Bulletin of the

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and
Minnesota Medical Foundation

Arteriovenous Malformations of the Brain

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I. ARTERIOVENOUS MALFORMATIONS OF THE BRAIN

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Arteriovenous malformations of the brain represent a small but important proportion of intracranial lesions seen by the neurologist and neurosurgeon. MacKenzie[6] in 1953 reported that such anomalies constitute only one per cent of all brain tumors admitted to the National Hospital in London. Such a percentage is supported by the fact that 41 verified malformations have been seen in the University Hospitals from 1934 to 1954. The incidence of verified lesions at this institution has increased in recent years with the increasing utilization of cerebral angiography (Figure 1). In the first fifteen years of the period studied for this report, 18 malformations were seen, while the remaining 23 were diagnosed in the last five years.

One of the first major reports in the literature concerning these malformations was by Dandy[3] in 1928. He reported on eight cases of his own and on twenty-two collected from the literature. He found that approximately forty per cent of the untreated patients died of a cerebral hemorrhage. In 1948 Olivecrona and Riives[9] reported on sixty-five cases and stated, "Most, if not all, patients die of hemorrhage, or are completely incapacitated." In 1951 Bassett[1] reported eighteen cases with eleven having evidence of a subarachnoid hemorrhage. He stated, "In no instance was the effect as devastating as that from the subarachnoid or intracerebral hemorrhage accompanying rupture of an aneurysm." Bassett[1] also stated that these patients suffer from frequent recurrent hemorrhages, but rarely are they fatal. In view of the conflicting opinions expressed in the literature it was felt that an additional study was warranted. This report concerns forty-one cases of verified arteriovenous malformations seen at the University Hospitals from 1935 to 1954. Only those cases are included where the diagnosis was verified by surgical exposure, angiography, or postmortem examination. Complete clinical data are included and analyzed in order to provide a basis of comparison with other reports in the literature.

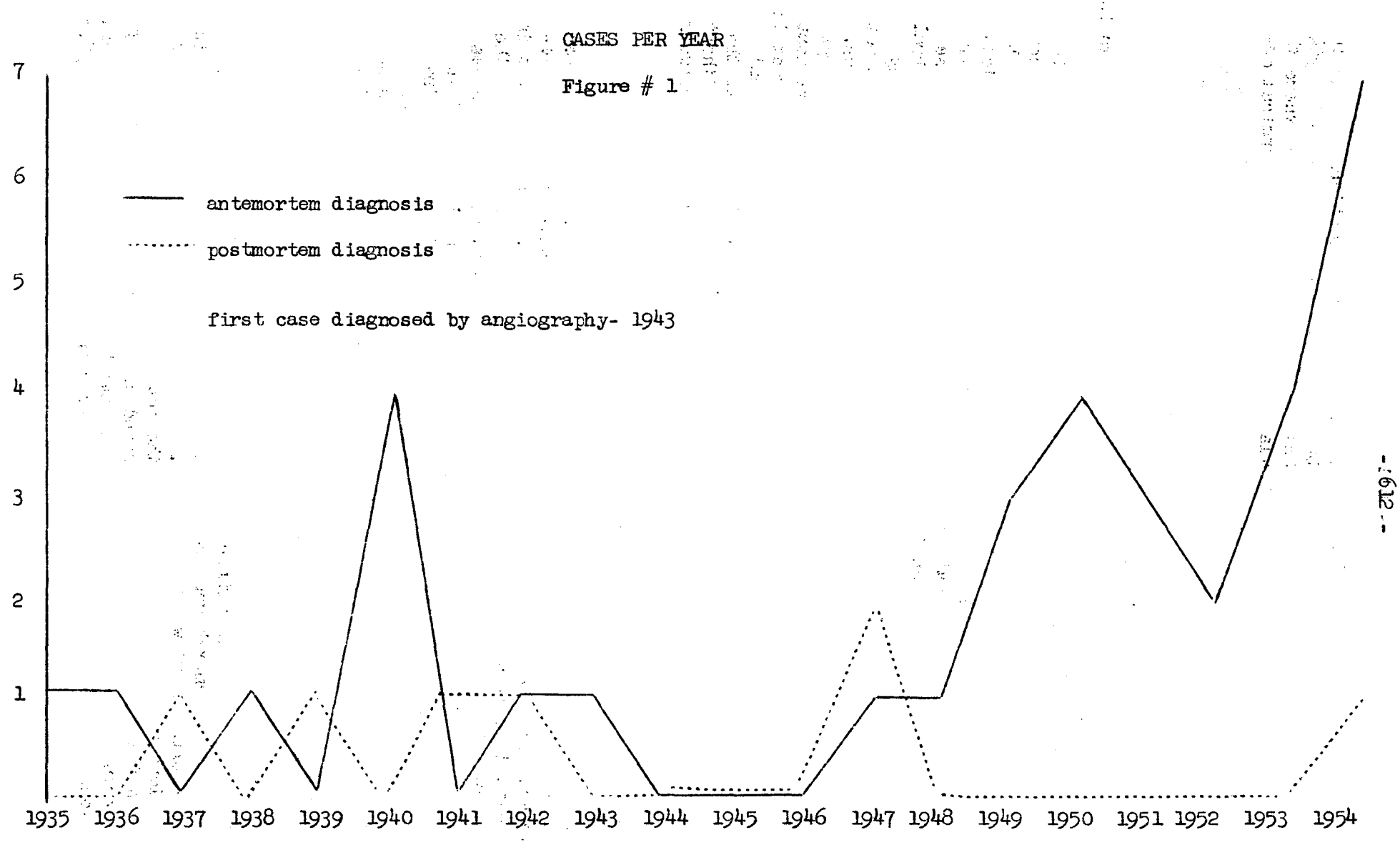
These lesions are generally held to be congenital in origin and result from persisting abnormal communications between the embryonic arterial and venous circulation. There is brain tissue between the vessels of the malformations which serves to differentiate them from true vascular tumors (Noran[7]). These malformations are often called cerebral angiomas, a term which is misleading since they are not neoplasms and do not grow in the way that a neoplasm grows. They increase in size but only through a gradual dilatation of their component vessels. This dilatation is thought to be the result of continuous abnormal pressure transmitted directly into the venous channels from the artery without the usual intervening capillary network.

Incidence

The sex incidence was equal in twenty-five males and twenty-five females reported by MacKenzie.[6] The incidence in males was twice that in females in the cases reported by Olivecrona.[9] Males only were reported by Dandy[3] but the series was small and this predominance was obviously due to chance. The University of Minnesota Hospitals series contained twenty-six males and fifteen females.

