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Hydrocephalus

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I. HYDROCEPHALUS

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Many procedures have been attempted in the treatment of hydrocephalus, but a satisfactory solution has not yet been attained. This report is primarily concerning the treatment of hydrocephalus. The pathology of hydrocephalus is reviewed since a knowledge of it is essential for an understanding of treatment. Various procedures employed in the past for the control of hydrocephalus are reviewed. Some experiments performed by one of the authors (GH) are reported and our experience in the treatment of hydrocephalus at the University Hospitals is reviewed.

Frequency

From 0.7 (71) to 7.8 (95) per thousand new born children are hydrocephalic.

Heredity

In certain strains of mice, recessive hereditary traits cause hydrocephalus[76]; also certain breeds of dogs are prone to develop hydrocephalus[60]. In man, heredity apparently is not an important factor. Multiple hydrocephalic births are rare in the same family unless there is associated spina bifida[8,76] or toxoplasmosis.

Pathology

Hydrocephalus due to cerebral atrophy is not included in this discussion. Only hydrocephalus characterized by dilatation of the ventricles due to increased ventricular pressure is considered. A simple classification of hydrocephalus is the following:

A. Non-communicating hydrocephalus. This involves blockage to the flow of cerebrospinal fluid at the foramina of Munroe, in the third ventricle, aqueduct of Sylvius, fourth ventricle or foramina of Luschka and Magendie. This obstruction may be due to:

1. Developmental abnormalities present at birth:

   a. Obstructions of the aqueduct of Sylvius account for one-half of the cases of obstructive hydrocephalus in infants[21]. There are several varieties:

      (1) Forking of the aqueduct in the form of multiple poorly connected channels, incapable of adequately conducting fluid[7,76]. Symptoms appear early in most cases but may occur as late as the tenth year.

      (2) Stenosis of the aqueduct, consisting of simple narrowing of the aqueduct. This is not very common.

      (3) Neuroglia septum across aqueduct. This is extremely rare, only five cases being noted by Russell[76]. In one case there was a complete septum at postmortem allowing no fluid to pass through the aqueduct, yet the child lived to be eight years old.

      (4) Gliosis of the aqueduct. This condition is characterized by subependymal gliosis and is progressive in nature, but the true etiology is not clear. Eighteen cases were reported up to 1940[76].

      (5) Compression of the aqueduct due to vascular abnormality such as superior cerebellar artery aneurysm[75], or thalamic cyst[62].

   b. Hydrocephalus with spina bifida and meningocele. The true etiology of the hydrocephalus is thought by some

to be loss of the absorbing power of the meningocele sac[68], by others, obstruction at the foramen magnum due to developmental traction on the brain stem, the distal cord being anchored at the meningocele sac (Arnold-Chiari deformity)[54]. However, the brain stem may develop a similar deformity when there is no traction[76], and the Arnold-Chiari defect may produce hydrocephalus without associated spinal defect[1,34]. When the myelomeningocele is present, removal of fluid from the sac may precipitate hydrocephalus, the high intracranial pressure forcing the brain stem into the foramen magnum as a "plug", perpetuating hydrocephalus by blocking the flow of cerebrospinal fluid at the foramen magnum[27,80], but this is not a constant occurrence[36].

c. Platybasia narrows the foramen magnum and thus produces obstruction[76].

d. Achondroplasia, because of unknown factors, causes obstruction[76].

e. Congenital atresia of the foramina of Luschka and Magendie[11,22].

2. Non-communicating traumatic. This is almost always due to hemorrhage and scar tissue formation at the foramina of exit of cerebrospinal fluid from the ventricular system due to wounds[6], cerebellar hematoma[10], or birth trauma. The reaction is always one of aseptic inflammation in addition to obstruction.

3. Non-communicating neoplastic. Obstruction occurring after infancy is due, in 95% of cases, to cysts or tumors[74]. They may be variable in type and location, but of course those in the ventricles or near foramina or in the aqueduct obstruct most readily.

4. Non-communicating inflammatory. This type of obstruction above the fourth ventricle is frequently due to brain abscess[2], ependymitis or other inflammatory lesion[14]. Russell[76] states that probably one-half of the cases of chronic hydrocephalus in children are due to post-meningitis inflammation, usually at the base, the meningitis attack at times being very mild. It may be pyogenic or granulomatous. Of the granulomas, tuberculosis is most common producing an obliteration of the basal cisterna. Monilia, torula, toxoplasma and cysticercus are rarely the etiological agents and usually they effect primarily the base. In many cases of posterior fossa arachnoiditis, the etiology is not clear[41].

B. Communicating hydrocephalus. In this type there is obstruction to the flow of cerebrospinal fluid after it leaves the foramina of Luschka and Magendie, that is, there is communication between the ventricular system and the spinal subarachnoid spaces. The obstruction may be in the basal cisterns, at the tentorial notch, the subarachnoid space over the cerebral hemispheres, or at the arachnoid villi and Pacchionian granulations.

Communicating hydrocephalus may be due to decreased absorption of cerebrospinal fluid or possibly to increased production.

1. Increased production. It is not certain that this is ever the cause of hydrocephalus. Its occurrence is supported only by inconclusive observations of hydrocephalus occurring with the following lesions:

a. Otitic hydrocephalus, a poorly defined entity usually involving ear infection with thrombosis of the lateral sinus[76,86,87].

b. Pressure upon the lateral sinus, as from tumor[76].

c. Hypertrophy of the choroid plexus, an extremely rare condition[26].

