

**Staff Meeting Bulletin**  
**Hospitals of the » » »**  
**University of Minnesota**

**Degenerations of**  
**The Macular Region**

STAFF MEETING BULLETIN  
HOSPITALS OF THE . . .  
UNIVERSITY OF MINNESOTA

Volume XI

Friday, February 2, 1940

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INDEX

	<u>PAGE</u>
I. LAST WEEK . . . . .	200
II. MOVIE . . . . .	200
III. ANNOUNCEMENTS	
1. LECTURE . . . . .	200
2. LECTURE . . . . .	200
3. CONTINUATION STUDY COURSE . . . . .	200
4. BABIES . . . . .	200
IV. DEGENERATIONS OF THE MACULAR REGION . . . . .	
. . . Karl Benkwitz . . .	201 - 215
V. GOSSIP . . . . .	216 - 217

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during the school year, October to May, inclusive.

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William A. O'Brien, M.D.

I. LAST WEEKDate: January 26, 1940Place: Recreation Room  
Powell HallTime: 12:15 to 1:15 P.M.Program: Movie: "Polar Trappers"The Vitamin B Complex  
Olaf Mickelson  
Frederick W. Hoffbauer  
Wm. H. Hollinshead  
Evrel LarsonDiscussion:C. J. Watson  
G. O. Burr  
Wallace Armstrong  
B. A. Watson  
J. L. McKelvey  
G. W. AndersonPresent: 149  
- - -Corrections:

1. Bibliography for The Vitamin B
- 
- Complex, Vol. XI, No. 14, p. 198:

Meiklejohn, A. P. and Kark, R., 1939,  
"Pigment Excretion in Pellagra,"  
New Eng. Med. J., Vol. 221, p. 519.

2. Mr. Andrew "Scotty" McGilp should
- 
- have been Mr. Andrew Fischer "Scotty"
- 
- McGilp. It was not their third
- 
- child; it was their fourth child.
- 
- It was a boy.

Gertrude Gunn  
Record Librarian  
- - - - -II. MOVIETitle: "Ether Bed"Released by: Visual Educ. Dept.  
- - -III. ANNOUNCEMENTS1. LECTUREDr. Erwin R. Schmidt,  
Professor of Surgery, University of  
Wisconsin will give a lecture at the  
regular monthly meeting of the Hennepin  
County Medical Society in its quarters  
on the top floor of the Medical Arts  
Bldg., Feb. 5, 1940 at 8:00 p.m.  
Subject - "Peritonitis Secondary to  
Disease of the Appendix." Staff members  
are cordially invited to attend.  
- - -2. LECTURESigma Xi - Friday,  
February 2 - "Sulfanilamide and Related  
Chemicals in the Treatment of Infectious  
Diseases" - Dr. Wesley W. Spink.  
Orchestral music - 7:45 - 8:15.  
Lecture 8:15. Cyrus Northrop Memorial  
Auditorium.  
- - -3. CONTINUATION STUDY COURSEProblems of Newborn  
and Premature Care, February 8 - 10,  
- - -4. BABIESSon of Dr. and Mrs.  
Manuel Ramos, born January 19. Weight  
7½ pounds. Dr. Ramos, who is a fellow  
in pediatrics, is from Manilla, P. I.  
- - -

IV. LESIONS IN THE MACULA AND AREA  
CENTRALIS AS A CAUSE OF  
VISUAL IMPAIRMENT

Karl Benkwitz

Of all the retina only that part designated the macula concerns central vision where our acute visual sensations are recorded. This is a small pinpoint area seen on fundoscopic examination temporal to the optic nerve by two disc diameters. As seen with the ordinary ophthalmoscope it is relatively avascular with terminal branches of the superior and inferior temporal central retinal vessels spreading out about it, in ring-shape fashion, demarcating the Area Centralis. It is in the center of the Area Centralis that one can see the macula with its small yellow foveal reflex. The Area Centralis is well nourished by capillaries from these vessels, but the fovea is a capillary-free area. This foveal avascular layer is nourished especially by the choriocapillaries of the choroid, although of course nutritional fluid comes by diffusion and imbibition from the surrounding retinal vessels. This indirect nutrition makes the macular area prone to degeneration of a special character. Also, at the macula the nerve fiber layer of the retina spreads apart. Characteristic only of the macula is the thick layer of ganglion cells. At the fovea both the cone layer and the outer nuclear layer are thicker than elsewhere and bulge inward; the inner nuclear layer is extremely thin and the nerve fiber, ganglion cell and inner plexiform layers disappear leaving a funnel-shaped sunken depression in the retina.

Pathological lesions which involve the macular area of the retina impair the central visual function in part or in toto according to their severity. Some lesions may be temporary and go onto spontaneous healing, others are intermittent and recur while the majority once present remain throughout the life of the individual. The papillomacular bundle may also be affected by ascending degeneration, starting at the macula, causing temporal atrophy of the disc.

When the central vision is lost, the patient sees no better than 20/200 and is unable to read fine newspaper print. The form field which describes the status of the peripheral vision remains normal. Various entoptic phenomena are described by these patients, such as metamorphopsia, i.e., objects are seen with abnormal curvatures and outlines as viewed in multiple curved mirrors of the amusement park midways. Micropsia and macropsia are noted in which objects are seen respectively smaller or larger than their normal size. Often the patients are aware of central positive scotomas of variable size in proportion to their macular lesion. In some instances there is a temporary hyperopia which when corrected with the proper convex spherical lens restores central vision to nearly normal. Usually the general health of the patient is excellent and the oculist is consulted only for improvement of the impaired central vision. The general ocular examination most often discloses no pathology except for the insidious macular lesion. And, often when such a lesion is identified all attempts fail to show concurrent disease.

The lesions bear special study and frequent reexamination to give a real evaluation of their character. The pupils are dilated with mydriatics and the fundi are studied with the ordinary ophthalmoscope or with Vogt's red-free light method which gives a greenish hue to the fundus and makes apparent macular lesions, which are often yellow and iridescent, but lost in the surrounding yellow of the fundus and the brilliant light reflex. Also, the binocular ophthalmoscope gives us a stereoscopic picture showing elevations or depressions if present, and, similarly, the slit-illumination of the giant Friedenwald ophthalmoscope offers this diagnostic aid. Serial photography has been employed, both plain and stereoscopic, for diagnosis and for follow-up records.

During the past two years a group of 112 cases of various types of macular degeneration seen in the eye clinic and on the wards of this hospital have been studied. It is from this group that we

wish to summarize and show slides representing most types of disease of the macula and Area Centralis.

