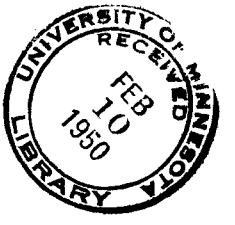
Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation



Electrokymographic Study in Congenital Heart Disease

Volume XXI | Friday, February 10, 1950 | Number 16

BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
and
MINNESOTA MEDICAL FOUNDATION

Volume XXI | Friday, February 10, 1950 | Number 16

INDEX

Published weekly during the school year, October to June, inclusive.
Annual Subscription Rate - $3.00

Address communications to: Staff Bulletin, 332M University of Minnesota Hospitals, Minneapolis 14, Minn.

I. AN ELECTROKYMOGRAPHIC STUDY OF THE PULMONARY PULSATIONS IN CONGENITAL HEART DISEASE

Joseph Jorgens
John W. LaBree
Forrest H. Adams
Lloyd George Veasy

The purpose of this paper is to present a new application of the electrokymograph which should aid in the diagnosis of certain types of congenital heart disease by the evaluation of the pulmonary blood flow. This evaluation is based on the electrokymographic record of vascular pulsations in the periphery of the lungs. We feel that it is not only important to ascertain the type of congenital heart anomaly, but also to determine whether or not the anomaly is of such a degree that the patient will benefit materially by surgery. This can be determined in part by estimating the pulmonary blood flow. If it is inadequate in a patient with cyanotic congenital heart disease, an arterio-venous shunting procedure is probably advisable. On the other hand, in those cases of cyanotic heart disease with an adequate flow of blood to the lungs there is a question as to whether the patient will benefit by a shunt procedure.

Also it may be of value to know the pulmonary blood flow in acyanotic congenital heart conditions. A large pulmonary blood flow may indicate a large intracardiac shunt from the left to the right side of the heart. The size of the shunt could be estimated in this manner. If the shunt is small in a patient with a patent ductus arteriosus, then the strain on the cardio-vascular system probably will be slight. Although at the present time it is the accepted practice to operate on all patients with patent ductus arteriosus regardless of age and the presence or absence of cardiac disability, the size of the shunt might be a better indication for surgery.

The amount of pulmonary blood flow in congenital heart disease is largely determined by the presence of a shunt. Usually an increased pulmonary blood flow exists when there is a shunt of blood from the left to the right side of the heart such as is present in an interatrial, an interventricular septal defect, an anomalous pulmonary vein draining into the right side of the heart, or a patent ductus arteriosus. On the other hand, a decrease in the pulmonary flow is due to a right to left shunt and some type of an obstruction to the flow of blood into the pulmonary artery. This condition usually exists in tetralogy of Fallot, tricuspid atresia, tricuspid stenosus, pure pulmonary stenosis and in a certain type of truncus arteriosus.

The present methods for estimating the amount of pulmonary flow are the following:

1. Physical examination
2. Roentgen examination
3. Roentgenkymography
4. Angiocardiography
5. Heart catheterization
6. Electrokymography

On physical examination a decreased pulmonary second sound suggests a decreased flow and an accentuated second sound an increased flow or pressure in the lesser circulation. On roentgen examination the size of the pulmonary arteries, its branches and the right ventricle may be indicative of an unusual amount of blood flowing to the lungs. Roentgenkymography and fluoroscopy can aid by establishing the presence of unusual pulsations of the pulmonary arteries. Althcugh it does not determine pulmonary flow, one of the better methods of determining right to left shunts is the measurement of the circulation time from arm to tongue combined with a reference arm to lung time. Angiocardiography can establish definitely a shunt from the right ventricle into the aorta and also shows the size of the pulmonary artery branches. However, it is not an entirely innocuous procedure.

- - - - - -

From the University of Minnesota Cooperative Heart Demonstration Unit, a cooperative project between the University of Minnesota and the U. S. Public Health Service.

Heart catheterization at the present time is the most valuable procedure for learning about the intracardiac physiology, but it is quite expensive and time consuming. Therefore, it would be invaluable to develop additional methods of examinating the pulmonary circulation. The study of the lung pulsations recorded by the electrokymograph was undertaken with this in mind.

The lung pulsations are primarily the pulsations of very small vessels within the pulmonary parenchyma. These pulsations are caused by the volume pulse wave arriving in the small vessels such as the arterioles. They are initiated by the contraction of the right ventricle. Blood is forced into the pulmonary artery which dilates to accommodate this additional blood. This zone of dilatation of the pulmonary artery moves toward the peripheral branches and is known as the volume pulse wave. The main factors influencing the shape and size of this wave are the volume of blood ejected per stroke from the right ventricle and the rate of ejection of this volume. For example, if for some reason the right ventricle ejected only half the usual quantity of blood during one contraction, obviously the pulmonary artery would not dilate as much, and the peripheral volume pulses also would be small. The electrokymograph records a summation of the individual volume pulse waves of the small vessels in a volume of lung and is able to record this summation.

At this university, Dr. Herbert M. Stauffer began a study of the pulmonary parenchymal pulsations and reported in 1948 that reduced or absent pulsations over the pulmonary parenchyma appeared to exist in some cases of tetralogy of Fallot.[1]

The pulmonary parenchymal pulsations were first described by Dr. Maurice Marchal[2] of France in 1946. He found them to be invisible to fluoroscopy, roentgenography, roentgenkymography and clinical examinations. These pulsations, he found, could be temporarily stopped by increasing the intra-alveolar air pressure above the pulmonary arterial pressure in some subjects and reduced in amplitude in others.[3] Gillick and Schneider[4] found similar results in 1949. Fleischner, Romano and Luisada[5] in this country recorded these lung pulsations and suggested that they might be a composite tracing, partly venous, partly arterial, similar to a plethysmogram. The lung pulsations are invisible to ordinary fluoroscopy because the pulsations are of vessels that are very small and the present intensification of the fluoroscopic image is not adequate for their detection. The roentgenkymograph can show the pulsations of the pulmonary artery and the hilar shadows in a single plane, and is of definite help in establishing the presence of a "hilar dance."

