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

Optic Neuropathies

Volume IX Friday, April 8, 1938 Number

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
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Volume IX | Friday, April 8, 1938 | Number 23

INDEX

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Financed by the Citizens Aid Society

William A. O'Brien

I. LAST WEEK

Date: April 1, 1938

Place: Recreation Room
Nurses' Hall

Time: 12:15 to 1:15

Program: Movie: "The Milk Parade"

Roentgen Therapy
K. W. Stenstrom
C. B. Nessa
H. H. Jensen
A. L. Abraham

Present: 115

Gertrude Gunn,
Record Librarian

II. MOVIE

Title: "A Night at the Movies"

A Robert Benchley Short

Released by: M-G-M

III. AUTHOR

EDWARD BURCH, was born in St. Paul, graduate of Princeton University, 1928, medicine at Johns Hopkins University, graduate of 1933. Internship 1933-34 and residency 1934-37, Johns Hopkins Hospital. Now associated with his father, Dr. Frank Burch, Professor and Head of the Department of Ophthalmology, University of Minnesota, in the practice of ophthalmology in St. Paul. He has the privilege of presenting the first staff conference from this service.

IV. NEXT TWO WEEKS

1. Next Friday, April 15th, being a legal holiday, there will be no staff meeting.

2. The following Friday, April 22nd, is the scheduled date for the regular meeting. It is planned to invite Dr. Mont Reed to address the staff on Thursday, April 21st. Dr. Reed will be here as guest of the St. Paul Surgical Society. Unless you hear to the contrary, the meeting will be held April 21st instead of April 22nd.

V. CITIZENS AID SOCIETY LECTURE

Will be held April 12th at 8:15 P.M. in Medical Science Amphitheatre. The subject will be "Ovarian Hormones in Relation to Cancer of Female Genitalia." The speaker will be Edgar Allen, Professor of Anatomy at Yale. This lecture, given each year, is a scientific treatise on some phase of malignancy. The Citizens Aid Society, consisting of Mrs. George Chase Christian, Franklin Crosby, W. P. Christian, and Charles Case, gave the money for the Cancer Institute, much of its equipment, the recent therapy apparatus, the funds to support this bulletin, and, in addition to many other things, a fund for a lectureship each year. We should indeed be grateful to them, as are the many patients whose lives have been spared as a result of their generosity and thoughtfulness. In this connection it is interesting to note the recent designation of our hospital as an approved training center for graduate students under the National Cancer Fund for Aid in the Diagnosis and Treatment of Malignancy.

VI. DESK MAN

CHARLIE HAYDEN called a father the other night to inform him that his wife had twins. Papa replied, "The deuce you say."

VII. OPTIC NEUROPATHIES - A Review

Edward Burch

The various disease entities which may give rise to pathologic alteration of the optic nerve are so protean that any discussion of the optic neuropathies embraces not only the field of ophthalmology but those of neurology, neuro-surgery, endocrinology, toxicology, internal medicine, otology and pediatrics in greater or lesser degree as well. Before passing to a discussion of the various pathologic conditions of the optic nerve, a brief summary of its anatomy is in order.

The optic or second cranial nerve is in actuality no nerve at all but a fibre tract connecting two portions of the brain. Arising in the chiasm it runs as a flattened band forwards and outwards to the optic foramen. As it approaches the globe it assumes a more oval shape. At a point halfway from the chiasm to the foramen it becomes invested with its pia-arachnoid sheath. As it passes through the foramen it acquires its dural covering. Its total average length is five centimeters and anatomists have divided it into intracranial, intracanalicular, intraorbital and intraocular portions. Within the cranial vault it lies upon the diaphragma sellae where it is in relation to the hypophysis. It then traverses the anterior portion of the cavernous sinus. Emerging from the optic foramen it enters the orbit where it courses for three centimeters before entering the eyeball. Within the orbit its medial aspect is in relation to the sphenoid and posterior ethmoid sinuses. At its point of entrance into the orbit it becomes surrounded by the cone of external ocular muscles and lies in close proximity to the third and sixth cranial nerves, the sympathetic and naso-ciliary nerves. The central retinal artery and vein enter the nerve from nasally and below at a point about ten to twelve millimeters behind the eye. They traverse the subarachnoid space for a short distance before passing into the fiber bundles. When the nerve pierces the globe, the fibers normally lose their myelin sheath. The intraocular division of the nerve passes through the outer two coats of the globe and appears as the papilla or nerve head where it joins the layer of nerve fibers of the retina.