First to be considered are the macular lesions referable to TRAUMA to the eye. First of this group is the lesion Comotio Retinae (Berlin's Traumatic Macular Edema). Several hours after a blow on the anterior surface of the eyeball a grayish-white spot appears in the macular region, usually the point opposite the point of application of the force. It is larger in size than the disc, the margins are indistinct, and the center area is redder. In severe cases the area around the disc may appear cloudy and hemorrhage may develop in the retina. Vision is reduced to 20/50 or 20/200 with the presence of a central scotoma for the first 24 hours, but soon begins to improve and within 5 to 6 days vision, fields and fundus pictures are normal, though some fine pigment and yellow-red patches may later appear as a permanent change.

The next traumatic lesion is that of Rupture of the Choroid which is seen fairly frequently. It is really a rupture of the lamina vitrea of the choroid, which retracts, leaving a crescentic area in which the sclera is visible. These can be produced either by direct impact or by concussion. The ruptures are usually at first concealed by the hemorrhage, but when this is absorbed, the rupture appears as a large white band or patch with clear-cut or irregular edges and sharp or ragged tips. Pigment shows later on the surface and margins. This is the common baseball and basketball injury to the eye. If the lesion involves the macula a central positive scotoma results with visual acuity of 20/200 or less, and this remains as a permanent sequelae.

The final traumatic lesion is a "Hole" in the Macula. A peculiar result of concussion acting directly on the eyeball is a round or oval rupture of the retina which, with the ophthalmoscope, looks like a hole in the center of the macular region. The bottom of the hole is depressed in elevation with the level of the retina and has a reddish color, contrasting so strongly with the rest of the retina that the first impression is that of hemor-

rhage. The margins are clear cut. Sometimes the hole contains yellowish cholesterol dots and the surrounding retina may be grayish in color and appear edematous or as though a slight detachment of the adjacent retina existed. Usually the lesion is  $1/3$  to  $1/2$  the size of the disc, but not necessarily always right at the macula. When at the macula, a central positive scotoma and 20/200 vision exists as a permanent finding. Boxers are prone to any one of these injuries though their frequency of occurrence is small.

The next group of macular lesions are present because of an INFLAMMATORY REACTION. Most all macular lesions are confused with inflammatory conditions and treated as such when inflammatory lesions at the macula are relatively infrequent, only 32 cases out of 112 were studied by us. First of this group to be considered are the Conglomerate Tubercle and Focal Exudation at the Macula. In chronic tuberculosis, isolated macular involvement may occur either as a large focal lesion with exudation or as a conglomerate tubercle. It usually covers a large area, two or more times the size of the disc and has a grayish color with diffuse margins, and is somewhat raised. The margins of the lesion often show hemorrhages. Vitreous opacities are usually present. In other cases there is a diffuse yellowish decoloration within which small, round nodules may be found. These patches heal, leaving a large, atrophic and pigmented scar simulating macular coloboma.

This type of central chorio-retinitis occurs in persons without signs of active pulmonary tuberculosis, but with positive Mantoux and latent foci in the lymph nodes. It is more common in young people and the lesion has a chronic course; often both eyes are affected.

The solitary or conglomerate type represents a tumor-like mass composed of great numbers of miliary tubercles which have coalesced. In this condition the lesion is accompanied by acute symptoms of inflammation, due to caseation and absorption of large amounts of bac-

terial products. The posterior portions of the globe may go on to perforation.

Next of the inflammatory lesions are the Metastatic Macular Isolated Foci of Chorio-retinitis. These occur in endogenous infections (pyemia, septicemia, meningitis, acute infections, etc.) where septic emboli detach from the focus of suppuration and are carried by the blood stream to the vessels of the retina and choroid--often terminating at the macula or the Area Centralis. They are capillary emboli, and an inflammatory lesion develops which may lead to suppuration and necrosis.

With the ophthalmoscope the exudate of acute plastic choroiditis appears upon fundus examination as a yellowish-red or yellowish-white spot about 1/2 to a large size of the disc. The retina at first is raised over the lesion. Later pathological pigment appears in the fundus--surrounding the lesion. After several weeks the exudate absorbs and in its place the choroid and retina lose their normal structure and are transformed into connective tissue. The color of the focus changes more and more, the white background of the sclera becoming increasingly visible--called the atrophic spot. Some large choroidal vessels may be seen within the atrophic area. Recurrences or acute exacerbations with a plastic exudate surmounting the atrophic area do occur.

Mantoux tests are negative. No evidence of chronic or active tuberculosis can be found on general and roentgenological examination, but often non-specific foci can be demonstrated which after their eradication the inflammation subsides to form a patch of chronic focal chorio-retinitis. Dental and sinus foci of infection are the frequent offenders. The term metastatic as used here has no reference to neoplastic growths.

The third large group of lesions found at the macula has been described under different names by numerous clinicians in various parts of the world, but probably all fall in the same category, i.e., IDIOPATHIC CENTRAL RETINAL DETACHMENTS. The Germans and Japanese designate these cases as Serous Central Chorio-retinitis

while at the Wilmer Institute of Ophthalmology Walsh and Sloan refer to the condition as Idiopathic Flat Detachment of the Retina at the Macula, and more recently Gifford has described a similar condition as Central Angiospastic Retinopathy. The interest here is so keen since it is in this group that complete recovery usually returns as to normal vision, fields and fundi.

The typical lesion is that of a clear elevation of the retina off the choroid by a transudate sometimes containing small cholesterol-like, pinpoint deposits. In some instances the periphery of the lesion is well demarcated by a sharp white line in the retina and radially directed streaks of light reflex leave their margin. The retina itself shows no abnormalities and the lesion is for some unknown reason confined to the macula. There is usually a relative scotoma for colors and metamorphopsia, micropsia and macropsia are present. As the lesion heals, the retina flattens, hyperopia decreases and the relative scotoma disappears. The vision, if corrected with the proper convex optical lenses, remains normal, but the glass correction can be discarded when the pathology disappears. The ocular media remain clear. There are no retinal vascular abnormalities. The etiology in this country is attributed to foci of infection and allergy, while the Germans and the Japanese believe this a tuberculous exudate rather than a transudate or edema. Occasionally the lesion persists in a subacute or chronic stage for many years with partially impaired vision and relative scotomas. The young and middle-aged adults, chiefly men, are affected. A group of investigators in this country and also in Germany choose to call this form Juvenile Macular Exudative Retinitis because of the youth of the patient, usually negative medical findings, moderate chronicity without great reduction of vision as compared to the appearance of the retinal lesion, a tendency to recovery of vision earlier than cure of the lesion, and finally in many cases recovery of normal vision with a normal or almost normal appearing macula and the absence of hemorrhage.