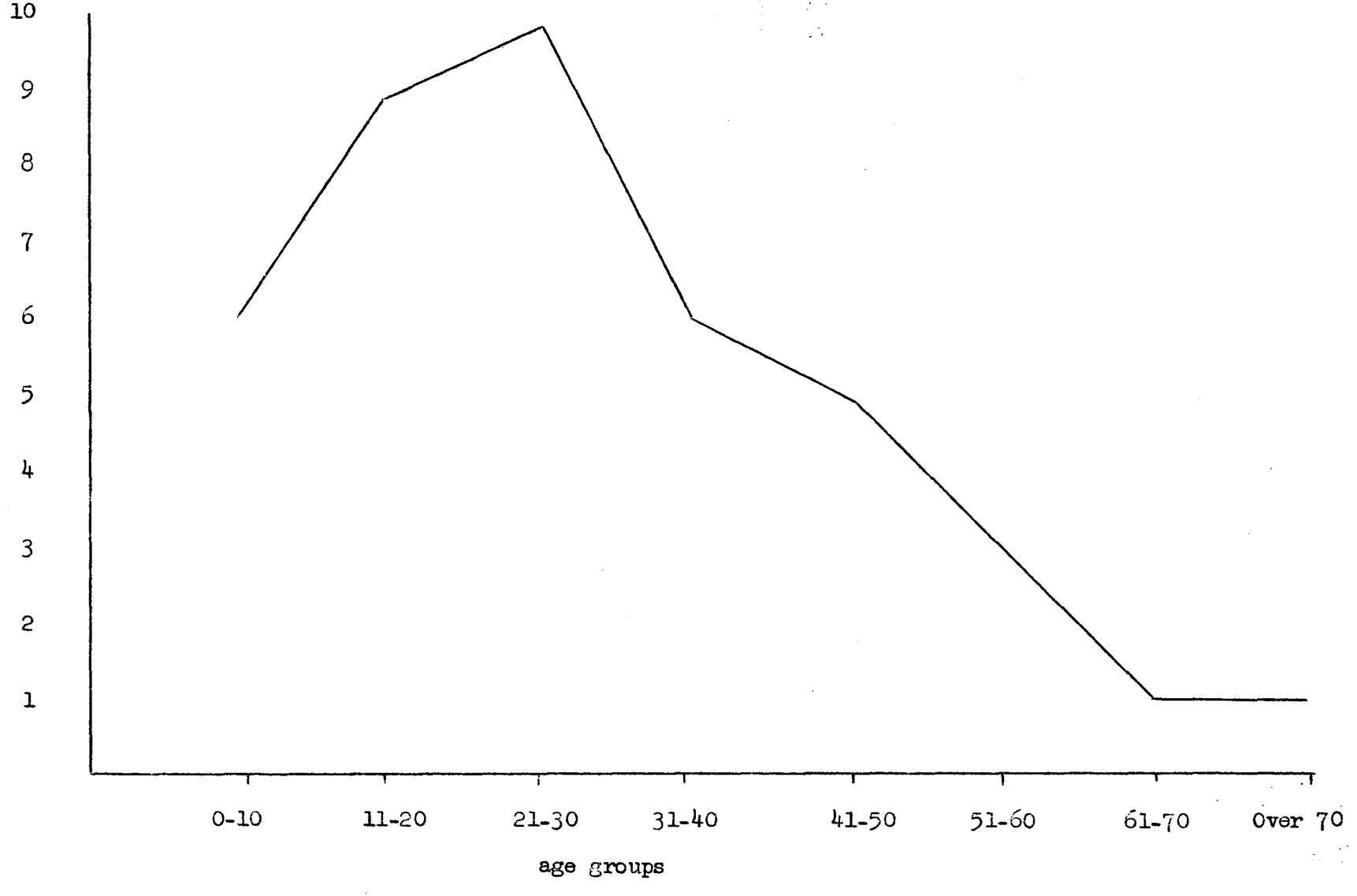
Symptoms

Symptoms referable to arteriovenous malformations may begin at any age, but it is more common to see the onset in the younger age groups. MacKenzie[6] found 58 per cent to have the onset of symptoms between the ages of eleven and thirty years. Our series also had 58.5


CASES PER YEAR
Figure # 1
antemortem diagnosis
postmortem diagnosis
first case diagnosed by angiography- 1943
7
6
5
4
3
2
1
1935 1936 1937 1938 1939 1940 1941 1942 1943 1944 1945 1946 1947 1948 1949 1950 1951 1952 1953 1954

AGE INCIDENCE

Figure # 2



per cent with the onset of symptoms in this age group (Figure 2). The youngest case was diagnosed at the age of six weeks following the parents' observation of rapidly increasing head size. The lesion was in the left cerebellar hemisphere where it had produced a block of the flow of cerebrospinal fluid. The oldest case was admitted following a subarachnoid hemorrhage at the age of seventy-four years but had noted a subjective bruit eight months before. The diagnosis was confirmed by angiography.

The most commonly recorded symptoms are hemorrhage, seizures, headache, mental deterioration, bruit, or the sudden onset of a hemiparesis or a hemiplegia.

Chart 3

Symptoms

	Number	Percentage
Hemorrhage	24	56%
subarachnoid 15		
intracerebral 9		
Seizures	17	41%
Headache	10	25%
Mental Deterioration	8	19%
Bruit	6	15%
Visual disturbances	3	
Hydrocephalus	2	
Hypopituitarism	1	

Chart 4

Duration of Symptoms

1. Less than 48 hours	5
2. 48 hours to 6 weeks	4
3. 6 weeks to 2 years	16
4. 2 years to 32 years	16

As stated previously, recurrent hemorrhage is considered to be a common finding in the clinical course of this disease. Pilcher et al,[10] Bassett,[1] and MacKenzie[6] feel that the hemorrhage from these lesions is rarely fatal, while Dandy[3] and Olivecrona and Riives[9] hold that it is the most common cause of death. In our series, twenty-four cases (56%) had one or more hemorrhages. In seventeen it was the first symptom of the disease. Twelve (50%) of the twenty-

four had more than one episode of bleeding. One patient had four known subarachnoid hemorrhages before coming to operation. Four cases died almost immediately from the first episode while four more died after the second or third hemorrhage. The interval between bleeding episodes varied from seventy-two hours to twenty years. Two cases bled a second time five and six months respectively after the first episode. Four cases bled again one to four years later and four more bled at seven, eight, fifteen, and twenty years after the initial hemorrhage. In nine of the twenty-four cases, the hemorrhage was intracerebral. Seven of these suffered severe neurological damage or died shortly after the hemorrhage. The mortality rate from hemorrhage is approximately twenty per cent of the entire series or thirty-three per cent of all cases which had a hemorrhage.

