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Hypoglycemosis In Infancy

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I. A REVIEW OF HYPOGLYCEMOSIS IN INFANCY

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The purpose of the present program is to review the results obtained from an extension of our previously reported studies on spontaneous hypoglycemia in infants and young children[1]. The grave significance of the clinical problem of severe hypoglycemia in this age group and the value of early therapy have become more apparent as additional data have become available.

That normal full-term as well as premature infants tend to have lower fasting blood sugar levels during their first few days of life than those found in older infants, children or adults has long been recognized. In most instances this tendency is so mild and transient, however, that little attention need be given to it. On the other hand, more severe forms of spontaneous hypoglycemosis (hypoglycemic state or syndrome) with such manifestations as hunger, pallor, weakness, sweating, torpor or irritability, muscular tremors, transient strabismus and generalized convulsions, undoubtedly are far more frequent than we formerly believed. The vagueness of the manifestations and the frequent transitory nature of the disturbance make clinical diagnosis in the earliest stages somewhat difficult. Not only the mildness of the early symptoms, but their rapid response to the ingestion of small amounts of carbohydrate-containing food as well, reassures the nurse or parents to such a degree that medical assistance may not be sought until the symptoms become extremely severe. Such a condition in children, or even in adults, may remain mild or may disappear completely and never result in serious handicaps to the individual. The occasional occurrence of hypoglycemic symptoms in any age group might easily be misinterpreted as manifestations of a purely functional psychological disturbance or psychoneurosis, unless the patient or his physician is particularly aware of the possibility of a hypoglycemic state, or unless the blood sugar level determined in the course of a routine examination is found to be in the hypoglycemic range.

Severe, persistent hypoglycemia producing episodes of coma, repeated convulsions, and irreversible central nervous system damage, when allowed to continue, is fortunately much less common. However, as found by our experience, its occurrence under the age of five years, is far more frequent than that of diabetes mellitus and if allowed to go untreated, it may cause death or its residual effects on the nervous system of the living child may be devastating.

The primary pathological condition, which is presumably responsible for the spontaneous hypoglycemia, is at times readily ascertainable by menas of the history and findings from the physical examination, but must be confirmed by study of the carbohydrate metabolism. In many cases, however, elaborate diagnostic tests, including surgical explorations, do not yield a definitive explanation of the primary pathogenesis. These are patients suffering from so-called "idiopathic" hypoglycemia.

Any classification of hypoglycemic disorders must consider the central role of insulin[2], whether there is an absolute hyperinsulinism, as from tumors or hyperplasia of the pancreatic beta cells, or a relative hyperinsulinism secondary to deficiency of one or more of the factors opposing insulin action. This concept was emphasized in a previous publication[1]. The etiological classification by Wauchope[3] is in agreement with this concept.

CLASSIFICATION OF HYPOGLYCEMIA
(modified after Wauchope[3])

I. True hyperinsulinism (insulin excess):
   A. Hyperactivity of islands of Langerhans:
      1. Physiologic hyperactivity (induced by excessive carbohydrate intake).
      2. Hypertrophy or hyperplasia of islet beta cells. (compensatory or pathologic).

3. Tumor arising from beta cells.

B. Insulin administration.

II. Relative hyperinsulinism:

A. Lack of dextrose precursor substances:

1. Diminished absorption from gastrointestinal tract:
   a. Starvation.
   b. Excessive loss from vomiting, diarrhea, or fistula, with and without infection (combination of diminished food intake and increased metabolism).
2. Liver abnormalities (decrease in glycogen stores or mobilization).

B. Lack of development or breakdown of regulatory mechanism:

1. Normal newborn or premature infant.
2. Endocrine imbalance: adrenal, pituitary and thyroid insufficiency. (Possibly congenital absence or relative deficiency of hyperglycemic - glycogenolytic factor of alpha cells.)
3. Intracranial injury.
4. Idiopathic or cryptogenic form.

Included in the category of relative hyperinsulinism are the following: (1) extensive liver disease such as hepatic necrosis, atrophy or carcinomatosis and glycogen storage disease (von Gierke's disease); (2) destructive or atrophic lesions of the anterior pituitary; (3) adrenal cortical hypofunction and (4) conditions of starvation, increased loss or decreased absorption from the gastrointestinal tract and kidneys; (5) the absence of or decrease in pancreatic alpha cells[4] with decrease in hyperglycemic-glycogenolytic factor (glucagon[5]) may also result in relative insulin excess.

Into the category of relative hyperinsulinism may also fall the condition, or group of conditions, designated as "idiopathic" hypoglycemia. This group of hypoglycemic patients includes those in whom the usual diagnostic procedures reveal no specific organ, systen or hormonal etiology but who are found to have either increased sensitivity to injected insulin or impairment of the normal response to hypoglycemia. It is into this little-understood group that the largest number of hypoglycemic dhildren fall, according to our experience.

A brief survey was recently made of the cases of spontaneous hypoglycemia seen in the Pediatric wards of the University of Minnesota Hospitals. Hospital charts on all Pediatric patients having spontaneous hypoglycemia or illnesses likely to result in hypoglycemia were examined. This survey covered the period from 1935 to the present time.

Not included in the total of 38 cases found are those patients on whom a clinical diagnosis of hypoglycemia was made merely on the basis of history or physical findings without conclusive laboratory confirmation. Also excluded were those patients on whom a single blood glucose value fell within the hypoglycemia range but was either not repeated or was not substantiated by further determinations.

