain prodromes of the disease that wil will lead you to suspect it. Numbness,tingling,pains,light pains especially in the legs and feet. There is muscular weekness and the patient is a little confused mentaly. Or the disease may flare cut suddenly. The tenderness and other symptoms gradually increase. A temperature of 102°- 102° may come on and it lasts for a few days to a few weeks. The weekness also becomes mor marked. The patient gets more sensative and many of them are treated for rheumatism. There is tenderness in the arm,leg and especially in the for arm,fingers and hand. There is a numbness which goes on to pain. The skin over the extremities especially below the knees gets red,shiny,glossy and lo looks edomatus. The weekness of the patient goes on untill he drops down and is unable to get up again or otherwise help himself. He is paralysed. The anterior tibials and the extensor muscles of the forarm are the first and worst to suffer and this may help you in making your diagnosis. The patient suddonly gets wrist drop,foot drop or shoulder drop and certainngroups of muscles are affected. There seems to be a certain selective activity for certain nerves which is interesting. The affected muscles atrophy. The muscles of the face and eye may be involved,the muscles contreled by the 4,5, 6,and 7th nerves. There develops a neuritis which in a few weeks develops into a paraplegia when you get foot drop,~~xkm~~,especially in alcoholics or wrist drop in Pb poisoning. In some cases on foot or hand drops after the other in other cases all four extremities paralyse rapidly. Then comes on edema and atrophy. As to the reflexes,right at the beginning they are increased for 1 or 2 days probably then they become deminished and are finally lost. From the hyperasthesia it goes on to anesthesia. The anesthetic areas may be irregularly distributed over the body. Then they go on to hyperalgesis. At the same time the temperature and pains seem to be decreased,however,they are seldom entierly absent. The articular and tendon senses are deminished or even lost and often they cant stand at all. Then the reactions of degeneration come on and they may be partial or complete. At times in addition to the ocular neuritis there is retinal hyperasthesia and often an optic neuritis. The involvement of the pneumogastric nerve in this disease is rare but if it is involved there will be palpitation,sense of sinking,dyspnoea, disturbance of respiration,etc. In the first day or two the sphinctors are paralysed. The mantal disturbances which are present at fkrst may go on to delerium or stupor and some have dilusions and hallucinations. After the paralysis have occurd which in alcoholics go on to quadraplegia there come on the reactions of degeneration as loss of excitability to the faradic current at first,later loss of response to the galvanic current as woll. Contractures follow. The feet become extended abd stiff,the legs flexed and the patient is helpless.

Sensory Symptoms:- The hyperasthesia,anesthesia and paresthesia are important,don't forget them. As the disease progresses and the neuritis has gon on untill it produces instability of gate and posture it is called neuritic psudo tabes (he lifts his feet high,etc).

Vaso Motor and Trophic Symptoms:- These symptoms manifest themselves in the redness,edema,glossy aperance of the extremities and there may be profuse sweats.

Mental Symptoms:- These are often called Korsakoff's psychosis. T They rarely recover from this? There is a muttering at first and a sen se of prostration and forgetfulness. An examination of the blood will not give you any information;there is an early leucocytosis.

Pathology:- The pathology of this disease is deffinite. It is a disease of the peripheral nerves,rarely going higher although it may pass into the chord. The muscilo spiral and the anterior tibial nerves are the ones most often and most severely affected. Thoy have an inter stitial neuritis and this inflamation is assotiated with or followed by degenerative changes. There may be small hemorrhages into the sheaths of the nerves followed by an exudate and an infiltration of leucocytes into the nerve sheaths. Then there is muscular atrophy.

If the neuritis goes on to complete degeneration it dets up a fibrosis,leaving a fibrosed nerve.

DIAGNOSIS:-

Differentiate:- 1st. Diffuse myelitis.
2nd. Anterior polio myelitis.
3rd. Locomotor ataxia.
4th. Spinal meningitis.
5th. Acute ascending paralysis of Landry.

When you find patellar reflex absent think of -
Loco motor ataxia
Multiple neuritis. -in man.
Acute anterior polio myelitis. - in children.
Myelitis.
Landry's progressive paralysis : pernitious multiple neuritis.
Beriberi , it is a toxic neuritis.

Where the patellar reflex is present think of ?
Paresis
Multiple sclerosis
Spinal meningitis.
Neurosthenia.
Cerebral lesion.

PROGNOSIS:-

If the disease is due to alcohol they rarely get well as they keep on drinking. They are predisposed to intercurrent infections as tbc. pneumonia,etc. If they cut out the alcohol for 1 - 2 years they stand a chance of getting well permanently.

TREATMENT:- This is not difficult. Keep the patient at rest Wrap them up in hot water compresses. At times lead,wator and laudnum may give releif. Menthol linament may do good. Asperin 10 grs. 3 - 5 times a day. For the pain give morphia 1/4 gr. or codine. Sometimes pryramidom,5 - 10 grs. Salcylates are also indicated. Purge the pation tiCnt with calomel and salines every once in a while. After the acute stage give strichnine and As. Apply early electricity.as ni the other nervous diseases. Keep up thd strichnine for some time. Gine KI 5 - 1 grs. a day. Feed him well and watch his hygiene and he will get well in 6 mo. to a year.

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THE CREATORS OD COMPETITION

3rd Term Pathology.

INTERNATIONAL

LEGAL CAP

20 SHEETS

3/15/'09.

B. Diseases of the brain, Outline of:-

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IV. Inflammations or encephalitis.
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2. Chronic, interstitial encephalitis.
3. Multiple sclerosis.
4. Chronic meningeoaencephalitis, or Dementia paralytica.

V. Infectious granulomata.
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VI. Tumors.
1. Glimoma.
2. ASarcoma.
3. Carcinoma.
4. Cholesteatoma.

VII. Cysts.

E. Diseases of the Brain.

I. Anatomical considerations:-

In order that one understand the diseases which affect the brain and the results which are to be expected a more or less definite idea of the anatomy must be had. The brain, as you know, is divided into the cerebellum and the cerebrum, either of which has two hemispheres. The pons verolei and the medulla oblongata, the latter is continuous with the upper end of the spinal cord. Of the coverings and the ventricels we have already spoken. Sections through the cerebrum and the cerebellum reveal to the naked eye, two colors, grey and white, spoken of as the grey and white matter of the brain.

Microscopically. The substance of the nervous system is made up of neurons and neurologia, both of which are derived from epiblast, or ectoderm, and are therefore, more closely related to the epithelial structures than to the connective tissue structures. The neurologia may be looked upon as the supporting substance of the brain, although epiblastic in origin, but it corresponds in function and changes to the other connective organs met in the bory. It is made up of a multitude of small cells, with round nucloi, in the cytoplasm of which are enumerbale small fibrills, which in the adults may or may not be connected with the neurologia cells. The neurologia cells are divided into two classes, the spide cells, having straight unbranched fibers, and the mossy cells consisting of many branched fiberills. The neurons are the parenchymatous part of th central nervous system and consist of ganglion cells and certain processes. The ganglion cells are found in the grey matter of the brain; in the great basal ganglia: in the ganglia connected with the spinal nerves: in the sympathetic ganglia and in certa ganglia of the organs of special sense. They vary in size from 4 microns to 135 microns, the longest probably being in the motor areas of the spin cord. The shape varies depending upon the way inwhich the processes come off. If there is only one process = unipolar; if two processes = bipolar: is many processes = multipolar. Most of them come under the latter group. The ganglion cell processes, which are really but extensions of th cytoplasm, are divided into two groups, the dendrites and the axones or axis cylinders. The dendrites come off from one part of the cells, extend a short distance and subdivide dichotomously (into two)into numerable little branches, upon which are small projections = Gemmules. From another part of the cell comes the axis cylinderand depending upon the fu ther behavior of these axons we subdivide the cells into 2 groups: I. whe the axis cylinder becomes a fibrile of the nerve. II. Those where the axi cylinder after extending a shortdistance breaks up into numbers of littl fibbillae. These axis cylinders after preceeding for a short distance ar encased in myelin or the white substance of Schwann, which serves as a protection for them. They are classified into association fibers = which run to the different parts of the same hemisphere: Comissural Fibers = whi run across to the opposite hemisphere; and the Projection Fibers = which b come parts of nerves.

The ganglion cell has no cell membrane, but is made up of a reticulum of cytoplasm, which takes more of the acid stainand in that cytoplasm are numerous substances which take the basic stain or better the stain with such substances as toluidoin blue or methylin blue. These substances are trigoid bodies and depending upon their arrangement, cells are variously classified. An important thing to remember, however, is that th trigoid substances are absent from the part of the cells from which the axone is given off. The nuclei of the different ganglion cells behave somewhat differently producing another basis of classification:- in the or form it rakes a basic stain, in another the nucleus probably takes the acid stain, or the nucleolus is deeply stained. Many of these cells, esp iall as one gets along in years, contain a yellowish brown pigment, whose importance is unknown. Remember that a neuron, then, is made up of a centralganglion cell or cell body and the processes, the dendrites and axi cylinder, and this constitutes the unite of the central nervous system.

The white substance of the central nervous systemis made up of support ing neurologia with the axones surrounded by myelin; while the grey matter is made up of ganglion cells and their processes, all held together by neurologia. In the cerebral cortex we may recognize four layers; an outte most one on the surface which is the molecular layer and is made up of neu logia and the dendritic processes derived from the genglion cells of the

subjacnet layer (2). the layer of small pyramidal cells. (3). The layer of large pyramidal cells (4) and the deepest of all is a layer of irregular shaped cells the polymorphous cells and and it is from the cells of this layerthat most of the axones, which go to form the nerves, arise.

Cerebellar grey matter:

This consists of three layers, (1). A molecular layer containing a few small cells on its outerside and some large basket cells on its inner side. (2). a thin layer of Purkinje cells, these are very large cells with many processes, the only ones in the cerebellum which give rise to axones going to the nervoes. (3). Granule layer, consisting of any cells whose nuclei take basic stains.

II. Malformations of the brain:-

These are farily frequent especially when one takes into consideration the lesser degrees.

1. Hyperplasia:-

by some this is called hypertrophy, a poor term, here the brain becomes much lar er than normal, the increase, however, being chiefly in the neurologia, causing the white susbstance to be increased, encroaching upon and destroying some of the grey matter. This is usually congenital, but it may begin in young adult life. If it has started before the ossification of the sutures, the crainial bonesare likely to be spread wide apart, as in congenital hydrocephalus, giving the appearance of a large head with a small face. If the process has begun after the ossification of the of the sutures there will be erosion of the inner plate and thinning of the bones. Sooner or later the case ends in death, with symptons of chroinic compression.

2. Anencephalia:-- Complete absence of the brain is rare and imcompatable with life. It is usually associated with absence of the cranium and with failure of closure of the spinal canal, this being represented by a shallow groove down the back with here and there remnants of muscle and tissue. Partial agnesia is sometimes present = absence of parts of the brain as portions of the poles of the cerebrum which more frequently affects the anterior pole and is compatable with life.

3. Agyria:-- This is the term applied to total absence of some of the well recognized convolutions of the brain and is not infrequent.

4. Microcephlia:-- This is an abnormally small brain. It usually affects the cerebrum but may also include the other parts. The cranium is usually too small, the face relatively too large. As a rule, those tracts of the cord whose elements are derived from the cerebrum are also too small. The child may be born alive but ordinarily lives only a short time.

5. Microgyria:-- One sometimes sees brains in which some of the convolutions are too small and poorly developed.

6. Encephalocele:-- This is the herniation of the brain or parts thereof through an abnormal opening in the skull. If it is composed of the menongies alone it is called meningeocele: If however, the meninges and brain herniate through = meningeo-encephalocele, especially where there is much fliud present, if however the amount of fluid be scant, we sometimes simply apply the term encephalocele. Usually the herniation is through openings in the occipital bane and the mass hangs down as a sack on the back of the neck. However, in addition to this location it may be found at the root of the nose or at the base of the brain or along any of the sutures. If there is much herniation it is imcompatable with life.

7. Porencephaly:--At times certain parts of the brain are congenital absent leaving holes which fill up with fluid and these may be in the substance of the brain or they may reach the surface. If the latter, they produce conical depressions into which the visceral pia dips and may communicate with the ventricles. The symptons depend upon the part affected. From these congenital conditions one must carefully distinguish acquired cysts due to hemorrhage, softening, etc., to which we can apply the name pseudoporencephalon.

3/19/09.

III. Circulatory disturbances:

1. Anemia.

1a. Etiology:- Decreases in the blood content of the brain is seen in general anemias and cachexias; in sudden shock or in - surgical shock and at times in arterio-sclerosis of old age; sometimes occurs reflexly from the too rapid emptying of an over distended bladder or peritoneal cavity; perhaps in the use of such drugs as ergot; and an increase in the intercranial pressure due to an increase of fluid especially in the ventricles, as an internal hydrocephalis.

2a. Morbid Anatomy:-- This anemia may be either acute or chronic. In the acute form the brain substance is pale and the distinction between the gray and the white matter is not so clear and none of the minute r d dots of the cut vessels appear. Microscopically, one sees no change except the partial or almost co mplete absence of blood.

In the chronic form, where the anemia has lasted for a long time, the brain appears to be drier and smaller, the convolutions may be swollen and narrower and the sulci deeper. Microscopically, in this condition one may see some degeneration of the cortical cells, but this is usually not marked.

3a. Results:-- Such a condition may produce vertigo, drowsiness, perhaps irritability, if more severe there may be coma, perhaps ringing in the ears and spots before the eyes.

2. Hyperemia:--

1a. Etiology:-- A certain amount of this is physiological, during mental activity. Increases of the arterial blood are seen where the hearts action is excessive from any cause. It occurs in coughing, vomiting, with severe emotion, delerium, acute mania, in acute inflammations of the brain, and in nearly all the general conditions and diseases as rabies and tetanus.

2a. Morbid Anatomy:--The membranes are tense and their vessels are stuffed with blood. The cerebral vessels are also filled and show as fine red lines and can be traced much farther than ordinary. On section, the punctum vasculosa are more numerous and distinct.

Microscopically:-- In addition to the filled vessels one may find capillary hemorrhages into the perivascular spaces and in the more extreme degrees out for a very short distance into the brain substance. Further than this the picture will depend upon the accompanying process.

3a. Such an overfilling of the vessels tends to produce ruptur = apoplexy. Such an excess of blood in the brain may increase the size of the cerebral vessels which press on the nerve centers and produce headache.

3. Congestion:--

1a. Etiology:--The venous excess of blood in the brain may be due to any interference with the return flow of blood, as in thrombosis of the sinuses, in tricuspid insufficiency, or in pulmonary diseases of an obstructive charcater. At times tumors may press upon the venous trunks and exudates may also act also in the same manner.

2a. Morbid Anatomy:-- In this connection it is important to remember that the blood content of the veins of the brain after death may not be a true criterion of their condition during life, since by gravity much of this blood may be either draine away, so there will be less than should show or if the head be lowered the blood may settle into the veins, thus also giving a false impression. It is the rule to find the veins of the dependent parts more filled with blood than the anterio For this reason, at the autopsy, if the brain is to be examined we do this first before the large vessels of the thorax are cut and permit a draining away of the blood. The congestion may be general or for some local cause acting upon the veins of Galen, it may be confined to the ventricles.

Grossly, the veins are full of blood, the brain appears too large and there is an excess of fluid in the ventricl s and in the interpial spaces. The increase of the venous blood may be so great as to produce a slightly bluish color in the white substance. There is no characteristic microscopical picture.

3a. Results:-- If the congestion be intense it may produce headache or even coma and if it continue for ant considerable time it willl almost surely produce edema of the brain.

4. Edema of the Brain:

4. Edema of the Brain.

1a. Etiology:-- A local edema occurs about tumors and areas of softening or it may be general in severe acute hyperemai, in congestion, in such dropsical diseases as chronic nephritis or failure of compensation, at times in hydrocephalus when the fluid soaks through the tissues, as it were: and in the alcoholoic wet brain.

2a. Morbid Anatomy:-- As a rule, the pia is also edematous and an excess of fluid is to be expected in the interpial spaces and in the ventricles. The brain tissue is foo soft, juicy, undergoes early postmortem changes and gentle pressure along the line of the vessels may express a little fluid. The convolutions are flatter and moister while michroscopically the perivascular spaces are distended, the nervous tissues show vacuolated spaces and the ganglionic cells not infrequently show absorption of fluid, swelling or treu degenerative changes.

3a. Results:-

Coma is not unusual and death may result.

5. Hemorrhages into the brain:- Hemorrhage into the brain is divided into 2 forms :(1). Puntctate, where they are small and numerous: (2). Massive, where they are large and usually single.

1a. Punctate hemorrhages. These are failry common.

1b. Etiology:- A certán degree of this occurs in nearly ever case of severe hyperemia, also in inflammationtions, either directly of the brain itself, or those extending in from the pia. They may occur in certain dissases as small pox, pernicious malaria, etc. And at times in concussion one may find a number of of very small hemorrhages instead of larger ones.

2b. Morbid Anatomy:- Usually the grey matter suffers more than the white and on cutting into it one finds numbers of little soft red areas which can be washed away by a gentle stream of water.

Microscopically these hemorrhages appear as little lines following along and lying within the perivascular lymph spaces or extending for a short distance into the brain substance, thus destroying a certain amount of it. 3b. Results:- As a rule the hemorrhages are not extensive enough to produce clinical symptoms.

2a. Massive Hemorrhages:- In considering the large hemorrhages of the barin it is helpful to subdivide them into apoplexy or the non-traumatic and into the traumatic.

Apoplexy or non-traumatic hemorrhages of the brain:- This is a more or less massive hemorrhage into the brain substance which has come about from the non-traumatic rupture of the vessels.

In the etiology we must consider the predisposing and the immediate or exciting causes. We nenver have an apoplexy occuring from a normal vessel, there must have been some condition atedating. By far the most frequent is atheroma or arterio-sclerosis and this from any cause. Important here are the little miliary aneurisms along the cerebral arteries and their branches. Then too, fatty degeneration of the vessel walls may predispose to he orrhage. This is seen sometimes in certain posions as in phosphorous posioning and snake venom. At times in the young, acute infections may produce the fatty degeneration and thus explain the occasional examples of apoplexy in young children. Given then this weaken vondition of the vessel, the immediate cause is some marked, usually sudden increase in the blood pressure. This may be due to violent emotions, as an outburst of anger, perhaps excessive mental effort: excessive physicial activity, but the most interesting of all are those frequent cases which occur during the sleep. Here we are not able to say what the exciting of the increased blood pressure is, but it is probably due to an excessive mental activity as dreams or night mares, which do not rise to the conscious mind.

Morbaid anatomy:- As to the site of preference -- the most of the cases occur along the course of the branches of the middle cerebral artery, especially those to the corpus striatum and the potic thalamus. Indeeed the greater number occur in the internal capsule. Next in order of frequency is the cerebral cortex, pons, cerebellum, centrum ovale, middle cerebella peduncle, crus cerebri and the medulla, named in the order of their frequency of occurence. When the area is sectioned the hemorrhage may show as one mass or seem to be made up by fusion of a number of areas, the latter, however, may be due to the irregular distribution of the blood in the brain substance. If the patient has died during the hemorrhage, which is very rare, the blood will be found still fluid. As a rule, several hours or even days elapse from the time of the rupture of the vessel to the death of the patient and here one will usually see the central part to be distinctly clotted, either firm or jelly like. The size of the hemorrhage will vary very much from the size of the end of one's finger up to one which involves and destroys w while lobe.

If the hemorrhage has occured in the internal capsule, the intervening tissue may be broken through, and the blood be discharged into the ventricle.

In the early cases, microscopically the hemorrhage shows that the afeected part of the brain will be entirely degenerated and for a varying distance out xxxx the brain tissuex will be infiltrated with blood and shows degeneration in dinimishing degrees. If the patient does not die of the hemorrhages a number of changes at the site of the hemorrhage are possible. The clot acts as local irritant, setting up a fibrous hyperplasia, by which a capsule is formed, enclosing the hemorrhage. Within this capsule the clot shrinks, the more fluid part is absorbed and in favorable cases it becomes completely organized, leaving only pigmented scar tissue. In other cases after the incapsulation, the blood may become broken down, and more fluid and remain as a cyst, the contents of which may show the blood pigment or this latter may be deposited in the capsule leaving the fluid clear or only slightly tinted. Another possibility, which is an unfortunate outcome, is the subsequent additon of pyogenic organisms giving rise to a cerebral abscess.

As to the immediate results:-- death frequently occurs, but usually not under 12 to 24 hours after the rupture. In additon to this varying degrees of paralysis, usually hemiplegia on the side opposite to the appoplexy, may be found. The focal symptons and the clinical manifestations will vary with the site and depend upon the area destroyed. It is by these thatwe attempt clinically to locate the hemorrhage.

As to the remote effects---- the hemorrhage is followed by a degeneration or a Wallerian along the /x/x/s/ /o/f/ /t/h/ lines of the axones, which are interfered with. As recovery occurs a gradual return of the function, at least to a fair degree, will be noted in favorable cases. This is due to regeneration of the axones where their ganglionic cells have not been destroyed.

3a. Traumatic Hemorrhages of the Brain:--

1b. Etiology:-- Here there has been a definite injury to the head, as a blow, fall, or some penetrating wound. There may or may not be an accompanying fracture. The hemorrhage may occur in the brain substance immediately under the site of the injury or on the opposite side. Where it occurs on the opposute side it is frequently due to a distinct fracture, at times, however, there may be the injury and the hemorrhage withoue such a fracture. The following will probably serve to explain the condition:------ The skull is an elastic box and when it receives a blow that is transmitted around the sides, causing a bulging out of the bane at this place. The brain, however, being softer does not follow so quickly and meets, as it were, the skull on its rebound.

2b. Morbid Anatomy:-- The brain tissue in such traumatic hemorrhages is usually lacerated primarily or along with the break in the vessels. The hemorrhage may be single or multiple, but aside from the location, the laceration and the cause it resembles apoplexy. Then too, the traumatic hemorrhages of the brain are nearly always accompanied by meninge al hemorrhages into the subdural space. The subsequent course of events is like that of apolexy.

3b. Results :-- Depending upon the site, the extent of the hemorrhage, the amonut of laceration of brain tissue, and the degree of compression of the meningeal hemorrhages the results will vary.

6. Thrombosis and Embolism.

1a. Etiology:-- These are due to the same causes which operate in other parts of the body. Thrombosis is due especially to athroma or syphilitic endarteritis. In embolism, the obstructing substance may be any one of the common forms of embolis, more frequently a bit of blood clot or of heart valve from an aortic valvulitis, or a part of a clot broken off from a thrombosed vessel.

2a. Morbid Anatomy:-- The thrombosis may occur any where in the brain but most frequnetly in the basilar arteries; while embolism is most frequent in the arterys of the Sylvian fissures, 80% occurring here. This is due to its being more directly in line with the blood stream from the left heart. Embolism does not occur in the basilar artery because this is formed by the union of the two vertibral arterys and is larger thsn either contributing trunk.

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3a. Results:--The thrombus or embolis practically always results in an infarct. But if it be a thrombus, forming so slowly that a collateral circulation may be established no change may ensue or only temporary symptons or changes.

7. Infarcts.

1a. Etiology:-- Infarcts of the brain are due to thrombosis and embolism from any of their causes. Since most of the small arteries of the brain are end arteries these two processes, thrombosis and embolism, are nearly always followed by infearction.

2a. Morbid Anatomy:--

1b. Location:-- These infarcts are seen in the areas supplied by the arteries which are plugged up. The sites of preference of these were given above.

2b. Forms:-- As a result of the cutting off of nutrition we get e degeneration or softening of the brain known as encephalomalacia. Depending upon the color, in the gross, we divide these into red, yellow and white softenings. The underlying changes are essentially the same in all. The differences in color are due to the varying amounts of hemorrhage or blood present and the varying degres of degeneration in this blood.

1c. The red softening contains much blood and corr sponds to the red or hemorrhagic infarcts in other parts of the body.

Grossly, when one opens the skull cap the veins will be seen distended with blood and as a rule, there is an excess of cerebro-spinal fluid, especially in the sulci . Now, on sectioning the infarcted areas we come upon an irregularly shaped portion in the midst of which is a red or dark red grumous fluid and about this is a zone in which the brain tissue is distinctly soft and pulpy, the red color gradually fading a way and edema is much in evidence. Ordinarily one can not make out the cone shape. The size varies according to the caliber of the vesel, from one to two cms, to probably almost one third of a hemisphere.

Microscopically, one finds great numbers of r,b,c, in various stages of disintegration; the ganglionic cells braoken down into a granula fatty material or they may appear only as ghost of cells with fat globul Smaller rounded cells filled with fat droplets (compound granule cells), are seen and these are probably leucocytes which have wandered in in an attempt to remove the debris and have become involved in the necrosis. Othere better preserved leucocytes will be seen, espocaillya bundant about the margin. As one leaves this central necrosed portion the ganglic cells show edema: and degeneration of the exones and broken up neuroglia fibers are made out. A little later one may see cholestrin crystals wity some corpora amylicia.

2c. Yellow Softening:-- This represents, in most cases, the later stge of red softening where the blood has been broken do down and much of the pigment removed leaving a distinct yellow color from the small of pigment and the fatty change. Sometimes, also, it is possible for the white softening to be transformed into the yellow by the subsequent deposition there of altered blooddpigment. In the yellow softening very much the same gross picture prevails as described for the red form, save for the color. Then, too, while the area may be somewhat larger, it is usually more definitely circumscribed and the zone of softened brain tissue aroud it is not so wide.

Microscopically, in the yellow softening, the cells are more completely degenerated, blood pigment is seen, many fat globules, with perhaps not even the ghosts of cells. Here, too, one may get chl~~ŕŕŕŕŕ~~ bholestrin crystals and perhaps crystals of fatty acids and the corpora amylacia. About the central part is an area in which the blood vessels a hyperemic and an infiltratio n of leucocytes, both polys and lymphocytes (especially lymphocytes), and evidences of hyperplasia in the neuroglia cells. Perhaps with this one may see an occasional true fibrous tissue bibroblast, but this not to a marked degree.

3c. White Softening:-- This corresponds to the anemic infarct elsewhere and results from the edema and liquifaction necrosis of a part of the brain deprived of its blood supply and is most typically seen in the white substance of the brain.

Grossly, one sees an irregulary shaped area filled with a milky white fluid surrounded bu ragged walss from which project the little free ends of fibers and the small blood vessels. This fluid part may resemble pus grossly, but it is usually too white for ordinary pus and sometimes distinctly thin.

Microscopically, one sees that it is composed of granular anf fatty detritis, compound granule cells, nuclei of neuroglia cells, and later the corpora amylacia, with some leucocytes. The same boundary zone of diminishing edema and degeneration of the brain substance is seen about the area.

3a. Results:-- As to the immediate result---- death may ensue from the involvement of important parts and the clinical symptons are very like those of hemorrhage, especailly apoplexy, the exact featrue depending upon the size and the location.

Remote Results----If death does not result, the area may become encapsulated by proliferation of the neuroglia tissue. Here, the contents may have its fluid part removed, when the area shrinks and this may become completely organized by the penetration of the fibrous tissue or neuroglia tissue, usually the latter. At other times the more solid parts may be broken down and removed, leaving the fluid contents to form a cyst. This fluid may be clear, all the pigment having been deposited in the wall or it may still be brownish fro m the blood pigment. An occasional result is the calcification .

IV. Inflammation, Cerebritis or Encephalitis.

1. Acute Encephalitis:-- Of this there are four forms: acute traumatic, simple acute focal, acute hemorrhagic, and acute suppurative.

1a. Acute Traumatic Encephalitis:--

1b. Etiology:-- Here there has been some form of injury to the brain, as by a penetrating wound or laceration of the brain substance by a blow on the head, or by fragments of bone in the depressed fractures, or any other cause which serves to produce a definite injury to the brain. In this form we exclude those where there has been a subsequent infection with bacteria. This form, then, merely represent the inflammatory reaction produced by irritation of the destroyed brain tissue.

2b. Morbid Anatomy:-- Grossly we will see only the result of the destruction of the brain tissue, with this there is apt to be more c less hemorrhage. The subsequent changes are practiaclly indenticle with those which follow traumatic hemorrhage of the brain and indeed thare is very little reason for recognizing this as a separate process, save for the sake of logical completeness in discussion.

Microscopically, one finds the degenerated ganglionic cells, axones and neuroglia; disintergrated r,b,c, the vessels are hyperemic and there is a zone of invading leucocytes; later proliferation of neuroglia will be seen and this will finally result in the encapsulation or organization of the area. The results depend upon the site and extent of t the injury.

2a. Acute Simple Focal Encephalitis:--Here, we have an inflammation of many localized areas of the brain without suppuration.

1b. Etiology:-- This process occurs in the course of many acute infection as influenza, typhoid, diphtheria, rabies, sometimes in acute articular rheumatism and in a few cases it is thought to have followed sun stroke. Some of these cases show in the lesions the same microorganism which is responsible for the gemeral disease present. In other cases a different micro organism is found and here the general disease serv serves to lower the resistance of the part of the brain affectd, thus permitting the action of these other microorganisms. Then again, the toxins which are found in these acute infections can be absorbed and carried to the brain and by their action alone produce this form of encephalitis.

2b. Morbid Anatomy:-- Throughout the brain are many little areas which vary in size from microscopic to a few mm. When large enough to be seen they show as little red softened areas, usually distinctly softer than the tissue in simple softening and more red. At a later stage the blood is absorbed and the areas are paler. It is probably that grossly, at least in the greater number of cases, one could not make the diagnosis from the gross appearance alone, as he might confuse this with miliary abscesses, perhaps miliary tubercles, etc..

Microscopically, however, during the earlier stages one finds the areas showing very hyperemic blood vessels and at times capillary hemorrhages and collection of lymphocytes. A little later the ganglionic and neuroglia cells af the part are degenerated and some cells larger than the ordinary neuroglia cells, but perhpas derived from them, mke their appearane At the same time one usually finds a distinct number of plasma cells. If the area be not large, healing may occur by the formation of a small cyst or a scar. Where the areas have occurred just under or very near the pia, this latter may become adherent to the brain.

3b. Results:-- The clinical manifestations are very irregular because of the wide distribution of the areas and frequently these symptons are obscured by those of the general disease in the course of which this affection has occurred

3a. Acute Hemorrhagic Encephalitis:-- This is a disease very similar to the above, although more severe and not so frequently met. It is characterized by little localized areas of inflammation in var ious parts of the brain, in which areas hemorrhages are found.

1b. Etiology:- This is much the same as that given for simple acute focal encephalitis and indeed in most points it seems to be but a more severe stage of the other.

2b. Morbid Anatomy:-- Sometimes both the brain and the cord are involved, giving rise to the name encephal-myelitis h emorrhagica" If the bulk of the inflammation is located in the nuclei of the floor of the fourth ventricle and about the aqueduct of Sylvius, the nuclei of the third, fourth, and sixth crainal nerves, the condition is known as polio-encephalitis superior. If, further behind that, involving the nclei about the fortu ventricle with the seventh to the twelfth nerves, = polio-encephalitis inferior. This expression "polio-encephalitis" signifies an inflammation chiefly confined to the nuclei at the base of the brain. Sometimes with this the anterior cornua of the cord are involved giving rise to the name polio-encephalo-myelitis". Sometimes the involvement is chiefly confined to the motor nuclei of the bulb and we get the condition known as acute bulbar paralysis. In all these conditions the fundamental changes are the same, the distinctive names being given to indicate the site of the chief lesion.

Grossly we find little areas varying in size from a pin head to the end of a pencil, soft, very red, pulpy or sometimes pink and stippled with little red dots. The appearance of the pia will vary depending upon any accompanying leptomeningitis which is frequently associated, so that we nearly always see the near by pia turbid with injected vessels.

Microscopically:-- we find the vessels full of blood, the perivasc perivascular sheaths full of lymphocytes and these are getting out into the tissue, the neuroglia infiltrated with red blood cells or are the seat of distinct hemorrhages. The ganglionic cells are swollen, the tigroid bodies and nuclei are altered in theri staining, their axons swollen and fragmented.

Results:-- Each of these specially named processes will produce different clinical pictures because of the different parts involve

4a. Acute Suppurative Encephalitis:-- Here we have an a cute inflammation of the brain substance due to an infection with the pyogenic organisms. It may be either diffuse or localized, the latter more frequently being called abscess of the brain.

1b. Etiology:-- As a rule, the diffuse form of this disea disease occurs of the superficial parts of the brain and is secondary to a purulent meningitis. Here the microorganisms penetrate the cortex along the lines of the lymphatics and the perivascular lymph spaces. However, the localized form may arise from infected emboli or thrombi, or may be due to the subsequent infection to an injury to the brain, or may be produced by the extension of a suppurative bone disease as when the bone is involved from an otitis media or from the skull bones . Multiple abscesses may be formed in the brain in pyemia and at times the absces es formed in pneumonia or in the chronic suppurative inflammations of the lungs and in these cases it is probable that the organisms have been transmitted to the brain through the retropharyngeal lymphatics to produce an abscess at the base of the brain. The mast frequnet organisms found are the micrococci, strptococci and pneumococci.

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2b. Morbid Anatomy:-- As suggested before, there are two forms, diffuse and localized.

(a). The diffuse form affects the cortex and its effects are seen in an edema of the gray matter, injection of the vessels, softening of the brain substance and sometimes capillary hemorrhages, The involvement in this form rarely goes very deep into the gray matter and almost never involves , to any material degree, the white substance.