Seizures are often reported to be focal in nature and are said to be one of the most frequent features of this disease. Seventeen (41.5%) of the cases in this series had seizures during the course of their illness. In ten cases, seizures were the first symptoms. Four of the seventeen had grand mal attacks without a localizing component and two had grand mal seizures with localizing features. One patient had an aura of spots before her eyes suggestive of an occipital lobe lesion. This subsequently was confirmed. The other patient had occasional postictal paralysis on the left side, pointing to a lesion in the right Rolandic area which subsequently was confirmed. Three cases had psychomotor seizures, and all three had lesions in the temporal lobe. Seven cases had typical Jacksonian seizures, six motor and one sensory. The one sensory and four of the motor seizures were accurately localizing for a lesion in the pre-Rolandic area. However in two the typical Jacksonian motor march proved to be misleading inasmuch as one had seizures beginning in the left hand with spread to the left half of the body but the lesion was situated in the right occipital lobe. The other with a similar motor march had the malformation in the temporal lobe. The remaining case suffered from an aura of nausea for five years and eventually began to have adversive seizures indicating a frontal lobe lesion but it was found to be in the temporal lobe. Consequently it can be seen that ten of the seventeen patients with seizures had a localizing feature in their seizure pattern which was accurate, three exhibiting falsely localizing features, and four had no component of any kind which could be interpreted as localizing.

Headache was observed in ten cases as a prominent symptom. MacKenzie[6] stated that there is frequently a typical

Chart 5

Seizures

Total Cases 17

Nature of Seizures	Number of Cases
Grand mal without localizing component	4
Grand mal with a localizing component	2
Jacksonian	7
Temporal lobe	3
Adversive	1

syndrome closely resembling migraine except that the aura is often prolonged and may persist even after the headache has developed, a rare event with true migraine. He also noted that many cases exhibit headaches, which if one sided, are persistently so, and are on the side of the angioma. In our series only one case had a headache which resembled migraine. The remaining nine patients with headache demonstrated no particular pattern. They complained of severe generalized headaches without any premonitory symptoms. One case had severe bifrontal headaches for fifteen years before developing Jacksonian seizures which led to an investigation and diagnosis. Another was known to have severe generalized headaches for twenty-one years associated with slowly progressive mental deterioration and hypopituitarism. The other seven cases had moderately severe headaches for periods of one to eight years before developing other symptoms causing them to seek medical attention. There were three cases with headaches of one to three weeks' duration which were not included in the above figures, since the headache was due to increased intracranial pressure from obstruction of hemorrhage.

Mental deterioration has been mentioned as a feature of this disease. Norlen[8] states that it is common and often severe. On the other hand, MacKenzie[6] found it to be rare and did not record any instance of severe deterioration in his series of fifty cases. The usual explanation is that deterioration is secondary to hypoxia of the cerebral tissue as a result of the shunting of blood flow by the malformation. However, it also may be a sequel of cerebral destruction following hemorrhage and the formation of an intracerebral hematoma. In particular, such damage to the frontal lobes is likely to produce mental changes. Eight of our series were shown to have mental deterioration of a significant degree. One patient had an acute personality change lasting three weeks which was considered to be the result of a large hematoma in the right frontal lobe. The other seven cases exhibited slowly progressive changes varying from mild confusion to a severe organic psychosis which required institutionalization. None of these seven had any evidence by history or by examination of a hemorrhage. Two did have a chronic cerebrospinal fluid obstruction with low-grade increased intracranial pressure which may have contributed to the mental changes. Figure 6 demonstrates the nature and duration of the mental changes.

The presence of a bruit is mentioned as frequent by observers in these cases. MacKenzie[6] states that a bruit was heard in 24 of the 48 cases in whom it was listened for and he considered it to be a

Chart 6

Age	Nature of Deterioration	Duration	Presumptive Cause
57	Mild confusion	1 year	Hypoxia
15	Severe truancy problem	1½ years	Hypoxia
21	Severe intellectual impairment	16 years	Hypoxia and chronic hydrocephalus
40	Moderate confusion, memory loss	5 years	Hypoxia
32	Moderate confusion	1 year	Hypoxia, chronic hydrocephalus
27	Severe intellectual impairment	1 year	Hypoxia and partial replacement of both frontal lobes by malformation
55	Severe psychosis, paranoid	12 years	Hypoxia
25	Moderate confusion	3 weeks	Large frontal lobe hematoma

sign of considerable diagnostic importance. Actually it is not diagnostic, since it can be heard in very vascular tumors such as angioblastic meningioma and in the presence of a carotid-cavernous sinus fistula. In our series eighteen cases had no record of the presence of a carotid-cavernous sinus fistula. In our series eighteen cases had no record of the presence or absence of a bruit. In the other twenty-three cases six had an audible bruit and seventeen did not have a bruit audible to the examiner. Only two cases were recorded as subjectively noting a bruit. Here the duration of this complaint was two months in one and eight months in the other patient. Large lesions and superficial lesions are, according to MacKenzie,[6] most apt to have a bruit and a deep midline lesion, even if large, is less likely to be audible. However, in our series many of the large and superficially placed lesions did not have an audible bruit. Of the six cases where a bruit was heard, four were large (3 cm. or more in size) and were superficial; one was medium sized (1-3 cm.) and superficial; and the last was large but deeply placed in the midline. Seven of the seventeen noted to have no bruit had large superficial lesions.

Location

Arteriovenous malformations may involve any vessel of the brain but appear to show a predilection for the component parts of the middle cerebral arterial circulation. There were twenty-one malformations arising from the middle cerebral artery, eight from the anterior cerebral artery, six from the posterior cerebral artery, one directly from the internal carotid artery, and one from the anterior choroidal artery. Nine malformations were in the posterior fossa with seven arising from a cerebellar artery and two from the basilar artery. In all, there were forty-six separate malformations recorded since two cases had multiple lesions. One of these had four distinct malformations. The location of the lesions as to depth seemed to correlate somewhat with the nature of the symptoms. All but one of the patients with seizures had a malformation that was superficial and involved the cortex. The deep midline supratentorial lesions usually manifested themselves initially by hemorrhage or mental deterioration. The posterior fossa malformations produced obstruction to the flow of cerebrospinal fluid, local signs of cerebellar or brain stem dysfunction or hemorrhage.