2. Due to decreased absorption of cerebrospinal fluid.

a. Developmental abnormalities of the subarachnoid spaces present at birth may account for some cases of hydrocephalus[3], but it has not been conclusively demonstrated that this does occur. Diet deficient in folic acid has, in rats[66,73], produced hydrocephalus which may be of this type. Congenital aneurysms may give rise to repeated subarachnoid hemorrhages and it has been demonstrated that repeated hemorrhages can produce asceptic meningitis with obliteration of the subarachnoid space. Repeated traumatic subarachnoid hemorrhage may, of course, produce the same result. Brain stem swelling, as the result of trauma, may produce obstruction at the tentorial notch.

b. Traumatic. Subdural hematoma may produce hydrocephalus from interference with circulation and absorption of the cerebrospinal fluid[67]. An incidence of 28% has been reported[48].

c. Neoplastic. This is a rather rare entity and is caused by diffuse infiltration of the meninges by malignant cells (gliomatosis, sarcomatosis, secondary carcinoma). Usually, the basal cisterns are affected more than the subarachnoid space at the convexity. Posterior fossa tumor may cause obstruction at the tentorial notch[28].

d. Inflammatory. An inflammatory reaction due to bacteria, virus or parasite, or foreign material obstructing the basal cisterns is most common, but the subarachnoid spaces over the convexity may be involved. Gargoylism, because of the abnormal irritating lipoid may cause adhesions over the convexity[76].

As hydrocephalus develops[68,69], the cerebral white matter is destroyed first, beginning proximally in the lateral ventricles and progressing toward the block. The septum pellucidum becomes fenestrated and the corpus callosum thinned. The third ventricle is ballooned out in the region of the lamina terminalis and may give rise to visual field changes from pressure upon the optic chiasm[44]. The choroid plexus may be atrophied or normal. The ependyma is often destroyed and subependymal gliosis is usually present. The ventricular wall may rupture[85] with formation of false diverticuli, especially out over the superior part of the cerebellum[70,76]. Rupture of the head has been reported[4]. Ventricular dilatation is, to some extent, reversible when pressure is relieved[79].

The microscopic changes consist of variable amounts of demyelinization and loss of tissue in the white matter, loss of ependyma and development of subependymal gliosis. Aberrant cortical tissue may lie in the white matter, nerve cells may be irregularly distributed, and there may be disturbed cortical lamination[12]. The collaterals of the long nerve fibers of the white matter may be torn off and compound granular corpuscles formed to engulf the disconnected fibers.

Treatment

The treatment of hydrocephalus has been primarily directed toward:

a. Reducing the production of cerebrospinal fluid.

b. Increasing absorption of cerebrospinal fluid chiefly through facilitating its exit from the ventricular system.

Let us examine the first of these methods.

To decrease the production of cerebrospinal fluid, two surgical methods

have been employed:

a. Carotid artery ligation.
b. Destruction of the choroid plexus.

Carotid artery ligation was employed as early as 1898[89] and as recently as 1922[33]. This procedure has been reported, but not definitely proven, to have arrested the progress of hydrocephalus by decreasing blood flow through the choroid plexus. There is danger of brain ischemia with resulting hemiplegia from carotid ligation.

Destruction of the choroid plexus has been attempted by removal[17,19,24], coagulation[25,71,77,78], or x-ray radiation[23,89]. Coagulation is the procedure most extensively employed and has produced satisfactory arrest of the progress of hydrocephalus in from 20 - 50% of patients operated upon. Radiation of choroid plexus has been thought to produce cures of hydrocephalus but convincing proof is lacking.

Medical treatment to reduce the production of cerebrospinal fluid such as thyroid extract[23], has not been of benefit.

To facilitate the flow or absorption of cerebrospinal fluid, medical treatment has been tried and found ineffective. Injection of hypertonic solutions (intravenously or into the gastrointestinal tract)[31,89] produces only a temporary reduction in the cerebrospinal fluid pressure. Dicumarol[51] (to promote lysis of subarachnoid adhesions), theobromine sodiosalycilate[23,56] (to decrease surface tension of the blood and cause it to absorb fluid), have all been tried without lasting value. Postural drainage[68] (upright position) may be of some benefit in mild hydrocephalus.

Other forms of treatment which have been tried are the following:

1. Surgical removal of obstruction.

2. Open drainage.

3. Closed drainage.

   a. Attempts have been made to drain cerebrospinal fluid from the ventricle:

      (1) into the subdural, extradural, and extracranial spaces.

      (2) into the subarachnoid space.

      (3) into a body cavity.

      (4) into the venous system.

   b. Attempts have been made to drain cerebrospinal fluid from the subarachnoid space.

      (1) into a body cavity.

      (2) into the venous system.

4. Cervical sympathectomy (to increase absorption in arachnoid villi).

To facilitate the exit and absorption of cerebrospinal fluid, removal of any obstruction (as tumor or hematoma) is indicated if this is surgically possible[1,5,24,27,33,41,74]. If the obstruction is removed, the results are usually good. In some cases of obstruction of the aqueduct of Sylvius, a catheter, silver spiral, or plastic tube has been passed through the aqueduct, later removed, or left in place permanently with relief of obstruction[18,89]. In general the mortality has been high and results unpredictable, although Leksell[53] and Norlen[63] have recently reported encouraging results with a wire spiral inserted into the aqueduct.

Open drainage: Repeated punctures of ventricle or subarachnoid space eventually result in infection[24,89]. Open continuous drainage through tubes or along hairs inserted as a drain also terminate in infection.

Closed drainage from the ventricle into the epidural or subdural space or the extracranial soft tissues has been attempted. Using various kinds of tubes and drains, the ventricles have been

connected with the epidural and subdural space[23], the subgaleal space, the temporal muscle[43], and the intraorbital fat[89]. Good results were sometimes reported in isolated cases.

