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# ACTH and Cortisone In Allergy

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# I. ACTH AND CORTISONE IN ALLERGY

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Allergy, in its real implications, is a term used to designate the abnormal altered tissue response of certain people to the stimuli of their environment whether these be physical, chemical or psychic.[1] It is the abnormal reaction of their cells to the world about them. From this point of view it is evident that a great many conditions that we ordinarily do not think of as allergic could be included in that category. It is also evident that certainly to the laity and almost as certainly to the medical profession, the term "allergy" is restricted to that group of altered reactivity diseases that have a marked hereditary tendency, hyperreactivity of smooth muscle, immediate wheeling skin reactions or flaring, production of specific antibodies or reagins, and symptoms of vasomotor origin. It has become increasingly clear in recent years that even in these restricted conditions commonly regarded as allergic where hypersensitivity may play a very striking role, it is the pattern of tissue response that is fundamentally important. The manifestations may be initiated by a wide variety of stresses and stimuli.[2] In the words of Selye "the abnormal adaptive response of the agent is the major cause of the disease".[3] With the development and use of ACTH and cortisone came the hope that these hormones might favorably alter this fundamental, unfavorable biologic response of the allergic person.

Before assaying the role of ACTH and cortisone in the management of allergic problems, a brief resume of our present concepts and orthodox methods of treatment would be indicated. A hypothesis in science, certainly in medicine, need not be 100% correct to be of great help especially in the field of therapeutics.

In the allergic individual, we first must have the so called allergic state --a condition often inherited in which the patient is more likely to develop the symptoms of allergy than do others in exactly the same environment. It is the soil in which allergy in the usual sense can flourish.[4] We have further the capacity in these people to become sensitized so that upon re-exposure to an allergen they respond with the production of antibodies. The union of the antigen and antibody results in the release of a toxic agent--histamine or histamine-like--which in these patients causes a variety of symptoms of vasomotor origin.[5]

TABLE I

| |
|---|
| Allergic state + exposure to an allergen or stimulant = sensitized state |
| Sensitized state + allergen = antibody production |
| Antibody + allergen = toxic product |
| Toxic product = symptoms |

Treatment of allergics, irrespective of the shock organ involved, is based on an accurate diagnosis of a true hypersensitive state and interference with these various hypothetical processes going on in the allergic individual. Everything that itches, wheezes or sneezes is not allergy, not even in the allergic. The ideal approach would, of course, be an attack on the fundamental asthmatic state but unfortunately in allergy, we know as little about that fundamental factor as does the cardiologist about coronary disease, the psychiatrist about mental disorders or the gastro-enterologist about cirrhosis of the liver. We do not know why the guinea pig is easily sensitized while the rat is very resistant. The next method of approach would be prophylaxis of the asthmatic state. While heredity is important in allergy, eugenics is very hard to put in practice and is of very little actual help. A more practical method is to tell people with a background of that type to avoid commonly known sensitizing agents such as pollens, fungi, animals, dust, antigenic injections, and prolonged emotional and physical trauma.

The third method is avoidance of the causative allergens or stimuli which, of course, implies recognition of these etiologic factors by history, physical examination, laboratory procedures, skin tests, and therapeutic trials----the most important being history. This may be accomplished by removal of the patient from the allergens, placing a barrier between the patient and allergen, or removal of the allergen from the patient.

The next method of treatment is by modifying the antibody formation or the allergen-antibody reaction. While nitrogen mustard, X-ray and isotopes[6] have been tried to modify the antibody response, these methods are dangerous and at present, impractical. The most successful therapy at this level has, of course, been by specific hyposensitization. This method apparently acts by production of blocking or thermostable antibodies which interfere with the union of the antigen and the thermolabile antibody on the shock tissue cell.[7] Another theory is that these injected specific antigens stimulate the alarm mechanism with resulting amelioration of symptoms due to ACTH.[3]

The next method of approach in this theoretical concept is to neutralize the histamine or histamine-like substance resulting from the union of antigen and antibody. While, as pointed out by Dragstedt,[8] histamine release is at least a major factor in the causation of allergic symptoms, it is probably not the only factor. It is because of this that Sir Thomas Lewis[9] called the factor "H Substance" and said "I shall speak of an H substance and in using it shall mean any substance or substances liberated by the tissue cells which exert on the minute vessels and nerve endings an influence culminating in the 'triple response'." While the histamine theory is plausible, the antihistamines, as reported here in 1949, although of a great deal of help in some minor allergic disorders, have proven of very little practical help in the more severe complicated conditions and in particular, asthma.

The last method of treatment is symptomatic. These are too numerous to mention and include the parasympathetic depressants, the sympathetic stimulants, the iodides, anti-bariums, etc.

