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University of Minnesota Hospitals
and
Minnesota Medical Foundation

Problems of Oxygen Therapy

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BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
and
MINNESOTA MEDICAL FOUNDATION

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I. PROBLEMS OF OXYGEN THERAPY

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1. Causes of Hypoxia

A. Decreased oxygen in the inspired air.
B. Interference with oxygen flow through respiratory tree.
C. Decreased ventilatory power
D. Interference at pulmonary alveoli.
E. Decreased carrying power of the blood for oxygen.
F. Circulatory insufficiency.
G. Enzymatic toxicity in the cell.

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A. Decreased oxygen and inspired air

In modern medicine it is only rarely that a decrease of the oxygen tension of the inspired air is important except in the field of anesthesia. In mountain climbing or high altitude flying, a decrease of oxygen tension may be a factor, but under normal circumstances in the practice of medicine we are not concerned with it. At sea level, with a barometric pressure of 760 mm. of mercury, the oxygen tension of the air is about 158 mm. Hg. The oxygen tension of the alveolar air is approximately 101 mm. Hg.

B. Interference with oxygen flow through respiratory tree

Of all the causes of hypoxia likely to be seen medically, interference with flow of air to the lung is one of the commonest. Complete obstruction of the airway will cause death in a few minutes. However, in many conditions it is common to have partial obstruction of the airway which handicaps the patient in his repiration and limits his activity, yet which goes uncorrected for a prolonged period of time. The type of obstruction may be considered according to the site at which it occurs.

Pharyngeal obstruction is due either to paresis of the musculature of the pharynx or to lodging of foreign bodies in the pharynx. Neurological conditions, such as hemiplegia with bulbar palsy, poliomyelitis, or myasthenia gravis, traumatic lesions, severe infections, all may cause a paresis or paralysis of the musculature of the pharynx. In these cases there is either collapse of the pharyngeal airway, or the pharynx is unable to clear secretions from the airway by swallowing. The tongue may fall back against the posterior pharyngeal wall, partially or completely blocking the airway. Consequently the airway remains small, and resistance to the flow of air is increased. Under normal circumstances, secretions draining into the throat are swallowed promptly so they do not accumulate in the lower pharynx around the glottis. Examination of the normal lower pharynx will show that there are no secretions present in the piriform sinuses nor back of the throat. When there is paresis of the muscles of the pharynx, puddling of secretions occurs and one or both piriform sinuses may be found to be full of mucus at all times. As saliva accumulates in the lower pharynx, the patient must exercise care in breathing so that he does not inhale it into the trachea. For this reason the patient alters his respiration so that he keeps his glottis closed as much of the time as possible. He breathes with short, shallow breaths to avoid aspirating saliva.

Patients who have pharyngeal paresis may find that they must use the lower muscles of the pharynx as well as the muscles of the trachea to guard against aspiration of fluid. Because of this intermittent blocking of the airway these patients tend to remain underventilated at all times. When the patient has a muco-purulent postnasal secretion the problem of maintaining an open airway is more difficult. Choking and coughing occur more frequently as these secretions collect in the lower pharynx and cannot be removed. For this reason, patients who have difficulty in maintaining a pharyngeal airway find that the difficulty is aggravated by upper respiratory

infections which produce a post-nasal discharge.

Food which increases the amount of mucus secreted in the saliva also will increase the problem of maintaining an open airway. This is particularly true of foods which contain milk. Milk stimulates the secretion of mucus in saliva. Patients who have a severe paresis of the pharyngeal muscles should not be fed milk foods. Gradually as the patient learns to handle his secretions, milk can be added to his diet again.

Laryngeal obstruction also may interfere with the flow of air to the lungs. Bilateral paralysis of the abductors of the vocal cords is rather rare even in bulbar poliomyelitis. When it occurs the vocal cords fall together in the midline and the patient is unable to inhale. Under these circumstances, an artificial airway is the only means of maintaining such a patient. In bulbar poliomyelitis this has been seen on a number of cases. The paralysis may be temporary with the patient gradually regaining enough control so that he is able to keep his airway open. In a few cases a permanent tracheotomy has been necessary.

More common than abductor paralysis is reflex closure of the glottis to prevent aspiration of fluid when there is paresis of the lower pharynx. Any mechanical stimulation of the larynx or trachea will cause reflex closure of the glottis and block the airway. Thus when patients have flooding of the lower pharynx with saliva which begins to trickle down into the larynx, the patient will have paroxysms of choking which interfere with normal respiration. To avoid this the vocal cords are approximated reflexly except during a short inspiratory phase. This protective reflex may contribute materially to asphyxiation of the patient. At the same time that there is reflex closure of the vocal cords, there is inhibition of deep breathing to avoid aspiration of material. These two conditions combine together to produce a severe hypoxia which may result in cyanosis. Obviously the only answer to this condition is either to maintain the airway free of saliva by adequate suctioning, or to produce a satisfactory artificial airway so that the puddling of saliva in the pharynx does not interfere with adequate respiration.