Physical Findings

The neurological damage caused by these malformations was extremely variable and was secondary to hemorrhage, hypoxia, thrombosis in the abnormal

Chart 7

Location

Artery of Origin	Number of Cases
Middle cerebral artery	21
Anterior cerebral artery	8
Posterior cerebral artery	6
Cerebellar arteries	7
Basilar artery	2
Internal carotid artery	1
Anterior choroidal artery	1
Total	46

vessels, replacement of tissue by the lesion, or obstruction to the flow of the cerebrospinal fluid. Four cases were considered to have no neurological deficit on examination. Fifteen patients had a hemiparesis or hemiplegia secondary to 1) hemorrhage with or without an intracerebral hematoma; 2) thrombosis in the vessels of the malformation; or 3) a malformation located in the posterior frontal area. Eight of the fifteen cases with paralysis also exhibited a cortical type sensory defect on the contralateral side. Twelve cases had increased intracranial pressure as a result of obstruction to the flow of cerebrospinal fluid or from the effect of an intracerebral hematoma. Eight cases had cranial nerve palsies, with six of these being a unilateral abducens paresis. Five of the latter had evidence of increased intracranial pressure, and the paresis was probably due to involvement of the abducens nerve throughout its long intracranial course. The other cases with abducens paresis had a malformation in the posterior fossa which probably encroached directly upon the brain stem or upon the nerve itself. Two cases exhibited other cranial nerve involvement, one being oculomotor paralysis and the other olfactory nerve damage. The former was associated with increased intracranial pressure and the latter was a sequel of a large bifrontal malformation with direct pressure upon the olfactory nerves. Seven cases were admitted in coma following a hemorrhage. Another was comatose as a result of status epilepticus. Five cases had hemianopsia, all five had a lesion in the occipital or temporal lobes with direct involvement of the optic pathways. Three cases exhibited cerebellar dysfunction, and all three had malformations in the cerebellum. As previously mentioned, eight cases showed evidence of mental deterioration. Hydrocephalus was seen in two of the children, both having obstruction of the aqueduct of Sylvius. There were associated congenital abnormalities in two cases; one with a lumbar myelomeningocele and the other with a large port-wine nevus over the occiput.

One patient with a large lesion located in the third ventricle had severe hypopituitarism.

Laboratory Data

The polycythemia often observed secondary to an arteriovenous fistula elsewhere in the body was not seen in this series. Likewise there was no tendency noted toward cardiac enlargement as a possible secondary effect of an overloaded circulation. Several authors[7,10] have mentioned that these lesions often produce calcification which can be seen on plain skull x-rays. Thirty-seven of the forty-one cases in this series had plain skull x-rays taken. The others expired within a few hours of admission before the x-rays could be obtained. Calcification of the malformation was observed in only two cases. One patient was 18 and the other 26 years old. Two cases showed evidence of increased intracranial pressure on the plain skull x-rays with erosion of the posterior clinoids. Two cases also were interpreted as exhibiting increased vascularity of the skull in areas which subsequently were proved to overlie the malformations. Air studies were obtained on several of the early cases in this series and were helpful only to the extent that they localized an intracerebral hematoma, except for one case where an intraventricular mass was seen. This proved later to be a tangle of abnormal vessels lying in the lateral ventricle. Carotid angiography was carried out on 26 cases, in addition two cases also had vertebral angiograms. Two of the carotid angiograms were technically unsuccessful. Only one angiogram which adequately demonstrated the intracranial vessels failed to show the malformation. Eight cases had bilateral carotid angiograms in order to study the arterial feeding vessels when the malformation was in the midline or to determine the extent of a bilateral lesion. One case had a hemiplegia immediately after an intracarotid injection of thorotrast. The malformation was demonstrated on the angiogram and was considered

inaccessible. The etiology of the hemiplegia is not clear. There were no untoward sequelae of angiography using diodrast. Postoperative angiograms were made in five cases. Three were performed to determine the effectiveness of operative occlusion of the feeding arteries; another because the patient remained quite disoriented after excision of the lesion; and the fifth because the patient continued to suffer from moderately severe headaches after surgery. The latter two cases showed complete excision of the malformation. In the first three done to demonstrate the condition of the feeding artery, one showed an estimated 90 per cent reduction in the malformation, another approximately 50 per cent reduction, and the third showed no filling of the lesion.

Therapy

The treatment of choice is total excision of the malformation. However, these lesions are sometimes the most formidable the neurosurgeon must attack. Pilcher[10] favors radical excision but adds that the surgeon must be able to recognize the limits of such possibilities and must have the courage to withdraw when faced with lesions beyond these possibilities. Procedures other than total excision have been offered which suggest the possibility of control of the malformation without the danger of uncontrollable hemorrhage or subsequent incapacitating neurological disability. Trupp and Sachs[13] have advocated obliteration of the abnormal vascular channels by lightly stroking the vessel walls with a low coagulating current. Norlen[8] has criticized this on the basis that these lesions are seldom entirely superficial and that the electrocoagulation does nothing about the deeper levels of the cone-shaped malformations. Occlusion of the feeding vessels has been performed with the hope that the abnormal channels will undergo clotting with resultant lessening of the danger of hemorrhage. Irradiation was proposed by Cushing[2] because at that time he found that these lesions were beyond any hope of radical extirpation but it is now generally agreed that the amount of x-ray necessary to obliterate the vessels produces excessive damage to the normal brain tissue (Bassett[1]).

Thirty cases were operated upon. Ten of the thirty were explorations only and had no definitive procedure because of the size or location of the malformation. Four of these ten are now dead. Two were operative deaths, another died of bronchopneumonia six years later. This patient had a severe hemiplegia and uncontrollable seizures throughout the entire survival period. The fourth patient died in status epilepticus three years after the exploratory operation. Of the six surviving cases, two are living three and six years, the former being asymptomatic and the latter having approximately one seizure every three months. The other four living after exploratory operations are total invalids, three have severe mental deterioration and one has a severe hemiparesis and global aphasia. The twenty cases receiving definitive surgery can be separated into a group having total excision and a group having occlusion of the feeding arteries with silver clips. There were fifteen patients with total removals. There were three operative deaths. Three more have died subsequently; the first $1\frac{1}{2}$ years postoperatively from a hemorrhage from a second malformation elsewhere in the brain, the second at $1\frac{1}{2}$ years following a nocturnal seizure, and the third two years after surgery from peritonitis after perforation of the sigmoid colon. The nine surviving cases have been followed for four months to eight years. Six are working and have minimal or no neurological deficit. Of the remaining three patients, one is still receiving rehabilitation because of a hemiparesis and is expected to return to school in the near future. The second has a moderately severe hemiparesis, is able to walk with a cane, but has not attempted any gainful occupation. The third case received extended hospitalization for tuberculosis, during which time he also was rehabilitated for a hemiparesis, and states that he has a job as a librarian beginning May 1, 1955. There were five

cases in which the feeding vessels were clipped. There have been no deaths in this group. They have been followed for three months to six years. Four are considered good results with no residuals except one who has a mild hemiparesis. All four are working. The fifth case still has, after six years, severe mental deterioration which was present prior to surgery.

The entire group receiving definitive surgery (20 cases) showed an overall mortality rate of thirty per cent with the operative mortality one-half of this total mortality. In evaluating the fourteen surviving cases, it was found that ten (71%) had a good result, three (21%) are fair, and one (8%) is poor. Of the group operated upon but not receiving definitive surgery (10 cases) there is an overall mortality of four cases (40%) again with the operative mortality one-half of the total mortality. Of the six surviving cases, only two (33%) are good results and the other four (67%) are invalids. Those who are still well continue to have the possibility of recurrent hemorrhage.