We have studied the pulmonary parenchymal pulsations of 43 patients with congenital heart disease in the last 9 months in an attempt to evaluate their worth in the diagnosis of congenital heart disease. A standard electrokymographic technique was used in 23 cases which were later proven by heart catheterization, angiocardiography, operation or autopsy. These cases, separated into anatomical groups, are presented in this report.

In order to understand how these pulsations are recorded it is necessary to describe briefly the electrokymograph.

Apparatus

The electrokymograph, designed by Henny and Boone,[6] consists of a power supply, 1000 volts, a 931 A photomultiplier tube, a small fluorescent screen measuring 22 square centimeters placed one inch from the surface of the photoelectric cell, and a filter designed to remove the 120 cycle current fluctuations produced by a full wave rectified fluoroscopic tube. The recording is made by a string galvanometer and a special camera* without any further amplification of the output of the photomultiplier tube. (Fig. 1)

*This camera was procured through the Greater University Fund.

Schematic Drawing of Apparatus

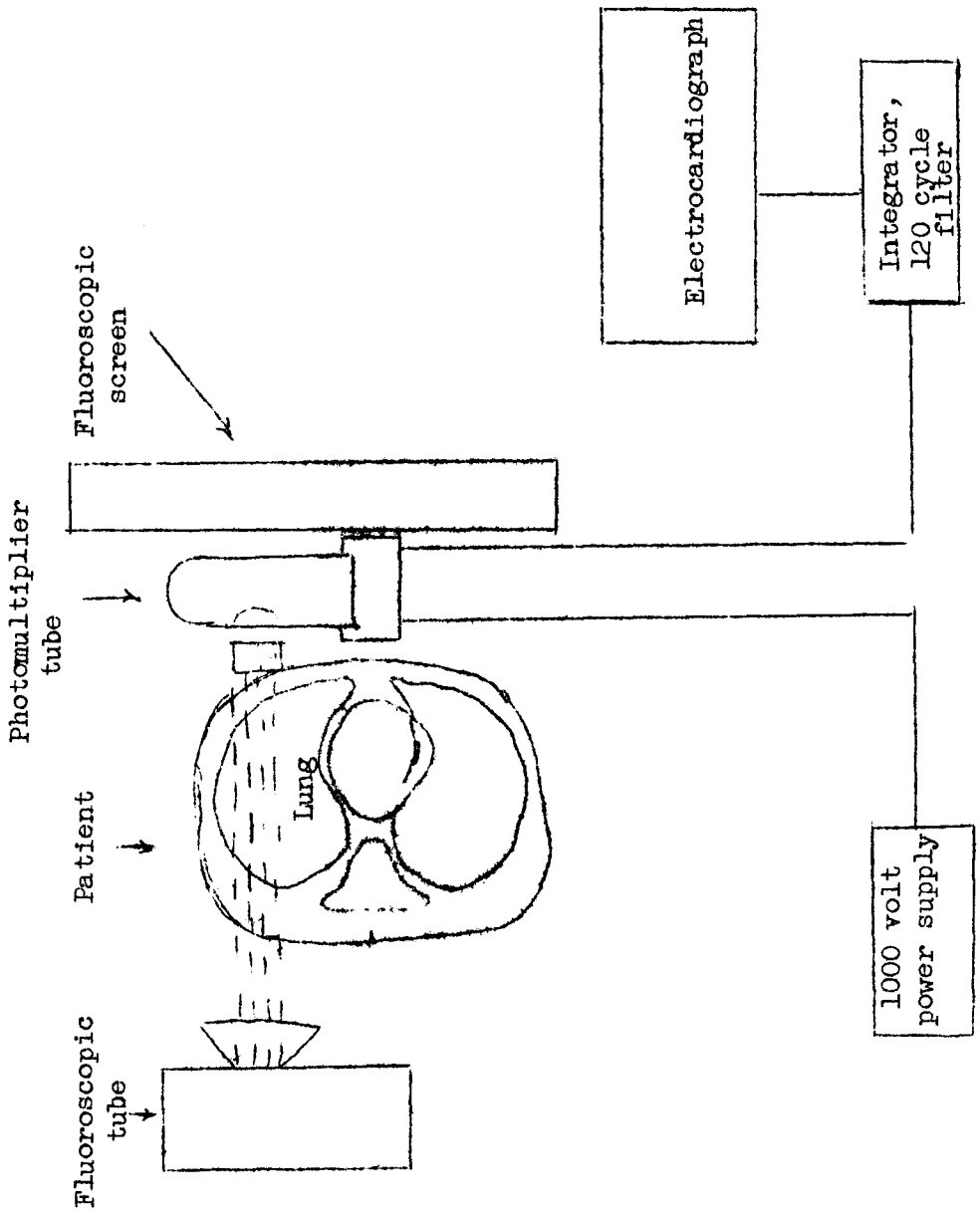


Figure 1

The photomultiplier tube is capable of measuring a very weak light source, and the amount of current output from the tube will vary linearly with the amount of light striking the tube's light-sensitive surface.[7]

Any variation in the amount of x-rays striking the small fluorescent screen will cause the amount of light from the screen, and, thus, the amount of current from the photoelectric cell, to vary.