In certain inflammatory conditions of the upper respiratory tract, there may be enough edema or inflammation of the larynx to cause obstruction to the flow of air into the lungs. Particularly in severe allergic reactions or in diphtheria, this laryngeal obstruction has proved to be a serious problem. Laryngeal tumors also may block the airway at the larynx enough to interfere with exchange of oxygen. Occasionally food or other foreign bodies are aspirated into the larynx and cause blocking, both by mechanical obstruction and by reflex stimulation of the larynx to cause spasm and closure of the vocal cords.

Tracheal obstruction may also be caused by inflammation or edema, similar to that causing laryngeal obstruction. Secretions and exudates on the wall of the trachea are removed in two ways. The ciliary action of the mucous membrane of the trachea carries exudates up to the lower level of the larynx. At that point, the ciliated mucous membrane ends and material must be coughed across the larynx. Therefore under conditions in which the ability to cough has been lost, that is, when there is disturbance of function of the vocal cords, the muscles of respiration, or the abdominal muscles, it is not possible to clear the trachea adequately of secretions. Accumulated secretions may interfere with movement of the air through the trachea or the secretions may eventually be aspirated into a bronchus, causing atelectasis.

C. Decreased ventilatory power

When the muscles of ventilation are weakened or paralyzed, there is a decreased ability to carry on respiration. The muscles of respiration may be divided into the primary respiratory muscles and the accessory respiratory muscles.

The primary muscles of respiration are the intercostal muscles and the diaphragm. Under normal circumstances, these muscles carry on the major part of all respiratory efforts. It is only under conditions of unusual exertion that the other muscles are needed for respiration. The common accessory muscles of respiration which are substituted frequently under conditions in which the primary muscles of respiration are ineffective are the sternocleidomastoids, the abdominals, the muscles of the pectoral girdle, the strap muscles in the neck and the glottis. The sternocleidomastoid muscles, pectoralis minor, and strap muscles of the neck function as muscles of inspiration. The abdominal muscles and the pectoralis major act as muscles of expiration. When a patient has these muscles available, he can learn to substitute them for the primary muscles of respiration.

The muscles of the glottis also may be used to aid in respiration when there is respiratory weakness. At the end of inspiration the glottis may be closed, to hold the inspired air in the lung. When the vocal cords open expiration occurs passively. This type of breathing is seen frequently when there is respiratory distress, and is accompanied by the so-called "respiratory grunt". Such breathing always indicates that there is an impairment of respiration.

Conditions which increase the rigidity of the thorax reduce the effectiveness of the muscles of ventilation, and may result in inadequate respiration. Thoracic rigidity may be increased by spasm of the intercostal muscles, the muscles of the pectoral girdle, or the abdominal muscles. Such spasm is seen frequently in acute poliomyelitis. This muscle spasm limits inspiration mechanically. Moreover, when these muscles are in spasm, respiration causes pain, and consequently respiration is inhibited reflexly in an attempt to avoid pain. Pulmonary congestion, emphysema, and pulmonary fibrosis also increase the rigidity of the thorax and the work that must be done in carrying on respiration.

D. Interference with oxygen transport across the alveolar membrane

If there is thickening of the alveolar membrane or accumulation of fluid in the alveoli, there is an increased barrier to the absorption of oxygen into the blood. Since oxygen travels across the alveolar barrier by diffusion and since the rate of transfer is inversely proportional to the square of the distance the thickness of the alveolar barrier is important. With pulmonary congestion and minimal alveolar edema the distance for diffusion is increased and transfer of oxygen is slowed. With increasing pulmonary congestion and edema alveoli are completely filled and become nonfunctional as respiratory surface. Normally there are approximately 300 square meters of alveolar surface available for the absorption of oxygen. As pulmonary congestion occurs this effective surface area is cut down progressively, and with it the oxygenation of the arterial blood. As congestion progresses, the distended blood vessels cause collapse of the alveolar sacs. Loss of aeration and decreased oxygen tension in the alveoli results in increased permeability of the alveolar epithelium to plasma protein and red cells.[1] There is an increased filtration of plasma and red cells into the alveolar spaces, producing typical pulmonary edema. If enough of the alveolar surface becomes involved the patient dies from asphyxia.

There are a number of factors that contribute to pulmonary congestion. Of these hypostatic congestion due to immobilization of the patient on his back is probably one of the commonest. Patients who are helpless for any reason, such as patients paralyzed with polio or hemiplegia, patients who are in a cast and unable to turn, or patients who are prostrate with illness or are under an anesthetic will develop hypostatic congestion in the dependent part of the lung in a few hours. This hypostatic congestion is reversible if the patient is turned, but may be progressive if the patient remains immobilized in the same position. For this reason, it is essen-

tial that patients who are unable to turn themselves should have assistance in order to turn completely to one side or the other at least every hour so that the dependent portion of the lung may be up for an interval of time and the blood congested in it may be allowed to drain away.

