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

Arachnoiditis

Volume XIII Friday, December 5, 1941 Number 9

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

Volume XIII | Friday, December 5, 1941 | Number 9

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Published for the General Staff Meeting each week
during the school year, October to June, inclusive.

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Alumni and Friends.

William A. O'Brien, M.D.

LAST WEEK

Date: November 21, 1941

Place: Recreation Room
Powell Hall

Time: 12:15 to 1:10 P.M.

Program: "Body Section Roentgenography"

Discussion
L. G. Rigler
Kenneth Olson

Present: 133

Gertrude Gunn,
Record Librarian

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Date: November 28, 1941

Place: Recreation Room
Powell Hall

Time: 12:15 to 1:05 P.M.

Program: "Highlights of the Football Season"

Present: 165

Gertrude Gunn,
Record Librarian

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II. MEETINGS

1. SEMINAR IN PATHOLOGY

December 8, 1941, Monday, 12:30, Room 104, Institute of Anatomy. Bronchial Adenoma - J. B. Arcy.

2. SEMINAR IN PHYSIOLOGY

December 11, 1941, Thursday, 8:00 P.M., Room 116, Millard Hall. Subject: Denaturation of Proteins Including Surface Denaturation - H. B. Bull, Northwestern University.

3. DIVISION OF DERMATOLOGY

Fourth Annual Meeting, American Academy of Dermatology and Syphilology, at the Waldorf Hotel, New York City, December 14-18, 1941.

The Use of Hormones in Dermatology - Francis W. Lynch
Tuberculosis, Tuberculids and Sarcoids - Henry E. Michelson
Dermatoscleroses - Paul A. O'Leary
Dermatopathology - Hamilton Montgomery
Diagnosis and Treatment of Lichen Planus Carl W. Laymon
Practical Use of Vitamins in Dermatology and Syphilology - Paul A. O'Leary
The Role of Allergy in Tuberculosis of the Skin - - Henry E. Michelson and others.

4. CENTER FOR CONTINUATION STUDY

Diseases of Infancy and Childhood - December 15-20, 1941.

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III. EVENTS

Basketball

Milliken University vs. University of Minnesota, Field House, December 6, 8:00 P.M.

North Dakota State vs. University of Minnesota, Field House, December 13, 8:00 P.M.

Creighton University vs. University of Minnesota, Field House, December 20, 8:00 P.M.

Hockey

London A.C. vs. University of Minnesota, Minneapolis Arena, Dec. 11-12, 8:30 P.M.

Christmas Vacation

(Academic) - December 19 to Jan. 4 inclusive. No staff meetings or bulletin published during this period.

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IV. OFFICES

CLYDE M. CABOT, M.D.

Announces the opening of his Offices at
948 Medical Arts Building
Minneapolis, Minnesota
Ophthalmology and Otolaryngology
Bronchoscopy and Oral Surgery

BRidgeport 6566.

ARACHNOIDITIS (DIFFUSE PROLIFERATIVE LEPTOMENINGITIS)

Alex Blumstein
A. B. Baker

Introduction

Chronic or subacute low grade leptomeningitis, cystic and adhesive in nature, is a clinicopathologic condition known by a number of names in medical literature (arachnoiditis, serous meningitis, meningitis serosa circumscripta vel cystica, arachnoiditis adhesiva circumscripta and diffusa). For the sake of simplicity and because of widely accepted usage in this country, the term arachnoiditis is used in this title though it is obviously inaccurate.

The cystic structures are not cysts in the true sense of the word. They are in reality collections of cerebrospinal fluid walled off by meningeal adhesions. The fluid within the cysts is often under great tension. The leptomeningitis may be predominantly cystic or predominantly adhesive. The extent of the arachnoiditis varies from a well localized lesion to a diffuse process involving the entire cerebrospinal axis. There is, however, a definite tendency for the process to be either cerebral or spinal. Arachnoiditis may be primary or it may be secondary to pathologic changes in adjacent structures.

In some instances the etiology can be ascertained. This is especially true in the localized (circumscribed) form where trauma has been shown to play an important role. The clinical picture, the course, and the response to treatment is extremely variable, depending on the location, the extent and the cause of the process.

Cerebral Type

The cerebral type of arachnoiditis is usually of a focal type but rarely it may be diffuse. Demel collected reports of 40 cases of the localized type. The clinical picture was that of brain tumor. In 16 cases the lesion was in the posterior fossa. Recovery followed in approximately 90 percent of cases operated on. Horrax reported 33 cases simulating cerebellar tumor; 28 were followed from 1 to 9 years; all showed improvement or complete relief from symptoms. Lillie reported 3 cases of prechiasmal syndrome produced by arachnoiditis. He indicated the possibility of preoperative diagnosis and the favorable results frm proper surgical treatment.

Spinal Type

For purposes of discussion, spinal arachnoiditis may be divided into the localized and the disseminated types. Generally, writers on the subject of spinal arachnoiditis group all the cases together regardless of the extent of the process. This gives an exceedingly varied and complex clinical picture. The tendency to put all cases into a single group is in part due to the clinical difficulty of differentiating the 2 types in some cases. However, as Stookey points out, the subject would be less confusing if a real effort were made to place the disseminated and localized types in separate categories for the purpose of analysis. In the following discussion an effort will be made to distinguish between the circumscribed and the disseminated types. If that is not possible, the term arachnoiditis will not be qualified.

