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Cerebral Angiography

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I.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

February 27 - March 5, 1949

No. 237

Sunday, February 27

9:00 - 11:30 Surgery Grand Rounds; Station 22, U. H.
Pancreatitis; William Rogers; Rm. M-109, U. H.

Monday, February 28

8:00 - Fracture Rounds; A. A. Zierold and Staff; Ward A. Minneapolis General Hospital.

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; M-109, U. H.

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

11:00 - 11:50 Roentgenology-Medicine Conference; Staff; Veterans Hospital.

11:00 - 11:50 Physical Medicine Seminar; E-101, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.

12:00 - 1:00 Physiology Seminar; Permeability of Insect Skib; Glenn A. Richards; 214 M. H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:20 Pathology Seminar; The Diagnosis of Carcinoma of the Stomach by Examination of Gastric Washings; Warren C. Hunter; 104 I. A.

12:30 - 1:30 Surgery Problem Case Conference; A. A. Zierold, C. Dennis and Staff; Small Class Room, Minneapolis General Hospital.

1:30 - 2:30 Surgery Grand Rounds; A. A. Zierold, C. Dennis and Staff; Minneapolis General Hospital.

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

4:00 - 6:00 School of Public Health Seminar; Some Nursing Problems of Foreign Countries; Elizabeth M. Brackett, International Health Division, Rockefeller Foundation; 113 MeS.

*4:00 - 6:00 Kellogg Lecture; Immunity and Hypersensitivity; D. R. Mathieson, Mayo Clinic; Powell Hall Amphitheater.

* 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.

4:00 - Pediatric Seminar; Brain Tumors; Adrian Jensen; 6th Floor, Child Psychiatry, U. H.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; D. Creevy and H. M. Stauffer and Staffs; M-109, U. H.

Tuesday, March 1

8:30 - 10:20 Surgery Reading Conference; Small Conference Room, Bldg. I, Veterans Hospital.

9:00 - 9:50 Roentgenology Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and Robert Hebbel; Veterans Hospital.

12:30 - 1:20 Pathology Conference; Autopsies; Pathology Staff; 102 I. A.

1:00 - 2:30 X-ray-Surgery Conference; Auditorium, Ancker Hospital.

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

*2:00 - 4:00 Kellogg Lecture; Rickettsial Diseases; Herald R. Cox, Lederle Laboratories, Pearl River, N. Y.; Todd Amphitheater, U. H.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans Hospital.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:00 - 5:30 Physiology-Surgery Conference; Experimental Heart Failure; C. Lillehei & J. R. Bobb; Eustis Amphitheater, U. H.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Dr. Lipschultz & Staff, General Hospital; Powell Hall Amphitheater.

Wednesday, March 2

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515, U. H.

8:30 - 9:30 Clinico-Pathological Conference; Auditorium, Ancker Hospital.

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans; Room 1AW, Veterans Hospital.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker and Joe R. Brown; Veterans Hospital.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

12:00 - 12:50 Radio-Isotope Seminar; Blood Volume and Tissue Volume Studies with Radio-Sodium and Radio-Chlorine; J. C. Wang; Rm. 212 Hospital Court, Temporary Bldg.

3:30 - 4:30 Journal Club; Surgery Office, Ancker Hospital.

4:00 - 5:00 Infectious Disease Rounds; Powell Hall Amphitheater.

Thursday, March 3

8:15 - 9:00 Roentgenology-Surgical-Pathology Conference; Craig Freeman and H. M. Stauffer; M-109, U. H.

8:30 - 10:20 Surgery Grand Rounds; Lyle Hay and Staff; Veterans Hospital.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-109, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:50 Surgery-Radiology Conference; Daniel Fink and Lyle Hay; Veterans Hospital.

11:00 - 11:50 Urology Seminar; The Technique of Biopsy of the Liver and Its Result; Robert Green; E-101, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

11:30 - 12:30 Clinical Pathology Conference; Steven Barron, C. Dennis, George Fahr, A. V. Stoesser and Staffs; Large Class Room, Minneapolis General Hospital.

12:00 - 1:00 Physiological Chemistry Seminar; The Nature of the Circulating Thyroid Hormone; Eleanor Berman; 214 M. H.

1:00 - 1:50 Fracture Conference; A. A. Zierold and Staff; Minneapolis General Hospital.

2:00 - 3:00 Errors Conference; A. A. Zierold, C. Dennis and Staff; Large Class Room, Minneapolis General Hospital.

4:00 - 5:00 Bacteriology and Immunology Seminar; Rickettsial Infections; Herald R. Cox, Lederle Laboratories, Pearl River, N. Y.; 214 M. H.

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

5:00 - 6:00 X-ray Seminar; Anomalies of the Aorta; Barnard Hall; Todd Amphitheater.

