Bulletin of the University of Minnesota Hospitals and Minnesota Medical Foundation

Hypotensive Anesthesia

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I. HYPOTENSIVE ANESTHESIA

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INTRODUCTION

The production of hypotension for the purpose of securing an ischemic surgical field may be accomplished by:

1) reducing the patient's blood volume,
2) decreasing his peripheral resistance, or
3) a combination of both.

Gardner[1] performed the first operation in 1946 upon a patient whose arterial pressure was reduced to a hypotensive level. His technique for reduction of blood volume by arteriotomy was fully described by Hale[2] in 1948.

Decreasing the peripheral resistance was first employed by Griffiths and Gillies[3] in 1948. They used "total spinal block" and postural pooling to accomplish a bloodless surgical field. In 1950 Enderby[4] introduced a second method of decreasing the peripheral resistance, namely, autonomic ganglion blockade. This technique has become the most popular since it is considered physiologically safer, technically simpler, and is readily reversible.

The hypotension resulting from a decrease in peripheral resistance presumably allows adequate tissue blood flow and oxygenation. In contrast, the hypotension resulting from a reduction in blood volume is accompanied by some increase in peripheral resistance. This, in turn, decreases tissue blood flow and oxygenation, producing a state similar to impending surgical shock.

To date, over 21,000 operations, employing the hypotensive technique, have been performed with a mortality rate of 0.2 per cent.

The following studies were conducted jointly with the neurosurgical division in an attempt to evaluate the efficacy and safety of induced hypotension and to develop, if possible, standards which will insure the safety of the patient.

MATERIALS AND METHODS

Studies were carried out on 14 patients either prior to or during surgery. There were 8 males and 6 females ranging in age from 7-67 years.

Investigations were conducted in the recovery room on 5 patients prior to surgery, because certain data were difficult to obtain during a surgical procedure. The other 9 patients were studied throughout the actual operations.

All patients were premedicated with morphine or codeine and atropine or hyoscine prior to the induction of anesthesia. Anesthesia was induced with Pentothal-curare mixture (Pentothal 25 mgm. and d-tubocurarine 0.75 mgm. per cc. of solution). The patients were intubated and then connected to a semi-closed circle absorption system. Anesthesia was maintained by additional increments of Pentothal-curare mixture and a flow of 1000 cc. each of nitrous oxide and oxygen. Respiration was controlled and augmented by manual bag compression throughout the entire anesthetic period.

Hypotension was induced with hexamethonium bromide (C6) which was given intravenously in doses of 25 milligrams of the ion.

Following the induction of hypotension in the operative cases, the nitrous oxide was discontinued so that 100 per cent oxygen could be supplied. The preoperative study cases were left on the original mixture of nitrous oxide and oxygen.

Arterial and venous pressure studies were recorded on a multichannel electronic recorder (Sanborn Polyviso) equipped with strain gage amplifiers. Arterial pressures were transduced by means of a model P23A Statham strain gage and the venous pressures by means of a model P23B strain gage. The overall catheter-

manometer-recording system produced a uniform frequency response curve from 0 - 16 c.p.s., and then fell off sharply as the frequencies increased.

The venous strain gage was adapted to a 10 cm. length of Polythene catheter, which was threaded through a 15 gage needle into the external jugular vein. The catheter was directed toward the heart. The strain gage was fastened so that its diaphragm was level with the distal opening of the catheter. The necessity of applying a correction factor, because of unequal levels, was thus eliminated. The position of the gage remained fixed in relation to the vein. The pressure tracings, therefore, represent the actual peripheral venous pressure at the site of the catheter opening wihtin the jugular vein.

Electrocardiograms were recorded on a single channel direct writing Cambridge electrocardiograph equipped with a relay controlled remote marker, thereby furnishing a signal mark on the pressure tracing at the time each ECG was taken.