Microscopically:-- The neuroglia tissue will be seen infiltrated with leucocytes in which the polys predominate, the ganglionic cells are swollen, edematous and are apt to show slight degenerations. The vessels are full of blood and the perivascular spaces are distended with leucocytes

(b). Coming now to the more important abscess of the brain, we may find them numerous and small as the result of a pyemic process or ther may be one fairly large, as the result of infected emboli or from some

destructive bone disease or from the extension of a purulent thrombosis in one of the sinuses. The cerebrum is the site of preference and the cerebellum next in frequency. In the larger more typical forms one finds a central area of fluid or semifluid purulent material, white or slightly blood tinted from the hemorrhage. These are either surrounded by a zone of degenerated, softened, edematous, perhaps somewhat hemorrhagic brain tissue, or if they have lasted longer there may be a kind of capsule formed by the proliferation of the neuroglia, however, the tendency to healing is very slight. The abscesses tend to spread and get larger by the gradual involvement and breaking down of the adjacent brain tissue. If, of much size, they may produce a distinct increas in the intercranial pressure whereby the convolutions are apt to be flattened. The degree of involvement of the pai depends naturally upon the proximity of the abscess to this membrane. Very fr equently, even though the abscess may be distinctly within the brain substance, one is apt to find a more or less localized inflammatory condition of the pia either simple or more frequently purulent.

Microscopical examination of such an abscess shows the soft fluid part to be made up chief ly of polys, the causative microorganism and the end products of degeneration of the brain tissue. Around this comes the zone of inflamned brain substance, which in its description corresponds to that given in the diffuse form.

3b. Results:-- If these areas be of any considerable size there is rarely any real healing. The process usually gets larger and larger until so much important structure is destroyed tahat death occurs. Even in the pyemic form of brain abscess, the general condition of the patient usually kills him before healing occurs. In the diffuse form also, the underlying cause is almost always of such severity that death will ensue. The clinical manifestation will depend upon the size and situation of the abscess.

2. Chronic Interstitial Encephalitis.

This is a condition in which there has cocurred a chronic hyperplasia of the neuroglia. Many forms, of necessity, must be grouped under this term.

1a. Etiology:-- We may say that this chronic hyperplasia occurs under a number of conditions. (1) It may involve all of the brain or all or a greater part of one hemisphere, leading to an atrophy of that part. Here it is spoken of as a diffuse or disseminated chronic interstitial encephalitis also sometimes called " lobar sclerosis". The cases included here are probably due to an underlying congenital defect in the arteries supplying the part. (2) It may occur in limited and circumscribed areas as a terminal stage in other diseases as in congenital syphilitic children, or about a congenital defect as in porencephaly. (3) It may follow the little areas of acute focal encephalitis where these are healed. (4) It may be one form of syphilis of the brain. (5)Healing of the little tubercular areas may produce it. (6) In chronic degenerative chorea (Huntington's Chorea), degenerative changes occur in the anterior pale and in the motor areas and these may be followed by a sclerosis of these parts. (7)There is a group of cases in which there occurs althrough the brain and cord little areas of sclerosis which constitute the disease entity of multiple or insular sclerosis which will be described separately.

2a. Morbid Anatomy:-- Where the process is diffuse or disseminated, a distinct atrophy or hypolpasia occurs. The involved area is much too small. At times a distinct space of even one cm exists between the dura and the parietal pia. The visceral pia is adherent to the brain and when removed the convolutions are seen to smaller, the sulci and fissur broader, but frequently too shallow. This part of the brain feels distinctly too firm and does not alter its shape on handling. It also cuts with increased resistance. The gray matter is too thin and not sufficiently well marked off from the white substance. This latter, the white substance, is usually the chief seat of this sclerosis but the process is not by any means confined to it.

Microscopically:-- the neuroglia and the connective tissue are distinctly increased. The ganglionic cells and their pro cesse, both the dendrites and the axones are atrophied. One finds the little corpora amylacia which have occurred from the degeneration of the cells and with these an occasional compound granule cell (Cluge's Corpuscles or cells). Along with this there will practically always be seen chronic inflammatory or degenerative changes in the blood vessels, but one usually has little difficulty in making out that these are secondary. All of the smaller areas of sclerosis referred to under the etiology will show similar changes but here one nearly always finds the ganglionic cells and the myelin sheaths degenerated.

3. Multiple sclerosis:- also called insular sclerosis.
This is a chronic disease of both the brain and the cord, of uncertain oiology and charactorized by multiple widely scattered little areas of sclerosis.

1a. Etiology:- As indicated above the cause is not known. Some claim that it followslate after some of the acute infections, as gripp, scarlet etc, where it has been preceded by the simple acute focal oncephalitis. Others claim that syphilis is a prominent cause and it is known that syphilis can produce a condition similar to this. In some cases there seems to be a distinct hereditary or family tendency.

2a. Morbid anatomy:- scattered here and there through the brain and cord, at times more numerous in one than in the other are many little grey patches of grey hardening, varying in size from pin head to 3-4 c.m. in diameter though the large size is rare. Areas of all size will be found in the same case. The white matter of the brain is more often affected than the grey, but the latter does not entirely escape. A marked feature is that there is no mrked roularity in the distribution of these areas and they show no relation in occurence to the motor areas, nervous tracts and the basal ganglia. If they are in the cortex the pia is frequently adherent over them, when it may be so thick as to obscure the area under it or still sufficiently thin to allow the little areas to be seen. On sectiong the brain a distinct difference is noted as the knife goes through o e of these hard patches. They seem to show a special tendency to be of larger size than in the pons and the medulla oblongata. On section they show as localized greyish, slate colored or greyishred patches and on fixation in Mueller's fluid they stand out more clearly as yellowish areas. In the cord the cervical and Lumbar enlargements, as a rule, show more of the areas.

Microscopically one finds an excess of neurologia tissue, frequently distinct amounts of connective tissues, the myelin sheahs are destroyed so that the naked axones run through the area. At times though not frequently all the axones will have disappeared. If is believed by some that the axones that one does see have come in as a result of regeneration of them. Toward the periphery of the area will be found compound granule cells, corpora amylacia, bits of myelin and fat droplets and in most cases the blood vessesl are seen to have undergone a hyaline degeneration with a thickening of the wall. In some cases these changes in the blood vessels are so marked as to leadsome observers to regard them as the fundemental lesion.

3a. Results:- These cases never recover. He presents the clinical picture of volitional tremors; the scanning speech, where the words are spoken slowly and he accents different syllables; also nystagmus of both eyes is present.

4. Chronic meningoo-encephalitis, or general paralysis or parasis of the insane or domentia paralytica:-
This is a chronic disease of the brain and cord with their membranes, producing a certain form of mental disturbance with a gradual onset of various paralyses, more or less complete.

1a. Etiology:- It occurs most frequently in males, coming on between the ages of 35 and 55 and in by far the greater number of cases syphilis will be found (75-90%). Combined with syphilitic infection as a predisposing cause, an important elemtn seems to be mental strain as from business worry.

2a. Morbid Anatomy:- In a typical case grossly we see that the dura is adherent to the skull and not infrequently is the seat of a chronic internal hemorrhagic pachymeningitis and this most frequently occurs over the convex part but it may spread to the dura of the base and of the spinal cord. The pia is very frequently thickened, either showing as lines along the blood vessels or as diffuse patches. The endothelia on the inner side of the visceral pia is proliferated, frequently producing such adhesions to the cortex as to be stripped with difficulty tearing the brain substance. The vessels of the pia are surrounded by a zone of infiltrating lymphocytes. The brain itself may seem to be alrger, due to an accompanying internal hydrocele. It may be, however and usually is, decidedly smaller and atrophic. When the pia is removed the convolusions are apt to be less well marked off and when section is made into the brain tissue it is seen to be too pale and the grey matter of the cortex is distinctly too thin. The vascular changes are seen with the naked eye. The distension of the blood vessels and their perivascular spaces produce a porocity or cribriform appearance. The ventricles are

always too large, sometimes distinctly so, and the eppendema and the choroid plexus are covered with little granulations. The basal ganglia, the pons and the medulla usually show no gross changes, but the dira about the oblongata may be thickened. In the cord the pia is too adherent to this structure, more escpcially over the posterior columns and corss sections of the cord, even in the fresh state, may show degeneration changes in to posterior columns.

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Microscopically:- The brain, especially in the fraontal lobes, show changes in the ganglionic cells, their dendrites and their axones. The glial tissues is much increased. The changes in the ganglionic cells brought out by proper staining, consists of a swelling and a cloudiness of the cytoplasm, later a shrinkage and atrophy of this and formation of granules which stain deeply; the nucloi of these cells are too pale. Excessive pigmentation of the ganglionic cells with vacuolation of them may occur. The axones show an irrgularity of contur and the medullary sheath is gone, either partly or entirely. The degeneration affects first the radiating and longitudinal fibers of the cortex. The dendrits are swollen, shorter, and shrunken. Special staining of the neurologia shows that the number of the nucloi is increased and t our fibers too dense and numerous. With this one sees an increase of the connective tissue, which has grown in along the pial vessels. In advanced cases the vessles show marked changes, the walls thickened, adventitia proliferated, but ~~xkx~~ with all this the lumena are increase or dilated, and the perivascular spaces are much dilated and in extreme cases produce the porocity mentioned above.

In the cord the changes are usually those of the combined systemic disease involving chiefly the posterior and lateral columns. At times, however, it is so combined to the posterio columns as to resemble so closely Tabes dorsalis that a differentiation in the cord apone cannot be made. It is possible however that the tabes dorsalic may have preceeded the chronic moningo-encephalitis. In most cases the crainial and periphoral spinal nerves show degneneration.

3a. Results:- This is the condition most frequently present when we say that the patient is suffering from softening of the brain. There is no recovery. The onset is gradual and for the clinical symptoms you are referred to your lectures on mental and nervous diseases.

V. Infectious granulomata.

1. Tuberculosis-

T,b,c, of the brain may appear as milliary tubercles or a large solitary tubercles.

(a). The milliary form is found in general miliary t,b,c, where the pia shows a number of miliary tubercles. The cortical parts are the ones most frequently involved, although at times these show as little grey or yellowish whit areas with soft centers, frequently with a slight area of hemorrhage about them and microsco cally they present the typical appearance and surrounded by a zone of inflammatory reaction.

(b). The solitary tubercles in the brain are at times called tyromata. These may appear independenly of t,b,c of the meningeso. It is more frequent in children than inadults and is almost always secondary to t,b,c, elsewhere, especially of the lymphatci nodes. The bacteria reach the brain through the blood vessels. The lesions show as rounded areas varying from those that can just be seen with the naked eye, to as large as a hen egg, rarely larger. They are greyish yellow, with caseous center frequently showing a zone of miliary tubercles about the and they grow by the breaking down and fusion of these. There is rarely any attempt to wall it off, but there may be.

2. Syphilis of the brain:-

If it be remembered that in the third stage of acquired syphilis that we have a marked tendency to arterioasclerosis and that the syphiliti virus frequently show a marked prediliction for the brain andthe cord, it will be weel understood that syphilis as a cause may underlie many chronic conditions. At these lesions are so located that we can recognize a clinical and pathological entity as in the chronic meningoo-oncephalitis then again on the other hand the lesions may be so irregular in their distribution and produce such a series of mixed symptoms as to render a classification and identification impossible. Indeed when one sees these cases presenting such irregular symptoms, he is at once justified in considerinf the possibility of syphilis being the underlying cause.

Coming now to the more distinctly recognized lesions, let us speak of 2 forms:- (a) Syphilitic oncephalitis; (b) The solitary Gumma.

(a) This is a name given to the condition in which multiple gummatous formations occur in the grey and white substance of the brain, most frequently in the grey. Here the little gummata form along the lines of the blood vessels, consisting first of lymphocytes, plasma cells and an occasional giant cell. Then comes the hyaline formation in the vessels and the casoation of the masses of the cells. Healing of this may produce little localized areas of sclerosis, very like and probably frequently identical with the multiple or insular sclerosis described. Before the complete sclerosis they may show as little areas with a grey or white center and a greyish red periphery. Considering the gross appearance of these alone it is impossible to distinguish them from miliary tubercles. But in the latter, of course, we always find a distinct primary focus of t,b,c, in the general autopsy. The symptoms in these cases is very like those in multiple sclerosis.

(b) Solitary Gumma; While the word solitary gumma means only one gumma present, yet we may have 2-3 of fair size. They vary from 1 to 10 c.m. in diameter, the latter size being extreme and rare. At the P. M. one does not frequently find a gumma of the brain, at least in these days. This is due to the greater tendency of t e present day physicians of recognizing syphilis and instituting the treatment, at least sufficient treatment to prevent the formation of the gumma. Then, too, the gumma of the brain usually produce such symptoms as to compell attention and treatment.

Grossly:- the consistency of the gumma is rarely uniform. the periphery is soft, odematous, and red from the excess of blood and the central part is drier, paler, firmer, with less blood, perhaps hyaline or showing cheesy areas. The outlion is frequently irrgeular.

Microscopically:- In the central part we see the poorly staining granular or hyaline material, with a considerable amount of fibrous tissue. Then in the outer zone are the fibrous tissue fibroblasts, with an occasional giant cell, and plasma colls. There is some degeneration in the ganglionic cells and evidences of degeneration of the glial colls. The blood vessels in and about the gumma practically always show either a chronic endarteritis, frequently producing obliteration of the lumen or it may be a markedproliferation of the adventitia. Such a gumma may press on and destroy or involve important centers or tracts or it may produce such a blocking of the circulation as to cause real cerebral softening.

VI. Tumors of the brain:-

These may be either primary or secondary, the latter are such as one would expect in a wide spread involvement with secondary tumors as sarcoma or cancers spreading through the blood vessles. Their importance lies in the hoastening death of the patient otherwise doomed because of the metastatic depositos in other parts of the body. No special description of these is required.

1. Glioma;- This tumor is found only in the central nervous system and in such prolongations of it as the retina. It probably the most frequent brain tumor, especially in children and in young adults. It may occur any where in the central nervous system, usually however, in the cerebral cortex, and frequently it is not well marked off. As a rule, it is solitary; from 1-12 c.m. in diameter. The consistency of the tumor depends upon its age; its vascularity; and the amount of degeneration. When small, it is usually firm and greyish white. The larger ones may have a greyish red appearance, from the many blood vessels or may show areas of hemorrhagic softening. As a rule there is not much breaking down of the adjacent brain tissue and here the differentiation of the tumor and the normal structure is more difficult. In certain cases, however, the degeneration occurs more rapidly about the tumor's periphery and the tumor may thus seem to be completely isolated from the nervous structure.

Microscopically:- one can usually make out that the tumor has arisen from a multiplication of the glial tissue. In the simpler forms the fibrille are seen closely interlaced and running in every conceivable direction, while the glial cells are also increased. In these the nucleus may be single, small, round, or oval, but other cells will occur which are larger and contain more than one nucleus. Cells which have been mistaken for ganglionic cells, but these ganglionic cells do not occur in these tumors except accidentally. Nerve fibers may be met in these tumors and

and if in considerable numbers = neuroglioma. But these can not be distinguished grossly. As one reaches the margin of the growth the tumor appearance gradually merges into the normal structure. The blood vessels are numerous and large, their walls frequently showing degenerative chang At times the nuclei are so numerous and so conspicuous as to remind one of sarcoma and thus have been called gliosarcoma, a term which should not be used since these have not arisen from the proliferation of connective tissue . As a rule, the glioma produce less destruction of the brain t tissue by pressure than any other tumor of the brain. Pressure symptons may be present and in other rarer cases the bone may be eroded.

2. Sarcoma:--In adults this tumor is perhaps the most frequent and in total numbers comes next in frequency to the glioma. Remember that they are connective tissue derivatives and must therefore arise from the pia or prolongations of it into the brain tissue or spread in from the bone or arise in the adventitia of the blodd vessels. The sarcoma may be small, round, hard, white and surrounded by an incomplete capsule. Such a form of sarcoma is difficult to differentiate from the glioma. Anf other form may be large, diffuse, infiltrating and vascular. Such a tumor as this may be so large as to occupy almost an entire hemisphere. Sometimes in the smaller form there may be enough well formed fibrous tissue to justify calling it a fibro-sarcoma. Otherwise the microscopical appearance of sarcoma of the brain is the same as that of similar tumors elsewhere. If there are many wide blood vessels = "angio-sarcoma". There may or may not be much destruction of the brain tissue by pressure or invasion.

3. Carcinoma:__- Primary carcinoma of the brain may arise from the epithelia of the ependyma or choroid plexus of the brain, or pituitary body and spread to the brain tissue. All these are very rare.

Secondary cancers are more frequent.

4. Cholesteaoma:-- This is a rare tumor containing cholestrin and made up of somewhat consentric layers of flat cells. It occurs most frequently in the meninges but has been found in the brain tissue.

VII. Cysts.

1. Dermoid Cysts:-- These are very rare, arise from the menenges and invole the brain by contact.

2. Parasitic Cysts:-- Such cysts as those produced by the cysteccr cus and the hydatid may be found in the brain or chorcid plexus.

3. Cysts produced by softening:-- These have been referred to fro time to time as with tumors, infarcts etc.. Also in the congenital defect such as porencephalia.

Lecture 4/2/'09.

C. Diseases of the Spinal Cord, Outline of---

I. Anatomical considerations.
   1. Gross Anatomy
   2. Minute Anatomy.
   3. Functions.
      1a. Of the gray Matter.
      2a. Of the White Matter.

II. Malformations.
   1. Amyelia.
   2. Micromyelia.
   3. Diastemyelia.
   4. Unusual length
   5. Heterotopia.
   6. Spina bifida.
   7. Hydro myelia.
   8. Syringomyelia.

III. Circulatory Disorders.
   1. Hyperemia.
   2. Congestion.
   3. Anemia.
   4. Varicose Veins.
   5. Hemorrhages.
      1a. Punctate.
      2a. Massive.
      3a. Hematomyelia,

IV. Inflammations.
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      1a. Hematogenous purulent.
      2a. Myelitis secondary to purulent leptomeningitis.
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I. Anatomical Considerations.

1. Gross Anatomy:__- As we know, the cord is that part of the central nervous system which lies in the spinal canal, well inceased by the vertebra and surrounded by three membranes: the internal pia is more firmly attached to the cord than the analagous mambrane is the the brain; the parietal pia is atta ched to the vesce ral pia but does not dip into the posterior fissure; the dense dura, which is adherent neither to the bany canla nor to the cord. It must be remembered however, that the dura sends extensions out along with the spinal nerves. The cord presents two swellings, the cervical and lumbar and from the latter the cord rapidly narrows to the conus terminalis, this narrowing being produced by the giving off of a great number of nerves forming the chordae equinae. Two sets of nerves are connected with the cord, anterior and posterior, one set of each coming off at the same level = anterior and posterior spinal roots. Section through the cord shows an anterior and a postrios fissure, the latter being the deeper and in the gray commissure will be found what remians of the original central canal of the cord, usually colored and represented by a few ependymal cells. This is only a potential canal.

Contrary to the arrangement in the brain the white matter is on the outside and the gray matter on the inside. The gray maater on cross section roughly represents the latter "H", with two broader anterior cornu and two smaller more ponted posterior horns. The two halves are united by a narrow gray commissure. While anatomically the white matter is uniform and in ordinary microscopical sections of a normal cord no differentiation can be made out, yet physiologically and pathologically we can determine that it is divided into a number of tracts. Close to the anterior fissure comes the direct pyramidal tract; surrounding the anterior cornu is the anterio-lateral ground bundle and this, by some, is further subdivided into a motor and a sensory part: More superficial to this comes the natrio-lateral ascending and descending tract of Gower's; Outside, and somewhat posterior, is the crossed pyramidal tract; Still outside of that and more superficial is the direct cerebellar tract; a little area close to the posterior cornu is the tract of Lasseur; lying next to the posterior fissure is the posterio-median tract or the column of Goll; Between that and the posterior cornu is the postrior-lateral tract or the column of Burdach.

2. Minute Anatomy:-- Roughly speaking, the white matter is made up of axones going too or coming from the brain and the ganglia. In the gray matter of the anterior cornu we have some very large ganglion cells (multipolar) whose axones extend out through and form a part of the -anteri spinal root. From the level of the seventh cervical to the third dorsal is a group of ganglion cells lying in the neck of the postrior cornu and this is known as the column of Clark.

3. The Function.

1a. Of the grey matter; as suggested, the anterior consists of these large multipolar cells whose axones extend into the anterior nerve roots and with these are distributed to the periphery of the body. These cells are concerned with motion, so that any lesion of the anterior cornu will produce a paralysis. Then too, a lesion of the anterior horn gives rise to trophic disturbances, so some of the cells at least must have a trophic function. The posterior horn receives fibers from the posterior nerve roots and ganglia and are concerned more with sensation. The cells of Clark's column give rise to axones which get into the anterio-lateral tract of Gower's and into the posterio-lateral or direct cerebella tract.

2a. Of the White Matter;-- The columns of Goll and Burdach are sensory in function and are physiologically and anatomically connected with the posterior roots and their spinal ganglia, from which most of the fibers of the posterios columns have their origin.

Sensory stimuli, then, from the surface of te body are transmitted first to the spinal ganglia and through them or by them are transmitted along the fibers of the columns of Goll and Burdach, therefore any lesion which occurs between the spinal ganglia and the posterior columns or in the posterior columns will produce a degeneration in the diraction of the conduct of the impulses, therefore ascending. The sensations transmitted here ahve to do chiefly with the sense of touch and coordination in station and gait, therefore any lesion would disturb these. The diract and coossed pyramidal tracts are exclusively motor in their function. Their fibers originate in the motor areas of the brain, so that any lesion in these motor areas or along the course of the fibers from them will produce a degeneration in these tracts. The antrio-lateral tract or the column of Cowers conducts pain and temperature sensations. It shows both ascending and descending degeneration. The dirоct cerebellar tract is, as it name indicates, directly connected with the cerebellumn and conducts sensation which controls the equilibrium. Its fibers show asceding degeneration.

II. Malformations.

1. Amyelia:-- Complete abseene of the spinal cord occurs only with like condition of the brain. It may be represented by a few tags of nerve tissue attached alnong a broad oprn groove in the back.

2. Micromyelia:-- In connection with certain defects in the development of the brain one may sometimes find a spinal cord which is congenitally too small. This is usually due to defects in the development of par ticulat tracts. This may give rise to one form of asymetry of the cord. The usual form os asyemmery, however, arises where there is a failure of the fibers to cross over or decussate form one side to the other , as they should do. But inthis latter case the tot al soze of the cord is ablout normal.

3. Diastemyelia:-- Here we have a congenital condition where a part or all of the cord has its two halves separated, usually encased in separate membranes, sometimes lying in the same bony canla or perhaps even separated the one from the other by a distinct bony pa rtition. A somewhat similar condition, at least grossly, is the truely double cord as is found in the double monsters, but here both cords have theoir two sides complete.

4. Unusual length of the Cord:-- At times there is a congenatal prolongation of the cord tissue into the sacrum.

5. Heterotopia:-- This name is applied to the condition where bits of the gray matter is misplaced i,e, found in unusual sites in the white matter, even on the surface of the cord. In most cases these are connected with the cornu by a narrow stalk and the cornua will not show any material alteration in appearance,

6. Spina Bifida:-- Not infrequently there is a lapsis or failure in development of the posterior part of the spinal canal, usually in the lower lumbar or sacral region. This allows the meninges to bulge through and present as a more or less eminence under the skin, which may be covere thicklu with hair. In the rarer cases this skin covering may be entirely wanting allowing the thick granular membranes to presetn themselves directly to view. If the membranes alone with the fluid pouches through = meningeocele; if some of the narvous tissue comes thorugh aslo = myelocele.

7. Hydromyelia:-- This is a condition in which the central canal is dilated, lined by ependymal epithelia and contains the cerebro-spinal fluid. This is more frequently circumscribed, not extending the entire lenght of the cord and can now be distinguished from other pathological conditions by the character of the lining. Sometimes it presents diverticula out into the cord substance , but these also will be lined by epitheli This condition most frqquently accompanies internal hydrocephalus and is usually congenital, but in some cases it seems to have been acquired. The milder degrees show no symptons but the more marked forms may produce symptons similar to those which will be indicated in syringomyelia. Sometimes the expression "internal hydromyelia" is applied to this condition and an excess of fluid surrounding the spinal cord is called "external hydro myelia".

8. Syringomyelia:-- This is a pathological cavity in the cord not lined by epyndemal epithelia. The cause of such cavities is not known. The most fre quent site is in the cervical enlargement near the central canal and in the gray substance of the posterior part of the cornu. From this situation it is liable to extend into the cornu. The white matte in the vicinity is usually spared but it may be destroyed later as the cavity increases in size.

The cavity contsins fluid which is like the cerebrospinal fluid, even though it may not connect with the central canal. The fissure about the cavity is compressed and the cord is usually flattened. The posterior ar lateral tracts frequently indergo changes. There is a loss of sense of pain and temperature, while that of touch is preservad. This is explained by the fact that the sensations of pain and temperature are conducted along Gower's tract the fibers of which come from the opposite side of the cord and cross in the white commissure and are here involved.

Microscopically:-- We have a cavity surrounded by evidences of proliferated neuroglia and in the zone about this we have braoken down nerve tissue, degenerated ganglion cells, much destruction of the myelin sheaths and some corpora amylacia. The secondary degenerations in the tracts will depend upon the site of the lesion. Sometimes favorable sections will show degenerated changes in Gower's tract above the site of th lesion but these are not manifest in the gross appearance, only microscopically.

Lecture 4/5/'09.

III. Circulatory Disorders.

1. Hyperemia:-- True hyperemia of the cord perhaps only occurs in connection with inflammation. It is difficult to say at the post mortem whether or not a cord is hyperemic. This is due to the ease with which t blood enters and leaves the cord after death.

2. Congestion:- The veins of the cord, especially the posterio venis, at the post morten are nearly always filled with blood, due to the gravity settling of the blood into them because of the position of the ca Therefore, while we may find much blood in the veins, it is not infrequen ly impossible to say whether or not such a condition existed before death We know very little of the causes or results of congestion.

3. Anemia:- This is found in all of the general conditionsin which the blood is too poor in r,b,c, or is anemic. In pernicious anemia we get usually a marked degree which in some cases is diffuse, but seems more prone to affect the posterior and lateral columns. In this pernicious anemia we may get very few symptoms, depending upon the lesions but sometimes they resemble those which are found in combined sclerosis and they are notably irregular in thier distribution. Rather a marked feature of such anemic degeneration is that the grey matter and the ganglion cells escape.

4. Varicose Veins:-- In certain cases groups of veins lying on the surface pf the cord become distinctly varicosed, irrgularly dilated and torotorous, even so much so as to remind one of the vessels found in a plexiform angioma. The cause is not known. Suc dilated vessels may produce pressure and degeneration.

5. Hemorrhages:--

1a. Punctate hemorrhages:- Sometimes in the acute infectic especially of the more severe character, examination of the cord will show numerous small pin point capillary hemorrhages, irregularly distributed. These are of relatively of small importance.

2a. Massive hemorrhages:-- The massive hemorrhages are almost always due to some sort of trauma. In the cord we practically never have a hemorrhage comparable to the cerebral apoplexies. In other words the blood pressure of the cord never gets high enough to produce the so-called spontaneous rupture of the vessels. Such a comparatively large hemorrhage produces destruction of the fibers and the cells of the affected area and secondary degenerations are to be expected. The locati of the hemorrhage is determined by the symptoms which the patient exhibits The healing of these hemorrhagic areas takes place by neurologia proliferation, at times converting the mass into a pigmented scar-like area or perhpas a capsule is formed, the contents later liquified and then it is converted into a cyst.

3a. Hematomyelia:-- Sometimes the blood from a hemorrhage escapes into the central canal of the spinal cord and is then called hemato-myelia. Here the accumulation is usually slow, perhaps due to an ozzin from the vessels, and not to a rapid flow of blood. The blood mass is sually surrounded by eppendymal epithelia, which gives a clue to its location. Here the results are the same as those of hydromyelia, unless there has been destruction of much of the cord tissue.

IV. Inflammations of the spinal cord:-

The boundary line between true inflammations of the spinal cord and the simple degenerations of that substance is by no means clear and distinct and considerable confusion has arisen from this. Where the inflammation chiefly affects the white subs-

tance = Leukomyelitis: if of the grey matter = poliomyelitis; The term myelitis is rather loosely used to indicate sometimes one, sometimes the other and sometimes it is applied to an involvements of both the grey and the white matter. Where the entire thickness of the cord is involved the condition is known as transverse myelitis. If a considerable lenght of the cord is affected = Diffuse; if it is only a small area = focal: if many small areas or foci = disseminated.

1. Leukomyelitis, also sometimes called myelitis.

1a. Hematogenous purulent:-- Here we most typically have an abscess of the cord of multiple abscesses, the infection having been brough through the blood. The formation os a single abscess of any size is of uncommon occurence, and even the occurence of miliary abceses is rare. Either one of these may, however, be met secondary to bronchiectasis or purulent processes in the lungs or in the genito-urinary tract or to a dysentery followed by a liver abscess; sometimes in pyemia, and at times secondary to purulent spinal meningitis. In this last cases, however, abscess formation is not usually the sequence.

Grossly:- The situation of the abscess is most frequently near the central canal, this site being determined by the fact that it is here that the vessels are beginning to get so small that the bacteria lodge in these sites and here too the arrangement of the tissue elements is looser more easily allowing a collection of pus. These abscesses involve some of the grey matter and may also extend into the adjanct white matter. After one has removed thecord and has placed it flat on the table, as the finger is run from one end of the cord to the toher the abscess area is distinguished by a distinctly softened feel as compared to the more resistant sensations imparted by the normalcord. This is by no means characteristic of the bascess alone, for it may be due to degeneration also. When one cuts into the abscess area the semifluid material oozes out an the clean cut is impossible. The area is usually whitish or grayish, containing a semifluid purulent material and the adjacent cord structure is degenerated and softened, perhaps reddish gray from and excess of blood.

Microscopically:-- The degenerated central part , the semi fluid contents, shows many dead polys, the causative organisms, many fat globule remnants of myelin sheaths and compound granule cells. The surrounding tissue shows the usual forms of degeneration to wit; swelling of the axones, degeneration of the myelin, perhaps some infiltration with leucocytes, and if many ganglion cells have been involved they are also degenerated. With these the blood vessels are likely to be distended with blood and the perivascular spaces chpked with leucocytes. Where the destruction is not too great and not too acute, if the pyogenic organisms die out, as they may, the area may be healed by neuroglia proliferation. If, however, the vascular changes are more pronounced, the destruction of the tissue more acutethe connective tissue which forms a part of the prolongations of the pia into the cord and that in connection with the advetitis of the blood vessels begins to proliferate, then the area is healed by the filling in of adult fibrous tissue. This later contracts, leaving a depressed place in the cord. In this respect, the healing by real connective tissue differs from the healing by neuroglia proliferation because the newly formed neuroglia do not show the same tendency to retraction as does the connective tissue. This, however, is about the only gross difference and one can not always tell grossly which form of healing has occurred. Some importance is attached to the character of the material healing such areas in that it is much easier for the regenerated axones to penetrate through the neuroglia than through connective tissue and there is not so much likelyhood that a subsequent contrsction will later destroy them. Where there occur many miliary abscesses these may heal and leave little scar like areas of white sclerosis. Following the abscess formation, if death does not occur, secondary degenerations will follow in the invloved tract.

2a. Myelitis secondary to purulent Leptomeningitis:-- Here, as the name suggests we have an inflammation of the cord substance which has followed a purulent inflammation of the spinal pia. This may be be epidemic, traumatic, ot from any of the causes given for purulent leptomeningitis, nor does the process differ in nature with the various causes, except in degree. The involvement of the cord in these cases is chiefly peripheral.

Grossly:-- The cord appears swollen, red, much too soft and when cut the periphery may bulge too much or even be almost creamy.

Microscopically:-- one finds the vessels full of blood, varying degrees of fattyxxx degeneration of the myelin sheaths, the fibers are swollen and granular, later disintegrated. If many of the ganglion cells are involved, and this is not frequent, they also show a breaking up, first of the tigroid substance, then a solution of the nucleus and finally a degeneration of the cytoplasm, which becomes swollen, cloudy granular or vacuolated from the fat. ith this one finds an excess of leukocytes, while about the only change in the neurologia will be some evidence of proliferation and the glial cells are too numerous. The extent of involvment along the cord as well as the depth to whic it goes will depend upon the degree and the severity of the leptomeningitis and upon the length of time it has been present. In the secondary changes which follow there is likely to be an irregular ditribution, perhaps not at all marked, even in appraently severe cases.

5a. Transverse myelitis without local foci, secondary to injury and acute infections. This is sometimes called acute myelitis. It was referred to here as a transverse myelitis because of the tendency to involve the width of the cord, grey and white matter together.

1b. Etiology:-- Here we have to acknowledge a great many causitive agents. Injury seems to underlie a great many of them. This may be a distinct breaking into the spinal canal, as by a fracture, but it may arise from concussion from a jar or fall or continued jolting. Here we must include some of the cases of railroad spine. It is true that a great many of these conditionscalled railrodd spine are really hysteria or neurasthenia; sometimes absolute fraud: but some of the cases are a distinct myelitis. Here also come cases which occur during the acute infections as small pox, rheumatism, typhoid fever and typhus fever, dysentery, gripp, pneumonia, hydrophobia and gonorrhoea. Sexual excess, alscholism, spurressed menses and exposures to cold have at times been given as the cause, but these portably act only as predisposing causes. Whether acute syphilis can produce this condition is still a disputed question. Certain cases do seem to arise, however, in the aarly stages of syphilis and present a clinical picture loke those of other cases of a cute myelitis and thus seem to depend upon sylhilis as the cause.

2b. Morbid Anatomy:-- The extent of the distribution as well as the general appearance of the cord will vary remarkably in different cases. The lesion may show in one segment of the cord or extend over several segments. The part is distinctly soft, especially to the touch. If the process has been very ecute the substance is almost fluid, perhaps of a chocolate or red color from the admixture of degenerated blood. But if the process be slower the area is likely to be grayish white or red and semisolid. On cross section of the fresh cord the edges about the affected area bulge too much and the differentiation between the gray and the white matter is more difficult.