The lesions in the cases of Central Angiospastic Retinopathy resemble in every way those of Serous Central Chorio-retinitis except for the retinal vascular evidence of spasm of the smaller arterioles supplying the macular region with no evidence of generalized hypertension.

In these cases histories disclose symptoms of coldness and paraesthesias of the extremities. Blanching of hands and feet in cold atmospheres, trophic changes such as flushing of extremities in dependent positions and pallor in elevated position. Temperature readings of the skin of extremities in draft-free rooms are lower than the usual 4 to 6 degrees F. from those taken of the abdomen, and after block of the tibial nerve greater rises than normal are recorded beyond the block. Oculometry readings show a decrease which is evidence of decreased arterial flow. Capillary microscopy of the bed of the toe and the finger nail shows decrease in number of visible capillaries by 30 to 40%. Those present are markedly diminished in calibre and show the phenomena of "plasma-skimming" in which segments of capillaries are empty of red blood cells, while closely packed cells are seen in that portion of the loop proximal to the narrowed segment. Here the Wassermann and Mantoux tests are negative. In smokers the effect of smoking on the extremities and the retinal arterioles is striking, often precipitating an attack of loss of central vision.

The circulatory origin of the condition makes for the transient attacks, with complete recovery and frequency of recurrences of macular subretinal edema, and temporary visual impairment often correctable with hyperopic optical corrections.

Often these types of cases are mistaken for cases of retrolubar neuritis because of the difficulty of viewing and recognizing the pathology at the macula.

The next large group of macular lesions are those of the DEGENERATIVE group. The first type of lesion under this grouping is the Juvenile and Senile Macular Disciform Degenerations. The Juvenile group has just been referred to. The Senile group consists, as the name implies, of

disc-shaped lesions in the macula and the area centralis. They are found usually in old adults, 50 to 70 years of age, and both eyes are involved though not necessarily both at the same time. The vision because of the large size of the lesion, 1 to 2 and 3 times the size of the disc, is reduced to 20/200 or less with the presence of a large absolute scotoma. The remaining fundus examination reveals a normal optic nerve and retina. The retinal, and if visible, the choroidal vessels usually show advanced arteriosclerotic changes. The lesion is characterized by the presence of an elevated gray mass in the macula with deep hemorrhage, pigmentary changes and frequent white, punctate areas of degeneration in the surrounding retina. Twelve cases were reported at the University of Iowa Eye Clinic in one year.

Sclerosis of the choroidal vessels, occurring with generalized vascular disease is believed to cause metaplasia and hyperplasia of the pigment epithelium with the formation of a mass resembling connective tissue between the choroid and retina. From a few cases observed throughout their course the lesion seems to begin with multiple hemorrhages in the macular area. Disciform degeneration when observed usually appears in the macular region as a yellowish-white or gray opaque mass situated beneath the transparent retina. The lesion may be only slightly elevated or it may reach an elevation of 5 to 6 diopters. Many of the lesions are disciform, but they may assume almost any form. Marginal hemorrhages and deposits of pigment are frequently noted. The surface may be uneven; hemorrhages and clumps of pigment may be present. Small cystic areas may be present in the retina over the lesion.

The lesion may be confused with choroidal malignant melanoma and often eyes are mistakenly enucleated because such a diagnosis has been made. These lesions show no tendency to disappear and vision and central field defects are permanent while peripheral vision remains normal.

There is a degenerative group of

macular lesions called Central Retinopathy due to Drusen. In middle age and later periods of life nodular thickenings of the lamina vitrea membrane of the choroid may occur which are known as verrucae or "drusen." They are little round, light or yellowish-white spots of, or a little larger than, a medium size retinal vessel which usually are not associated with pigment changes. They are found around the papilla and in the periphery but most frequently concentrated at the area centralis and macula. These spots are due to colloid deposits on the lamina vitrea and are probably produced by the retinal pigment epithelium. In some instances they reach such a large size that they push the pigment epithelium apart and become visible in the fundus as highly iridescent, crystalline, gold-like particles interspersed with clumps of retinal pigment. Often no visual disturbance is produced, but when they become so numerous in the macular region they produce pressure atrophy of the visual cells with pigment accumulation and vision is reduced to 20/40, 20/65 or 20/200 with the presence of an associated central scotoma. Form fields remain normal. Changes are all permanent. English authors have recognized a group of such cases in which numerous hyaline bodies and extensive retinal macular degeneration exists on a hereditary basis and have called the condition Doyme's guttate choroiditis.

We have found abnormalities of cholesterol metabolism in these cases with fasting blood cholesterol determination elevated up to 375 mgm. per 100 c.c.

Retinitis Circinata is a condition of degeneration of the retina in which numerous white spots and hemorrhages develop about the macula in the area centralis. The lesion as its name implies assumes the shape of a ring or wreath or an ellipse, sometimes complete but usually interrupted. The white areas are composed of a conglomeration of small dots which coalesce, forming elongated patches with cremated margins. Between them, scattered in an irregular manner small or large hemorrhages are present. Inside the white wreath the retina is diseased and sooner or later the macula undergoes a

special degeneration with pigmentary changes and sometimes disciform degeneration. An absolute scotoma then results with 20/200 the best vision obtainable.

Marked arteriosclerosis of the retinal vessels is most often present especially the small arterioles supplying the area centralis. The retina shows on section cystic degeneration of the external plexiform layer containing amorphous masses of exudate and fat-laden cells and areas of hemorrhage. In milder cases the wreath of white spots may disappear but the rule is that they remain unchanged for years. The carinate arrangement of the lesions may sometimes be present in cases of diabetic retinosis.

Angioid streaks of the retinae is a term used to describe a peculiar distribution of dark colored broad lines in the retina about the optic nerve. From this circular streak around the disc, allied streaks take their origin and run more radially out toward the periphery. The streaks vary in color from reddish to dark brown. They lie behind the retina and before the larger choroidal vessels. No connection is made between them and the vascular system. Most cases of angioid streaks of the retina are accompanied by extensive lesions in the macula. Three stages have been described in the development of lesions in the macula:

First stage:--the angioid streaks are found without any visible lesion in the macula and associated with normal vision.

Second stage:--a rather sudden decrease in visual acuity occurs, which is due to detachment of the retina in the macular area. Beneath this detachment, according to Wilde, is a subretinal transudate.