Circulation times were done according to the method of Knutson et al[5]. This method employs the aid of the oximeter. Methylene blue (0.8 mgm. per kgm.) is injected rapidly into the antecubital vein. The time required for the dye to reach the ear, as indicated by a fall in the oximeter reading, is referred to as the arm to ear circulation time. The normal arm to ear circulation time by this method is 13.4 seconds. Maximum dye concentration appears at the ear in 28.6 seconds.

ARTERIAL PRESSURE STUDIES

The average onset of action following a single intravenous injection of C6 was 29 seconds. The peak of action was attained within 3-5 minutes.

A single 25 mgm. intravenous dose produced an average drop in systolic pressure of 48.8 per cent and a drop in diastolic pressure of 44.2 per cent. These figures represent the average of the falls in blood pressure of all the patients studied. The duration of hypotension obtained by a 25 mgm. dose varied from 15-50 minutes.

Several factors directly influenced the level of hypotension attained, namely, the dose of the drug, the depth of anesthesia, the position of the patient, the state of the blood pressure prior to the administration of C6, normovolemic and oligemic shock and positive pressure respiration.

Dosage.-Initial doses were usually 25 mgm. and additional increments the same. The total dosage varied from 25 mgm. to 225 mgm. In some cases precipitous falls in pressure to dangerously low levels occurred with single 25 mgm. doses. On the other hand, one patient received a total dose of 225 mgm. of C6 and yet failed to attain a satisfactory hypotensive level.

Preoperative studies showed maximum pressure falls with the first 25 mgm. dose of C6. Subsequent doses failed to produce the low level attained by the initial dose. There is a tendency for the blood pressure to assume a slightly higher level than that reached following the initial dose. Early in the series it became evident that there was a point beyond which the pressure could not be reduced. In general this level is attained with 25-50 mgm. and additional dosage only serves to prolong the effect.

Depth of anesthesia. - Pentothal-curare anesthesia appeared to compliment the hexamethonium in maintaining hypotension. The fact that all of the patients were under anesthesia may account for the relatively large percentage falls in blood pressure accompanying a single 25 mgm. dose of C6.

Position of the patient.-The majority of the patients undergoing surgery with induced hypotension were in a 10 degree Fowler's position prior to the administration of hexamethonium. For this reason, it was not possible to determine to what extent the postural pooling of blood in the lower extremities contributed to the overall fall in pressure. In six cases, studies were carried out on anesthetized patients with C6 induced

hypotension to determine the role posture played in producing a further pressure fall. Seven observations were made tilting the table to 10 degree Fowler's position. The average fall in systolic pressure was 22.6 per cent and in diastolic pressure 15.3 per cent. Five observations tilting the table to 15 degree Fowler's position revealed an average fall of 39.7 per cent in systolic and 31.9 per cent in diastolic pressures.

State of blood pressure prior to C6 administration.-This clinical series contains too few cases to be of statistical value regarding the relationship between the falls in pressure produced by hexamethonium and the pre-existing pressure levels. However, several interesting observations were made.

Three patients could be classed as normals. In these subjects the average fall in systolic pressure was 36.2 per cent and in diastolic pressure 37 per cent.

Six patients were classed as hypertensives. In these, the average systolic fall was 46.4 per cent and diastolic fall 42.6 per cent.

Six patients had increased intracranial pressure prior to surgery. This group underwent an average systolic drop of 48.9 per cent and diastolic drop of 47.6 per cent.

Normovolemic and oligemic shock.-One patient was in severe normovolemic shock at the onset of surgery. Cerebral decompression restored his blood pressure and hemorrhage started. Twenty-five mgm. of C6 produced a 68.1 per cent fall in systolic pressure and a 66.1 per cent fall in diastolic pressure.

Another case was in a state of compensated oligemic shock at the time of C6 administration. A fall of 61 per cent occurred in the systolic pressure and 55.5 per cent in the diastolic pressure.

Positive pressure respiration.-Ordinary intermittent positive pressure respiration has very little effect upon the arterial pressure of patients under C6 blockade. Augmented respiration varies the systolic pressure about 5 mm. Hg. The diastolic pressure shows even less variation. Apnea simply eliminates the respiratory waves in the arterial tracing.