Microscopically:-- Varying degrees of degeneration are met in the myelin sheats, nerve fibers, neuroglia tissue and ganglion cells. These changes are much the same as those previously described under the degenerations and of very like those met in acute encephalitis.

A special word about the myelitis in rabies is probably in order here. Two general classes of cases occur. In both the vesse ls are filled with blood and some hemorrhages are seen. One form, however, shows a very marked hyperemia, especially noticable in the gray matter but is al so seen to some extent in the white matter. With this there are leukocytic accumulations along the course of the vessels toward the central canal. These accumulations of leukocytes constitute the rabic tubercles. In the second form the hyperemia is less marked but the softening of the tissue is more pronounced. Here, also, we may have some hemorrhage, as from an eroded vess l and these hemorrhages are likely to be most prominent in the posterior horn and about the central canal but they may also show in the anterior horn. Similar changes to these are also found in the meddulla.

3b. Results of Transverse Myelitis:-- The amount of secondary degeneration is variable and will follow the distribution of the chief lesion. Healing by neuroglia proliferation may occur.

4a. Pressure Myelitis:-- Here we have a myelits due definitely to the pressure of a tumor, an eroding aneurysm, cysts, misplaced vertebrae as in caries of the spine or in fractuer. This myelitis is usually not an acute process ,save some of those cases due to the pressure of a fractured spine. The pressure produces a destruction of the cord under it and the microscopical picture here is that of the degenerwtions already described

except that we are apt to have a prominent proliferation of the neuroglia cells and an increase of their fibers.

The important results are that the axones are cut off from their ganglion cells and thus we get ascending or descending degenerations, depending upon whether it be sensory or motor tracts upon which the press is exerted.

2. Acut Anterior Poldomyelitis.

This condition is an inflammation of the anterior horns of the gray matter of the cord. Because of its greater prevalence in children it is sometimes referred to as infantile paralysis or the essential paralysis of children.

1a. Etiology:-- As just suggested, it is a condition almost entirely confined to children, usually about the age of the second dentition. But a certain number of cases has been found in adults and as some claim, in lower animals as well. A Peculiar feature is that while a great number of cases seem to arise sporadically, yet it may develop onto a distinct epidemic. For this reason it is thought to be an infection of some sort but a definite specific organism has not been found. The diplococcus intracellularis meningitimus has been isolated from the cerebrospinal fluid in some of the cases. But in other cases other organisms have been found and in many of them no organisms at all are discovered. The distribution of the lesion makes it extremely probable that the infectious agent is carried through the ventral system of blood vessels.

2a. In considereing the morbid anatomy it is more convenient to describe the cord first at an early stage during the height of the disease and then much later when the secondary degenerative changes have taken place. As already indicated it is the anterior horn of one side which is chiefly affected. This may be only throuhout one segment of the cord but frequently it is of much wider distribution even along the whole length of the cord. The favorite sites seem to be the lumbar and cervical enlargements. In its earlier stage when the cord is removed the pia over the affectedarea is hyperemic and this part of the cord has a softened feel. On section through it the entire gray matter seems hyperemic and s/a small petechial hemorrhages are seen in the affected hron. Even in the fre fresh cord and at this stage, the horn may seem too small and this is especially brought out if the cord be fixed for some time in Muller's fluid Along with this decrease in size the distinction between the gray and white matter at this part is to a considerable extent lost. However, if it is properly stained microscopical sections will show the tru nature of the process. The blood vessels going to the anterior horn are full of blood, their perivascula spaces packed with a zone of lymphocytes. The punctate hemorrhages will be found in the severer cases. Marked changes will be see in the ganglion cells. Many of these are shrunken, distorted in outline; thier nuclei eccentric or breaking down: the tigroid substance is much brok enup; disarranged or entirely gone and many of these degenerated ganglion cells are surrounded by a zone of small lymphocytes. Other ganglion cells in the region may be swollen and cloudy, theri nuclei gone and their shape is lost. The dentrites of the degenerated and degenerating cells are lost, their axones are varicosed and their myelin sheaths are degenerated and fatty. This degeneration of the axones extends throughout their course, so that a peripheral nerve degeneration is to be expected. In the anterior horn a certain number of the ganglion cells and their ansones will remain normal.

In the older cases the anteriorlateral aspect of the cord over the original site of the involvement is distinctly smaller than the opposite side and the anterior spinal herve roots which come off from this part of the cord are too small. When the cord is cut through here the decrease in size of the anterior horn is plainly seen, at times it seems entirely gone.

Microscopically:-- Section now shows very few ganglion cells, while the area is filled in with proliferated neuroglia and connective tissue. The blood vessels which are left are thick walled but their lumina are distended. The nerve fiber s which should originate in this ap part are gone or represented only by very slender delicte lines. Alon, with the above description it must be remembered that the ganglion cells of the anterior horn are not destroyed uniformly,i,e:certain cell groups seem more prone to involvement and are therefore affected very constantly. On the other hand certain groups seem to escape, notably that column which is situated toward the inner anterior tip of the hron and which controls the muscles of the back, These are nearly always preserved so that in the paralysis and atrophy the muscles of the back may remin in good condiditon. Following such changes in the cord as the above certain secondary changes depenent upon them are to be expected.

At times there is a distinct atrophy of parts of the motor areas of the cerebral cortex, perhpas resulting from disuse. The muscles supplied by the fibers originating in the degeneratied horn undergo trophic chnages: a degneration and atrophy, and frequantly a replacement by fat, either as a distinct fatty degeneration, but more frequently distinct adipose tisuse. The bones ad the blood vessels of the part, may after a time, undergoa distinct atrophy, but this is probably from disuse. The muscles remain flaccid and paralyzed but electrical stimuli show reactions of degeneration.

Results:- Acute anterior poli myelitis does not frequently kill. The clinical symptoms consist of a progressive atrophy of certain of the muscles, the situation of which depends upon the location of the lesion. A certain amount of recovery is possible, not through the regeneration of the ganglion cells destroyed, but through the adaptation of another part or of other cells. The atrophy and degeneration of the muscles produces contractures of various sorts.

3. Acute ascending paralysis or Landry's paralysis.

This is a name given clinically to a group of cases in which the paralysis develops and extends upward, that is the muscles of one leg are first affected, then those of the other leg, and so on until the muscles of respiration perhpas degulition are affected, when death occurs. The basis of this process is an acute parenchymatous degeneration of toxic or infectious origin, involving the peripheral motor neurons, indeed it is a form of neuritis.

1a. Morbid anatomy:- This is very variable. At times there is such an involvement of the anterior horns spreading upward, as to suggest the name acute ascending poliomyelitis. This condition is not always seen. Along with this inflammatory changes are likely to be met in the grey matter of the cord, medulla, pons, cerebellum and cerbrum. Various degeneration in other organs of the body as t e liver, kidney, heart, etc, occur so constantly that they would seem to indicate that the condition is due to a general systemic toxemia, which for some reason, unknown, has centered upon the central motor neurons.

Recovery can result but death usually ensues by involvement of the muscles of respiration.

V. Primary degnerations of the psinal cord. Here we have a more or less complete degeneration of certain systems of axones or tracts of the cord, affecting the whole length without a distinct injury severing its axones and sometimes without a primary degeneration of the ganglion cells which give rise to the fibers. The tracts affected are thosw which conduct impulses to and from the brain.

1 Posterior Sclerosis, also known as tables dorsalis, locomotor ataxia. The terms posterior sclerosis and tabes dorsalis are misnomers and were given to the disease at a time when it was thought that the sclerosis of hardening of the posterior column was the primary lesion. Locomotor ataxia is simply a clinical name given because of this comon clinical feature. Posterior sclerosis, then is a disease in which the peripheral sensory neurons slowly degenerate.

1a. Etilogy:- Tabes dorsalis is found much more frequently in man than in women, (10 to 1) , its age of preference is between 30 and 40 years, rarely over 20 and usually it has distinctly developed before the age of 50. The definite underlying cause is not known but syphilis is present in by far the greater number of cases, the percentage being variously given from 50 tp 90% pf all cases. Some observers hold that tabes dorsalis never arises in a patient who has not had or does not have syphilis. It arises somewhere between 6 and 15 years after the primary lesion. The lesions are not distinctly syphilitic in nature or in histology and may be due to the toxins which have arisen as a result of the general metabolic disturbances due to the syphilis. Exposure to cold, over exertion, excessive use of alcohol, excessive sexual indulgence, probably act only as predisposing causes.

2a. Morbid Anatomy:--The appearance of the cord both grossly and minutely will vary with the age of the lesion. In some cases it is the dorsal portion of the cord, i,e: that part of the cord which corresponds to the thoracic vertebrae, which is chiefly involved. In other cases the greater part of the lesion is seen in the dorso-lumbar region and again one may find that the chiefest amount of involvement and the earlierst is in the cervical part of the cord, producing the socalled high or cervical form of tabes.

In order that the lesions found in posterior sclerosis may be some what more intelligible let us consider as briefly as possible some of the facts about the posterior spinal ganglion. Let it be remembered that posterior sclerosis is not a lesion primarily of the cord, but it is an affection of the peripheral sensory neurons and that the degenerations met in the cord are secondary to this. From the ganglion cells in the posterior spinal ganglia comes off a single process which soon divides into two branches, first; a peripheral brainch which extends out to the periphery of the body, skin muscles and koints. This is really the dendrite of the ganglion cell and transmits impulses-to the cell ganglion cell and it is the only instance in the body where a dendrite becomes medulated. The second, or central portion of this process, the real axone of the cell, conducts impulses away from the ganglion cell. This central process enter the spinal cord proper in the postero-lateral portion at or near the posterior horn, then it divides again into two processes, one a short descending branch which descends in the cord for a segment or two, and another longer ascending branch whose final distribution will be given later. As soon as this axone enters the cord it gives off a prominent collateral which enters and makes up the colum of Lissauer. Remmember that in this column of Lissauer we have it divided into two parts by the posterior limb of gray matter, so that a portion of it lies external to the posterior horn and if degenerated, as it frequently is in posterior sclerosis, may confuse one into thinking that we have a degeneration of the lateral column. The main central part, the ascending branch of the axone in the lower part of the cord; i, e, those arising from the sacral and lumbar posterior roots, pass quickly through the column of Burdach and constitute the greater exogenous part of the column of Goll. Just here, it is well to call attention to the fact that practicaly all of the tracts of the cord are made up of exogenous fibers or fibers which arise some where out side of the cord and endogenous fibers, those arising within the cord, chiely from the ganglion cells of the gray matter. In the dorsal region these central fibers for a time traverse the column of Burdach particularly just inside the posterior horn, then pass into the column of Goll and run along its posterios portion. In the cervical region, especially high up, these central axones remain in the column of Burdach and finally arborize around the large cells in the cuneate nucleus. The fibers which have come up from the column of Goll in a similar manner arborize around the cells in the nucleus gracilis. Throughout theri course both the ascending and descending branches are constantly giving off collaterals which help tp increase the size of the posterior tract. Now while the distribution just mentioned for the central part of theis axone will hold for the longer branches, there are shirter ones from other cells in the spinal ganglia which enter the gray matter and arborize about the ganglion cells.

ith these points in mind let us consider somewhat the gross and minute features of a case of posterior sclerosis. In the early stage differs materially from that seen in the very late stage of a long standing case.

The earlier cases:-- On removal the dura is normal but the pia is usually somewhat thickened over the posterior columns in the area chiefly involved and it may be somewhat adherent but this is not a common feature. The posterior column may feel a bit too hard and on proper fixation in Muller's fliud some areas of degeneration show up as grayish patches. In the early stage these areas show up as different parts of the posterior column at different levels of the cord.

Proper microscopical preperation will better reval the exact part involved. In the dorso-sacral region the column of Lisauer will be completely degenerated. In the dorsol portion two areas of degeneration will show:--One in the posterior portion of the column of Goll, another in the column of Burdach just internal to the posterior horn. In the cervical region the degeneratio n will be found almost confined to the column of Goll, especially its outer and posterior part. In this description we have chosen a case where the lesion has involved especially the dorso-sacral periperla sensory neurone. In the case of High or cervical tabes, while the column of Goll may show some degeneration, we are quite likely to have the column of Burdach especially degenerated.

Choosing now a case of long standing:-- The cord on removal will be distinctly flattened, the degenerwtion of the posterior columns very marked, practically complete. The spinal pia often shows as a dense white band over the posterior column, it is quite adherent and this part of the cord (posterior column) feels distinctly hard. On section it is grayish red in contrast to the white color of the other parts of the white matter of the cord and microscopical section will show degeneration to have involved almost all of each of the posterior columns. A neuroglia and connective tissue proliferation gives us the sclerosis, which originally named the disease.

disease. Two parts of the posterior columns are likely to escape except in the extreme or very long standing cases. One of these is at the anterior part of the column of Goll, lying next to the posterior commissure and made up chiefly of endogenbus association fibers. The other areas, a small triangular area, lying just over the pia posterioly and next to the posterior median fissure. Careful examination, even in the early stage, reveals changes in the posterior spinal ganglioa, consisting of a degeneration and atrophy of the ganglion cells. With these degenerations will be seen in the peripheral dendrites of these cells. We are now ready to consider the fundamental changes in tabes dorsalis. Three possibilities present themselves:-

(a). The primary fundamental change may be a degeneration in the peripheral dendrites or the peripheral nerves. If these be degenerated, impulses no longer are carriod through them to their cells. So the cell itself may undergo changes as a result of which the central axnoe degenerates.

(b)The ganglion cells of the posterior spinal ganglia may degenerate primarily. Resulting from this there will naturally follow a degeneration in these processes.

(c) This locat s the lesion in or about the posterio nerve root, between the spinal ganglia and the spinal cord. Each one of these has its adherents. It seems to me that the bulk of evidence tends rather to point to the peripheral degeneration as the fundamental change.

Results:- (1). The Agryll Robertson pupil= loos of accomodation to light but not to distance. (2) The lightening or lancenating girdle pains and hot flushings. These are reflex. (3). Loss of tactile sense and the inability to make accurate movements, he sways when his eyes are closed and ealks stepping high as though in cotton or in feathers. (4) A loss or disturbance of the reflexes. (5). Usually late various trophic changes as dystrophies or arthropies etc, occur.

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2. Friederich's Ataxia, or hereditary Spinal ataxia.

1a. Etiology:- The cause of this disease is not known. In some cases it seems to have been inherited or probably a better way of stating this is that it is prone to attack more than one member of a family, and to occur in successive generations.

2a. Morbid Anatomy:- The typical cord is much too small, especially in the cervical region and this diminution in size seems to effect all the constitutents.

Microscopically: there is a degeneration found in the posterior and in the lateral fracts;the degeneration being especially complete in the columns of Goll: frequently complete in the Crossed Pyramidal tracts and in Clarke's column, while a considerable part of the direct pyramidal tract and the columnof Burdach is still in tact. The column of Gower's may be much involved. Atrophic and degenerative changes may occur in the anterior horns.

3a. Results:- In early childhood or infancy there begins to occu movements of a choreic nature:nystagmus, disorders of speech: trembling: lack of co-ordinationand often a lateral curvature of the spine.

3. Amyotrophic Lateral Sclerosis:-

This is a primary progressive degeneration involving both the cortico-spinal and the spino- peripheral nerves, e.g., degeneration will be seen in the motor or pyramidal tracts and in the anterior horns.

1a. Etiology:- The cause is not known.

2a. Morbid Anatomy:- There is a degeneration in the fibers of the pyramidal tracts, more especially the corssed pyramidal, hence the name lateral sclerosis, and secondarily a degeneration in the ganglion cells of the anterior horn and their corresponding nerves.

Grossly:- In a weel established case of amyotrophic lateral sclerosis the cord is too small, especially along the cervical enlargement. The dura undergoes no change except perhaps a thickening late in the disease: the pia also is fairly normal, exceptit may be too adherent. The cord feels distinctly hard and on section of a fresg cord the lateral pyramidal tract are grey or light grey instead of white. The anterior horn will probably show no gross lesion except that it may be too soft and too red. The changes thus mentioned may be followed up into the oblongata. The Hypo-

glossal and Glossopharyngeal nerves may be small and shrunken and like changes are to be expected in the naterior nerve roots. Also one sees a muscular atrophy with a deformity and fixation of the joints.

Microscopically:- Proper preparation and staining will show a degeneration in the crossed pyramidal tract, but the neruologia tissue is very little increased. In certain cases some degeneration may me manifest in the columns of Goll, thus approaching the type of combined sclerosis. In these sections the general size and shape of the anterior horn are kept, but many of the cells are degenerated and the tissue is too loose. The axones are small and shrunken. Not infrequently careful study will reveal a degeneration of some of the pyramidal cells of the cerebral cortex, thos of the motor area. The axones of these diseased cells will also be degenerated. When this occurs a certain amount of degeneration will show also in the internal capsule.

Changes in the muscles originally controlled by these diseased fibers are those of simple atrophy with a substitution of adipose tissue for the muscular substance. Sometimes, however, the change in the muscle fibers is that of a fatty degeneration. Atrophic or completely degenerated muscular fibers will be found side by side with others unchanged.

3a. Results:- There occurs a muscular weakness, fibrillary twitching, later on atrophy occuring first usually in the arm, later a weaking and spastic condition of the legs. The tendon reflexes are increased and Babinski's sign is present. If the medulla is affected then we have a Glosso-Labio- Pharyngeal or bulbar paralysis, but this usually occurs late.

4. Primary Lateral Sclerosis or Spastic Paraplegia:- Similar to the above, Amyotrophic Lateral Sclerosis, is this spastic Paraplegia, but here the corssed pyramidal tracts are involved without the disturbance in the anterior horns. It has been denied by some that such a condition exists, wile still others hold that this is but an early stage of amyotrophic form. There are two general groups of these cases:- (1). Infantile or congenital (2) The adult form.

(1). In the infantile of congenital form we have a partial or complete absence of the motor part of the cerebrum, an early or congenital hemorrhage into the motor area or internal capsule. Following this the lateral pyramidal tract is degenerated and some involvement is seen in the direct pyramidal tract. It is to be noted here that the degeneration may be confined to one side, producing a hemiplegia and to all intents and purposes the lesion produced is simliar to that which will be described under the degeneration secondary in origin. In such cases there is very little tendency to the proliferation of the neuroglia and so the cord is distinctly too small especially about the lateral tracts.

RESULTS:-- The infant early shows a spastic contraction of the legs and of the arms and legs, later more distinct cerebral symptons as strabismus, epileptic attacks etc.

(2) In the adult form the on come of the trouble is very insiduous and there is a slow degeneration of the direct and crossed pyramidal tracts. Here the disease usually occurs between the 20 and 40 year and has been variously attributed to syphilis, injury, poisoning and infectious diseases. A gradual muscular weakness with spasticity, Babinsky's sign, increased reflexes occurs but with no involvement of the rectum and bladder and no sensory disturbance.

5. Combined sclerosis or Ataxic Paraplegia or Combined Posterior Lateral Sclerosis:-- Here there occurs a degeneration in the posterior and lateral columns.

1a. Etiology:-- Concussion, injury to the spine, pernicious anemia, syphilis, lead poisoning, and even malari a have been assigned as causes. 2a. Morbid Anatomy:-- The cord is distinctly too small, the diminution in size benig chiefly over the posterior and lateral portions. This part is distinctly harder than it should be. On fresh section, the degenerated columns are gray or grayish whits. The tracts more frequently involved are the columns of Goll and Burdach, Clark's column and consequently the direct cerebellar tract and also the crossed pyramidal tract. Thus we have a combination of the lesion of tabes dorsalis and lateral sclerosis, however, the involvement of the posterior columns here is rarely so great as in tabes dorsalis and also the process usually begins in the pyramidal tracts and later involves the posterior ones.

3a. Results:-- It begins with a spastic paraplegia as under the primary lateral sclerosis, then later there are the shooting pains,

the ataxia and other symptoms of ordinary Tabes.

6. Chronic Anterior Polio-Myelitis.

This is sometimes called Progressive Muscular Atrophy of the Aron-Duchenne type. Some authors hold that a distinction should be drawn between the progressive muscular atrophy and chronic anterior polio myelitis, but in all these cases there is a usual and important involvement of the anterior horns with a degeneration of their ganglion cells, with the axones and axiscylinders. So that cilinically an antemortem diagnosis is praactically impossible and even anatomico examination of the cord may show no difference. So that for practical purposes one may consider them the same process.

1a. Etiology:- This condition may follow prolonged muscular exertion, concussion, perhaps occurs late, many years after a distinct attacj of the acute Anterior Polio Myelitis. But with all these the etiology is quite obscure.

2a. Morbid Anatomy:- The size of the cord is practically unchanged as a rule. Fixation in Muller's fluid may show an alteration in size and shape of the anterior horns, but not necesarily. Some hold that when this change does show that it is chronic anterior polio-myelitis and where it does not show = progressive spinal muscular atrophy.

Microscopically:--The ganglion cells are atrophic and degenerated, showing much too few ganglion cells to the section. This change usually begins and is most marked in the upper dorsal and cervical regions and from here spreads up and down. It may involve the nuclei in the medulla, producing symptons of bulbar paralysis. With the degeneration of the ganglion cells of the anterior horn there follows a degeneration of theri axones and then the muscles atrophy, first degeneratin and being convertad into a fatty detritis, perhaps only a fatty sarcolemma remains. Some times the atrophy is simple and a lipomatosis occurs.

3a. Results:- These begin slowly with a weakness and a flaccidity, usually of the smaller muscles of the hand, then the arms, the the shoulders and the trunks. It may later involve the diaphram or those muscles supplied by the Vagus, the Glossopharyngeal and Hypoglossal nerves and thus end fatally.

VI. Secondary Degenerations of the Spinal Cord.

When there is a separation of the axone or dendrites from their corresponding ganglian cells or whenever the ganglion cells die, these axones and dendrites degenerate. If it is due to a definite solution of the continuity, either degeneration of by actual truama, the degeneration occurs distinctly in the direction of cunduct of the impulses. In certain cases, however, where the lesion is toxic, the first sign of the degeneration seems to occur in the part of the process fartherest removed from nutrition, most distant from the ganglion cells and gradually extends to the cell. This, however, does not seem to be constant. From the statements just made and keeping in mind the constitution of the tracts of the cord, it will be seen that we have an ascending and a descending degeneration, depending upon whether it is sensory or motor axones or cells involved.

1. Descending degeneration of the Cord.

This will be found in the pyramidal tracts. It is possible, however, to find a lesser degree of descending degeneration in the tract of Gowers. If the lesion be in the motor area of the cerebrum, in the coron radiata, in the internal capsule, in the pons, or in the medulla there will be a descending degeneration of the direct pyramidal tract on the same side as the lesion and a degenerwtion of the crossed pyramidal tract on the opposite side. While this general statement holds true, more careful examination has shown that there is a degeneration of some of the fibers of the lateral pyramidal tract on the same side as the centr lesion, thus proving that all of the fibers do not decussate. The lesion which produces such a secondary degeneration may be hemorrhage, directly into the substance of any of these parts or pressure upon them by tumors, hemorrhage, abscess formation, etc. Late in such a descending degeneration there may occur some changes of the nature of a simple atrophy in the ganglion cells of the anterior hron. Now suppose we have a lesion which involves the transverse axis of the cord at some place as a transverse myelitis, injury, tumor, or fractured bone, it is evident here that both motor and sensory cells will be cut off from their cells, so that below the lesion we have a descending degeneration in the direct and crossed pyramidal tracts, sometimes also a careful examination will show some descending degeneration in a little comma shaped tract lying at the inner

side of the column of Burdach.

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2. Ascending degenerations of the spinal cord:- These affect the columns of Goll and Burdach, these fibers being cur off from the posterior spinal ganglia. There will also be an ascending degeneration of somewhat variable extent in the direct cerebellar tract because these fibers are cur off from the cells of the columns of Clarke, and this degeneration here may be traced as high up as the restiform body of the Oblongata. Suppose the injury has occured in the Corda Equina, either from injury in the lower lumbar of Sacral vertebrae or perhaps due to a tumor formation, then a secondary ascending degeneration will occur dur to the cutting off of the fibers from the ganglion cella of the posterior Spinal Ganglia. This resembles very much the degeneration in Tabes Dorsalis . In the Lumbar cord almost all of the posterior columns, except a small median zone and a part close to the posterior commissure, are degenerated. This degeneration gets smaller as we go higher, until it is almost wholly confined to the column of Goll.

VII. Infectious Granulomata.

1. Tuberculosis of the Cord:- T,b,c, of the cord substance itself is quite rare. In some cases where there has occured a t,b,c of the meningese, the periphery of the cord is involved by direct extension, producing a t,b,c, meningo-myelitis. This probably the most frequent t,b,c involvement of the cord, since the meningese may be involved themselves by a direct extension of such a t,b,c, involvement of the spine in Pott's Disease. However, in addition to this t,b,c, meningo-myelitis, we may find solitary tubercles or multiple miliary tubercles.

Solitary Tubercles:- This has usually come about from t,b,c, of th meningese but it is so localized that one does not give to it the name tuberculous meningoencephalitis. It rarely reaches a large size. It presents practically the same appearance and inflammatory reaction about it as that of tuberculosis of the brain. It may produce a subacute myelitis with destruction of the tissue involved directly, in which case we would expect a subsequent degeneration.

Miliary form:-- /// In miliary tuberculosis of the cord the bacteri are brought to the blood by the blood stream and it is usually found in connection with tuberculosis elsewhere, most frequently a genaral miliary tuberculosis, however, a miliary tuberculosis of the cord alone hae been described. In this form the gray matter of the cord is involved by preference because of the distribution of the blood vessels. We have produced a form of acute myelitis. In addition to the small tubercles of charscteristic histology there is an infiltration of leucocytes along the perivascular lymph spaces, proliferation of the neuroglia, with some degeneration of the ganglion cells, either in the anterior or posterior horns. Careful examination may show a small amount of secondary degeneration but this is usually not marked. It cannot be made out with the naked eye because these cases usually do not last long enough for extensive secondary degenerations to occur. Nor is the distribution of these seconda degenerations regular, because of the irregular placings of the little tubercles.

2. Syphilis of the Cord:-- Definite gumma may be found in the cord substance. They are rare and are very apt to be accompanied by changes in the pia. Such a gumma shows as a grayish white area followed by a secondary degeneration. They present the usual microscopical features. The most frequent syphilitc lesion of the cord is a chronic syphilitic meningeo myelitis in which the major part of the involvement shows in the menenges and in the blood vessels. The involvement of the blood vessels mau be circulscribed or diffuse. The circumscribed areas will probably be surrounded by small areas which are very like gumma or at best the early stages of gumma. On the other hand, the vese ls may be involved diffusely, one part showing advanced stages of degeneration while another part will show active cellular proliferation. While the arteries are shiefly affectd the veins are also involved.
As to 4esults:ef this there is a thickenig and obliteration with or without thrombosis or a thinning and dilatation with an aneurysn which may rupture later. The involvement of the pia in these cases is especially likely to be most prominent in the cervical or in the dorsal region. The pia is thick and adherent, either diffusely or losally and may show the degenerative changes of gumma. When one attempts to remove it from the cord it tears the substance of that part and then shows as a thick tough dirty white membrane, the inner part rough form the torn tags of tiss tissue. In the cord itself one finds the periphery distinctly harder to

the touch, white, and microscopical examination shows the connective tissue extension of the pia much thickened and with this one sees the alterations in the blood vessels described above. There may be a swelling and degeneration of the compressed nerve fibers. The myelin sheaths may show fat droplets and the neuroglia be somewhat increased. These secondary degenerations vary with the extent and location of the part involved. Along with this the spinal nerve roots, both anterior and posterior, are quite apt to be compressed by the thick pial extensions about them.

Results:--One may have few or many symptons depending upon the extent of the lesion. These disturbances are apt to be a combination of motor and sensory ones with perhaps an involvement of the rectum and bladder and especially is this true if the involvement of the cord in a transverse axis has been sufficient to approach the type of a transverse myelitis.

VIII. Tumors of the SPINAL CORD.

1. Glioma:-- This is the most important primary tumor of the cord. It is apt to be of the infiltrating form, elongated, and extending over several segments of the cord, at times involving the nerve roots, making them larger and harder. The growth of such a tumor in the cord is apt to produce curious distortions in shape and perhaps looks something like an hypertrophy. Otherwise the glioma of the cord resembles both grossly and minute ly the glioma of the brain.

2. Primary Sarcoma of the Cord:-- This is a very rare tumor. It may be a simple sarcoma or what is aclled a gliosarcoma.

3. Secondary Malignant Tumors:-- Both cancers and sarcomas occur but they are not apt to reach a large size.

Parasitic Cysts:-- Due to the Echinococcus and Cystecirrus may occur in the spinal cord but they are rare.

The Peripheral Nervous System: Outline of:

A. The Canglia of the Cranial and Spinal Nerves.
   I. Anatomical Considerations-.
   II. Degenerations.
      1. Of the cranial nerve ganglia.
      2. Of the Spinal narve ganglia.
B. Peripheral Nerves.
   I. Anatomical considerations.
   II. Circulatory Disturbances.
      1. Hyperemia,
      2. Hemorrhages.
      3. Edema.
   III. Atrophy.
   IV. Degenerations.
   V. Regenerations of injured nerves.
   VI. Inflammations or Neuritis.
      1. Acute Interstitial.
      2. Chronic Interstitial.
      3. Parenchymatous.
         1a. Poly neuritis.
   VII. Infectious Granuloma.
      1. Tuberculosis.
      2. Syphilis.
      3. Leprosy.
   VIII. Tumors.
      1. Neuroma.
      2. Sarcoma.

A. Ganglia of the Cranial and Spinal Nerves.

I. Anatomical Considerations. The general make up of the ganglion of the cranial nerves and those found in connection with the posterior spinal roost is essentially the same. Surrounding these ganglia is a fibrous capsule whis is a continuation of the perineurium of the nerves which aenter and leave the ganglia. Within this capsule the ganglion cells are arragged somewhat in layers or systems and consist of three general types of cells: by far the greater number of then are the unipolar cells, having one process which soon divides in a T or Y shape. One of these divisions constitutes the peripheral portion, really a dendrite, and medulated, however, and transmitting impulses from the periphery of the body to this ganglion cell. The pther proces, in reality the axone, also medullate, conducts impulses away from the cell, enters the spinal cord alo g the posterior nerve roots, there divides into two branches, one short descending branch going for a segmant or two; the other longer, ascending and variously distributed, chiefly to the posterior column. An important collateral from this ascending branch enters and helps to form the chief part of

column of Lissaur. The second type of cell, also unipolar, has a short process which does not leave the ganglion. The third type, scant in number, is an ordinary multipolar cell very much like the cells of the sympathetic ganglia. The axones which enter these ganglia arborize about the ganglion cells. The processes conduct sensory impulses.

II. Degeneration of the Cranial Ganglia.

1. These ganglia may be involved by direct extension of a neighboring process or may become degenerated as the re sult of an ext sion of a degeneration of the peripheral sensory axones. In a number of cases persistent trifacial neuralgia the gasserian ganglia has been found much changrd. The myelin sheathes and nerve fibers degenerate, the ganglion cells atrophy and the connective tissue is so proliferated as to give rise to a distinct sclerosis. Similar changes are occasionally met in t other ganglia of the cranial nerves.

2. Degeneration in the Spinal Ganglia:-- This occurs in tabes dorsalis and its connection there has already been suggested. These ganglia may also be involved by a direct extension of such processes as tubercular cares of the spine, extension of tumors etc.. Then, too, distinct changes have been found in these ganglia in herpes zoster, probably of a causative nature. In large spinal ganglia have also been met acromegal but the relation is not understood.

B. Peripheral Nerves.

I. Anatomical Considerations. The parenchyma of a peripheral nerve is the bundle of axones from ganglion cells. These may be either direct extension from the pyramidal cells of the cerebral cortex or from the cells of the basal ganglia, or form the cells of the gray matter of the cord or processes from the posterior spinal ganglia. In addition to this many of them contain or may be entirely made up of the socalled sympathetic fibers from the ganglion cells of the sympathetic ganglia. The neuraaxone in the medullated form is surrounded by the myelin sheath, then the neuralemma, a delicate mambrane, in which certain nuclei appear a fairly regular intervals. These are gathered into little bundles surrounded by the endoneurion (connective tissue), these into larger bundles held together by the perineurion and the entire nerve enclosed by the perineurion. The axis cylinders have various terminations in the periphery of the body, the sensory ones their own perculiar nerve endings, and the motor ones to the muscle fibers by a different character of ending.

II. Circulatory Disturtances.

1. Hyperemia: hyperemia of the peripheral nerves occurs in the acute inflammations and follows injuries.

2. Hemorrhages:- These usually occur from direct injury, perhpas from extreme distension of the blodd vessels and in certain of the infectious diseases with a hemorrhagic tendency. The importance lies in the compression that they produce or in subsequent organization by fibrous tissue which may retract and compress the nerve fibers.

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3. Edema:- This occurs in the nerve trunksas they traverse areas of inflammation. It may be sufficient to produce pain from the compression of them.

III. Atrophy:- This is very closely related to degeneration. The atrophy may occur from pressure, as by tumors, cysts or at times from chronic inflammatory changes.

IV. Degeneration:- as suggested before whenever an axone is cut off from the ganglion cell to which it belongs there occurs a degeneration in the direction in which the the axone transmits impulses. In addition to this a certain amount of degeneration occurs in the direction toward th axone's ganglion cell. This extends back to the nearest uninjured node Ranvier. The amonunt of ba kwaed degeneration which occurs will vary with the amount of injury and the character of the injury to the nerve trunk. If it be a clean cut incission very little backward degeneration occurs. If, however, the injury e xtends over a greater area as in cauterization, injury, or compression, the amount of this retrograde degeneration will be greater, nerver extending, however, further back than the first injured node of Ranvier. The peripheral degeneration extends to the uttermost distribution of the axone. The first change in the nerve can be made out only by closr study. It consists of the breaking up of the axone into a number of minute fibrillae with a scattering of these about in the myelin.