Friday, March 4

8:30 - 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.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:20 Medicine Grand Rounds; Staff; Veterans Hospital.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:00 - 12:00 Surgery-Pediatric Conference; C. Dennis, O. S. Wyatt, A. V. Stoesser and Staffs; Minneapolis General Hospital.

11:30 - 12:50 University of Minnesota Hospitals General Staff Meeting; Carcinoma of the Breast--An Analysis of 626 Cases Referred for Roentgen Therapy; Harvey W. Stone and Halvor Vermund; Powell Hall Amphitheater.

12:00 - 1:00 Surgery Clinical Pathological Conference; Clarence Dennis and Staff; Large Classroom, Minneapolis General Hospital.

1:00 - 1:50 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

1:00 - 3:00 Pathology-Surgery Conference; Auditorium, Ancker Hospital.

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

4:00 - 5:00 Electrocardiographic Conference; George N. Aagaard; 106 Temp. Bldg,, Hospital Court, U. H.

Saturday, March 5

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; Station 20, U. H.

8:30 - 9:30 Surgery Conference; Auditorium, Ancker Hospital.

8:00 - 9:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor, West Wing, U. H.

8:00 - 9:00 Surgery Literature Conference; Clarence Dennis and Staff; Minneapolis General Hospital, Small Classroom.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-101, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 11:30 Surgery-Roentgenology Conference; Movies; Todd Amphitheater, U. H.

9:00 - 12:00 Neurology Conference; Veterans Hospital Annex, Fort Snelling.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:00 - 12:00 Anatomy Seminar; Effects of Androgen upon Castrated and Normal Fetuses, Rachel L. Fralick; Cytology of the Pancreatic Islet Cells, Lewie O. Ingersoll; 226 I. A.

II. CEREBRAL ANGIOGRAPHY

Lyle A. French
Paul S. Blake

Cerebral angiography is the procedure in which the intracranial vascular system is visualized roentgenographically following the intracarotid injection of radiopaque dyes. The procedure was first used in 1927 by Egaz Moniz[28], a Portuguese neurologist. It was not immediately accepted nor extensively used until the few years before World War II when it received widespread acceptance in Europe. It became accepted in this country after the war. Because of the inherent toxicity of the dyes originally used, the risk of the procedure did not at first seem to be worth the added information obtained, but after the pioneers demonstrated the great utility of the procedure and introduced less toxic dyes, cerebral angiography as an accessory diagnostic aid in intracranial lesions came into frequent general use. Included in this report are the experiences with angiography at the University Hospitals and Veterans Hospital, Minneapolis.

Before Moniz used angiography in his clinic, he experimented extensively with animals and cadavers. He used many bromine, strontium, and iodine salts before finally selecting a 25% solution of sodium iodide for use on his patients. This drug was not ideal because it could not be sterilized and it had to be freshly prepared before injection. There were also many unpleasant and alarming reactions from it such as pain, headaches, convulsions, and temporary hemiplegias. Using sodium iodide, Moniz had an overall mortality of 2%. In 1931 Löhr and Moniz simultaneously began using thorotrast, a 25% colloidal suspension of thorium dioxide which has the same viscosity as blood. This drug caused few, if any, immediate side effects and its use became generalized. However, thorotrast is radioactive. It is not excreted from the body but is picked up and retained by the reticulo-endothelial system. Some concern over the radiation effect was felt since sarcomas in rats could be caused by the injection of the drug, but those who used it felt that by restricting the amount of thorotrast to 30 cc., the long term toxicity of the drug would be negligible. No ill effects ascribable to the use of thorotrast were reported by the early workers[4,24]. However, Jacobson and Rosenbaum[15] have reported extensive fibrosis in the reticulo-endothelial system several years following its injection, and MacMahon, Murphy, and Bates[27] reported a patient who developed a sarcoma of the liver twelve years after the injection of thorotrast. Because of this radioactivity, thorotrast is used less and less frequently for cerebral angiography. The mortality after angiography with thorotrast is 1.3%[7].

Haussler[12], in 1940, reported that ethyl triiodo stearate offers good contrast, is excreted rapidly, and has little toxicity when used in angiography. Un fortunately, the drug is not available in this country.

Torkildson and Engeset[5] in Sweden, and Gross[10] in this country began the use of diodrast in 35% solution. This dye fills the smaller vessels better than thorotrast but does not give the excellent roentgenographic contrast obtained with thorotrast. Most patients experience some mild reactions to its injection, such as a murmuring sound, a flushing of the face or pain over the side of the head. Very occasionally, demented and confused patients react violently after its injection, and occasionally convulsions have occurred[5]. These severe side effects are very infrequent, and 35% diodrast is the dye now most widely used. No deaths have been ascribed to diodrast reactions in cerebral angiography.