Drainage from the ventricle into the subarachnoid space has met with some success. Initially an attempt was made to drain into the subarachnoid space by removal of a block of brain tissue[89], but the meninges became adherent and the hydrocephalus progressed. Attempts were also made to establish a communication between ventricle and subarachnoid space through the corpus callosum, also between lateral ventricle and the subarachnoid space by omental strips[23,50], some of which were reported to have been successful in arresting the progress of the hydrocephalus.

More recently the third ventricle has been brought into communication with the subarachnoid space through puncture of the floor of the third ventricle. Dandy[20,21] was the first to make such an opening in the floor of third ventricle. This operation has subsequently been modified by Dandy and others[35,58,59,82,89,91,92]. These modifications include variations in approach by elevation of the frontal lobe or the temporal lobe or through the ventricles with single or multiple openings made in various parts of the floor of the third ventricle.

Successful arrest of the hydrocephalus has been produced in from 50 - 100% of cases operated. Avulsion of the choroid plexus, which at the same time opens the lateral ventricle into the cisterna ambiens, has been done in a small number of cases with favorable results[45].

Several methods using tubes to connect the ventricle with the subarachnoid space have been devised, the most widely used being the Torkildson procedure[89] reported in 1939. This involves connecting the lateral ventricle by means of a rubber catheter with the cisterna magna. Reports indicate that it has been quite successful in relieving hydrocephalus due to a block of the cerebrospinal fluid pathways between the lateral ventricle and cisterna magna[29,40,90]. Another similar procedure, reported in only two cases, is the connection of the occipital horn with the cisterna magna by means of a tube passing through the tentorium[72].

Drainage from a ventricle into a body cavity: Recently, drainage of the ventricular system into the peritoneal cavity has been practiced extensively by Cone[13] and still more recently by Jackson, Schofield, and others[49]. In a considerable experience, it has been found that blockage or dislocation of the tube frequently occurs in infants but not in adults.

The most recent method is that of Nosik[64,65] whereby the temporal horn of the lateral ventricle is connected, by means of a plastic tube, with the middle ear. This procedure has not been done in a large number of cases, but it has been successful in relieving hydrocephalus. There is, of course, the possibility of having infection extend from the pharynx to the meninges, but so far experience has not shown this to be a serious hazard. The longest cure thus far reported is three years and this child has had measles, colds, and sore throat without spread of the infection to the meninges. It appears to be a most satisfactory method, but more experience with longer observation is necessary to evaluate it.

Drainage of the ventricle into the venous system (saggital sinus, veins of scalp or jugular vein[89] has been unsuccessful because of tube blockage with clotted blood and periodic bleeding into the ventricle. Drainage into the thoracic duct has been unsuccessful in dogs[47].

Drainage from the lumbar subarachnoid space into body cavities has been confined to the peritoneum, ureter and pleural cavity.

Drainage into the peritoneum, by means of a tube from the lumbar subarachnoid space was first done by Ferguson in 1898, later by Heile[39], Cushing, and others[89,24]. Good results were obtained in a few cases. Variations using intestine, silk threads, or rubber tubes were

later tried[23]. More recently Cone[13], later Jackson[49] and others have drained the spinal subarachnoid space with a plastic tube passed subcutaneously around the flank to the peritoneal cavity. The results have been more promising than in the ventriculo-peritoneal shunts.

Drainage of the lumbar subarachnoid space into the ureter has received a great deal of attention since it was first performed by Heile[38] and recently popularized by Matson[57] and others[9,24,52,94]. It involves connecting the lumbar subarachnoid space with the ureter by means of a plastic tube. Usually the kidney is removed. Adequate pressure relief has been obtained in the small number of cases done to date. Electrolyte must be added to the infant's diet because of that lost through the evacuated cerebrospinal fluid.

Drainage into the pleural cavity[39,89] in two cases so far reported has failed because of obstruction in the pleural cavity.

Drainage from the spinal subarachnoid space into the venous system has been beset with the same difficulty of tube blockage as has drainage from the ventricular system to the venous system[23,37,89].

Cervical sympathectomy[89] has failed to increase absorption.

In general, concerning these numerous procedures employed over a long period, it is to be noted that many procedures were tried only a few times and then abandoned. Initial reports, however, were nearly always favorable. It is also true that most procedures have been directed toward the obvious mechanical aspects of the problem. The fundamental physiology of production and absorption of cerebrospinal fluid is still unsolved.

Experimental Hydrocephalus

Experimentally, hydrocephalus has been produced by a number of methods, but nearly all have been inconstant and there has been a high mortality rate in the experimental animals. They may be divided into:

1. Those methods having as their intention the blocking of the foramen of Munroe or the aqueduct of Sylvius.

2. Those producing adhesive arachnoiditis or mechanical obstruction in the subarachnoid space of the posterior fossa or convexity.

In the first group, leuconite and starch injected into the ventricle has been used to block the foramen of Munroe[88]. The aqueduct of Sylvius has been blocked with cotton[16], cotton soaked in lampblack[42], by stinulation of ependymal growth by x-ray radiation[83], and by cellophane[46].

The posterior fossa and cerebral subarachnoid spaces have been blocked by producing experimental meningitis[30], foreign bodies in post fossa ("sponge tent")[15], and an iodine strip around the midbrain[22]. Lampblack and india ink have been injected into the ventricle or cisterna magna[32,61,81,93]. Kaolin has been injected into the cisterna magna[55], and also thorotrast[84].

Experiments

Animal experimentation on the production and treatment of hydrocephalus was carried out by one of us (GH) during the past year.*

Hydrocephalus was produced in dogs by two methods. The first was that of Ingraham et al[46], involving the placement of a small cylinder of cellophane in the aqueduct of Sylvius. Adult dogs were used. A suboccipital craniectomy was done and the fourth ventricle opened. A small cylinder, formed by winding cellophane upon a segment of ureteral catheter, was inserted by means of a curved wire carrier into the aqueduct of Sylvius. The cellophane should produce an inflammatory reaction with slow, progressive occlusion of the aqueduct. This method was found not to be entirely satisfactory in our hands, the production

* This work was done at the Montreal Neurological Institute under the direction of Dr. W. V. Cone.

of even moderate hydrocephalus being unusual.