The primary diagnosis in the 38 patients known to suffer from spontaneous hypoglycemia were as follows:

1. Adrenal insufficiency		6
a. Addison's disease	5	
b. Congenital adrenal hyperplasia (female psudohermaphrotitism)	1	
2. Glycogen storage disease (von Gierke's type)	3	3
3. Galactosemia		1
4. Hypothyroidism (cretinism)		1
5. Panhypopituitarism (dwarf)		1
6. Solitary beta cell adenoma of pancreas		1
7. "Idiopathic" hypoglycemosis		25
		38

In a large majority of these young children with hypoglycemia, it may be noted that a specific etiologic diagnosis could not be made. In reviewing these cases we were unable to find evidence of diminished absorption from the gastrointestinal tract, renal glycosuria, or hepatic disease (see classification above). We have, assumed that these cases

fell into the category of relative hyperinsulinism due to lack of development or breakdown of the normal carbohydrate regulatory mechanisms. Metabolic studies on the patients with glycogen storage disease and galactosemia were presented at the Hospital Staff Meeting last year. The one child with a pancreatic adenoma was successfully operated on by Dr. Wangensteen[6] and has been completely symptom free since surgery.

Those patients having adrenal insufficiency were treated with specific replacement therapy (adrenocortical hormones), as was the cretin (thyroid extract), with the expected rise in their fasting blood glucose levels. ACTH therapy was ineffective in these cases.

The 25 patients with "idiopathic" spontaneous hypoglycemia include 18 males and 7 females. When these patients were grouped according to the age of onset of definite hypoglycemic signs or symptoms, it was found that most of them had their initial difficulty in the period of infancy, varying from a few days of age to 2 years.

Age at onset of clinical hypoglycemia

Birth	-	2 weeks	4	
2 weeks	-	6 months	7	
6 months	-	2 years	10	21
2 years	-	5 years		3
5 years	-	10 years		1
10 years	-	15 years		0
				25

The presenting complaints of these patients indicate that we probably see only the most severe of hypoglycemics in a Hospital clinic. A far greater number of the less severe cases are either never properly diagnosed, recover spontaneously or automatically adjust their own diets by voluntary food selection in such a manner as to avoid serious difficulty.

Presenting complaints in the infantile group:

Convulsions	19
Coma with convulsions	3
Staring, pallor, strabismus, etc.	3

Eleven (11) of the patients included as "idiopathic" hypoglycemics showed a familial or hereditary factor in the etiology. We are aware of the fact that this distribution between hypoglycemic patients with and those without the hereditary trait for this abnormal metabolic tendency is not representative of hypoglycemic patients generally. Although the genetic factor has occasionally been referred to vaguely by other workers, we know of no other study in which the occurrence of multiple cases in a family has been clearly demonstrated.

Treatment of patients with spontaneous hypoglycemia should obviously be aimed at correction of the underlying etiologic disorder when this can be determined. In cases of true hyperinsulinism, due to insulinogenic tumors of the pancreas, surgical extirpation undoubtedly offers the best hope for permanent relief. Partial pancreatectomy may be required in hyperplasia of the pancreas. Specific replacement therapy is undoubtedly indicated in cases of adrenal, thyroid or pituitary insufficiency. Dietary measures, such as the use of a high protein, relatively low carbohydrate diet and the avoidance of prolonged fasts may also aid in prevention of recurrence of alimentary hypoglycemic symptoms encountered most frequently in adults (as described by Conn[7]). Patients with "idiopathic" hypoglycemia, whose symptoms are transient and very mild, usually respond well to a planned dietary regime alone. Other patients with severe protracted hypoglycemia due to unknown cause have often been treated by subtotal pancreatectomy with response varying from transient to complete and permanent relief of hypoglycemia. When this relief is but temporary and severe hypoglycemia recurs, the more conservative measures may produce better results than they did before operation. Alloxan was used successfully in one severe case by Talbot and co-workers[8]. However, patients in this category, especially infants, may not require partial pancreatectomy, according to our more recent experience. As reported previously, therapy with adrenocorticotropin (or Cortisone and hydrocortisone in much larger doses) has proved highly effica-

cious in such cases.

Since our first staff meeting report (October, 1949) on the effects of ACTH in five cases of severe, non-Addisonian, spontaneous hypoglycemia, other clinicians in widely scattered regions have reported therapeutic results similar to our own and further observations of the kind have also been made in this Clinic. The primary purpose of the present report is to summarize the latter.

Follow-up studies on the five young patients previously reported have yielded a number of noteworthy facts, as follows: (1) The importance of the newly observed genetic factor has become still more striking in the etiology in these patients. The mother of three of them, all males (., ., and .), subsequently gave birth to a female infant ( ), who developed classical signs of severe and persistent hypoglycemia during the first few days of life. A second female infant was born to the parents of the other two children (and .). Up to the present time (age 9 months) she has continued to show normal blood sugar and no symptoms of hypoglycemia. However, two male cousins of J.G. and , . and ., likewise born in the interim, developed typical signs of severe hypoglycemia at the ages of four months and twelve months respectively. These family trees have been reported elsewhere[9]. (2) Histological sections of the pancreas of each of the two children, . and ., (brother and sister), subjected to subtotal pancreatectomy, when stained by the special technique of Gomori, showed the absence or extreme sparcity of alpha cells in the islets of Langerhans[4].

This anomaly is interpreted tentatively as a possible cause of the hypoglycemia in these two patients in light of the belief of some authorities that the hyperglycemic-glycogenolytic factor (HGG or glucogan), isolated from the pancreas, may be elaborated by the alpha cells. Comparable examinations of pancreatic sections from sporadic cases of supposedly "idiopathic" spontaneous hypoglycemia have not shown this or any other anomaly of the pancreas. None of the other familial cases in our series have been subjected to operation because of their satisfactory responses to therapy with adrenocorticotropin. (3) , the most severely affected patient in the original series, was treated with ACTH in gradually diminishing doses of the hormone administered at gradually increasing intervals of time over a period of one year, without use of a special diet. (See Table.) At the end of that time, October, 1950, the therapy was discontinued without recurrence of abnormal fasting blood sugar levels. The patient's schedules of mental and physical growth and development were maintained at entirely normal levels throughout the year of treatment and have continued so without further therapy up to the present date (October, 1953). No complications referable to the small doses of hormone used were ever observed and no tendency to refractoriness developed.