Third stage:--the transudate is organized by connective tissue, and a pale grayish macular disc is formed; occasionally hemorrhage in the fundus is identified. Vision is reduced to 20/200 and an absolute scotoma is formed. The optic disc, retinae and, if visible, choroidal vessels show no visible alter-



ations. There has been a recent discovery of their frequent association with a rare skin lesion, Pseudoxanthoma elasticum, which suggests that both conditions are due to a degeneration of elastic tissue.

#### Cystoid and Honeycomb Macular Retinosis.

This condition is best viewed with red-free light and often it is the forerunner of macular "Holes" in cases not subjected to trauma or infection. It is usually found in the old adults but occasionally in the younger adults. The yellow area at the macula appears to be divided into numerous round polygonal, honeycomb-like spaces whose walls are represented by white lines. When these spaces become larger, true cysts are formed--cystoid degeneration. If the septa between the cysts ruptures, then a "hole" is formed in the macula. Sometimes hemorrhages are seen in the cysts. The condition is seen often in old arteriosclerotic people. Cystoid degeneration is a progressive disease. A central scotoma is present and vision is reduced to 20/50 or 20/200. This defect is permanent and the scotoma enlarges with progression of the lesion.

Myopia. In certain cases of myopia degenerative changes occur, while these are more common in the higher degrees of myopia and in persons past middle age, they may occur in myopia of only moderate degree and may begin at a relatively early age. The commonest change is an atrophy of the pigment epithelium in the macular area. This usually appears as a light area often surrounded by pigment. In high degrees of myopia the bulging backward of the posterior pole may be seen by a bending of the vessels at its edges (posterior staphyloma). These fundus changes, while often referred to as myopic choroiditis, have nothing to do with inflammation and must be considered as a form of degeneration due to thinning and stretching of the inner coats of the eye as it enlarges. In the course of this progress hemorrhage may occur in the retina leaving further atrophy and pigment displacement. Vision again is reduced to 20/200 with the presence of an absolute central scotoma; the peripheral vision remains normal.

Solar Retinosis. Exposure of the eyes

to the direct sunrays or to strong electric lights even for a few seconds may produce serious damage to the macula. The refractive media of the eye act as an ordinary convex lens, concentrating the sunrays in its focus and producing a "burning" of the retina. Insufficient protection of the eyes during an eclipse is the most common cause of solar burns. Looking at powerful electric lights may have similar effects; workers in electric plants, or those using acetylene torches show degrees of visual impairment varying from persistence of after images to transitory or central scotoma with impaired vision to 20/50, 20/65 or 20/200. Distortion of objects is often present. The lesion is permanent. The pathological lesion found in the macula consists of small black pigment dots and yellowish-white spots or a maroon colored area with a gray central patch.

Arteriosclerotic Senile Macular Degeneration is associated usually with definite arteriosclerotic retinal vascular findings. It is often considered in part associated with the senile hereditary macular degenerative changes. The retina at the macula shows small foci of pigment accumulation interspersed by areas of apigmentary degeneration, called "cobblestone" degeneration. Arteriosclerosis of the retinal arteries and veins is prominent; also, the choroidal vessels show similar changes. The lesion if found in old adults producing loss of central vision in part or completely.

THE HEREDITARY CEREBROMACULAR ABIOTROPHIES are the next group of macular lesions to be described. They consist of a group of diseases showing familial and hereditary tendencies with concurring retinal macular and cerebral pathology. Cerebral pathology may be absent in many cases. The word abiotrophy has been coined to signify the degeneration and death of certain cellular elements at a certain stage in the life cycle, due to pre-existing hereditary factors. In each condition included in this group a specific group of cells is predominantly affected, and the change makes its appearance at a certain

period of life.

First of this group are the cases of Amaurotic Familial Idiocy of which two divisions now exist. First the Infantile Form (Tay-Sach's Disease). Inheritance is of the dominant variety here and the Jewish race is almost exclusively affected. The disease begins in the third to the sixth month of life, and death usually occurs from one to two years later. The retinal changes consist in a white ring of intense edema surrounding the foveae centralis which appears cherry-red by contrast. The edema is due to swelling and destruction of the ganglion cells which are most numerous in this region. This pathological process involves also the brain and the spinal cord again with degeneration of their ganglion cells. Optic atrophy of the disc is present as the disease progresses. The infants appear to be blind or nearly so. Rapid mental and physical deterioration are evident at the same time and progresses until death. Both eyes are involved. The second division is called the Juvenile Form (Spielmeyer and Vogt disease). In this type the symptoms of degeneration do not make their appearance until four to eight years of age, and are much slower in onset. The complexus is recessive, inherited, family disease; and the predilection for the Jewish race is less marked than in the infantile form.

The general symptoms are: increasing motor weakness ending in complete paralysis (familial cerebral diplegia), epileptic seizures, and lastly, idiocy. Vision fails gradually until the patient is completely blind. Death may come anywhere from the tenth to the twentieth year of life.

The retinal lesion, when first seen, has usually existed long enough so that a coarse granular black pigmentation of the macular area has occurred. Pigment is present in the periphery, exclusively in certain cases, the picture resembling pigmentary degeneration of the retina, while in other cases both peripheral and macular areas are affected. The disc becomes atrophic and retinal vessels are normal. Central scotoma is present which increases in size and vision is

20/50 or 20/200 and progresses to blindness. Both eyes again are involved.

Finally we have the Hereditary Cerebro-macular Degenerations. In this condition local spontaneous retinosis occurs in the macula, appearing at different periods of life. The fundus picture consists of a moderate edema of the macular region which is soon replaced by the appearance of fine pigment granules and often of shiny lipoid deposits. The macular lesion may be one-half to one or two times the size of the disc and though elevated slightly in some cases the demarcation from the surrounding retina is most difficult. Vision is usually reduced to 20/200 and large absolute central scotomas are present. The characteristics of these cases are: 1. The involvement of both eyes, which show extremely similar pictures. 2. The hereditary or familial character of the disease. Transmission is usually made by the affected females. Consanguinity is frequently found. The retinal vessels and periphery of the fundus are normal. Although no ophthalmic picture can be considered typical for each group, the fundus details are very similar in cases occurring in the same family and usually start at the same age.

Another form of fundus change resembles a coloboma of the macular region and appears as a large round or irregularly oval, yellowish white patch with clear cut margins and peripheral pigmentary deposits.