Hydrocephalus was then produced in puppies by the cisternal injection of lampblack. It has been found that only a few types of lampblack will produce hydrocephalus. Fortunately, the first type of black tried was effective. One-half to 1 cc. of a 10% saline suspension was injected into the cisterna magna taking care to mix it well just prior to injection. The lampblack spread throughout the basal cisterns and through its bulk together with the adhesive arachnoiditis produced by it, obstruction was produced. Moderate to severe hydrocephalus occurred in all animals and, in general, they tolerated the procedure well, only one animal being lost from the injection.

Extensive hydrocephalus having been secured, attempts were made to connect the dilated ventricular system with the pleural cavity. Ingraham et al[47] had previously tried this but the tubes they inserted uniformly became blocked. It was not determined if the pleural cavity reacted to block the absorption of cerebrospinal fluid.

In the experiments here reported with various techniques, Portex and Polythene tubing were passed from the posterior portion of the lateral ventricle, through a trephine hole in the posterior parietal region, then subcutaneously down to the thorax and into the pleural cavity, either posteriorly through a defect formed by resecting a portion of the eighth rib, or anteriorly through a similar defect in the third rib. It was necessary to insert the tube into the thoracic cavity for a distance equal to that from the second rib to the diaphragm, in order to prevent dislocation of the tube out of the pleural cavity. The status of the chest cavity was followed with periodic x-rays and the continued functioning of the tube by periodic ventricular pressure measurements. Cell counts of the ventricular fluid were taken whenever infection was suspected.

Results

Nearly 60% of the shunts were complicated at some time by sepsis along the tube. If the sepsis extended into the thorax, it followed the tube and became a well encapsulated abscess.

About 50% blocked at the ventricular or thoracic end, or both. The blocking material in the chest was fibrin (at times in the form of a sac); in the ventricle it was fibrin and glial elements.

When the animals were sacrificed, 20% of the tubes were functioning, the longest functioning period being 29 days after operation. In none of these was there excess fluid in the chest.

Examination of the chest cavity revealed a small amount of fibroblastic reaction of the pleura adjacent to the tube, but no generalized reaction. In cases where a tube had been inserted but no fluid entered the chest cavity, the reaction was almost entirely simple compression of the pleura and adjacent lung from the weight of the tube.

It seems definite that the chest easily handled the fluid from the ventricle and that the reaction of the pleura to tube and fluid was minimal. The operated area, however, seemed prone to infection. The negative intrathoracic pressure (at times as high as -90 mm. H_2O) may have promoted tube blockage.

Microscopic examination of the brain and cord in these animals revealed an arachnoiditis from the lampblack in the subarachnoid space. No unusual reactions were found in the brain except that the gliosis around a cellophane plug in the aqueduct was surprisingly small.

We have, so far, felt hesitant to try drainage into the pleural cavity in humans largely because of the possibility of infection.

In the years 1938 to 1949 inclusive, fifty-two patients have received one or more operations primarily for the relief of hydrocephalus. The various combinations are tabulated below:

Coagulation of choroid plexus alone	19
Lamina terminalis puncture alone	11
Torkildson procedure alone	11
Decompression of Arnold-Chiari deformity	3
Creation of artificial foramen of Magendie	1
Lamina terminalis puncture and Torkildson procedure	3
Lamina terminalis puncture and decompression of Arnold-Chiari deformity	1
Coagulation of choroid plexus and lamina terminalis puncture	1
Coagulation of choroid plexus and Torkildson procedure	1
Coagulation of choroid plexus and decompression of Arnold-Chiari deformity	1

Coagulation of Choroid Plexus

Twenty-two patients with communicating hydrocephalus received coagulation of the choroid plexus. In each case before the choroid plexus was coagulated it was demonstrated that there was communication between the ventricle and subarachnoid space by the passage of dye or air between the ventricle and lumbar subarachnoid space. One of these patients had previously an ineffective Torkildson for what later proved to be a communicating hydrocephalus of toxoplasmosis. In two patients an obstructive hydrocephalus was, previous to the coagulation, converted into a communicating hydrocephalus by lamina terminalis puncture and decompression of an Arnold-Chiari deformity respectively.

The etiology of these cases of communicating hydrocephalus was toxoplasmosis in four patients, meningitis in five, metastatic tumor in one, encephalocele in one, and myelomeningocele in two. In nine no cause was demonstrated.

Eight of the twenty-two patients are known to be dead. Two died on the first postoperative day in hyperthermia. In the third, intracranial pressure was controlled only a few days and then returned in such magnitude that a cerebro-spinal fistula developed with subsequent meningitis and death. Three of the patients now dead were controlled for a few months only to have a recurrance of the hydrocephalus and death. Postmortem examination in one of these revealed a metastatic Wilms' tumor. Another patient, after several months of control, was temporarily out of balance but then in balance until death two years after operation. Death was evidently due to severe neurological deficits. Finally, one child remained in mild imbalance for one year but otherwise developed normally until two years postoperatively when he had the first of a series of disabling intracranial events diagnosed at home as "strokes". After the second of these episodes he was reported to have been paralyzed on the right side and aphasic. He died seven years after operation during one of these attacks.

Of the fourteen patients living when last seen, four cannot now be located. One of the four was in very poor condition when last seen.