Technique of Injection

The procedure is done under nembutal sedation and procaine infiltrated locally; it is wise to infiltrate the carotid bifurcation thoroughly with novocain solution to eliminate or abolish the carotid sinus reflex. The dye is in-

jected into the common carotid artery. In most clinics the artery is exposed and the puncture of the artery is made under direct vision. There is a definite trend toward inserting the needle percutaneously. In this clinic the procedure has been to attempt a percutaneous injection and if not successful to surgically expose the artery. Many surgical techniques for exposure of the artery have been described; in this clinic the common carotid is isolated by making a short transverse incision above the thyroid cartilage at the anterior edge of the sternocleidomastoid muscle. A seventeen or eighteen gauge needle is then inserted into the vessel. An attempt is made to push the point of the needle to the posterior part of the artery and then to turn the bevel of the needle posteriorly so that the majority of the dye, as it is injected, joins the stream of blood flowing up the internal carotid artery. A hard rubber tube with a three way stopcock is connected to the needle and a slow stream of saline is kept flowing through the needle until everything is in readiness when 10-15 cc. of the dye is forced into the carotid as rapidly as possible. Just before the injection is complete, an exposure is made to demonstrate the arterial tree, and three seconds later a second exposure is obtained in order to visualize the venous channels. The circulation time from common carotid to internal jugular through the internal carotid is about four seconds, and through the external carotid this time is about six seconds[23]. Some people slow this circulation down by compressing the jugular during the procedure[21], others by occluding the common carotid below the needle[2]. So far we have felt that these measures are unnecessary and complicate the procedure. By occluding the carotid Moniz reported that the anterior cerebral artery failed to fill in 30% of cases. Gross[11] does not occlude the artery and reports 100% filling of the anterior cerebral. The apparent reason for this is that lowering the pressure in one internal carotid allows blood to flow across the midline from the opposite side of the circle of Willis and this non-dye containing blood fills the anterior cerebral. Two injections are made to allow an arteriogram and venogram to be made in both antero-posterior and lateral positions. Dyes[2] and Timins[36] have a method whereby antero-posterior and lateral skull x-rays are made on the same injection. Our x-ray plates have a portable Bucky grid attached to them but are changed by hand. Caldas has described a radio carousel whereby six exposures are made in six seconds. Sanchez-Perez[35] has described a spring box which delivers up to three cassettes as fast as they can be removed. Variable x-ray technique has been employed. In this clinic we use long exposures, up to one and one-half seconds for the laterals and three seconds for the antero-posterior views with 20 ma. and 75 kv., because we use diodrast, a relatively poor contrast medium. These long exposures increase the chances of visualizing dye in all vessels. The total radiation per angiogram which is delivered to the operator's hands has been computed as .3R[5]. Some clinics doing angiography extensively have used lead shields to protect the operator's hands[5,29].

The technique of injection of the vertebral artery is similar to that of the carotid except for the site of injection. The vessel is visualized as it arises from the subclavian artery. It is the first branch and traverses in a cephalad direction. The needle is inserted as described above and 10-20 cc. of diodrast injected and roentgenograms made. Both lateral and antero-posterior views are obtained.

Angiographic Anatomy

The internal carotid artery is seen entering the skull and ascending to the infraclinoidal area where it bends sharply forward and backward in the cavernous sinus (the carotid siphon) and then ascends to its supraclinoidal portion which bends forward and divides into its two main branches, the anterior and middle cerebral arteries. These branches go in opposite directions - the anterior cerebral and its branches are on the

medial surface of the hemisphere and the middle cerebral artery after coursing through the Sylvian fissure divides into branches over the lateral surface of the hemisphere.

The first intracranial branch of the carotid is the ophthalmic artery which arises at the anterior knee of the carotid. The next branch is the anterior choroidal which arises as the carotid passes the clinoids. The anterior choroidal branch courses posteriorly in an almost horizontal plane. Sometimes the posterior communicating artery can be visualized and, in 15% of the cases, the posterior cerebral artery is visualized indicating that a part of its blood supply comes from the internal carotid.

The anterior cerebral artery, after reaching the medial surface of the hemisphere, divides into two principal branches which arch around the corpus callosum and continue posteriorly into the parietal area. These are the pericallosal and callosal marginal branches. There are smaller, frontopolar, branches which course anteriorly on the medial surface of the frontal lobe.

The first branch of the middle cerebral artery, after it reaches the lateral surface of the brain, is the ascending Rolandic - a candelabra shaped artery terminating in the rolandic area. Three branches of the middle cerebral course diagonally along the Sylvian fissure - (1) the post-temporal artery which has a curve, (2) the middle or angular artery, and (3) the post-parietal branch.