The periods of incidence of the disease are:

- (a) appearing at birth (congenital type)
- (b) from 6 to 8 years of age, at the beginning of the second dentition (infantile type)
- (c) in the period between 14 years when puberty begins, and 20 years, when skeletal growth ends (juvenile type)
- (d) in adults from 50 years on (beginning of senile involution) (Senile type)

The cerebral lesions are usually asso-

ciated with the congenital and occasionally the infantile types. Epileptic seizures often occur followed by complete idiocy or the patient may have just the mental status of a moron. In the juvenile and the senile forms the eyes alone may be affected.

The pathological changes at the macula consist in an abiotrophy of the cones and a migration of pigment granules from the pigment epithelium to the surface of the retina or the granules completely disappear.

The Senile Type occurs at a much more advanced age and is very slowly progressive. A hereditary predisposition to the condition undoubtedly exists and is probably the only known etiological factor. Arteriosclerosis has been considered a factor, and accounts for a certain number of these cases but it is seldom more marked than in other persons the same age with no macular lesions. The condition is most common of those just mentioned, being one of the most frequent causes of disappointing visual results following cataract extraction. It is marked with gradual loss of central vision to 20/200, with the appearance of central absolute scotoma, while the peripheral fields remain normal. The fundus picture shows a fine granular appearance at the maculae which become more coarsely pigmented in later stages. Red-free light discloses peculiar multicystic or honeycomb-like structures at the macula in many cases. Rarefaction of the retina may occur with an area the size of the disc in which choroidal vessels are visible.

Some references refer to senile cases in which memory fails, speech becomes difficult, patients are irritable and are in a pitiable mental state.

Coloboma of the Macula is the last of the macular lesions of our classification. Coloboma in its typical form, is the result of faulty closure of the fetal fissure in the embryo. This causes a complete defect in the pigment epithelium and choroid in an area below the disc which usually extends to the ora serrata-called coloboma of the choroid. Ophthal-

moscopically it is seen as a large white semi-oval area with its rounded end toward the disc and its base below.

Coloboma of the macula is a similar defect in the choroid at the macular area. It is round or oval, usually with a sharply defined border surrounded by pigment. Pigment is often seen scattered across the defect. A large central scotoma is present and vision is reduced to 20/200 or less. It has been explained as due to nutritional deficiency or intra-uterine inflammation at the time when this area is undergoing development, but has occurred in certain families so frequently as to constitute a hereditary anomaly. Coloboma of the optic nerve, of the iris or eyelid often are concomitant findings.



Bibliography

1. Gifford, H.  
A Case of the juvenile form of family amaurotic idiocy.  
The Ophthalmic Record. 21:577-595, 1912.
2. Spielmeier  
Juvenile form of amaurotic family idiocy.  
Neurol. Centralbl. 1906, p. 2.
3. Batten, F. E.  
Cerebral degeneration with symmetrical changes in the maculae in two members of a family.  
Trans. Ophth. Soc., U. Kingdom. 23: 386-390, 1903.
4. Vogt, A.  
Juvenile form of amaurotic family idiocy.  
Monatschr. f. Psych. u. Neurol., 18:163, 320.
5. Vogt, A.  
Juvenile form of amaurotic family idiocy.  
Monatschr. f. Psych. u. Neurol. 18:403 (Nov.) 1907.
6. Vogt, A.  
Juvenile form of amaurotic family idiocy.  
Arch. f. Kinderheilk. 51:1.
7. Behr, Carl  
Juvenile form of amaurotic family idiocy.  
Monatschr. f. Psych. 28:327.
8. Behr, Carl  
Die Heredodegeneration der Makula.  
Klin. Monatsbl. f. Augenheilk. 65:465-505, 1920.
9. Gifford, Stanford  
Central retinopathies.  
Paper presented at the Center for Continuation Study, University of Minnesota, Post-graduate course in Ophthalmology, 1939.
10. Gesell, Arnold and Blake, Eugene  
Twinning and ocular pathology with a report of bilateral macular coloboma in myozygotic twins.  
Arch. Ophth. 15:1050-1071, 1936.
11. Hughes, Nicholas  
Macular disease in children.  
Trans. Ophth. Soc. U. Kingdom. 51:516-520, 1931.
12. Evans, P. J.  
Familial macular coloboma.  
Brit. J. Ophth. 21:503-506, 1937.
13. Vogelsang, K.  
Macular coloboma in like twins.  
Klin. Monatsbl. f. Augenh. 98:322-327, 1937.
14. Sorsby, Arnold and Oliver, John O.  
Macular coloboma; historical report.  
Brit. J. Ophth. 23:724-729, 1939.
15. Sorsby, A.  
Familial central choroidal angioid sclerosis.  
23:433, 1939.
16. Sorsby, Arnold  
Congenital coloboma of the macula, together with an account of the familial occurrence of bilateral macular coloboma in association with apical dystrophy of hands and feet.  
Brit. J. Ophth. 19:65-90, 1935.
17. Wexler, David and Last, Murray  
Colobomas of the optic nerve and of the macula; a microscopic study.  
Arch. Ophth. 20:787-797, 1938.
18. Samuels, Bernard  
Papilledema with cystic degeneration.  
Am. J. Ophth. 21:1242-1259, 1938.
19. y Troncoasa, Manuel U.  
Diseases of the macula. Internal diseases of the eye and atlas of ophthalmoscopy.  
Philadelphia, F. A. Davis Co., 1937.