When a constant positive intrapulmonary pressure of 6 mm. Hg is applied to an apneic patient, there is practically no change in arterial pressure. However, application of a high intrapulmonary pressure (15 mm. Hg) results in a marked decline in arterial pressure with complete disappearance of the pulse pressure. Release of the pressure results in a gradual return to the starting level without a tendency to overshoot.

EFFECT OF HYPOXIA AND HYPERCARBIA ON PATIENTS UNDER THE INFLUENCE OF C6

Normal individuals and patients under moderate Pentothal-curare anesthesia show a rise in arterial blood pressure when subjected to hypoxia or hypercarbia. The question arose as to whether this pressure response would still obtain in the presence of C6 blockade.

The arterial pressure response to hypoxia in patients under the influence of C6 is illustrated by the following example. When the oxygen tension was reduced to 80 mm. Hg (10 volumes per cent) for a period of 4 minutes, the arterial pressure gradually fell from 85/65 to 74/57. After the fourth minute 100 per cent oxygen was administered and the pressure returned to the starting level.

The response to hypercarbia is illustrated by the following example. Ten per cent CO_2 was administered for a period of 5 minutes. By the end of this period the alveolar CO_2 tension was 70 mm. Hg. No change in arterial pressure occurred.

During the period of hypoxia, the heart rate increased from 94 to 104. Under the influence of hypercarbia, the heart rate fell from 110 to 92.

VENOUS PRESSURE STUDIES

Findings.-The initial effect of C6 on

the venous pressure varied. Some patients experienced a transitory fall while others had an increase and still others, no change at all. This variability in initial response was dependent principally upon the position of the subject at the onset of action of the drug. Fowler's position favored a fall in venous pressure while the level and Trendelenburg positions favored a rise.

Once the full effect of C6 was attained, a close parallelism between the venous and arterial pressure followed. Alterations in the patients' positions, fluctuations of the anesthetic levels, subsequent doses of C6 and the administrations of pressor agents produced similar alterations in both venous and arterial pressure.

Effect of position.-Falls in jugular venous pressure of 15-25 cm. of H_2O, were noted when patients were changed from a level to a 15 degree Fowler's position. Increases of 20-30 cm. of H_2O occurred when the patients were tilted into 15 degrees Trendelenburg.

A study of the effect of positional changes on the patient's jugular pressure before and after C6 administration made it evident that the responses were nearly equal under both conditions. On the other hand, the arterial pressure responses to changes in position were minimal before the administration of C6 and became exaggerated following C6.

DISCUSSION

The arterial and venous pressure tracings clearly indicate that the anesthetized patient's response to C6 is very dramatic. Within less than a minute, the systemic circulation is converted from a high pressure system to a low pressure system. As previously pointed out, low pressure systems are not considered harmful when they are the result of a decreased peripheral resistance[6]. The complete sympathetic paralysis produced by C6 dilates all the vessels under autonomic control, producing a markedly decreased peripheral resistance. Under those conditions tissue blood flow is increased. When the oxygen tension of the inhaled atmosphere is elevated, it would seem logical to assume that tissue oxygenation is adequate[7]. However, this situation does not always obtain since patients in the dilated vascular state have better tissue circulation in the dependent portions of their bodies than they do in the elevated portions. Clinically this was demonstrated on several occasions. Hypotensive patients, undergoing neurosurgical procedures, frequently were placed in a 15 degree Fowler's position, since the operative site should be the most elevated portion of the body to secure a bloodless field. On several occasions severe cerebral cyanosis developed while the toenail beds remained pink. This constitutes striking evidence of unequal tissue blood flow in the various segments of the body. When vital organs are among the elevated portions, it is imperative that an accurate knowledge of the intra-arterial pressure of the vessels supplying the organs be known. Enderby[8] recognized the danger of cerebral hypoxia and stressed keeping the head dependent.