Other patients of the original series have been treated much less consistently with ACTH than , because of the less-than-ideal cooperation of the parents in some instances, and because of the expense involved, but all have shown marked improvement without resort to partial pancreatectomy. One additional two-month-old infant with a severe degree of hypoglycemia and numerous convulsions, who was found to respond entirely satisfactorily to therapy with corticotropin during a week-long test period, was subjected to partial pancreatectomy because it was necessary for her parents to return to their home which was in an inaccessible region in northern Canada. Relief from all symptoms and signs of hypoglycemia was prompt following the operation but improvement lasted only three weeks, when the fasting blood sugar returned to hypoglycemic levels and convulsions recurred. Corticotropin was again found to be efficacious in maintaining normal fasting blood glucose levels

During the past 18 months six additional young infants with spontaneous hypoglycemia of severe grade (two sporadic cases of unknown etiology, one typical cretin, and three belonging to two of our affected families) have been treated with

ACTH, all but the cretin, over a period of several months each to determine the long-time effect of the hormone without the confusing influence of partial pancreatectomy. The results of these case studies are summarized below.

Case 1 - ___. The patient with cretinism, an infant less than three months of age during the period of study, appeared to be totally athyreotic. The degree of fasting hypoglycemia was marked. Repeated therapeutic tests with adrenocorticotropin, before thyroid replacement therapy was instituted, showed the patient to be totally unresponsive to the hormone. Nor was there a significant increase in nitrogen excretion during the period of its administration.

The sugar tolerance curve obtained by Dr. Ulstrom was of the prolonged hypoglycemic type. ACTH therapy alone had no measurable effect on the shape or the height of the curve.

When thyroid extract was given instead of the ACTH, however, the fasting blood glucose level rose within a few days to normal and remained normal so long as this treatment was continued.

Repetition of the intravenous glucose tolerance test, after the optimum response to thyroid therapy had been attained, showed the curve to rise to normal at about the fourth hour of the test instead of continuing at a hypoglycemic level for a much longer time as it had done previously. When ACTH was given in addition to thyroid therapy, the repetition of the glucose tolerance test showed the fall in the curve to be less precipitous and less extensive and the return to normal much more prompt. The results of this single series of tests were tentatively interpreted as indicating the probability that thyroid function is essential for gluconeogenesis, at least in cretinism, and that exogenous ACTH (or adrenal cortical hormones) may be essential for optimal effects in the cretin. The patient's general progress appeared to be satisfactory, however, as a result of thyroid therapy alone.

Case 2 - , sister of , , and (hypoglycemic siblings), was first admitted to the University of Minnesota Hospital at the age of 5 days with the diagnosis of severe spontaneous hypoglycemia (probably congenital) made on the first day of her life by our alert colleague, Dr. W. S. Wright, who found her first blood glucose to be 22 mg per 100 ml. at the hospital in which she was born. Her brief history indicated that on the second day after birth she became irritable, then lethargic and had a generalized convulsion. The blood sugar, determined near the time of the seizure, was found to be 9 mg per 100 ml. ACTH therapy was instituted promptly and regular formula feedings were begun. The fasting blood sugar on the third day was found to be 85 mg per 100 ml. No further manifestations of hypoglycemia were observed before the patient was transferred to the University Hospital for more intensive study.

When ACTH therapy was discontinued, the fasting blood sugar fell to 15 mg per 100 ml., confirming the original diagnosis of spontaneous hyperglycemia. Upon resumption of ACTH therapy the blood sugar values again rose to more nearly normal levels. Subsequently, after a number of abrupt interruptions of her therapy for additional metabolic studies, she began to manifest diminished blood sugar response to the same dose of hog ACTH given originally, as if she might be developing an antihormone reaction or had acquired some impairment of her thyroid or adrenocortical function. The responsiveness to corticotropin obtained from the sheep pituitary gland was found to be no greater than that from the hog. Addition of thyroid therapy or cortisone resulted in no increase in her sensitivity to ACTH.

Since she showed no serious symptoms of hypoglycemia, despite fasting blood sugar levels between 30 and 52 mg., she was discharged to her home without hormone therapy. At that time she suffered from an intractable, low grade fever presumably due to an infection of the urinary bladder for which various antibiotic and sulfonamid drugs were given with gradual improvement.

After a period of several months at

home without hypoglycemic symptoms, she again began to show manifestations of hypoglycemia including two convulsions. Upon her return to the Hospital at that time the fasting blood sugar values were found to be much lower than before discharge. ACTH in gel in moderate doses was then found to relieve symptoms completely and maintain the fasting blood sugar levels between 47 and 83 mg per 100 ml. When last seen, the patient had continued to remain symptom-free on small doses of the ACTH gel preparation once daily. Her growth and development are reported to be essentially normal.

Case 3 - a cousin of . and ., was admitted on Feb. 2, 1953 at the age of 4 months. He was apparently well until two weeks prior to admission at which time he was noted to have twitchings of his arms and legs before his morning feeding. This was relieved by the meal. A minor convulsive episode recurred several days later. Because the family was aware of the possibility of hypoglycemia, a blood sugar was determined promptly. This was found to be 44 mg per 100 ml. (Folin-Wu method). He had one further episode consisting of incoordinated eye movements and myoclonic jerks prior to admission. Two further fasting blood sugar determinations made elsewhere were reported as 52 and 47 mg per cent. (Folin-Wu method). He was placed on a high protein diet of strained meats and egg yolk and referred to the University of Minnesota Hospital.