E. Decreased carrying power of the blood for oxygen

Oxygen is carried in the blood in solution in the plasma and also in chemical combination with the hemoglobin. Hemoglobin will combine with 1.34 cc. oxygen per gram of hemoglobin. The saturation of hemoglobin with oxygen is proportional to the tension of oxygen in solution. (Fig. 1). The curve is sigmoid. Hemoglobin approaches 97% saturation at the normal alveolar pO_2 of 100 mm. Hg. As the pO_2 is decreased there is a relatively flat decline of hemoglobin saturation so that at a partial pressure of oxygen of 60 mm. Hg. the hemoglobin still is 90% saturated. Below that level the unsaturation of hemoglobin occurs more rapidly. At a pO_2 of 40 mm. Hg. the hemoglobin is 75% saturated; at 30 mm. Hg. it is 60% saturated; and at 20 mm. Hg., 35% saturated. This dissociation curve provides that for a decrease of pO_2 from 60 mm. Hg. to 20 mm. Hg., 55% of the oxygen is released from the hemoglobin.

Oxygen is carried to and from the hemoglobin in solution. The hemoglobin acts as a reservoir for oxygen carried in the blood. The oxygen saturation of the hemoglobin at all times depends on the tension of oxygen in solution in the plasma, which in turn will depend on the oxygen supply from the lung or the oxygen demand by the tissues.

In anemias the amount of hemoglobin in the blood is decreased, decreasing the reservoir for storage of oxygen. Consequently when the tissues demand oxygen the supply in the blood is exhausted more rapidly than normal. Also, hemoglobin may be rendered useless for respiration by forming stable combinations with other compounds. Among these are compounds which change the ferrous iron of hemoglobin to ferric iron to produce methemoglobin, such as methylene blue, nitrates, chlorates, and benzine. The sulfa drugs produce sulfhemoglobin as a stable compound. Carbon dioxide combines with hemoglobin to form carboxyhemoglobin. Carboxyhemoglobin is a reversible compound, just as is oxyhemoglobin. However, carbon monoxide has 250 times the affinity for hemoglobin that oxygen has. Consequently, the relative amounts of carboxyhemoglobin and oxyhemoglobin will depend on the partial pressures of the two gases and the affinity of each gas for hemoglobin. In the presence of small amounts of carbon monoxide in the inspired air, appreciable amounts of carboxyhemoglobin will be formed. Conversely, by increasing the oxygen tension in the inspired air, more oxyhemoglobin will be formed and carboxyhemoglobin will dissociate.

F. Circulatory insufficiency

Circulatory insufficiency, whether caused by cardiac decompensation or peripheral vasoconstriction, decreases the circulation through the capillaries. Stagnation of blood in the capillaries results in a decrease of oxygen tension of the plasma and desaturation of hemoglobin. Diffusion of oxygen from the capillary to the cell is proportional to the oxygen tension gradient. As this gradient diminishes, the transfer of oxygen diminishes. This results in hypoxia of the cell. Cellular metabolism is impaired progressively with increasing hypoxia until at a pO_2 of approximately 20 mm. Hg. the vital centers of the brain can no longer function. Since these centers must function continuously during life, the brain pO_2 must always be higher than 20 mm. Hg. The pO_2 of the capillaries must exceed this tension in order for diffusion of oxygen to occur. For that reason we must consider 20 mm. Hg. pO_2 as the end point of diffusion. An arterial pO_2 of 100 mm. Hg. (hemoglobin saturation 96 per cent) produces a diffusion gradient to the cell of 80 mm. Hg. When the hemoglobin saturation of the capillary is decreased to 90 per cent the pO_2 is 60 mm. Hg. and the diffusion

gradient and rate of transport of oxygen from the capillary to the cell is 50 per cent of normal. When the hemoglobin saturation is decreased to 80 per cent the plasma pO_2 is 45 mm. Hg. and the diffusion gradient and rate of oxygen transport from the capillary to the cell is 31 per cent of normal. Unless this relationship between hemoglobin saturation and oxygen transport is understood, the value of the former is likely to give a false sense of security regarding the status of oxygenation in the vital centers.

Tissue edema might be considered another factor in circulatory insufficiency. Edema in the tissues increases the distance through which diffusion must occur and thereby decreases the adequacy of the supply of oxygen to the cells. Again since the rate of oxygen transferred from the capillary to the cell is proportional to the oxygen tension gradient and this gradient falls off proportional to the square of the distance from capillary to cell, when edema occurs there is a pronounced decrease in the rate of oxygen transfer to the cell.

2. Oxygen Therapy

A. Provision of an adequate airway

Air flows through the respiratory tree, as through any airway, according to certain physical principles. Poisseuille's law describes these relationships.

$$V = \frac{P_{\pi} r^4}{8 \ln}$$

- V = volume flow of gas
- P = pressure gradient across the tube
- r = radius of the tube
- l = length of the tube
- n = viscosity of the gas

This law is derived from the more basic law that volume flow is proportional to pressure divided by resistance.