Several factors contributed to the large reductions in arterial pressures encountered in this series. All of the patients were anesthetized with Pentothal-curare anesthesia before C6 was administered. There can be little doubt that the anesthesia potentiated the effect of C6. The continuous direct recordings of the intra-arterial pressures might account for the greater falls because of the increased accuracy of the direct method. The majority of the patients were not normal; some were hypertensive; others had increased intracranial pressures; still others were in impending shock. All of these abnormal physiologic states rendered the patients more susceptible to C6.

Contrary to the findings of others[9, 10, 11, 12], relatively normal patients in the supine position experienced large reductions in arterial pressure following the administration of C6. Pentothal-curare anesthesia was the only significant variable in this instance.

The maximum fall in arterial pressure was usually achieved with the initial dose, providing it was large enough. Immediately following the initial drop, the pressure rose to a slightly higher level where it remained stabilized, even though additional C6 was administered. This initial maximum fall may be related to the suddenness of the change in circulatory dynamics. The acute peripheral vaso- and venodilatation may allow blood to pool momentarily with a resultant reduction in its return to the right heart. Cardiac output would then be reduced markedly at this point because of diminished cardiac filling. Presumably the circulatory system would then accommodate to the lowered pressure as arterial blood gradually pushed through the capillary beds, increasing the venous blood volume and pressure. As the venous pressure is restored, the volume of the blood entering the right heart would increase and the cardiac output would rise. Once the cardiac output becomes stabilized to the reduced peripheral resistance, additional C6 will not alter the pressure. (This statement assumes that the initial dose of C6 was large enough to effect maximum vascular dilatation. Under this condition, the peripheral resistance will remain constant unless the blood volume is reduced.)

Positive pressure respiration has been advocated to reduce the arterial pressure to levels lower than those attained with C6 alone[13]. Experimentally these results could not be confirmed when reasonable pressure was employed. (Manual bag compression ranged from 4 to 10 mm. Hg.) Very strong positive pressure respiration is necessary to produce a venous stasis which is sufficient to reduce cardiac output and lower the pressure. The advisability of this maneuver is questionable because the circulation time of the brain would be increased by the obstruction of the cerebral venous return.

Relative maintenance of the diastolic pressure has been reported by others[12]. They attributed this to the increased heart rate which preceded the drop in pressure. Stroke volume was reduced, thereby conserving the diastolic level. In this series the percentage falls in systolic and diastolic levels were about equal. Several explanations may account for this discrepancy in results. First, most of the patients had rapid heart rates because of atropine premedication prior to C6 administration and secondly, a more accurate method of recording arterial pressure was employed.

The variations in behavior of the venous pressure, following the initial injection of C6, have several possible explanations. When C6 is administered to a patient in Fowler's position, the jugular venous pressure will promptly fall since the sudden peripheral venodilatation permits a momentary mass pooling of blood in the dependent veins. At this time the blood, returning to the right heart, will be mainly from the segments which are elevated above the heart. Consequently, a fall in jugular venous pressure results. Patients in the supine or slight Trendelenburg position, did not experience an initial fall in the jugular venous pressure.

The unpredictable degree of hypotension was emphasized in the pressure studied. Elderly patients with hypertension underwent large percentage falls; young individuals with normal pressures experienced moderate falls. Fowler's position significantly contributed to the degree of hypotension obtained. These factors, coupled with clinical observations, led to several obvious assumptions. First, there must be a critical hypotensive level. Secondly, the brain and heart may become hypoxic if the pressure is allowed to fall below the critical level. Thirdly, Fowler's position will alter the critical pressure level, particularly in relation to the cerebral blood flow.

Studies designed to establish a rough critical pressure level are in progress and a method for maintaining the pressure level has been developed. The following pages represent a preliminary report of the studies.