Age and Sex

Elkington reported 41 cases of arachnoiditis collected at National Hospital, Queens Square. There were 30 males and 11 females. The youngest case was 23 years of age; the oldest 65. The average age of the patients when first seen was 45 years. The age grouping was as follows:

Under 20	-	0 cases
20-30	-	6 "
30-40	-	6 "
40-50	-	14 "
50-60	-	10 "
60-70	-	5 "

One of the cases reported by us is that of a 17-month-old boy. Elkington refers to the case of an 8-year-old boy reported

Heller. In Elsberg's 38 cases, men and women were equally affected.

Causes

As to causes, Elkington's 41 cases are divided in the following manner:

9 cases	-	Injury. The interval between injury and symptoms varied from 3 weeks to 24 years. The severity of the injury was inconstant.
4 "	-	Syphilis
2 "	-	Meningococcic meningitis
4 "	-	Gonorrhea
16 "	-	"Systemic" infection
18 "	-	No known cause.

Ten of 12 cases of spinal arachnoiditis reported by Stookey had had typhoid fever, influenza, encephalitis, or meningitis. Trauma as a forerunner of arachnoiditis (localized?) is reported particularly by Mauss and Krüger. In 54 laminectomies in cases of individuals who suffered war wounds, they found arachnoiditis 23 times. In 14 of the 23 cases there was direct trauma of vertebrae. In 9 cases there was no direct vertebral trauma. The authors attributed these cases to concussion associated with momentary dislocations of the vertebral bodies. They reported good results from operative intervention. As to causes of the disseminated type, Selinsky reported that 5 of 8 vases had had pneumonic or pleuritic infections. Mackay reported 5 cases of the disseminated type; 3 seemed to result from some form of acute meningitis - one 5 weeks, one 17 years, and one 5 years previous to the arachnoiditis. In 2 cases there was no known cause. Barker and Ford reported a case of the disseminated type in which arachnoiditis set in during convalescence from lymphocytic choriomengitis. Laboratory studies revealed the virus.

Clinical Picture

There is a gradual onset without fever. Invariably the disease assumes a subacute or chronic course. In Stookey's 12 cases of arachnoiditis, 6 had symptoms for more than 7 years. In Elsberg's series, there was a history of symptoms from 1 to more than 9 years. The disseminated type produces a syndrome of multiple involvement of the spinal nerve rootlets, both anterior and posterior, but predominantly posterior. PAIN IS THE MOST DISTINCTIVE SYMPTOM. It usually commences over one or more spinal segments and later becomes bilateral and spreads over a wide cutaneous area. The pain often has a burning, constricting quality and is frequently influenced by posture, movement of the spine, straining and coughing. The pain may occur over widely separated areas and may be accompanied by hyperesthesia or diminished sensation. The objective sensory disturbances are often vague, bizarre or inconstant in distribution. Diminished reflexes and muscular atrophy which can best be explained on the basis of anterior rootlet involvement are not infrequent. If the cauda equina is involved there is atrophy and hypotonia of the legs. Bladder and rectal disturbances come late in the disease; as a rule there is urgency long before incontinence sets in.

There may be slight or, in later cases, considerable involvement of the spinal cord. A cyst may produce signs and symptoms of compression like a true neoplasm. If the cyst is part of a disseminated process, there will be signs simulating extramedullary tumor in addition to the features of multiple rootlet involvement. This will lead to slowly progressive weakness and spasticity of the extremities, impaired sensibility, exaggeration of tendon reflexes, pathologic plantar responses, and impairment of sphincters. Constriction of the spinal cord by adhesions may cause a similar picture.

Spinal Fluid

In 50 per cent of Elsberg's cases, cells and total protein were well within normal limits. Stookey did manometric studies in 10 cases; 5 had almost complete block and 5 showed partial block. Even in some cases of subarachnoid block, the protein content of the fluid was normal and xanthochromia was absent. Increase in cells is rare in arachnoiditis. In Elkington's series, cytology was done in 26 cases with normal results in

2. In one case there were 50 and 42 lymphocytes per cu. mm. on 2 separate occasions.

X-ray Studies

In Selinsky's 8 cases of disseminated arachnoiditis all showed scattered arrest of iodized oil after cisternal injection. The arrest of lipiodol at multiple levels is pointed out by a number of writers. Schwartz and Deery describe the small linear shadows due to droplets of oil spread over portions of the meninges. Scattered droplets of oil in the subarachnoid space generally retain a globular shape. However, when the oil lodges against thickened dentate ligaments and subarachnoid fibers, small linear horizontally placed shadows are produced on the film.

Diagnosis

Robertson reported 5 cases of arachnoiditis. He made the preoperative diagnosis in 3 of the cases, using the following points:

1. Extensive area affected by the pain which was of burning quality and characterized by periods of remission.

2. The intimate relation of pain to posture (such as raising the arm).

3. Evidence of dissemination of the process.

4. Evidence of involvement of motor roots (atrophy and diminished reflexes).

The pain is intense and is frequently associated with hyperesthesia. In two of our cases the pain was intractable and demoralizing.