It should be noted that the major factor in resistance is the bore of the tube: resistance is proportional to r^4. The importance of reduction of the cross-sectional area of the airway can be understood when we consider the following:

Diameter	Volume of gas exchange
1	1
1/2	1/16
1/3	1/81
1/4	1/256

For this reason to cut resistance as much as possible, any airway should have the largest diameter that is feasible.

The resistance is directly proportional to the length of the tube. Therefore all tubing should be as short as possible to avoid increasing the resistance.

Resistance is also related to the viscosity of the gas breathed. About 1/3 of the work of normal breathing is due to resistance to flow of air. When the flow of air is fast enough to cause turbulence, the resistance goes up with the square of the rate of flow. Below the critical velocity at which turbulence occurs, resistance increases proportional to the rate of flow. Helium is of value in oxygen therapy only in conditions in which there is turbulent flow of air. Under normal conditions of streamlined flow helium does not flow with less resistance than air. However, during turbulent flow the increase in resistance is less for helium mixtures than it is for air or pure oxygen.[2] The increased resistance due to marked constriction of the airway such as occurs in asthma is due to turbulent flow of air. This resistance may be reduced by breathing helium-oxygen mixtures rather than air. However, if the airway is of normal size the critical velocity for turbulence is approximately 300 liters per minute. This rate is rarely exceeded in normal respiration. So with an airway of normal size helium mixtures are of no advantage.

The maximum velocity of airflow and consequently maximum turbulence occurs in the upper airway at the glottis. A large proportion of the resistance also occurs at that level. At a respiratory exchange of 6 liters per minute, 58% of the resistance to airflow is in the upper airway. At the rate 60 liters of air per minute respiratory exchange, 71% of the resistance is in the upper airway.

These physical considerations of the factors influencing resistance in the airway may be summarized: that any airway should have as large a bore as possible and be as short as possible in order to provide minimal resistance.

The normal airway through the pharynx and larynx will not be altered in length but may be appreciably altered in cross-section if there is collapse of the tongue into the posterior pharynx or accumulation of secretions to cut down the size of the airway. It is important that the airway be maintained open by suctioning and positioning to prevent an increased resistance to the flow of air. If the normal airway cannot be maintained, an artificial airway should be used. The tracheotomy is a relatively simple procedure which can be performed quickly to provide an adequate airway. Large tracheotomy tubes should be used. For adults a number 5 or number 6 tube should be used to avoid increased resistance. A number 4 tube should be used for all except the smallest children.

In calculating resistance to respiration we are accustomed to think of respiratory exchange as being approximately 8 liters of gas per minute. However, we often neglect the fact that this gas must flow both into and out of the lung each minute. Moreover, we may forget that inspiration or expiration occurs for only a fraction of the total cycle time. Under normal circumstances, inspiration lasts about one second. There are 16 such inspirations per minute with normal, quiet respiration. If the normal minute volume of respired air is 8 liters, the tidal volume is 0.5 liter, and the rate of airflow into the lung during inspiration will be 30 liters per minute. With fever or exertion, the minute respiratory volume may be increased above 8 liters, and the inspiratory rate may rise to 50, 60, or even 100 liters per minute during inspiration. With these facts in mind it is easy to see that even a moderate constriction of the airway may cause respiratory distress. For adequate respiration air or oxygen must be inspired intermittently at a rapid rate.

To breathe oxygen through a catheter into a tracheotomy tube at a constant rate of 4 to 6 liters per minute will not provide adequate ventilation. The rate of flow of oxygen is not fast enough during the period of inspiration to allow filling of the lung and the catheter in the tracheotomy tube obstructs the free flow of air to and from the lung. Consequently the lung will be underventilated. The oxygen tension of the alveolar air will diminish and the carbon dioxide tension will increase. This results in asphyxia. Consequently, if the upper airway is not adequate attempts to supply oxygen through a tracheotomy by means of a catheter will do more harm than good.

A catheter used to suction secretions accumulated in the tracheotomy tube or trachea also may be dangerous if not used properly. During the time that the catheter is in the tracheotomy tube there is obstruction of ventilation and asphyxia develops. Oximeter studies have shown that prolonged suctioning may result in a degree of hypoxia (75% hemoglobin saturation) which may be dangerous to the patient.[3] Therefore, the catheter should be inserted quickly, suction continued for only two or three seconds, and then the catheter removed to allow ventilation of the patient. If it is necessary to suction again this may be repeated a number of times with intervals for respiration.

The oxygen humidifier apparatus which is used at this hospital is illustrated in Figure II.[4] Use of this apparatus provides humidified oxygen in the desired

concentration to the tracheotomy of the patient. It is necessary that oxygen introduced into the tracheotomy tube be humidified to prevent drying of the tracheal and bronchial mucous membranes. The content of oxygen in the inspired air can be altered by attaching a tank of oxygen and a tank of air or of 80% helium-20% oxygen through a Y tube to the humidifier bottle of this system. 100% oxygen is obtained directly from the oxygen tank. Lower concentrations of oxygen are obtained by dilution with air according to the following table:

90% O_2 = 5 liters O_2 + 1.0 liter air.