CIRCULATION TIMES

The arm to ear circulation time was

correlated with the arterial pressure in 2 study cases. In each case the circulation time varied inversely with the pressure level. The pre-hexamethonium circulation time was 14 seconds. (This is very close to the normal 13.4 seconds established by Knutson et al[5].) Following an initial fall in systolic pressure from 174 to 66 mm. Hg, the circulation time was nearly doubled.

The circulation time during induced hypotension has been reported as normal by other workers[4]. However, they were estimating the time by clocking the reaction period of drugs injected intravenously.

ARTERIOVENOUS OXYGEN DIFFERENCE OF THE BRAIN

The AV oxygen difference across the brain was determined in 2 cases in the following manner: with the brain exposed, heparinized samples were collected under anaerobic conditions from the middle cerebral artery and the superior sagittal sinus before, during and following C6 induced hypotension. Total blood oxygen contents were determined by the manometric method of Van Slyke and Neill. The heart rate remained constant in both cases and is not included in the following table.

CASE	SAMPLE	B. P.	$(A\text{-}V)O_2$
	I	168/108	8.9
A	II	54/46	10.9
	III	136/100	5.9
	I	126/92	6.9
B	II	66/39	7.6
	III	96/66	5.7

ANOXIA PHOTOMETER STUDIES

Variations in arterial pressure did not significantly alter the ear oxygen saturation while the patients were in the supine position. However, when the patients were tilted to Fowler's position, the oximeter readings fell significantly. The tilting experiments were repeated several times in each case to determine the consistency of the results. When strong positive intrapulmonary pressure was applied continuously for 3 minutes, severe drops in the oximeter reading occurred.

The findings are difficult to interpret when one considers the purpose for which the anoxia photometer was intended. Theoretically, this instrument measures "arterialized" capillary blood. Under normal conditions the oxygen saturation of the blood in the ear capillaries should remain constant as long as no alteration in pulmonary ventilation occurs. Neither pulmonary ventilation nor inhaled oxygen tension was allowed to vary during the tilting experiments, yet the oximeter reading fell. This might be interpreted as a variation in blood thickness, however, the model 17A Anoxia Photometer has a compensating circuit which largely nullifies this error in the normal range of adult ears.

One interpretation of the depression in oximeter readings, resulting from placing the hypotensive patient in Fowler's position, could be stagnation of the ear capillary blood. This interpretation would be consistent with the circulation times and the AV oxygen difference previously mentioned. Strong positive intrapulmonary pressure reduced the oximeter reading markedly. In this instance, venous obstruction and a reduced cardiac output are both present. Stagnant anoxia must be the explanation of the fall in ear oxygen saturation during this maneuver.

DISCUSSION

The few circulation times, AV differences and oximeter studies, carried out on patients with C6 induced hypotension, indicate that the blood flow of vital organs may be reduced to such an extent that severe hypoxia results. This is particularly true of the elevated portions when postural drainage in combination with hypotension are employed to secure a bloodless field.

Crumpton et al[14] studied cerebral circulatory and metabolic changes in hyper-

tensive encephalopathy patients who were under treatment with C6. They found a 29 per cent reduction in the mean arterial pressure and a 30 per cent decrease in cerebrovascular resistance. The cerebral flow and oxygen uptake of the brain were not significantly altered. They noted also a significant decrease in the internal jugular oxygen saturation during hypotension which indicated a lack of cerebral metabolic adjustment to the reduced blood pressure.

The findings in this investigation are in agreement with the above except for the cerebral blood flow. Though cerebral blood flows were not done, the circulation times and AV oxygen difference indicate that they would be decreased. This difference is readily explained by the comparatively small fall in mean arterial pressures reported by Crumpton et al. To substantiate further our belief that both cardiac and cerebral hypoxia may develop if the arterial pressure is permitted to fall below the hypothetical critical level, electrocardiographic and electroencephalographic studies were done.

ELECTROCARDIOGRAPHIC STUDIES

Both Katz[15] and Wiggers[16] have advocated the use of serial ECGs for the detection of myocardial hypoxia. Alterations in the shape of the T wave and/or depression of the ST take-off, are considered indicative of myocardial ischemia.

