Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation

Strangulation Obstruction

Volume XXIV | Friday, December 19, 1952 | Number 11

BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
and
MINNESOTA MEDICAL FOUNDATION

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Published weekly during the school year, October to June, inclusive.

The Bulletin is sent to members of the Minnesota Medical Foundation.
Annual membership fee - $10.00.

Address communications to: Staff Bulletin, 3330 Powell Hall, University of Minnesota, Minneapolis 14, Minn.

I. ROENTGEN OBSERVATIONS IN STRANGULATION OBSTRUCTION

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INTRODUCTION

Further reduction in the mortality rate of acute intestinal obstruction must come from better management of the two conditions which cause most present-day deaths: strangulating obstructions and markedly distended late cases of simple obstruction. Better techniques of surgical decompression will permit surgeons to save even late cases of distention.[8,9,10,33,34] The mortality of strangulating obstructions, however, can be reduced only by earlier diagnosis.

It is the common practice to operate upon most cases of intestinal obstruction as soon, after the diagnosis is made, as the patient can be prepared to withstand the surgical procedure. The diagnosis of mechanical intestinal obstruction without elaboration is, therefore, specific enough in most instances. Two groups of problem cases remain. The first group contains those cases of strangulating obstruction in which not only is the strangulation not revealed but the signs of obstruction are disguised. The second group is composed of those cases which should be treated non-operatively, if possible, and would be so treated if it were certain that strangulation was not present. This group includes the poor operative risks, the partial small bowel obstructions (especially if there have been several previous operations), and the inflammatory lesions in which an obstructive component is present. Dennis[8] believes that 35 to 40 per cent of small bowel obstructions could well be treated by non-surgical means if we could but select the proper cases with unfailing accuracy.

DEFINITION

An obstruction is considered to be of the strangulating type if, in addition to a block in the intestinal continuity, there is evidence of compromise of the blood supply. The degree of compromise varies from slight compression of the mesenteric veins, manifested at operation by a slight bluish discoloration of a segment of the bowel, to frank hemorrhagic infarction and gangrene.[14] In most cases of internal strangulating obstructions, a "closed loop" is produced. The bowel lumen is narrowed or blocked at two points by a single constricting lesion which also compresses the mesentery and its vessels. The lumen of the bowel between the two points of compression is open. It is possible to have only a partially closed loop with sufficient vascular compression to produce infarction and certainly one can find almost completely closed loops without any evidence of strangulation at all. (Figure 3). The incarcerated loop tends to be fixed in position. Small closed loops are quite fixed while long loops may have considerable mobility.

Strangulating obstructions may result from a volvulus, an incarcerated hernia, passage of the small bowel through an abnormal opening in the mesentery such as is produced by certain surgical procedures, or a twisting of the bowel around a band such as a Meckel's diverticulum. The most common cause is the presence of adhesive bands at two separated points producing a double obstruction.

PATHOLOGIC PHYSIOLOGY

Whatever the specific cause of the incarcerated or twisted loop, there are two groups of effects: the first arises because the bowel content has stopped moving forward and the second because the bowel is anoxic, either as a result of passive congestion or of ischemia. The effects probably develop in somewhat the following manner. The luminal obstruction is followed by an increase in peristaltic activity with concomitant pain. Gas and fluid accumulate above the point of obstruction. If the lumen at the upper end of the incarcerated loop is only partially obstructed gas will enter the loop. Sometimes, especially in the presence of severe vascular occlusion, there is immediate

spasm of the intestines and the superior segment of the bowel will be devoid of gas or will contain a surprisingly small amount. The presence of moderate amounts of gas or the presence of fluid plus moderate amounts of gas within the fixed loop indicates the likelihood of an incomplete obstruction. At the same time as the lumen is occluded, the mesenteric blood vessels are compressed. The veins are narrowed first, producing a rise in venous pressure but not arterial pressure. For the most part, save for rupture of thin-walled capillaries and veins, extravasation of blood into the lumen of the strangulated bowel and into the free peritoneal cavity is produced early by the elevated capillary pressure working against an impeded venous return. As soon as the venous pressure rises there is progressive anoxia of the bowel musculature producing first hypermotility and then spasm. This is followed, as anoxia increases, by flaccidity and dilatation. The late result of both the distention and the compromised circulation is a thickened overstretched bowel wall, deficient in tone and contractility.[4,13] Frank necrosis and perforation of the intestines may supervene.