The duration of the symptoms is usually much longer in arachnoiditis than in cord tumor. The initial pain frequently extends over a larger area than in cord tumor (for example, an entire extremity). The distribution of the pain is difficult to reconcile with a single compressing lesion. Anesthesia is generally not so pronounced as in spinal tumor, unless there is marked constriction. A history of previous subarachnoid infection is helpful in the diagnosis of arachnoiditis.

The arrest of iodized oil at multiple levels is a most important diagnostic aid. Subarachnoid block without xanthochromia or increased protein is a highly suggestive corroborative finding in arachnoiditis.

The onset of arachnoiditis is much slower than in encephalo-myelo-radiculitis. In the latter there is rarely subarachnoid block and the course is very much more benign. In encephalo-myelo-radiculitis there is, as a rule, absence of muscle atrophy and fairly complete recovery despite the severity and extensiveness of the nervous system involvement. In arachnoiditis there is rarely cell-protein dissociation.

Evidence of upper and lower motor neuron involvement in arachnoiditis may lead to a mistaken diagnosis of amyotrophic lateral sclerosis. This occurred in one of our cases and in a case reported by Robertson. However, pain is rarely a prominent part of amyotrophic lateral sclerosis and subarachnoid block practically never occurs.

Treatment

Elsberg states: "Whatever may be the cause for the leptomeningeal adhesions, it can not be denied that the adhesions may disturb the functions of the spinal cord and nerve roots by direct pressure, and by interference with the vascular supply of the cord aggravate a pre-existing intramedullary lesion. Therefore, in the present state of knowledge, exploratory laminectomy is generally indicated."

"The adhesions are most often found on the posterior and lateral aspects of the spinal cord and especial attention must be paid to the emerging nerve roots, and any bands constricting them must be divided."

In referring to x-ray therapy for disseminated arachnoiditis, Selinsky states:

> "In my experience, no other non-surgical therapeutic measure has exerted such a favorable influence. The spine is crossfired with high voltage radiation at the various levels indicated by the sensory disturbance. One or more series of treatments are given, depending on the response to therapy. If indicated, the series of treatments is repeated at intervals of six weeks. Fractional treatments are given at each sitting and consist of from 100 to 150 roentgens (in air) until a total of 800 roentgens is administered."

In one patient 3 courses of treatment were given.

Prognosis and Results

Operation was done in Mackay's 5 cases of the disseminated type; 2 died, 2 were unimproved, and 1 was clinically benefitted. According to Selinski:

> "High voltage roentgen therapy exerts a definitely ameliorating effect which is variable in duration. Recurrences of the pain may be relieved by a repetition of the treatment."

He reported "good results" in 4 of 8 cases.

From a pathologic study of our cases of disseminated arachnoiditis, the outlook for therapeutic improvement from either x-ray or surgical intervention after the process has been long established, does not appear at all promising. In our 4 cases the cord damage appeared to be due to vascular involvement with associated tissue destruction rather than compression of the nervous tissue by the thickened meninges.

Elsberg states, "The outlook after operation will depend to a considerable extent upon the intramedullary changes that have occurred. If the adhesive process is well localized and the symptoms have not been of more than 1 or 2 years' duration, the patient may be relieved of all or almost all disturbances by the operation." "If the adhesive process extends over a great part of the spinal leptomeninges, the outlook for improvement is small. However, some of these patients may be improved if by good fortune or good judgment, a part of the cord which has been most compromised by the adhesive process has been exposed and adhesions which compressed the cord have been divided or the contents of a cyst evacuated." Elsberg analyzed 38 cases from his personal experience. In more than one-half the patients, little or no improvement followed the surgical intervention. "In about 25 per cent of the patients in whom the adhesive process appeared localized with or without compression of the cord by bands, and in whom the symptoms were of less than two years' duration there was considerable improvement in the spastic paraplegia and the sensory disturbances, so that bedridden individuals were able to be up abd about again; bladder disturbances, when they existed, were not relieved, and the patient remained an invalid."

In about 25 per cent of Elsberg's patients the relief was complete or almost complete, so that the individuals were able to return to their work and usefulness. In the majority of these patients the relief was permanent.

Pathologic Findings

In chronic diffuse leptomeningitis there is grossly a definite thickening of the leptomeninges throughout the entire length of the spinal cord. Usually this involvement is not uniform, being more marked at certain levels and in certain regions at a single level. In the more involved areas the membranes are extremely opaque, completely obliterating the underlying structures.

In Elkington's series, 18 of 41 cases showed at operation a loculated collection of fluid amounting to actual cysts. The most conspicuous abnormality was in the arachnoid which was often milky and opaque in appearance and contained areas

irregular patchy thickenings. The pia-arachnoid was bound to the dura, the cord, and the nerve roots by adhesions. In some of the cases there was a complete obliteration of the subarachnoid space.

Since there are very few complete autopsy studies reported in this condition, we shall limit the histopathological descriptions to the study of our own cases.