The current output of the phototube is passed through a capacity-coupled circuit in which there is a filter for removing the 120 cycle fluctuations.

Technique of Recording

Under fluoroscopic guidance, the photomultiplier tube with its small fluores-

cent screen is placed just above the right diaphragm near the costophrenic sulcus. The small fluorescent screen is approximately 52 cm. from the fluoroscopic tube, this distance being kept as constant as possible. The subject is in a supine position on the fluoroscopic table. The central beam of the fluoroscopic tube is directed through the subject's chest to the small fluorescent screen in front of the photoelectric cell. The shutters of the fluoroscope are coned down to the area of the small fluorescent screen. A carotid pulse recorder is placed about the subject's neck for a timing device. This pulse is projected by an optical system into the same camera. An assistant standardizes the string galvanometer before each recording, turns on the power supply and sets the potentiometer control to a standard setting. The subject is instructed in avoiding a Valsalva maneuver while holding a deep inspiration. The fluoroscope is set at 76 kilovolts and 3 milliamperes. The subject takes a large breath and holds it. The camera records a tracing of the variations in the current output of the photomultiplier tube.

Interpretation

The recorded tracing is a densigram of the chest area above the right diaphragm. When the right ventricle contracts, blood is forced into the periphery of the lungs. This additional blood absorbs a certain quantity of roentgen rays and, therefore, fewer x-rays will strike the fluorescent screen, less light is emitted by the fluorescent screen and less current will be produced by the photoelectric cell. During the cardiac cycle, varying amounts of blood will be present in the lung periphery, and the densigram gives a record of this changing amount. The densigram is purposely made in an area away from the hilar region so that the pulsations of the large vessels do not contribute appreciably to this recording. Figure 2 is a schematic tracing of a normal pulsation obtained over the lung parenchyma and of the simultaneously recorded carotid pulse-wave. The upward deflection of the carotid pulse occurs within .02 second after the beginning of left ventricular systole. Usually in the normal lung pulsation two upward curves are present. The upward deflection indicates increased density, and the downward deflection decreased density of the chest. The larger curve occurs in ventricular systole and begins about .04-.08 second after the carotid pulse records systole. This large upward thrust is due primarily to the arrival in the periphery of blood ejected from the contracting right ventricle. A deep inspiration is held by the subject, thus the changing density must be due only to changing blood volume. Of course, there is changing blood volume in the chest wall, i.e., in the muscles, skin and subcutaneous tissues, which also may take part in the changing density. However, placing the photoelectric cell over tissues equivalent in thickness to the chest wall with no amplification does not bring out the pulsations that are found over the lung fields. This is probably because the density in these tissues is considerably greater and the blood volume change is relatively not as great.

The large upward thrust represents an increased inflow of blood into the lungs over the blood flowing out of the lungs. This upward thrust probably represents primarily a volume pulse in the smaller arterial vessels.[8]

The pre-systolic upward curve is difficult to explain. It occurs about .12 second before the carotid, registers systole and possibly is due to the damming back of the blood into the lungs during the left atrial contraction.

Because of the many variables in the calibration of the electrokymograph and differences in sizes of the subjects, the height of the upward curves has not been calibrated in terms of cubic centimeters of blood volume change. The height, therefore, only indicates a relative amplitude of one tracing compared to the next.