REVIEW OF LITERATURE

Reinus[27] studied the cases of mechanical small intestinal obstruction resulting from adhesions seen over a ten-year period at Lenox Hill Hospital, New York City. He attempted to determine the reliability of the commonly accepted clinical and laboratory evidences of strangulation. The material was divided into simple and strangulating obstructions and the latter were separated into the "imminent strangulations", in which the intestine showed any signs of stagnant anoxia, and the "obstruction with strangulation", in which the strangulating process had terminated in infarction. One might call these early and late strangulating obstructions.

Analysis of the physical findings revealed essentially the same changes as have been emphasized by Wangensteen[33] and grouped as the signs of irritability of the parietal peritoneum. Of the clinical laboratory findings, McKittrick and Saris[23] stressed the importance of a rising leukocyte count. In Reinus' material, the leukocyte count was felt to be of little help. The results of roentgenologic diagnosis in his series are of great interest. In no case was the presence of strangulation suggested following roentgen examination. In addition, it is noteworthy that while a correct diagnosis of mechanical obstruction was made in 48 out of 51 cases of simple obstruction, even the presence of obstruction, per se, was not evident in three out of six cases of late strangulating obstruction and in one out of three cases of early strangulating obstruction.

In the surgical literature little emphasis is placed upon the value of roentgenologic signs of strangulation. Cope[6] (1948), Ferguson[11] (1949), Ficarra[12] (1949) and Cole[5] (1950), among others, have written excellent papers on intestinal obstruction, with attention to the diagnosis of strangulation, without any definite indication that roentgen evidence of strangulation is sought or is of value. Indeed many radiologists have felt with Middlemiss[24] that "the problem of the state of an obstruction, acute or chronic, simple or strangulating, is a clinical entity rather than a roentgenological problem."

Many years ago one of us[29] was impressed with the possibility that roentgen evidence of strangulation might be sufficient to permit the separation of such cases from simple obstruction. Continuing experience suggested that some cases of strangulating obstruction demonstrated a rather localized distention of two segments of bowel. In many instances the gas-filled loops of bowel took on a characteristic appearance which was described as the "coffee-bean" sign because the gas-filled lumina were separated by a fairly broad, dense band caused by the edematous bowel walls or possibly by a small amount of peritoneal fluid between the loops of bowel. The presence of fixation, as shown by lack of movement of a loop of bowel on several films made in different positions, was also emphasized.[28]

Frimann-Dahl, in 1944[15], and again in 1951[17] described another roentgen sign. A soft tissue density suggesting a soft tissue mass, caused by intestinal loops almost completely filled with fluid, was considered indicative of strangulating obstruction. He did not believe that localized gaseous distention could occur in a strangulated loop of bowel. In 1949, Crowley and Winfield[7], in an extensive review, stated that gaseous distention of an isolated loop was almost pathognomonic of strangulating obstruction. They added that the finding might not be recognized because of the presence of other gas-filled loops and that it was therefore better seen early. It was their feeling that "the presence of a single loop, if found together with the clinical diagnostic features, may generally be regarded as the strongest evidence of strangulating obstruction." This was in agreement with observations one of us made earlier.[29] Berry[2] described essentially the same findings while Totten[32] discussed and presented an illustration of fluid-filled loops in a patient with a strangulating obstruction. Hunt[19,20] and Lockwood, Smith and Walker[22] noted the absence of normal mucosal markings in a distended segment of small intestine. They stated that, when present, this is a pathognomonic roentgenologic sign of strangulation or embarrassment of the blood supply.