Electrocardiographic studies were done on several of the cases. All cases showed changes indicating myocardial hypoxia. To illustrate, with a blood pressure of 127/86 the tracing shows that the ST segment takes off from the isoelectric line and the T wave is of normal shape and voltage. A tracing taken at a pressure of 85/62 indicates slight depression of the ST segment with some flattening of the T wave. Further tracings made with the patient in a 15 degree Fowler's position, and with an arterial pressure of 65/55 showed definite ST depression and flattened T waves.

While the pressure was still reduced (85/65), the oxygen was increased from 50 to 100 per cent. A tracing was made following 2 minutes of 100 per cent oxygenation. The ECG again reverted to normal. These changes indicate myocardial hypoxia during hypotension.

ELECTROENCEPHALOGRAPHIC STUDY

Electrocorticograms and simultaneous arterial pressure tracings were made in an attempt to determine, indirectly, the extent of cerebral hypoxia during the hypotensive state.

A Grass model 3C console electroencephalograph, equipped with an ink writing recorder, was employed. The kymograph speed was 3 cm. per second and the machine was calibrated at 100 microvolts per cm.

Our findings were consistent with a depression of cortical activity as the arterial pressure is progressively reduced. The gradual depression in the high voltage, fast activity in all leads was striking. In one instance when the arterial pressure reached 55/45, virtually all activity had ceased. At this point, the pressure was elevated with a vaso- pressor agent and the cortical activity immediately increased.

CONCLUSION

Hexamethonium induced hypotension for the production of ischemic neurosurgical fields is not without danger. Although the neurosurgical procedure is greatly facilitated, the risk of cerebral and cardiac hypoxia is ever present. The indiscriminate use of hypotension has already led to inexcusable fatalities and more will occur in the future if this method continues to be employed as a routine convenience to the surgeon, rather than a benefit to the patient.

Cerebral ischemia is incompatible with adequate cerebral oxygenation. Therefore, it is impossible to prevent all bleeding during neurosurgical procedures; only diminution of surgical hemorrhage can be accomplished.

The procedure should not be attempted

without a continuous recording of the intra-arterial pressure and at no time should the systolic pressure be permitted to fall below 70 mm. Hg.

The findings indicate that Fowler's position markedly increases the danger of cerebral hypoxia. For this reason, the patient is much safer in the level, supine position.

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II. MEDICAL SCHOOL NEWS

Coming Events

March 26	Special Lecture; "Trace Elements in Biochemistry and Medicine"; Dr. Bert L. Vallee, Associate in Medicine, Harvard Medical School, and Research Associate, Department of Biology, Massachusetts Institute of Technology; Owre Amphitheater; 4:00 p.m.
March 27	Tape-Recorded Symposium: Is the Concept of Science Different in Biology than it is in the Physical Sciences? Owre Amphitheater; 3:00 p.m. (This is a tape recording of a discussion which was held on December 8, 1952, at the meeting of the Boston Society of Biologists. The participants were Dr. James B. Conant, Harvard; Dr. Phillip G. Frank, Harvard; and Dr. Paul Weiss, University of Chicago.)
April 2	Special Lecture; "On the Possibility of Vector Analysis in Electrocardiography"; Dr. Hans Schaefer, Professor of Physiology, University of Heidelberg, Germany; Veterans Administration Hospital, Building 1, Conference Room; 8:15 p.m.
April 3	Special Lecture; "Coronary Circulation"; Dr. Hans Schaefer, Professor of Physiology, University of Heidelberg, Germany; 129 Millard Hall; 4:30 p.m.
April 6-11	Continuation Course in Proctology for General Physicians
April 16-18	Continuation Course in Gynecology for Specialists
April 27-29	Continuation Course in Gastroenterology for General Physicians
April 28	Clarence M. Jackson Lecture; "Gastro-Intestinal Symptoms with Particular Reference to Motor Disturbance"; Dr. Chester M. Jones, Clinical Professor of Medicine, Harvard University Medical School, Boston; Owre Amphitheater; 8:00 p.m.
April 29	Family Doctors' Day; Powell Hall Recreation Lounge; 12:00 o'clock noon.