Of the remaining ten, five are at least of average mentality, but two of them are disabled by spastic lower extremities. Two of the ten cases appeared to have a normal intelligence but are so shy because of severe physical defects that it was difficult to estimate their mental capacity; three are completely disabled by both mental deficiency and serious physical defects.

The above results do not give a true indication of the effectiveness of surgery. In twelve of the eighteen adequately followed patients, the hydrocephalus was controlled throughout the period of observation except that in three there was a short period of recurrent imbalance. Three additional cases were controlled $1\frac{1}{2}$, 3 and 6 months respectively. Only one case was not controlled at any time.

It would, therefore, appear that coagulation was successful in more than one-half the cases in arresting the progress of hydrocephalus, but may survive with severe neurological deficits. They are apparently due to the hydro-

Chart I

Etiology and follow-up data on 22 cases of communicating hydrocephalus in which choroid plexus was coagulated.

Etiology	Status	Postop. follow-up	Duration of control of hydroceph.	Physical status	Mental status
Toxoplasmosis	L	10 months	10 months	poor	poor
"	L	42 months	42 months	poor	poor
"	L	2 months	0	-	-
"	L	72 months	72 months	fair	fair
Meningitis	L	48 months	48 months	fair	poor
"	D	84 months	84 months	-	-
"	D(postop.)	-	-	-	-
"	L	2 weeks	2 weeks	-	-
"	D	5 months	1½ months	-	-
Tumor	D	4 months	3 months	-	-
Encephalocele	L	3 months	3 months	good	good
Myelomeningocele	L	90 months	90 months	fair	good
"	L	30 months	30 months	poor	poor
Unknown	L	1 month	1 month	fair	good
"	D(postop.)	-	-	-	-
"	D(18 days postop.)	5 days	-	-	-
"	L	12 months	12 months	good	good
"	L	7 months	7 months	fair	good
"	D	24 months	24 months	-	-
"	D	18 months	6 months	-	-
"	L	24 months	24 months	good	good

cephalus or the lesion causing the hydrocephalus and could be eliminated by a more restricted selection of cases for operation.

In general, we selected for coagulation of the choroid plexus cases in which it was decided, through history and physical signs, that the imbalance between secretion and absorption of fluid was not great. Preoperative intraventricular pressure and thickness of the cerebral wall were also given some consideration. Preoperative ventricular pressures were recorded in ten patients and at operation the thickness of the cortex was recorded in eleven. There were instances of relief of hydrocephalus in the face of high preoperative intraventricular pressure and instances of failure in the presence of low intraventricular pressure. The cortical thicknesses varied from 0.5 cm. to 2.5 cm. The patient with the 0.5 cm. cortex had a good result and that with a 2.5 thickness had a poor result. Observations

were too few to draw any definite conclusion as to the value of pressure and thickness of cortex in the selection of patients for operation.

Decompression of Arnold-Chiari Deformity

Six infants with myelomeningoceles complicated by non-communicating hydrocephalus have had suboccipital exploration; in five an Arnold-Chiari deformity was found and decompressed; in the sixth patient an absence or occlusion of the fourth ventricular foramina was found. An opening was made, the hydrocephalus arrested and she is now five years of age but has spastic lower extremities and severe mental retardation.

One of the five infants with Arnold-Chiari deformity expired of operative shock. In the other three hydrocephalus was uncontrolled; one had so many congenital anomalies that nothing further was done and expired a few months later; the second expired $1\frac{1}{2}$ months after a lamina terminalis puncture which gave temporary relief; the third has already been listed in the coagulation series. Her hydrocephalus was eventually controlled by bilateral choroid plexus coagulation and at eight years of age she is somewhat retarded mentally and has a spastic paraplegia.

The three living children who had suboccipital exploration all had evidence of leg paralysis preoperatively, all had repair of the myelomeningocele, all now have severe residual neurological defects.

Lamina Terminalis Puncture

Sixteen patients had lamina terminalis punctures alone or in combination with other procedures for relief of non-communicating hydrocephalus.

Six patients had obstruction due to tumor; in three the probable cause was benign aqueduct obstruction; five were associated with encephalocele or myelomeningocele, one had toxoplasmosis and in one, even autopsy did not reveal the cause.

The pertinent data on these patients is given in Chart 2.

In four patients the follow-up is inadequate; in three of these the pressure was controlled when last seen.

Nine patients are now dead. Five died before leaving the hospital. Two patients with associated non-communicating hydrocephalus and myelomeningocele were temporarily relieved ($1\frac{1}{2}$ to 7 months) but died with recurring pressure. The other two now dead failed to receive any benefit from the procedure.

There are three living patients with adequate follow-up; two were children with presumed tumors whose hydrocephalus was controlled only after roentgen therapy; the third had benign aqueduct obstruction and hydrocephalus was not controlled until Torkildson procedure was done.

Torkildson Procedure

In fifteen patients a Torkildson procedure was carried out: Ten for obstructing tumors, two had toxoplasmosis, one a benign aqueduct obstruction, and in two postmortem did not reveal the cause of the obstruction.

These patients are summarized in Chart 3.

Chart 2

Etiology and follow-up data on 16 cases of hydrocephalus in which the lamina terminalis was punctured for hydrocephalus.

Etiology	Status	Follow-up	Control of hydroceph.	Age Group
Unknown	D(postop.)	-	no	adult
"	D	-	no	adult
"	D(postop.)	-	no	child
"	D	inadequate	no	adult
"	L	4 years	only after x-ray Rx	child
"	L	8 months	" " " "	child previous table
Benign obst. aqueduct	L	5½ years	only after Torkildson	adult
"	L	inadequate	effective	adult
"	D(postop.)	-	-	infant
Encephalocele	D(postop.)	-	-	infant meningitis
"	L	inadequate	effective	infant
Myelomeningocele	L	inadequate	effective	infant
"	D	1½ months	temporary effect	infant
"	D	1 year	7 months	infant
Toxoplasmosis	D	4 years	ineffective	infant
Unknown	D(postop.)	-	ineffective	adult previous table

Chart 3

Etiology and follow-up data on 15 cases of hydrocephalus on which Torkildson procedure was done for hydrocephalus.