About this time, within 36 to 48 hours, the myalin is found to be broken up into fat droplets and soon the last vestige of the axone is gone, with only the neurelemma left. Much later little /////// concentric bodies like the corpora amylacia, only smaller, are to be found.

V. Regeneration of Injured Nerve:-- Taking as a type the regeneration of a nerve which has been severed, one finds that in between the cut ends ther is first a hemorrhage, a clot, then becoming organized by the growing in of the connecti e tissue epitheliods, their elongation into fibroblasts, an d the penetration of capillary blood vessels. At the same time phagocytes come in and begin to remove the debris and of this debris an important part is the fat droplets which have resulted fro m the degeneration of the myelin. At the same time the nuclei of the newrelemma beg begin to multiply, this change being most pronounced in the proximal end o of the nerve. The cytopasm of these cells elongates and occupys a position of the former myelin within the neurelemma. At the same time the axones begins to elongate from its proximal end, pushes through the cytopasm of the proliferating neurolemma cells on through this young fibrous tissue until it beaches the old neurolemma when it rapidly proceeds to the further extremity of the former nerve fiber. As this axone penetrates through the cytopasm of the proliferated neurolemma cells it undergoes a differentiation into myelin and new neurolemma where necessary, i,e, between the cut ends of the nerve fibers. Thus the continuity of the fiber is restored and it may again resume its function, later, however, the contraction of the newly formed fibrous tisuue may by the pressure produce a subsequent degener ation or pressure atrophy of the nerve fiber, thus explaining some of the cases where the function has been restored only to be lost again.

VI. Inflammations and Neuritis.

This may be either acute or chronic and it is somewhat arbitrarily divided into interatitial and parenchymatous neuritis, depending upon whether it is the interstitial connective tissue or the nerve fiber primarily involved. At times an etiological division of neuritis is made, into the traumati c, toxic, infectious and chachectic. In the latter division, however, the lines are not closely drawn and the division is a clinical one rather than pathological. The inflammation may invole one nerve or many ne ves, the latter condition being called poly neuritis" or "multiple,neuritis".

1 Acute Interstitisl Neuritis.

1a. Etiology:-- It may arise from the direct extension of a nearby pathological process of an inflammatory nature: from trauma: exposure to cold; at times it occurs apparently from the action of rheumatic poisons: some of these cases of infectious neuritis are of this sort as beri beri.

2a. Morbid Anatomy:-- The involvement may be of one nerve through out the greater part of its extent as the facial nerve, or it may be confined to a small part of the nerve trunk, or it may be a multiple neuritis. The ne ve is swollen, soft and pink.

Microscopically:-- The vessels of the perineurion are full of blood, the peri- and endoneurion are infiltrated with leucocytes, which are polys if the process be suppurative; or lymphocytes if it is simple. Sometimes definite little foci of polys may be found in the suppurative form, constituting little abscesses in the nerve. The fixed conective tissue cells may show some proliferation. A lways in these cases there will be a dggree of degeneration in the myelin, breaking it up into fet droplets. The axones may swell, split up or break up and disappear. If of a mild grade, complete recovery may result. If there has been much connective tissue proliferation or if there be repeated attacks this will become a chronic interstitial neuritis.

2, Chronic Interstitial Neuritis.

1a. Etiology:-- As just suggested this form frequently follows repeated attacks of the acute interztitial form but it may follow one attack. It frequently occurs in syphilis and at times comes about from apparaently no cause, =cryptogenic. It is sometimes met in aged people, probably here an arterio-sclerosis underlies the condition.

2a. Morbid Anatomy:-- One or many berves may be involved. The involved nerve is harder than normal, dense and white,

Microscopically:-- The connective tissue is increased, its cells proliferated, the vessel walls are thickened and at times the lumina are pratically of completely obliterated. The nuclei of the neurolemma may be incresed in number, the nerve fibers are degenerated many of them entirely gone; at times represented only b a thin irregular or collapsed neurolemma. In this condition one is apt to meet with secondary degenerations in the muscle fibers supplied = a degenerative atrophy.

Perhpas there will also be a degeneration of some of the spinal tracts, as in the posterior columns. A spinal variety has been described under the name chronic hypertrophic interstitial Neuritis in which there is such a great increase in the interstitial tissue that the nerves are much too large, and may be felt under the skin as thick cords. Here the muscles suffer a degenerative atrophyland ascending degenerations are frequent in the spinal cord.

3. Parenchymatous Neuritis.

1a. Etiology:- This is due to some toxic substance circulating in the lood or the lymph. These substances may arise from within the body, as in the acute infections, notably in diphtheria and to a less extent typhoid; they may also be produced in gout, diabetes, and uremi On the other hand the poisonous substances may come from without the body, as in the cases of neuritis which sometimes follow ptomain po other food poisonings, alcohol and the metalic poisons such as Pb, As, Cu, and H

2a. Morbid Anatomy:- The early changes are rahter of a degenerative nature, than truly inflammatory. In these conditions we are more apt to have a number of nerves affected, especially those of the lower limbs, much less often only one important trunk may be involved. The nerves become firmer, smaller and greyer.

Microscopically:- The degeneration is ssen to affect first the myeli and later the axones. The myelin breaks up into irregular masses which may or may not be connected by little bridges of myelin. Then it becomes converted into fat droplets. The axone swells and breaks up and disappears. The neurilemma collapses, its nucloi may proliferate. Compound granule cells appear and the little amyloid bodies may show but usually the concentric arrangement is not distinct. The amount of vascul and interstitial change noted is usually at first slight, then as the degeneration progresses, this becomes more prominent. The connective tissue proliferates and in the end we have a picture very much the same as in chronic interstitial neuritis. Following these late changes may come muscular atrophy and perhaps degenerative changes in the ganglion cells of the anterior spinal horns.

VI. Infectious Granulomata.

1. Tuberculosis:-

Tuberculosis of the nerves is practically always due to a direct extension. It is most frequently seen in the spinal and cerebral nerves roots following a neighboring tubercular pachymeningiti Small tubercles are to be found in the connective tissue of the perineurium and there will be some secondary degeneration in the nerve fibers.

2. Syphilis:- Small gummata are sometimes found in the cranial nerves. More frequently syphilis gives rise to a sort of chronic interstitial neuritis, which is especially prone to affect the spinal nerve roots. Here we are apt to have a secondary degeneration from the pressure on the nerve fibers.

3. Leprosy:-

The involvement of the nerves by leprosy, which produces to so-called anaesthetic form, and it is the small cutaneous nerves which are almost exclusively affected. Careful examination of these reveals within the epineurium, along with the peri- and endoneurium the large vaculoated, sometimes multinuclear lepra cells, containing numerous lepra bacilli. The presence of these bacilli produce a connective tissue proliferation and the formation of a sort of agranuloma. Following such a condition the nerve fibers degenerate and thus give rise to the areas of anaesthesia and to the trophic changes so characteristic of leprosy.

VIII. Tumors.

1. Neuroma:-

1a. False Neuroma:- those are also sometimes called fibroneuromata. These not infrequently occur as thickened nodules along the course of the nerves, perhaps hundreds of them occuring in the same case. Here on mieroscopical examination one finds that it is not really a tumor, but merely an excissive proliferation of the fibrous tissue, more or less localized, separating widely the individual fibers. The nerve fibers do not frequently show any proliferation in these tumors, but more frequently undergo a pressure degeneration. A somewhat similar condition, at times, occursin the cut ends of nerves in the amputation stumps, where a more or less rounded mass made up of an intricate tangle of nerve fibers is found. These are frequently called amuptation neuromata, explaining a certain number of painful amputation stumps.

2a. True Neuromata, which have occured from the proliferation or an increase in the number of the nerve fibers with a mimimal amount of connective tissue are rare. They are small in size and may be made up of medulatedor non-medulated fibers, but not malignant.

2. Sarcoma:- In rare cases sarcoma has arisen from the connective tissue of the nerves. They do not often reach a large size; more frequently of the spindle cells variety and do not differ in their histology from the usual spindle cell sarcoma.

Lecture 4/26/'09.

Diseases of the Joints, Outline of :-

I. Anatomical considerations.
II. Circulatory changes.
  1. Hyperemia.
  2. Hemorrhage.
  3. Edema.
III. Traumatism.
  1. Strains and Sprains.
  2. Dislocations.
IV. Permanent Malformations.
  1. Congenital.
  2. Acquired.
    1a. Ankylosis.
V. Inflammations.
  1. Acute.
    1a. Simple acute serous synovitis.
    2a. Acute Fibrinous or sero-fibrinous Synovitis.
    3a. Acut Suppurative Arthritis.
  2. Chronic Inflammations.
    1a. Chronic Serous Synovitis.
    2a. Chronic Purulent Arthritis.
    3a. Arthritis Deformans.
    4a. Gouty Arthritis.
VI. Infectious Granulomata.
  1. Tuberculosis.
    1a. Etiology.
    2a. Morbid Anatomy.
    3a. Results.
  2. Syphilis.
VII. Tumors and Loose Bodies.
  1. Fibroma.
  2. Lipoma.
  3. Loose Bodies.

I. Anatomical Considerations.

A joint consists of the articular ends of two or more bones, capsule and ligaments retaining these bones in postiton and the synovial membrane which lines the cavity and provides the synovial fliud as a lubricant. The articular ends of the bones are covered with cartilage, in nearly all cases hyalin. The ligaments and capsule are made up of dense white fibrous connective tissue with a certain amount of elastic tissue, while the synovial membrane in its outer part consists fibrous and elastic tissue which merge or fuse with the surrounding joint structure, while the inner part next the cavity is covered by flattened endothelia.

II. Circulatory Changes.

1. Hyperemia. This occurs in the early stages of acut inflammat: also cones about as the result of trauma as with sprain or dislocation. In such a condition the synovial membrane instead of its glistening yellowish white appearance is pink or red and may show little hemorrhagic areas.

2. Hemorrhage:-- The most important cause of hemorrhage in the joint structures is traumatism. In the severer forms, of course, usually some bad injury; also in certain of the blood discrasias as in hemophilia we have blleding into the joint structures and the joint cavity. In those acute infections where there is a tendency to hemorrhages we may have the joint affected. Small hemorrhages, usually insignificant arise in the course of acute inflammations of the joints. In any of these cases the hemorrhage may manifect itself in the tissues making up the joint, as the ligamnents and the synovial membrane. In others cases in addition to this we have the blood escaping into the joint cavity and if of any considerable amount it is scalled "hemarthron". Where the blood escapes into the cavity, if there is a small amount of it it will merely tinge the synovial fluid or streak it; but if lage in amount the blood may entirely replace or seem to replace this synovial fluid and such amounts will interfere distinctly with the movements of the joint.

Then too, the presence of the blood in the joint seems to increase the tendency to subsequent infection and inflammation. If the hemorrhage be not too great it may be absorbed, the only trace remining being the blood pigment deposited in the lining membrane. If greater in amount and a distinct inflammation has not supervened, organization of this clot with fibrous tissue may occur, producing a kind of ankylosis.

5. Edema:-- A certain amout of edema in the tissues making up the joint is a constant feature in the acute inflammations. An excess of fluid in the cavity may occur in acute or chronic synovitis or arthritis. This fluid is usually clear, of a yellowish color, thin, and if there be much of it the condition is known as "hydrops articularoum, or dropsy of the Joint".

III. Traumatism.

1. Strins and Sprains:-- These consist of more or less stretching or perhaps tearing of te ligaments and capsule without a displacement of the articular surfaces of the bone. In practically all these cases a certain amout of hemorrhage occurs from the torn vessels and this may extehd into the joint cavity. Incomplete repair of the torn tissues may produce a permanet weakness of the joint; sometimes in the organization of the blood which has escaped excessiev amounts of fibrous tissu e are formed, which may produce a form of ankylosis, usually of the capsular variety. Then too, after a strain or sprain acute synovitis or arthritis may occur.

2. Dislocations.:-- Here we have a misplacemetn of the articular surfaces of the bones. When it is complete =luxation; If incomplete = s subluxation. Such dislocations may be 1spontaneous, 2congenital or 3traumatic.

Under the spontaneous dislocations are placed those which have occurred from some slight cause acting on a joint previously weakened by disease. This may be produced by an accumulation of fluid in the cavi causing a stretching and weakening of the capsule and ligamnents as in serous synovitis; or it may be a destrution of parts of the bone. Then too as a result of disease the fossae into which the heads of the bones should be placed may become too shallow, allowing an easy dislocation.

In the congenital form we find it nearly always in the hip joint and this may come about from a primary hypoplasia of the hip joint whereby the acetabulum is too shallow or too small for the head of the femur. From abnormal positions of the foetus in utero the head of the femur may be forced out of another wise normal acetabulum.

Traumatic Dislocations:-- This is the most frequent pathological process found in connection with ~~~~ the joints. As a result of violence the supporting tissues are torn, hemorrhage takes place and acute inflammation ensues. If the dislocation be reduced this inflammation rapidly subsides, but if it continue, however, certain permanent deformities occur.

IV. Permanent Malformations.

1. Congenital Malformations of Joints depend upon the congenital arrest in the development in the bony parts of joints or upon abnormal pressure exerted particularly upon the articular surfaces of the bones. Here there results such deformities as club foot: When if the foot is turned in = pes varis; if out = pes valgus; if the foot is extended and he rests upon the ends (anterior) of the metatarsal bones = pes equinus; if the foot is flexed and the body is supported on the feel = pes calcaneous: or it may be various combina tions of these. In addition to the congenital defects of the feet we may get clubbed hands ~~ with various deformities or knock knee = genu valgus; or bow legs = genu varum. In addition to these defects being congenital certain of them may be acquired.

2. Acquired Malformations:also formation of new or false joints:-- Where a dislocation is not reduced and the part has not become immobile a false joint may be fomed. As a result of pressure atrophy a space is hollowed out in the bone upon which the articular head of the dislocated bone rests. About this is an ossifying periostitis which builds up a wall of bone thus deepining the cavity. About the margins of thsi cavity fibrous tissue is formed. which becoming very thickened takes the place of the ligaments of the original joint. Sometimes a portion of tathe original ligamnets may be utilized. Cartilage arises from a metaplasia of the periostium and the cavity becomes lined with a syhovial membrane, thus in som cases an almost perfect new joint is formed. After a resection of a joint and the approximation of the bones these may be united by fibrous, cartilagenous or bony tissue. The bony union is the surgeons disired end. If the ends be approximated and held so and the vitality of the patient be sufficient bony union occurs in exactly the same way in which a ftactured bone is healed. Sometimes, however, when the vitality is low or where the parts are not firmly fixed a fibrous or caetilagenous uhion occurs produc-

producing the flail joint.

1a. Ankylosis:-- This is a complete or partial union of the bones at the joint so that movement is impared or prevented. It results from interosseous attachments as the result of chronic inflammation. If the cartilage be eroded from the ends of the bone and the bones be held in position for some reason a bony union is likely to occur, preventing any movement and it is known as true ankylosis. If a certain amount of disturbance of the ends of the banes is allowed the bony repair does not occur and fibrous tissue or cartilage results. This permits a certain deg degree of movement at the joint, though always limited, and the condition is known as false ankylosis. Sometimes the loss of movement is due to an extreme thickening of ths ligaments and capsule a bout the joint = capsular ankylosis.

V. Inflammations. These inflammations may be confined to the synovial membrane = synovitis ; or it may involve the cartilage, ligaments and capsule = arthritis and at times it extends to the bones themselves = panarthritis. Clinically the term arthritis is applied to any or all of these.

1. Acute.

1a. Acute simple serous synovitis:-

1b. Etiology:- A degree of this always occurs in such trauma to the joints, as sprains, contusions and dislocations. A rather more severe dgree of this is found in acute articular rheumatism and gonorrhoeal rheumatism, frequently assumes this form. The presence of loose bodies in joints, which being cought between the articular surfaces, may produce an acute synovitis. Tumors nearthe joint or enclosed foci of osteomylitis may produce this form of synovitis. Practically all the cases of acute arthritis of any form start in first as acute serous synovitis.

2b. Morbid Anatomy:- The joint cavity is filled with an excess of clear, yellowish, thin fluid, not so thick and glairy or viscid as the normal synovial fluid. Perhpas in this little flocculi o f fibrin may be found, but these are inconspicuous. Especially in the traumatic cases some blood will be admixed. The synovial membrane ins injected reddened, swollen, and the synovial folds and fringes are especially swollen and red. In the more severe cases the joint structures round about will be swollen, edematous, red and hot from the excess of arterial blood.

3b. Results:- Upon the removing the cause such an inflammation usually subsides, undergoing complete recovery, ot it may pass on into a distinct arthritis. A certain number of these cases show a distinct tendency to return, passing into a chronic stage. Even without recurrent attacks such an acute inflammation as this may gradually merge from the acute to subacute and then the chronic inflammation.

2a. Acute Fibrinous or sero fibrinous synovitis:-

The cause here is practically always an infection either with the micrococcus of articular rheumatism, gonnococcus or other pyogenic cocci. Here in addition to the changes indicated for the above synovitis, one finds a distinct layer of fibrin on the serous membrane. This fibrin here is the prominent feature and while there is an excess of fluid usually, in a few cases there may be little fluid present, producing the dry fibrinous form. In this cases the fibrin is likely to organize with f brous tissue, producing a chronic arthritis or an obliteration of the joint cavity = Ankylosis.

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Lecture 5/ 3/ '09.

3@. Acute Suppurative Arthritis:-

Here there is an involvement of the synovial membrane, cartilage and other joint structures.

1b. Etiology:- It is always bacterial, the pyococci, including the streptococci, gonnococci, pneumococci, B. Coli, and B. Typhosus. These are the ones usually at fault. These may gain entrance through an injury to the joint or may come about from the direct extension of a neighboring process: and lastly the process may be hemotogenous the baceria being brought by the blood and in the purulent arthritis, which may be found in gonorrhoeal rheumatism, also as a complication of pneumonia, typhoid fever, and puerperal sepsis.

2b. Morbid Anatomy:- this may affect one or more than one joint. It probably begins as a synovitis, but very quickly become a panarthritis. There is an intense hyperemia of the joint structures, an exudate of polys into the cavity, and marked destruction of it.

Microscopically:-- the tissues are infiltrated with polys and with this the other marks of a severe inflammation will be seen. The skin over the koint is red, tense, shiny and white. If the pus is not quickly evacuated and erosion of the cartilage will occur , perhaps more or less of the bone will be involved so that in some cases the whole of the epiphesys will be destroyed. At the same time areas of necrosis are seen in the ligaments and capsule. These weaken the joint so that spontaneous displacements easily occur, or the adjacent tissue may be involved by the purulent exudate extending along the tendons and the tendon sheath even into the bursae or possibly reach the surface forming fistul tracts and dischagging pus.

3b. Results:--If the process is stopped early before there is much perosion good repair will result. If, however, the articular cartilages are eroded and the bone involved more or less ankylosis is to be expected when the process heals.

2. Chronic Inflammations.

1a. Chronic Serous Synovitis:-- This is most frequently due to a continuation of an acute synovitis or to recurrent attacks of it. When the process becomes chronic the exudate is likely to be large. The fliud may be thin or thick, at times almost gelatinous, perhaps containing masses of fibrin and it is always rich in albumen. The synovial memebrane and folds become thickened, the fringes may be long and polypoid, the cartilages may be covered by or converted into fibrous tissue. A large amount of fluid distending the cavity, stretches the ligaments and capsule, weakens the joint so that spontaneous dislocation is possible.

2a. Orhonic Purulent Arthritis:-- This is always due to bacterial infection and follows the acute setous arthritis. The various erosions and other changes have been sufficiently mentioned under the acute form. The cartilage may become converted into fibrous tissue, fused and ankylosis is a frequent sequel. This ankylosis may be almost or entirely capsular or the un on may be fibrous or even bony. Along with thi there is nearly always some involvement of the neighboring periostium which thickens; bone is later laid down here, so that we have the ends of t the bones variously distorted and ragged.

3a. Arthritis Deformans.

Synonyms:-- Chronic Osteo-arthritis, chronic arthritis, Rheumatoid Arthritis, Dry Arthritis, Atrophic Arthritis, and that of the vertebrae is known as spondylitis Defromams.

1b. etiology:-- Two theroys are held, (a) that it is of nervous origin, (b) that it is an infection. In the latter case some say that there is a definite causative organism; others , that it may be due to any one of a number. As a matter of fact the real cause is not known. It may be mono articular or poly articular. Some of those cases involving one joint seem distinctly tracable to such injuries as sprains, contusions, fractures, etc.

2b. Morbid Anatomy:-- As just mentioned it may be mono or poly articular. The mono articular form most frequently affects the hip, then the knee, the shoulder, and the elbow in the order named. Probably then the joints of the hands and of the feet and the vertebrae are next most frequently affected. The poly articular form is seen most frequnently in women passed the age of thirty and affects the small joints of the extremities, at times the vertebrae. In the early stage of the disease it is a panarthritis. The cartilage increases

small joints of the extremity, at times the vertebrae. The early stage of the disease is a panarthritis. The cartilage increases in thickness, its matrix becomes fibrillated especially near the free surface. Later there is an absorption and necrosis of this thickned cartilage in places, especially those parts expsed to pressure. About the margins of the cartilage th e tissue proliferates, bone or lime salts deposit , producing irregularities,. The synovial villi increse in size, frequently containing much fat, extending in masses into the joint to form the socalled arbo rescent lipoma. The medulla of the bone at the end becomes absorbed, the lacumae increse in size, which decreses the density and here mucoid or gelatinous areas may be met. Along with the increzase of the bony tissu about the magins will come an atrophy of the parts exposed to pressure. They are absorbed so that the normal appearance of the joint may be completely lost. The excessive amount of bone formation and fibrous tissue overgrowth produce partial or complete ankylosis or the bony absorption leads to dislocation or distoriton of the joint and the thinning of the bon substance makes fracture easy. As the movement of the joint becomes restricted, the muscles and other tissues atrophy, rendering the deformity more evident. Then too, bits of bone or synovial villi may break offf, forming loose bodies in the joint.

Spondylitis Deformans or Arthritis Defromans of the Spine:-- This produces an absorption of the anterior parts of the bodies of the vertebrae resulting in a sttoping position of the body. The edges of the vertebrae become connected by bony deposits , irregular bony masses are formed, the bodies of the vertebrae decreased in size and the whole converted into an inflexible column with various deformities.

4a. Couty Arthritis or Gout Arthritis Uratica.

1b. Etiology:-- The underlying cause of the gouty deposits is an abnormal metabolism whereby the urates are not properly handled. Some hold that the slowed circulation produces local necrotic areas and the change in the tissue reaction here produces deposist of salts frm a blood plasma overloaded with them.

2b. Morbid Anatomy:-- The salts are usually sodium biurate but the urate of Ca, Mg, and NH_3, and later on some of the carbonate. may be found. In most cases it is the metatarso phalangeal joint of the great toe first involved, but it may also be seen in the ahnds, fingers, knee, etc. The sodium biurate is deposited in the form of sharp needles or granules in the matrix of the cartilage and its cells, inthe synovial membranes, in the ligaments and sacsule, frequently in the joint cavity and this deposition, which is paroxysmal in character, is followed by a hyperemia and acut inflammation. In severe cases the cartilage may become necrosed, the synovial membrane and capsules thivkened producing chronic changes frequently of a severe degree. Later on considereble deposition of these salts occurs in the tissues about the joint, producing an enlargement, thus the tophi. These, by erosion, may reach the surface and some of the material may be discharged. At the same time as the urates are depositied in the joint they may begin to appear elsewhere, as in the cartilage of the ears, skin, subcutaneous tissues, eyelids, kidneys, etc. As the acute paroxysm subsides the acute inflammation of the joint passes off leaving, however, the greter part of the deposit.

VI. Infectious Granulomata.

1. Tuberculosis:-- This is quite frequent and is also called "White Swelling".

1a. Etiology:-- Certain of these cases arise first in the synovial membrane, the bacteria having carried there by the blood either from a definite active recognized tuberculous focus or from some latent obscure focus as the bronchial or mesenteric nodes. Where this begins in the synovial memebranes = arthropathic. The greater number of tuberculous joints, however, arise secondarily to tuberculosis of bone = osteopathic. Here the original tuberculous involvement of the bone is apt to be near the articular surface, so that extension to the joint is frequent and easy. Childhood, before 15, is the period of preference. In most cases there is a history of injury. This may act by producing an area of lowered resistance where the bacteria may lodge and grow or it may produce the breaking down of a latent bone tuberculosis.

2a. Morbid Anatomy:-- Tuberculosis usually involves only one joint, the hip, knee, or ankle, in order of frequency, bu t it may be the shoulder, wrist or elbow. Then t o, the tubercular arthritis is frequently a prominet feature in the tuberculosis of the vertebrae or Potts' disease of the spine. In a general disseminated tuberculosis we may get miliary tubercles in the joints. These are of no clinical significance.

Of the chronic process, there are two general forms:

(a)Chronic Casecus Tuberculosis:-- This, when arising as a synovial tuberculosis or form the bone, rather early becomes a panarthritis. The synovial thickening and proliferated bone shows much granulation tissue, perhaps projecting out and filling the cavity. At this stage the tissue microscopically shows ordinary tuberculous granulation tissue with the formation of tubercles, a farin number of giant cells, but few bacilli. This stage is sometimes called fungoid tuberculosis. Now ensues an extenzsiev caseation of the granulation tissue breaking down and filling the cavity with granular caseous material. The cartilages are ulcerated, the joint capsule becomes tuberculous and sinuses may extend to the surface, the socalled "cold abseesses of the joint". Little rounded masses of altered fibrin , the socalled "rice bodies", are sometimes found in the cavity.

Results:-- (1) Healing , which may give rise to ankylosis, with luxation or subluxation; (2) Wide spread miliary tuberculosis; (3) Extension along the bones gives rise to a tuberculous osteo-myelitis: (4) a not infrequent complication, especially after the sinuses are formed, is a secondary in fection with the pus organisms filling the cavity with pus and increasing the rapidity of the destruction.

(b). Chronic Fibrous Tubercular Arthritis:-- This may affect a number of joints simultaneously or in succession. It is essentially a chronic fibrous hyperplasia and may resemble arthritis deformans. A slight amount of caseation is always present but not frequently marked. The tissues about and of the joint show much fibrous increase, serous fluid fills the cavity and ankylosis may result from a union of the articular surfaces. The danger of systemic distribution is much less in this form.

2, Syphilis of the Joint:-- This may occur either in congenital or acquired syphilis. In the congenital form we have eitera a chronic serous synovitis with much fliud in the cavity or a fibrous increase with some ulceration of the cartialge giving rise to joint deformities. In the acquired syphilis a serous synovitis of rather acte nature may occur during the stage of the secondary eruption, especially in the sterno clavicular joint. In the tertiary stage, in rare cases, we may find gummata of the synovial membranes or it may extend in from the neighboring rissues. Healing of these leaves irregular stellate scars with nodular joint surfaces and deformities.

VII. Tumors and LOOse Bodies of the Joints.

The joint may be involved secondarily by the extension of tumors from other parts. Primary tumors are rare. The fibroma may occur and the lipoma arborescense of arthritis deformans has been m entinoned.

Loose Bodies:--These are sometimes met in the joint of the elbow, wrist, hip, shoulder, and ankle, but perhaps nine tenths of then occur in the knee. They consist of detached parts of the articular cartilage, synovial membranes, fragments of bone, calcified fibrin masses or the rice bodies, proliferated fibrous tissue etc. They may give rise to serious difficulties by getting in between the surface and locking the joint or they may produce serous synovitis.

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there are usually tubercles in the adhesions, in case of acute tubercular pleurisy. This may occur from direct extension of the disease in which case you will find and effusion, either serofibrinous, purulent, or hemorrhagic. Pyo-pneumo-thorax, from perforation of a cavity or suppurating process is not infrequent and is such cases there often an absent leucocytosis.

Symptoms: You may not notice the onset and there may be a large exudate without symptoms of it. Usually there is a little cough and it may be marked. If there is a co-existant diseaseof the lungs, the symptoms will be associated with that and will be more marked. If subcrepitant rales, which are dry and fine, are present, and with any other symptoms of tuberculosis, the positiveness of your diagnosis will be increased. Later there will be lungs signs or symptoms of acute miliary tuberculosis and these will clear up the local symptoms which were mild at first. Sometimes these cases with an exudate clear up: the exudate is absorbed, the pleura becomes thicker and aherent and then there will be no signs remaining.

Tuberculosis of the Pericardium.

The same lesions affect this that affect the pleura and peritoneum. The effusion may be small with no signs, or it may be large or Hemorrhagic. In chronic cases the mebranes are thickened and have tubercles and cheesy masses scattered over it. Tubercular percarditis is less frequent than tubercular plourisy.

This is rarely a primary affection but is usually secondary to pulmonary, pleural, mediastinal, or bronchial tuberculosis, and especially is this ture in children, but it may occur at all ages. If there is a near by tubercular focus, it may invade the pericardium e.g spine, or ribrs.

Diagnosis.: This is difficult. There is no inflammatory leucocytosis and this will help to cut out the inflammaions proper. If you have a history of the disease in the family or in the person, or you can locate the disease in other parts of the body, you are safe in your diagnosis. In the chronic form, where it may be a part of a general serous membranous tuberculosis, you may over look it or suspect it. You may suspect it when it is associated with the lungs, pleura, peritoneum, or glands.

It may occur with or without effusions. More frequently it is the dry and adhesive form and this produces peculiar changes in the heart, as hypertrophy and dilitation. The signs of an adherent pericardium may be present, as, dullness extending as high up as the sternum and around the aorta, because of the chessy masses at the base of the heart. Cases with a simple effusion ~~xixi xix~~ as serofibrinous or hemorrhagic, will give signs like those of dilation of the heart and other pericardial effusions.

Tuberculosis of the Peritoneum.

In Miliary Tuberculosis and also in the chronic forms of pulmonary tuberculosis, frequently the peritoneum is involved, being studded with little greyish white tubercles. If there is an intrstinal ulcer of the tuberculous type, the peritoneum may also show these little greyish white tubercles. It occurs like the other Serous Membranous tuberculosis = the acute miliary form without the exudations as above mentioned.

In the chronic forms there are large grwoths and they tend to ulcerate and break down; sometimes this is walled off and becomes sacculated. The exudate is usually purulent or sero-purulent.

~~IX~~ Chronic Fibroid Tuberculosis

This may occur with insiduous symptoms or it may be the final stages of acute miliary tuberculosis. There will be little or no exudate and the tubercles may be hard and pigmented, and the surfaces adherent

Etiology: The disease may be primary and local; it may pass from the intestines; or from the Fallopian tubes; or the Seminal Vesicles; or from the prostate. Abdominal diseases ~~xxx~~ or diseases that cause trouble in the circulation in the peritoneum and the intestinal tract, as cirrhosis of the liver, ovarian tumors, hernial secs, and traumatism to the abdomen, have predisposing influences.

The usual age is from 15 to 40, frequent, it may beunder 10 years or over 40, and then you will think of carcinoma. Negroes are prone and females are more liable.

Symptoms: They may be latent or there may be none at all. If there is a hernia or ovarian disease you will have the symptoms of this and over look the tuberculosis. The fever sometimes runs up as high as 103 or 104; or it may be subnormal or it may range about 99. The pulse is variable, small and rapid; there are abdominal pains, diarrhoea, and sometimes vomit. Sometimes these cases go on a resemble typhoid fever, with the above symptoms accompanied by progressive emaciation and anemia.

If signs of effusions occur there will be,usually, an irregular fever with sweats; tympanites, due to a loss of tone caused by an inflammatory infiltration of the gut. Ascites occurs in these cases which may be purulent or hemorrhagic. Sometimes you may feel hard masses or hear friction sounds about the umbilicus.

Sometimes the symptoms are very slight, little anemia, only a dragging on" fever, pains and abdominal distention. In some of these cases the skin becomes pigmented and looks like Adison,s disease, except that it occurs in splotches and spots over the abdomen.

This disease often simulates tumors of the abdomen and is frequently associated with them. The omentum may be rolled up, forming a firm elongated mass in the upper abdomen and attached to the transverse colon. The same occurs in carcerous peritonitis but is more frequent in tuberculosis; the nodular masses and tympanites occur in ruberculosis. The omentum may sink into the Right Illiac fossa and form a tumor there. When adhesions and accculations from in the intestines, mesontery, and abdominal walls the diagnosis is difficult. These nodules or tumors may lie in the pelvis, in the mid-line, of to onesides, and they may,of course be associated with Fallopian disease.

There is a rare disease inwhich the intestines become thickened, retracted, and shortened, which forms a tumor which feels like a solid mass between the palpating hands.

In children,especiallly, with tubercular peritonitis, the mesenteric glands are affected and ascites may co-exist.

Unless there is tuberculosis in the other organs there is difficulty in the diagnosis, especially when there is no fever, or pain or only a slight abdominal tenderness. But if you have the emental tumor with the other symptoms the diagnosis is easy. Always look out for cancer, especially after the cancerous age.

Diagnosis of Saccular Exudation of tuberculous lesions from Ovarian Tumors:-

Consider:- History of tuberculosis in the family or patient; there is an irregular fever and pain in tuberculosi, while these are not marked in ovarian tumors; with Tbc there are gastrointesinal disorders but less frequently in ovarian tumors; the Tbc tumors are not well defined, while the ovarian tumors generally are; also examine for the firm contracted coils of the intestines so frequently occuring in tbc; Examine some of the centrifugated fluid for the tbc. bac.

Differentiate the following:-

(1). Hernia: Here there is a sudden local pain; absolute constipation; fecal vomiting; tympanites; but no ascites.

(2). Volvulus:- which gives its own characteristic symptoms.