Of the original group of 41 patients, 11 had no operation. Seven of these eleven expired within forty-eight hours after admission to the hospital. All seven were moribund on admission and no diagnostic or therapeutic procedures were feasible. Of the remaining four, one died on the twenty-seventh hospital day in status epilepticus without an antemortem diagnosis of arteriovenous malformation. Another died as a result of a subarachnoid hemorrhage two years after the diagnosis of an inoperable lesion was made. Of the non-operated group, one patient has been followed for four years and has a severe hemiparesis, aphasia and marked mental deterioration. The other case exhibiting marked mental deterioration at the time of ther release from the hospital in 1943, has been lost to follow-up.

Discussion

From the information derived from this series several points of interest arise. The greater incidence in males, as noted by Olivecrona[9] and Dandy[3] is partially

Chart 8

EVENTUAL OUTCOME

Type of procedure	operative deaths	death from malformation	death - other cause	Alive working	Alive invalid
Exploration only (10)	2	1	1	2	4
Total excision (15)	3	2	1	8	1
Obliteration of arterial supply (5)	0	0	0	4	1
No operation (11)	-	7 (immed.) 2 later			1 1(?)
Total (41)	5	12	2	14	8

supported by our figures. The symptom complex which MacKenzie[6] feels is strongly diagnostic was seen rarely in our series. MacKenzie states that a history of focal seizures followed by a subarachnoid hemorrhage or focal seizures and subsequent sudden severe hemiparesis without any evidence of hemorrhage is highly suggestive of the diagnosis. In our series, ten patients had seizures as an initial complaint with only four having an eventual

hemorrhage. Four others had a severe hemiparesis after the onset of seizure. Thus, only eight cases have a diagnostic history by this criteria of MacKenzie's. Certainly such a historical sequence is helpful, but in our experience, it appears too infrequently to depend upon it for diagnosis. The sequence of hemorrhage followed by seizures observed in five cases of our series is not at all diagnostic since the cortical irritation from subarachnoid bleeding from any source may initiate the seizures. The history of atypical migraine mentioned by MacKenzie[6] to be frequent in the presence of arteriovenous malformations was seen in only one of our forty-one cases. A history of subarachnoid hemorrhage and a bruit heard on auscultation of the head is strongly suggestive, but only six of our cases were recorded as having an audible bruit. There is no entirely reliable explanation why certain large and superficial lesions do not produce a bruit. The probable source of the sound is from the effects of the currents produced by swirling and eddying of the blood in the abnormal channels. Possibly the configuration of the vessels in some arteriovenous malformations is such that the necessary currents are not produced. The use of the electronic stethoscope in these patients may possibly elicit bruits not formerly audible with the conventional stethoscope. Calcification in these malformations is infrequently seen on plain skull x-rays. Evidently there is no increasing tendency for these lesions to calcify with age since calcification was seen in two cases eighteen and twenty-six years of age but not in any older cases.

The mortality figures from hemorrhage are less than the commonly accepted figures of forty per cent for rupture of an intracranial aneurysm (French and Blake[4]). However, disability from uncontrollable seizures, marked mental deterioration, severe hemiparesis or aphasia in addition to the twenty per cent chance of hemorrhage makes it imperative that every possible attempt be made to remove or obliterate these malformations. The fact that ten of these forty-one cases were explored and the lesion felt to be inoperable indicates that these lesions may present a most formidible problem in the treatment. Some of these ten were operated early in the series when angiography was not extensively employed and an arteriovenous malformation was unexpectedly exposed without knowledge of its extent or feeding arteries. In addition, the possibility of multiple malformations as seen in two cases presents a major problem in therapy.

There were three cases of arteriovenous malformations with concomitant aneurysms which were not included in this report since it was not possible to determine which lesion was the cause of the symptoms. The possibility of multiplicity of these malformations or of associated aneurysms provides a strong indication for complete preoperative angiographic study. Angiography also provides information of inestimable value for planning the surgical attack on the malformation. The use of combined hypotension and hypothermia at the time of operation may, through control of hemorrhage and protection of nervous tissue should temporary occlusion of the intracranial circulation become necessary, render more lesions operable in the future.

Conclusion

A series of forty-one verified arteriovenous malformations of the brain has been studied and their clinical features and treatment compared with other reports in the literature. The symptom complexes seen in this disease are infrequently diagnostic. Often this type of lesion is not even suspected clinically. Final diagnosis or verification of the diagnosis is permitted by intracranial angiography. The prognosis of untreated arteriovenous malformations is very poor, and radical surgical excision or obliteration should be carried out whenever feasible.

REFERENCES

1. Bassett, R. C.
"Surgical Experiences with Arterio-

venous Anomalies of the Brain"
J. Neurosurg., 8:59-74, 1951.

2. Cushing, H. and Bailey, P.
"Tumors Arising From the Blood Vessels of the Brain"
Springfield, Ill., 1928, Charles C. Thomas, Publisher.

3. Dandy, W. E.
"Arteriovenous Aneurysms of the Brain"
Arch. Surg., 17:190, 1928.

4. French, L. A. and Blake, P. S.
"Subarachnoid Hemorrhages and Intracranial Aneurysms"
Journal-Lancet, 70:459-466, 1950.

5. French, L. A. and Peyton, W. T.
"Vascular Malformations in the Region of the Great Vein of Galen"
J. Neurosurg., Vol. XI, No. 5, pp. 488-498, 1954.

6. MacKenzie, I.
"The Clinical Presentation of the Cerebral Angioma"
Brain, 76:184-214, 1953.

7. Noran, H. H.
"Intracranial Vascular Tumors and Malformations"
Ph. D. Thesis, University of Minnesota, pp. 59-71, 1944.

8. Norlin, G.
"Arteriovenous Aneurysms of the Brain"
J. Neurosurg., 6:475-494, 1949.

9. Olivecrona, H. and Riives, J.
"Arteriovenous Aneurysms of the Brain"
Arch. Neurol. and Psych., 59:567-602, 1948.

10. Pilcher, C. et al
"The Surgical Treatment of Intracranial Angiomatous Malformations"
Surgery, 28:342-349, 1950.

11. Ray, B. S.
"Cerebral Arteriovenous Aneurysms"
Surg., Gyn., Obstet., 73:613-648, 1941.

12. Sugar, O.
"Pathological Anatomy and Angiography of Intracranial Vascular Anomalies"
J. Neurosurg. 8:3-22, 1951.

13. Trupp, M. and Sachs, E.
"Vascular Tumors of the Brain and Spinal Cord and Their Treatment"
J. Neurosurg., 5:354, 1949.