Interpretation of angiograms appears at first to be confusing, but if one remembers that all the main arteries are on the external surface of each hemisphere, and that changes in their position indicate changes in the shape of the surface of the brain, it is obvious how deflections in their course are used to localize brain tumors.

The venograms show the superior and inferior sagittal and the straight sinuses, the internal cerebral veins, the vein of Galen, and the great veins of Galen, and the great veins over the surface of the brain. These great veins over the surface are: (1) the ascending veins which flow into the sagittal sinus and, (2) the anastomosing veins of Trollard (anteriorly) and Labbe (posteriorly). Displacement of these veins may be of confirmatory value in the localization of intracranial masses[5,20].

When the vertebral system is injected, the vertebral artery is visualized as it traverses through the transverse foramina and then as it enters the skull. It then joins with the vertebral of the opposite side to form the basilar artery which is visualized lying on the anterior surface of the pons. The numerous pontine branches of the basilar are not visible. Occasionally the cerebellar branches can be distinguished. When the basilar artery reaches the circle of Willis, it divides into two branches, the posterior communicating and the posterior cerebral. The former is seldom visualized but the latter can be distinctly seen as it courses posteriorly and superiorly over the occipital lobe.

Venograms taken after injection of the vertebral artery may reveal the great vein of Galen, the straight and transverse sinuses, and the anastomotic veins entering the transverse sinus.

Clinical Interpretation

Aneurysms: The most frequent locations of aneurysms of the cerebral vessels are at the bifurcations of the large basal vessels, such as the junction of the internal carotid with the anterior or middle cerebral artery. Less frequent are aneurysms at the junction of the carotid with the anterior or with the posterior communicating artery or on the internal carotid artery itself.

With arteriography, both the location and size of the aneurysm may be ascertained. Occasionally the size of the aneurysm as seen in the angiogram is considerably smaller than that seen at the time of operation. This is due either to a clot of blood within the

aneurysm or to the neck of the sac being only partially filled with the dye. Occasionally the neck of the aneurysm may be so small that none of the contrast media enters the sac in which case the aneurysm is not visualized.

The internal carotid artery enters the cranial cavity through the foramen lacerum. It remains extradural until it reaches the level of the anterior clinoid process. Extradural aneurysms rarely burst because they are well protected by this overlying dural sheath. Clinically they resemble slowly growing neoplasms and must be differentiated from parasellar tumors. An aneurysm in this location (extradural portion of the internal carotid artery) encroaches on the third, fourth, fifth, and sixth cranial nerves so that various combinations of palsies of eye movements and hypaesthesia over the face occur. Occasionally direct pressure by the aneurysm on the optic chiasm or nerve results in visual abnormalities. The aneurysm may produce erosion visible in roentgenograms in the parasellar region. A definite differential diagnosis between aneurysm and parasellar tumor can usually be made preoperatively by angiography.

Aneurysms arising on the internal carotid artery or its branches, after the artery has pierced the dura at the level of the anterior clinoid process, may produce no symptoms until they rupture. They are usually located on the posterior aspect of the carotid siphon.

The surgical treatment of intracranial aneurysms consists of either occluding the stalk or neck of the aneurysm or by trapping the aneurysm by occluding the vessel both proximally and distally to the lesion. If the aneurysm is located below the origin of the posterior or anterior communicating arteries, the therapy of choice is ligation of the carotid artery in the neck which occludes the proximal flow of blood and then occluding the vessel intracranially distally to the aneurysm. Ligation of the carotid artery in the neck does not embarrass an aneurysm arising distally to the communicating vessels. Aneurysms in this location are best treated by an intracranial approach.

Brain Injuries: Löhr[24] has studied angiographically 1000 patients with skull fractures. He classifies brain injuries as (a) "commotio cerebri" and (b) "contusio cerebri." In the commotio cerebri group he found very narrow constricted vessels on the injured side; he interpreted this as due to brain swelling or possibly to vascular spasm. In the contusio cerebri group he observed an increased resistance to injection of the dye and that the vessels were broad and flat. Angiograms in patients with subdural or extradural hematomas revealed in the antero-posterior view, the vessels to be displaced away from the lateral wall of the skull. Löhr[24] and Kristiansen[19] states that all acute head injuries should have an angiogram - others[38,5], even though they are great exponents of the angiogram, believe that bilateral trephines are more logical in acute head injuries. Most investigators have agreed that in chronic posttraumatic states angiograms are indicated.

Brain Tumors: Brain tumors are localized by (1) displacement of the normal vessels supplying the brain, (2) enlargement of afferent vessels to a tumor or efferent vessels from a tumor area, (3) pathological changes of blood circulation within the tumor itself[5].