The subarachnoid space of the brain is continuous with that of the optic nerve which terminates in a culdesac whose anterior portion does not extend beyond the lamina cribrosa.

The optic nerve itself consists of 800-1200 bundles of fibers separated by septa derived from the pia-arachnoid sheath. The bundles consist chiefly of visual fibers which are axones of the ganglion cells of the retina and whose synapses connect with those of the external geniculate body. In addition there are pupillary fibers having ultimate connections with the nucleus of the third nerve and retino-motor fibers. Whether these are also commissural fibers between the two retinae and trophic fibers in addition is not definitely known. The individual fibers have a myelin but not a neurilemmal sheath. Lack of the sheath of Schwann explains the inability of optic nerve fibers to regenerate. From the macular region passes an important group of fibers, the papillomacular bundle. This group, intimately concerned with central vision, constitutes about one-third the total number of fibers. At the nerve head they occupy a wedge-shaped sector on the temporal side. As they pass backwards they lie at first lateral and a trifle below the others, but later occupy a more central position. The vulnerability of this group of nerve fibers is a point of the utmost practical consideration.

The papilla receives its blood supply from the central artery of the retina. The optic nerve in most of its intraorbital portion is supplied by both the central retinal artery and ramifications from the posterior ciliary vessels. The portion of the nerve within the foramen and the intracranial segment are supplied by the ophthalmic, anterior cerebral, and internal carotid arteries.

Of the lesions affecting the optic nerve, papilloedema or choked disc is of considerable interest, not only because it reflects the state of intracranial pressure but also because of

the widespread speculation which has arisen as to the means of its production. Among the first to speculate upon the mechanism of papilloedema were Gowers and von Graefe. The former adhered to a toxic-inflammatory basis while the latter advanced the theory that it was brought about by pressure on the cavernous sinus. Parinaud held that choked disc was merely a continuation of cerebral edema. Another possible explanation, that of some nervous irritative influence on the retinal vessels, has also been propounded. These conjectures are chiefly of historic interest. At the present time it is generally believed that the cerebrospinal fluid, when under great pressure, interferes with the venous return by compressing the central retinal vein as it crosses the intervaginal space. This view is held by Paton, Holmes, and Lauber among others. During the past year, however, Jeffers, Griffith, Fry and Fewell, in a series of interesting experiments upon rats, have been able to demonstrate that papilloedema may be due to an increased flow of fluid from the subarachnoid space to the eye rather than to a blockage of the venous return. It is quite possible, however, that all cases of choked disc are not produced by the same mechanism.

As the optic nerve enters the globe, its trunk fits tightly in the scleral ring which is a tough, unyielding structure. If the volume of fluid in the optic nerve sheath is increased the nerve is unable to expand laterally and must do so anteriorly in an axial direction. This causes a "mushrooming" forward of the papilla.

In the early stage of papilloedema there is a blurring of the neuroretinal margins, which is most marked at the upper and lower poles of the disc. The papilla is usually grayish red. The veins are engorged and slightly tortuous and there is often an edema of the entire retina. As the condition progresses the whole disc becomes swollen and elevated. The neuroretinal border is blurred at the entire periphery, the physiologic cupping of the nerve-head is obliterated and the papilla assumes a more translucent appearance. The veins become tremendously swollen, appear much darker and make a sharp bend over the margin of the disc. Finally, hemorrhages and exudates appear in the region of the disc, later spreading toward the periphery of the retina which, by this stage, has become markedly edematous. Newly formed vessels may appear on the papilla. In about one-fifth of the cases there will sooner or later appear a more or less complete star-shaped macular figure.

The subjective symptoms of papilloedema are surprisingly few. Some patients complain of transient blurring of vision. Occasionally there are spells of almost complete amaurosis lasting a few moments. Vision is usually well preserved until the later stages. The visual field changes consist of an enlargement of the blind spot with a slight contraction of the peripheral field.