CLINICAL DATA

In order to evaluate the possibilities and limitations of roentgen diagnosis in cases of strangulation, 334 small bowel obstructions seen between 1937 and 1952 in the University of Minnesota Hospitals were studied. Thirty-four instances of internal strangulating obstructions were found. This is an incidence of 10 per cent compared with 5.5 per cent in Baumgartner's series[32] and 18.5 per cent in Frimann-Dahl's group[17]. Twenty-six cases were available for study and these form the basis of this analysis. There were 21 cases due to peritoneal adhesions, two cases in which there was small intestinal volvulus and peritoneal adhesions around an anterior colostomy stalk, two cases of volvulus alone and one case of jejunal volvulus complicated by the presence of four enteroceles along the anti-mesenteric border of the jejunum.

Table I indicates the frequency of the roentgenologic signs. In almost 60 per cent of cases the closed loop is demonstrable. Fixation of a portion of the small intestines is noted in 50 per cent and loss of mucosal markings in 50

TABLE I

ROENTGENOLOGIC FINDINGS IN 26 CASES OF STRANGULATING OBSTRUCTION

Closed loop		15
A. Gas distended ("coffee bean" sign)	7	
B. Fluid-filled ("pseudo-tumor" sign)	8	
Fixation of loop		13
Loss of mucosal pattern		12
Absence of gas above obstruction		6
Lack of decompression of localized segment following suction		3
Presence of small amount of gas in colon		17

per cent. Three cases showed only the signs of simple obstruction and in one even mechanical obstruction could not be diagnosed.

ROENTGENOLOGIC SIGNS

The roentgen diagnosis of strangulating obstruction may be missed if the entity is not specifically sought or if one considers that there is a single sign which should be present in every case. Strangulation obstruction is produced by a fairly constant anatomical derangement, the incarcerated or twisted intestinal loop. This, however, initiates physiological disturbances which are progressive or changing in character and varying roentgen signs mirror the various phases of the pathologic process.

The evidence of an incarcerated loop may be of two types. If the loop is only partially closed it will be gas-filled or gas and fluid filled. In the horizontal film, gas will be seen in the two distended limbs of the incarcerated loop and the gas shadows will be separated by the apposed intestinal walls, probably somewhat edematous, and therefore producing a thicker shadow than normal. (Figures 1,2,3). This has been called the "coffee bean" sign.[29] If the incarcerated loop is completely closed it will contain little or no gas. Intestinal gas is either swallowed air (72 per cent), a diffusion product into the intestines from the blood stream, or the result of intestinal putrefaction.[1,33] A completely closed loop obviously will not admit the gas which descends from the upper portion of the bowel and therefore the largest source of intestinal gas is excluded from the incarcerated loop. It will contain, for the most part, only the bloody transudate resulting from the process of strangulation. A small amount of swallowed air may be found in a completely closed loop, if the process of closure developed over a period of time, beginning with a partial obstruction. Roentgenograms of the abdomen will then show the fluid-filled closed loop as a somewhat rounded or oval-shaped soft tissue density (Figures 4,5,6) -- the "pseudo-tumor" sign.[15] While the "coffee bean" sign is harder to distinguish when there is gas in the superior segment of the small bowel, the "pseudo-tumor" sign is enhanced by the presence of gas in the upper reaches of the small intestine.

Fixation of the involved loop is the third of the signs usually observed (Figures 5,8,9,10). Films should be made with the patient in the erect, supine and lateral decubitus positions in order to bring this out. Lack of movement of the loop is strongly in favor of a diagnosis of closed loop obstruction. In the presence of a large amount of gas in the upper small bowel, fixation may be somewhat difficult to determine. Short incarcerated loops will tend to show a higher degree of fixation than longer loops.