Before describing the pathologic changes in chronic diffuse leptomeningitis, it might be advisable to briefly review the structure of the normal spinal pia-arachnoid. The spinal pia is composed of a relatively thin layer of intertwining collagenous fibers which are closely adherent to the surface of the cord. The thickness of this layer varies normally from 5 to 15 microns. The pia is very vascular and contains a large number of blood vessels. Along these vessels one can normally observe scattered groups of mononuclears, which often increase in number in inflammatory conditions. The arachnoid is also a thin net-like membrane about three times as thick as the pia and measuring about 30 to 40 microns. It is a relatively avascular membrane composed of collagenous fibers and lined on both its inner and outer surfaces by a thin cellular endothelial layer composed of a single irregular layer of peculiar squamous-like cells which can usually be identified only with special silver stains. There is a definite space between the pia and arachnoid called the subarachnoid space. The latter is traversed by numerous fibrous strands that extend from the arachnoid and are attached to the pia (arachnoid trabeculae). When the leptomeninges are studied on fixed tissue, many of the above details cannot be identified. Often the subarachnoid space is greatly narrowed and the leptomeninges are so intimately related that they often must be identified as a single structure. They then appear as interlacing bundles of collagenous fibers, the inner portion being vascular and probably representing the pia while the outer portion is relatively avascular, somewhat thicker and represents the arachnoid. The total thickness of the leptomeninges normally would vary from 35 to 55 microns.

Histologically, also, there is a diffuse but irregular thickening of the leptomeninges; their combined thickness often varying from 85 to 215 microns (2 to 5 times normal). Either of the 2 membranes may be more severely involved, but often they are so intimately related that it becomes impossible to positively identify the separate membranes. In most areas there is a complete obliteration of the subarachnoid space although occasionally in the less severely involved areas this space can still be identified although greatly reduced in size. In some cases the thickened leptomeninges may become so extensive that the process extends outward to obliterate even the subrudal space, the thickened membranes merging with the inner dural layer which, however, can usually be verified by its more dense structure. This thickening of the leptomeninges may or may not extend inward to involve the membranes within the anterior and posterior commissures of the cord.

The involved pia-arachnoid shows definite structural variations. It is usually definitely acellular and comprise of dense collagenous tissue. Often it becomes partially or even completely hyalinized, loosing all structural characteristics and assuming a most homogeneous appearance. A few connective tissue nuclei can be found in such cases but even these are pyknotic. Scattered collections of mononuclears are occasionally present.

The spinal vessels show a most variable degree of involvement. The radial vessels surrounding the cord are invariably compressed by the thickened membranes and many are completely occluded. The degree of vascular involvement frequently is in direct proportion to the thickness of the pia-arachnoid. The vascular pia, in some cases, appears entirely devoid of vessels while in other cases it shows a definite reduction in its vascularity. The sulcal arteries are usually completely surrounded and often compressed by the thickened membranes resulting, in many cases, in a definite vascular narrowing. In a few

areas these vessels are completely occluded and produce a focal softening or even a complete central cavitation within the spinal cord. Usually the smaller paracentral arteries escape damage, but in an occasional area even these vessels become compressed and occluded.

The spinal rootlets as they penetrate these thickened membranes may remain uninvolved, but more commonly they are narrowed, compressed and show definite pathologic alterations. Demyelinization is invariably present with a variable destruction of nerve fibers and a secondary fibrous tissue replacement of the involved areas. In some rootlets almost half the fibers appear to have been replaced by a secondary fibrosis.

The spinal cord usually shows some changes at some of its levels. These alterations appear to be directly related to the degree of vascular involvement. The meningeal thickening never appears sufficient to produce cord compression. The cord shows a moderate swelling of the myelin sheaths, often with some patchy vacuolization. In some cases, vascular occlusion produces definite focal areas of softening which involve large areas of a single cord level. Even complete central cavitation may occur, producing a typical syringomyelic picture. The nerve cells are usually uninvolved although moderate changes may occur in the more severe cases, consisting of swelling, chromatolysis, fragmentation and even complete disappearance. In some cases petechiae occur throughout the gray matter of the cord.

Case I

[redacted], a 57-year-old white male. On September 1, 1937, while fixing a tire, he stumbled backwards and struck the lower part of his back against a cement curb. He got up without assistance and continued his work. During the following days the base of his spine felt sore, usually becoming worse at night. However, he continued to work regularly. On September 14, two weeks after his injury, he noticed muscular twitching in the right thigh. The next day he had pain and numbness in the back and about the right hip. On the next day, while trying to rise from a stooped position, he lost control of both legs. This was followed almost immediately by severe pain extending down to both feet. In 3 or 4 hours his legs became completely paralyzed and remained so for about 5 weeks. He had bowel and bladder incontinence for the first 3 weeks of that period. He had a patchy sensory loss from the umbilicus down. After 3 weeks, his urinary difficulty disappeared. However, he did not regain rectal sensation. After 5 weeks, control of the legs began to return. At the same time he noted gradually increasing stiffness of the legs. A "burning feeling" was associated with the stiffness and return of motion. He was able to walk with some support. He continued to have pain in the lower back and legs.

About 15 years previous to the difficulty already described he suffered a slight head injury. Six weeks later he had dizziness and pains in the neck; the dizziness was brought on by rotation of the head and he had some difficulty maintaining his balance. He had no paralysis, and the symptoms disappeared in three weeks.