The arteries of the brain lie on the surface and normally take a tortuous course as they follow the cerebral convolutions. A space occupying tumor will tend to displace the vessels away from its center. Also as the tumor enlarges, the overlying cortical convolutions become flattened and the arteries become stretched and thin, separated from each other, and follow straight courses around the area of localized enlargement[22]. The cerebral veins may be displaced by the tumor mass or engorged due to the slowing of cerebral circulation

resulting from increased intracranial pressure, but the extreme variability of the venous drainage in normal brains makes their displacement only of confirmatory value in diagnosing space occupying lesions[5].

Tumors produce characteristic abnormalities visible in angiograms. The basic changes will be described briefly: many intermediate forms, of course, exist. In the antero-posterior view the ascending portion of the anterior cerebral artery is examined for displacement across the midline by a tumor lying laterally in the hemisphere. The horizontal portion of the anterior cerebral is then examined for possible elevation by a tumor mass such as a hypophyseal tumor lying beneath it. The horizontal portion of the middle cerebral artery may be elevated by tumors in the anterior portion of the temporal lobe. This is important in distinguishing these tumors from those in the posterior portion of the temporal lobe which do not disturb this portion of the middle cerebral artery[5,22]. The carotid siphon may be straightened or displaced indicating a lesion in the vicinity of this structure. In the lateral views an upward or downward displacement by a tumor of the pericallosal and Sylvian vessels may be visualized. This displacement occurs appropriate to the location of the tumor, either above or below the major vessels. Centrally located tumors cause direct lateral pressure and will not produce an appreciable shift in the position of these vessels. In the latter case, a stretching of the arteries with elimination of the frequent curves can be significant. Vessels may be displaced in a circular fashion around a tumor. If there are no vessels within the circle, a cyst or abscess or an astrocytoma must be considered[5].

Ventricular enlargement, either congenital or acquired, will cause straightening and stretching of all vessels but most noticeably those in the Sylvian group. In addition, the arch of the anterior cerebral artery around the ventricle may be widened[5,8,25]. The venogram may reveal the carotid veins to be small due to compression resulting from the increased intracranial pressure.

For the purposes of discussion, List[21] divides tumors into two groups: those located above and those located beneath the Sylvian fissure. Tumors located above the Sylvian fissure: If the tumor is located parasagitally in the frontal region, the lateral view may reveal downward and backward displacement of the anterior cerebral artery, but if the tumor is located well within the substance of the frontal pole, there may be only minimal changes characterized by tautness of the pericallosal branches and slight depression of the Sylvian group. In the anteroposterior view, lateral displacement of the anterior cerebral artery may be seen. Unless pathological tumor circulation or definite arching of vessels around a lesion are visualized, a more accurate diagnosis of frontal tumors is made by means other than angiography.

The accuracy of localization of tumors in the posterior frontal and frontoparietal regions by angiography is good. Tumors in these regions may depress the Sylvian group of vessels and localized vascular arches partially encircling the tumor may be visualized. Tumors localized to the Rolandic area may stretch and distort the callosomarginal artery. The pericallosal vessels as visualized in the lateral view are seldom displaced unless the lesion is located parasagittally. In posterior frontal or frontoparietal tumors, the antero-posterior view may show the anterior cerebral artery dislocated across the midline, but this is not as consistent as in tumors located more anteriorly. In anteriorly placed tumors this dislocation of the anterior cerebral artery is a smooth arc from the base of the skull to the inferior border of the falx cerebri, while in parietal and frontoparietal tumors the dislocation is apt to form an acute angle with the point located at the origin of the frontopolar artery[6]. The carotid siphon is often pushed downward and backward. Tumors located beneath the Sylvian vessels: Tumors located beneath the Sylvian

vessels elevate this group of vessels. If the tumor is in the tip of the temporal lobe (including meningiomas of the sphenoid wing) the first part of the middle cerebral artery may ascend sharply then arch backward in a plateau. In the antero-posterior view the horizontal part of the middle cerebral artery may rise at an angle to reach the convexity of the hemisphere instead of the normal horizontal course to the lateral wall of the skull. This displacement of the Sylvian vessels will not occur if the tumor is located further posteriorly.

The carotid siphon may be stretched by tumors in its vicinity. Distortion of the supraclinoidal part of the carotid, before it divides into the anterior and middle cerebral branches, is common in tumors of the suprasellar region or of either the frontal or temporal lobe. The infraclinoidal part is seldom disturbed except by tumors in the immediate locality, such as meningiomas of the medial third of the sphenoid wing and by hypophyseal tumors[29,5].