While the normal small intestine shows a continuing autoplastic change in the contour and appearance of the valvulae conniventes, distended small intestine contains fixed semi-circular or circular valvulae. When stagnant anoxia occurs there is loss of tone of the muscularis mucosa and the fixed valvulae disappear leaving a smooth or formless bowel lumen. This is a fourth sign of strangulation and can be demonstrated either within the gas-filled incarcerated loop or within the bowel just above, providing there has been sufficient distention to compromise the intramural circulation. (Figures 2,8).

That strangulating obstructions can occur without evidence of abdominal distention has been emphasized by Wangensteen[33]. Reinus' review indicates that while distention was present in 80 per cent of the simple obstructions and 90 per cent of the early strangulating obstructions, it was found in only 40 per cent of the late strangulations. The roentgenologic counterpart of these clinical findings is the absence or scarcity of small intestinal gas above the proximal end of the strangulating loop. (Figures 1,7,9,10). In the present series this was found six times.

Chesterman[3] reproduced this situation experimentally in cats and tried to find the explanation. He placed a celluloid

window in the abdominal wall of the experimental animals and removed enough omentum to allow a clear view of the small intestinal segments which were to be studied. Strangulation was produced and relieved by means of a one centimeter wide cloth tape brought out of the abdomen through a stab wound. Here it was fastened over a lead button so that the process could be watched and the degree of strangulation controlled. Pressure was applied gradually over a period of one or two minutes and the degree of pressure was varied in different experiments. Every degree of vascular obstruction was produced from slight venous obstruction alone to complete venous and arterial occlusion.

It was demonstrated that mild venous obstruction produces effects that differ little from those of non-strangulating obstruction. Severe venous obstruction, especially if associated with arterial obstruction, produces marked alteration in the motor function of the rest of the small intestine. "There is not only immediate spasm of the strangulated loop but also of the gut above and below it, which frequently affects the whole of the small intestine and even the pylorus, although the effect on the stomach is variable. After a time, which varies from a few minutes to several hours, the spasm lessens and intestinal movements recommence, ... but in spite of prolonged vigorous contractions much distention does not take place until retrograde venous thrombosis has occurred or peritonitis is present."[3]

Chesterman believes that retrograde venous thrombosis is the primary cause of marked bowel distention, whether proximal or distal to an acutely strangulated loop of small intestine. If this process extends further on the distal side, dilatation of the bowel is then found distal as well as proximal to the strangulation. These findings were confirmed not only by strangulating a loop of bowel but also by damaging the veins away from the strangulated loop, so that thrombosis took place with results similar to those described above. The bowel in these cases contains fluid and little if any gas.

Clinically, it is precisely these cases which are the most dangerous and a careful routine of film analysis would indicate that even films showing few or no signs of mechanical obstruction should be scrutinized for signs of strangulating obstruction. This fact and its implications for roentgen diagnosis have been emphasized in earlier publications from this department[29] and by Frimann-Dahl[15,17].

The inference to be drawn from these observations would seem to be that the venous return from a trapped loop of intestine may be impeded without a concomitant block of the bowel lumen. Wangensteen[33] has stated that "transport (of gas and fluid) is possible through a strangulated loop of bowel which at operation may give every evidence of loss of viability." It is significant that in this series moderate amounts of gas were found in the colon in nine cases and very small amounts in eight additional cases. (Figure 9). This suggests either that the obstruction is not complete or that the evacuation of the distal segment of the bowel, which usually occurs with the onset of cramps in the simple obstruction, is less complete in strangulations.

While suction siphonage is not used if there is any likelihood that strangulation obstruction is present, several cases have been seen in which continuous siphonage was instituted in patients thought to have simple obstructions, only to find that a single loop could not be decompressed. These cases have been operated upon promptly after this observation and closed loop obstructions have been found. (Figure 11). It should be emphasized that in cases where suction treatment is being used, the failure to evacuate one loop of bowel while all remaining loops are evacuated is strong evidence of a strangulating obstruction.