He was admitted to the University Hospitals on December 23, 1937, at which time the neurologic examination revealed: hearing loss on the right; hyperactive deep reflexes in the legs with bilaterally positive Babinski signs; positive Chaddock and Oppenheim signs on the right; left patellar and bilateral ankle clonus; weakness and spasticity of the legs; incoordination of the legs with loss of position sense and diminished vibratory sensibility; absence of pain, temperature and light touch sensibility from the 12th thoracic segment down. The rest of the physical examination was essentially negative.

The spinal fluid examination revealed a pressure of 10 mms. of mercury with no rise on jugular compression; protein, 56 mg. per cent; no cells, and serologic tests negative for syphilis. Urinalysis and routine blood studies were normal.

X-ray report: "Lipiodol examination of the spine was done radiographically [illegible] after the injection

of 2¼ cc. of lipiodol into the cisterna magna. At the beginning of the examination all of the lipiodol was in the upper cervical spine and cisterna. After putting the patient in the upright position the lipiodol moved very slowly through the cervical canal and met a temporary obstruction at approximately the level of the 1st thoracic vertebra. After approximately 4 to 5 hours in the upright position there was still lipiodol retained at the level of the 6th thoracic, 11th thoracic and 2nd lumbar vertebrae. A few droplets had dropped down into the sacral canal. In one of the lateral views at the level between the 1st and 2nd lumbar vertebrae there was a suggestion of posterior bulging into the canal from the intervertebral disc. Findings suggest an arachnoiditis together with the possibility of multiple ruptures of the intervertebral discs. A definite diagnosis can not be made." (Dr. Harold Peterson).

On 1/11/38, the lamina of the 1st and 2nd lumbar and the 12th thoracic vertebrae were removed, and there was apparently some encroachment on the cord at this level. There was no pulsation of the cord. The conus region was exposed. The rootlets seemed to be very thick and heavy as though they were swollen. A slightly protruding intervertebral disc was found. It did not appear to be ruptured or prolapsed in the usual manner. It was simply compressed back into the spinal canal for an elevation of about 3 or 3½ mm. Through an incision in the anterior surface of the dura part of the disc was excised and part of it was curetted out. A small amount of lipiodol seemed to be held in position just above the protrusion. The rest of the lipiodol could not be evacuated by changing the patient's position. (Dr. W. T. Peyton).

The postoperative course was very stormy. On 1/18/38, the blood culture was positive for hemolytic streptococci. The patient developed uncontrollable abdominal distension. Despite treatment by sulfanilamide, nasal suction and other measures, he failed to rally and died on 1/20/38, about 5 months after the onset of his illness.

Pathological studies: The leptomeninges surrounding the entire cord was thickened, both membranes being about equally involved and measuring from 55 to 210 microns in thickness. The subarachnoid space was narrowed and in some areas completely obliterated. The pia-arachnoid appeared avascular, acellular and somewhat hyalinized. Not even the remnants of pyknotic nuclei could be detected. The arachnoid trabeculae were narrowed and thickened. The membranes within the commissures were also greatly involved. The sulcal arteries were completely surrounded and compressed by the thickened meninges, producing a definite vascular narrowing. Even the smaller paracentral arteries were partially compressed by the extensive meningeal changes.

The rootlets as they passed through the thickened meninges were greatly constricted and appeared to have undergone partial destruction. They contained a great decrease in the number of myelin sheaths with a secondary fibrous tissue replacement of the destroyed elements. Almost half the rootlet fibers appeared to have been replaced.

The spinal cord showed a moderate demyelinization which was particularly marked in the posterior columns. The nerve cells appeared structurally intact.

Case II

. was 51 years of age at time of death, November, 1940. In May of 1937, the patient first noted an itching sensation in band form at the level of the nipple line. At the time of examination, a burning and smarting sensation replaced the itching. The sensations were intermittent, coming in the form of attacks as frequently as every 5 or 6 seconds on one side or the other. Two days after the onset of [illegible] difficulty, his appendix was [illegible] at another hospital. [illegible] after operation the right leg became numb and he experienced some difficulty in walking. In a few weeks the left lower extremity became numb.

stated that when he crossed his legs in bed he was unable to determine which leg was on top. He first entered the hospital 3 months after the onset of his illness. His chief complaint was numbness and loss of sensation in the legs from the hips down to and including his feet. He also complained of a constricting sensation around his waist.

His past history and family history were essentially negative. In March of 1937, the patient wrenched his back while lifting a heavy box. He was not able to work for 2 weeks.

Physical examination was negative except for the following neurologic findings: The cranial nerves were essentially normal except for diminution in visual acuity on the right. He was unable to read large new print with the right eye. The upper extremities were normal, except the right triceps reflex was more active than the left. Only the right upper abdominal reflex was elicited. There was spasticity of the legs, with hyperactive deep reflexes and positive Babinski's bilaterally. There was mareked weakness of the right leg. Vibratory sensibility, muscle pain and position sense were normal in the left leg but markedly impaired in the right leg. Light touch and pin prick was felt normally down to the 3rd rib on the right and the 4th rib on the left; below these levels there was patchy loss of superficial sensation. There was no sphincter impairment.