(3). Enteritis:- No tumors, no ascites, no tuberculosis in other parts of the body, and there are mucous discharges from the bowel.

PULMONARY TUBERCULOSIS.

Also called "phthisis pulmonalis, consumption ".

It occurs in classical form in the following three types:-

1. Acute Tuberculaer Pneumonic Phthisis or Acute Phthisis.
2. Chronic Ulcerative Tuberculosis.
3. Fibroid Phthisis.

According to the mode of infection the lesions produced are of two types (a). Sometimes the bacilli reach the lung thru the blood or lymph vessels. Then the primary lesion is usually in the tissues of the alveolar walls, in the capillaries, in the epithelium of the air cells and in the fibrous septa between the lobules. The lodgement of the bacilli produces an irritation and there results small grayish miliary nodulae involving several of the alveolar spaces. These nodules consist of round cuboid uninuclear cel of epitheloid character. There maybe only a local tuberculosis or a gener tuberculosis of the lungs, depending upon the number of bacilli. The tubercles maybe uniformily scattered thru out both lungs and form a part of a general miliary tuberculosis, or they maybe confined to the lungs, or to one lung. In these tubercles there is an arrest of cell division, fibroid formation or sclerosis of the tubercle, and they maybe completely transformed into fibrous tissue. Some of them caseate near the center of the tubercle, while at the periphery the trouble is extending by proliferation of the epitheloid and lymphoid cells. Sometimes these tubercles become confluent or conjoined, and in this way form diffuse areas which undergo caseation and softening. Sometimes there is a very intense infection of some local region thru the blood vessels, and the tubercles are very thick and the intervening tissue becomes intensely inflammed and the air cells are filled with the products of the desquamated inflammation and a good man lobules maybe involved. Some claim this mode of infection to be the principal mode of infection of the lung.

(b). Next is where the bacilli reach the lung thru the bronchi by inhalatio or by aspiration. The small bronchi or bronchioles are especially affecte and the distribution is in lobular form. The tuberculous masses are large from the first, are more diffuse, and may involve an entire lobe or an entire lung. They form areas of nodular broncho-pneumonia, and little granulations occur outside the bronchial tubes - the socalled " peri-bronchial granulations". The same process of caseation, ulceration, and cavity formation, together with sclerosis and encapsulation and healing, make up the essential elements going on in these areas.

1. Acute Pneumonic Tubercular Tuberculosis.

Also called " Phthisis florida", or by the laity "Galloping Consumption" It occurs at any age, but in children it is often diagnosed as broncho-pneumonia. It maybe primary, or it maybe secondary to a localized tuberculosis of the lung, pleura, peritoneum, or other organs. It seems that men are oftener affected. It sometimes takes the form of pneumonia, in which one lung or one lobe is involved, and the involved part is heavy, airless, and the pleura shows a thin exudate. Section of the lung shows hepatization, perhaps cavities, and tubercles scattered thru the consolidated tissues, and also yellow-white masses which are caseated tubercles. These maybe surrounded by an intensely injected or consolidated tissue. Anemic or dry areas may occur separated by areas of red inflammed or hepatized tissue. The tubercles maybe scant and escape notice. The other lung or the bronchial glands may exhibit tuberculosis. As the process gets older, these grayish-white masses appear. The affected lobe or lung is large, firm, and maybe converted into a dry yellowish-white mass. If the disease lasts two or three months there is softening at the apex with the formation of cavities.

Symptoms. It may begin abruptly with a chill, usually in those previously in good health, and simply following exposure to cold or some debilitating circumstance. Its onset is quite similar to pneumonia, with pain in the side, fever, cough, prostration, perhaps hemorrhage for a day or two, and expectoration usually mucoid at first but later becoming rusty. The sputum may contain the bacilli or it may not for some time. There is dyspnoea and perhaps evidences of suffocation. The temperature rises quick to 104 and usually remits and is continuous, but it maybe hectic.

By the end of the first week, there are night sweats, rapid emaciatikn greater prostration, and expectoration is more abundant and muco-purulent or greenish-yellow. The physical signs would be the same of those of a pneumonia. There would be dulness on percussion, increased fremitus on palpation, increased vocal resonance of auscultation, with suppression of the respiratory murmur. Later there maybe bronchial breathing, and as the tissue breaks down and cavities form you get gurgling rales and amphoric breathing. Without the presence of the tubercle bacilli being demonstrated, a diagnosis of lobar pneumonia would be made, until failure of the crisis to appear. A pneumonia is expected to resolve in two or three weeks, and if it don't do so, you had better be suspicious. Usually by the end of the second or third week there will be elastic tissue and tubercle bacilli in the sputum. These cases run a very rapid course, usually from two weeks to three months. Some casees are recorded as running less than one week. If it persists longer than three months it assumes the chronic form.

Diagnosis. Clinically the onset and symptoms are those of lobar pneumonia, but certain things would arouse your suspicion, as a tuberculous family history, hemorrhages, previous bad health, recurring chills, fever rather more remittant form than occurs in pneumonia. You may mistake bronchiectasis for it, but the fever and loss of flesh are not so great, and the other symptoms of a dilated bronchus will appear.

Broncho-Pneumonic Form of Acute Phthisis.

This if more frequent in children and forms the large majority of cases of the galloping type. It is an acute caseous broncho-pneumonia, starting in the smaller tubes, which become blocked up with cheesy material, while the air cells of the lobule are filled with the products of a catarrhal pneumonia. These areas early have a grayish-red color, but later become opaque and caseous and yellow-white in appearance. If contigous areas fuse an entire lobe maybe consolidated, but usually it is only in the lobules with crepitating places between. The bronchial glands maybe enlarged and contain tubercle bacilli, and they may suppurate and form an abscess. Cavities occur here in the same way and by the same process as before. Haemoptysis may produce an extension of the pneumonic process or an aspiration pneumonia. The contents of the cavity may be brought out and then drawn into the finer tubes, and with this, fever, dyspnoea, and signs of a diffuse broncho-pneumonia with a rapid course. This may occur in ordinary cases of tuberculosis. In children the bronchial glands swell and press on the lung and produce indentations, and there maybe direct extension of the tuberculous process. In still other cases the caseous broncho-pneumonia involves only a few group of lobules closely associated together or different lobules situated thru out the lungs. The size of these consolidations depend on the size of the bronchi involved. Some have grayish-red tubercles and secondary infaction may occur.

Symptoms. These are varyable. Adults maybe in good health but ~~usually they~~ are overworked, run down, anemic, neurasthenic. Sometimes hemorrhage is the first symptom. There maybe repeated chills, high temperature, rapid pulse, increased respiration, and loss of strenght of flesh.

The physical signs are some what uncertain and indefinite at first. Later on, frequently at the apex, there is impairment of resonance. The breath signs at first are a little harsh, broncho-vesicular, or the slightest tendency to bronchial breathing. b There maybe various rales and the sputum will show the bacilli. By the third week the typhoid stage will intervene and death occur before your diagnosis of tuberculosis is made. ~~In these cases there is early onset with the above symptoms and the disease confined to the lung or spread to both.~~ There are chills, sweats, fever and emaciation.

Then in 6 or 8 weeks the symptoms abate and you think it pneumonia recovering, but it is simply becoming chronic. In children this disease frequently follows measles, whooping-cough, and perhaps la grippe in adults as well. In children, the child is taken suddenly sich while teething or during convalescence from some acute infection, and the temperature runs up rapidly, the cough becomes severe, there is consolidation in one or both apices, rales appear there, and death may occur in a few days. The lung shows signs of areas of broncho-pneumonia, and scattered here and there areas of tubercular nodules. On section it may look like broncho-pneumonia, but microscopically tuberculosis is revealed and tubercle bacilli are frequently present in the bronchial glands. During convalsecence from some acute infection, like measles, whooping-cough, scarlet fever the child is taken ill with fever, has a cough, dyspnoea, and some times in two weeks these symptoms subside, but instead of getting well the child goes from bad to worse and becomes pallid weakened and looses weight. Rales are heard about the bases. There are areas of defected resonance and perhaps above these a little hyper-resonance. Sweats occur, a hectic fever, and this may end in death or go on and persue the chronic form.

2. Chronic Ulcerative Tuberculosis.

Here in the large majority of cases the lesions go on to ulceration and softening and cavity formation. It is at first a pure tubercular infection, but later becomes a mixed infection with streptococci, staphylococci, or pneumonococci, and here you get septic symptoms from the infected foci.

Pathology. There are a large number and variety of lesions, comprising diffuse tuberculous infiltration, caseation, pneumonic areas, cavities, enlargement of the bronchial glands which maybe caseated, with the bronch in a state of catarrhal inflammation, and the pleura maybe invaded. Most often the lesions are in the apeces and the disease proceeds downward, usually worse in one lung and more tissue is here involved. It follows distinct routes. It has been found in a large number of cases that when the upper lobe is attacked by the primary lesion, the lesion is not at the extreme apex but about one to one and a half inches below the summit of the lung, and nearer the posterior and external borders. The lesion tends to spread downward probably by inhalation. You may not be able to make out the diseas from the front, while examination from behind may reveal it. In front this lesion is opposite a point just below the center of the clavicle, and it extends along the anterior aspect of the upper lobe, along a line one to one and a half incehs from the inner ends of the 1st, 2nd and 3rd intercostal spaces. A second spot often overlooked, where the primary lesion begins, is below the outer third of the clavicle in the second interspace. The extension is downward and outward. The middle lobe becomes involved usually following disease of the upper lobe on the same side. The first infiltration is about 1 inch to 1-1/2 inch below the posterior end of its apex and at a spot on the chest wall opposite the 5th dorsal spine. Hence always examine carefully this posterior apex in all cases. This lesion spreads downward and laterally along the line of the interlobular septa, along the line of the vertebral border of the scapula, when the elbow is raised above the level of the shoulder. Sometimes the disease spreads To the apex of the opposite side, but usually not until the lower lobe of the first side is involved. Lesions of the base are not very often primary. About 1-500 cases occur. The lesions may become arrested at the apex in chronic cases, and progress elsewhere in the lung.

Pathology and definition of the lesions:

(1). Miliary Tubercles, which are distributed by dissemination of aspirated tubercular matter thoughout the air cells or into the walls of the smaller bronchi. They are also distributed by the lymph and blood currents, and especially by the lymphatics, in which the tubercle are located in a radial manner about the old foci. Sometimes these foci are in the vessel walls and in this way they becomes scattered. You may have many miliary tubercles that are hard and firm at the base of the lung or lower lobe, while there may be cavities in the apex

The most of these cases, however, are consituted by the tubercular broncho-pneumonic type. Here the process originates in the small bronchioles and the air passages becomes blocked with the products of inflammation and caseation. If a section of the lung is made these small bronchioles show up as small orifices plugged up with cheesy masses surrounded by caseous nodules.

(2). Now in these pathological conditions the processes of ulceration, caseation, and softening occurs in the bronchila wall and frequently forms a small cavity.

(3). But in most cases of chronic ulcerative pulmonary tuberculosis there is a formation of new fibrous tissue. This is known as sclerosis. These fibroid changes may be extensive. In many cases the caseaous areas are completely surrounded by a fibrous capsule.

(4). In this firbous capsule calcification may occur and result in healing, and the formation of lung stones. And miliary tubercles may occur around these.

(5) A pneumonic process may occur, in which the inflammed alveoli are filled with epitheliood cells. This consolidation may extend for some distance about the focus: and the consolidated areas may fuse to form a uniform consolidated area. The pneumonia may be tubercular in origin or it may be a simple inflammation. In appearance it may be exactly like red hepatization, or closely resemble a simple infiltration. In othere cases the cut surface is yellow or white or opaque. When there is much consolidation early in the disease, it is due to this pneumonic infiltration.

(6). Cavities or Vomica:- These are formed by necrosis and ulceration in the lung tissue. This begins, usually in the walls of a tubercular bronchus, which becomes dilated from the retained secretions and necrosis and ulceration result. This extends into the surrounding tissue. There may be many cavities formed and these unite: tometimes they communicate with a bronchus and sometimes not; and the may occur in a mass of consolidated material away from a bronchus.

These cavities in the form of fresh ulcers in the acute tubercular phthisis where there is no limiting membrane formed. Their walls are soft, ulcerating, necrotic masses and often produce pneumothorax. They are greatest in the upper and posterior parts of the urper lobe. Sometimes they are called porgressive, in contradistinction to the chronic ulcerative pulmonary Tbc., which is called non-progressive. As a general rule, in chronic ulcerative pulmonary tbc, cavities are always present if the disease is far enough advanced. Here the limiting wall is well defined; and there is a secretion of pus. These cavities consist of obliterated bronchi

These cavities not infrequently, have little threads running across them. Thesethreads consist of obliterated bronchioles and blood vessels. The exudate from these cavities is purulent, like the sputum. There may be a gangrene present, in which case you get a fetid breath. In most of these cases there are several cavities, but the cavity may single and alrge enough to almost absorb a whole lobe or the entire lung. The latent cavities are those that are not active, such as are seen at Post Mortem. These are usually small and surrounded by a dense fibrous tissue.

The lining membrane of these cavities is quite smooth and looks like mucous membrane.

A lagge bronchus with consolidations about it will give the same physical signs and sounds as a cavity.

As the blood vessels become obliterated by ulceration of inflammation hemorrhage occurs, Some of these blood vessels have aneurisms formed on their walls, these are small and are significant only in the production of hemorrhage. Usually,however, there is a surround the blood vessel a zone of sclerotic tissue.

The pleura is nearly always involved in chronic ulcerative pulmonary

This occurs early and the pleura may be thickened and dense and firm, or it may be thinned. The pleurisy is not always tubercular in origin, but sometimes it is a simple inflammation. But when it is tubercular, there may be caseous nodules through out the membrane. Serous, purulent, or hemorrhagic, effusions may occur into the cavity and not infrequently pneumothorax or pyopneumothorax occurs.

If the process begins in the smaller bronchioles these become filled with secretion and debris, and as it extends up, the tubes become dilated = bronchiactasis. The mucous membrane of the bronchioles near by, or of those over which the secretion is aspirated or flows, shows a catarrhal inflammation and sometimes ulceration.

The bronchial glands are swollen and edematous, and may contain miliary tubercles. They undergo caseation or softening with the formation of pus; sometimes they calcify.

The other organs of the body, as the brain, liver, spleen, kidneys, bowels and pericardium are usually involved. The liver may be enlarged and undergo fatty changes. It may also contain amyloid areas, as well as the spleen, kidneys, and mucous membrane of bowel.

Intestinal tuberculosis frequenylt occurs and is nearly always accompanied by diarrhoea.

Sometimes the endocardium is affected and there may be tubercular bacilli in the vegetations.

The larynx is frequently ulcerated; the vocal cords and epiglottis may be destroyed.

Modes of onset of tuberculois are important, because they are so frequently overlooked.

Usually it comes on slowly and has a gradual and progressive course. It may however, be at first latent, in which case the patient does not complain of well defined illness and there is considerable progress before the disease is recognized. There may be no well marked local lesions or a cavity may form before a doctor has been consulted. Indeed you may recognize tuberculosis in other organs of the body, as in the bones, glands, intestines peritoneum, etc, before you recognize it in the lungs.

One of the most frequent forms in which occurs and the symptoms of which the patient complains of first, is indigestion and dyspepsia, and anemia, and stomach troubles. The stomach becomes irritable, vomiting is frequent and free, with acid eruptations. In young females there are ofter symptoms of chloranemia, palpation of the heart, slight afternoon fever, and amenorrhoea.

Next is the type in which the patient complains of attacks of malaria. There are chills, fever, and sweats. These occur with considerable regularity and the patient is drenched with quinine before the true disease is recognized.

Sometimes the first symptoms is that of pleurisy. It may be of the dry form at the apex, with a simple friction rub. Sometimes there is the sharp stabbing pain of acute pleurisy, followed by an exudate with its symptoms of cough, fever, etc, and the effusion may disappear but the signs do not clear up. About 1/3 of all the pleurisy cases with effusions develop tuberculosis.

The laryngeal lesions are very rarely primary, but most often they are secondary to chronic ulcerative pulmonary tbc.

Sometimes hemoptysis is the very first symptom and there are tubercles in the lungs but they are not recognized.

Sometimes the cervical glands may be enlarged for months or years, then suddenly with considerable rapidity there is an involvment of the lungs.

Again the lung symptoms may be quiescent and the glandular symptoms are very active.

Most cases of chronic ulcerative pulmonary tbc begin with an acute catarrhal cold, catarrh of the naso-pharynx. There are cough, asmathic breathing anf wheezing rales.

Symptoms of chronic pulmerative pulmonary tbc:-

these will depend upon the stage it is found in, whether nodular, ulcerative, or cavity formation. They are not definite or uniform and the symptoms of all three stages may be found in one lung. they vary from the signs of small consolidations or small cavities up to a dif-

fuse infiltration with large consoliations. If the disease begins slowly and insiduously the patient feels a gradual loss of strength, general ill health.

It may follow any of the acute infections as influenza, typhoid, measles, whooping cough etc, and when you are expecting recovery from these the signs of ill health remain. There is increased debility, emaciation, loss of apetite, and then the physical signs usually associated with fever.

If the disease begins abruptly there may be chills, and fever, and hemorrhages with or without pleural symptoms.

Classified local signs--

(1) Pain. This may be absent, moderate, or severe; it may occur early or late; it is usually due to the accompanying pleurisy. In character it is sharp and stabbing; its intensity is greatly increased on coughing, deep inspiration; vomiting gives great pain. It is usually located on the lower and lateral parts of the thorax, but it may be in the apex, in which case it is not so severe. There may be pain between the scapulae behind. Be careful in these cases not to mistake an intercostal neuralgia, also exculde muscular rheumatism of the throacic walls.

(2) Cough:- This one of the most frequent and early symptoms. It may be absent or slight. A curious fact is that it does not depend upon the extent or severity of the lesion, but upon the personal equation of the patient. The cough is usually dry and hacking at first, but later as necrosis, ulceration and mixed infection occur, the sputum is increased and becomes purulent, while at first it was simply a glary mucous. If there be a cavity filled with secretion the cough may be paroxsymaland worse in the morning. If the larynx is involved the cough is hoarse and husky. It may be so sevre as to cause insomnia and an almost constant vomiting = Morton's Cough. When this occurs there is of course rapid emaciation and rapid loss of strength.

(3), Sputum:- It may be scanty or none at first but later become very profuse; it may be only streaked with blood or very hemorr hagic. Sometimes there are little Sago-like masses coughed up from the alveoli cells. When a cavity has formed the sputum is more likely to be purulent and contain purulent masses which may be green, yellow or ~~xxx~~ opaque, and sink in water = coin lumps.

Microscopically:- If examined early the sputum will ne found to contain alveoli epithelia, pus, blood, fat drops, elastic fibers and ~~xx~~ bacilli. The last are the most important and the only absolute scientific index of tuberculosis. When destruction of the lung tissue occurs you must exclude abscesses and gangrene. When these occur there isusually a bad odor, especially with gangrene, and the odor is frequently sweethish, as well as fetid. The calcerous areas of degener a tion are sometimes coughed up. There may be one or many and varying in size from a pea to a cherry. They may produce ulceration or result from ulcerations.

(4) In from 60 - 80% of cases of this form of tbc there is hempptysis. It may be a mere staining or quite a profuse hemorrhage. When it is a stainigg, it occurs from the hyperemia; and when it is profuse it is due to the ulceration of blood vessels is a necrotic area or cavity. The hemoptysis may occur frequently or at long intervals. Sometimes there is a capillary oozing from the capillaries in the wall of a cavity. When this occurs you will have the "rusty sputum". Bleeding frequently occurs in young apparently healthy patients without injury, trauma, and without a family history of tbc. Very frequently these cases show no bacilli in the sputum. Sometimes hemorrhages occur after violent exercise or trauma, and on examination of the chest nothing will be found, but after 2 or 3 days bacilli will appear in the sputum. This hemoptysis is most apt to occru in men and it recurrs in most cases. Sometimes it is due to a rupture of an aneurism on the pulmonary artery and it comes without warming or without other symptoms. Mental excitement, emotional disturbances, exercise and violent coughing all aggrevate the hemorrhage. It is curious fact that small hemorrhages give relief, but large ones cause anemia and often produce death. The blood may be mixed with saliva and be frothy; in color it may be bright or dark red. If the blood has previously passed into the bronchioles it may be coughed up as moulds.

(5). Dyspnoea.

This is a very important symptom. It may be small or very great in amount, depending upon the extent of the disease, the intoxication, fever,, and whether it occurs in the early or later stages when the muscles are atrophied, great emaciation and a consequent less demand for oxygen. Usually the respirations vary from 20 to 30 per minute. This rate is intensified by exertion, mental excitement, trauman, rapid extension of the disease, occurence of broncho-pneumonia, condierably by miliary tuberculosis, and markedly by pneumothorax, emphysema, cyanosis, and failure of compensation of the heart. The same is ture of enlargement of the heart or loss of compensationfrom any cause.

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Symptoms of Chronic Ulcerative tuberculosis:-

(1). Fever:

This may be the initial symptom and become very important. Sometimes there is an entensive involvement of the lungs with a normal or subnormal temperature; or there may be a very high temperature with but slight local lesions. The fevere is for themost part constant at some period of the day in the earlier stages when the tubercles are forming, although it may not be high. When the tubercles soften and caseate, the temperature goes higher, and accomapnying this there is emaciation, debility which is rapid and progressive. The temperature is usually at its maximum between 2 and 6 P. M., and at its minimum between 2 and 6 A. M. It may be continuous, remittant, or intermittant. Very early it is usually continuous. The remittant and intermittant types may occur early or late or they may alternate, depending upon the character of the lesions and the stages of the disease. In the remittant form the temperature is always above normal. It varies usually between 2 and 3 degrees between the maximum and the minimum.

The intermittant type is frequent, and especially is it characteristic of cavities, pus or mixed infections. It howver, may occur in the beginning and suggest malaria. When TBC does begin this way, with fever, sweats, perhaps chills, it is due to a sepsis, and the temperature usually begins to rise is the morning or just before noon, and then it reaches its maximum by 8 P. M. and slowly falls to its minimum point b etween 2 and 8 A. M. Sometimes the temperature goes down very low even to 96-5 or less. This is the type os temperature that is associated with severe sweats and have been known for a long time as Hectic.

(2). Sweats: Most the chronic ulcerative tubercular cases have sweats. They are rather a bad omen. The may occur at any time; most frequently in the stage of cavity formation. As to the time of the day,- they most usually come on in the early morning,hours when the temperature is lowest, or they occur during the relaxation of sleep. They vary from a very slight moisture to a perfect drenching. they are most frequent in the late stages and some cases do not have them at all.

(3). Pulse:- It is increased in frequency, especially with the fever. It is usually soft, compressible, regular and perhaps full. The rapidity ofthe pulse is important in the diagnosis of early TBC, for it is continuously above normal without local signs or constitutional symptoms. As suppuration occurs, the pulse becomes softer and more compressible and later weaker and smaller toward the end.

(4) Emaciation:

This symptoms indicates the reason this disease is called "Concumption". This is usually marked; the loss of weight is gradual and progressive, tho there they be periods of improvement with an increase in weight or one may hold one's own. Patient seldom die during the time they are ganing in weight. The loss in weight is in muscular and fatty tissue, andthis may be very great toward the end until the patient is nothing but skin and bone. This emaciation is due to the fever, toxemia, loss of apetite, feeble digestion and assimilation.

(5). Anemia: This is an evidence of the decreased nutrition; usually there is pallor of the skin, lips, and mucous membranes. The blood has nothing characteristic; early there may be a chloranemia; the HB is decreased; later when there is a mixed infection and

cavities, there is an increase of leucocytes, which may be as high as 50,000, with an excess of polys. This is due to a secondary infection of the other organs.

(6)Progressive weakness and debility

PHYSICAL SIGNS:

(1) Inspection:-

the shape of the chest is sugestive butthis is growing less so, for a great many cases look perfectly well whem their sputum will show the tbc bacilli. The characteristic chest is long, narrow, with intercostal spaces, more vertical ribs, and the scapulae stick out like wings. These chests are thin and flattened antero-posteriorly. Near the sternum may be very prominent and the sternum may show a funnel shaped depression. One clavicle may be more prominent than the other and the spaces above and below the clavicle are apt to show marked depressions. There is defective expansion of the chest; this occurs early and is important. This symptom is best elicited by looking down the chest wall and having the patient inthe recumbent position.

A wide cardiac impulse in the 2nr, 3rd. and 4th spaces on the left side is apparent in cases where the left apex is diseased and the lung is retracted.

(2). Palpation:-

There is deficient expansion. To elicit this you have the patient take a full, slow, steady inspiration.

The tactile fremitus is increased whenever there is infiltration, at the apex or base, and it is also much increased over cavities unless the pleura is greatly thickened, provided the cavity has free excess to a bronchial opening.

(3). Percussion:- The resonance is impaired over areas of Tbc infiltration, whether studded with nodules or caseated, and of course greatly impaired in fibroid cases. The note may be increased over cavities when they are empty ot greatly impaired, even to dullness or flatness when the cavity is full of fluid. With small scattering of tbc foci, you may get no change in the resonance. If you get deficient resonance above or just below the clavicle early in the disease, it is an important sign, because this is the first place to be infected. To get a perfect elicitation always compare the two sides; examining the patient while taking the ordinary breaths, both on inspiration and expiration, because that modifies the note.

In Tbc consolidations you get a tympanitic or tubular note. In fibroid cases it will be flat.

You may get a cracker-pot resonance over a cavity when it has thin walls, is near the surface, and the chest walls are thin, especially at the apex or just below the clavilce or behind in the supra- and infraspinous areas of the scapulae.

Sometimes in these cases, especially in the later stages, there is tenderness of the muscles and skin, and the slightest stroke may produce spasmodic contractions of the muscles.

All these signs depend upon the vibratory capacity of the chest wall.

(4). Auscultation:- As there is little air going into the air spaces the breath sounds are decreased. Here comapre the two sides of the chest on deep expiration after coughing to remove the exudate from the Bronchi and Trachea. Sometimes, early in the disease, you may not hear any insipratory sounds at all, while the expiratory will ne normal or prolonged and slightly tubular. Sometimes the murmur is harsh, or "cog-wheel and interrupted".

As consolidation increases, the breathing becomes harsher and more bronchial.

Rales depend upon the associated bronchitis. Early on inspiration or cough you will get a dry crackling or moist subcrepitant rale. As the bronchitis increases, the rales change and become louder, bubbling,and if they are dry you get simply a clinking sound.

In cavities you may hear a gurgling, resonant or cavernous sounds, or even the amphoric breathing. The unaffected portion of the lungs will have harsher breathing. Vocal resonance is increased in all stages.

During consolidation or cavity formation broncophany and some times whispered pectriloquy may be heard. You may get a pleural friction at any time and this may be limited above the scap

times whispered pectoriloquy is heard.

You may get a pleural friction sound and this may be limited above the scapulae, it may be either anterior or posterior, or laterally or be heard all over the chest.

When the layer of pleura over the heart is involved and also the pericardium, the "pleuro-cardial friction is heard. This is differentiated from the pericardial friction by having the patient stop breathing.

When the lung over the heart is consolidated and the heart is acting forcibly, striking on the consolidation and if there is an opening into a bronchus, you get a whiffling sound knwon as the " cardio-respiratory murmur." Systolic murmurs in the Pulmonary arteries are frequently heard and are supposed to be due to a contraction and retraction of the pleura.

Cavity Signs:-

These depend upon the size, the location from the surface, the secretion, whether or not there is an open bronchial connexion, the consolidation, much thickening of the pleura and effusions. Some times a cavity is overlooked. If there is no pleural thickening, the cavity is not large, chest walls thick and the cavity is deeply situated, then a normal note is gotten. But if the cavity is near the surface, filled with air, you get a tympanitic or amphoric note.

Special Signs:-

(1) Wintrich's Sign:- The percussion note is louder and higher, sometimes even tympanitic and if the mouth is open, the tongue protruded and quite breathing it is better heard. You will get a contrary sign if the mouth is closed. An interrupted wintrichs sign is obtained when the cavity has a free bronchial connexion.

(2). Gerhadt's Sign:- If a cavity communicates with a bronchus the tympanitic note changes with the change in bodily posture.

(3) C rack-pot:- This is elicited over large, superficial, thin walled cavities, and is best heard with the mouth open and with a quick stroke. It is characterized by a hissing chinking sound.

(4). Cavernous Sounds (So-called) This is a hissing, blowing, tubular, or amphoric note, and may occur in large cavities on inspiration and expiration.

(6) Rales:- These are usually coarse, bubbling, gurgling, loud, and sometimes even metalic. Of course, if the cavity is dry there are nor rales.

(7) Vocal resonance is increased always and whispering pectoriloquy is frequently heard.

(8). A-Succassion:- may be heard is a very large cavity filled with air, and fluid. The coin-percussion sound is heard frequently over very large cavities.

(9) A clear ringing valvular heart sound may be heard, especially in large dry cavities. Don't mistake the pseudo-cavity signs. These are produced by consolidation near a bronchus, by bronchiectasis. They high pitched, tympanitic, tubular, cavernous, and are very similar to the cavity signs.

COMPLICATIONS:-

(1). Respiratory System:- (a) Larynx is frequently involver. Sometimes this is the very first symptom, giving a husky voice, pain on swallowing, wheezy or asmathic cough, no expectoration and the patient is continually becomeing wweaker. There may be aphonia dysphasia, which is very painful and distressing when there is extensive ulceration about the glottis and pharynx. There is an associated bronchitis with inflammation of the pharynx and the regurgitation of fluids or food through the nose. Pneumonia is a termination of the cases, and it may take a regular or atypical course.

(b) Emphysema- of the unaffected lung and this may come on slowly and there may be a rupture causing a general emphysema.

(c) Gangrene of the lungs:- sometimes occurs especially in the walls of the cavities.

(d) The pleura is generally affected in these cases. It may be dry at first and it may heal leaving a thickened adherent surface These adhesions may be small and localized or there may be bands of adhesions interfering with the action of the lungs, or the adhesions may be general. There is frequently effusions into the pleural cavi-

ty often before the tbc in the lungs is recognized. The effusion may be purulent or hemorrhagic, someyimes it is milky. It may be encapsulated by adhesions. Pneumothorax is common and is fatal in 24 hrs. generally, but it may last for weeks or months, in this case it is accompanied by pyo-pneumothorax, which may be quite slow in its course.

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(2). Cardio-Vascular Sytem:-

(a) The heart's impulse is visible over a larger area and nearer the sternum. It may be heard in the 2, 3, and 4th, interspaces, especially when the lung retracts. The heart may be drawn up if there is much retract in the upper lobe and the apex beat is heard higher up. Systolic murmurs are heard over the heart and especially at the Pulmonic orifice. And sometimes there may be a Tricuspid murmur. This does not always indicate disease of the valves or endocardium, for it may be a functional murmur due to the state of the blood. The murmur may be rough and harsh without disease of the endocardium. It is held by all aurthors that pulmonary stenosis and all other congenital lesions abou the Pulmonary orfice, predispose to tbc, while those at the Mitral orifice are antognistic.

(b) Pulse:- For a long time the pulse may be full and soft, with a low arterial tension, but as the disease progresses it becomes weak, feeble and smaller. Sometimes the capillary pulse shows in the back of the hands and nails.

(c) In the blood and glandular system there is an early anemia, which belongs to the chloranemia type. The blood plates are increased. Later when there are cavities, suppuration and fever, the blood shows a marked leucocytosis. The W. B. C. may be increased to 50,000, chiefly polys.

An eosinophilia is considerable favorable by some and unfavorable by others.

(3). Gastro-Intestinal system:- The tongue may show all the febrile characteristics. It may be covered with a white or yellowish fur; or it may be clean and red. It is strangely clean, considering the nature of the disease. Ulcers and Thrush may develop in the later stages. Pharangeal tubercles may form, break down and form ulcers.

The saliva is impaired in its digestive power; the apetite poor; and there may be intense thirst. There may be symtpoms of a chronic gastritis, while tuberculosis of the stomach is rare. Ulcers of the catarrhal type may form. The mucous membrane is engorged and it may undergo and atrophy. At times the muscular tonus is decreased and the stomach then dilates.

The loos of apetite, nausea and vomit may be due to an associated gastritis; or sepsis; or it may be due to Morton' cough.

Early in the disease and especially in the nervous, there is hyperacidity. But later the acid is deficient in amount.

Intestines:- Constipation is the rule in the early stages, but diarrhoea generally occurs later. It may be due to tubercles in the bowel from swalloed sputum; or to catarrhal ulcers; or to amyloid changes.

The large bowel also shows ulceration. Sometimes there is extensive ulceration in the Illeum without diarrhoea. Ulcers, Haemorrhoids, and fistulae-in-anno are very frequent.

(4). Nervous system:- We all know of the cherful and hopeful disposition of the tubercular patients. But finally some other patients develop insanity, which usually takes the form of melancholia, associated with frightful illusions of a persecuting or tormenting nature. But frequently the mind remains clear to the end.

Nervous symptoms:- are generall those of a meningo-encephalitis, or the tubercles may be so located as to produce aphasia, hemiplegia. A basilar meningitis may occur, sometimes of the cerebral type, but most frequently the cerebro-spinal.

A neuritis may develop and produce a paralysis of the arm or leg, usually of the extensors, and occuring late.

(5) Genito-Urinary System:- Increased ~~xxixx~~ albumin, either febrile or due to an actual nephritis. As amyloid changes are ~~frequently~~ present you will get an increase in albumin, casts and frequently.

An associate dropsy also may occur.

Cystitis occurs as does pyelitis with pus and some times with blood.

The hematuria may be due to congestion of the kidneys and not to an actual renal lesion. Sometimes the bacilli are found accompanying the pus in the urine.

The testis frequently present a tubercle and this may be the first symptom of an on coming tuberculosis. The tubercle is usually located in the epididymus.