80% O_2 = 4.5 liters O_2 + 1.5 liters air.

70% O_2 = 4.0 liters O_2 + 2.0 liters air.

60% O_2 = 3.0 liters O_2 + 3.0 liters air.

50% O_2 = 2.5 liters O_2 + 3.5 liters air.

Air or 20% oxygen in helium should be used to dilute oxygen if lower than 100% concentrations of oxygen are desired. Pure nitrogen should never be used since if the oxygen tank runs empty and pure nitrogen is supplied to the patient he will die of anoxia in a few minutes. It is desirable to give the patient a sufficient amount of oxygen in the inspired air so that his hemoglobin is 97% saturated at all times. This will produce an arterial pO_2 of 100 mm. Hg. Usually patients are maintained adequately on a concentration of fifty to seventy per cent oxygen in the inspired air. If there is evidence of cellular hypoxia from local edema, it may be desirable to increase the arterial oxygen tension, by increasing the oxygen content of the inspired air.

Since the rate of diffusion is proportional to the oxygen tension, it is possible to increase the rate of transportation of oxygen about five-fold by shifting from air to pure oxygen. In pulmonary edema this may mean the difference between complete saturation of hemoglobin and incomplete saturation of hemoglobin. At the capillary it may mean the difference between adequate oxygenation of the cell so it can perform its normal metabolism and severe hypoxia which may lead to the death of the cell.

Summary

The causes of hypoxia have been considered. The importance of maintaining an airway of sufficient cross-sectional area to allow free flow of gas to the lung has been stressed. If a normal upper airway cannot be maintained, a tracheotomy with a large tube provides an adequate substitute. All tubing connected to a tracheotomy should be of large bore.

The saturation of hemoglobin with oxygen is determined by the tension of oxygen dissolved in the plasma. The plasma oxygen tension also determines the rate of diffusion of oxygen from the capillary to the cell. Small changes in hemoglobin saturation indicate relatively great changes in oxygen tension. Breathing gas mixtures containing high concentrations of oxygen increases the rate of diffusion of oxygen to the cell.

References

1. Warren, M.F., Peterson, D.K., and Drinker, C.K.
The effects of heightened negative pressure in the chest, together with further experiments upon anoxia in increasing the flow of lung lymph.
Am.J.Physiol.137:641-48, '42.
2. Dean, R.B. and Visscher, M.B.
The kinetics of lung ventilation.
Am.J.Physiol.134:450-67, '41.
3. Elam, J.O., Hemingway, A., Gullickson, G. and Visscher, M.B.
Impairment of pulmonary function in poliomyelitis.
Arch.Int.Med.81:649-65, '48.
4. Kubicek, W.G., Holt, G.W., and Kottke, F.J.
Physiology of respiration as applied to the treatment of bulbar poliomyelitis.
Arch.Phys.Med.,29:84-88, '48.