Choked disc must be differentiated from optic neuritis and pseudo-neuritis. To attempt a differentiation from the ophthalmoscopic picture alone is attended with the utmost difficulty in most cases. While visual acuity is but slightly impaired in papilloedema, there is usually a marked loss of central vision in optic neuritis because of the presence of a central scotoma. It is therefore incumbent upon the clinician to make careful studies of the visual acuity, central and peripheral fields. Light adaptation is unimpaired in choked disc but there is usually some impairment in optic neuritis. In optic neuritis there is commonly pain on motion of the globe; this is not the case in papilloedema. There tends to be less elevation of the nerve head in optic neuritis than in choked disc but the physiologic cupping disappears at an earlier stage in optic neuritis. In pseudoneuritis, a congenital condition, the disc appears abnormally red, the margins are hazy, the vessels may be overfilled and a high degree of hyperopia or astigmatism is present. There are never exudates or hemorrhages, there is no impairment of vision, no visual field changes and the condition remains static. One finds the binocular ophthalmoscope of considerable assistance in studying the finer details of

changes in and about the nerve head.

Having established a diagnosis of choked disc, it becomes necessary to determine its etiology. By far the most frequent cause of papilloedema is a space-occupying lesion within the intracranial vault. Such a lesion may be a tumor of the brain, either primary or metastatic, abscess, gumma, tubercle or aneurysm. By far the most frequent single cause of choking is tumor. It is said to be the cause of four-fifths of the cases of choked disc. Except in tumor of the frontal lobe, the papilloedema is usually bilateral, and it tends to occur earlier and to be more pronounced in tumors of the posterior fossa.

Inflammatory lesions are responsible for some cases of papilloedema. Among the more common may be mentioned epidemic encephalitis, tuberculous, gummatous, and purulent forms of meningitis. In these diseases an inflammatory exudate or membrane, which forms about the openings of the fourth ventricle, blocks the escape of cerebrospinal fluid into the subarachnoid space. An internal hydrocephalus with increased intracranial pressure and papilloedema results. Another type of meningitis, benign lymphocytic, which is believed to be a virus disease and which closely simulates tuberculous meningitis with respect to the spinal fluid findings, has recently received attention as a causal agent in choked disc.

A cause of choked disc, which has received scant attention until the last few years and yet which is of extreme importance to recognize clinically, is serous meningitis. It is a misnomer to speak of this entity as an inflammation in the light of extensive and conclusive investigation by Woodhall. He has demonstrated, after a study of two hundred dural preparations, that there is a tendency for the superior longitudinal sinus to empty into the right lateral sinus and for the straight sinus to empty into the left lateral sinus. In such an event a torcular sinus is lacking and there is no adequate communication between the two lateral sinuses. Therefore occlusion of one lateral sinus results in obstruction of the venous return from either the superior longitudinal or the straight sinus. He has found that there is frequently a great discrepancy in the size of the two lateral sinuses and that in some instances one is lacking. It is therefore patent that obstruction of the larger of the two sinuses will result in cerebral edema with intracranial hypertension and papilloedema. This is the situation which obtains in many of the cases of papilloedema of obscure etiology which are spoken of as pseudotumor cerebri. These cases show papilloedema as a result of the increase in intracranial pressure. There are no localizing neurological signs and the spinal fluid is normal except for the increased pressure. Many of these cases are unrecognized clinically. The diagnosis is facilitated if a Queckenstedt test is made.

Choked disc is also caused by purely orbital conditions such as tumors, progressive exophthalmos of Naffziger which occurs in association with hyperthyroidism, lead poisoning, skull injury, deformity of the cranium such as oxycephaly and has been reported as the sequela of allergic states which bring about cerebral edema with marked intracranial hypertension. It also occurs as a complication in about one-third of all cases of cavernous sinus thrombosis.