20. von Grolman, G.  
Recent observations on central nervous chorioretinitis.  
Arch. de Oft. de Buenos Aires. 13:235, 1938.
21. Edgerton, A. E.  
Report of a case and review of the literature.  
19:463-469, 1936.
22. Kohler, A. R. and O'Brien, C. S.  
Disciform degeneration of the macula.  
Arch. Opth. 13:937-959, 1935.
23. Davis, W. T. and Sheppard, Ernest  
Juvenile macular exudative retinitis-- (Junius-Kuhnt).  
Arch. Opth. 13:960-970, 1935.
24. Junius, Paul  
Erscheinungsformen und Ablauf der juvenilen Retinitis exudative macularis.  
Ztschr. f. Augenh. 70:129, 1930.
25. Junius, Paul and Kuhnt, Hermann  
Die Scheibenförmige Entartung der Netzlaufmitte.  
Berlin, S. Karger, vol. 7, 1926.
26. Walsh, Frank and Slaon, Louise  
Idiopathic flat detachment of the macula.  
Am. J. Opth. 19:195-208, 1936.
27. Kitahara, Sakae  
Über klinische Beobachtungen bei der Japan häufig vorkommenden Chorioretinitis centralis serosa.  
Klin. Monat. f. Augenh. 97:345, 1936.
28. Prevec, S.  
Central retinal detachment.  
Klin, Monatsbl. f. Augenh. 100:222-235, 1938.
29. Streiff, E. B.  
Serous central chorioretinitis and its differentiation from central angioneurotic retinitis.  
Klin. Monatsbl. f. Augenh. 103:524-530, 1939.
30. Verhoeff, F. H. and Grossman, H. P.  
Pathogenesis of disciform degeneration of the macula.  
Arch. Opth. 18:561-585, 1937.
31. Würdemann, Harry Vanderbilt  
Formation of a hole at the macula; light burn from exposure to electric welding.  
Am. J. Opth. 19:457-460, 1936.
32. Gifford, S. R. and Marquardt, Gilbert  
Central angiospastic retinopathy.  
Arch. Opth. 21:211-228, 1939.
33. Goedbloed, J.  
The syndrome of Groenblad and Strandberg; angioid streaks in the fundus oculi, associated with pseudoxanthoma elasticum.  
Arch. Opth. 19:1-8, 1938.
34. Lloyd, Ralph I.  
Hereditary macular degeneration.  
Am. J. Opth. 19:216-221, 1936.
35. Nathan, David  
Maculo-cerebral degeneration.  
Am. J. Opth. 21:1029-1031, 1938.
36. Collins, E. T.  
Hereditary ocular degenerations-- "ophthalmic abiotrophies."  
International Congress of Ophthalmology, Washington, D. D., 1922 (Apr.) pp. 103-143.
37. Day blindness and color blindness with macular degeneration.  
Duke-Elder Text Book of Ophthalmology. London, Henry Kimpton. Vol. 1, 1938, p. 977.
38. Tree, M.  
Familial hyaline dystrophy in the fundus oculi or Doyme's family honeycomb choroiditis.  
Brit. J. Opth. 21:65-92, 1937.
39. Evans, P. J.  
Familial macular colobomata.  
Brit. J. Opth. 21:503-506, 1937.

40. Gifford, S. R.  
A text book of ophthalmology  
Philadelphia, W. B. Saunders Company  
1938, pp. 297-304.
41. Lowenstein, Arnold  
Die Tuberkulose des Auges.  
Berlin, Germany, Urban and Schwarzen-  
berg, 1924.
42. Wurdemann, Harry V.  
Ophthalmoscopic studies of the  
macula lutea. Contributions to  
Ophthalmic Science.  
Jackson's Birthday Volume. Menasha,  
Wis., George Banta Publishing Company,  
1926, pp. 264-273.
43. Oatman, E. L.  
Diagnostics of the fundus oculi.  
The Southworth Company, vol. 3, pp.  
55, 56, 57, and 59.
44. Weeks, J. E.  
Macular retinitis. Diseases of the  
eye.  
Lea & Febiger, 1910, pp. 471-477.
45. Pyle: Macular and posterior polar  
regions. In: System of Ophthalmic  
Practice. chapt. 15, p. 296.
46. Cornwall, L. H.  
Heredity in nervous and mental dis-  
eases.  
Paul B. Hoeber, Inc., vol. 3, 1923,  
pp. 114-124.
47. Behr, Carl  
Die Anatomie der "senilen Makula"  
(der senile Form der makularen  
Hereditäregeneration).  
Klin. Monatsbl. f. Augenh. 67:551-  
564, 1921.
48. Wright, Lieut.-Col. R. E.  
Familial macular degeneration.  
Brit. J. Ophth. 19:160-165, 1935.
49. Perera, C. A.  
Retinitis pigmentosa with "hole" in  
the maculae; report of a case.  
Arch. Ophth. 20:471-474, 1938.
50. Braun, R.  
Pathological anatomy of disciform de-  
generation of the macula.  
Arch. f. Augenh. 110:534, 1937.
51. Shoemaker, W. T.  
Serous exudate beneath the retina  
clinically simulating sarcoma of  
the choroid or ciliary body.  
Ed. Jackson Contributions to Oph-  
thalmic Science. Menasha, Wis.,  
George Banta Publishing Company.  
1926, pp. 146-148.
52. Blue, Robert  
Family degeneration of the macula  
lutea. Section on Ophthalmology  
of J. A. M. A., 1919, pp. 191-202.
53. Clark, H. S.  
Familial macular degeneration with  
and without dementia, with a re-  
port of two new cases of the type  
with dementia.  
Section on Ophthalmology of J.A.M.A.  
1918, pp. 104-120.
54. Feingold, Marcus  
Progressive macular degeneration  
in three members of a family.  
Section on Ophthalmology of J.A.M.A.  
pp. 312-325  
Arch. Ophth. 45:532-543, 1916.
55. Borsellino  
Macular degeneration and bilateral  
microphthalmos in three brothers.  
Brit. J. Ophth. 20:598, 1936.
56. Mann, Ida C.  
Certain conditions of the macular  
region usually classed as colobo-  
mata.  
Brit. J. Ophth. 11:99-116, 1927.
57. Batten, R. D.  
Angioid streaks, and their relation  
to a form of central choroidal  
disease.  
Brit. J. Ophth. 15:279-289, 1931.
58. Crawford, J. W.  
A hole in the macula.  
Pacific Coast Oto-Ophth. Society.  
1933, pp. 26-35.
59. Dykmann, A. B.  
Angioid streaks of the retina with  
association of hemorrhage into the  
macula and pseudoxanthoma elasti-  
cum.  
Pacific Coast Oto-Ophth. Society.  
1933, pp. 36-49.