DISSOCIATION CURVE OF HUMAN BLOOD

WITH VARIOUS CO_2 PRESSURES

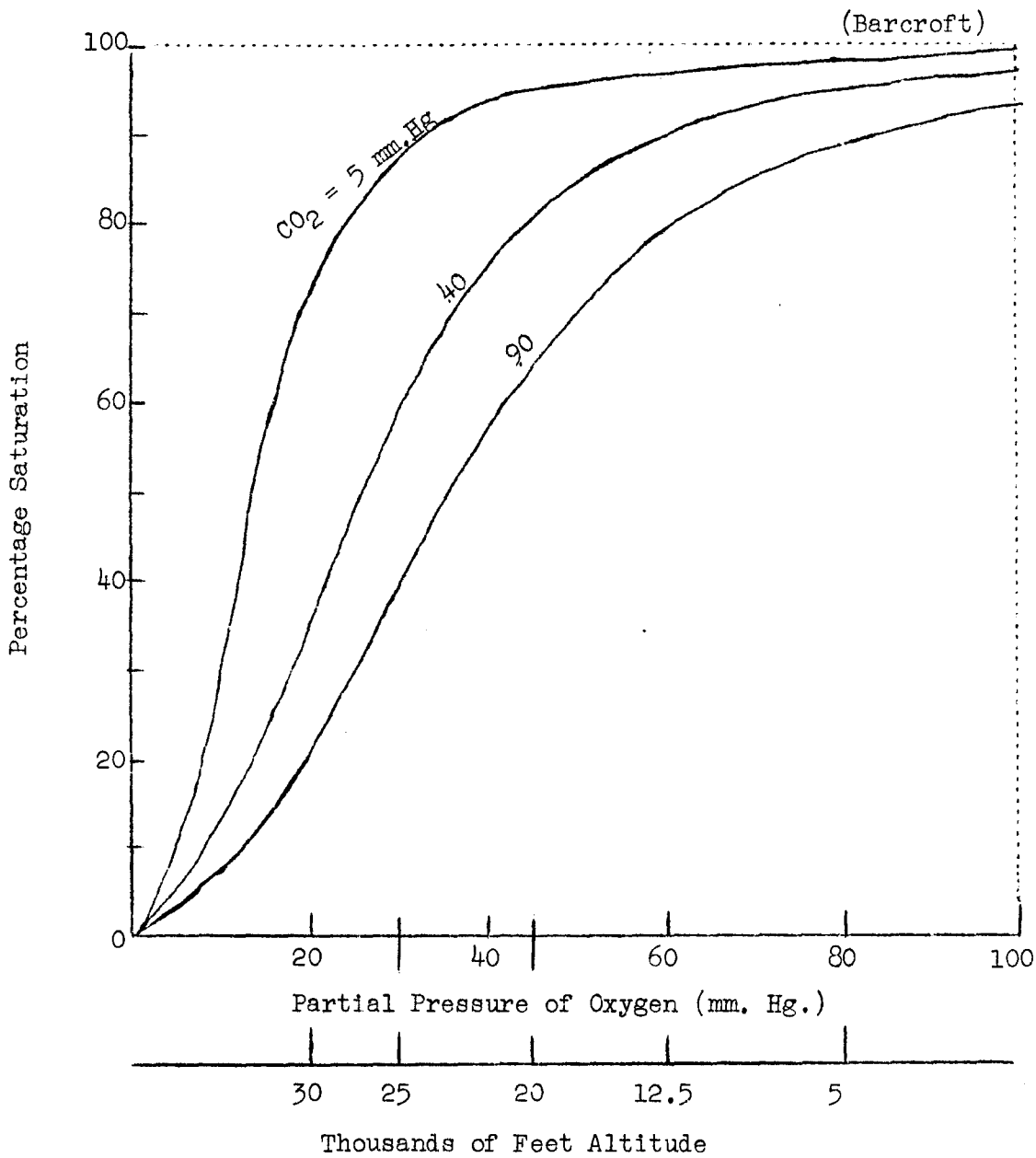

Figure I. Dissociation curve of hemoglobin at various carbon dioxide pressures (after Barcroft).

The normal pCO_2 is 40 mm. Hg. but will be decreased by hyperventilation. The partial pressure of oxygen on the abscissa is shown in relation to the effect of altitude on alveolar pO_2 to demonstrate the great significance of small changes of hemoglobin saturation in relation to the amount of oxygen available to the cell.