The treatment of papilloedema consists chiefly of removal of the primary cause. Since brain tumor is by far the most frequent cause, cases usually fall into the hands of the brain surgeon. In inflammation and toxic cases and those due to obstruction of the venous return from the brain, such as serous meningitis, it is imperative to ward off secondary atrophy by either subtemporal decompression or repeated lumbar puncture. If a tumor of the brain is suspected, particularly a subtentorial tumor, lumbar puncture should never be attempted, for the sudden release of pressure may cause a herniation of the vital structures in the medulla into the foramen magnum with resultant respiratory failure and death.

The histopathological findings in

choked disc consist of distention of the optic nerve sheath, degeneration and finally gliosis of the nerve fibers, thickening of the vessel walls with round celled infiltration, bowing forward of the lamina cribrosa and edema of the papilla, especially nasally where the fiber bundles are much less compact.

The second large group of optic neuropathies for consideration are those ascribable to inflammatory and toxic lesions of the nerve itself. Most unfortunately there exists great confusion regarding the terminology of this class of lesions. The British have long been accustomed to speak of swelling of the nerve head due to increased intracranial pressure as optic neuritis. In this country we employ the term papilloedema or choked disc. This confusion is increased by the use of the term papillitis, which has also been used interchangeably with both papilloedema and optic neuritis by some authors. It would seem more rational to reserve the term optic neuritis for those conditions commonly associated with inflammatory and toxic states of the nerve and to further divide the optic neuritis when there are clinical signs of inflammation at the disc as evidenced by discoloration, swelling, loss of the physiologic cup, and obscuration of the neuroretinal margins into the axial type with involvement of the papillomacular bundle and the periaxial type without involvement of the papillomacular bundle.

In the axial type there may or may not be swelling of the disc, depending upon the proximity of the lesion to the papilla. If swelling is not present, but a central field defect is found, the condition is usually termed retro-bulbar neuritis. Optic neuritis is further classified as acute or chronic.

By far the majority of acute types of the disease are due to some demyelinizing process of the nerve. It is held by some neuropathologists that all demyelinizing states are really one and the same disease. However, four main groups are recognized clinically. The first, most important and commonest, is disseminated sclerosis. The second is neuromyelitis optica or Devic's disease where the optic nerve lesions are associated with transverse myelitis, the spinal cord lesions often appearing at a later date. The third is encephalitis periaxialis diffusa or Schilder's disease. In this disease one encounters progressive cortical blindness and deafness with marked spasticity, death finally supervening. Acute disseminated encephalitis following vaccinia and chicken-pox also displays an affinity for the optic nerve and demyelinization similar to that seen in disseminated sclerosis is present.

Optic neuritis as an initial monosymptomatic manifestation of multiple sclerosis deserves special mention. It should be borne in mind that optic neuritis either with or without swelling of the nerve head may be the first indication of disseminated sclerosis. Whereas formerly the para-nasal simuses were incriminated as the etiological factor in numerous cases, evidence continues to accumulate that many patients who have been subjected to intranasal surgery have later developed classical signs of multiple sclerosis. Indeed, it seems not overbold to state that in the majority of cases of acute optic neuritis in a patient between the ages of fifteen and fifty some form of demyelinizing disease will be found to be the responsible agent. Various British neurologists have stated that seventy-five per cent of patients in the fifteen to fifty age group suffering from optic neuritis are afflicted with disseminated sclerosis. Such a percentage is undoubtedly too high, but analysis of the etiological agents in optic neuritis as carried out by J. Moore offers convincing proof that demyelinizing disease must be accorded first place by a wide margin in the incidence of optic neuritis.

The periaxial form of optic neuritis without central scotoma occurs chiefly in basilar meningitis. The process extends down the intervaginal space enveloping the nerve and giving rise to a clinical picture from the ophthalmological standpoint very similar to papilloedema. The swelling tends to be less marked, however, and the spinal fluid is not under increased pressure. It is most frequently en-

countered in epidemic meningococcus meningitis.

Optic neuritis with central scotoma has been reported in connection with febrile conditions such as herpes ophthalmicus, influenza, pneumonia and septicemia. This is uncommon, however.