II. MEDICAL SCHOOL NEWS

Coming Events

May 9 - 13	Continuation Course in Electrocardiography for General Physicians
May 10	Duluth Clinic Lectureship; "The Relationship of Achylia Gastrica to Pernicious Anemia;" Dr. William B. Castle, Professor of Medicine, Harvard University Medical School, Boston; Mayo Memorial Auditorium; 8:15 p.m.
May 11	Department of Medicine Annual Banquet; Calhoun Beach Club Hotel; 6:30 p.m.
May 12	Medical Six O'Clock Dinner; Coffman Memorial Union Ballroom; 6:30 p.m.
May 16 - 21	Continuation Course in Proctology for General Physicians
May 18	Minnesota Medical Alumni Faculty-Student Luncheon; Junior Ballroom, Coffman Memorial Union; 12:15 p.m.
May 23	Minnesota Medical Foundation Annual Luncheon; Junior Ballroom, Radisson Hotel; 12:30 p.m.
May 23 - 25	Minnesota State Medical Association Annual Meeting; Minneapolis Auditorium.
May 26 - 28	Continuation Course in Surgery for General Surgeons

* * *

Medical Six O'Clock

An annual event of real interest to all of us in the Medical School will be held on Thursday evening, May 12. The MEDICAL SIX O'CLOCK DINNER, sponsored by the Medical Inter-Fraternity Council, will take place in the Main Ballroom of Coffman Memorial Union beginning at 6:30 p.m. Featured speaker this year will be Dr. Philip S. Hench, Professor, Section of Medicine, Mayo Foundation, and Nobel Prize winner, who will give a talk entitled "X Marks the Spot". Master of ceremonies for the dinner will be Dr. Gilbert S. Campbell, Instructor, Department of Surgery, and an interesting evening is in store for all who attend.

Students, faculty members, and all friends of the Medical School are urged to obtain their tickets from the Dean's office or Inter-Fraternity Council representatives.

* * *

Foundation Luncheon

The Minnesota Medical Foundation will hold its annual luncheon meeting on Monday, May 23, in the Junior Ballroom of the Radisson Hotel in Minneapolis. The luncheon is held in conjunction with the annual meeting of the Minnesota State Medical Association which will take place from May 23 to 25.

The principal speaker at the luncheon this year will be Dr. Cecil J. Watson, Professor and Head, Department of Medicine, University of Minnesota Medical School, who has recently visited several countries in the Middle East under the auspices of the State Department. His subject will be "A Middle East Medical Journey."

All members of the Foundation, their families and friends, are cordially invited to attend the luncheon. Tickets are $2.25 and may be obtained from the office of the Secretary-Treasurer, 1342 Mayo Memorial.

* * *

(continued on next page)

Dr. Castle to Visit Campus

The Medical School will play host to a most distinguished scientist next week. Dr. William B. Castle, Professor of Medicine, Harvard University Medical School, Boston, will be the Duluth Clinic lecturer for 1955 and will be in Minnesota from May 9 to 13. The Duluth Clinic Lecture itself will be presented on Tuesday evening, May 10, in the Mayo Memorial Auditorium at 8:15. Dr. Castle's subject that night will be "The Relationship of Achylia Gastrica to Pernicious Anemia." Dr. Castle will lecture in Duluth on Thursday evening, May 12.

The Duluth Clinic Lectureship, an annual event sponsored by the Duluth Clinic, provides an opportunity for us to have outstanding scientists spend several days on our campus each year. During this time the lecturer delivers one or two formal lectures; and, perhaps even more important, he spends considerable time in the laboratories and in informal seminars where he may stimulate and encourage the younger members of our faculty in their programs of investigation.

* * *

Faculty News

Dr. Leo G. Rigler, Professor and Head, Department of Radiology, presented a lecture on "Roentgen Observations on Carcinoma of the Lung" at the Tenth Annual Convention of the Homer G. Phillips Hospital Internes Alumni Association in St. Louis on May 3.

Several members of the Student Health Service staff attended the annual meeting of the American College Health Association which was held in Colorado Springs, Colorado, from April 28 to 30. Those attending included Doctors Ruth Boynton, Donald Cowan, and Robert Hinckley, and Mr. Edward Dvorak and Mr. Richard Bond.

The recent meeting of the American College of Physicians which was held in Philadelphia from April 25 to 29 attracted a number of the members of the faculty of the Department of Medicine: Doctors Wesley W. Spink, F. W. Hoffbauer, Robert B. Howard, Herman J. Wolff, and Paul S. Hagen. Dr. Spink presented a paper entitled "Adrenocorticotrophic Hormone and Adrenal Steroids in the Management of Infectious Diseases" and also participated in a Clinical-Pathological Conference.

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Publications of the Medical School Faculty

Dempsey, M. E. and Wilson, R. H.: Normal Human Oxygen Saturation of the Arterial Blood as Determined by Gasometric Analysis. J. Lab. & Clin. Med., 43:791, 1954.

Flink, E. B.: Problems Arising in the Treatment of Adrenal Insufficiency with Cortisone and Hydrocortisone. Minn. Med., 37: 623, 1954.

Flink, E. B. and Zimmermann, Bernard: Fluid and Electrolyte Balance I. Basic Considerations. Minn. Med., 37: 545, 1954.

Flink, E. B.: Fluid and Electrolyte Balance. III. Medical Considerations. Minn. Med., 37: 715, 1954.

Schultz, A. L. and Sandhaus, Sol: Clinical Value of the Plasma Butanol-Extractable (Thyroxine) I^{131} in the Diagnosis of Hyperthyroidism and Myxedema. J. Clin. Endocrinology and Metabolism, 14: 1062, 1954.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

May 9 - 14, 1955

Monday, May 9

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - Tumor Conference; Doctors Hitchcock, Zimmermann, and Stenstrom; Todd Amphitheater, U. H.

11:30 - 12:30 Physical Medicine and Rehabilitation Staff Seminar; Rating Scale for the Psychiatric Patient in O. T.; Peter Briggs; Heart Hospital Theater.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - Physiology Seminar; Effect of Total Body Irradiation on Electrolyte Balance; W. O. Caster; 214 Millard Hall.

1:00 - 2:00 Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler; Todd Amphitheater, U. H.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker, and Staff; U. H.

1:30 - 3:30 Dermatology Hospital Rounds; H. E. Michelson and Staff; Dermatology-Histopathology Room, C-394, Mayo Memorial.

4:00 - 6:00 Anesthesiology Conference; F. H. Van Bergen and Staff; Todd Amphitheater, U. H.

4:30 - Public Health Seminar; National Health Developments in Finland; Miss Armi Hallsten-Kallia; 100 Mayo Memorial.

4:30 - Pediatric-Medicine Infectious Disease Rounds; Stations 33, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Ancker Hospital

8:00 - 9:00 Pediatric Contagion Rounds; Richard Lein; Contagion 5.

8:30 - 10:30 Medical and Surgical Chest Conference; Dr. Gehlen and Staff; Auditorium.

9:30 - 12:00 Visiting Staff Rounds.

10:00 - 12:00 Surgery Grand Rounds; Begin Floor E4.

11:00 - 12:00 Pediatric Rounds; Harry Orme; Contagion 1.