Results

The subjects used for "normals" were

Schematic Drawing of Lung Pulsations

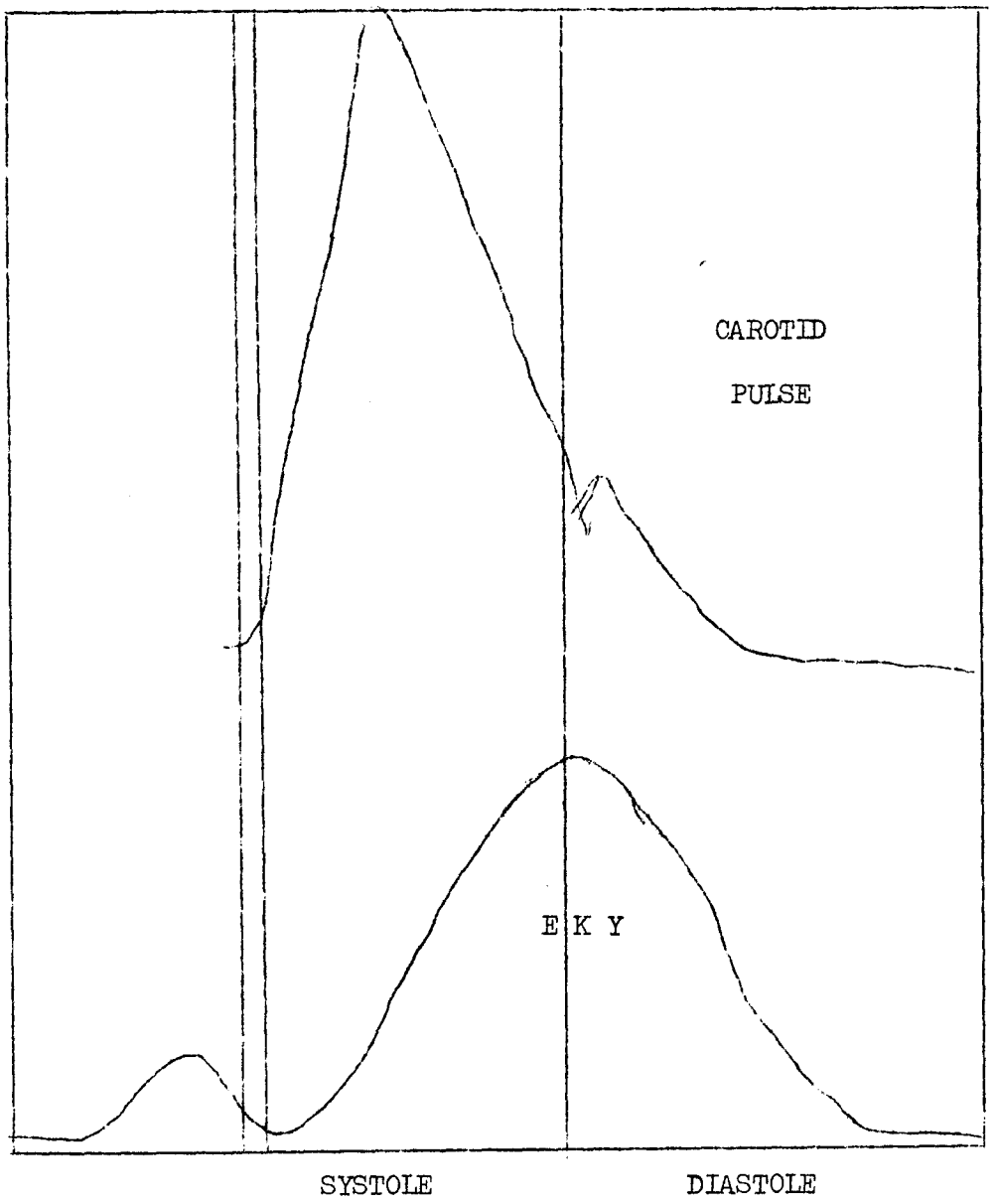


Figure 2

twenty-nine children under 15 years of age. They were not strictly normal since they comprised part of the hospital population. They had no apparent cardiac disabilities, however. The range in value of the height of the systolic upward thrust was 3 mm. to 14 mm. with an average of 7.3 mm. It is necessary to recall that this has no quantitative value, but only serves as a reference point in comparing other groups of pathological cases. Six patients with patent ductus arteriosus showed a range of 5 mm. to 7 mm. for the systolic upward deflection with an average of 6.1 mm. The shape of the curves did not vary appreciably from the normal. Occasionally no pre-systolic wave was visualized. There was no delay in the time of arrival of the volume pulse at the periphery. From this small series of cases it does not seem possible to distinguish them from the normal or from the next group, the interatrial septal defect. It is hoped that with a larger series, characteristics may be recognized which will be indicative of this condition.

Five patients with interatrial septal defects showed a range of from 8 mm. to 12 mm. in the height of the systolic upward deflection and an average value of 10 mm. There was no delay in the time of arrival of the volume pulse at the periphery compared to the normal. The pre-systolic wave appeared smaller because of the increased height of the systolic wave.

The most interesting series consisted of 12 patients with pulmonary stenosis with various associated anomalies. Of these 12 cases, one had a systolic upward thrust of 6 mm., two with 4 mm., and the rest were about 3 mm. in height. The average was 2.8 mm. While this group is small, it is suggestive that the height of the lung pulsations, especially in this group, may have diagnostic importance, when the electrokymograph is more adequately calibrated. Five of this group had delayed arrival time of the volume pulse at the periphery. The same carotid pulse recorder was always used as the timing device. In the normal series, the volume pulse arrived between .04 and .08 seconds after the beginning of the foot of the carotid pulse wave. In these five the arrival time occurred .10 seconds or more after the foot of the carotid pulse wave. Two of these pulse volumes did not reach the periphery until .16 second after carotid pulse wave.5

Tracing one is typical of those we have obtained in patients without known lung or heart pathology. The two upward curves are present, the larger one occurring in ventricular systole, and the smaller one occurring in the pre-systolic period. The vertical time line represents the beginning of the foot of the carotid pulse wave for all the tracings. The recordings have been traced for this paper and represent actual size and shape of the curves.

Figure two is a tracing obtained from a patient with an isolated pulmonary stenosis. This patient, R.S., was 15 years old, had a low exercise tolerance and was acyanotic. There was a systolic thrill over the left sternal margin and a harsh systolic murmur heard best over the fourth left interspace. The blood pressure in the right arm was 120/70. The roentgen findings showed a very large pulmonary artery, and the question arose as to whether the peripheral vascular markings and hilar vessels were normal or increased in size. The electrokymographic study of the peripheral pulsations of the lung revealed a diminution in the size of the peripheral pulsations as compared with the normal. The shape of the curves was difficult to make out. A diagnosis of pulmonary stenosis was made, and this was later proven by heart catheterization to be an isolated pulmonary stenosis.

., 12-year-old male, acyanotic, complaining of fatigue, had a systolic thrill over the fourth left interspace. A loud, long, rumbling systolic murmur was heard over the entire precordium with the maximum intensity in the fourth interspace. A short diastolic murmur was heard over the mitral area. The roentgenological examination showed an enlarged heart of the right ventricular type. The pulmonary artery, root shadows, and peripheral pulmonary vascular markings were markedly enlarged. The roentgen findings, therefore, were consistent with an interatrial septal defect, however, the possibility of patent ductus arteriosus still existed. An electrokymographic study of the periphery showed large pulsations which were consistent with an interatrial septal defect. A large systolic deflection is more characteristic of an interatrial septal defect than of a patent ductus arteriosus. Heart catheterization confirmed the diagnosis of interatrial septal defect. Tracing three shows the pulsations obtained. The pre-systolic wave never returns to the base line, but is combined with the following upward thrust of the volume pulse.