Syphilis may give rise to a true optic neuritis with papillomacular bundle involvement and a complement-fixation test is desirable in all cases of optic neuritis. The optic nerve involvement occurs in the late secondary stage of the disease, is characterized by its fulminating course and rapid deterioration of vision despite active treatment. Optic neuritis may also occur as a neuro-recidive phenomenon in inadequately treated patients or in individuals who allow their therapy to lapse, particularly during the secondary stage of the disease.

There are probably some cases of optic neuritis which are attributable to disease of the paranasal sinuses. It should be remembered, however, that remission of the optic neuritis following intranasal procedures does not constitute proof of the etiological relationship, for optic neuritis is notoriously subject to spontaneous remissions, and at a later date neurological examination may reveal absent abdominal reflexes, nystagmus, scanning speech and other classical signs of disseminated sclerosis.

Although foci of infection in the teeth, tonsils and middle ear have been cited as possible causes of optic neuritis, there is very little evidence to show that such foci have any connection with the optic nerve condition.

Local orbital conditions such as abscess, cellulitis, and periostitis may rarely cause optic neuritis. Within the past two years the radiologists have called attention to an obscure osseous pathological state affecting the internal table of the skull in the frontal region known as hyperostosis frontalis interna. The x-ray picture is quite characteristic. A few reports of this condition as a cause of optic neuritis are on record.

Pituitary tumor, in the early stages, may give rise to a retrobulbar neuritis with central scotoma. Although as the tumor enlarges the characteristic bitemporal field defect becomes manifest, the possibility of pituitary tumor should be borne in mind.

In adolescent females at the time of the menarche there occasionally develops a retrobulbar neuritis. It may also come on during lactation. It is believed that these forms of the disease are in some way associated with swelling of the pituitary gland.

In a recent number of the Archives of Ophthalmology, Clay and Baird reported a series of unclassified cases of optic neuritis which they felt might be due to a virus affection of the nerve. Various Chinese authors have also reported numerous cases, occurring chiefly in young males in the northern Chinese provinces, in which none of the usual etiological factors could be demonstrated, although vitamin deficiency has been postulated as the cause.

The chronic forms of optic neuritis are chiefly due to specific toxins. Of these alcohol and tobacco are the chief offenders. As a rule, the patients are over fifty and diabetics seem unusually predisposed. Occasionally quinine, various arsenic compounds, salicylic acid in its various forms, filix mas, optochin, thallium and trichlorethylene may cause toxic amblyopia. In this connection it is interesting to note the recent report in the J.A.M.A. of a case of optic neuritis occurring during the administration of sulfanilamide.

Diabetes may also very rarely cause retrobulbar neuritis. One of the most interesting types of the chronic form of the disease is Leber's hereditary optic atrophy. Although it is generally said to be transmitted through the female who escapes the disease there are a few well-authenticated cases of the condition occurring in women. The familial incidence nearly always establishes the diagnosis.

The diagnosis of optic neuritis

rests upon the appearance of the nerve head, and particularly upon a careful study of the central field of vision. In axial neuritis there may or may not be swelling of the disc, but to establish the diagnosis one must demonstrate a defect in the central field of vision, either relative or absolute. The amount of visual loss will depend upon the size, position and density of the central scotoma. In periaxial or perineural optic neuritis the central field is normal and there may or may not be a concentric contraction of the peripheral field. The diagnosis is made in cases of basilar meningitis where the process has given rise to swelling of the optic nerve head while the cerebrospinal fluid pressure remains normal.

Any evaluation of therapeutic procedures in optic neuritis is difficult because of the fact that many forms of the disease are subject to spontaneous remissions. For many years strychnine and pilocarpine sweats were regarded as the most efficacious form of treatment. When sinus disease was regarded as one of the chief causes, countless patients were subjected to intranasal surgery. Recently the use of intravenous triple typhoid vaccine has been widely advocated, particularly in multiple sclerosis. Quinine by mouth has also gained some advocacy in the therapy of disseminated sclerosis.

In recent months, Carroll of the Institute of Ophthalmology at the Presbyterian Hospital, New York City, has claimed phenomenal improvement in tobacco-alcohol amblyopia by the use of a well balanced diet supplemented by concentrated vitamins and liver extract. During the period of treatment patients were permitted to continue the use of alcohol and tobacco without restriction. The vitamin concentrates employed were brewer's yeast, wheat germ and cod liver oil. Carroll made a careful study of eight patients and felt that the improvement in vision was too prompt and too striking to be attributable to any cause but the dietary and vitamin-liver regime.