12:30 - 2:30 Surgery Out-Patient Clinic; Room 8.

Monday, May 9 (Cont.)

Ancker Hospital (Cont.)

2:00 - 3:00 Routine EKG Interpretation; Dr. Sommers and House Staff; Medical Record Library.

2:30 - 3:00 Discussion of Problem Case; Auditorium.

3:00 - 4:00 Surgery Journal Club; Classroom.

3:00 - 4:00 Lectures on Electrocardiography; Ben Sommers; Auditorium.

4:00 - 5:00 Medical Clerk Journal Club; Auditorium.

Minneapolis General Hospital

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station 11.

10:30 - Orthopedic and Fracture Rounds; Drs. John Moe and O. J. Campbell; Station 20.

11:00 - Pediatric Case Discussions; Erling Platou; Station 8.

12:30 - Surgery Grand Rounds; O. J. Campbell, Station 21.

1:30 - 2:30 Tuberculosis Conference; J. A. Myers; Station 8.

2:00 - Pediatric Rounds; William Krivit; Stations 4, 5, & 6.

Veterans Administration Hospital

9:30 - Infectious Disease Rounds; Drs. Hall, Zinnemann, and Doe.

1:30 - Cardiac Conference; Drs. Smith, J. Brown, Hoseth, Simonson, and Farquhar; Conference Room, Bldg. I; Rounds immediately following conference.

Tuesday, May 10

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; Samuel Feinberg, John A. Anderson and Staffs, Eustis Amphitheater, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 104 Jackson Hall.

12:30 - 1:30 Physiological Chemistry Seminar; Magnetism and Molecules - From Fundamentals to Hemoglobin; Everett Dulit; 214 Millard Hall.

12:30 - Anatomy Seminar; Experimental Hepatic and Renal Necrosis in the Rat; Joel G. Brunson; 226 Jackson Hall.

3:30 - General Physiology Seminar; 323 Zoology Building.

3:30 - Pediatric Seminar; Certain Aspects of Diphtheria; Dr. Bridges; 1450 Mayo Memorial.

4:00 - 5:00 Pediatric Rounds on Wards; John A. Anderson and Staff; U. H.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

Tuesday, May 10 (Cont.)

Medical School and University Hospitals (Cont.)

5:00 - 6:00 X-ray Conference; Presentation of Cases from Ancker Hospital; Drs. Aurelius and Clemett; Eustis Amphitheater, U. H.

*8:15 p.m. Duluth Clinic Lectureship; "The Relationship of Achylia Gastrica to Pernicious Anemia;" Dr. William B. Castle, Professor of Medicine, Harvard University Medical School, Boston; Mayo Memorial Auditorium.

Ancker Hospital

8:00 - 9:00 Pediatric Rounds; Dale Cumming; Contagion 1.

9:00 - 10:30 Visiting Staff Rounds.

9:00 - 12:00 Practical Diagnostic Clinic; Harry Orme; Out-Patient Department.

11:00 - 12:00 Medical X-ray Conference; J. R. Aurelius; Auditorium.

2:30 - 4:00 Routine EKG Interpretations; Resident Staff.

4:00 - 5:00 Medical-Pathological Conference; W. F. Mazzitello, Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Elizabeth Lowry and A. Bridge; Station 5.

10:00 - Cardiac Rounds; Paul F. Dwan; Classroom, Station 4.

10:00 - Psychiatry Grand Rounds; R. W. Anderson, Station 3.

12:30 - 2:30 Dermatology Rounds on Clinic; Carl W. Laymon and Staff.

1:00 - Tumor Clinic; Drs. Eder, Coe, and Lipschultz; Classroom.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Surgical Conference Room, Bldg. 43.

8:30 - Hematology Rounds; Drs. Hagen and Wexler.

8:30 - Surgery Journal Club; Conference Room, Bldg. I.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery-Tumor Conference; D. Ferguson and J. Jorgens.

1:00 - Review of Pathology, Pulmonary Tuberculosis; Conference Room, Bldg. I.

1:30 - Combined Medical-Surgical Chest Conference; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

4:00 - Thoracic Surgical Problems; Conference Room, Bldg. I.

5:00 - Fluid Balance Conference; Conference Room, Bldg. I.

5:30 - Physiology Seminar; Surgical Conference Room, Bldg. 43.

Wednesday, May 11

Medical School and University Hospitals

11:00 - 12:00 Pathology-Medicine-Surgery-Pediatrics Conference; Todd Amphitheater, U. H.

Wednesday, May 11 (Cont.)

Medical School and University Hospitals (Cont.)

12:30 - 1:30 Radioisotope Seminar; Mr. Robert Salmon; Betatron Room in Cobalt Underground Section, U. H.

1:00 - 2:00 Dermatology Clinical Seminar; F. W. Lynch; 300 North Clinic.

1:30 - 3:00 Pediatrics Allergy Clinic; Albert V. Stoesser and Lloyd Nelson; W-211, U. H.

3:30 - 4:30 Dermatology-Pharmacology Seminar; 3rd Floor Conference Room, Heart Hospital.

4:30 - 5:50 Dermatology-Infectious Disease Seminar; 3rd Floor, Conference Room, Heart Hospital.

5:00 - 6:00 Radiology Residents' Lecture; Congenital Heart; Joseph Jorgens; Todd Amphitheater, U. H.

5:00 - 5:50 Urological-Pathological Conference; C. D. Creevy and Staff; A503, Mayo Memorial.

5:30 - 7:30 Dermatology Journal Club and Discussion Group; Hospital Dining Room.

7:30 - 9:30 Dermatology Seminar; Review of Interesting Slides of the Week; Robert W. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; J. Noble; Auditorium.

11:00 - 12:00 Pediatric and Contagion Rounds; Harry Orme; Contagion 1.

11:00 - 12:00 Medicine Resident Rounds; W. F. Mazzitello.

3:30 - 4:30 Pediatric Surgery Conference; Harry Orme; Auditorium.

Minneapolis General Hospital

8:30 - 9:30 Obstetrical and Gynecological Grand Rounds; William P. Sadler and Staff; Station 30.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station 31.

11:00 - Pediatric Rounds; Erling Platou and Richard Raile; Station 6.

12:30 - Pediatrics Staff Meeting; Classroom, Station 4.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Surgical Conference Room, Bldg. 43.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

9:00 - Gastro-Intestinal Rounds; Drs. Wilson, Zieve, Ferguson, Brakel, Vennes, Nesbitt and Sadoff.

10:30 - Psychosomatic Conference; C. K. Aldrich; 7th Floor, Bldg. 43.

12:30 - Medical Journal Club; Doctors' Dining Room.

12:30 - X-ray Conference; J. Jorgens; Conference Room, Bldg I.

Wednesday, May 11 (Cont.)

Veterans Administration Hospital (Cont.)