Laboratory studies, including tests for syphilis on the blood and spinal fluid were negative. There was no evidence of subarachnoid block. The spinal fluid was clear and colorless, contained 40 mg. of protein per cmm. and no cells. The gold curve was 022110000.

X-ray studies of the spine with the use of lipiodol were negative.

About 3 weeks after his hospital admission, the patient experienced some improvement. His legs improved in strength and his sensory disturbances were not as severe as previously. He was discharged in September after 1 month of hospitalization. However, his improvement was of brief duration, and he was readmitted in October, 1937. In November, 1937, a laminectomy was performed in the region of the 7th cervical and 1st thoracic vertebrae. There was no obstruction. The cord appeared normal. The arachnoid seemed thickened in several places. His postoperative course was uneventful and he was discharged unimproved. He was followed in the outpatient department from that time until his final hospital admission in 1940. He complained bitterly of constant itching and burning sensations. He was unable to sleep, lost weight and gradually became weaker. He seemed to improve following a course of deep x-ray, but his improvement was again of short duration. His weakness became so marked that it was necessary to hospitalize him again in September, 1940. At that time he complained of bladder and rectal incontinence of 3 or 4 months duration. The last 6 months previous to admission it was difficult for him to walk because of weakness of the legs. He had last reported 2 weeks prior to admission. He first had numbesss of both hands and then his left hand became paralyzed. Neurologic examination showed spastic paralysis of both legs and atrophy of the small muscles of the hands. The left hand was flexed at the wrist and the fingers were flexed to form a cupping effect. Both arms showed a patchy superficial sensory loss. The patient developed signs of urinary tract infection. He became weaker, developed a pneumonic process in the right lung, and expired onNovember 6, 1940, about $3\frac{1}{2}$ years after the onset of his illness.

Pathologic findings: Autopsy revealed a pneumonic process in the right lung. Serial sections of the brain revealed no gross abnormalities. The dura was normal except for thickening in the operative region. The arachnoid showed yellowish, thickened, indurated areas averaging about 5 mm. in diameter and scattered throughout the thoracic and lumbar areas. There was atrophy of the upper thoracic and cervical regions of the spinal cord with small areas of hemorrhagic shoftening.

Microscopic studies showed a moderate diffuse thickening of the pia-arachnoid throughout all levels of the cord. This alteration was most marked in the pia,

which measured 15 microns in thickness, was very acellular and contained very few vessels. The arachnoid was less severely involved but was also definitely thickened, especially in the anterior and posterior aspects of the cord. The extensions of these membranes into the commissures showed a similar structural alteration and thickening. The rootlets were completely surrounded by these thickened meninges but showed no structural alterations.

The sulcal arteries were for the most part uninvolved. In a few areas their lumens were narrowed. In some of the sacral segments these vessels appeared completely occluded and had produced a focal softening within the cord. Sections through the upper sacral and lower lumbar levels revealed a complete central cavitation of the cord. The tissues around this cavitation were fragmented but showed a minimal degree of cellular reaction.

The spinal cord contained a diffuse dilatation of the myelin sheaths and some swelling of the anterior horn cells. Numerous distended vessels and petechiae were present throughout the gray matter of the cord.

Case III

, a white male 57 years of age at time of his death in August, 1940.

The patient was admitted to the General Hospital in January, 1940, complaining of pain and weakness of the left leg, numbness and a sensation of cold in the toes and inability to walk. The symptoms began seven months previously with pain in the left ankle. In a few weeks the pain began to spread gradually involving the calf, knee, thigh, and hip on the left side. The pain was steady, aching in character, and made worse by cold. It was noticed that his toes would readily become damp and cold. This was more marked in the left foot. Several months after the onset of the illness, the patient developed marked weakness in the left foot. For a few months previous to admission he had urinary frequency.

Physical examination revealed a well developed, obese white man who did not appear to be acutely ill. His feet were cyanotic and cold to the touch. The upper extremities were essentially normal. There were fibrillary tremors in both thighs and a flaccid paralysis of the left leg with absent deep reflexes and foot drop. The right leg was very weak. There were patchy scattered areas of hyporthesia.

Laboratory studies showed a hemoglobin of 91 per cent and a white blood count of 6,050. The spinal fluid was clear and contained 50 mgm. per cent of protein and no cells. The pressure and response to the Queckenstedt test were normal. Serologic tests for syphilis on blood and spinal fluid were normal. An air myelography was negative.

Course: The patient complained constantly of pains in his legs. In May, 1940, about 1 year after the onset of his illness, there was definite atrophy of both legs from the hips down. At that time he complained of paraesthesias in both hands. In June he developed lobar pneumonia which developed into a chronic unresolved process. In August a neurologist recorded atrophy of the muscles of the arms with marked weakness and flaccid paralysis of both legs with marked atrophy. The patient gradually became weaker, developed pneumonia on the right side, and expired in August, 1940, about 15 months after the onset of his illness.

Pathologic findings: The spinal cord and meninges were grossly normal. The vessels at the base of the brain revealed a moderate degree of arteriosclerosis. Serial sections of the brain showed no gross abnormalities.