Induced hyperpyrexia with the vapotherm, electric cabinet or short-wave apparatus has not to date proved markedly successful in optic neuritis.

Several years ago the various vasodilator drugs began to be employed intravenously in the treatment of retrobulbar neuritis, particularly the toxic forms of the disease. Of this class of drug the nitrites and acetyl-choline were recommended and Duggan reported prompt and rather complete recoveries in his series of cases.

Recently a powerful acetyl-choline derivative, mecholyl, has been prepared by Merck. This drug is administered by mouth and, although it has not been used over a long period of time, the results to date are encouraging.

Mecholyl should not be employed in the face of cardiovascular disease. When given orally its range of safety is considerably greater than when given intramuscularly. It is a powerful vasodilator, it lowers the intraocular tension, brings about pupillary contraction, causes marked generalized sweating and increases the tonus of the gastrointestinal tract. Its effect is greatly enhanced if the drug is given in combination with prostigmine, but as yet the full possibilities of this method of therapy have not been sufficiently standardized to permit recommendation of their combined use.

The optic atrophies constitute the third large group of optic nerve conditions which are to be discussed. If one omits the atrophy secondary to glaucoma and the ascending type consequent upon purely retinal lesions there remain for consideration the primary and secondary forms.

In primary or simple optic atrophy, whatever the cause, the appearance of the nerve head presents a well known picture. The color of the disc varies from extreme pallor to waxy yellow. The margins of the papilla are clear cut, there is an atrophic cupping due to atrophy of nerve fibers which permits the observer to view the interdigitations of the lamina cribrosa, the so-called "stippling" of the nerve head. The retinal arteries are narrowed in the later stages. Two types of secondary atrophy are recognized: first, consecutive atrophy following

papillomacular bundle disease where there is temporal pallor of the disc with atrophic excavation chiefly confined to the temporal side; and second, the atrophy secondary to papilloedema or inflammation of long standing. Here the disc assumes a dirty gray or whitish hue. Fibrous tissue proliferation causes a filling in of the physiologic cup, the neuroretinal borders are obscured and poorly defined, the blood vessels are contracted and often accompanied by perivascular sheaths of glial scar tissue for some distance as they leave the papilla.

In the primary forms of atrophy the peripheral fields show contraction first for color, particularly red, and finally for form. The vision diminishes rather gradually as a rule and there may be astonishing disparity between the appearance of the disc and the amount of visual acuity.

In consecutive optic atrophy there exists a large central defect with loss of central vision and the peripheral fields may also be constricted. In atrophy following papilloedema the fields are markedly contracted for form and color, if indeed any vision remains.

Primary or simple atrophy may be due to various causes, of which tertiary neurosyphilis is the most common etiological agent. It occurs with much greater frequency in tabes with dementia paralytica. Tumors in the region of the optic chiasm, chiefly of the hypophysis, are also responsible for a fair percentage of cases. In these instances the visual field defect characteristically consists of bitemporal hemianopsia although in the earliest stages a central scotoma may be present to confuse the clinical picture. X-ray studies of the sella turcica are usually helpful in establishing the diagnosis. Occasionally one encounters the so-called Foster Kennedy syndrome which gives rise to primary optic atrophy on one side with papilloedema on the other side together with anosmia indicating an olfactory groove neoplasm of the frontal lobe. Direct pressure on one optic nerve causes the primary atrophy and this serves to indicate the position of the tumor. The pressure of sclerotic internal carotid arteries on the nerves at the chiasm may cause atrophy with a bi-nasal field defect. This condition is exceedingly uncommon.

Breaks in the line of conduction due to trauma such as skull fracture and gunshot wounds give rise to simple atrophy. After a complete severance of one nerve, several weeks will elapse before nerve head changes become manifest, although there is total and immediate loss of vision in the affected eye. Hemorrhage in the nerve sheath due to blows on the head must also be mentioned as a possible cause of atrophy.