A group of signs of strangulation has been noted which are less frequent in occurrence and less specific in character. These are obstructing lesions showing long fluid levels (Figure 12), large amounts of fluid in the loops (Figure 7),

or solitary segments of bowel distended out of proportion to the remainder of the intestine (Figure 9). They suggest the possibility of strangulation and should initiate a search for the signs previously described as well as for clinical confirmation of the diagnosis.

Five cases of small intestinal volvulus were found in the series reported here. In none of them were there signs to suggest a twisted loop such as one sees in cecal or sigmoid volvulus, nor was there any evidence of loops radiating from a single point. There were no solitary gas-filled loops in an arcade pattern as described by Levine and Solis-Cohen[21] and Ripstein and Miller[30]. Two cases showed a positive "pseudo-tumor" sign, three showed fixation of loops and two revealed a segment of small intestine completely devoid of mucous membrane markings.

ILLUSTRATIVE CASE REPORTS

Case 1. "Coffee-bean" sign.

., a 53-year old woman, was admitted on 2-22-42 because of vomiting and lower abdominal pain. Her illness began with a fainting spell approximately 48 hours earlier. The vomiting had persisted until the time of admission. Pain was not constant and was generalized across the lower abdomen.

The abdomen was mildly distended. The percussion note was tympanitic over the upper abdomen and dull below. There was rigidity over the lower abdomen and tenderness to deep palpation. The uterine cervix was displaced to the left. The right anterior quadrant of the pelvis was filled with a soft cystic mass, the upper borders of which could not be outlined because of the patient's inability to relax. There was another cystic lobulated mass in the cul-de-sac. Both masses were very tender to palpation.

A tentative diagnosis of a twisted ovarian cyst was made and immediate laparotomy was advised. She was taken to the operating room that evening and a loop of strangulated ileum, 18 inches long, was found bound into the pelvis by adhesions. Resection was performed and an end-to-end anastomosis of the ileum was made.

Case 2. "Pseudo-tumor" sign.

., a 71-year old man, was admitted to the hospital because of severe pain in the right lower quadrant of the abdomen. The pain was colicky and recurred at five minute intervals. He had had epigastric pain for three weeks prior to admission.

The abdomen was diffusely rigid. There was spasm and tenderness in the right lower quadrant. A few bowel sounds were heard on auscultation. An appendectomy scar was present. The hemoglobin level was 16.3 grams. The white blood count was 10,700, with 92 per cent polymorphonuclear leukocytes.

Operation revealed a knuckled loop of ileum in the right lower quadrant, bound down by adhesions. There was decided injection of the bowel along its mesenteric border and several petechiae were seen. After removal of the obstructing adhesions, bowel color returned to normal and resection was not considered necessary. The postoperative course was smooth and the patient was discharged on the seventh day after operation.

Case 3. Fixation of Intestinal Loops.

, a 69-year old woman, was admitted to the hospital on 9-25-42 because of constipation, abdominal cramps and vomiting of four days duration.

The lower abdomen was distended and intestinal loops were palpable. There was no visible peristalsis and no abdominal rigidity. The hypogastric region was decidedly tender. This finding was confirmed on pelvic and rectal examination.

Operation revealed a strangulating obstruction of the distal ileum with early gangrene. The terminal three feet of ileum were resected. Convalescence was very satisfactory.

Case 4. Loss of Mucosal Pattern.

, a 27-year old man, was admitted to the hospital on 7-22-40 because of cramping lower abdominal pain and vomiting of 24 hours duration. He had had an appendectomy two years before.

The abdomen was rigid and there was rebound tenderness, most marked in the right lower quadrant. Physical findings of a mass in this area were equivocal. Borborygmi were auscultated.

Operation that evening revealed gangrene of two feet of the terminal ileum as well as a large amount of bloody, foul peritoneal fluid. Adhesions were responsible for the strangulating obstruction. The involved segment was resected and the two limbs of small intestine were exteriorized. One month later the ileostomy was closed. The patient made an uneventful recovery.