Tumors in the posterior part of the temporal lobe may elevate the first part of the Sylvian group of vessels in lateral views, although in antero-posterior views the horizontal part of the middle cerebral artery may not be displaced. The exact location may be shown by a concavity of the Sylvian vessels over the tumor. Occipital lobe tumors may produce a straight line elevation of the Sylvian group of vessels and occasionally displace the posterior extension of the pericallosal vessels.

The displacement of vessels as described above is a valuable method of localizing tumors, but the visualization of the tumor circulation not only more accurately defines the tumor's position but it may also make possible a histological diagnosis of the thpe of tumor. Engeset[5] believes that a more accurate histological diagnosis can be obtained by angiography than by frozen sections of the tumor. He feels sufficiently confident with angiography to ascribe a lesion as inoperable from its angiographic appearance.

Moniz was the first to describe variations in tumor circulation depending upon the histological type of tumor. He believes that gliomas do not disturb normal cerebral vessels because of their infiltrative nature but this has not been born out by reports of other investigators.

Glioblastoma multiforme: Tönnis was the first to describe the angiographic appearance of the glioblastoma multiforme. At operation he noticed arterial blood in the veins surrounding these tumors and in angiograms pointed out the frequency of intraneoplastic arteriovenous aneurysms. Because of the rapid circulation through these tumors due to these fistulae, tumor circulation may be visible in the arteriogram and not in the venogram. Several cases have been reported in which the tumor circulation of glioblastoma were seen on venograms[13,5], but the angiographic diagnosis of glioblastoma multiforme from a roentgenogram exposed so late that the normal venous channels are filled is precarious. The circulation of most other tumors will appear on venograms. The glioblastomas have an abundant vascular supply with the vessels irregularly arranged and of irregular calibre. They show irregular concentration of dye in the veins[5] and between the vessels are small irregular spots which may be miliary aneurysms. Large arteriovenous aneurysms and one or two abnormal veins leading away from the tumor area may be visible[13]. The circulation may vary from a faint spotted haze of weak contrast accumulation with small pinhead sized vessels which cannot be followed for over one centimeter to more pronounced vascular changes with huge loops of vessels two or three centimeters in length[26]. Occasionally vascular changes are present only at periphery of the tumor indicating extensive central necrosis. On the venogram in these cases there is either no dye left in the tumor area or only a light capillary haze.

Metastatic tumors: The distinction between glioblastoma multiforme and metas-

tatic carcinoma may, at times, be difficult but usually it can be made. Metastatic carcinomas, like glioblastomas, have an abundant vascular supply with frequent arteriovenous aneurysms and irregularity of vessels. The presence of normal vessels arranged annularly about the lesion with just a faint haze of circulation in the tumor in the arteriogram but with a maximum circulation within the tumor visible in the venogram is characteristic[5].

Astrocytomas: Astrocytomas seldom show any vascularity in either the arterial or venous phase. Some investigators believe that vascular lakes and intraneoplastic circulation visible in venograms is indicative of an astrocytoma, but most reports do not agree with this. According to Engeset, astrocytomas characteristically show a marked dislocation of normal vessels around the tumor with practically no vessels within the curved enclosed area. Vessels seen in the tumor are usually straight and even. Oligodendrogliomas, abscesses, cysts and cholesteatomas cannot be distinguished from astrocytomas angiographically, but with help of clinical symptoms and signs and plain roentgenograms, the diagnosis can usually be made.

Meningiomas: Meningiomas present a typical picture on the angiogram. They almost always receive part of their blood supply from the external carotid artery, and if this source of blood in the tumor can be demonstrated, it is practically pathognomonic. If the tumor gets all of its blood supply from the external carotid and the internal carotid alone has been injected, the appearance of the normal vessels circling around the xtracortical tumor is often diagnostic. In the arteriogram, when the tumor circulation is filled, enlarged vessels leading up to the tumor but ending in brush-like fans at the edge of the tumor may be seen.[8] The vessels are all of constant calibre and have constant dye concentration. Because the circulation through meningiomas is slow, the venogram reveals a diffuse accumulation of dye within the tumor sharply demarcating it from the surrounding brain tissue. Arteriovenous fistulas within the tumor are rare, and irregularities in size of vessels are not seen in meningiomas; if these features do occur in what otherwise appears to be a meningioma, a sarcomatous type of meningioma or a metastatic carcinoma must be considered. Furthermore, if there is pathological change visible in the vessels, there is the possibility of the tumor being a glioblastoma multiforme[5]. Gliomas, however, never receive their blood supply from the external carotid circulation which is of differential significance.

Vascular tumors: Angiomatous lesions may produce Jacksonian seizures or subarachnoid hemorrhages. On angiograms they may be confused with a glioblastoma because they contain large arteriovenous fistulas. However, angiomas rarely cause a shift of the midline structures or a ventricular deformity as do the glioblastomas.