, male, age 13, cyanotic since birth with a low exercise tolerance, showed watch crystal formation of the nails. A systolic thrill was present over the base of the heart, and a systolic murmur was heard over the entire precordium. The roentgenograms showed a large right ventricular type of heart with an enlarged pul-

monary artery segment, large vascular root shadows, and prominent peripheral vascular markings. Clinically, the differential diagnosis lay between a mild degree of tetralogy of Fallot and an Eisenmenger's complex. The electrokymographic study showed large-sized pulsations of the peripheral vessels indicating that there was no pulmonary stenosis present. Tracing four shows the actual copied tracing. Heart catheterization proved this case to be an Eisenmenger's complex. So, it appears possible by means of electrokymography to distinguish between a tetralogy of Fallot and an Eisenmenger's complex.

----, female, 10 years old, cyanotic from birth with a low exercise tolerance, showed watch crystal formation of the nails. A thrill was felt over the entire precordium, and a systolic murmur was heard best in the second and third left interspaces. The roentgen study showed a large aorta with absence of the main pulmonary artery shadow. The hilar vessels were within normal limits in size as were the peripheral vascular markings. The apex of the heart was somewhat elevated. Again a question existed as to exact diagnosis. The electrokymographic study (tracing five) showed a diminution of the pulsations in the periphery of the lungs. In fact, it was difficult to make out which upward deflection represented the systolic thrust. The pre-systolic wave and systolic wave appeared equal in height. Delay in the arrival time of the volume pulse was difficult to establish. By these characteristics a diagnosis of pulmonary stenosis was made. Heart catheterization and angiocardiography favored truncus arteriosus over tetralogy of Fallot. This again proved the electrokymograph capable of detecting abnormal pulmonary flow.

male, 16-years-old, a cyanotic with retardation of growth and fatigue had a systolic murmur heard best over the second and third left interspaces. The pulmonary second sound was greater than the aortic second sound. The blood pressure was 95/50 in the right arm. Roentgenogram showed a large heart of the right ventricular type, large pulmonary artery, large root shadows and prominent peripheral vascular markings. Doubt existed as to the definite diagnosis. The electrokymographic study revealed large pulmonic pulsations in the periphery. (Tracing six). The pre-systolic wave was evident. The systolic upward thrust appeared somewhat steeper than usual. There was no delay in the arrival of the volume pulse at the periphery. The findings were consistent with an interatrial septal defect. Heart catheterization proved this case to have an anomalous pulmonary vein emptying into the right atrium. Whether there existed a concurrent interatrial septal defect could not be proven. In this case, the electrokymogram of the lung pulsations was not able to reveal the exact diagnosis but did suggest the presence of a left to right shunt.

This next case is that of a young girl, , 9 years of age, acyanotic and easily fatigued. A continuous thrill was palpable over the second and third left interspaces. A high-pitched systolic murmur was heard over the apex. A loud, continuous machinery murmur was present over the second left interspace. Roentgenological diagnosis showed a heart that was within normal limits in size with a large pulmonary artery, root shadows and increased vascular markings. Although the diagnosis of patent ductus appeared apparent, heart catheterization was performed. Electrokymographic studies showed only fair-sized pulsations of the peripheral lung field near the right base. Normal-sized pulsations appear to be more typical of patent ductus arteriosus than interatrial septal defect. In this case, the pre-systolic wave was inconspicuous. The findings were then consistent with patent ductus arteriosus. Heart catheterization proved the existence of a patent ductus arteriosus.

From these cases it is apparent that differences in the tracings obtained by electrokymography exist. It also appears reasonable to believe that soon characteristic findings will be recognized for each of the congenital anomalies and this new instrument will prove to be a valuable adjunct in the diagnosis of congenital