Admission physical and neurological examinations were completely negative. Laboratory findings were all normal, except for two consecutive fasting blood glucose values which were 16 mg per 100 ml. After an initial short control period, he was placed on plain corticotropin (ACTH), 10 mgms every 6 hours for 12 days. His fasting blood glucose promptly rose to normal levels. On this dose hyperglycemic levels were inadvertently produced. The corticotropin was, therefore, reduced from 40 to 20 mg. per day for ten days, after which additional 15 mg. daily of ACTH gel was given for 22 days without return of hypoglycemia. While on therapy no further clinical hypoglycemia was noted. Because this infant developed a frank, clinical picture of Cushing's syndrome due to overdosage with ACTH, the dosage of the hormone was cautiously decreased over an additional period of two months from 10 mg to 1 mg. daily. The ACTH gel was then discontinued entirely. The baby was placed on a high protein, relatively low carbohydrate diet with one small feeding being given at 4:00 a. m. to insure the absence of morning hypoglycemia while the complication subsided. Under the latter regime his fasting blood glucose fell slowly to a range between 17 and 43 mg. per cent, and on two occasions drowsiness, uncoordinated eye movements and muscular twitchings were noted. He was then placed on ACTH gel, 2.5 mg. daily for an additional month. His fasting blood sugar during this period varied from 108 to 186 mg. per 100 ml. (Folin-Wu), as reported from the laboratory of the family physician. Since that time (one month ago) he has received no hormone therapy and has had no evidences of hypoglycemia. The Cushingoid signs have disappeared and the infant now appears to be physically and neurologically normal. It is obvious now that this young infant was inadvertently given too large doses of the hormone during the first few weeks of his treatment.

Case 4 - was admitted to the University Hospital May 28, 1953 at the age of 3 months with a diagnosis of spontaneous hypoglycemosis made by a colleague in St. Paul. He was delivered by Cesarean section at the end of an uneventful full-term pregnancy, the sixth child in the family. Twenty minutes after birth he had a generalized convulsion. Following this he had from 10 to 30 convulsions daily. He was treated with anticonvulsant medications for approximately one month with little or no success before a blood sugar determination revealed the existence of marked hypoglycemia. He was therefore placed on ACTH therapy by the referring physician with complete cessation of convulsions. Side effects of ACTH therapy (secondary Cushing's syndrome) over a two month period were feared and the patient was referred to this Hospital for further evaluation and treatment.

His family history was negative except

for the fact that his father's uncle had had convulsive episodes in infancy suggestive of hypoglycemia which were apparently relieved by ingestion of sugar.

Physical examination of ., a well-nourished and well-developed infant, revealed no abnormality except for a slight suggestion of excessive corticotropin therapy. The first blood glucose drawn on admission was 15 mg. per cent. Therefore, the patient was immediately placed on ACTHAR gel, 10 mg. every 12 hours. The next blood glucose, drawn 36 hours after corticotropin therapy was begun, was 148 mg. per 100 ml. Over a 19-day period the dose of the drug was gradually reduced to 1 mg. per day. The fasting blood glucose during that time ranged between the extremes of 30 to 180 mg. per cent. The patient was symptom-free and the slight evidence of side effects to corticotropin disappeared. While this patient was on one mg. of ACTHAR gel per day, the blood glucose fell to levels as low as 24 and 15 mg. per cent. When the dose was increased to 2 mg. per day, the fasting blood sugar generally remained normal, but occasionally dipped into the hypoglycemic range. He was discharged on this dose and subsequent blood sugar levels have ranged from 60 to 110 mg. per cent. When last examined in September, 1953, he was found to be an alert, happy infant with only slight delay in motor development. He occasionally has slight drowsiness and irritability which quickly respond to sugar by mouth. This is not certain evidence of hypoglycemia. It would appear that this patient may soon be able to dispense with the hormone therapy.

Case 5 - , a 15-month-old boy, was first seen on November 26, 1952. He had been regarded as normal up to the age of 9 months when he had a generalized convulsion. Following this, he had frequent, severe and protracted seizures. After one particular series of seizures he was noted to have unsteady head and hand movements, which have persisted. He was treated with antiepileptic medications with little, if any, benefit. One month before admission to the Hospital, he began having episodes of irritability followed by prolonged stupor. The blood sugar was determined and found to be "very low". He was then placed on Cortisone and phenobarbital by the local physician. On this therapy the irritability persisted, but there was a decrease in the frequency of convulsions.

His family history was noncontributory. Physical examination revealed a severely retarded, somewhat athetoid infant with no other obvious defects. The blood sugar on admission was 38 mg. per cent and on subsequent days ranged from less than 15 to 30 mg. per cent. An intravenous glucose tolerance test (0.5 gm/kg body weight) revealed a fall of the curve to hypoglycemic levels at 3 hours where it persisted.

On Dec. 16, 1952 he was tested on Cortisone therapy, 12.5 mg. every 6 hours. No effect on the blood sugar was observed with this dosage of Cortisone over a period of 11 days. The dose of Cortisone was then doubled. Ten days at this dosage resulted in no change in fasting blood sugar levels. On Jan. 12, 1953 ACTH, 10 mg. every 6 hours, was started. Later this was changed to 20 mg. each 12 hours of ACTHAR gel. On ACTH his blood glucose level rose abruptly to the normal range and throughout the treatment period of 36 days varied from 54 to 98 mg per cent. When the ACTH was discontinued, his blood sugar fell very slowly and after one month reached levels of 42 to 50 mg. per cent. On March 18, 1953 he was placed on hydrocortisone (Compound F), 10 mg. every 8 hours for 6 days, without elevation of his fasting blood glucose values. The dose was then increased to 40 mg. every 8 hours with a prompt rise in the blood glucose to the normal range. After 8 days of treatment the hydrocortisone was discontinued and he was again placed on ACTHAR gel, 10 mg. twice daily. Since that time, his fasting blood sugar levels have remained above 70 mg per cent. He was re-admitted July 7, 1953 - 45 days after discharge, with definite side effects of overdosage of adrenal corticoids and ACTH apparent. The ACTH dosage was tapered over a one-week period when it was discontinued. Since then, he has had no fasting blood sugar values below 50 mg. per cent. There has been no change noted

in the athetoid movements or the retardation. Despite the apparent "cure" of the hypoglycemia, the child still has frequent myoclonic and petit mal-like seizures, which have been well controlled with barbiturate therapy. Nevertheless, he does not stand alone or talk at the age of 20 months. The neurological complications developed by this infant are considered to have resulted from the prolonged period of severe hypoglycemia and convulsions before adequate therapy was instituted.