* * *

Dr. Scherer Selected as Markle Scholar

The Directors of The John and Mary R. Markle Foundation recently announced the selection of Dr. William F. Scherer, Assistant Professor, Department of Bacteriology and Immunology, as a Markle Scholar in Medical Science. This scholarship pays a total of $30,000 over a five-year period. Dr. Scherer becomes the fourth member of our faculty who has been so honored. Previous recipients from Minnesota are Doctors George E. Moore, Robert A. Good, and Leonard F. Peltier. The entire faculty joins in extending congratulations to Dr. Scherer on this well-deserved award.

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Alumni Notes

Robert H. Alway, '37 BS; '40 MD, has been appointed as Professor and Head of the Department of Pediatrics at the University of Colorado School of Medicine. Since 1949 Dr. Alway has been Associate Professor of Pediatrics at Stanford University School of Medicine. He assumed his new position with the Colorado institution on January 1.

Dr. Mildred Schaffhausen, interning at Minneapolis General Hospital, was awarded the scholarship citation and $100 from the American Women's Medical Association, in December.

(Continued on next page)

American Medical Education Fund Drive

H. E. Drill, M.D.

Chairman of Minnesota Division and
Sixth Councilor District and
Member, Board of Trustees, Minnesota Medical Foundation

As State and Sixth Councilor District Chairman of the American Medical Educational Fund Drive, I would like to bring to your attention the pertinent details relating to this extremely important and worthwhile project sponsored by your American Medical Association.

State Chairman of this educational endeavor met with the A. M. A. committee in Chicago on January 25, 1953. Those in attendance were given a comprehensive outline of the aims and objectives of the Medical Educational Fund. The success of this project has broad and far-reaching implications which could well determine the future policies of our medical schools as well as to indicate to the American people and our government how far we as doctors are willing to go in defense of the private practice of medicine in this country. We are fortunate in having the enthusiastic support and financial backing of many private industrial enterprises.

I should like to say here that I believe after making a study of the AMEF, that it is a worthy cause. The medical schools of our country are the foundation of America's health. They turn out the doctors, medical scientists, and researchers essential to economic and social progress. They train the medical manpower needed to staff our hospitals, health programs, and the expanding practice of industrial medicine. To lower the standards of teaching or to reduce the medical manpower would be disasterous.

Careful and exhaustive studies indicate that our 79 approved medical schools will require additional annual subsidies amounting to approximately ten million dollars. The funds must come either from government or private sources. It is obvious from the report submitted by the committee sounding out American business, that private industry prefers the latter. Both business and industry have already subscribed generously to this medical educational fund. However, many more thousands of dollars have been pledged and will be forthcoming, contingent upon the extent to which doctors of medicine will respond in support and defense of their chosen profession.

Perhaps you are now asking, where does the need for such a large annual fund lie? Medical training is by far the most expensive field of education. The rapid increases in costs have been staggering. The average cost of training a doctor has doubled in twenty years to $10,000. Tuition fees, raised 165% since 1940, pay only one-fifth of this cost. Other factors contributing to the increased cost are 1) lengthy period of training 2) costly laboratory facilities 3) high ratio of teachers to students 4) complicated training techniques arising from recent scientific advances. Why doesn't each state-supported school increase its budget request to cover its needs? The answer to this is the same as it always has been. Drastic cuts in budgets submitted to the state legislatures are common. Furthermore, budgets are set up for specific uses. Herein lies the difficulty--lack of unrestricted funds. Unrestricted funds can be used to augment the salaries of worthy underpaid faculty members, to provide laboratory facilities, to improve and expand out-patient services for medical students, etc.

How are we as doctors in active practice to benefit?