Microscopic sections revealed a diffuse but irregular thickening of the spinal leptomeninges, which measured from 80 to 175 microns and produced a partial obliteration of the subarachnoid space. These membranes were extremely fibrous and [illegible]ly acellular although they did contain a few nests of mononuclear cells. The vessels surrounding

the cord were markedly compressed by the thickened meninges within which they were enmeshed. Some of the vessels were almost completely occluded.

The rootlets as they passed through the involved membranes did not appear to be extensively altered although in some of the lumbar segments these did appear to be a mild compression of the posterior rootlets with some replacement of the destroyed elements by connective tissue.

The cord appeared intact, neither the white nor gray substance showing any changes.

Case IV

(This case is being reported through the courtesy of Dr. A. H. Wells, Duluth). (. was a male child who was 17 months of age at the time of his death in May of 1940. The patient was dead on admittance to the hospital and there is, therefore, scant clinical information. He had been in a hospital about 2 months before his death. At that time it was noted that the child had been sick for 2 weeks with fever and loss of weight. He had developed cough and dyspnea and had refused food for the last two days before admission. The physical examination revealed rales in both lungs and extreme malnutrition. The reflexes were normal.

A physician who had attended him reported that the child developed normally until 11 months of age, when he first showed symptoms. In a short time he was unable to sit up and could not eat well. Two months before death he developed a severe bronchopneumonia. Following that illness he became very much weaker; he lay in bed, hardly moving a muscle.

Pathologic findings: The autopsy, performed 3 hours after death, revealed a bronchopneumonia and acute pancreatitis. Postmortem blood chemistry showed a blood sugar of 30 mg. per cent, and a urea nitrogen of 47.3 mg. per cent.

Microscopic sections revealed a fibrosis of the pia-arachnoid throughout the entire spinal cord. These membranes were greatly thickened and had resulted in a complete obliteration of the subarachnoid space. In many areas the collagenous pia-arachnoid fused imperceptibly with the dura thus also obliterating the subdural space. The arteries surrounding the cord were compressed but not completely occluded by the meningeal involvement. The nerve rootlets were encircled but not particularly altered. The only changes seen in them were scattered areas of myelin swelling with an early formation of geometric figures due to the breakdown of the neurokeratin network.

There was a decrease in the number of nerve cells in the anterior horn, the remaining neurons being pale, fragmented or shrunken. Many ghost cells were observed. Numerous petechiae were present within the gray matter and especially near the dorsomedial cell columns. A small cystic area was encountered in the gray commissure lateral to the central canal. The white matter of the cord appeared to be intact. There was a little swelling of the myelin which was most pronounced in the marginal region of the lateral columns.

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VI. RECOMMENDATIONS

The following recommendations are made to the physicians of the United States by the committee on food and nutrition of the National Research Council:

Improvement of Flour and Bread

A procedure to improve the nutritive quality of flour and bread: For the time being synthetic vitamins are utilized to restore essential substances to white flour and bread. Thiamine, nicotinic acid or nicotinic acid amide, and the mineral iron are the substances now being added. Ultimately processes of milling may be developed which will preserve the vitamins and minerals originally in the wheat in a flour acceptable to consumers. The purpose of this recommendation is to insure immediately a supply of these vitamins of the vitamin B Complex (thiamine and nicotinic acid) which are essential for the satisfactory oxidation of carbohydrate foods. The required levels for these vitamins, as well as for iron, will approximate those found in whole wheat flour and bread. (Many millers and bakers have made such products available under the designations "enriched" flour and "enriched" bread. Regulations for their control will soon be promulgated by the Food and Drug Administration, through the Federal Security Agency.)

Use of Iodized Salt

The widespread use of table salt iodized to a standard level of one part of potassium iodide or equivalent per 10,000 parts of salt is recommended. (Iodized salt has been available for many years, but its use of late has unfortunately declined; the incidence of endemic goitre is on the increase. Conclusive evidence confirms that harm does not attend the use of iodized salt by persons otherwise receiving an adequate supply of iodine.)

Addition of Vitamin A to Oleomargarine

A recommendation has been adopted to promote the general fortification of oleomargarine with vitamin A to a level of

9000 International Units per pound. (Such oleomargarine is now replacing unfortified oleomargarine.)

Summary

The Committee believes that use of whole wheat bread and of butter should be encouraged by vigorous educational effort. Where these natural foods are not used, enriched flour and bread and properly fortified oleomargarine should be preferred over unenriched white flour and bread and oleomargarine not containing vitamin A. Use of iodized salt is also to be emphasized. These nutritionally improved products will greatly facilitate the planning of satisfactory diets.

These recommendations have the endorsement of the Council on Foods and Nutrition and of the Board of Trustees of the American Medical Association. Your personal endorsement of them will help more. Strong leadership in the campaign for improved nutrition must come from physicians; your active support of the nutritional activities of your community is essential.

Acceptance and application of the recommendations of the National Nutrition Conference and of the actions of the Committee on Food and Nutrition by all the people is required. If our great Nation is to preserve its heritage, Americans must have a diet that more nearly meets the Recommended Dietary Allowances advocated by the National Nutrition Conference. (See Jour. A.M.A. June 7, 1941) Nutritious food may well determine the effectiveness of the national defense. The people must understand; they look to you for guidance.