Tracings of peripheral vascular pulsations

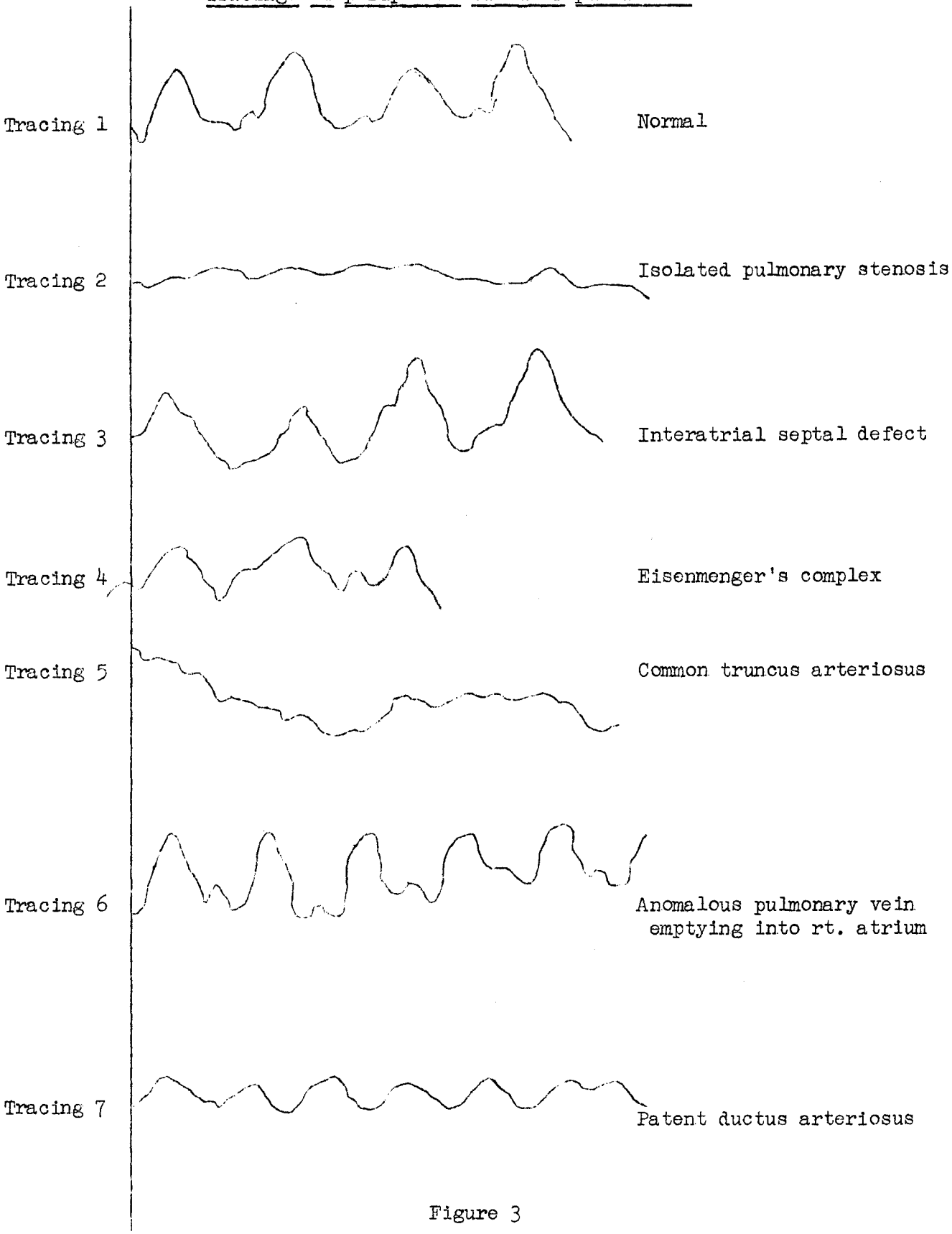


Figure 3

heart disease.

Discussion

The tracings are of interest because they show variations from what appear to be normal lung parenchymal curves. These tracings present suggestive evidence differentiating between left to right and right to left shunts. However, as yet the series of tracings are too small, and the many variables too great for us to be positive that any one curve is characteristic of a particular congenital defect. Timing of the arrival of the volume pulse may be also of diagnostic value. In patients with marked pulmonary stenosis it appears that there exist typical tracings with low deflections. However, some patients with only mild degrees of pulmonary stenosis have tracings that are normal in size and shape.

The height and shape of the curves are dependent upon certain physiological factors of the pulmonary circulation and upon the characteristics of the electrokymographic unit.

Obvious physiological factors are:

1. Right ventricular stroke volume and ejection curve
2. Elasticity of the large pulmonary arteries
3. Vascular characteristics of the volume of lung under direct observation:
   a. Vasomotor tone and elasticity of the arterioles
   b. Number and size of arterioles and arteries
4. Pressure waves in the veins reflected from left atrial systole and other mechanical cardiac events.

Any mechanism which will change the relationship of the above factors will influence the outflow and inflow into the lungs and may change the shape and height of the curves.

From these factors it is possible to postulate that a large systolic upward deflection would occur if an increased stroke volume of blood were ejected at an increased rate of ejection, or if the large arteries were more rigid than usual, or if a normal or decreased vasomotor tone existed in the arterioles, or if there existed some impediment to the normal flow of blood out of the capillaries. If this reasoning is valid, a Lutembacher's syndrome should give one the highest systolic upward deflections possible to record. Conversely, a small right ventricle due to an infundibular stenosis would have a very small stroke volume output. This has been true in a single case studied recently. If in tetralogy of Fallot a large percentage of the blood is shunted into the aorta, again the pulmonary blood volume should be low, and the volume pulse probably will be small. This was true in five instances where such studies were made. In one case, however, the systolic upward deflection was definitely normal in height, the other four were at or below the lower limits of normal.

The shape and height of the curve are also dependent upon the following factors:

1. Quality and quantity of x-rays emitted by the fluoroscopic tube.
2. The distance from the tube to the fluorescent screen.
3. The distance from the small fluorescent screen to the light-sensitive surface of the photoelectric cell.
4. The characteristics of the photomultiplier tube and its power supply.
5. The characteristics of the filter, integrators and the capacity-coupled circuit.
6. The thickness and density of the patient.
7. The characteristics of the string galvanometer.
8. The position of the electrokymographic head in respect to the chest, this varying with different sized and shaped individuals and different diaphragm levels.

All these factors were made as standard as possible when these tests were per-

formed. Obviously, it was impossible to standardize the thickness and density of the patient. An attempt was made to use filters as a method of standardizing some of the other above factors, but it was not satisfactory with the capacity-coupled circuit being used. Plans have been made to build a direct-coupled amplifier circuit with an automatic gain control and calibration monitor. Variations in the size of patients, in the fluoroscopic tube output and in the electrical characteristics of the phototube circuit would be eliminated, and it appears possible to calibrate exactly the height of the curves obtained in cubic centimeters of blood. Thus, if the amount of lung tissue surveyed is closely estimated, the density change is related to the cubic centimeters of blood per cubic centimeter of lung tissue. This value can be used in estimating total pulmonary blood flow by taking recordings over various portions of the lung fields. Obviously, the total pulmonary blood flow is not completely mirrored in the electrokymographic tracings for two reasons: first, not all parts of the lung can be surveyed, and, secondly, a portion of the pulmonary blood flow will only change the velocity of flow without dilating the small vessels.[9] In spite of these limitations, a fair estimate of the pulmonary flow will be available.