Arteriosclerosis: Cerebral arteriosclerosis is not an indication for angiography in itself but it produces abnormalities which are sometimes visible in patients suspected of other lesions. There is a loss of the physiological loops and curves of normal arteries; they become straight and there is considerable variation in calibre with narrowed and enlarged regions or even aneurysms. A sudden break in continuity of an artery due to an intravascular block is said to be characteristic of arteriosclerosis[25].

Thrombosis: Carotid thrombosis is an entity which recently has been recognized as a fairly frequent cause of headache, psychic disturbances, hemiparesis, and aphasia. The maintenance of blood supply to the involved areas is insufficient because of poor collateral circulation. No dye can be injected into a thrombosed carotid artery, of course, but an angiogram done on the opposite side may reveal filling of both hemispheres through the collaterals in the circle of Willis. The middle cerebral may not be as well filled on the involved side as in the anterior cerebral artery. Ligation of the carotid artery on one side causes from 80% to

100% increased flow in the opposite carotid. Löhr[24] states that this can be demonstrated in angiograms of the uninvolved side by an enlargement of the vessel. Reichert[34] resects a thrombosed carotid artery feeling that he thereby increases the contralateral flow.

Indications

The indications for angiography in this clinic are not closely defined because it is felt that the full value and significance of the procedure is not as yet known. In general it is used whenever it is felt that such a study would provide knowledge valuable in diagnosis and treatment of the intracranial lesion. Up to the present time, it has been limited to use in patients with suspected aneurysms or neoplasms, Whether it will be used at a later date in patients with hydrocephalus, trauma, arteriosclerosis, or other diseases must await further evaluation.

Angiography is used routinely in patients with subarachnoid hemorrhage in order to ascertain the site of the vascular rupture. It is often done bilaterally because lateralizing signs may be minimal and the side first injected reveals no abnormality. Poppen injects patients with subarachnoid hemorrhage twenty-four to forty-eight hours after aneurysmal rupture for he feels that one cannot inject the dye fast enough through an 18 gauge needle to significantly raise the pressure within an aneurysm. Others prefer to wait four to six weeks following the accident in order to provide time for the site of rupture to heal. In this clinic, the interval between hemorrhage and angiogram has been progressively shortened until it has now been reduced to four days. The reason for shortening this interval is twofold: (1) to decrease the period of hospitalization, and (2) to treat the aneurysm before recurrent rupture with possible fatal hemorrhage.

Angiography is done on patients with suspected brain tumors in whom the side of the lesion is known and in whom ventriculographic studies are deemed inadvisable or are found to be unsatisfactory for tumor localization. In addition, angiography is done on patients in whom it is anticipated that it would be advantageous to know either the histological type of tumor or the source of the blood supply to the tumor.

Results

Included in this report are one-hundred consecutive angiograms performed at the University Hospitals or Veterans Administration Hospital from 1939 to 1949. The majority of them have been made during the last three years. The carotid artery was injected in ninety-six and the vertebral in four cases. From 1939 to 1945 the open technique of injecting the vessel was used. As the technical aspects of injection of the contrast media as well as the timing and amount of roentgen exposure became standardized, the percutaneous or closed method of injection has been used in increasing proportions. In the last thirty angiograms, twenty have been made with percutaneous injections, and only after the percutaneous method has been unsuccessful has the open method been used in these recent cases.

Thorotrast was used as the contrast media in ten cases, neoiopax in nine, and diodrast in eighty-one cases. The use of thorotrast was discontinued because of the dangers of radioactivity even though it seemed to provide greater contrast than other media. The use of neoiopax was discontinued because its injection was associated with a convulsive seizure in four of the nine cases in which it was used. A thirty-five per cent solution of diodrast has been the media of choice because of the paucity of reactions observed with its use. Irrespective of the type of solution injected, fifty per cent of the patients experienced disagreeable sensations over the side of the head with visible ipsilateral flushing of the face. Two per cent of the patients experienced tingling in the contralateral extremities. Serious complications in the one-hundred cases were as follows:

Convulsions - 5 patients
Hemiparesis - 2 patients
Deaths - 2 patients

The convulsive seizures all occurred with the use of neoiopax except in one case in which diodrast was used. The seizures were considered secondary to immediate and direct irritation of the vessel wall by the injected media.

Hemiparesis occurred in two patients, in one it was transient (6 hours) but in the other it was persistent. In the latter case the cause of the hemiparesis was considered due to the faulty technique of inadvertently injecting clotted blood into the carotid artery, which blood clot may have produced an area of cerebral ischemia.