Case 6 - , 18-month-old son of one of our Pediatric staff members and cousin of . and ., was admitted to the University of Minnesota Hospital on September 25, 1952 for more intensive study of his spontaneous hypoglycemia, which he was known to have had in less severe form since the age of 12 months. At the latter mentioned time, this apparently healthy infant was observed on one occasion to have a transient episode in which his "eyes rolled back and were unsteady". This was before a meal terminated the abnormal reaction. A similar episode recurred at the age of 15 months and again at 17 months. The last attack was accompanied by urinary incontinence and mild diarrhea. All attacks occurred just prior to a meal.

When studied by his father and associates at the Northwestern Hospital, the fasting blood glucose levels were found to be very low. Glucose tolerance tests showed the curves to be comparatively low, falling to hypoglycemic levels early and persisting for many hours. The physical and mental status were normal essentially.

ACTH therapy was instituted on Oct. 2, 1952 when the fasting blood sugar levels varied between 23 and 40 mg. per cent during a preliminary control period. On the second day after treatment was begun with 10 mg. plain ACTH every 6 hours, the fasting blood glucose was found to be 50 mg. per 100 ml. Fasting glucose values ranged between 70 and 90 mg. per 100 ml. during the following 9 days, after which medication was interrupted for a trial period of 7 days. The fasting blood sugar values again fell to hypoglycemic levels, varying between 25 and 40 mg. per 100 ml.

When ACTH therapy was resumed the daily dosage was reduced to 25 mg. of the longer acting ACTH gel given in two equal doses at 12-hour intervals. The blood sugar again rose to normal levels on the third day and remained normal thereafter while the dosage of ACTH gel was gradually reduced to 5 mg. once daily. Subsequently the hormone was given in a dosage of 2 mg. every second day. The patient has remained entirely symptom-free. His physical and mental development and growth have been entirely normal. Within the near future the hormone will be withdrawn completely for a test as to his need for further use of the therapy.

COMMENT

The foregoing brief report of our recent studies on spontaneous hypoglycemia calls for additional comment to emphasize certain points which we regard as being of special importance.

It has become quite apparent to us that the frequency with which severe spontaneous hypoglycemia occurs is far greater than was formerly suspected, particularly its occurrence in young infants. The condition is undoubtedly misdiagnosed in many infants or even in older individuals. That severe brain damage results from prolonged hypoglycemia and frequently recurring, long lasting convulsions or coma, as described by Dr. A. B. Baker[10] and others in pathological studies following insulin-induced shock, is illustrated clearly by two of the most seriously neglected patients in our series of cases.

The latter were both known to be entirely normal infants, as regards their mental and motor development, up to the age of eight or nine months, that is, before the onset of their severe hypoglycemia. Lack of correct diagnosis allowed both infants to suffer from severe hypoglycemic reactions including convulsions for many months before the immediate cause of their symptoms was recogniz-

EFFECTS OF PROLONGED TREATMENT OF SEVERE SPONTANEOUS HYPOGLYCEMOSIS

WITH ACTH IN DECREASING DOSES (. Age 13 months)

Age	Ht.	Wt.	Days	ACTH		Fasting Blood Glucose* mg. per 100 ml.		
				Dose	Hrs. bet.			
mo.	cm.	kg.	Obsvd.	mg.	doses	high	low	mean
13	73.2	7.5	10	0	---	28	8	18
			4	9	6	94	48	78
			10	0	---	58	38	47
14	73.3	7.8	2	0	---	30	24	27
			3	9	6	90	40	72
			16	18	48	67	38	53
15	74.0	8.5	171	15	48	68	28	52
20	78.5	10.1	96	10	48	60	30	51
			120	20	96	62	38	55
26	85.9	11.1	32	10	96	68	44	56
30	90.0	12.4	166	0	---	85	43	53

* Samples taken at maximal time after ACTH administration

ed and effective therapy was instituted. It has long been the practice of physicians, ourselves included, to attribute such symptomatology to probable birth injury, even when the obstetrical history gave no definite support to such an assumption. Subsequently recognized residual damage to the nervous system, often manifested by athetoid or spastic movements, as well as severe motor and mental retardation, has appeared to confirm the mistaken original diagnosis of "traumatic epilepsy from obstetrical injury". While the latter is undoubtedly a genuine entity of frequent occurrence, we now believe that many inadequately studied cases of spontaneous hypoglycemia are inadvertently being relegated to that category.

The seriousness of the tragedy involved in a misdiagnosis in such an infant with severe hypoglycemia is heightened by the knowledge that means for early diagnosis and treatment are now available. Proper sampling of the blood at the proper time for determining the glucose level will reveal the presence of hypoglycemia with certainty. It must be recognized that the level of sugar varies considerably throughout the day, often requiring that several samples be investigated before a final conclusion regarding the diagnosis can be drawn. Many physicians, who may suspect the existence of hypoglycemia on clinical evidence in patients having convulsions, are accustomed to draw a sample of blood during or immediately following a generalized seizure. In such an instance the true diagnosis is not infrequently eliminated from the physician's mind by this single test, because the glucose value reported by the laboratory is found to be within the limits of nor-

mal. It should be remembered that a generalized convulsion due to any cause may itself result in an elevation of blood sugar, unless the glycogen stores in the muscles, as well as those in the liver, have previously been almost completely depleted. Blood samples should be obtained, therefore, before seizures as well as before meals.

The importance of the genetic factor in the etiology, especially in those patients with otherwise undetermined cause, is emphasized by our experiences with three entirely unrelated families. In one family, not included in previous reports from our own clinic, two members of a set of triplet girls have spontaneous hypoglycemia. These two afflicted individuals are identical or single-ovum twins. The third non-identical member of the triplet, at the age of 7 years, has shown no evidence of a hypoglycemic tendency, even when subjected to special tests for the same. The siblings and other members of a family containing a patient with demonstrated hypoglycemia should be examined for the hypoglycemic trait.