On the same individual by comparing records taken at different times, a good estimate of the differences in pulmonary flow should be obtained. In the introduction of this paper the value of knowing the amount of pulmonary flow in acyanotic and cyanotic congenital heart disease was discussed. It may also be of importance in other cardiac conditions, for instance, it is probable that blood rushes back into the lungs in mitral regurgitation much as blood rushes into the neck vessels in tricuspid regurgitation. This should be possible to record in the lung pulsations. Since the pulmonary flow and the cardiac output is the same in normal subjects over a period of time, electrokymography probably can help in determining the beginning of cardiac failure by the lowered cardiac output and serial recordings may aid in showing the extent of recovery under therapy. The electrokymographic recording of lung pulsations appears to have a wide field in clinical practice as well as in purely investigative studies.

Summary

In an attempt to develop procedures for the evaluation of the pulmonary blood flow in congenital heart disease, the electrokymograph has been used. A brief description of the apparatus, technique of obtaining such pulsations and interpretation of a normal tracing is reported. Tracings are shown in six cases of different types of congenital heart disease. It is felt that further study of these pulsations will make it possible to distinguish between intracardiac and extracardiac shunts, to distinguish between increased and decreased blood volume flow to the lungs, and to distinguish between the blood coming from the heart directly or from collateral circulation. The many variables, both physiological and electrical, make interpretation difficult. It is hoped that with a new electrokymograph now being built the electrical features will be completely standardized, and a more critical investigation of the changes due to pathological physiology will be permitted.

ACKNOWLEDGMENTS

We are appreciative of the guidance of Dr. Leo G. Rigler and Dr. Herbert M. Stauffer who initiated this investigation. We also appreciate the cooperation of Dr. Paul F. Dwan and Dr. Morse J. Shapiro for referring patients to us.

This study would not have been possible without the excellent technical assistance of Barbara Bruner and La Vonne Bergstrom.

References

1. Stauffer, Herbert M.
Roentgenology of Congenital Heart Disease.
Bull. of U. of M. Hosp. 30:486-503, (June 11) '48.

2. Marchal, Maurice.
De l'enregistrement des pulsations invisibles due parenchyme pulmonaire ainsi que des pulsations cardiovasculares par la Kinedensigraphie.
Arch.du Mal du coeur, 30:345-359, (Sept.-Oct.) '46.

3. Marchal, Maurice.
Methode de mesure do la pression arterielle de l'artere pulmonairo chez l'homme par les rayons X.
Compt.rend.Acad.sc.225:394-396, (Aug.25) '47.

4. Gillick, Fred G. and Schneider, Jurg.
Electrokymographic Studies of Lung Field Pulsations with Exhalation against Pressure.
J. of Applied Physiology, Vol. 2: No. 1 (July) '49.

5. Fleischner, Felix, Romano, Francisco, J. and Luisada, Aldo A.
Studies of Fluorocardiography in Normal Subjects.
Proc. Soc. Exper.Biol.and Med. 67: 535-539, '48.

6. Henny, George C. and Boone, Bert R.
Electrokymograph for Recording Heart Motion Utilizing the Roentgenoscope.
Amer.J.of Roent. and Radium Ther., 54: (Sept.) '45.

7. Engstrom, Ralph W.
Multiplier Photo-Tube Characteristics: Application to Low Light Levels.
J. Optical Soc. of Amer. 37:420-431, (June) '47.

8. Hertzman, A. B., Randall, W. C., and Jochim, K. E.
Estimation of cutaneous blood flow with photoelectric plethysmograph.
Am.J.Physiol.145:716-726, (Mar.) '46.

9. Krogh, August
The Anatomy and Physiology of Capillaries.

II. MEDICAL SCHOOL NEWS

Coming Events

February 16 - E. Starr Judd Lecture - "Growth in the Field of Anesthesia" - Dr. Henry K. Beecher, Harvard University Medical School; Medical Sciences Amphitheater, 8:15 p.m.

February 16-18 - Continuation course in Cancer for General Physicians.

March 6-8 - Continuation course in Gastro-Intestinal Diseases for General Physicians.

* * *

Faculty News

Dr. Leo Rigler, Professor and Head of the Department of Radiology, has returned recently from Washington, D. C., where he served as consultant in radiology for the Armed Forces Institute of Pathology. During his stay Dr. Rigler presented lectures and conducted conferences in various hospitals in Washington area, which are a part of the Armed Forces medical installations.

Dr. Harold S. Diehl, Dean of the Medical School of the University of Minnesota, and Dr. George N. Aagaard, Director of Postgraduate Medical Education, attended the annual Congress on Medical Education and Licensure in in Chicago, February 5, 6, and 7. The Congress, which is held annually, included the meetings of the Advisory Board for Medical Specialties and the Council on Medical Education and Hospitals of the American Medical Association. Panel discussions which were presented and were of great interest to those attending were, 1) The Evaluation of the Qualifications of Candidates for Certification by American Boards in Specialties; 2) The Place of the Specialties in Undergraduate Medical Education.