(Continued on next page)

The medical schools of our nation are graduating over 6,000 new doctors every year--doctors with new knowledge, new techniques, and vast new resources.

It should be clear to every doctor that if medical schools continue to operate without proper finances it will seriously damage the teaching system and ultimately lower the standard of medical practice in the United States. American doctors cannot afford to permit the weakening of their professional standards which are the backbone of a nation that enjoys the highest health levels in the world today, nor can they permit the creeping paralysis of government control to envelope their schools and ultimately dictate the standards of medical practice.

Give serious thought to this venture, in which each one of us must play a part, whether we give generously according to our means, or not at all. Your contribution can be designated to the school of your choice, in which instance every dime of it will be channelled back to that school by the National Fund. Funds given directly to any medical school do not become a part of the National Fund and cannot, therefore, reflect professional support, which in turn would have a bearing upon contingent donations from business and industry. Undesignated gifts will be prorated among the 79 medical schools.

Bear in mind, contributions to the AMEF are tax exempt. Therefore, you can afford to be generous by allowing yourself a reduction in your taxes at the same time you are supporting a mighty worthy cause.

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Publications of the Medical School Faculty

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III.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

March 23 - 28, 1953

Monday, March 23

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.

12:30 - 1:30 Physiology Seminar; 214 Millard Hall.

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

4:00 - Pediatric Seminar; The Care of the Premature Infant; William Bevis; Sixth Floor West, U. H.

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

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

4:30 - 6:00 Physiology 114A and Cancer Biology 140 -- Research Conference on Cancer, Nutrition, and Endocrinology; Drs. Visscher, Bittner, and King; 129 Millard Hall.

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

Ancker Hospital

8:30 - 10:00 Tuberculosis and Chest Conference; Auditorium.

2:00 - 3:00 Surgery Journal Club; Classroom.

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.

Monday, March 23 (Cont.)

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.

1:30 - Cardiac Rounds; Drs. Ebert and Berman.

2:00 - Psychosomatic Rounds; Bldg. 5.

4:00 - Cardiac Conference; Drs. Ebert, Berman and Simonsen.

Tuesday, March 24

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

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

Minneapolis General Hospital

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

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; Sta. A; Willard White, et al.

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.

Tuesday, March 24 (Cont.)

Veterans Administration Hospital (Cont.)

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

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

9:30 - Infectious Disease Rounds; Dr. Hall.

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 - 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.

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

Wednesday, March 25

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.

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.

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

Minneapolis General Hospital

8:30 - 9:30 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.

Wednesday, March 25 (Cont.)

Minneapolis General Hospital (Cont.)

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

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

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.

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

2:00 - 4:00 Infectious Disease Rounds; Main Conference Room, Bldg. I.

4:00 - 5:00 Infectious Disease Conference; Wesley W. Spink; Conference Room, Bldg. I.

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

Thursday, March 26

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

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 - Special Lecture; Trace Elements in Biochemistry and Medicine; Bert L. Vallee; Associate in Medicine, Harvard Medical School, and Research Associate, Department of Biology, Massachusetts Institute of Technology; Owre Amphitheater.

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

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.

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.

Thursday, March 26 (Cont.)

Minneapolis General Hospital (Cont.)

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

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

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

1:00 - House Staff Conference; Station I.

2:00 - 4:00 Infectious Disease Rounds; Classroom.

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

Veterans Administration Hospital

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

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

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

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

Friday, March 27

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

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; A Study of Degenerative Changes of the Cervical Spine in Relation to Age; Russell Blanchard; 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 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.

Friday, March 27 (Cont.)

Minneapolis General Hospital (Cont.)

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

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

1:00 - Medical Pathology Slide Conference; E. T. Bell and D. F. Gleason; Conference Room, Bldg. I.

Saturday, March 28

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; Station 54.

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. Goldish and Bolin.
11:15 - 12:00 Morphology . . . . . Dr. Aufderheide.

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* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.