Etiology	Age group	Status	Follow-up	Control of hydroceph.	
Tumor	adult	D(postop.)	-	no	
"	child	D	7 months	temporary	Tube blocked at post
"	adult	L	1 year	yes	x-ray
"	adult	D	2½ years	1½ years +	x-ray
"	adult	?D	inadequate	no	
"	child	D	3 years	yes	later operation for craniopharyngioma
"	adult	L	2 years	yes	x-ray
"	child	D(postop.)	-	no	plugged,adjusted
"	child	L	9 months	yes, after x-ray	poor until x-ray
"	child	L	8 months	no	lamina terminalis and x-ray
Toxoplasmosis	adult	L	later proved to be due to lack of absorption - see coag.		
"	child	L	15 months	yes	
Benign lesion of aqueduct	adult	L	2 years	yes	
Unknown	adult	D(postop.)	-	no	lamina terminalis
"	adult	D(postop.)	-	-	-

Seven patients are known to be dead and one other unrelieved of hydrocephalus was moribund when last seen. Four deaths were postoperative. Three of the eight were controlled for three to eighteen months before death.

Of the seven living patients, four had tumors and have lived eight months, nine months, one year and two years respectively. In two of them hydrocephalus was not controlled until they had received x-ray therapy. Two with benign obstruction are alive and well fifteen months and two years after operation. The other living patient was later found to have a communicating hydrocephalus with toxoplasmosis and was controlled by coagulation of the choroid plexus.

In summary, the Torkildson procedure, done in fifteen patients, was effective in controlling the hydrocephalus permanently in five and temporarily in three.

Ventriculo-Mastoidostomy

Seven ventriculo-mastoidostomies have been performed in the last four months.

The first infant had a large myelomeningocele. The patient was admitted at the age of five days but operation was deferred until the granulating sac would heal. There was no neurological deficit on admission but in the next few days paralysis of the sphincters and the legs set in and soon appeared complete. During the next month the sac did not heal and the child developed hydrocephalus with rapidly enlarging sac and head. One month after admission a ventriculo-mastoidostomy was performed inserting a polythene tube through a trephine opening in the right posterior parietal area into the right lateral ventricle. This was carried subcutaneously to the second trephine opening just behind and over the ear and inserted into the mastoid antrum.

Postoperatively the child drained fluid profusely from the nasopharynx when on its face and the head collapsed and the myelomeningocele sac collapsed and healed rapidly so that repair could be done one week later. Two months after operation the head is the same size it assumed after operation.

Two newborn infants with encephalocele and hydrocephalus have had ventriculo-mastoidostomy performed with immediate success. In one of them an ulcerated sac promptly healed making its repair possible.

One child with communicating hydrocephalus following meningitis and one child with communicating hydrocephalus of unknown cause have had ventriculo-mastoidostomy.

A one year old child who had bilateral choroid plexus coagulation during the fifth and sixth months of life for communicating hydrocephalus and had been well controlled for five months when it again developed signs of pressure and an enlarging head and in whom dye tests now demonstrate obstruction, had a ventriculo-mastoidostomy with relief of pressure.

The seventh case was a $2\frac{1}{2}$ year old child with a craniopharyngioma extending into the third ventricle to obstruct the flow of cerebrospinal fluid between the lateral ventricles and between the lateral ventricles and aqueduct. Bilateral ventriculo-cisternostomy tubes were inserted. Fluid drained into the posterior fossa but accumulated there apparently due to tumor blocking the basal cisterns in the region of the floor of the third ventricle. A cerebrospinal fluid fistula developed in the wound above the posterior fossa. Eventually a ventriculo-mastoidostomy was done, but the fistula had become large, meningitis ensued, and the child died. Immediately after operation dye injected into the lateral ventricles came through into the pharynx but later when meningitis became established it did not do so. Failure in this case was obviously due to factors not related to the ventriculo-masyoidostomy.

Discussion

Coagulation of the choroid plexus has been quite successful in controlling the progress of communicating hydrocephalus. The mortality, 14%, has been lower than in other procedures. Unfortunately the patients salvaged by coagulation were on the whole a disabled group, but this morbidity appears to be due to the effect of the hydrocephalus before it was relieved or to the disease which caused the hydrocephalus rather than to the operation. There is, of course, a possibility that trauma of operation could be a factor.

Suboccipital decompression for Arnold-Chiari deformity was successful in only one of six cases. This operation should apparently be discontinued.

Puncture of the lamina terminalis resulted in the hightest postoperative mortality (31%) and was almost uniformly ineffective in permanently relieving the hydrocephalus. This operation should apparently be discontinued.

Torkildson ventriculo-cisternostomy was effective in most cases in which it was done for aqueduct obstruction. But two tubes became obstructed, several cases were erroneously thought to be obstructive when the operation was done

but later proved to be non-obstructive. It has also failed because in addition to aqueduct obstruction there has been associated obstruction of the basal cisterns in the region of the tentorial notch. Torkildson ventriculo-cisternostomy apparently has merit in properly selected cases. The postoperative mortality was 27%.

Ventriculo-mastoidostomy has been done in seven cases all in the last few months. The result has been uniformly good except in one case which developed meningitis from a pre-existing cerebrospinal fluid fistula. More experience and a longer period of follow-up will be necessary to evaluate this procedure, but at the moment it appears to be more satisfactory than any of the operations evaluated in this report.