Dr. Diehl gave a preliminary report to the Congress on Medical Education in Great Britain. The report was the result of the survey which Dr. Diehl made together with Dr. Loren R. Chandler, Dean of the Stanford University Medical School, and Dr. Stanley E. Dorst, Dean of the University of Cincinnati Medical School, at the request of the American Medical Association.

* * *

Dr. Henry K. Beecher to Give E. Starr Judd Lecture

Dr. Henry K. Beecher, Chief of the Department of Anesthesia, Massachusetts General Hospital, and Dorr Professor of Research in Anesthesia at Harvard Medical School, will deliver the annual E. Starr Judd lecture in surgery on Thursday, February 16, at 8:15 p.m. in the Medical Sciences Amphitheater. The subject of Dr. Beecher's address will be, "Growth in the Field of Anesthesia". The E. Starr Judd lectureship, which was established in November, 1933, annually brings to our campus distinguished scientists and teachers in surgery and its related fields. All physicians are cordially invited to attend the lecture which will also be open to the public.

Dr. Beecher will also participate in a continuation course in Cancer for general physicians of Minnesota and North Dakota presented at the Center for Continuation Study, February 16-18. He will discuss "Anesthesia in Surgery of Thoracic Tumors" with this group of practicing physicians. The course will emphasize present concepts in the diagnosis and management of the various types of neoplasms. Clinical and full-time members of the staff of the medical school and the Mayo Foundation will form the faculty for the course.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

February 12 - February 18, 1950

No. 277

Sunday, February 12 - H O L I D A Y

Monday, February 13 - H O L I D A Y

8:00 - Clinical Research Club Meeting; 1- Blood Pyruvates in Liver Disease; Donald M. Amathuzio, Department of Medicine. 2- Thalamotomy; Morton Feferman, Department of Neurosurgery; Eustis Amphitheater, U. H.

Tuesday, February 14

8:15 - 9:00 Roentgenology-Surgical-Pathological Conference; Craig Freeman and L. G. Rigler; M-109, M. H.

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

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

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

11:00 - Contagion Rounds; Forrest Adams; Sta. L, General Hospital.

12:30 - Pediatric-Surgery Rounds; Drs. Stoesser, Wyatt, Chisholm, McNelson and Dennis; Sta. I, Minneapolis General Hospital.

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

1:30 - 2:30 Pediatric Psychiatry Conference; R. A. Jensen and Staff; 6th Floor, West Wing, U. H.

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

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

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

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

4:00 - 5:00 Physiology-Surgery Conference; Ether-Anesthesia and its Physiological Effects; Dr. Knight, et al; Eustis Amphitheater.

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

5:00 - 6:00 Porphyrin Seminar; C. J. Watson, Samuel Schwartz, et al; Powell Hall Amphitheater.

Tuesday, February 14 (Cont.)

5:00 - 6:00 X-ray Conference; Presentation of Cases by Veterans Hospital Staff; Drs. Fink and O'Loughlin; Todd Amphitheater; U. H.

Wednesday, February 15

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

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

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

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

11:00 - Pediatric Rounds; Erling Platou; Sta. I, General Hospital.

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

12:00 - 1:00 Radio-Isotope Seminar; Radio-Isotopes in Neurosurgery; C. M. Caudill; 113 Medical Sciences.

12:15 - Staff Meeting; Main Classroom, General Hospital.

3:00 - Pediatric Rounds; E. J. Huenekens; Sta. I, General Hospital.

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

4:00 - 5:00 Infectious Disease Rounds; University Hospital, Todd Amphitheater.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; E-101, U. H.

Thursday, February 16

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

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

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

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

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

11:30 - Pathology Conference Clinic; Main Classroom; General Hospital.

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

12:00 - 1:00 Physiological Chemistry Seminar; Proteins in Dental Enamel; W. D. McBride; 214 M. H.

Thursday, February 16 (Cont.)

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

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

4:15 - 5:00 Bacteriology and Immunology Seminar; Preliminary Evaluation of the Tuberculostatic Activity of Streptomycylamines; Robert Patnode; 214 M. H.

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

5:00 - 6:00 X-ray Seminar; Hodgkin's Disease of Bone; Dale Parshall; Todd Amphitheater, U. H.

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

Friday, February 17

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

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

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

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

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

11:00 - Pediatric Rounds; Erling Platou; Sta. I, General Hospital.

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

11:45 - 12:50 University of Minnesota Hospitals General Staff Meeting; Hyaluronidase Inhibition by Serum in Skin Disease; Melvin L. Grais and David Glick; Powell Hall Amphitheater.

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

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

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

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

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

3:00 - 6:00 Demonstrations in Cardiovascular Physiology; M. B. Visscher, et al; 301 M. H.

Friday, February 17 (Cont.)

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

4:15 - 5:15 Electrocardiographic Conference; Rhythm and Arrhythmias A-V Block; R. Berman; 106 Temp. Bldg., Hospital Court, U. H.

5:00 - 6:00 Otolaryngology Seminar; Review of Current Literature; Dr. Strand - Discussor, Dr. Tangen; Todd Memorial Room, U. H.

Saturday, February 18

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

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

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

9:00 - 11:30 Psychiatry Conference; Lobotomy; Dr. Schiele; Powell Hall Amphitheater, U. H.

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

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

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

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

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

11:00 - Contagion Rounds; Forrest Adams; Sta. L, General Hospital.

11:00 - 12:00 Anatomy Seminar; Physiological Relations of the Hypophysis and Thyroid; Arthur E. Sethre; Secretion of Androgen by the Ovary; Howard Dale; 226 I. A.