As, by very definition, allergic diseases are diseases of adaptation, resulting from man's reaction to his surroundings. Selye's[3] concept of the general adaptation syndrome, whether entirely correct or not,[11] is of a great deal of help especially in evaluation of the role of ACTH and cortisone.

TABLE II

Allergen ⟶ sensitized reticulo-endothelial system = antibodies

Allergen + Antibody = toxic substance

Toxic substance ⟶ Hypophyses = ACTH

ACTH ⟶ Adrenals = Cortisone and DOCA

The antigen stimulates the reticulo-endothelial system especially the plasma cells to produce antibodies. The resulting toxic agent resulting from the reaction of the antigen and antibody, according to Selye, directly or indirectly stimulates the hypophysis to produce ACTH. ACTH in turn stimulates the adrenals to produce cortisone and DOCA. If the response is too violent or the gluco-corticoids are inadequate, allergic reactions result. As the gluco-corticoids mitigate the response of the shock tissues to stimuli and the mineralo-corticoids increase the responsiveness, the degree of sensitivity would depend on the balance between these two hormones.

As I pointed out in a report on the use of ACTH and cortisone in asthma in 1951,[12,13] it was immediately evident from the effects of ACTH and cortisone, that they would have profound effects in the sensitivity states. This was confirmed before and since then by many reports, especially on acute symptoms and for relatively short periods of time.[14, 15,16,17,18,19,20] Whether as pointed out by White[21] and Sayers,[22] these hormones

act by (1) interference with release of or the toxic action of anaphylactagenic substances produced in the antigen-antibody reaction, (2) alteration in the relative concentration of antigen and antibody in the tissues or factors which influence the combination, (3) alteration in cell permeability through action on hyaluronidase, or (4) alteration in tissue response to the antigen-antibody reaction; it is very evident that early in the course of treatment of allergic individuals with ACTH and cortisone, there are changes of induced hyperactivity of the adrenal cortex. These need not be here re-enumerated. In evaluation of the effect on chronic severe allergic cases here reported, however, we must also and especially consider the euphoria induced, the increased appetite and neuropsychiatric changes.

As would be deduced from the effects and confirmed by the literature,[23] absolute contraindications to the use of ACTH and cortisone are few but they should be used with caution in diabetes mellitus, psychotic disorders or emotional instability, cardiac failure, during major surgery, severe infections, myocardial infarction, pulmonary embolus, cerebral accidents, osteoporosis, tuberculosis, syphilis and peptic ulcers.[12,13] Very important is the question of the effects of long continued administration and large dosage. In general, the response is neither rapid nor long continued. The effects are reversible when the hormone is discontinued. And it is indeed encouraging to know that in Cushing's disease, due to a unilateral tumor of the adrenal cortex, the other adrenal cortex may undergo atrophy, but its function returns even years later after the tumor is removed. Beyond that, it is neither necessary nor desirable to give large doses for a prolonged period.[24] The immediate undesirable clinical effects reported are edema and hypertension, masking of symptoms and signs of infection, decreased localization of infections processes, diabetogenesis, perforation of gastrointestinal ulcerations, precipitation of psychosis, muscle weakness, osteoporosis, hirsutism, loss of head hair, acne, pigmentation, Cushing's syndrome, impaired wound healing, and increased coagulability. The effects are probably due to the undesirable metabolic effects of protein catabolism, $Na^+$ and water retention, $K^+$ loss, derangement of carbohydrate metabolism, Ca and $PO_4^{---}$ depletion and depletion of other essential constituents of protoplasm.[25,26] The effects are usually not marked certainly in the dosage used in allergy and are easily controlled by discontinuance or reduction of the amounts prescribed. Sodium limitation, especially in patients receiving in excess of 10 mg. of ACTH or 50 mg. of cortisone daily, is indicated, while in many receiving large dosage, restriction to less than 300 mg. daily may be necessary. A high protein diet of 120 to 200 gm. daily with adequate calories and high in potassium is logically advised especially in undernourished individuals.[27] Very infrequently diuretics may be indicated. While the weakness noted at times has no constant relation to low serum levels or resulting ECG changes, it has at times been desirable to give potassium chloride especially when the level is below normal or when the hormones are to be given in large doses or a prolonged period of time. Estrone and progesterone or testosterone will frequently prevent the most annoying of the symptomatic side effects in menopausal women where they are most frequent. Above all, suboptimal dosage at the lowest possible level will minimize these undesirable features in the use of ACTH and cortisone.[12]

In the present state of knowledge and expense, these hormones should be used in allergics only where the diagnosis is well established and where the other means of therapy already reviewed do not adequately control the disease. It is by now certainly evident that ACTH and cortisone, whatever the mode of action, are blocking agents and have no specific effect. Beyond that before starting therapy the relative expense, hazards and contra-indications must be taken into account not only in short term but also in the probably necessary long term use of these agents.