60. Doyne, R. W.  
Peculiar condition of choroiditis occurring in several members of the same family.  
Trans. Ophth. Soc. United Kingdom. 19:71, 1899.  
Further notes on family choroiditis.  
Trans. Ophth. Soc., U. Kingdom. 30:274-276, 1910.
61. Kraupa, Ernst  
Die Kongenital luetische zentral Retinochoroiditis.  
Ztschr. f. Augenh. 89:204-208, 1937.
62. Marshall, C. R.  
Entoptic phenomena associated with the retina.  
Brit. J. Ophth. 19:177-201, 1935.
63. Pavia, J. L.  
Edema de Burlin y alteraciones cono-retinianas consecutivas.  
Rev. Oto.-Neuro.-Oftal. 9:289, 1934.  
Abstr. in Brit. J. Ophth. 19:689, 1935.
64. Arnold, A. Max  
Weitere Beiträge zur Kenntnis der cystoiden Maculadegeneration (Bienenwabenmacula) mit Bemerkungen zur Technik des rotfreien Lichtes.  
Arch. F. Ophthal. 122:299, 1929.
65. Neulen, E. N.  
Central retinal hemorrhage, synchronous with onset of menstrual period.  
Pacific Coast Oto-Ophth. Society. 1925, pp. 31-34.
66. Davenport, R. C.  
Senile macular exudative retinitis.  
Trans. Ophth. Soc. U. Kingdom. 46:137-158, 1926.
67. Dobson, Margaret  
Diseases of the macula, seen with red-free light.  
Trans. Ophth. Soc. U. Kingdom. 46:158-167, 1926.
68. Batten, R. D.  
Macular disease, with special reference to acute primary macular disease.  
Trans. Ophth. Soc. U. Kingdom. 41:411-413, 1921.
69. Hay, P. J.  
Nutritive supply of the macular region in the light of a case of embolism of the inferior temporal artery.  
Trans. Ophth. Soc. U. Kingdom. 41:481-485, 1921.
70. Whitehead, A. L.  
Persistent central scotomata following exposure of the eye to direct sunlight during the solar eclipse.  
Trans. Ophth. Soc. U. Kingdom. 42:278-280, 1922.
71. Williamson-Noble, F. A.  
Macular lesions associated with thrombosis of the central retinal vein.  
Tr. Ophth. Soc. U. Kingdom. 43:287-296, 1923.
72. Batten, R. D.  
Symmetrical destruction or absence of retina in macular region? Correlated to the maculo-cerebral degeneration of adolescence and infancy. Tr.  
Tr. Ophth. Soc. U. Kingdom. 42:109-110, 1922.
73. Stock, Wolfgang  
Pathologische Anatomie des Auges. Pub. Ferdinand Enke, Verlag Stuttgart, Germany, pp. 133, 144 and 151.
74. Hughes, C. A.  
Bilateral central choroiditis.  
Trans. Ophth. Soc. U. Kingdom. 58:438-439, 1938.
75. Lawson, Arnold  
An unusual variety of macular hemorrhage, the case also exhibiting an anastomosis between the retinal and choroidal venous circulation.  
Tr. Ophth. Soc. U. Kingdom. 30:278-280, 1910.
76. Hepburn, M. L.  
Hemorrhage at the macula.  
Trans. Ophth. Soc. U. Kingdom. 30:82-84, 103; 1910.



77. Paton, Leslie  
Rare form of macular lesion with extensive retinal exudation and degeneration.  
Tr. Ophth. Soc. U. Kingdom. 22:174-176, 1912.
78. Moxon, Frank  
Hole in the macula associated with Tay's dots.  
Tr. Ophth. Soc. U. Kingdom. 31:260, 1911.
79. Collins, F. T.  
A case of widespread exudation internal to the choroid and beneath the retinal vessels, giving rise to a white reflex.  
Tr. Ophth. Soc. U. Kingdom. 31:112-115, 1911.
80. Nettleship, E.  
Some cases probably allied to Tay's infantile retinitis (amaurotic family idiocy).  
Tr. Ophth. Soc. U. Kingdom. 28:76, 1908.
81. Pusey, Brown  
Family degeneration of the macula lutea with suggestion as to its cause.  
Tr. Ophth. Soc. U. Kingdom. 14:364-370, 1915.
82. vander Hoeve, J.  
Schadigegen des Auges durch Lich. senile Linsentrübungen und senile macula-degeneration.  
Graefe Arch. f. Ophth. 98:49-66, 1919.
83. Parson, H. W.  
Affections of the eye produced by undue exposure to light.  
International Congress of Med., London, 1913.
84. Oeller, H. J.  
Atlas of rare ophthalmoscopic conditions and supplementary plates of the atlas of ophthalmoscopy.  
Wiesbaden, J. F. Bergmann, 1900.
85. Lisch, K.  
Rare forms of tapetoretinal degeneration.  
Klin. Monatsbl. f. Augenh. 98:498-504, 1937.
86. Bailliart, Schiff-Wertheimer and Miroux  
Lesions of the macula of capillary origin.  
Bull. Soc. d'opht. de Paris., 1935, p. 180.
87. Gjessing, G. A.  
Senile macula degeneration.  
Zeitschr. f. Augenh. 56:79, 1925.
88. Die Marzio, Quirino  
Fundus oculi diagnostica oftalmoscopica.  
Stab. L. Salomone, Roma via Ostiense. 75., 1937.
89. Gissy, C. S.  
Ein weiter Fall von "Präretinalem Oden."  
Ztschr. f. Augenh. 57:423-429, 1925.
90. Rieger, Herwigh  
Ein Beitrag zur Kusuistik der tapeto-retinalen Degeneration (Heredodegeneration der Makula mit Beteiligung der Peripherie bei drei Brüdern).  
Ztschr. f. Augenh. 57:429-463, 1925.
91. Werdenberg, E. F.  
Blendungsretinitis nach Sonnenfinsternis Beobachtung.  
Ztschr. f. Augenh. 30:273-316, 1911.
92. Deutschmann, R.  
Doppelseitige angeborene Lochbildung i. d. Macula.  
Beitr. z. A. 87:585, 1914.
93. Oatman, E. L.  
Maculo-cerebral degeneration (familial).  
Am. J. M. Sc. 1911. (April).  
Abstr. in Ophthalmoscope. 9:852, 1911.
94. Lutz, Anton  
Ueber eine Familie mit hereditärer-familiärer Chorio-Retinitis.  
Klin. Monatsbl. f. Augenh. 49:699-703, 1911.

95. Jennings, J. E.  
Symmetrical degeneration at the macula in three children of the same family.  
Am. J. Ophth. 26:296, 1909.
96. Weisenberg, V.  
Ueber einen eigenartigen Fall von akuter Chorio-Retinitis mit Gelbfärbung der Macula.  
Klin. Monatsbl. f. Augenh. 65:517-527, 1920.
97. Mayou, M. S.  
Cerebral degeneration with symmetrical changes in the maculae in three members of a family.  
Tr. Ophth. Soc. U. Kingdom. 24:142-145, 1904.
98. Stock, W.  
Ueber ein bis jetzt noch nicht beschriebene Form d. familiärer auftretenden Netzhaut degeneration bei gleichzeitiger Verblödung und über typische Pigment-degeneration d. Netzhaut.  
Klin. Monatsbl. f. Augenh. 45:225-244, 1908.
99. Family cerebral degeneration with macular change.  
Quart. J. Med. (July) 1914, and Medical Chronicle. (Sept.-Oct.) 1914. Quoted from Ophthalmoscope. 13:77, 1915.
100. Stargardt  
Ueber Familiäre progressive Degeneration i. d. Maculagegend d. Auges.  
Ztschr. f. Augenh. 30:95-116, 1913.