1:30 - 3:00 Metabolic Disease Conference; Drs. Flink and Shapiro.

3:30 - Urology Pathology Slide Conference; Dr. Gleason; Conference Room, Bldg. I.

7:00 - Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, May 12

Medical School and University Hospitals

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Room 3.148 Mayo Memorial.

11:00 - 12:00 Cancer Clinic; K. Stenstrom, B. Zimmermann; Todd Amphitheater, U. H.

12:30 - 1:30 Physiology Seminar 210; Transport; Selected Topics in Advanced Permeability; Nathan Lifson; 214 Millard Hall.

12:30 - 1:30 Endocrine Seminar; Studies on Hypoglycemic States; Irvine McQuarrie; 271 Lyon Laboratories.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

4:00 - 5:00 Anesthesiology Seminar; F. H. Van Bergen and Staff; Room 100, Mayo Memorial.

5:00 - 6:00 Radiology Seminar; Discography in Evaluation of Lumbar Disc Lesions; Jack Friedman and M. Z. Goldner; Eustis Amphitheater, U. H.

*6:30 p.m. Medical Six O'Clock Dinner; Coffman Memorial Union Ballroom.

7:30 - 9:30 Physiology 211 Seminar; Selected Topics in Heart and Circulation; Hemodynamics; M. B. Visscher and Robert Evans; 271 Lyon Laboratories.

Ancker Hospital

8:00 - 9:00 Pediatric Clinical Staff Conference; Contagion Classroom.

9:00 - 10:00 Pediatric Contagion Rounds; Alexander Stewart, Contagion 5.

9:30 - 10:30 Medical Grand Rounds; Auditorium; Visiting Staff Rounds immediately following Grand Rounds.

11:00 - 12:00 Pediatric X-ray Conference.

11:00 - 12:00 Medicine Resident Rounds; W. F. Mazzitello.

2:00 - 3:00 Routine ECG Interpretation; Ben Sommers; Medical Record Library.

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station 4.

10:00 - Psychiatry Grand Rounds; R. W. Anderson and Staff; Station 3.

11:30 - 12:30 Clinical Pathological Conference; John I. Coe; Classroom.

12:30 - 2:30 Dermatology Rounds and Clinic; Carl W. Laymon and Staff.

1:00 - Fracture X-ray Conference; Drs. Campbell and Moe; Classroom.

Thursday, May 12 (Cont.)

Veterans Administration Hospital

8:00 - Experimental Surgery Laboratory Meeting; Conference Room, Bldg. I.

8:30 - Hematology Rounds; Drs. Hagen and Duryea.

9:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

9:00 - Surgery Ward Rounds; D. Ferguson and Staff; Ward 11.

11:00 - Surgery-Roentgen Conference; J. Jorgens; Conference Room, Bldg. I.

1:00 - Infectious Disease Conference; Conference Room, Bldg. I. (Rounds immediately following conference).

4:00 - 5:00 Seminar on Radioisotopes in Medicine; Radiological Safety; Conference Room, Bldg. I.

Friday, May 13

Medical School and University Hospitals

8:00 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

11:00 - 12:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Eustis Amphitheater, U. H.

11:45 - 12:50 University of Minnesota Hospitals Medical Staff Meeting; Glaucoma; Richard C. Horns; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

1:00 - 2:00 Physiology Seminar 212; Selected Topics in Respiration: Respiratory and Circulatory Effects of Hypothermia; E. B. Brown; 214 Millard Hall.

1:30 - 2:30 Dermatology Grand Rounds; Presentation of Cases from Grouped Hospitals (University, Ancker, General and Veterans) and Private Offices; H. E. Michelson and Staff; Eustis Amphitheater, U. H.

2:30 - 4:00 Dermatology Hospital Rounds; H. E. Michelson and Staff; Begin at Dermatological Histopathology Room, C-394 Mayo Memorial.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

3:30 - 4:30 Dermatology-Physiology Seminar; 3rd Floor Conference Room, Heart Hospital.

4:00 - 5:30 Chest X-ray Conference; Chest Staff and Charles Nice; Todd Amphitheater, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hanson and Staff; E-534, U. H.

5:00 - Urological Seminar and X-ray Conference; A-503, Mayo Memorial.

Ancker Hospital

8:00 - 9:00 Pediatric Rounds; Charles Steinberg, Contagion 1.

Friday, May 13 (Cont.)

Ancker Hospital (Cont.)

10:30 - 11:30 Pediatric Contagion Rounds; Richard Smith; Contagion 1.

11:00 - 12:00 Contagion Rounds; Harry Orme; Contagion 5.

2:00 - 3:00 Routine EKG Interpretation; Resident Staff.

3:00 - 4:00 Medical-Surgical-Pathological Conference; Auditorium.

4:00 - 5:00 Medical Journal Club; Conference Room, E5.

4:00 - 5:00 X-ray Surgery Conference; Auditorium.

Minneapolis General Hospital

10:00 - Otolaryngology Conference; Robert A. Priest, Large Classroom.

10:30 - Pediatric Surgical Conference; Tague Chisholm and B. Spencer; Classroom, Station 4.

12:00 - Surgery-Pathology Conference; Drs. Campbell and Coe; Classroom.

1:00 - 2:00 ECG Conference; Boyd Thomes and Staff; Classroom, Station 4.

2:00 - 4:00 Clinical-Medical Conference; Thomas Lowry; Classroom, Station 8.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

11:00 - 12:30 Psychiatry Case Conference; Werner Simon; Psychiatry Department, VA Hospital Annex.

12:30 - Urology X-ray Conference; X-ray Department.

1:00 - Autopsy Conference; E. T. Bell; Conference Room, Bldg. I.

2:00 - Pathology Slide Conference; E. T. Bell; Conference Room, Bldg. I.

Saturday, May 14

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 9:30 Pediatric Grand Rounds; Eustis Amphitheater, U. H.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; Alexander R. Margulis, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:00 Obstetrics and Gynecology Rounds; J. L. McKelvey and Staff; Station 44, U. H.

10:00 - 12:00 Otolaryngology Seminar on Current Literature; L. R. Boies and Staff; Todd Memorial Room, A-675 Mayo Memorial.

Saturday, May 14 (Cont.)

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

9:30 - 11:00 Medicine Grand Ward Rounds; W. F. Mazzitello.

11:00 - 12:00 Medical Clerk Case Conference; W. F. Mazzitello.

Minneapolis General Hospital

8:00 - Urology Staff Conference; T. H. Sweetser; Main Classroom.

9:00 - Psychiatry Grand Rounds; R. W. Anderson; Station 3.

9:30 - Pediatrics Rounds on all Stations; R. B. Raile.

11:00 - 12:00 Medical X-ray Conference; O. Lipschultz, Thomas Lowry and Staff; Main Classroom.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - Medical X-ray Conference; Conference Room, Bldg. I.

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* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.