Two patients expired following antiograms. In the first patient the diodrast infiltrated the carotid sheath but failed to enter the intracranial vessels. Because of his preoperative apprehension, local procaine infiltration anaesthesia was supplemented with intravenous pentathol solution. The cause of death was not ascertained at autopsy; clinically it was presumed to be due either to an overly sensitive carotid sinus reflex or to laryngeal spasm secondary to the anaesthesia. The second patient that expired had an angioma of the right cerebellar hemisphere. Immediately following the injection of neoiopax, he had a generalized convulsive seizure, became comatose and expired eight hours later. At autopsy no cause of death was demonstrated.

Chart I. Data on 100 Consecutive Angiograms

Indication	Total No.	Unilateral	Bilateral	Positive	Negative
Subarachnoid hemorrhage	42	23	19	6	36
Suspected brain tumor	53	53	0	10	43
Head injury	2	2	0	1	1
To study circulation of tumor	3	3	0	3	0
TOTAL	100	81	19	20	80

Chart I reveals the indications and results of the angiograms included in this series. The majority (53) were performed on brain tumor suspects; in thirteen (24%) of these there was angiographic evidence of the location of the tumor. The histological type of tumor was meningioma in six cases, angioma in one case, glioma in five cases, and granuloma in one case. In three patients angiograms were made in order to study the blood supply of previously verified tumors. The purpose of the study was to ascertain the most feasible method of surgical attack and in all cases it did furnish the desired data. Angiograms were made on forty-two patients with evidence of subarachnoid hemorrhage. In six (14%) the location of the aneurysm was determined. Two patients with head injuries were subjected to angiography and in one there was evidence of a subdural hematoma. In the other patient the study was normal.

Summary

1. A series of one-hundred angiograms is reported.

2. Demonstrable evidence of a tumor was present in 24% of the patients with suspected brain tumors.

3. Demonstrable evidence of an aneurysm was present in 14% of the patients with subarachnoid hemorrhage.

4. Angiography has been of definite value as an accessory diagnostic aid.

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III. MEDICAL SCHOOL NEWS

Doctors Bell and Nathanson to Give Special Lectures

On Wednesday, March 2, Dr. E. T. Bell, Professor of Pathology, will deliver the annual Clarence M. Jackson Lecture at 8:15 p.m. in the Auditorium of the Museum of Natural History. Dr. Bell's subject will be the "Pathology of Diabetes."

It is fitting that this tribute should be paid to Dr. Bell by Phi Beta Pi medical fraternity, sponsors of the lectureship. A dinner in honor of Dr. Bell will precede the lecture.

On Thursday, March 3, the annual George Chase Christian lecture on cancer will be presented by Dr. Ira T. Nathanson. This lecture on the subject, "Hormonal Alteration of Advanced Cancer of the Breast," will be presented at 8:00 p.m. in the Medical Sciences Amphitheater. Dr. Nathanson is a member of the cancer commission of the Harvard University Medical School and a member of the staff of the Massachusetts General Hospital.

He will also participate during his visit to Minneapolis in the course in Cancer for Non-Metropolitan Physicians of Minnesota which will be given at the Center for Continuation Study on March 3, 4, and 5. Dr. Nathanson's presentation in the cancer course will be on the subject of "Lymph Node Metastases."

Course in Physical Medicine to be Offered at the Center for Continuation Study

On March 28, 29, and 30, a course in Physical Medicine for General Physicians will be offered at the Center for Continuation Study. This course is intended to acquaint physicians with the benefits which can be obtained with the application of physical medicine to common medical problems. Lectures and demonstrations will be presented on the various forms of physical therapy. General emphasis will be placed throughout the course on those methods of treatment which the physician himself can administer. Symposia will be held on the use of physical medicine in rheumatoid disease and in fractures. In the symposium on rehabilitation, special emphasis will be placed on rehabilitation of the hemiplegic patient. Another subject which should be of particular interest to general physicians will be physical medicine in the treatment of the neuroses.

* * *

New Minn. Medical Foundation Members

Dr. Herbert W. Schmidt, 1514 Durand Court, Rochester
Dr. R. P. Hallin, Worthington
Dr. Richard O. Burmeister, Welcome

Kellogg Foundation Lectures

The following lectures will be given during the week of February 28. All medical students, interns, nurses, technicians, dietitians, and physicians are cordially invited to attend these lectures. A special invitation is extended to University Fellows.

Dr. D. R. Mathieson (Mayo Clinic)	"Immunity and Hypersensitivity"	Monday, February 28, 4:00-6:00 p.m. Powell Hall Amphitheater.
Dr. Herald R. Cox (Lederle Laboratories, Pearl River, N.Y.)	"Rickettsial Diseases"	Tuesday, March 1, 2:00-4:00 p.m., Todd Amphitheater.