In our previous Staff Meeting Report on this subject (1949), one case ( ) of hypoglycemia, presumably due to pituitary insufficiency, was briefly referred to as having been "cured" of this aspect of his disorder by intensive intramuscular administration of a crude multiple-hormone preparation from the pituitary gland (Polyansin-Armour, no longer marketed). The patient, a pituitary dwarf, was referred to us in 1940 primarily because he had been suffering from "nervousness" and frequent morning convulsions for several months. Since Young[11] had shortly before reported the production of permanent diabetes mellitus in rabbits, and later in other species of animals, by repeated injections of a crude extract of anterior pituitary gland substance, an attempt to repeat the experiment in this patient up to the point of countering the fasting hypoglycemia without producing diabetes was made. After the daily dosage of the polyvalent extract was cautiously increased to approximately three times that recommended by its manufacturers, the patient's fasting blood sugar was found to rise from the average level of 20 mg. per 100 ml. to between 50 and 93 mg. and, with this return of the fasting levels of blood glucose to normal, there was cessation of the convulsions. Although the medication was discontinued after a few weeks, because no measurable growth or genital response was produced, convulsions or other symptoms of hypoglycemia have never recurred and no signs or symptoms of diabetes have been observed by the parents during the subsequent time (12 years). Growth hormone is now known to be capable of producing diabetes by its effects on peripheral tissues, especially the Islets of Langerhans.

The old data from that inadequately studied case are reviewable briefly here because they may have some significance for the interpretation of our most recent observations. It will be observed that, contrary to our earlier expectations, the beneficial effects of ACTH therapy do not appear to be temporary only, as in the case of insulin therapy in diabetes mellitus. Virtually all of t the non-Addisonian, nonthyreoprevic patients with severe hypoglycemia, who have been treated consistently with adrenocorticotropic hormone, have shown the tendency to require smaller and smaller doses of the substance to maintain normal fasting blood glucose levels over a period of months. In a number of unusually severe cases so treated, it has been possible to withdraw medication entirely without the need for special dietary regulation and without recurrence of hypoglycemia, for example, . , . , . and . Some of the latter, after a few months of treatment, have been off ACTH for too short a time to justify final conclusions as to permanent "cure", but hope is justified by the result in the case of B.G. who has shown no hypoglycemia or symptoms since medication was discontinued 3 years ago. She has continued to grow and develop according to the normal schedules for her age.

The bodily changes by which ACTH (or cortisone and hydrocortisone) effects these results are not known with certainty, but the reported effects of these

hormones on the beta cells of the pancreatic islets, appears to be the most likely clue at the present time. Much more work remains to be done before a full explanation of our observations to date can be made.

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II. MEDICAL SCHOOL NEWS

Coming Events

October 15-17	Continuation Course in Diseases of the Chest for General Physicians
October 21	Continuation Course in Dental Problems in Medicine for General Physicians
October 22-24	Continuation Course in Rehabilitation for General Physicians
October 26-31	Continuation Course in Radiation Therapy for Radiologists

* * *

Greetings

If called upon to select a favorite season of the year, the majority of our Faculty would very likely nominate Fall. Minnesota's autumn loveliness, the hunting season, football games, and the resumption of social activities all prove to be invigorating--and at times distracting--influences. Most stimulating, however, is the return of students to the classrooms, laboratories, and hospital wards, for it is the discomfiting questions posed by students, more than any other experience, which keep us constantly aware of the gaps in our knowledge and which impel us to attempt to narrow those gaps.

For this reason and for many others, it is a real pleasure to extend greetings and best wishes to each member of the entering Freshman class and to each student in the Sophomore, Junior, and Senior classes. We wish also to extend greetings to members of the Faculty and to all Friends of the Medical School.

To our new interns and to our fellows and staff members who have joined the faculty since the close of the last academic year we would like to express a special word of welcome. We are sure that they will derive pleasure from their associations here and from the opportunities they will have of serving patients and students.

This year promises to be a particularly significant one from the standpoint of the expansion of our physical facilities. The Elias P. Lyons Laboratories will be ready for occupancy in the very near future, and the formal dedication will be held next February. Beyond that we are looking forward to July 1, 1954, when the Mayo Memorial Building is scheduled for completion. Indeed, an interesting year is in store.

Minnesota Medical Foundation Day

Thursday, October 1, was Minnesota Medical Foundation Day, perhaps the most memorable Foundation Day yet. Activities got underway at 2:30 p.m. in the Campus Club where a coffee hour for freshman medical students, sponsored by the Medical Students' Advisory Council, was held. Students and faculty turned out in force. The freshmen students heard brief talks by Dr. William Maloney, Assistant Dean; Dr. Wesley W. Spink speaking for the Foundation; and Miss Elsa Proehl, senior medical student speaking for the M.S.A.C.

The annual Foundation Day Lecture highlighted the day's activities. This year's lecture was given by Dr. Thomas Hale Ham, Professor of Medicine, Western Reserve University School of Medicine, Cleveland, Ohio, who spoke on "Looking Ahead for the Physician" at 4:00 p.m. in Owre Amphitheater. A capacity audience heard Dr. Ham deliver a stirring address in which he discussed the future of medicine in a most graphic manner.