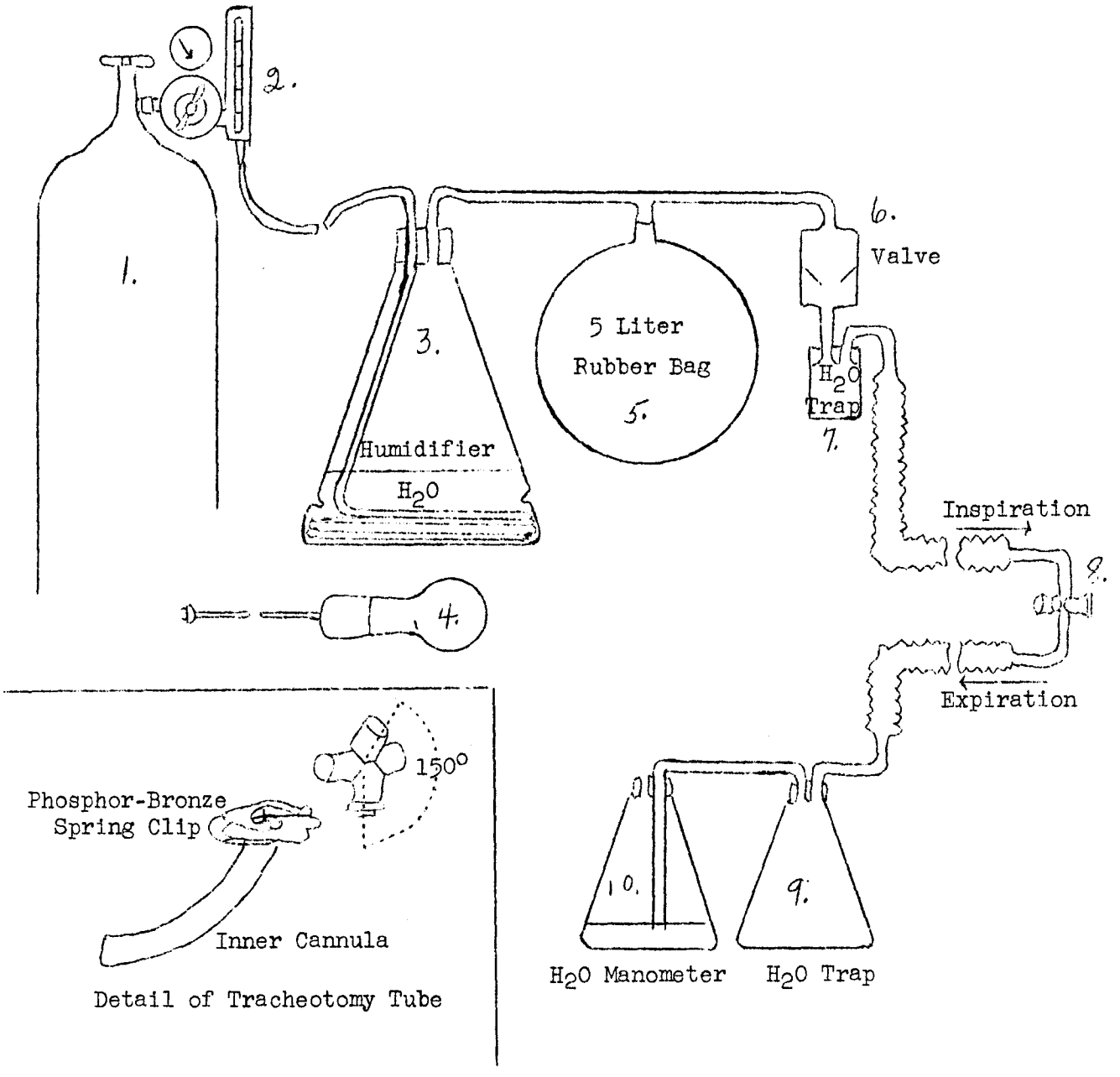

Figure II. Diagram of oxygen humidifier apparatus showing oxygen tank[1] with reducing valve and flow meter[2] calibrated in liters per minute, humidifier bottle[3] containing water and an air disperser.

A light[4] below the humidifier bottle raises the temperature of the water to about 34° so that the gas is more nearly saturated as it reaches the lungs. The 5-liter rubber anesthesia bag[5] is used as a gas reservoir, allowing the patient adequate volume for respiration. This bag should be nearly distended at all times during normal operation of the equipment. The one-way valve[6] prevents the back-flow of expired air into the rubber reservoir. Below the one-way valve is a water-trap[7] to catch moisture which accumulates by condensation. A one inch corrugated rubber tubing leads to the patient. This large-size tubing is used to decrease the resistance to flow of gas. The tubing is

connected by a plastic L-tube to one of the arms of the tracheotomy cross-connector. From the other arm of the cross a similar tube leads into the expiratory circuit. An empty bottle[9] acts as a water-trap to prevent aspiration of water into the tubing or trachea if the patient takes a deep breath when coughing. Water in the last bottle[10] serves three purposes: (1) It provides a small amount of positive pressure against which the patient breathes; (2) it acts as a valve to prevent back-flow of room air into the system; and (3) it bubbles with each respiration, indicating that normal respiration is occurring. The tracheotomy tube that is used with this apparatus is modified so that on the inner cannula there is a spring clip to hold the cross connector. The connector has two horizontal and a vertical arm. To the two horizontal arms are connected the large rubber tubings in the humidifier circuit. A rubber cap is placed over the vertical arm. This cap is removed whenever the oxygen supply is turned off or when it is necessary to suction the patient. By the use of the vertical arm in this way it is possible to suction the patient without discontinuing his oxygen therapy.

II. MEDICAL SCHOOL NEWS

Coming Events

May 22-27 - Continuation Course in Proctology for General Physicians.

May 25 - Special Lecture: "Review of Chemical and Pharmacological Investigations on Cardiac Glycosides", Dr. Arthur Stoll, University of Basle, Switzerland, Medical Science Amphitheater - 4:00 p.m.

May 25 - Medical Six O'Clock Club Mixer and Dinner for medical students and faculty, Main Ballroom, Coffman Memorial Union - 6:00 p.m.

June 12 - Minnesota Medical Alumni Association and Minnesota Medical Foundation Dinner - Spaulding Hotel, Duluth, Minnesota (during annual meeting of Minnesota State Medical Association) - 6:00 p.m.

* * *

Medical Six O'Clock Dinner

Many medical school alumni and faculty will recall with pleasure the annual meetings of the Medical Six O'Clock Club. These dinners for undergraduate medical students and faculty were always highlights of the school year. Entertainment, good fellowship, and informality prevailed and an opportunity was afforded for faculty and students to have fun together. Unfortunately, the Medical Six O'Clock Dinner was a casualty of the war. Many faculty members expressed keen disappointment over the fact that these friendly gatherings were no longer a part of the Medical School scene.

It is, therefore, welcome news that the Medical Interfraternity Council has taken over the task of sponsoring and promoting the Medical Six O'Clock Dinners. The first mixer and banquet at which, incidentally, wives are most cordially welcome, will be held at 6:00 p.m. on Thursday evening, May 25, in the main ballroom of Coffman Memorial Union.

The committee in charge has promised that laughter and good fellowship will abound. All clinical, full-time faculty members and their wives are cordially invited to join with the student body for what will prove to be a gala occasion.