6 Cutaneous system:- The skin is dry and harsh, sometimes local tubercles appear on the hand accompanied by pigmentation of the chest and other parts of the body. This pigmentation is called chloasma phthisicorum, and it occurs most frequently when the peritoneum is affected. Brown or black stains occur on the chest or back and are known as pityriasis versicolor.

The hair is long, dry and harsh and sometimes very early truns grey over the chest.

The fingers are clubbed at the ends.

There is pallor, cyanosis; or a deep red sppt on the cheeks. These occur late in the disease.

A general cutaneous emphysema is not infrequently seen and is due to a tupture.

The chest muscles are irritable and painful on percussion, especially late.

There is a hypertrophy of the mammary glands, especially in men, and may occur only on the affected side. It is a chronic Interstitial mastitis and non-tubercular.

Diagnosis:- It is important that this bemade early. It is made upon:-

(1). The general symptoms:- as failing health; loos of apetite, loss of weight, anemia, cough, evening temperature, and hemorrhage. If all of these are present then you have a strong case.

(2) Local signs:- (a)- deficient expansion which is easily noticed below the clavicles. (b)- dullness on percussion; decreased vesicular murmur, which is sometimes entirely absent with quiet respiration on inspiration but immediately on expiration you will get a prolonged expiration or a broncho-vesicular murmur, in places where it ought to be simply vesicular. A fraction rub may sometimes be both felt and heard. (c) Tubercular bacilli in the sputum. One examination may not reveal the bacilli, hence it should be repeated again and again. The bacilli may come from a very small focus in the lungs which will give no physical signs. Again there may be no sputum, in these cases give a few small doses (10grs) of KI. Elastic fibers may appear in the sputum due to destruction of the lung tissue by ulceration.

(d) Tuberculin has been successfully used. In 75% of cases it gives a positive reaction early. In this method you should start on small doses during the afebrile periods and get a febrile reaction within the first 24 - 48 hours. A temperature of 101 is considered diagnostic. This method of diagnosis is impaired by the fact that other diseases as, syphilis, leprosy, actinomyces, chlorosis, and Hysteria give the febrile reaction.

(e) X-Ray pictures may reveal foci which are so small or so located as to produce no physical signs, and they may be detected 3-4 weeks before the bacilli appear in the sputum.

Differential diagnosis:-

(1). Bronchitis:- this is especially troublesome when both sides are affected. There isusually no afternoon temperature nor other consitutional symptoms. The rales are larger and more diffuse and vary more than in tuberculosis. Unless the tubes are full of secretion there will be no dullness on percussion.

(2). Bronchiectasis:- especially in the cavity stage. The symptoms are practically the same and unless the bacilli are found the diagnosis is almost impossible.

XXX(3). Fibroid Phthsis: Occurs in three forms:-
(a) Pure fibroid phthsis without tubercles.
(b) Tubercular fibroid phthsis- primarily tubercular and secondarily fibroid.
(c) Fibro-Tubercular, which is primarily fibroid and secondarily tubercular.

The Tubercular-Fibroid type sets in and pursues a slow course. It may be secondary to Tubercular Broncho Pneumonia, or Chronic Tubercular pleuritis, or to chronic Ulcerative tuberculosis. It may be limited to one apex and be surrounded by dense fibrous tissue, with a thickened pleura; or it may be in the lower lobe and become sclerotic. The lung may give the appearance of sclerosis with but little or no evidence of tuberculosis, but the bacilli will usually be found in the walls of a cavity or in an old encapsulated caseous area; or there may be tuberculosis of the other organs or the bornchial glands. The bronchi will be dilated due to the induration and contraction. The right ventricle of the heart is often dilated and hypertrophied, and sometimes the whole heart.

This form of trouble is chronic running from 10 - 30 years and the patient will appear to have fair health.

The lung tissue is hard, dense, alveoli obliterated; it resists cutting and creaks; the section is smooth, dry and greyish. Sometimes t he fibrous tissue undergoes caseation. The pleura is usually thickened and adherent. The unaffected parts of the lung tissue are frequently emphysematous.

Symptoms:- The onset is insidious, usually with a cough, and this increases with very severe paroxysms of coughing in the morning. Later it is accompanied by purulent expectoration. When the bronchi become dilated the sputum is frequently fetid in odor. Dyspnoea is very marked on any exertion. There may be little or no fever during the entire course of the disease. The emciation is slow and slight.

Physical signs:- (a) Inspection:- the chest is sunken, the shoulder on the affected side is lowered; when there is left sided tuberculosis the heart may be displaced and the impulse felt if the 2, 3, 4th, interspaces. (b) Palpation:- diminished expansion; tactile fremitus decreased; (c) Percussion:- dullness, even to flatness. (d) auscultation:- enfeebled respiratory murmurs, especially the vesicular, and at the apex the respirations may be cavernous; at the base bronchial and distant.

Prognosis:- This is a disease that lasts from 10- 30 years. The other lung may become actively affected and this will produce death sooner. Death may immediately follow a hemorrhage. Prolonged suppuration with diffuse amyloid changes in the spleen, kidney, liver and bowels make a bad outlook. If the right heart has diffculty in pumping the blood through the fibrosed lung there will be dilitation and slowed circulation with dropsy as a secondary result.

(2) Differentiate: Chronic Bronchitis, which is frequently on both sides and has no cavities and no bacteria.

Complications:- Lobar pneumonia, which is a frequent cause of death. Sometimes Broncho-pneumonia. Typhoid fever, erysipelas, eruptivers as measles, scarlet etc. These usually preceed, however, sometimes they follow or occur simutaneously. Fistula-in-anno is usually tubercular and an operation is usually followed by a rapid termination.
Chronic Nephritis and Arterio-sclerosis may occur.
Heart lesions as endocarditis.
A rthritis which is usually chronic. (The above complications are complication s of pulmonary tuberculosis.)

Concurrent infections in pulmonary tuberculosis:- Micrococcus lanceolatus, streptcoccus pyogenese, staphyloccus aurens. Tuberculosis is therefore a combined infection. The pneumonia may be due to the bacilli, their toxins, or to a secondary infection. Certainly the pus organisms hasten the disease and the breaking down of the tissue.

Modes of Death in Pulmonary Tuberculosis:-

(1). Asthenia- with a gradual increasing debility until death.

(2). Asphyxia- this is frequent in acute Miliary Tbc and the acute Pneumonic Phthisis, but is more rare in the chronic forms.

(3). Syncope- this usually occurs with extra exertion and results from fatty degeneration of the heart. Some will faint immediately after a severe hemorrhage. Embolism and thrombosis of the Pulmonary artery produce rapid fainting and death, which may also result from pneumothorax.

(4). Hemorrhage- this is usually due to the ulceration of a large blood vessel or an aneurism in the wall of a cavity.

(5). Cerebral symtpoms:- such as coma, which may be due to a meningitis, less often to a uremia. Remember that uremia may occur with nephritis in tuberculosis. Death in convulsions is rare. Thrombosis of the cerebral sini may occur, giving the symptoms of meningitis.

2-21-08.

TUBERCULOSIS OF THE ALIMENTARY TRACT.

(1) Lips:- are rarely affected with tuberculosis, but when is does occur it is in the form of ulcers, which are sensative and look like a chancre or an epithelioma. It may be the primary focus, but most often is it secondary to Pulmonary or laryngeal tuberculosis.

Diagnosis:- made by scraping off some of the ulcer and staining for the bacilli or by the inoculation in to the lower animals.

(2). Tongue:- is frequently subjected to tuberculous lesions. They appear, usually in the form of small granular bodies along the edges or back part of the tongue and form ulcers, which are irregular, hard, with distinctly uneven edges, they are usually rough and caseous and may extend. Their course is usually serpentine and extensive. These tubercles appear as greyish white masses. There are other organs affected, but the glands at the angles of the jaw are not enlarged. The ulcer does not yield to KI.

Diagnosis:- Same as above.

(3). Salivary Glands:- Rarely affected.

(4). Hard and Soft palate:- become studded with tubercles from direct extension or by infection from throwing infected sputum up from the larynx during coughing.

(5). Tonsils:- are usually the dorr of entrance when the cervical glands are involved. They undergo an infiltration and become studded with tubercles; the gland enlarges and has the same appearances as in other inflammations.

(6) Pharynx:- miliary granulations occur usually from extension from the larynx. This produces a very severe dysphasia. Adenoids in children are usually tuberculous from the beginning, and they may look like the ordinary adenoids.

(7). Oesophagus:- Rarely affected.

(8). Stomach:- this is a rare affection and is almost unknown as a primary affection, but the secondary form may occur.

(9). Intestines:- This may occur as a primary focus in the mucus membrane but most often it is secondary to Pulmonary Tuberculosis. Sometimes it is induced by a direct extension from a tiberculous peritonitis.

The primary form most often occurs in children, with enlargement and caseation of the mesenteric glands, or it may result in a peritonitis. It is rare in adults, however, it may begin in the Coecum and looklike appendicitis. Either hemorrhage or peritonitis may be the very first symptom of this condition, and they are due to ulceration. Sometimes the ulcers, heal, cicatrize, and narrow the lumen of the bowel.

When this occurs as a secondary infection, it is due to Pulmonary Tuberculosis in about 50% of cases. They most often occur in the Ileum, but they may occur in the Coecum or colon. They begin in

solitary follicles, agminated glands, or mucous membrane. The affected areas undergo infiltration, caseation and ulceration, and the lesion may be extensive.

In the Ileum and Pyer's Patches the ulcers are ovoid, but in the Colon they are round or transverse. The ordinary characters of these ulcers are:- Irregular, rarely oval or ovoid or in the long axis of the bowel; they are more usually transverse and completely surround the inner coat of the bowel; the base is infiltrated or it may be caseous. The submucous and muscular coats are usually involved, and there may be small tubercles on the serous coat. Perforation and peritonitis may occur, or the ulcers may heal, resulting in multiple stricture and stenosis.

Tubercular lesions in the coecum near the Ileo-Ceecal valve are important because they may form hard movable tumors in the right Iliac fossae, or the tumor may be immovable and tender and resemble a cancer. This produces stricture of the bowel, with alternating constipation and diarrhoea. Such cases may be permanantly cured by an operation. Sometimes a fecal fistula forms, with chronic thickening and appendicitis. The tubercular bacilli appear both in the feces and the discharges from the fistula.

(9). Rectum:- Fistula-in-anno is tuberculous in 5% of cases. This lesion may result from extension from the peritoneum, Fallopian Tubes, peritoneum or from the mesenteric glands (especially in children). The coils of the intestines are matted together with caseating and suppurating tissue, and perforation may result.

(11). Liver:- is always involved in acute miliary tuberculosis. (a) The tubercles may be small and microscopic in size. (b) In chronic tuberculosis the liver may be affected with large nodules and miliary tubercles.

(c). A single caseated mass may occcur, either formed by fusion of a number of small tubercles or be originally one focus. Here hepatitis or peri-hepatitis may supervene, especially in children.

(d) Tuberculosis of the bile ducts:- this is not infrequent. It forms little tubercles and the liver is greatly enlarged. Ir is really a tubercular colangitis. A section will show little cavities containing bile stained pus.

(e) Tubercular cirrhosis of the liver;- the increase of fibrous tissue may be only slight and chiefly in the connective tissue, accompanied by fatty changes and milliary tubercles. In the more chronic forms there is really a fibrous overgrowth, but this is rare.

Symptoms:- perhaps ascites; may be a tumor; and if there is an associate hepatities or peritonitis then there will also be the symptoms of these.

TUBERCULOSIS OF THE BRAIN:-

(a) acute miliary processes causing a meningitis and acute hydrocephalus. (b) chronic meningeo-encephalitis, most frequently localized and containg small tubercles. (c) Solitary tubercles occur both in the acute and chronic forms.

The symptoms of the chronic forms are those of a growing tumor, being insiduous and most frequently occuring in the young. They are usually secondary to a tuberculous focus in other organs, as the bones and glands. Named in the order of frequency the tubercles occur:-(1) in the cerebellum, (2) Cerebrum, (3) pons. There may be one or many, varying in size from a pea to a walnut in size; geryish yeelow, caseous, firm or hard, surrounded by dense connective tissue, which is white in color. These tubercles may under go calcification. They may be attached to the meningese at the bottom of a fissure and look as tho they were imbeded in the brain tissue. They may occur along the Longitudinal Sinus, producing presssure and thrombosis. A tuberculous tumor may excite a surrounding meningitis.

In the localized mingeo-encaphilitis there is tuberculosis of the pia also. The tubercles may penetrate the arteries, and as the grow the nou rishment is diminished from the pressure on the arteries, and softening of the brain tissue results. This condition may produce a heniplegia or aphasia.

TUBERCULOSIS OF THE SPINAL CORD:

The same as in the brain. An acute meningitis is usually cerebro-

spinal, and is secondary.

TUBERCULOSIS OF THE GENITO-URINARY SYSTEM:

All the rogans may be affected successively or simultaneously. The course is usually rapid and usually occurs where there is latent disease somewhere else. There may be bacilli circulating in the blood and will invade the kidnay, however it is usually secondary to other localized foci.

In the urinary system the infection is due to the following:-

(1). Heredity: the bacilli can be found in the fetus, and in the testicles of the newly born.

(2). Infection may be fromother foci, as the lungs and bronchial glands, and here the bacillis enter thru the blood.

(3). Direct extension from the peritoneum, especially in the femal from the Fallopian Tubes or from the Rectum, bladder, uterus or vagina. Vertebral tuberculosis may be followed by tuberculosis of the kidnay as a direct extension of the disease.

(4). Infection from without, as by, coitus or the bacilli may pass from the rectum into the urethera or vagina.

Etiology:- Age 20-40 years; Sex. male, 3 to 1. Look for the smegma bacillus. Sediment the ruine and innoculate into rabbits or guinea pigs and in 3 to 5 weeks you will get definite lesions. By the staining methods you can exclude the smegma bacillus, especially by use of Carbol Fuschin and then decolorizing with alcohol.

(1). Tuberculosis of the Kidnay:-

In general miliary tubercles there are tubercles in the kidney. It ma be primary or secondary; uni- or bi-lateral; and the bacilli may enter through the blood current. In chronic tuberculosis you will h ave nodules in the kidney appearing as a pyelitis. It affect the pelvis, medulla and cortex. The extension may be either up or down bu most frequently down.

Pathology:- The lesions begins at the apex of a pyramid, exten to the calises and then to the pelvis, and an early pyonephrithis may occur. The extension may be into the ureters or even to the prostate. The same changes in the foci occur here as elsewhere.

The kidney tissue may destroyed and replaces with cheesy or caseous deposites. If the bacilli enter through the blood stream, then the lesions are limited to the cortex. In this cases there are nodules and caseous areas, but little distruction of renal tissue.

In pyelitis there is ulceration, suppuration and caseation of the mucous membrane. Usually both kidneys are affected, but one more seriously than the other; or the affection may be unilateral. The capsule is adherent.

Symptoms:- The saem as those in pyelitis. There are no very early symptoms which are characteristic, ans the pus may be the only one present. If the bladder is also affected then you will have frequency of urination, and sometimes tenesmus. There may be pain in the affected side, which resembles renal cholic. It is dull in character, with remissions and exacerbations. Hematuria always occurs sooner or later in these cases. Tube casts, pus cells, and bacteria may be found in the urine and if there is much destruction of tissue then cheesy masses will appear.

General symptoms:- Chills, irregular fever, emaciation,. The lung ar e frequently involved and sometimes a general miliary tuberculosis of the whole body will supervene.

Physical signs:- (1) Inspection- a tumor is rare. (2) Palpation: tenderness, perhaps enlargement of the whole kidney or distension of the pelvis.

Frequently there is pyelitis from the caculi. The urine is frequently acid and this distinguishes tuberculosis from cyctisis.

Differentiate:- (1) Calculous Pyelitis: here the pain is mor severe and sudden, larger tumor, and hemorrhages are more frequent. Failing to find the bacilli in the urine, look for foci in some of the other organs, byt the inoculation of tuberculin tests.

(2). Tuberculosis of the bladder and Ureter:- Rarely primary, but usually secondary, especially to the pelvis of the kidney. It may come from the testes, usually extending through the Seminal Vesicles, Ureters and the prostate first.

Symtpoms: Are principally those of cystisis; slow in appearande, and apparently without cause. If there is ulceration you will get hemorrhage and bacteria.

Tuberculosis of the Prostate and Seminal Vesicles:-
This occurs in the form of nodules, varying in size from a pea to a bean. There is great pain and irritation when the bladder is involved. Caseation and perforation may occur.

Tuberculosis of the Testes:-
Occurs both primarily and secondarily, usually secondary, especially in children but may also occur in adults.
Symptoms: those of a painful, protract orchitis. A tumor is generally present, but is overlooked by the patient.
Diagnosis:- Differentiate:- Syphilis: here the body of the gland is more frequently involved and there is less pain; irregular nodule and the Vas Deferens not involved. KI don't do any good. Tuberculosis of the peritoneum is frequently associated and a General miliary tuberculosis usually occurs.

TBC of the Tubes, Uters, and Ovaries:

The Fallpian tubes are most frequently affected, either primarlily or secondarily. Here there is salpingitis, enlarged tubes, cheesy contents, and thickened walls. There may be adhesions of the fimbria or the uterus may be involved. It is usually bi-lateral, and it may occur in children. Abscesses may form, then rupture and set up a peritonitis.

Ovaries:- This is invariably a secondary infection. It is characterized by abscess formation and ulceration.

Uterus:- Not frequently involved, but is generally secondary to pulmonary of pelvic tuberculosis. It is really a tubercular endometritis. The mucous membrane is thickened at the fundus and tubercles form in the muscular tissue. Sometimes the disease passes into the Vagina and it may also affect the aplcenta.

Diagnosis:- age, heredity, other lesions; examine for the bacilli.

Tuberculosis of the Mammary Glands:-
This is rare. IT may be primary or secondary. Age 40 to 60, occurs in women nearly always.

Symptoms:- Fistulous ulcerations, discharges, cold abscesses, may be multiple. The axilary glands are most frequently involved. The pain is sharp and laceolate. It is usually chronic and you can generally find the bacilli.

Tuberculosis of the Circulatory System:-
(1). Myocardium:- Shows miliary tubercles in the acute forms of the disease; caseous tubercles are rare. There is sclerosis and the infection may be from the Mediastinal glands.
(2) Endocardium:- Rare; most frequently secondary and has an associated mixed infection. There are usually bacteria in the vegetations.
(3) Arteries:- The larger arteries are primarily rarely affected; usually by direct extension from without, however, it may occur in the walls of the blood vessels. The muscular coat and the intema are thickened and hence the lumen in narrowed. Ulceration from the outside into a vein or an artery from a Bronchial or mediastinal gland is most frequent. It may produce general diffuse tuberculosis of the lung or a general miliary tuberculosis of the entire body.

Prognosis of Tuberculosis:- Depends upon the individual and family diathesis, severity of the disease, associated diseases and complications determination if the individual and his financial condition. It is decidely more favorable when there is no hereditary taint, with previous good constitution, good health especially combined with good health and good apetite and digestion. Cases that are diagnosed early, suitable climate, out door life, rather insiduous onset than sharp attacks, with little fever and with much pneumonia have a better prognosis. Those that sleep and eat well do better. Cases that begin with pleurisy last longer and do better.

Bad indications:- High fever, poor apetite and digestion; progressive emaciation; frequent hemorrhages; insomnia; pain, sweats; diarrhoea; persistant Diazo reaction in the urine; complications and associated diseases, as: chronic nephritis, Gastritis, intestinal

tubercu losis, involvement of the larynx and acute pnumonia. The disease is frequently not so bad in those that contract it in old age, but here it runs an insiduous course and may not be recognized or shorten life very much. It is especially destructive to children, when it takes the cerebro-spinal form. The disease may pass into the lymph glands or bones and remain later and localized for a long time and then flame out when the proper environment is present .

Treatment:- (1). Prevention:- (a), Education of the doctors nurses, and public atlarge. The latter is best done by public lectures and lectures in the public schools by a well qualified and informed Dr. (b). A ll cases of Tbc should be reported to the public board of health. (c). Hygiene: prevtent permiscous spitting; (d) a great deal of attention should be paid to foods, dairy products; and also to the cows. There ought to be sanitoria, both public and private for the care of these patients. Care for the sputum either by disinfection, sputum cups or flasks.
(2). Isolation:- Every case should be isolated in his own room, with individual bedding and as little furniture as possible.
(3). Tuberculous families: all the delicate children should be taken care of because they are liable to have colds, sorethroats, catarrh, bronvhitis, and tonsilits, and these all predispose to the contraction of tbc. Prevent these children from getting their feet wet, andmake them live an active, outdoor, open air life, and wear comfortable clothing accoding to the changes in the weather. They should have an exceedingly nutritious diet. The chst may be drenched twice a day with cold water to stimulate the expansion of the lungs. Look out for the acute infections, as measles, whooping cough, and gripp, and also anemia. Give a tonic of: Cod Liver Oil; Syrup of Iodide of Iron and arsenic. And remove all enlarged tonsils.
(3) General Treatment:- Tuberculois of the bones, and lympahatics will sometimes heal spontaneously as pulmonary Tbc sometimes does. This healing is perfected by sclerosis, calcification or encapsulation A thickened pleura may sometimes check the spread of a lung lesion. If there is serious fever the patient should be confined in bed, as exercise does harm. Let them have lots of pure fresh air, and a low temperature does no harm.

Climate: This is sometimes a matter of considerable importance. Consider:- (1). Purity; (2) Equability. (3) Sunshine; (4) Dryness; (5) Altitude. A high altitude is better for some: others thrive in a dry warm climate; still others convalesce in a warm moist climate. An equable temperature is demanded for changes in the temperature are bad for these patients. A relativerly low temperature is better than a relatively high one, becase the former is stimulant, while the latter is a sedative. A rarefied temperature at high altitudes stimulates the respiration and does good by enlarging the air cells, sometimes enlarges the chest, and breathing capacity. But it is universally conceded that patients have to remain continuously in these climates for permanent relief. Sutiable cases for high altitudes are those where the diagnosis is made early; no serious lesions, little emaciation, and no cavitiesor desturction in the lungs. Thin and emaciated patients, with severe lung lesions and of a nervous temperament do better at low altitudes, even though it is cold. In recommending a change of climate always consider the financial side from the patient"s standpoint.

Feeding is especially important: Nausea and vomiting are frequently troublesome, and may occur early. Sometimes they are due to medication with Cod Liver oil and Creosote, in this event you must stop them. Sometimes a change of climate, decrease or entirely stopping the diet for a time; living in the open air and exercise will decrease the nausea and vomit and increase the digestion. If these are very traoublesome it is better to take smaller amounts of food at shorter intervals, as milk, eggs, butter milk, broths, albumin water etc, and later when the patient gets stronger, the diet may consist of fish, fowl, and as much fat as possible. The French wash out the stomach with cold water and give one quart of milk, one egg and 100 grams of finely ground meat three times a day. Raw eggs either sucked or taken with milk are fattening. Tr. Of Nux Vomica (10 M)m ~~dilute~~

Nitric or Nitro-Muriatic acid, preparation s of malt, CompounfTr. of Gentia will increase the apetite and digestion. If there ssa feeble digestion with rapid pulse and weak then give whiskey.

(5) Medicinal Treatment: Cod Liver Oild is the best and first drug given. It increases the nutrition and is especially good in bone and gland tuberculosis. Begin with 1/2 dr. and gradually increase the dose to 1 - 2 drs. It should be given t.i.d. It may cause an irritable stomach, nausea and diarrhoea. Cow's cream amy be substitued especially in children.
Creosote has no specific action but is attended with good efects given both internally and by inhilation. Begin with ℞ 1 M doses either in milk or in capsules and increase the dose to the tolerant point or a little below this. It may be given in solution with alsohol and some flavoring extract. When there is an irritating cough it may be taken by inhalation either alone or combined with alcohol, glycerine, terbine or some of the tar preparations. A good ℞ for inhalation is equal parts of Alcohol, Chloroform and creosote.
Tonics: Syrup of Hypophosphites 1 dr; Fowler's Solution- begin with 3 -(5 M, and increase to the tolerent point, which is indicated by puffy eyes, irritable stomach or diarrhoea. Compressed air with creosote has been used in some of the sanitoria with varying results.
Treatment of Special symptoms:-
(1) Fever:- When the temperature is above 100 rest in bed out in the open air, or in a well ventilated room. Give a few doses of Acetanalid or phenacetin, usually, however, sponging with tepid water is the best. (2) Sweating:- Dilute Aromatic Sulphuris acid 1dr. t.i.d. 1/100 Gr. atropine hypodermatically twice a day. Dovers powders 5grs. Zinc Oxide 5grs. If the sweating is accompanied with a cough then give 1/4 morphine hypodermatically; agaricin /℞ 1/8 gr. Or Use the alcohol sponges. Some do better when waering flannels. (3). Cough Creosote combine with alcohol and chloroform equal parts, or creosote alone; or compound Tr. Benzoin; or turpentine; Eucaleptus; alcohol, or formalin, poured on a pot of boiling water and inhaled through a cone with give great relief. If the cough is due to pulmonary cavities use counter irritants over the chest, in the form of hot turpentine stupe, hot mustard plaster, or paint on Tr. Iodine, but best is to apply adhesive strips. The laryngeal erosions may cause the cough, in this event paint them with cocaine solutions. For the night cough useHoffman's Anodyne; Heroine 1/8m or codine or Morphine. It the cough occurs in the morning a glass of hot water previously made alkaline by adding 1/2 or 1/4 teaspoonful of sodium bi-carbonate will give relief. Hot milk is also good. Aromatic Spts. of Ammonis, Ammonium Carbonate or Ammonium Chloride are also sometimes used. Always give morphine inspite of the presence of cavities for this will give sleep with a consequent increase of strength and better resistence The inhalations are better when there is a constant cough accompanied with secretions. (4) Diarrhoea:- Bismuth 20 grs; Dover's Powders 2-4 grs. If the lesions are in the lower bowel then give opium and starch water; or the lead and opium pills; or the acid diarrhoea mixtures; Salol, Beta Naphthol or some of the other intestinal antiseptics may be given. (5) If there is a dysphasia, due to the ulcers in the larynx, then paint them with cocaine solutions. (6) Vomiting Change of food or stop it entirely for a time; give calomel in broken 1/19 gr. doses every hour until one grain is taken. Also cerium oxalate; Chipped ice; hot applications to the abdomen.
(7). Hemoptysis:- Rest in Bed; Morphine 1/4; atropine 1/150; if there is aprofuse hemorrhage then give a saline infusion under the skin.

LEPROSY

is a chronic infectious disease produced by the bacillus Lepra. It is characterized by tubercles in the skin and mucous membranes = Tubercular leprosy: or by anaesthetic areas in the skin = Anaesthtic leprosy. Both of these forms may be combined lare in the disease.
Etiology:- It is due to the lepra bacillus, which is very much like th e tubercle bacillus.
Modes of infection.-
(1) Inoculation: this is more or less questionable, except by direct contact.with the lesions.
(2). Heredity:- This is also questionable.

(3) Direct contagion is positive, and inoculation may be received from abrasions, saliva, expectoration, secretions from the nose and throat, from the urine, and from the milk of a nursing mother. As the bacilli also occur in the dust infected areas and other abrasions are liable to be inoculated.

As to age- all ages are liable, but it is most frequent between the 20th and 40th year. Leprosy is rare in childhood.

Sex- about equal. A fish diet is said to contribute.

Pathology:-

The bacilli grow and develop in clusters in the tuberculous nodules; in the skin and mucous membrane; also in the anaesthetic and pigmented areas. The bacilli may be seen in the epitheloidcells and lymphocytes. These cells are known as "Lepra Cells". They originally come from the lymphatics abd blood vessels, and the change in character is due to the bacilli.

A connective tissue surrounds this mass and forms a granulomatous area. The bacilli may be found in the lymphatics, spleeen, liver, nerves, and frequently in the blood, and also in the nodules. The little nodules project from the skin, and because of the diminished blood supply they caseate, ulcerate, or sometimes absorbed. A dense connective tissue surrounds, which later contracts, producing great distortion of the surface. The characteristic "Lion Face" is due to this contraction.

The entrance of pus organisms will greatly increase the destruction of tissue. The mucous membrane of the conjunctivae, cornea, larynx, etc, all are gradually involved. They may from deep ulcers, sometimes the fingers and toes are lost.

In the anaesthetic form, there is a neuritis, due to an atrophy of the nerves fibers, which is produced by the bacilli.

Symptoms:- (1). Tubercular Leprosy: The incubation period is from 2 to 5 years, sometimes shorter, and sometimes longer. For a long times the patient may have vague prodrones suchas, drowsiness, chilliness, irregular fever, and debility. Then there occur areas of cutaneous erythema, which are sharpely defined and frequently hyperasthetic. This is known as Macular leprosy. After a time these areas become pigmented. Frequently these changes occur without the development of the nodules, then the areas become anaesthetic, the pig disappears and the skin becomes white. This is the Lepra alba. The erythematous spots most frequenlt appear on the face, arms, and knees. Accompanying them there is a loos of the hair, eye brows and lashes. Finally the mucous membrane of the mouth, throat, and larynx particularly become involved, with a consequent harsh voice, and probably aphonia in the later stages. The conjunctivae are frequently attacked, producing a leprous keratitis, resulting in loss of sight.

The tuberculous areas are reddish brown in color and the covering skin becomes thick and scaly.

(2). Anaesthetic Leprosy:- This form has no external resemblance to the other. Usually it beging with pains in the limbs, and appearance of areas of hyperasthesia or of numbness. Then the trophic changes soon occur; small bullae come out, which may disappear with ulceration or leave anaesthetic areas. There may be a loss of sensation without the formation of macules. Sometimes the nerve trunks become large and nodular.

2-28-08.

The fingers and toes may be lost; the muscles over the affected area may become atrophied. The disease is chronic and may run for years.

Diagnosis:- Sometimes this can be made early from the erythematuous spots; which are at first hyperasthetic, later anaesthetic, and still later the macules disappear entirely. If the nerves are nodular and enlarged, this is a positive diagnosis. The bacilli may be found by staining scrapings from the ulcers or the serum squeezed out of the excised nodules. The nasal secretions may also contain the bacilli.

Differentiate:- (1). Tubercular syphilis.
(2). Syringomyelia

Prognosis:- The disease is chronic, running from 10 to 30 years.

Treatment:- Isolation. In these colonies chaulmoogra oil is given in 1 to 2 dr. does t. I.d.;and with this treatment a good many re-

coveries have been reported. Gurjun oil in 5-10 M doses has been given b ut it is not so efficatious. Calmette's antivenene ijected hypodrematically in 10 -30 cm, doses has been followed with good results.

Febricula or Ephemeral fever,

also sometimes called simple continued fever. It is a short febrile attack with no local lesions but depending upon many causes.
(a fever that lasts not longer than 24 hours = ephemeral; while one that lasts longer than this, 1 to 2 days = febricula.)
Etiology:- (1). Gastro-Intestinal Type:- in which there is nothing very definitely known. But errors in diet, ingestion of tainted foods, and ptomains, catarrh in children or bronchial inhalation of foul odors as sewer gas, may contribute.
(2). Mild or abortive type- appears in all epidemics of typhoid, yellow, scarlet, and dengue fevers, influenza, measles also in rheumatism and erysipelas. Here the fever lasts for a few days without other symptoms.
(3). Nervous form- in whih sun stroke, heat exposure, nervous exhaustion, and neurasthenic conditions contribute.

Sy mptoms:-

The onset is sudden with chills and vomiting: in neurotic children a convulsion may occur. There is headache, loos of apetite, coated tongue, and the temperature rapidly rises to 101-2-3 and con tinues for 2 - 7 days. It ends, usually, by crisis. The urineis scanty and high colored; herpes frequently on the lips, and there may be irritability going on to restlessness even to delerium. The patient is constipated. The pulse is full and bounding. With the crisis there may sweats, diarrhoea or free urination.

The onset, instead of being abrupt, may be slow and gradual with malaise.

Di agnosis:-

Exclude all other fevers by their characteristic local signs and general symptoms. Remember that this fever has no eruption and pursues a short course.

Look out for scarlet fever, typhoid fever, malaria, tubercular meningitis, all the mild infections, a slight bronchitis and tonsilitis Always examine the throat for tonsilitis and look out for a skin eruption.

Prognosis: good.

Treatment:- if severe, rest in bed, otherwise confined to the house. Give a few doses of castor oil or calomel and keep the bowels open. For the fever, use hydrotherapy and cooling drinks. Give a few doses of Tr. Aconite, or Sweet Spirits of Nitre, or some of the coal tars.

Lead Poisoning:-

also called Plumbism and Saturism. It is a chronic intoxication due to the slow absorption of lead. It is characterized clinically by cholic, paralysis and frequently by cerebral symptoms.

Pathology:- There is normally a small amount of lead in some of the human tissues, but when an abnormal amount is taken lesions occur in the muscles, peripheral nerves, liver, kidneys and mucous membranes.

The muscles are pale, yellowish, fatty, and in advanced cases the y are fibrosed.

Peripheral nerves, show a parenchymatous neuritis, especially in the nerve endings of the muscles, and the nearer you get to the spinal cord the less marked is the lesion. Usually the cord is not affected at all. In the primary atrophic form the ganglion cells in the anterior cornu and degenerated.

Intestines: in the acute and fatal cases there is frequently a marked enterocolitis.

Vascular system: shows an arteriosclerosis, especially marked in the liver and kidneys.

Brain:- vessels are sclerosed and shows a meningitis.

Etiology:- The disease is wide spread and occurs especially among workers in lead as painters, plumbers, printers, sometimes in dress-makers. The personal susceptibily varies a great deal. Adults more liable than children, due to exposure; women more than men; and animals also have the disease. Acid wines and ciders after being in contact with lead are posionous. Hair dyes, false teeth, silk thread, candy and cake also sometimes contain enough lead to produce the condition. It may also be produced by continued medication wi th Lead.