Preceding his lecture, scholarships were presented to 14 students by Dr. O. H. Wangensteen, President of the Minnesota Medical Foundation. Scholarships of $500 each were awarded to six members of the freshman class who are graduates of state colleges in Minnesota. These scholarships, made available through the efforts of Dr. Donald J. Cowling, were awarded to the following students: Frank W. Van de Water, St. Paul--Carleton; Miss Genell Knatterud, Minneapolis--Macalester; George Crow, Hot-Springs, South Dakota--St. John's; John Nilsen, Rothsay, Minnesota--St. Olaf; Leland Fairbanks, Harmony, Minnesota--Augsburg; Jerome J. Scherek, St. Paul--St. Thomas. Six scholarships, also $500 apiece, were presented to members of the Sophomore, Junior, and Senior classes. Based on both need and merit, these scholarships were given to: Richard E. Anonsen, Minneapolis, Senior; Donald Mattson, Minneapolis, Senior; Russell Boelke, Minneapolis, Junior; Emil Schulz, Minneapolis, Sophomore; Norval Martensen, Tyler, Minnesota, Sophomore; Sidney Nerenberg, St. Paul, Senior. David Stenzel, Easton, Minnesota, Senior, received a $500 Foundation scholarship which was donated by the Catholic Physicians Guild. One scholarship of $750 was awarded to Royal Hayden, Red Wing, Sophomore, on the basis of a very outstanding academic record.

Recipients of the scholarships and members of the Foundation and their guests dined at the Campus Club at 6:15 p.m. An excellent dinner was followed by a brief business meeting. Dr. Karl W. Anderson was re-elected to the Board of Trustees. Doctors Leo G. Rigler and Charles E. Rea were nominated and elected to the Board to succeed Doctors E. T. Bell and Vernon D. E. Smith who were not eligible for re-election. Both of the latter have served the Foundation well, and gratitude was expressed to them for their help and unfailing interest.

Dr. Wesley W. Spink, Secretary-Treasurer, gave a resume of the Foundation activities; Dr. Vernon D. E. Smith reported on the accomplishments and plans of the Membership Committee; and Dr. Robert B. Howard gave a report on the "Bulletin of the University of Minnesota Hospitals and Minnesota Medical Foundation." The meeting closed with a brief and inspiring message from Dr. Wangensteen.

* * *

Medical Alumni Association Activities

The Minnesota Medical Alumni Association is planning several things of interest for the coming year. First, of course, is the Homecoming Week Program which includes the following activities:

Thursday, November 5, and
Friday morning, November 6 -- CONTINUATION COURSE IN MEDICAL JURISPRUDENCE (Center for Continuation Study) (Tuition fee: $15.00)

Friday noon, November 6 -- Staff Meeting -- SPECIAL HOMECOMING PROGRAM (University Hospitals) -(Luncheon served beginning at 11:45 a.m.)
Immediately followed by: ANNUAL MEETING OF THE MINNESOTA MEDICAL ALUMNI ASSOCIATION

Friday afternoon, November 6, and
Saturday morning, November 7 -- HOMECOMING CLINICS (University Hospitals)

The Class of 1932-33 is holding its reunion and will take an active role in the Homecoming Clinics.

Saturday afternoon, November 7, 1:30 p.m. -- HOMECOMING FOOTBALL GAME
Minnesota vs. Indiana

The Medical Alumni Association is also preparing a Medical Alumni Directory. All alumni of the Medical School have received or will shortly receive an announcement of plans for the directory and will be invited to purchase one. Since no Medical Alumni Directory has been published since 1942, it is expected that the proposed Directory will serve a real need.

Plans are being made also for another Medical Alumni Luncheon for Senior Medical Students which will be held next spring. The luncheon held last May was an unqualified success, and all who participated felt that it should become an annual event.

The Senior students who graduated in June acted on a suggestion made at that luncheon. On June 2 they met as a group and elected permanent class officers. We are pleased to offer congratulations to the following Alumni Officers of the Class of 1953: President - Dr. Thomas Kirschbaum; Vice-President - Dr. Nathan Sidley; and Secretary-Treasurer - Dr. Eldore Nash. We hope that subsequent classes will follow suit.

* * *

New Faculty Members and Promotions

Joining our Faculty this year are: Doctors J. O'H. Tobin, Visiting Assistant Professor, and K. T. Brunner, Instructor, Department of Bacteriology; Dr. Bo Malmstrom, Instructor, Department of Physiological Chemistry; Doctors Richard W. Anderson, Associate Professor, Maynard Cohen, Assistant Professor, Harold Cohen, Research Associate and Gene Lasater, Instructor, all of the Department of Psychiatry and Neurology.

The following members of our Faculty have received academic promotions:

Promoted to Associate Professor

Wendell H. Hall	Department of Medicine
Robert B. Howard	Department of Medicine
R. Dorothy Sundberg	Department of Anatomy
Roy G. Holly	Dept. of Obstetrics and Gynecology
Richard W. Anderson	Dept. of Psychiatry and Neurology
Robert A. Good	Department of Pediatrics

Promoted to Assistant Professor

Leslie Zieve	Department of Medicine
Frederick H. Van Bergen	Department of Surgery
Leonard F. Peltier	Department of Surgery
Bernard Zimmermann	Department of Surgery
Samuel O. Cornwell	Department of Anatomy
Clarence J. Rowe	Dept. of Psychiatry and Neurology
Lewis W. Wannamaker	Department of Pediatrics

Promoted to Instructor

Horace Zinnemann	Department of Medicine
William Mazzitello	Department of Medicine

Promoted to Instructor (Continued)

William Goodnow	Department of Medicine
Neal Gault	Department of Medicine
Rudi Schmid	Department of Medicine
William D. Kelly	Department of Surgery
Roland D. Meader	Department of Anatomy
Richard G. Hibbs	Department of Anatomy
Rosalind Abernathy	Department of Pediatrics
Eleanor Colle	Department of Pediatrics
Charles Heath	Department of Physiology
Stuart W. Arhelger	Department of Surgery

We offer congratulations and best wishes to each of these individuals. Special congratulations are in order for Dr. Sundberg who also recieved her M.D. in June.