Tickets at $1.75 per plate may be obtained at the Medical School Office.

* * *

"Health Service and the Hoover Report" Title for Cowling Address

Dr. Donald J. Cowling, Chairman of the Mayo Memorial Commission, has announced that he will speak on the subject, "Health Service and the Hoover Report", at the June 12 dinner of the Minnesota Medical Alumni Association and the Minnesota Medical Foundation.

The dinner will be held at the Spaulding Hotel during the annual session of the Minnesota State Medical Association and will mark the opening of renewed activity for the Medical Alumni Association.

Dr. Cowling, who is Chairman of the Minnesota Committee for the Hoover Report, has a special interest in the subject and is well qualified to speak on the subject of such great interest to the medical profession.

Dr. Frank Elias, President of the Minnesota State Medical Association, will preside at the banquet.

Members of the medical profession and their wives are invited to attend this meeting. Alumni of the Medical School and members of the Minnesota Medical Foundation are especially invited to attend.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

May 21 - May 27, 1950

No. 289

Sunday, May 21

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

10:30 - 11:00 Surgical Conference; Surgery for Carcinoma of the Pancreas; Clarence Dennis; Rm. M-109, U. H.

Monday, May 22

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

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; M-109, U. H.

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

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

11:00 - 11:50 Physical Medicine Seminar; Plastic Surgery in Burns; Bernard G. Lannin; E-101, U. H.

11:00 - 11:50 Roentgenology-Medicine Conference; Veterans Hospital.

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

12:00 - 1:00 Physiology Seminar; Nutritional Requirements for Embryonic Development; Nelson T. Spratt; 214 M. H.

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

12:30 - 1:20 Pathology Seminar; Subject to be announced; 104 I. A.

12:30 - 1:30 Surgery Problem Case Conference; A. A. Zierold, C. Dennis and Staff; Small Classroom, Minneapolis General Hospital.

1:30 - 2:30 Surgery Grand Rounds; A. A. Zierold, C. Dennis and Staff; Minneapolis General Hospital.

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

4:00 - Public Health Seminar; The Health Officer Looks at Health Education; Russell Saxvick, North Dakota Department of Health, Bismarck; 113 Medical Sciences.

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

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

COME! COME! COME! - TO THE MEDICAL SIX-O'CLOCK MIXER AND DINNER - THURSDAY

Monday, May 22 (Cont.)

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staffs; M-109, U. H.

Tuesday, May 23

7:30 - 9:00 Fracture Rounds; General Hospital.

8:30 - 10:20 Surgery Seminar; 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; Eustis Amphitheater, U. H.

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

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

5:00 - 6:00 X-ray Conference; Presentation of Cases; Doctors Nessa and Anderson; Todd Amphitheater, U. H.

Wednesday, May 24

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

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

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

COME! COME! COME! - TO THE MEDICAL SIX-O'CLOCK MIXER AND DINNER - THURSDAY

Wednesday, May 24 (Cont.)

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans and Bernard O'Loughlin; 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; Some Investigations on Intermediary Metabolism with Heavy Carbon; N. Lifson; 113 Medical Sciences.

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

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

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

4:00 - 5:00 Infectious Disease Rounds; Main Conference Room, Bldg. 1, Veterans Hospital.

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

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

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

Thursday, May 25

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; The Terminal Peptides of Insulin; R. M. Fry; 214 M. H.

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

COME! COME! COME! - TO THE MEDICAL SIX-O'CLOCK MIXER AND DINNER - THURSDAY

Thursday, May 25 (Cont.)

*4:00 p.m. - Special Lecture; Review of Chemical and Pharmacological Investigations on Cardiac Glycosides; Arthur Stoll, University of Basle, Switzerland; Medical Science Amphitheater.

4:15 - 5:00 Bacteriology Seminar; Photodynamic Action of Methylene Blue on Vaccinia Virus; Henrick DeKruiff; 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; Portal Venography; Richard Bridenbaugh; Todd Amphitheater, U. H.

*6:00 p.m. - MEDICAL SIX-O'CLOCK MIXER AND DINNER: Main Ballroom, Coffman Memorial Union.

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, May 26

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; Aspiration Biopsy of Bone Marrow; R. Dorothy Sundberg; Powell Hall Amphitheater.

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

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

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

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

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

COME! COME! COME! - TO THE MEDICAL SIX-O'CLOCK MIXER AND DINNER - THURSDAY

Friday, May 26 (Cont.)

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

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

4:30 - 5:30 Journal Club; M-436, U. H.

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

Saturday, May 27

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

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

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

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

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

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

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

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

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

11:00 - 12:00 Anatomy Seminar; An Experimental Approach to the Problem of Functioning of the Fetal Thyroid, Arthur E. Sethre; Vital Staining of Tendon, Richard H. Swigart; 226 I. A.

* 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.

COME! COME! COME! - TO THE MEDICAL SIX-O'CLOCK MIXER AND DINNER - THURSDAY