The abosrption may be through the skin, lungs, or the stomach. There is free absorption through the lungs but perhpas most free thru the stomach. The deposition may be any of the tissues.

The elimination is thru the kidneys, skin and perhaps thru the bowels.

Symptom s of chronic lead poisoning:-
These may occur at any time from one month to years after the time of continued exposure.

(1) Anemia: the occurs early and is marked. The red cells and Hb are decreased about equally, but the Hb is no often lower than 50%. The red cells are pale, irregular in outline, and show an basophilic granular degeneration. Frequently a small leucocytosis is present ,- 12000 to 20000. Nucleated red cells may also be seen.

(2) Nutirti on is poor

(3). a blue line on the gums is seen in many cases. This is a deposition of lead sulphate in the gums, formed by the action of sulphureted hydrogen in the tartar, perhaps in the breath, on the lead deposited in the papillae of the gums. The line may appear, then disappear or it may remain for a long time. Do not mistake it for the line in "blue gum negroes", or for the blue line, which is somtimes seen, and can be easily brushed off. The blue line in chronic lead poisoning cannot be brushed off. This is best seen about the lower incisors, and canines.

(4). Cholic: this is a common symptoms and occurs about the uumbilicus. It is usually preceeded by gastro-Intestinal symptoms as constipation. It may be paroxysmal and relieved by pressure. The abdominal muscles may be retracted and rigid. Frequently a dull pain may remain between the paroxysms of the chloic. Vomit may also supervene. The heart's action and tension of the pulse may be increased dur ing the paroxysmal attacks of cholic. An associated diarrhoea may be present for years or months before the poisoning is recognized.

(5). Paralysis:- Are frequently seen, they may be localized or general. They are rarely the primary symtpoms.

(a) Wrist-drop- which is the most frequent, may be bilateral or unilateral, and varying in degree and accompanied with no fever. There are several forms:- Antio Brachial, in which the extensors of the fingers an d writts are affected. This is essentially a musculo-Spiral paralysis and produces both wrist and hand drop. If it is continuous some of the joints may become partially dislocated, and the tendon sheaths become filled with tumors, which are known as Gruebler's tumors. (2). Brachial form- here the Biceps, Deltoid, Brachialis- Antiacus, and Supinator Longus(Sometimes the Pectorals) are affected. This is usually bilateral and may be primary. Aran - Duchene type- in which the short muscles of the hand, especially those of t he Thenar and Hypothenar, perish away. Peroneal form- here the Peroneals, Extensos Communis Digitorium, Extensor Longus Pollis are paralyzdd, producing the stppping or steppage gait. The Laryngeal form, in which the adductors are affected, but this is rare.

(b) A general paralysis may be gin with the wridt drop and gradually involve the extremities. It may be sudden, occuring in a few days and ascending rapid and involving all four extremities, which become atrophied. Here the diaphram is also involved and results in death. This form is rare. The electrical response of the nerves may be present in varying degrees depending upon the severity of the lesions.

Accompanying this general paralysis are pains in the legs and joints, cramps in the muscles. The joint affection is known as saturine arthralgia. Usually however there is no impairment of the sensation. Tremors of a fine or course character may be present, beginning in the hands. They are usually constant and increased by volitional exertion.

(6). Cerebral symptoms:- These are numerous and important. Some times they are called lead-encephalopathy. They are delerium, coma, optic neuritis, neuro-retinitis, aphasia, convulsions, hemiplegia, amaurosis, hysterical symptoms and insanity. The most frequent are delerium and coma. These may occur suddenly and be accompanied by tremors and hallucinations. Epileptoid convulsions may occur also

Hemianopsia. The hysperical symptoms are most frequent in girls. Intense headache is a marked symptom. The insanity usually takes the form of mania or melancholia.

A saturine Gout has been described in which the heart is hypertrophied, blood vessels sclerosed; contracted kidneys; There is a high tension pulse accompanied by tremors and decreased elimination of area and uric acid. This condition was so named because is resembles gout and because it is supposed that lead favors the deposition of urates in the joints, especially those of the big.toe.

Diagnosisi:- is made upon the history of exposure and the clinical history given.

Differentiate:- (1). Alcoholic paralysis:- this most frequently occurs in the lower extremities; may affect all four limbs; and is accompanied by or preceeded by sensory disturbances.

(2) Other Cholics- as Renâ, Hepatic etc, by their oen particular characteristics.

(3). Some times appendicitis: or enteralgia from other causes.

Prognosis:- It is good in the mild cases where there are no serious changes in the muscles and arteries. But where there is a paralysis with degeneration, the prognosis is bad. Primary atrophy of the muscles is especially unfavorable. The same is true of lead encephalopathy.

Treatment:- Preyention; prophylactic measures should be observed by all those working in lead, incleanliness of the person, especially the hoands before eating. A respirator should be worn to prevent the inhalation of the lead ladened dust.

Medicinal treatment:- for the acute cholic attacks, give morphine, or apply hot turpentine stupes, or make pressure on the abdomen. For the constipation- give magnesium sulphate and keep this up. After the acute symptoms have disappear give KI (5-10 grs) three times a day.

For the anemia give iron.

For the paralysis- strychnine, massage, electricity, either galvanci or faradic.

If there is arterio-sclerosis with a high tension pulse give sodium nitrite (2-5grs), or nitroglycerine (1/100gr) three times a day.

Chronic Arsenic Poisoning:-

This is an intoxication due to the gradual absorption of arsenic. In the acute forms it is frequently produced by suicidal intent or by accident.

Acute Symtpons:- Intense pain in the stomach with cholic, diarrhoea and tenesmus, frequently collapse. Sometime after these symptoms paralysis may ensue.

Chronic ~~Symtpons~~ Form;-

Pathology:- Degeneration of the peripheral nerves; the anterior cornual cells may be affected. The liver and kidneys show a granular degeneration.

Etiology:- The individual predisposition varies very much. Neurasthenics who have a craving for tonics may contract the disease from continued medication. The same is true in chorea and anemia. Other sources of the poison are: complexion remedies, artificial flowers, wall paper, carpets, rugs, toys, some of the analin dyes, and the glucose in beer. Taxadermists and glass makers are especially liable.

Symptoms:- Anemia- loss of apetite, weakness, emaciation, dryness and irritability of the mucous membranes, especially the eyes nose, and throat; nausea, vomit, diarrhoea. The eyelids may be puffy or show above the brows. There is usually epigastric pain and tenderness, tenderness; gums red and bleed readily; sometimes salivation, parathesia as numbness and tingling about the fingers, alopecia and dysentery. The skin shows a marked pigmentation or bronzing resembling Addison's disease; also eczema, herpes, urticarcia. The urine shows albumin, casts, and sometimes blood.

The paralytic symptoms of those of peroneal form appearing first in the leg, later affecting the arms. There is muscular inco-ordinatin like ataxia; fine tremors especially in the arms. Contractures and the steppage gait may be noticed. Electrical responses of degeneration may appear before there is a loss of power in the muscles.

The arsenic is eliminated especially by the kidneys. Some show a


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great tolerance to arsenic; others not.

The complexion may be clear and waxy and accompanying this is a gloomy expression to the face, headache, neuralgia, hysteria or even hysteria.

The sensory disturbances are generally manifested by the paraasthesias, as described above. Erythromelagia is frequently seen.

Diagnosis: Depends upon the history with the above symptoms.

Differentiate:- (1) Lead poisoning.
(2) Alcohol poisoningn-

Prognosis:- Good.

Treatment:- Remove the source of poisoning; give a purge to aid in the elimination through the skin, Kidneys and bowels. Give KI (5-10 grs) three times a day. Prescribe a nourishing diet and tonics as strychnine and iron. For the paralysis- massage and electricity.

3-4-08.

SUN-STROKE.

also called heat exhaustion, prostration, insolation, thermic fever, heat stroke, and siriasis. It is a condition produced by long exposure to excessive heat. One form is heat exhaustion and the other is heat stroke or sun-stroke.

Etiology. It usually occurs in the Sun-stroke or Thermic fever type, in those who are working hard in the excessive heat of the sun, where the atmosphere is hot still and moist. Soldiers, farmers, brick-layers, masons, workingmen, working in the open sunskine are especially liable, and particularly those addicted to the use of alcoholics.

Heat exhaustion does not neccarily require the direct heat of the sun in its etiology, for it may occur at night and without the sun, in bakers, coal stokers, laundrymen, engineers and those people who are confined in hot close basements. This condition, or heat exhaustion, is produced by the action of excessive heat on the heat centers or the vaso motor centers in the medulla. It is claimed by some to be autotoxic in origin, and the heat is only a contributory cause.

Pathology:- There is an early and marked rigor mortis. The putrifactive changes of the body occur early becuase of the excessive temperature. The brain, membranes, and cord may chow a venous congestion as well as other organs of the body as the lungs, spleen, and the conjuctivae, which may show a marked engorgement. The left ventricle of the heart is usually contracted while he right ventricle is dilated.

The blood is fluid and dark; the r,b.c. are crenated and do not readily form rouleaux. Extravasations, in the form of petechiae, or ecchymoses, may occur into the skin, serous membranes of the cavities, sympathetic nerves and ganglia.

There are certain degenerative changes found in the liver, spleen, and nerves, especially in the parenchyma.

The toxic quality of the urine in increased.

Heat exhaustion with a lowered temperature represents a sudden vaso motor paralysis, while sun stroke is due to paralysis of the center in the Medulla that regulates the distribution and disposition of the heat in the body, heat more heat is produced and less is given off.

The symptoms of sun-stroke are divided in those (1) of the apoplectic or asphyxial type and (2) those of the hyperpyrhetic type.

Symptoms of the asphyxial or apoplectic form:- The onset may be sudden and terminate rapidly and fatally. In this case there is a sinking down with heart failure, dyspnoae, coma, perhaps convulsions. More frequently, however, with the excessive exposure to a very hot sun, the patient feels weak and dizzy, has a head ache of a throbbing character accompanied by dyspnoea, nausea, vomiting and a sense of oppression and depression, and sometimes by chromaropsia. Such a case may go on with his work for a while then sudden fall in a convulsion, followed by coma and finally death. Or the case may take a different turn, being characterized by restlessness, flushed face full rapid pulse (the artery feels full and bounding), the breathing is increased and may be labored or stertorous. The pupils are at first

dilated,later contracted. -There is frequency or urinationand constipation. The skin is hot, dry, and perhaps shows petechia. The tongue is moist. When delerium occurs it may be of the wild or manicial type.

The temperature rapidly rises to 102-6-8-10. In the fatal cases as the coma deepens the pulse becomes more rapis and ffeble, changing in character. The respirations are more hurried and shallow, succeeded by Cheyne-Stokes type and death follows in 24 - 36 hours or even earlier.

When the case is favorable the fever declines by lysis and it may take 2 to 3 days for conciousness to return.

Some of the more severe cases are followed by sequallae as, an inability to stand a high temperature; restlessness and other nervous disturbances; a failing memory and concentration of the mind may be impossible; and sometimes a neuritis.

(2)Symptoms of the Hyperpyrhectic type:- this is simply an exaggeration of the above with a more rapidly rising and higher temperature, which may be 110-12-15. The coma here is more rapid and the asphyxial symptome are very marked. Some of these cases are complicated by pneumonia, memingitis, nephritis with uremia; sometimes cardiac paralysis or cardio-respiratory paralysis.

Symptoms of Heat-exhaustion:- These may be gradual or sudden.
(a) Gradual- the onset is with faintness, dizziness, nausea, thirst, drowsiness, pain in the loins, stomach, or abdomen. There is a sensation of numbness and tingling over the extremities, later the y are cold,clammy, and show a pallor. Muscular weakness increasing to prostration, with a small, feeble, rapid pulse; sighing respiration, fainting and collapse. The temperature is subnormal and may be as low as 95. Conciousness is not so frequently disturbed.

Diagnosis:- is made upon a history of exposure; conditions surrounding the patient, and the presence of the above symptoms.

Differentiate:- (1) Alcoholism. (2). Apoplexy. (3). And anything that will produce coma such as uremia.

In diffenentiating sun stroke from the above consider:- high temperature, unconciousness, small pupils pupils, dry, hot, flushed skin also the history of exposure. While in differentiating heat exhaustion it is almost the direct opposite:- moist pale skin, shallow sighing breathing, pulse fast, small, and soft; subnormal temperature; senses retained or concious ness not deeply or so much clouded.

Prognosis:- Good in most cases; better in heat exhaustion thanin Sun-stroke.

(of heat prostration).

Treatment:- Prevention a well ventilated and cool sur roundings: abstainence from alcoholics; free bowels and active skin. Medicinal: If the temperature is very low, stimulants as a hot bath are indicated. Some of the ammonias as the amomatic Spts. (1-2 teaspoonfulls) or ammonium carbonate (5-10grs); strychnine 1/20-1/30 grs hypodermatically; digitalis or digitaline. Remove the patient from the causitive environment and put him in a coll well ventilated room.

Treatment of Sun-Stroke:- A rapid rise of temperature immediately requires a cold bath; cold pack; ice pad; ice to the head; or chuncks of ice may be rubbed over the body and face; an ice cold enema high into the rectum is generally followed by rapid lowering of the temperature.

If there is a marked cyanosis in very full blooded patients or even in convulsions, bleeding may be resorted to. Or give whifs of chloroform until the convulsions cease; or morphine 1/8 -1/4 hypodermatically. The salines or calomel in small doses should be given until the bowels are free.

It is not necessary to reduce the temperature below 100. As an afyer treatment- prevent complications and exposure to infections.

Anemia

This is a decrease in the quantity of the blood as a whole, and is called oligemia. A decrease of the r.b.c. alone is called oligocythemia: a decrease of the Hb = Oligochromemia; a decrease of the albumin and fluids especially = anhydremia.

(1) Local anemia The vis a tergo produces the circulation everywhere and there are variations according to the needs in the different parts of the body. This is under the control of the vaso motor system. Sometimes there is a deviation of blood from one system of vessels to another, which may be produced by external or internal conditions. A patient, who is not full blooded, may suffer an attack of syncope from local anemia of the brain, because the abdominal vessels are filled up with blood.

Emaciation, pain, reflex conditions as, removal of pressure suddenly may be exciting causes of local anemia. Enteroptosis may drain the blood away from the brain and cord, and produce a local anemia. In Raynaud's disease there is a marked anemia produced by a localized spasm of the blood vessesl in the diseased area.

Diagnosis: is made upon the pallor of the skin and mucous membranes, languor, dyspnoea, palpitation.

This local anemia, as well as some others are to be differentiated from:- (1). A condition of hereditary pallor in people who have good health. (2). A pallor in others, accompanied by nausea, pr oduced by the constant use of alcohol. (3) All other conditons that produce a local anemia, as Bright's disease, heart disease, and arterio-sclerosis. While all these are generally associated a general anemia, there may be a local anemia. (4). The pallor that is often ssen in the morphine and opium habit xxxxx especailly when the drug is removed, but as soon as the drug enters the symtem again the color to the face will return. (5). Also the pallor accompanying chronic metallic poisonings.

II. General anemia:- (a). Primary or essential; or cytogenic; (b) Secondary or symptomatic, which is the most frequent.

(b) Secondary Anemia: Etiology:- (1). Hemorrhages, either traumatic or spontaneous; rapid or gradual. Rupture of an aneurism or erosion of a large vessel may also be the cauitive agent. A secondary anemia most certainly follow post partum hemorrhages. Death may result from the rapidly lowering of the arterial blood pressure. Metrorrhagia, hemoptysis, purpura, or scurvy may allow the escape of enough blood to be followed by a secondary anemia.

The blood is rapidly regenerated in from 1 to 10 days. The watery elements and salts are withdrawn from the alimentary tract. The albumin is rapidly restored; the r.b.c. later and last of all is the Hb.

Pathology of the blood:- The r.b.c. are decreased in number; and poikilocytosis is more or less marked. Micro- and Macrocytes are seen. The corpuscles are paler and normoblasts occur very early, and even free nuclei may be seen. An absolute or a relative leucocytosis may also occur, in which there is an increase of the polymuclear neutrophilic leococytes, while the small monos are decreased. Of all the constituents the Hb is decreased the most. Sometimes megaloblasts are found. Polychromatiphilia and basophilia frequently occur.

The hemorrhages are repeated the regeneration of the blood is delayed; there is a very low Hb content and the lymphocytosis may occur.

(2). Secondary anemia produced by an excessive loss of albumin In Bright's disease, long continued suppuration, lactation, dysentery, and with rapidly growing tumors the albumin of the blood is greatly drawn upon. Here also the poikilocytosis is well marked; the corpuscles are smaller than normal; the normoblasts are decreased. There may be an early leucocytosis, but if the condition is continued the leucocytes may be decreased

(3). Inanition: as a cause of secondary anemia, xxxxxxxxxxxx decrease in the quality or quantity of the food or both; poor assimilation, sclerotic gastritis, cancers, dysphagia, or stricture.

Here the plasma is chiefly decreased.

(4) Toxic Anemia: Minerals such as lead, arsenic, mercury, and phosphorous may induce the condition. Organic diseases or infections which destroy the cells, as malaria, typhoid, and acute articular rheumatism and tuberculosis and diphtheria, may also be casitive agents.

Symptoms of secondary anemia:- Pallor; mental and muscular weakness; an early sense of fatigue; neuralgia and coolness of the skin, dyspepsia dyspnoea, palpitation, weak pulse, poor apetite, and and disordered digestion.

Prognosis:- Depends upon the cause, its affects, and the possibility of its removal.

Treatment:- Remove the cause, put the patient in the open air, and prevent fatigue and over work.

Give tonics as, Gentian, nux Vomica; Liquid extracts of Malt before meals. Prescribe a nourishing and easily digested diet.

Give Iron for the anemia, in the form of freshly made Blaud's pills(begin with 1 or 2 and then increase the dose) or Tr. ferric chloride (5-10m). or the syrup of ferric Iodide. And keep the bowels open with the salines of casçara.

II. General anemia (Con't) - (a) Primary anemias:- also called chlorosis or green sickness.

Etiology:- It occurs generall in young girls at the adolescent period,(12- 20 th. year); and most often in blondes. The condition is liable to recur and run for a long time. The time of preference is during the sexual evolution. Heredity plays some part especially in tuberculosis, and is associated with some deficient development of the circulatory system or genital organs. Unhygienic and ill-ventilated sourroundings, ill nourishment, and over work predispose, the same is true of emotions, tigh lacing and autointoxication.

Symptoms:- The onset is slow and marked by languor, emaciation, mental and physicial depression associated with irritability and a constant sense of fatigue. There is motor weakness, dydpepsia, fainting from the slightest cause, palpitation. Often headache occur early and veritgo may be marked, either with or without headache. constipation is usually present. The menses are irregular and decreased. The physical appearance frequently shows no emaciation and there may a considerable amount of subcutaneous fat retained, or it may be increased.

The surface of the skin has a greenish tint and the skin over the joints may be pigmented. This greenish tint differs from the muddy pallor of cancerous cachexia; from the yellow(lemon) color of pernicious anemia; from that blancked white aspect of hemorrhages; and from the saffron yellow color of jaundice. Sometimes the cheeks are red and the this is known a chlorosis rubror. The sclera are peraly or blueish white. The face is puffy and there may edema of the ankles.

Gastro-Intestinal symptoms:- The apetite is poor and frequently capricious, with a craving for dirt, chalk, slate and acids. The gastric juice is usually very acid and there is distress and pain after eating. Vomiting, regurgitation of food, gaseous eruptions may also occur. Frequently the stomach is dilated. There is nearly always constipation, sometimes diarrhoea alternates. There may be gastroptosis. The tongue is pale, flabby, dry and indented.

Circulatory symptoms:- dyspnoea, palpitation, syncope, vertigo. The heart is sluggish with a consequent coldness of the hands and feet. If the heart is dilated the transvere area of dullness is increased. A systolic marmur may be heard at the base or apex, usually at the base, it is soft in character. It may be heard hloudest at the Pulmonary area. Pulsations in the Carotids may be very marked. The bruit de diable or humming-top murmur may be heard in the Jugular veins. A thrombosis of the veins of the head, neck or extremities may occur and produce Pulmonary embolism .

The pulse is soft and full; sometimes fever occurs.

Among the nervous symptoms are: headache, hysteria, depression, Hyperasthesia, Tetanus Aureum; and the ability towrite upon the skin.

The urine is usually pale, of a low sp.gr. and free in amount.

JUNIOR & SENIOR PATHOLOGY. SESSION 1907-1908.

Lecture 1. 10/3/'07.

BLOOD CIRCULATORY APPARATUS

Diseases of the Pericardium, Outline of:-

I. Anatomical Consideration
- 1a Visceral Layer
- 2a Parietal Layer
- 3a Endothelial Lining
- 4a Character and amount of fluid

II. Circulatory Disorders
- 1a Hyperemia
- 2a Hematopericardium
- 3a Hydropericardium

III. Pneumopericardium

IV. Inflammations or Pericarditis
- 1a Acute pericarditis
  - 1b Etiology
  - 2b Varities
    - 1c Fibrinous
    - 2c Sero-fibrinous
    - 3c Purulent
    - 4c Hemorrhagic
- 1a Chronic Pericarditis
  - 1b Atermination of the acute
  - 2b Pericarditis Obliterans
  - 3b Milk Patches
- 3a Tubercular Pericarditis

V. Tumors of the Pericardium
- 1a Fibroma
- 2a Lipoma
- 3a Endotheliома
- 4a Sarcoma
- 5a Carcinoma
- 6a Cysts

I. Anatomical Considerations

The pericardium is the serous sack which envelops the heart. It consists of a strong, dense, fibrous tissue wall and an inner serous lining. Where this covers the heart directly the fibrous tissue is at a minimum and it is known as the visceral layer or pe epicardium. At the base of the heart around the great vessels this is reflected to form the outer wall or parietal layer of the pericardium, and here the wall is strong, dense, and inelastic. The sack is lined by a single layer of flat endothelia, hence it is a serous sack. Normally, at the autopsy, the sac contains a few cc (3-6) of a clear straw colored serum

II. Circulatory Disorders

1a Hyperemia

An excess of blood in the vessels of the pericardium may be found in acute inflammations, or at times in valvular diseases of th heart, with tumors, or aneurysms which increase the intra-thoracic pressure. Of itself it is usually of little importance

2a Hematopericardium

This is a term applied to an accumulation of blood in th pericardial sack. It may be due to a rupture of the heart or to a rupture of an aortic aneurysm, or an aneurysm of the pulmonary artery or of the coronarys. Sometimes a serous effusion into the pericardium may be so deeply blood stained as to look like blood. This is more often true of the pericardial sack than of any other serous cavity. This blood staining may occur in severe inflammations, with, tumors, or tuberculosi of the pericardium. After decomposition has set in in a dead body there is a transudation of fluid into the pericardium and this may be stained red with Hb (hemoglobin) from the decomposing blood. It is distinguished from hemorrhage in that the color is due to Hb and the cellular elements are lacking. Hematopericardium may result from a wound.

stained red with Hb from the decomposing blood. It is distinguished from hemorrhages in that the color is due to Hb and the cellular elements are lacking.
Hematopericardium may result from a wound.

3a Hydropericardium or Dropsy of the pericardium
It is the result of a long standing passive congestion of its vessels or it may be a part of a general anasarca in heart disease, kidney disease, etc. The accumulation of a clear straw colored serum may be large enough to interfere seriously with the movement or nutrition of the heart. A difference will be found depending upon whether the accumulation be rapid or slow. If slow the heart may learn to tolerate a large amount. But if rapid, death may quickly ensue.

III. Pneumopericardium

This is an accumulation of air or gas in the pericardial sack, a result from a wound made by a broken rib, or the penetration of foreign bodies from the oesophagus. It has occured from the rupture of a gastric or oesophageal ulcer into it. In a purulent pericarditis the exudate may undergo a decomposition with the formation of gases which may remain in the pericardium.

IV. Inflammations or Pericarditis

1a Acute Pericarditis

1b Etiology

It may be either primary where the irritant is carried to it thru the blood; or secondary, as the result of a direct extension of a neighboring inflammatory process. The primary form, which is the most important because the widest spread, occurs in a number of acute infections; ex, acute articular rheumatism, scarlet fever, small pox, severe grippe, and severe septic conditions.

In the secondary forms the inflammation may extend from the mediastinum, the lung, the oesophagus, the stomach, or even from the heart itself. Very frequently in the exudate of acute pericarditis no bacteria are found. These probably have been arrested in the pericardial tissue.

2b Varieties

1c In Acute Fibrinous Pericarditis or pericarditis sicca, the first change noted is a dullness or lack-luster appearance of the lining. At times a distinct fibrinous exudate may be made out grossly and this may increase till a thick layer is formed. In the latter case the movement of the heart may cause it to take on a rugosity or rough appearance to which the name "cor villosum" is applied. When microscopic sections of the pericardium in fibrinous pericarditis is made the inner surface is seen to be covered with a layer of fibrin of varying thickness. The endothelia are degenerated and desquammated. The sub-endothelia tissues are more or less infiltrated with small lymphocytes. The blood vessels are full, espocially the arteries, and there may be minute points of hemorrhage under the endothelial layer.

2c Acute Sero-fibrinous Pericarditis is usually an advanced stage of the above, to which has been added an accumulation of serum in the sack. This serum may be in such large quantity that it may seriously compress the larger air passages, the oesophagus, or the aorta. It may also displace the heart. The gross and minute features of the pericardium are similar to those just described, although as a rule they are more marked.

3c Acute Purulent Pericarditis is an usual condition. It may follow an ulcerative endocarditis, purulent myocarditis, or like inflammations of the adjacent structures. It can also occur in pyemia or septic emboli. The exudate consists of fibrin, serum, pus cells, bacteria, and more or less rbc (red blood cells).

4c Where the rbc are sufficient to color the exudate red it is then called Acute Hemorrhagic Pericarditis.

2a Chronic Pericarditis

1b Strictly speaking chronic pericarditis is not a disease entity, because it does not rise primarily, but follows one of the acute forms. In these acute forms the fluid part of the exudate is absorbed, the fibrin remaining to become organized into fibrous tissue by the formation of granulation tissue as the result of the penetration of Bibroblasts and new capillarys from the pericardial wall. Sometimes the two layers of the pericardial wall are glued together by the fibrin and this becomes organized to obliterate

to obliterate the sack thus we have pericarditis obliterans. If this gluing together and organization takes place at once point the constant movement of the heart may cause the fibrous adhesions to be drawn out into thin fibrous cords.

3c If a moderate amount of fibrous tissue is formed in the pericardium thin white patches are left which constitute the Milk Patches or "maculae albi". Lime salts may deposit in this fibrous tissue or exudate, inextreme cases enclosing the heart in a calcarious box made up of calcified plates. If the adhesions in chronic pericarditis are of any considerable extent the action of the heart is interfered with leading to congestion of various parts of the body or to hypertrophy of the heartwith its attending consequences.

3a Tubercular Pericarditis. This may be primary, but more often it is the result of a tubercular process from the lung, pleura, or mediastinal lymph nodes. It is usually fibro-caseous with a fibrous thickening of the pericardium and the formation of minute tubercles on a serous surface. Often there is an inflammatory exudate, the layer of fibrin completely covering and obscuring the tubercle. The tubercles may become fused and undergo caseous necrosis. Tubercular pericarditis is apt to be accompanied by hemorrhage, and also the two layers may become fused to obliterate the sack. Calcarious infiltration may occur. Healing may take place by the breaking down and absorption of the exudate. The symptons will vary depending upon the extent, and are similar to those of chronic pericarditis.

V. Tumors of the Pericardium

These are rare. Of the benign tumors fibroma and lipoma have been reported. Primary endotheliomata have been reported. The sarcomas and carcinomas are secondary either by direct extension or metastesis. Pedunculated cysts containing clear fluid have been known to occur, but are very rare.

Lecture 2 10/7/'07.

Diseases of the Heart: Cutline of-

I. Anatomical considerations
II. Congenital anomalies
III. Wounds and ruptures
IV. Aneurysm of the heart
V. Circulatory disorders
    1. Anemia
    2. Hyperemia
    3. Hemorrhages
    4. Thrombosis of the cavities
    5. Thrombosis and embolism of the coronary arteries
VI. Diseases of the Valves
    1. Acute endocarditis
        1a Simple acute endocarditis
        2a Mycotic or ulcerative (malignantx endocarditis
    2. Chronic Endocarditis
        1a Etiology
        2a Morbid anatomy
        3a Results
            1c Hypertrophy of the heart
                1d Lesions of the valves in relation to hypertrophy of the heart
                2d Aortic incompetency and stenosis
                3d Mitral incompetency and stenosis
                4d Summary of conditions leading to hypertrophy of the heart
                    1e Hypertrophy of right ventricle
                    2e Hypertrophy of left ventricle
                    3e Hypertrophy of both ventricles
                    4e Limitations of compensatory hypert
            2c Dilatation of the Heart
    3. Tubercular Endocarditis

VII. Stokes-Adams Disease
VIII. Degeneration of Myocardium
1. Cloudy Swelling
2. Fatty Degeneration
3. Fatty Invasion
4. Amyloid Degeneration
5. Hyaline Degeneration
IX. Fragmantation of Heart Muscle
X. Inflammations of Heart Muscle
1. Acute Myocarditis
2. Chronic Myocarditis = Chronic Interstitial Myocardit
1a Etiology
2a Morbid Anatomy
3a Results
4a Brown Atrophy
XI. Tumors
XII. Parasites

I. Anatomical Considerations.

The heart is a hollow muscular organ with four cavities. The wal have three layers. The inner or endocardium consists of a layer of flat endothelia placed on a very slight areola tissue. The mesocardium or myocardium is composed of the typical cardiac muscle with its crossed striations and branched fibers, and with round or oval nuclei situated deep within the sarcous substance. This muscle , which is arranged in layers, whose origin and insertion while seemingly definite and constant are hard to trace. The outer layer or pericardiumis composed of flat endothelia on a subendothelial connective tissue. The endocardium, by reduplication, forms the heart valves. The nutrition of the heart is maintained through the coronary arteries which come of. of the aorta just above the anterior flap of the aortic valve (right coronary), and the right flap posterior (left coronary). These coronary arteries finally sub-divide into practically terminal arteries. The blood is returned to the heart into the right auricle through the pulmonary sinus and by various small opemings into the right auricle. The lymphatics are well developed. Those from the right side terminating in the right lymphatic duct, while those from the left side empty into the thoracic duct.

The Nerve Supply. There are varous and numerous ganglionic centers in the ventricular and auriculo-ventricular grooves. Other ganglion cells have been found in the muscle itself.

Heart Sounds: There are two sounds which are represented by the syllables "lubb-dup". The first occurs at the beginning of the ventricular systole and is probably a combination of muscle vibration and the vibration of the auriculo-ventricular valves. It ends before the ventricular systole is completed and is deeper and longer than the second sound. The second soune occursat the end of the ventricular systole and is coincident with the closure of the semilunar valves. It is shorter and sharper than the first sound.

II. Congenital Anomalies.

These are due to one or both of two things, either failure of certain parts to develop properly, or they may be the sequellae of intrauterine inflammations of the foetal heart. The position of the heart may be shifted. The heart may be too small, called hypoplasia, a condition which may be associated with chlorosis. Any of the septa or valves may be wanting, and occasionally there is a complete absence, caleed acardia, which is met sometimes in monsters. The narrowing or partial occlusion of either the pulmonary artery or the aorta may be important. One of the most important congenital defects, because the most frequent, is seen in the failure of the foramen ovale to close properly. The venous blood becomes mixed with the oxygenated blood of the left auricle. These are the socalled "blue babys", and the disease is known as "morbus ceruleas". The well marked cases rarely live to adult life. The imperfect oxygenation of the bloodis evidenced by the blue colorwhich gives the name to the disease. And the imperfect circulation causes an almost characteristic thickening of the lips and nose, with a clubbing of the ends of the fingers.

III. Wounds and Ruptures.
Wounds which do not penetrate the entire thickness of the skin heart may be recovered from by a scar foemation. As a rule the penetrating wounds cause death by hemorrhage into the pericardium. Spontaneous rupture of the heart may possibly occur from some extraordinary strain on a normal heart; ex, extreme emotion or excessive muscular strain. It is said Christ died of a ruptured heart which is real "broken heart". Usually the heart must be weakened first by some disease. Of these Myomalacia cordis is common, which is a softening of the heart, as a rule from thrombis or embolism. Fatty degeneration, malignant endocarditis, and abscess are the most frequent. These processes being present and the heart being called upon to perform some extra service a distinct break of the wall may occur with death.

IV. Aneurysm of the Heart.
This is quite rare. It is usually found in the anterior wall of the left ventricle, where the wall has been weakened by disease. In myomalacia cordis from embolism an acute aneurysm may occur. If the part becomes the seat of a chronic fibrosis the dilatation may be gradual and thus an aneurysm form.

V. Circulatory Disorders.

1. Anemia: This of itself is of little importance because usually it is more or less transitory. It may be a part of a general anemia, or be due to a narrowing of the coronaries by fibrosis, or to the pressure of a pericardial effusion. If it continues it leads to fatty degeneration.

2. Hyperemia and Congestion: An excess of arterial blood (hyperemia) may be a part of an acute infections process, or of a local inflammatory mischief in the heart itself. Congestion, or venous excess, is usually found in a general congestion in valvular disease. Here the dilated veins stand out on the surface of the heart very distinctly.

3. Hemorrhage: It may be in the form of an infarct when a branch of the coronary artery is occluded. It may accompany a severe myocarditis, or surrounding abscess in the muscle. Hemorrhages may also be found in patients dying from severe acute infections; ex, sepsis or various poisons. In death by choking or strangulation subendocardial hemorrhages may be found.