In young children one occasionally sees pale discs associated with marked loss of vision for which there appears no assignable cause. Occasionally this condition is associated with a spastic diplegia. These cases have a congenital basis although the exact pathogenesis is not clear.

Of the consecutive forms of atrophy with temporal pallor, by far the majority are due to multiple sclerosis of some form of toxic amblyopia. The postneuritic type usually follows the choked disc of brain tumor.

There is one special form of optic atrophy which deserves careful consideration. This is the form due to the administration of tryparsamide. It may come on in particularly susceptible patients after the injection of even one or two doses. The patients often complain of visual sensations which they liken to "heat waves" before the eye. Visual loss may be quite rapid. The peculiar and characteristic contraction of the field of vision is almost invariably present. This consists of a loss of the upper and lower fields for form and color with good preservation of the nasal and temporal fields giving rise to a bizarre "oblong" shaped field.

Optic atrophy of the simple type may also occur as a result of poor nutrition of the nerve. Arteriosclerositic atrophy is perhaps more common in elderly patients than one suspects. Fundus examination will reveal marked

sclerosis of the senile decrescent type with a pale nerve head. Occlusion of the central retinal artery produces optic atrophy. The occlusion may be due to embolism or endarteritis of the vessel. This loss of vision is sudden. At first there is marked retinal ischaemia with a cherry-red spot at the fovea. Later the nerve becomes extremely pale and the retinal vessels become filiform in appearance. Pernicious anemia may give rise to a nutritional atrophy of the optic nerve and should be regarded as a potential cause in cases where the cause is not obvious. Often there are associated retinal hemorrhages sometimes with pale centers, the so-called "Roth spots." As a rule there are also indications of combined sclerosis of the posterior and lateral columns of the spinal cord. The blood picture of course serves to establish the diagnosis.

Exsanguination may bring about a marked pallor of the nerve head with visual loss, and for some reason which is not well understood severe gastric and uterine hemorrhage predispose to this form of atrophy more frequently than blood loss from other sources.

The therapy of optic atrophy obviously depends upon the cause. Omitting discussion of the treatment of forms such as those due to pressure where surgical intervention is clearly demanded brings up the question of the treatment of luetic atrophy.

The two principal methods of available treatment which have yielded worthwhile results in optic atrophy due to syphilis are the Swift-Ellis treatment consisting of the intraspinal injection of arsphenaminized serum and induced by hyperpyrexia. Both methods are attended by some risk to the patient. For the production of fever, malaria inoculata, intravenous typhoid vaccine, the electric cabinet, the vapotherm and the short-wave apparatus have all been employed. It is claimed by J. Earle Moore that malaria inoculata is the most effective therapeutic agent in atrophy of the optic nerves. Moore also maintains that malaria is of greater benefit to the patient from the standpoint of the spinal cord and cerebral manifestations of the disease.

During the past year or so the retrobulbar injections of atropine sulphate have been recommended in the treatment of both luetic and arteriosclerotic atrophy. Cordes reports a single case in which there was great improvement in the latter type of atrophy, but certainly this method of treatment in luetic cases has not proved worthy of further trial.

Recently Lauber and Sobanski have advocated the use of miotics and even fistulizing operations on the globe to combat luetic atrophy. The rationale for this procedure consists in their contention that the majority of tabetics suffer from hypotension, that the arterial pressure within the eye is therefore relatively too low in relation to the normal intraocular tension and that the optic nerve consequently is deprived of adequate nutrition. Although striking results have been reported from the Warsaw Clinic, this method of therapy has not gained widespread favor in this country. The French have likewise advanced the hypothesis that there is insufficient blood supply to the optic nerve in tabetic atrophy. They, however, believe the nutrition can best be restored by periarterial cervical sympathectomy. Leriche has been the chief protagonist of this school of thought.

The more conservative method of producing a better blood supply to the nerve through the use of vaso-dilators has been advocated. For this purpose intravenous nitrites, acetyl-choline and most recently, mecholyl, have been employed. To date an insufficient number of cases have been treated to permit evaluation of this form of therapy, but the method is deemed worthy of further clinical investigation.