DIFFERENTIAL DIAGNOSIS

Strangulating obstructions may present findings which simulate or are simulated by the following conditions: normal abdomen, paralytic ileus, simple obstruction, peritonitis, inflammatory or strangulating tumors, mesenteric arterial and venous thrombosis, and neurovascular conditions.

Careful attention to solitary gas-distended loops or to fluid-filled loops which might otherwise be overlooked will permit the differentiation of an early strangulation from a normal abdomen. Although there is often some gas in the colon in strangulating obstructions, there is no colic or rectal distention, which would tend to rule out paralytic ileus. The differentiation from simple obstruction depends upon the presence of the signs previously detailed. Separations of these two conditions may be difficult if the patient is seen very early -- for example, three to six hours after the onset of symptoms. Repeat examination in a few hours will probably indicate the proper diagnosis. Identification of other pelvic masses may offer some difficulty but the presence of a smoothly outlined, dense mass should speak, for example, for a twisted ovarian cyst rather than for a "pseudo-tumor" sign, as the latter is usually lobulated in outline. Mesenteric arterial thrombosis may present the picture of bizarre distention of both the small and large intestine ending sharply at the splenic flexure. This would follow the distribution of the superior mesenteric artery. A barium enema will demonstrate that there is no organic obstruction at this level, suggesting the presence of the vascular lesion. A spastic ileus may give a somewhat similar picture.[25] Mesenteric venous thrombosis results in transudation into the bowel wall, the bowel lumen and the peritoneal cavity. This is of diffuse character as opposed to localized fluid-filled loops in a strangulation obstruction.[31] At most, a few small gas collections are seen. There may be a homogeneous density over the abdomen with poor definition of the soft tissue outlines within the abdomen.

SUMMARY AND CONCLUSIONS

The earlier diagnosis of strangulating obstructions represents one of the most effective ways of lowering the current mortality rate of small bowel obstruction. Roentgenologic evidence of strangulating obstruction can assist very appreciably in the early diagnosis. The roentgen signs are dependent upon the presence of a closed loop of bowel distended by either gas or fluid and giving evidence of a fixed position. The findings in 26 cases were analyzed.

The signs which are strongly suggestive and when present should lead to the diagnosis are:

1. The "coffee-bean" shadow.
2. The "pseudo-tumor" shadow.
3. Fixation of a loop of bowel
4. Loss of the normal mucous membrane pattern within the closed loop or above it.

Additional signs of collateral value are:

5. Absence of small bowel gas in a case suspected clinically of small bowel obstruction.
6. The presence of unusually large amounts of fluid in the

lumen of the small bowel.
7. Long fluid levels far beyond the usual size.
8. Distention of a segment of the bowel far out of proportion to the remaining loops.
9. Absence of decompression of a localized loop following suction siphonage.
10. The presence of moderate amounts of gas in the colon despite the apparent evidences of small bowel obstruction.

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II. MEDICAL SCHOOL NEWS

Coming Events

January 8 - 10	Continuation Course in Anesthesiology for General Physicians
January 15	Continuation Course in Management of the Polio Patient
January 19 - 24	Continuation Course in Ophthalmology for Specialists
January 26 - 31	Continuation Course in Pediatric Neurology for Pediatricians, Neurologists, and General Physicians
February 2 - 4	Continuation Course in Clinical Chemistry for General Physicians
February 5 - 7	Continuation Course in Cancer Detection for General Physicians
February 12 - 14	Continuation Course in Cardiovascular Diseases for General Physicians
February 16 - 18	Continuation Course in Recent Advances in Diagnosis for Internists

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Continuation Course

A one-day continuation course in Management of the Polio Patient will be presented at the Center for Continuation Study on the University of Minnesota Campus on January 15, 1953. The recent epidemic of poliomyelitis has brought out the need of a course dealing with this disease. Management of all phases of the disease will be discussed, but emphasis will be placed on the care of the convalescent patient. The course will be presented under the direction of Dr. Frederic J. Kottke, Associate Professor and Head, Department of Physical Medicine, and the remainder of the faculty will include clinical and full-time members of the staff of the University of Minnesota Medical School.