Summary

The pathology of hydrocephalus is discussed. Literature concerning the treatment of hydrocephalus is briefly reviewed. Some experiments on the production of hydrocephalus in dogs and its treatment by drainage into the pleural cavity are described.

Experience in the treatment of hydrocephalus at the University Hospitals from 1938 to 1949 inclusive is reported and in addition some more recent experience with ventriculo-mastoidostomy.

In the above period, twenty-two cases were treated by coagulation of the choroid plexus. Three died in the hospital after operation (14%) and five died later. There was only one patient in which the hydrocephalus was not controlled at any time. Ten have been followed six months to six years and remain in balance. Two others remained in balance until they died of other causes two years and eight years after operation. The other six have either had only temporary control or have been followed less than six months. This method has, therefore, been quite successful in arresting the progress of hydrocephalus.

Decompression of the Arnold-Chiari deformity was not successful in five of six attempts and should be abandoned.

The lamina terminalis was punctured in sixteen patients. Five died in hospital after operation (31%) and four died later. This procedure was not effective in arresting the hydrocephalus in any of the patients who have had adequate follow-up.

A Torkildson ventriculo-cisternostomy was done in fifteen cases. Four died after operation (27%) and three others died later but since the operation was for obstruction due to tumor in ten patients, these late deaths are to be expected. This procedure was effective in controlling the hydrocephalus permanently in five cases and temporarily in three. Failure to control the hydrocephalus is known to have been due to blocking of the tube in two cases and erroneous diagnosis in a case of communicating hydrocephalus. Two of those dying in the hospital were also erroneously diagnosed since postmortem examination failed to reveal a cause for the hydrocephalus. The Torkildson ventriculo-cisternostomy was, therefore, quite effective in controlling the progress of hydrocephalus in obstructive hydrocephalus.

Recently seven ventriculo-mastoidostomies have been done with satisfactory decompression of the hydrocephalus in all except one case which developed meningitis from a pre-existing cerebrospinal fluid fistula. These operations were all done too recently to evaluate the long term results of ventriculo-mastoidostomy, but at the moment it would appear to be the most efficient operation for obstructive hydrocephalus.

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II. MEDICAL SCHOOL NEWS

Coming Events

March 1	Clarence M. Jackson Lecture: "Fractures About the Hip -- Early and Late Therapy," Carl E. Badgley; Museum of Natural History Auditorium; 8:00 p.m.
March 1 - 3	Continuation Course in Fractures and the Surgery of Trauma for General Physicians
March 26 - 28	Continuation Course in Pediatrics for General Physicians
April 2 - 6	Continuation Course in Urology for General Physicians and Surgeons
April 5 - 7	Symposium on Lupus Erythematosus
April 9 - 11	Continuation Course in Gynecology for General Physicians

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Dr. Carl E. Badgley to Give the Clarence M. Jackson Lecture

Dr. Carl E. Badgley, Professor, Department of Surgery, and Head, Division of Orthopedic Surgery, University of Michigan, Ann Arbor, Michigan, will deliver the annual Clarence M. Jackson Lectureship sponsored by the Phi Beta Pi Medical Fraternity on Thursday, March 1, 1951, at 8:00 p.m. in the Museum of Natural History Auditorium. His subject will be "Fractures About the Hip -- Early and Late Therapy."

During his visit to our campus Dr. Badgley will also participate in a continuation course in Fractures and the Surgery of Trauma which will be presented for general physicians at the Center for Continuation Study. Dr. Badgley's subject in the continuation course is concerned with fractures of the shaft of the femur and fractures about the elbow with nerve complications.

It may be noted that in connection with the course in Fractures and the Surgery of Trauma that accidents as a public health problem and as a legal problem will be stressed in addition to the clinical manifestations.

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Faculty News

Dr. Wallace D. Armstrong, Professor and Head of the Department of Physiological Chemistry, was the speaker at the February 21 meeting of the Minnesota State Dental Association. Dr. Armstrong's subject was "Fluoride and Dental Caries."

Dr. Grafton A. Smith left last week for the Navy Medical Corps. He will be stationed at the St. Alban's Naval Hospital in Long Island, New York.

Dr. Russell M. Nelson recently left for the Army Medical Center Graduate School in Washington, D. C.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Visitors Welcome

February 25 - March 3, 1951

Sunday, February 25

University Hospitals

9:00 - 10:00 Surgery Grand Rounds; Station 22.

10:30 - Surgical Conference; Todd Amphitheater.

Monday, February 26

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; M-109, U. H.

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

11:00 - 11:50 Physical Medicine Seminar; Scoliosis -- Spinal Fusion; John Moe; E-101, U. H.

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

12:00 - 12:50 Physiology Seminar; Cytological Changes in Experimental Liver Injury in Mice; W. Lane Williams; 214 Millard Hall.

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

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

4:00 - Public Health Seminar; 113 Medical Sciences.

4:00 - Pediatric Seminar; Brain Tumors; Dr. Sterrie; Sixth Floor West, U. H.

4:30 - 5:30 Dermatological Seminar; M-436, U. H.

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

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staffs; Powell Hall Amphitheater.

Monday, February 26 (Cont.)

Minneapolis General Hospital

9:00 - 10:00 Pediatric Rounds; Dr. Tobin; 5th Floor Annex.

10:00 - 11:00 Pediatric Rounds; Franklin Top; 7th Floor Annex.

1:00 - 2:00 Staff Meeting; Classroom, 4th Floor.

2:00 - 3:00 Journal Club; Classroom, Station I.

Veterans Administration Hospital

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

11:30 - X-ray Conference; Conference Room; Bldg. I.

1:00 - Metabolic Disease Rounds; N. E. Jacobson and G. V. Loomis; Bldg. I.

4:00 - Therapeutic Conference; Conference Room, Bldg. I.