* * *

Symposium

Sponsored by the Minnesota Heart Association and the University, a Symposium on Recent Advances in Cardiovascular Physiology and Surgery was held in Scott Hall on the University Campus from September 14 to 16. Over 650 physicians and scientists in allied fields registered for the program, and comments of those who attended were uniformly favorable. Speakers included: Doctors W. G. Bigelow, Toronto Canada; Clarence Crafoord, Stockholm, Sweden; Lewis Dexter, Boston; Charles T. Dotter, Portland; John H. Gibbon, Philadelphia; Robert P. Glover, Philadelphia; Dwight E. Harken, Boston; W.F.H.M. Mommaerts, Cleveland; Willis J. Potts, Chicago; John W. Remington, Augusta, Georgia; Robert F. Rushmer, Seattle; C. W. Sheppard, Oak Ridge, Tennessee; Torgny Sjostrand, Stockholm, Sweden; Olle Snellman, Uppsala, Sweden; Richard W. Stow, Columbus, Ohio; Henry Swan, Denver; H.J.C. Swan, Rochester, Minnesota; and Dr. E. Wetterer, Munchen, Germany. Many other well-known investigators also participated in the program. The annual George E. Fahr Lecture was presented by Dr. W. F. Hamilton who spoke on "The Physiology of Congestive Failure of the Circulation." The Fahr Lecture constituted an integral part of the Symposium.

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III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

October 12 - 17, 1953

Monday, October 12 (HOLIDAY)

Tuesday, October 13

Medical School and University Hospitals

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

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

12:30 - 1:30 Physiology 114C -- Respiration; E. B. Brown; 129 Millard Hall.

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

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

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

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 6:00 X-ray Conference; Presentation of Cases from Minneapolis General Hospital; Drs. Lipschultz and Heitzman; Eustis Amphitheater, U. H.

Ancker Hospital

8:00 - 9:00 Fracture Conference; Auditorium.

9:00 - 10:00 Medical X-ray Conference; Auditorium.

Minneapolis General Hospital

10:00 - Pediatric Rounds; Spencer F. Brown; Stations I and J.

10:00 - Cardiac Rounds; Paul F. Dwan; Classroom, Sta. I.

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

12:30 - Grand Rounds; Fractures; Willard White, et al; Sta. A.

12:30 - Neuroroentgenology Conference; O. Lipschultz, J. C. Michael and Staff.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

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

1:00 - Neurology Grand Rounds; J. C. Michael and Staff.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Surgery Staff Seminar; Medical Conference Room, Bldg. I.

9:30 - Infectious Disease Rounds; Drs. Hall, Zinneman, and Brown.

Tuesday, October 13 (Cont.)

Veterans Administration Hospital (Cont.)

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

10:30 - Surgery-Tumor Conference; L. J. Hay and J. Jorgens; Conference Room, Bldg. I.

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

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

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

Wednesday, October 14

Medical School and University Hospitals

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

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

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

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

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

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

12:30 - 1:30 Medical Journal Club; Library.

Minneapolis General Hospital

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

9:30 - Pediatric Rounds; Max Seham; Stations I and J.

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

11:00 - Pediatric Seminar; Arnold Anderson; Classroom, Station I.

11:00 - Pediatric Rounds; Erling S. Platou; Station K.

12:15 - Pediatric Staff Meeting; Classroom, Station I.

1:30 - Visiting Pediatric Staff Case Presentation; Station I, Classroom.

2:00 - 4:00 Infectious Disease Rounds; Sta. D.

4:00 - 5:00 Infectious Disease Conference; Wesley W. Spink; Classroom.

Wednesday, October 14 (Cont.)

Veterans Administration Hospital

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

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

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

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

4:00 - Combined Medical Surgical Conference; Drs. Flink and Hay; Conference Room, Bldg. I.

5:00 - Medical Journal Club; Conference Room, Bldg. I.

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

Thursday, October 15

Medical School and University Hospitals

9:00 - 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.

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

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

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 6:00 Radiology Seminar; Report of Meeting of American Roentgen Ray Society; Eustis Amphitheater, U. H.

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

Ancker Hospital

8:00 - 10:00 Medical Grand Rounds; Auditorium.

Minneapolis General Hospital

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

10:00 - Pediatric Rounds; Spencer F. Brown; Station K.

10:00 - Psychiatry Grand Rounds; J. C. Michael and Staff; Sta. H.

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

1:00 - Fracture - X-ray Conference; Dr. Zierold; Classroom.

1:00 - House Staff Conference; Station I.

Veterans Administration Hospital

8:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

Thursday, October 15 (Cont.)

Veterans Administration Hospital (Cont.)

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

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

1:00 - 3:00 Metabolic Disease Conference; Drs. Flink, Heller and Sherman.

Friday, October 16

Medical School and University Hospitals

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

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

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

10:30 - 1:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

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

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Surgical Treatment of Polyposis of the Colon by Total Colectomy; Richard Lillehei and Owen H. Wangensteen; Powell Hall Amphitheater.

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

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

4:00 - 5:00 124 Advanced Neurophysiology Lecture; Werner Koella and Ernst Gellhorn; 111 Owre Hall.

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

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

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

Minneapolis General Hospital

9:30 - Pediatric Rounds; Wallace Lueck; Station J.

10:30 - Pediatric Surgery Conference; Oswald Wyatt, Tague Chisholm; Station I, Classroom.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:15 - X-ray Conference; Oscar Lipschultz; Classroom, Main Bldg.

2:00 - Pediatrics Rounds; Robert Ulstrom; Stations I and J.

Friday, October 16 (Cont.)

Veterans Administration Hospital

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

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

2:00 - Autopsy Conference; E. T. Bell and Donald Gleason, Conference Room, Bldg. I.

Saturday, October 17

Medical School and University Hospitals

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

9:00 - 10:00 Infertility Conference; Louis L. Friedman, David I. Seibel, and Obstetrics Staff; Eustis Amphitheater, U. H.

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

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

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

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

11:30 - Anatomy Seminar; Abnormalities in Orchiectomized Fetuses and their Prevention by Androgen Therapy; L. J. Wells; 226 Institute of Anatomy.

Ancker Hospital

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

Minneapolis General Hospital

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

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

Veterans Administration Hospital

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

8:30 - 11:15 Hematology Rounds; Drs. Hagen and Hoseth;

11:15 - 12:00 Morphology . . . . Dr. Aufderheide; Conference Room.