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Dr. Utterback Joins Neurology Department at Iowa

On December 15 Dr. Robert A. Utterback, who has been an Instructor in the Division of Neurology, assumed his new position as Assistant Professor in the Department of Neurology at the State University of Iowa Medical School in Iowa City. We are sure that he will find his new post a stimulating one, and we all join in extending our best wishes for his success in it.

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Season's Greetings

With this issue the Bulletin suspends publication for the holiday season and will resume on January 9, 1953. We would like to extend to students, faculty, members of the Minnesota Medical Foundation, and all friends of the Medical School our best wishes for a pleasant Holiday Season and for a Happy New Year.

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III.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

December 22 - 27, 1952

Monday, December 22

Medical School and University Hospitals

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

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

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

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

12:15 - 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 - 5:30 Seminar on Fluid and Electrolyte Balance; Gerald T. Evans; Todd Amphitheater, U. H.

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

4:30 - Public Health Seminar; 15 Owre Hall.

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

Minneapolis General Hospital

9:30 - Pediatric Rounds; Eldon Berglund; Newborn Nursery, Station C.

10:30 - 12:00 Tuberculosis and Contagion Rounds; Thomas Lowry; Station M.

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

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. A.

1:00 - X-ray Conference; Classroom, 4th Floor.

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

Ancker Hospital

8:30 - 10:00 Chest Disease Conference

1:00 - 2:00 Medical Grand Rounds.

Veterans Administration Hospital

8:00 - 9:00 Neuroradiology Conference; J. Jorgens, R. C. Gray; 2nd Floor Annex.

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

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

2:00 - Psychosomatic Rounds; Bldg. 5.

Tuesday, December 23

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

12:30 - 1:30 Physiology 114D -- Current Literature Seminar; 129 Millard Hall.

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.

Ancker Hospital

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

8:30 - 9:30 Medical-Roentgenology Conference; Auditorium.

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

Minneapolis General Hospital

9:30 - 10:30 Obstetrics and Gynecology Staff Rounds; William P. Sadler and Staff; 301 Harrington Hall (Nurses Hall).

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

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

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

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

12:30 - Neuroroontgenology 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 - Infectious Disease Rounds; Dr. Hall.

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

9:00 - Liver Rounds; Drs. Nesbitt and MacDonald.

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

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

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

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

3:30 - 4:20 Autopsy Conference; E.T. Bell and Donald Gleason; Conference Room, Bldg. I.

Wednesday, December 24

Medical School and University Hospitals

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; [illegible]l 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 Staff; Todd Amphitheater, U. H.

1:30 - 3:00 Physiology 114B -- Circulatory and Renal System Problems Seminar; Dr. M. B. Visscher, et al; 214 Millard Hall.

4:00 - 5:30 Physiology 114C -- Permeability and Metabolism Seminar; Nathan Lifson; 214 Millard Hall.

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, U. H.

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.

2:00 - 4:00 Medical Ward Rounds;

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

Minneapolis General Hospital

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

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

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.

4:00 - Combined Medical-Surgical Conference; Conference Room, Bldg. I.

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

Thursday, December 25 (HOLIDAY)

Friday, December 26

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.

Friday, December 26 (Cont.)

Medical School and University Hospitals (Cont.)

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

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

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 Physiology 124 -- Seminar in Neurophysiology; Ernst Gelhorn; 113 Owre Hall.

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 - Pediatric Rounds; Robert Ulstrom; Stations I and J.

Veterans Administration Hospital

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

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

Saturday, December 27

Medical School and University Hospitals

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

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

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.

Ancker Hospital

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

Minneapolis General Hospital

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, Goldish, and Aufderheide.

11:15 - 12:00 Morphology . . . . . Dr. Aufderheide.