Tuesday, February 27

Medical School and University Hospitals

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

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

1:00 - 2:00 Physiology Seminar on Cardiac Metabolism; 129 Millard Hall.

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

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

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

5:00 - 6:00 X-ray Conference; Eustis Amphitheater, U. H.

8:00 - Journal Club; E-101, U. H.

Ancker Hospital

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

Minneapolis General Hospital

8:00 - 9:00 Pediatric Rounds; Forrest Adams; 4th Floor Annex.

8:30 - Pediatric Allergy Rounds; Dr. Nelson; 4th Floor Annex.

Tuesday, February 27 (Cont.)

Veterans Administration Hospital

8:45 - Surgery Journal Club; Conference Room, Bldg. I.

8:30 - 10:20 Surgery Conference; Seminar Conference Room, Bldg. I.

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

10:30 - 11:50. Surgical Pathological Conference; Lyle Hay and E. T. Bell.

10:30 - Surgery Tumor Conference; Conference Room, Bldg. I.

1:00 - Chest Surgery Conference; J. Kinsella and Wm. Tucker; Conference Room, Bldg. I.

1:30 - Liver Rounds; Samuel Nesbitt.

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

3:30 - 4:20 Clinical Pathological Conference; Conference Room, Bldg. I.

Wednesday, February 28

Medical School and University Hospitals

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Allen Judd and L. G. Rigler; Todd Amphitheater, U. H.

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

12:00 - 1:00 Radio-Isotope Seminar; Literature Review -- Present Status of Use of Radio Cobalt; H. W. Stone; 113 Medical Sciences.

4:00 - 6:00 Ophthalmology Seminar; Todd Room, 5th Floor, U. H.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater.

5:00 - 7:00 Dermatology Clinical Seminar; Dining Room, U. H.

8:00 p.m. Dermatological Pathology Conference; Todd Amphitheater, U. H.

Ancker Hospital

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

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

9:00 - 10:00 Pediatric Rounds; J. D. Tobin; 5th Floor Annex.

11:00 - 12:00 Pediatric Rounds; Franklin Top; 7th Floor Annex.

12:15 - Staff Meeting; Occupational Therapy Programs for Children; Betty Johnson; 4th Floor Annex.

Wednesday, February 28 (Cont.)

Minneapolis General Hospital (Cont.)

1:30 - Pediatric Rounds; E. J. Huenekens; 4th Floor Annex.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans and Bernard O'Loughlin; Conference Room, Bldg. I.

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

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

Thursday, March 1

Medical School and University Hospitals

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.

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

12:00 - Physiological Chemistry Seminar; Metabolism of One Carbon Fragments; H. M. Cavert; 214 Millard Hall.

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

5:00 - Bacteriology Seminar; 214 Millard Hall.

5:00 - 6:00 X-ray Seminar; Eustis Amphitheater, U. H.

5:00 - 6:00 Radiology Seminar; Radiation Therapy of Malignant Lesions of the Eye; John W. MacDonald; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatrics Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

*8:00 p.m. Clarence M. Jackson Lecture: "Fractures About the Hip -- Early and Late Therapy"; Carl E. Badgley; Museum of Natural History Auditorium.

Minneapolis General Hospital

8:00 - 9:00 Pediatric Rounds; Forrest Adams, 4th Floor Annex.

11:30 - Pathology Conference; Main Classroom.

1:00 - 2:00 EKG and X-ray Conference; Classroom, 4th Floor Annex.

2:00 - 4:00 Infectious Disease Rounds; 8th Floor.

4:00 - 5:00 Infectious Disease Conference; Large Classroom, 8th Floor.

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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.

Thursday, March 1 (Cont.)

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff.

9:15 - Surgery Grand Rounds; Conference Room, Bldg. I.

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

1:00 - Chest Rounds; William Stead.

Friday, March 2

Medical School and University Hospitals

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

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Carcinoma of the Stomach; Edward E. Mason; Powell Hall Amphitheater.

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

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

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

4:00 - 5:00 Clinical Pathological Conference; A. B. Baker; Todd Amphitheater, U. H.

4:15 - 5:15 Electrocardiographic Conference; 106 Temporary Bldg., Hospital Court, U. H.

5:00 - Urology Seminar; Potassium Ion Balance; Gerald Evans; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

9:00 - 10:00 Pediatric Rounds; J. D. Tobin; 5th Floor Annex.

9:30 - Surgery-Pediatric Conference; O. S. Wyatt and T. C. Chisholm; 4th Floor Annex.

11:00 - 12:00 Pediatric Rounds; Franklin Top; 7th Floor Annex.

Friday, March 2 (Cont.)

Veterans Administration Hospital

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

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

1:30 - Chest Conference; Wm. Tucker and J. A. Myers; Ward 62, Day Room.

3:00 - Renal Pathology; E. T. Bell; Conference Room, Bldg. I.

Saturday, March 3

Medical School and University Hospitals

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

9:00 - 9:50 Medicine Case presentation; C. J. Watson and Staff; E-221, U. H.

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

9:15 - 10:00 Surgery-Roentgenology Conference; J. Friedman, O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

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 - Anatomy Seminar; A Review of Some Histochemical Studies on Striated Muscle, Richard H. Swigart; The Use of Azo Rubin-S in Study of Hepatic Injury, Dennis J. Kane; 226 Institute of Anatomy.

Ancker Hospital

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

Minneapolis General Hospital

8:00 - 9:00 Pediatric Rounds; Forrest Adams; 4th Floor Annex.

11:00 - 12:00 Pediatric Clinic; Charles May; Classroom, 4th Floor Annex.

Veterans Administration Hospital

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

8:30 - Hematology Rounds; P. Hagen and E. F. Englund.