Russell M. Wilder, M.D., Chr.
Committee on Food and Nutrition
National Research Council

James S. McLester, M.D., Chr.
Council on Foods and Nutrition
American Medical Association.

VII. GOSSIP

The annual report of the Surgeon General of the Navy is always of interest to me. The death rate in 1939 was 2.10 per 1000. Motor vehicle accidents again played a prominent role, causing 50 deaths, including 1 suicide. Of the 42 attempted suicides, 32 were successful (all records indicate men try to do this less often than women, are uniformly more successful). Diseases accounted for 127 deaths, injuries for 181, and poisons for 6. There were 8.03 sick days per person and 19.67 for admission. In all 22% of the personnel was constantly on the sick list through the year. In 1937 punitive measures imposed on enlisted men for venereal disease were removed. The increase in venereal disease incidence from 1938 to 1939 was less than previously reported. The report is interesting because it reflects differential disease and death rates of specially selected groups. Note the absence of deaths from drowning. The age group 20-24 always shows suicide as one of the first five causes of death.

The American Medical Association found the cut of the Medical School in Trends in Medical Practice and Research of interest. They intend to run it in the student section of their journal together with a brief history of the school. Time, the news magazine, found the article on athletic injuries of greatest interest and requested our news department to send further photographs illustrating the preventive aspects of athletic injuries. Many readers found the double columns of interest. Eventually when this feature is smoothed out, all column notes will be on the outside margin and will directly relate to the material in the larger column on the inside. Nearly 6,000 issues of this number will have been mailed before the end of this week. They have gone to members of the Minnesota State Medical Association, our alumni, the medical students, physicians who have attended courses at the Center for Continuation Study, medical libraries, medical journals, and graduates in medical science who have earned a doctor of philosophy or master of science degree. In addition, many interested individuals and organizations have received copies.

Speaking of publications, the Homecoming News number of the Minnesota Alumni weekly was edited by Alois F. Branton, Jr., of Willmar, son of Dr. Alois F. Branton, Sr., executive secretary of the Minnesota Hospital Association and Willmar practitioner. Alois, Jr. has shown a distinct flair for journalism since his high school days. He assisted in editing last year's Alumni Homecoming number and this year was given complete charge. Many will recognize him also as the courteous, efficient young man who presides at the main information desk on certain hours of the day. He is a sophomore student at the University in the College of Science, Literature, and Arts.

The Doctors Mayo by H. B. Clapesattle is now off the press. The story of the Mayos is one of the fascinating tales of our day. It covers a period of 100 years of medical progress in Minnesota. (Note: The Minnesota State Medical Association has just issued a commemorative pictorial publication going back to the days of Christopher Carli who received his medical education at Heidelberg and came up the rivers of the new world to the territory that later became Minnesota. He arrived at the site of what is now Stillwater on May 24, 1841, and became the first civilian medical practitioner to settle permanently in the territory.) To commemorate the appearance of the Mayo book, the University of Minnesota officials, the University Press, together with the author (a woman), and the Mayo family will hold a reception for friends of the institution at the Coffman Memorial Ballroom on Wednesday, December 10. Invitations have been issued. The story is full of interesting tales concerning the development of the Mayo tradition and contains an excellent array of intimate pictures of the entire Mayo family, including many of their friends of earlier days. One of the most interesting characters in the book is the mother of Drs. Will and Charlie. The jacket contains a story of the University of Minnesota (and Press), including a cut of the new campus. The descriptive statement repeats the message cut in stone over the entrance of the auditorium facing the mall. "Founded in the faith that men are ennobled by understanding, dedicated to the advancement of learning and to the search for truth, devoted to the instruction of youth and the welfare of the State."

We are honored again. Health Service Director, Ruth E. Boynton, has been appointed a member of the sub-committee of the national committee on assignment and procurement to work with women physicians in the interest of national defense. Dr. Boynton has been selected for this purpose from a large list of women physicians in the United States because she so well represents the type of medical citizen who always works in the best interests of public welfare.

The Eustis Big Little News, Vol. I, No. 4, is off the press. This is a mimeographed publication sponsored by the children who are patients in the Eustis Hospital. It is one of the "freshest" of our publications. Every page is of interest, as it deals with persons and events as children see them. The following tribute to their chief is of interest:

"OUR CHIEF OF STAFF

Dr. Irvine McQuarrie is Professor of Pediatrics and head of the Pediatrics Department at our hospital. He is married and has three daughters. One of them is now attending the University of Minnesota. While at the University of Rochester in New York, before coming here, he worked with Dr. Eric Kent Clarke of the Psychiatric Clinic for Children. They worked together there for fourteen years.

Dr. McQuarrie has also traveled in various countries in Europe. He was visiting Professor of Pediatrics at Peiping Union Medical College in China.

His home is in Minneapolis and he likes it here very much. His greatest ambition is to have a good children's department. Photography is his hobby and his favorite sport is fishing.

Dr. McQuarrie says that the thing that irritates him most is anybody's interference with the smooth running of the Pediatrics Department. Next week Dr. McQuarrie is going to the University of Cincinnati as Visiting Professor.

He is very much interested in our newspaper and as he is a very busy man, we would like to thank him for taking the time and trouble to be interviewed.

Dorothy Chandler,
Age 16,
Willernie, Minnesota."