4. Thrombosis of the Cavities: At the autopsy in nearly all cases one finds blood clots of some sort in the cavities. The majority of these are not the result of thrombosis, since they have formed after death or during the agonal period and have no pathological significance. However, where death has been slow and the circulation slowed clots may form, especially if the wall be roughened from any cause. Of the PM (post mortem) clots the commonest form is that of the current jelly clot, which is soft, red, and not attached to the endocardium. These may form in any of the cavities, but are specially marked in the auricles. The clots formed just before death or in the agonal period may be red, but at times are yellowish or white and constitute the typical "chicken fat clot". These consist almost entirely of fibrin with very few cellular elements. And like the "current jelly clot" are not attached to the heart wall. At times they may extend into the vessels leading out of the heart, especially the pulmonary arteries. They may be hard to distinguish from true thrombi. The clots formed in thrombosis may be either red or white, but more often white. They are firm, lie in between the columna carnae, and are attached to the endocardium so that when they are removed the surface of the wall is dull and may be slightly roughened. Dilatation of the heart and anemia especially predispose to thrombosis by the slowing of the current through that part. If the thrombi become detached and lie free in the cavity thay form ball thrombi. The centers of any of these thrombi may soften and look like pus, but it is not pus. Occasionally layer or stratified clots are made by alternate slowing and quickening of the circulation. The clot has alternate red and white layers.

In any event when these clots are formed the action of the heart is hindered, and if important vessles become plugged sudden death may ensue. Parts of these thrombi may be swept on to be lodged as emboli in some small vessel, those from the left ventricle going to the brain as the site of preferance, since its vessels are more in a direct line. (The left middle cerebral is the most direct line). Among the diseases in which the circulation tends to be slowed, and which favors thrombosis are pneumonia, pulmonary tuberculosis (tbc), asthenic fevers and diseases of the myocardium. Acute endocarditis may favor such formation, and in fact a slight degree of fibrin formation is an almost constant feature of the disease. In chronic endocarditis also fibrin is deposited and clots may be formed on the roughened or calcareous valves.

5. Thrombosis and Embolism of the Coronary Arteries.

Thrombosis of the coronaries is more common than embolism because the openings of the coronary arteries are so paced behind the aortic cusps as to protect them from the entrance of emboli. The thrombosis oten follows athroma or syphalytic thickening. The results of thrombosis and embolism are much the same. The part supplied by the closed vessel is the seat of an ischemia and then as the tissue dies from lack of nutrition a white or anemic infarct forms. The commonest place for this to occur is in the left ventricular wall, either anterior or posterior near the apex (The part supplied by the left coronary). These infarcts are white, yellowish or gray in color, pyramidal in form, and project slightly above the surface in the early part of the process.

Microscopically:- the tissue does not stain well, nuclei gone, striations lost. If stained by osmic acid fatty degeneration is seen. If the area affected is small this degenerated material is absorbed and replaced by fibrous tissue. The primary softened condition is called "Myomalacia cordis". If the area is considerable extent the heart may rupture or an acute inflammation may ensue. When organization takes place the part shrinks and becomes cicatricial in character. This leaves a week inelastic place which is especially prone to undergo dilitation to form an aneurysm from the constant pressure of the blood within on this weakened tissue. Where infected emboli occur an abscess in the heart wall may form.

VI. DISEASES of the Endocardium.

1. Acute endocarditis.

While the term endocarditis strictly speaking refers to an inflammation of any part of the lining of the heart, yet it so frequently involves the valve leeflets that the term is usually that of as applying to the process here. While it is not done, it would be well if the more specific term of valvulitis would be used. Of the acute forms of endocarditis by far the more frecuent is the

1a. Simple Acute .

As to its ETIOLOGY it may be said to be practically always a secondary process, occurring in the couse of acute articular rheumatism more frequently than in any other disease. Next in order of frequency as a cause comes Scarlet fever, pneumonia, and puerperal sepsis. It must be remembered that it may be found in almost any suppuratve or infectious process. Various microorganisms have been isolated from the vegetations; ex, mic. pyogenese auris; Streptococcus pyogenese,; diplococci of pneumonia: and gonococci being among those most often met. Many others have been recorded but in all the others the causative relation has not been established.

With reference to the morbid anatomy of the simple form the common seats in order of frecuency are the mitral valve: aortic valve: pulmonary valve; endocardium of the left ventricle: of the left auricl and then the right ventricle. The part of the valve first involved is usually lying across the leaflet about 2 mm from the free edge. This is the line of closure and the little injuries incident to the closure of the valve probably predisposes this part to the process. At first the only change noted is a slight dulling of the surface and then a tiny row of bead like elevations appear, and if the process continues these elevations become very distinct, rough, and warty.

In this case the name verrucose endocarditis is used.

Microscopically:- The endocardium shows a proliferation of the endothelia with perhaps some degeneration, and beneath this comes an infiltration of small round lymphocytes. The little vegetations consist chiefly of fibrin which may be in flakes, granules, or net works of fibrillae. By proper staining the bacteria may be demonstrated. If the process continues there is a beginning proliferation of fibroblasts in the sub-endothelial tissue.

Prognosis:- If the process be very mild complete recovery may result. But more often there has been enough fibrous scar tissue formed to remain permanently, to be the basis of a subsequent attack. The simple acute may pass into the next form. Repeated attacks lead to chronic endocarditis to be described later. Do not forget that bits of these vegetations may form emboli, and frequently they are infected.

2a. Mycotic or Malignant Ulcerative Endocarditis.

This may be looked upon as an advanced stage of the simple acute. Here the destructive process is marked, a necrosis of the endocardium taking place giving rise to ulcers. Along with these ulcers there is usually a thrombosis with a deposition of much fibrin about the ulcers, or the ulcers may be entirely covered and hidden by it. These vegetations may assume a considerable size and are usually an important feature of the case. The destruction of the valves may be extensive leading to complete perforation, or to such a weakening of the valv that aneurysms of the leaflets later may take place. In all cases of malignant ulcerative endocarditis the presence of bacteria is important. They may be found growing on the surface of the ulcer and vegetations in great colonies. Here again is an opportunity for septic emboli.

Microscopically:- the endocardium is seen to have undergone extensive necrosis and there is an extensive round cell infiltration with multiplication of fibroblasts. This multiplication is specially true when the process has been grafted on a chronic valvulitis.

PROGNOSIS: Death may ensue and in bad cases usually does from interference with the heart action combined with the toxemia. Complete recovery is never possible. The fibrin may be absorbed and removed but it more often becomes organized by tthe penetration of fibro blasts and blood vessels to leave masses of scar tissue. The ulcers also heal by granulation and all of this fibrous tissue later contracting distotts the valves in all kinds of shapes.

2. Chronic Endocarditis.

1a. Etiology.

Any of the causes of acute endocarditis named above, if repeated, or if acting over a long time may be important in chronic endocarditis. Then, too, the possible effects of syphilis and alcoholism must not be lost sight of. It usually follows the acute process but can be primary or chronic from its very beginning.

2a. Of the MORBID ANATOMY in this chronic valvulitis there are two general forms. First, there may be a more uniform thickening of the leaflets with hard dense fibrous tissue and on the surface little hard vegetations or ridges of fibrous tissue, with perhaps the deposition of some lime salts. There may follow a uniform contraction to give rise to an incompetent valve. This is the less important form. In the second general form the growth of the fibrous tissue has not been uniform. Some of the new connective tissue cells continue to grow while others die. Thus the valve is thinned in places even to perforation and at other places it becomes rough and prominent with projections of fibrous tissue. Considerable fibrin may be deposited on the surfaces of these vegetations. After a time the process is further complicated by the shrinking and contraction of the valves with perhaps the deposition of line salts. The cusps become shorter, thicker, and fused to each other or to the heart wall. The chordae tendinea also take part in the process as do the papillary muscles, both of which may shorten and thus aid in the distortion of the valve. So there may finally result either an insufficiency of the valve or the lumen may be contracted so as to permit the blood to get through with added difficulty and so cause stenosis. Large ulcers may form on the valves with a deposit of considerable fibrin to make their resemblance to the mycotic endocarditis very close.

PROGNOSIS:- Recovery is naver possible. The process once formed always continues. And if death does not result from some intercurrent affection it will finally result from the effect on the heart and the circulation.

Lecture 4. 10/21/'07.

3a. Results.

1c. The most frequent consequence of valve lesion is hypertrophy of the heart. This hypertrophied is caused by an increase in size of the individual muscle fibers and also probably by a numerical increase. In all cases of hypertrophy the muscle tissue is usually redder, firmer, and more resistant to the knife than the normal. If some degenerations have intervened this may be changed so that the color is paler, the muscle flabbier, and the tissue too friable.

Microscopically:- the individual fibers are longer, their nuclei increased in size and often in number and usually there is an increase in the amount of fibrous interstitial tissue.

Simple hypertrophy is a thickening of the muscle without an alteration in the size of the cavities.

Concentric hypertrophy means the thickening of the wall with a decrease in the size of the cavity. It is very doubtful whether this ever actually occurs and whether the decrease of the cavity is not a post morton constriction of it.

Excentric hypertrophy is a thickening of the wall with an increase in the size of the cavity. It is really a hypertrophy with dilitation and this is the most common form. A simple dilitation of the heart would be an increase of the size of the cavity with out an increase in the thickness of the wall or even a thinning of the wall.

1d. Relation of Valvular Disease to Hypertrophy of the Heart.

Regardless of the nature of the lesion in the valve it is sure to throw more work on some part of the heart. For a while this call for extra workis physiological and is accompanied by increased nutrition just as any part of the body gets more nutrition by blood supply when it has more work to do. This increased nutrition usually leads to a building of more tissue and hypertrophy.

2d. Effects of lesions of the aortic valve. When the valve is so distorted as to fail to close properly and permits a backward flow or regurgitation of the blood it is called insufficiency or imcompetency. On the other hand, if the lumen of the valve is narrowed or contrcted so as to offer increased resistence to the outflow of the blood we have the condition of stenosis. Where the aortic valve is insufficient during diastole the blood instead of all going through the aorta in the general circulation as it should do, a part of it flows back into the left ventricle. This results in a deficiency of blood in the general circulation leading to a stimulation of the cardia c centers. The regurgitated blood in the left ventricle is in the way. Both of these lead to increased work on the left ventricle causing its hypertrophy and this for a while makes up for the insufficient valve. In stenosis greater effort on the part of the left ventricle is required to force the blood through the normal opening, thus again leading to hypertrophy of the left ventricle. In fact, in just such cases of aortic stenosis we find the largest hearts, some weighing 1800 gms or more. and it is to these we confine the name "cor bovinum" = beefy heart.

With such hypertrophy of the left ventricle the length of the heart is increased and the apex pushed downward and to the left.

3d. Mitral Insufficiency and Stenosis.

Here in case of insufficiency of the valve we have a back flow of the blood into the left auricle causing hypertrophy of that cavity in the effort to empty itself properly, and alos a dilitation to accommodate the extra blood. Such a regurgitation of blood into the left auricle prevents proper emptying of the blood from the lungs tending to an accumulation of blood in those organs. This is offset by increased work on the part of the right ventricle causing its walls to be thickened. Part of the blood regurgitated from the left ventricle will make that cavity do more work to keep the amount of blood in the systemic circulation up to the normal. The natural result is hypert.

Then too, in diastole the blood comes into the left ventricle with greater pressure because of the increased force of the right heart. this will finally tend to dilitation. So we have hypertrophy with dilitation.

In mitral stenosis the blood has difficulty in getting through the mitral valve. The left auricle must do more work so it hypertrophies and also dilates to accommodate the extra blood. The blood tends to dammed back into the lungs and the walls of the right ventricle must become stronger to force it through. In mitral stenosis not accompanied by insufficiency the left ventricle remains unchanged for a long time. In fact until the weaking of the right heart decreases the amount of nutrition furnished. At which time the wall of the left ventricle may degenerate and dilate. Where the hypertrophy is essentially in the right ventricle the breadth of the heart is increased the area of dullness exteds further toward the right. The apex of the heart becomes blunter and in extreme cases may even be formed by the right ventricle.

1e. Valve Lesios of the Right Side.

Uncomplicated valve lesions of the right side are not very common in adults, but when they do occur the results are similar to those outlined for the left side. There may be a congenital stenosis of the pulmonary artery leading to hypertrophy of the right ventricle.

Extra causes of cardiac hypertrophy.

1e. Hypertrophy of the right ventricle.

In addition to the valve lesions mentioned it may arise from any causes which increases the intra-pulmonic pressure, or the difficulty with which the blood gets through the vessels and capillarys of the lungs. Prominent among these are interstitial pneumonia, pulmonart tbc, emphyzema, chronic bronchitis, and in some forms of pleuritis, especially where wide spread adhesions have occurred.

2e. Hypertrophy of the left ventricle, from extra cardiac causes may come from a narrowing of the aorta, from an aneurysm of the thoracic aorta, from an increased in resistance to blood flow through the vessels the seat of endarteritis or arterio sclerosis. Very frequent cause of left ventricular hypertrophy are the forms of chronis interstitial nephritis. It has never been determined how this chronic Bright's causes hypertrophy, but is probably due to a combination of the effect of the accompanying arterio-sclerosis, and also to the added difficulty with which the blood is forced through the kidneys. It is evident when one considers the important part the elasticity of arteries play in maintaing circulation, that if they are changed into more or less rigid tubes by the addition of fibrous tissue, that the heart, especially the leaft ventricle, will be called on to use more force in sending the blood through the peripheral circulation.

3e. In certain general conditions both ventricles may be uni formly hypertrophied; ex, in exophthalmic goiter the heart is caused to beat faster and both chambers are hypertrophied. So too, in excessive muscular exercise: ex, athletes or laborers (dock workers) , or the excessive use of alcoholics, especially with beer drinking, will cause hypertrophy. The indulgence in excessive eating and drinking may lead to an increase of the size of the heart. Chronic adhesive pericarditis interfering with the heart's action causes hypertrophy of both ventricles.

4e. Limits of Compensatory Hypertrophy.

Regardless of the causes, and the course of the hypertrophy of the heart there will come a time when the amount of nuttrition furnished the muscle tissue does not keep pace with the extra work required. When this occurs the muscle tissue will undergo various degenerations and become weakened. This weakening of the heart is progressive in that still less nutriment is supplied. Then follows a series of congestions of various organs of the body with their attendant consequenves, called by the general term, "failure of compensation", and in the heart frequently dilitation.

2c. Dilitation of the Heart.

Dilitation of the hart is frequently accompanied by hypertrophy and may be one of two forms. Actual dilitation when it accompanies hypertropht for the purpose of accommodating more blood. And passive dilitation when there is no increase of the thickness of the wall but even an actual thinning of it. Such passive dilitation may be due to changes in the valves coming on so rapidly that the wall has not had time to thicken. This may later, however, be followed by a compensatory hypertrophy. Again, changes in the muscle, ex; infiltration from pericarditis; inflammations of the muscle; degenerations and infiltrations, especially fatty invasion; and atrophy of the muscle fibers may be important. An hypertrophied heart may later dilate from degeneration of its fibers. An acute inflammatory exudate in the lung or an acute pleuritic exudate may prevent the proper flow of blood through the vessels of the lung and so lead to rapid and even fatal dilitation of the right ventricle. Again, he may die from sudden dilitation of the right ventricle from a too rapid overloading of the stomach leading to an acute indigestion and dilitation of the stomach. The dilitation of the ventricle in this case was the result of the mechanical interference of the heart action by the distended stomach. The interference of the circulation through the lungs and general circulation, by pressing on the vessels, and possibly reflex nerve disturbances.

Results of valve lesion in organs ather than the heart: These are due to the circulatory disturbances and especially the congestions with edema, and the chronic fibrosis which accompanies stasis.

3. Tubercular Endocarditis.

This is a rare affection of the emdocardium, but may accompany tubercular pericarditis or be part of a general miliary tuberculosis. The lesions may occur as miliary tubercles, or these may fuse and undergo caseous necrosis. If they extend any distance into the muscle substance the wall may be weakened, leading to an aneurys of the cavity at that place. This is rare.

VII. Stokes-Adams Disease.

This is due to some interference with the wave of contraction as it passes from the auricle to the ventricle. His has shown that there is a narrow bundle of muscle fibers that pass from the auriculo septum to the ventricular septum and is called the "auriculo-ventricular bundle of His". And it is along this that the contraction waves pass. This disease is made manifest by the slow pulse and at times by a pulsation in the veins of the neck which is more frequent than the beat of the ventricle.

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This is accompanied by attacks of syncope with a very slow pulse. At times the lack of rhythm between the auricles and ventricles is considerable. The auricles beating 3-4 times to one of the ventricle. This is the condition known as "heart block". Frequently at autopsy no lesion of the bundle of His can be discovered. But various pathological conditions have been found, ex: tumors, gummata, sclerosis, etc. It can be produced experimentally by injury to heart in the neighborhood of the bundle. A real case of heart block is always fatal, the patient dying in syncope.

VIII. Degenerations of the Myocardium.

1. Cloudy Swelling.

In all cases of parenchymatous degeneration or cloudy swelling of the heart muscle the immediate cause is an infection. It occurs in diphtheria, pneumonia, typhoid fever, scarlet fever, occasionally rheumatism and less often in other acute infections. The elevated temperature and the toxic action are probable elements in its causation.

Morbid Anatomy: The effects are usually more marken in the left ventricle than in any other place and the process is practically always diffuse. The myocardium is pale, often turbid on section, and it is too friable.

Microscopically: The fibers are swollen, the striations indistinct or lost and the muscle may be too large and too pale. The cyto plasm of the fibers show precipitated albumens to give them a granular appearance. These granules clear up with acetic acid. This cloudy swelling may accompany an acute inflammation. If it persists fatty degeneration is almost sure to follow. Its result is to weaken the heart, with its attendant circulatory disturbances.

2. Fatty Degeneration.

In the etiology of fatty degeneration of the heart some toxic substance is present and it may be caused by deprivation of nutrition. The acute infections are the same as those mentioned in cloudy swelling. It also occurs in definite chemical poisonings, viz. P. As. Sb. of the chronic type poisoning, of course. Any severe parenchymatous degeneration may end in fatty degeneration.

Morbid Anatomy: The process may be either diffuse or localized. The latter is usually due to ischemia; ex. embolism or thrombosis of the coronarys. In the diffuse form the process involves almost every part of the heart. It is not uniform. Some areas of fatty degeneration may lie immediately adjacent to unaffected muscle. The part fatty degenerated is of a pale yellow color and this in contrast with the more normal red muscle in alternate streaks or patches give rise to the name "tabby cat" "pussy ?" or "tiger heart". The process is best seen in the papillary muscles of the left ventricle. The muscle tissue affected is much too friable, more so than in cloudy swelling. It is easily pinched through with the fingers. In rare cases the entire muscle is involved to give a diffuse yellow color.

Microscopically: The fibers are swollen, nuclei pale or lost, and the cytoplasm is converted into little round droplets of fat, stained black with osmic acid and does not clear up on addition of acetis acid. These fat droplets may be so numerous as to obscure the striations and the nuclei and show no tendency to fuse together.

Results: Rupture of the heart may take place if the ptocess be localized, but if it is diffuse the muscle is usually too weak to raise the intra-cardiac pressure sufficient to cause rupture. The dangers of the diffuse form are, that the patient may succumb to sudden syncope from failure of the heart to contract. Or there may be a gradual failing of the circulation brought about by weakness of the muscle. If the process is mild and not too many fibers involved, complete recovery is possible, the degenerated fibers being broken down and removed; New muscle fibers taking their place. If the lesion be more severe instead of replacement by muscle tissue, new connective tissue forms. The latter is nearly always the result in the localized form. In any event with fibrous tissue replacement we have a weakened heart which may tend to dilitation.

4. Amyloid Degeneration.

It may occur in the blood vessels, but is rare and unimportant.

5. Hyaline Degeneration.

It is usually seen in small areas giving it a glossy appearance. A special form, known as Zinker's hyaline degeneration, occurs occasionaaly in typhoid. In addition to the process in the abdominal muscles it is usually an accompaniment of cloudy swelling, and the muscles affected have the peculiar appearance of the flesh of fish.

3, Fatty Invasion.

The term "cardiac obesity" is often applied to this condition which is essentially an invasion of the epicardial fat in between the muscle bundlees, and in extreme cases between the muscle fibers themselves. The causes are those which lead to general obesity; ex, heredity, habitual overeating and over drinking, especially of alcoholic beverages, and of the latter, beer especially.

Morbid Anatomy: The increase takes place first at the sites where there is normally most fat,i.e. over the septum and over the right ventricular wall. On section, trabeculae of fat may be seen extending down into the muscle, in extreme cases to the endocardium, showing here as yellow patches.

Microscopically: The fat is seen to be entirely outside of the muscle fibers so that it is in no sense a degeneration. Thesis, however, nothing to prevent fatty degeneration from occuring in obese heart, but is an added process. The individual fibers may become atrophic from pressure. The result must always be a weakening of the heart from the mechanical interference with the normal contraction, and decrease in the number of muscle fibers. Circulatory disturbances; congestions etc., are sure to follow. Dilitation is a not infrequent complication.

IX. Segmentation of the Heart Muscle.

By this is meant a separation of the muscle fibers where they are normally cemented together.

This condition has baen found in acute infectioud diseases, in acute and chronis diseases of the central nervous system, and in sudden death from a number of causes. The muscle is often soft and friable and with an opaque appearance. It is possible, however, that the process may be post mortem, or that it may occur just beforc death, in the agonal period when the heart is putting forth extreme effort to act. Even though it does occur at such times as those mentioned, and hence should not be considered as a separate pathological process, yet it always indicates there is some trouble in the heart muscle.

Fragmentation of the Heart Muscle.

This is a tearing across of the individual fibers at places apart from their normal joinings. It occurs under the same conditions as segmentation, and nearly always the two occur together.

X. Inflammations of the Myocardium.

1. Acute Myocarditis.

The changes in acute myocarditis which are characteristic of the inflammation are referable to the interstitial tissue and the blood vessels. Any changes in the muscle fibers themselves, ex; cloudy swelling, etc., are secondary changes. It may be localized or diffuse. The acute diffuse myocarditis is met in the acute infections, more commonly diphtheria, scarlet fever, typhoid, acute rheumatism puerperal sepsis, and in influenza=grippe. It may be the result of the extension of an acute pericarditis, especially of the purulent form.

Grossly: The heart is too pale and too friable, points which are due to the accompanying cloudy swelling.

Microscopically: We have the swollen granular muscle fiber characteristic of cloudy swelling. But the essential changes is the round cell infiltration of the interstitial tissue and the hyperemia, with perhaps some diapedesis. After the process has lasted for some time we have a proliferation of the fibroblasts which will tend to the formation of new connective tissue. In this form there is no tendency to suppurative changes.

The acute localized myocarditis is practically always an acute suppurative myocarditis with the formation of abcess. It may rise in pyemia where we have the muscle studded with little gray miliary abseesses; or there may be larger abscesses either single ones or just a few which have resulted from an infected embolis of the coronarys. The latter are usually in the anterior part of the left ventricular wall near the apex. When sectioned the interior may be seen to contain a little pus.

Microscopically:- The adjacent muscle is degenerated and the pus is seen to consist of poly nuclear leukocytes, bacteria, and broken down muscle tissue. In the healing, if this occurs, the pus may become inspisated, removed, and replaced by scar tissue. In the miliary abscess the patient usually dies of the general pyemia before any such changes take place. In the larger abscess rupture of the heart may occur or the abscess itself may rupture into the pericardium, causing suppurative pericardidis, or into the heart cavity causing thrombosis or giving rise to septic emboli.

2. Chronic Interstitial Myocarditis = Chronic Fibrous Myocarditis.

This again is localized and diffuse.

1a. Etiology:- The localized form of interstitial myocarditis is due to the formation of scar tissue in the healing of an infarct from thrombus or embolis of the coronarys, or the healing of an abscess. The diffuse form may follow an acute myocarditis, or it may rise of itself independently, of an acute process when there is some changes of the coronarys; ex, endarteritis, or a failure of nutrition rising from chronic valvular disease. Then too, it may follow degenerations of the myocardium: ex, cloudy swelling, fatty degeneration, without a definite myocarditis antecedent. Often it is a reparative process, a fibrous hyperplasia and in reality not a definite inflammation. The immediate cause, then, is some disturbance of nutrition.

Remote causes are such diseases as may lead to a sclerosis of the arteries or a stasis of the blood in the heart. These are syphalis, chronic alcoholism, possibly exercise, old age, the gouty diathesis, and chronic valvular lesions.

Morbid Anatomy:- In the localized form we have an area of dense scar tissue, with may be some evidence of contraction about it. And microscopically the muscle tissue is entirely replaced bt adult fibrous tissue.

The diffuse form is wide spread, but very rarely uniform. To the naked eye the heart muscle presents patches or lines of white through the xxxxx med muscle. These are more common in the anterior wall of the left ventricle, and in the septum, and in the tips of the papillary muscles. The tissues have an extra toughness.

Microscopically:- Different parts of the heart may show the process in all stages.

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The onset will show newly formed fibrous tissue with many fibroblasts. Another almost complete replacement of the muscle with fibrous tissue. Still another section may show merely a round cell infiltration of the interstitial connective tissue. The latter is the earliest stage. Where the adult fibrous tissue is xxxxd formed it will be seen running in between the bundles and individual fibers. Frequently the muscle fibers will show degeneration as cloudy swelling or more often fatty degeneration. This is due to the pressure of the fibrous tissue. Where the process has taken place in a hypertrophied heart or is accompanied by hypertrophy, which is not unusual, the individual muscle fibers may be large, with large nuclei. The muscle is best preserved nearest the artery and here too the pxxxxxx formation of the extra fibrous tissue is at a minimum, indicating tha t the process is probably dur to decreased nutrition.

3.a. Results:- Always the heart wall is weakened at the site of the fibrous tissue formation. In the localized ot focal form this may lead to aneurysm. In the diffuse form it may be so prominent that the interference with the heart action leads to hypertrophy. If the wall be much weakened dilixxtation may ensue. In any event, the heart's action is weakened and disturbances of the circulation of greater or less importance is sure to follow. In certain cases paroxysms of intense cardiac pain may occur, forming one kind of "Angina Pectoris". Where the interstitial process is marked the action of the h heart is much more irregular than with straight valvular disorders, even though the murmur can not be made out. This irregularity of the heart's action is especially noted where the interstitial process affects the auricles, where it probably interferes with the continuity of the muscle and the consequent disturbance with the wave of contraction. i.e. it gives rise to heart block, even to such an extent as to be classed as Stokes Adams disease. This chronic interstitial myocarditis probably explains the irregular pulse seen in old persons with out other definite cardiac lesions.

4.a. Brown Atrophy of the Heart:- Brown atrophy of the heart is a special form of what Dr. Terrell thinks should be included under chronic interstitial myocarditis.

Here the heart is much smaller than normally, even being reduced to 1/3 its original weight. The tissue is dark red, the coronaries very tortuous, and the tissues quite resistant to the knife.

Microscopically:- There is an increase of the fibrous tissue throughout the wall, and this in contrast to the ordinary interstitial myocarditis is fairly uniform. The vessel walls are much thickened with added fibrous tissue. The individual cardiac muscle fibers are small and there is marked increase in the polar pigment. The latter does not take up the iron stains and is probably tissue pigment from broken down and atrophic muscle cells. This process usually occurs in old age, or any cachexia, or waisting disease. It is probably consequent on the arterio-sclerosis, or the congestion, or both.

3. Tubercular Myocarditis, or tuberculosis of the myocardium:- It is not frequent. It is most often a part of general miliary tbc and the patient dies before the tbc passes the miliary stage.

Syphalis:-- Gummata of the heart are rare, but may become of considerable size with much caseous necrosis.

XII. Tumors of the Heart.

They are not of much clinical importance because of their rarety and the practicable impossibility of diagnosing them during life.

The primary tumors are sarcoma, myxoma, fibroma, lipoma, and myoma Of the secondary tumors, which are more frecuent than the primary, carcinomas, and sarcomas have been observed.

XIII. Parasites of the Heart.

Echinococcus cysts may occur and rupture into the cavities. The cysticercus are the tenia soleum and the tenia saginata have been found.

cysticercus

Outline of diseases of the Blood Vessels:-

I. Anatomical considerations.
II. The Arteries.
    1. Retrogressive Changes.
        1a. Hypoplasia.
        2a. Atrophy.
        3a. Fatty Degeneration.
        4a. Amyloid Degeneration.
        5a. Hyaline Degeneration.
        6a. Calcarious Infiltration.
    2. Hypertrophy.
    3. Inflammations.
        1a. Acute Arteritis.
            1b. Acute productive Arteritis.
            2b. Suppurative Arteritis
        2a. Chronic Arteritis= Arterio-Sclerosis.
            1b. Etiology.
            2b. Morbid Anatomy.
                1c. Forms
                    1d. Circumscribed.
                    2d. Diffuse.
            3b. Results.
                1c. In the Vessels.
                2c. In the Organs.
            4b. Periarteritis Nodoso.
        3a. Tubercular Arteritis.
        4a. Syphilitis Arteritis.
    4. Aneurysm.
        1a. Etiology.
        2a. Morbid Anatomy and Classification.
        3a. Aneurysm of Different Vessels.
        4a. Results of Aneurysms.
        5a. Associated Conditions.
    5. Tumors of the Arteries.
III. Veins.
    1. Degenerations.
    2. Inflammations.
        1a. Acute Phlebitis.
        2a. Chronic Phlebitis.
    3. Phlebectasis= Varicose Veins.
        1a. Etiology.
        2a. Morbid Anatomy.
        3a. Results.
    4. Tuberculosis.
    5. Syphilis.
    6. Tumors.-
IV. Capillaries.
V. Vessels of New Tissue and of Tumors.
    1. Vessels of Organizing Tissue.
    2. Vessels of Tumors.
        1a. Of benign tumors.
        1a. Of Malignant Tumors.
            1b. Carcinoma.
            2b. Sarcoma.

I. Anatomical Considerations.

The arteries and veins consist of three coats variously developed depending upon the size and nature of the vessel. These are the intima= inner coat: Media= middle=muscular coat: adventia= theouter coat. In atypical artery the intima consists of anner lining of single layer of flat endothelium situated on rather dense subendothelia tissue, and then on an alastic membrane.

The media is made up of smooth muscle tissue.

The adventitia consists of a mixture of white fibers and elastic tissue blending more or less with adjacent structures.

The veins differ in that they are larger, less muscle and less elastic tissue. The nutrition of the vessels is derived from the vasa-vasorum which come off as branches from the arteries and run along the adventitia, their branches penetrating inward to supply food to all parts of the wall, save the endothelium. This latter is the only part

of the vessel which derives its nutrition from the blood stream flowing through the vessel.

II. Arteries.

1. Retrogressive Changes.

1a. Hypoplasia:- While strickly speaking this is not a retrgressive change, in that it is congenital, yet for convenience it is placed here. In this condition the hypoplasia the aorta and larger vessels are distinctly too small, and have always been from birth and is the result of foetal underdevelopment. The heart also may share in this condition. The hypoplasia may accompany "status lymphaticus", and may be found in chlorosis.

2a. Atrophy:- This atrophy of the vessels may involve the whole body or be confined to a single large vessel and its branches, or to the arterial system of a single organ. This atrophy always results from malnutrition. This malnutrition may be the result of general malnutrition of the body in any waisting disease. Or the malnutririon may result in the atrophy of some particular organ. It may also arise from a disease of the vessels themselves.

3a. Fatty Degeneration:-- This fatty dehenerration mat occur in any of the coats of the arteries, but is most often found in the intima, particularly in the subendothelia tissue. It is an important feature in athroma, to be described later. It may occur in large enough areas to be seen with the naked eye or it may require a microscopic examination to detect it. ~~The gggggggg~~ Grossly, where we can see it with the naked eye, the color is yelloish or yellowish white, well circumscribed, occurring in strips or patches perhaps of irregular shape. In severe cases there may be an actual erosion of the intima to form an ulcer= athromatous or fatty ulcer.

Microscopically:- The endothelial cells, if involved, and the cells of the subendothelia tissue are seen to be filled with little round fat globules. In extreme cases these dronlets are also extra cellular and infiltrate the tissues. Where the media is involved the muscle fibers contain fat drops. The result is a weakening of the vessel wall, leading to rupture or to aneurysm, or perhaps to calcarious infiltration. The aorta is most frequently involved, but the arteries elsewhere are by no means exempt.

4a. Amyloid Degeneration:--

The blood vessels, and of these the arteries of the part, are the first to be involved by an amyloid degeneration, and this is specially seen in the glomeruli of the kidneys, and in the arteries of the Malpigian bodies of an amyloid spleen. The process usually begins in the intima, then involving the media, them lastly the adventitia. From the blood vessels it spreads to the neighboring tissues, but probably does not involve the epithelium. Occasionally amyloid degeneration occurs in the larger arteries, ex: aorta, in the intima in such small patches that they can be recognized only by the peculiar staining of chemical reaction. Where the involvement is sufficient extensive to be seen with the naked eye, it presents a firm translucent, glassy, colorless appearance.

Microscopically:- it is structureless and almost homogeneous.

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5a. Hyaline Degeneration.

This may affect the intima, or more rarely the entire wall of the smaller vessels, to convert them, or the part affected, into a colorless somewhat glassy, structureless material, similar in gross appearance to the amyloid, but not giving the staining reaction of the latter. This process is very common in arterio sclerosis in the newly formed connective tissue.

6a. Calcarious Infiltration.

This is a commom occurrence in both forms of arterio ~~xxx~~ sclerosis. The process usually begins in the subendothelia connective tissue, and the lime salts may be deposited in granules or in calcarious plates which underly the endothelia like the scales of a fish. The media of the vessels may be involved, but it rarely occurs in the adventitia. At times the calcification may be so extensive in the ~~xxm~~ smaller arteries as to convert them into rigid tubes; ex, "pipe stem arteries" of old age. The process occurs in dead or dying tissue, especially if there is some blood supply for it.