## V. GOSSIP

Much talk of diet these days at the Center for Continuation Study. The influence of our teaching force in nutrition is evidenced by the fact that over one half of the group of 100 dietitians who are here at the course come from the Twin City area. One is impressed by the fact that the number of physicians who understand dietary factors in health and disease (with the possible exception of pediatricians) is small indeed. Our teaching group of physicians was characterized by "youthfulness and good looks." Some of them came through interest in metabolic problems (diabetes, nephritis, etc.), others through allergy, some through gastrointestinal diseases, and others by way of deficiency disease. There are a number of physicians in our state who are also interested in these questions; notably, E. L. Tuohy of Duluth, who might qualify as "passable in looks." The dietitians themselves did a good job of providing a decorative note in the classroom. The air is full of vitamins, with our newly found friend "B" coming in for a great deal of attention. A popular source is pork with "chops and ham" leading the chosen foods. Other less interesting sources are also stressed. As each person advances to the front to shift the vitamin emphasis back and forth, one is impressed by the sanity lever of the group. Dr. Phil Brown of Rochester was most philosophical about the low state of affairs in the physician's knowledge of dietetics. Especially did he warn the young ladies to nod respectfully when they were asked to put the patient on a bland diet, but on the side they were not supposed to do it, as very few physicians knew what a bland diet was (if they did they would never order it). Studies in another clinic indicated that surgeons had very little interest in feeding their patients. Control periods in which surgeons alternated with internists showed that the internist knew more. The college nutrition table in the Health Service had an interesting report. Last year's basketball regulars needed 5,000 plus calories a day to keep from losing weight. Athletic programs which did not include attention to diets in the past must have been either a physical or a financial handicap to the

young participants. Medical students have little opportunity to learn about dietetics in health and disease. We have no course in the subject except for short demonstration in pediatric and medical clerkships. One of the highlights of the week was the demonstration of the purchasing and maintenance of textiles. Farm wives excell our professional sisters in such knowledge due to sound teaching in various types of extension work. As a result, most mail order catalogues now list the results of such tests. Another interesting experiment had to do with the amount of meat on a chicken versus a turkey. Even though the whole chicken cost less, the whole turkey provided more meat because of the relationship between frame and insides to the whole. By the use of cheap cuts of meat and other economies, excellent food is provided at the farm school campus dining room for fifty cents a day for each student. The control of waste even involves the use of the outer leaves of the lettuce which are chopped up after being thoroughly washed. The historical evolution of the tin can was traced to a point over 100 years ago, when it was first used. The outbreak of the European war found us with only a four-months' supply of tin. (It has now been increased to 8 months.) The possible use of cellulose materials for storing foods is considered. Private industry forges ahead in new food uses. Government research agencies and private research groups follow the lead (this is characteristic of our governmental picture). A special corn which is now a leading product of a Minnesota cannery was created by the research groups on order from the cannery who knew that such a food product would fill a demand. The same is true of the giant green pea. The wide-spread construction of the locker system of storing foods is developed well in advance of our knowledge of how best to prepare foods for storage. It has also been found that we have a tendency to overheat our canned foods. Chemists can now determine the actual temperature required for each batch. Tough foods may result from too much calcium in the water, changing tender, delicious corn to a product not unlike field corn. New methods of freezing foods are also de-

scribed (all aimed at obtaining the finest ice crystals). Strawberries are now frozen by dipping them in chilled syrup before putting them in the freezing unit. They are said to closely resemble the fresh variety. It will be possible in the near future to secure the types of food we need at any time of year. The League of Nations, which has become interested in the question of nutrition finds the United States way in advance of other nations, but with still a great deal of progress to be made. Farmers, during seasons when cash crops returned sufficient money to buy food, got out of the habit of growing kitchen gardens. It has been estimated in this area that farmers could raise 8 $\frac{1}{2}$ % of their food needs. Only 45% is being raised. The rural women of Minnesota are organized under group leaders who themselves are not nutritionists but who receive their instructions in groups from dietitians. At the present time, every county in the United States is represented by some type of educational work in nutrition. Studies made of families on relief show that good food is the most important single factor in promoting child health. Discouraging are the reports which indicate that many families with sufficient funds to purchase proper foods for their tables do not obtain good nutrition because of ignorance, indifference, or antagonism. Women are slowly losing their leadership in bad food habits, and their place is being taken by men. The stubbornness of the male as to change in food habits is most noticeable in all reports. In two Ohio counties children are taught to care for rat colonies. The rats are put on experimental diets. The first group received the same lunch that the children brought to school -- white bread jelly sandwiches, cookies and coffee; in three other groups the diet was gradually increased to normal. As the children were from rural areas, breeding experiments were conducted. The result was that the children saw first hand the result of their own dietary habits on several generations of rats. At the close of school, the parents came to see the judging of these animals which was carried on in typical agricultural ring fashion. They were judged for size and development, coat, and special points.

The animals were then sacrificed, a number kept for a permanent exhibit, and the others retained for the biology group of the next year to dissect and weigh the skeletons. The allergic child received a great deal of attention. In a city of two million population, 282 diabetic children were found, but 14,000 allergic children were also located. The psychological factors involved in under and over nutrition are stressed. It is interesting to note that few real endocrine cases are observed by comparison. A thorough study of "appetite" is in line whenever under or overweight patients present themselves. Energy expenditure is probably more important in the underweights. In the case of one well-known person, who has an aversion toward eating too much, he was able to regain seven pounds of weight by resting and not by increasing food intake. Dietary factors in anemia are of special interest involving as they do an attempt to secure for babies and women a more satisfactory source of iron in their diets. Physicians are urged to revert to the custom of their medical forefathers in looking at the tongue; now to estimate the possible presence of deficiency disease. The skin you do not love to touch, hot burning feet, and poor circulation are all to be reconsidered in this new dietary light. One is of the opinion that dietitians are well-trained in basic knowledge of foods and practical applications. Medical students and physicians alike could profit by a better understanding of these subjects. Many diseases poorly understood in the past are of dietary origin. The public and much of the medical profession have undoubtedly gone off half cocked on the vitamin question. It is estimated that over one hundred million dollars a year is now being spent by the public for unnecessary products. If we were able to save this amount plus the unnecessary expenditure of fifty million dollars for cathartics we could go a long way toward providing good medical care for all. Oh, yes: desiccated food is ready for our next war!