With all these thoughts in mind and to minimize the dangers involved, the fol-

lowing procedures in Table III were carried out in all patients before therapy was started.

TABLE III
BEFORE THERAPY

1. Routine urinalysis
2. Blood, Hgb., Wbc., differential
3. Erythrocyte sedimentation rate
4. Blood N.P.N. or urea when indicated
5. ECG if indicated especially when over age 45
6. Thorn test with ACTH
7. Weight
8. Blood pressure
9. X-ray

During therapy the procedures in Table IV were done.

TABLE IV
DURING THERAPY

1. Urinalysis (at least weekly)
2. Blood sugar estimation (if patient is glycosuric)
3. Glucose tolerance test (if patient is hyperglycemic)
4. Erythrocyte sedimentation rate at weekly intervals (optional)
5. ECG to detect K depletion
6. Blood pressure
7. Weight

As I reported in 1951[13] when cortisone became more available and less expensive, the opportunity presented itself to give this hormone to ambulatory patients with severe, chronic, perennial asthma. All the usual specific and non-specific measures had been tried with little or no or very temporary effect and a progressively downward course in these semi invalid or invalid people. The initial dose was 200 mg. (100 mg. intramuscularly in each buttock). Thereafter the dosage was 100 mg. daily for six days. In the second and third week the dosage was reduced to 100 mg. three times a week. Here we discontinued treatment to see if we could get a remission. If none was obtained or if symptoms recurred gradually, treatment was again started. The dosage depended upon the response of the individual patient. No attempt was made to have the patient completely free of symptoms as long as he was comfortable. After obtaining a satisfactory effect, cortisone was given by mouth in equivalent dosage and with apparently approximately equivalent results. Cortisone acetate in the same form supplied for injection was mixed in milk or fruit juice until the drug was available in tablet form. As the effects of the orally given hormone is dissipated in 12 to 14 hours, it was prescribed in two or three divided doses.

TABLE V

| | |
|---|---|
| Number of patients | 30 |
| Ages | 15 to 62 |
| Males | 22 |
| Females | 8 |
| Duration of asthma | 3 to 32 yrs. |
| Diabetics | 2 |

Thirty patients with allergic asthma chosen for their extreme severity, chronicity and failure on the normal allergic regime were given cortisone in the manner described and under the above precautions and conditions. No patients with usual contra-indications were treated but for two asthmatics with mild diabetes mellitus. The ages varied from 15 to 62. Twenty-two were men and eight women. The duration of the disease varied from three to thrity-two years. The apparent causation of the allergy ran the gamut of allergens. Psychic factors were present as in all asthmatics to a greater or less degree.

TABLE VI

| RESULTS | |
|---|---|
| good | 22 |
| fair | 8 |
| poor | 0 |

The initial results in these cases, as I reported, were to me in the beginning really startling. Within a day to a week there was a marked diminution in wheezing with a feeling of well-being and energy. The cough lessened and in many cases almost disappeared by the second or third week. As the dosage was decreased, the euphoria and enthusiasm was decreased but the over all result was still very satisfactory. Results were evaluated according to the patient's own description and judgment and the over all picture including objective findings. Those who experienced 50% relief or more were considered to have fair results. Patients with mild or practically no symptoms were considered to have good results. By this criteria the initial results of treatment were good in twenty-two and fair in eight. In the eight that had only fair results there was more or less persistent cough but no frank severe asthmatic attacks---a real achievement in this type of case.

TABLE VII

| SIDE EFFECTS | |
|---|---|
| Increased appetite | 24 |
| Euphoria | 18 |
| Acne | 1 |
| Edema (transient) | 3 |
| Glycosuria | 2 |
| Furunculosis | 1 |
| Change of shock tissue | 1 |

The early side effects in this series of patients were not many and not serious. Increased appetite was noted in twenty-four though this had a tendency to decrease with decrease in dosage. Euphoria, mild and quite pleasant, was noted in 18. This also decreased after the first few weeks. Severe acne was noted in one which was controlled by penicillin. Transient edema was mild to moderate in three, but no medication was required beyond a reduction in sodium intake with a later reduction in the dose of cortisone. The only cases of glycosuria were in the two diabetics in whom insulin requirements were more than doubled during the first three weeks. When dosage was decreased, the insulin requirements returned to pretreatment levels. One case of furunculosis developed but subsided with use of penicillin. One case had a change of shock tissue and developed a severe urticaria which finally also responded to cortisone.

After four months treatment, one woman, age forty-four, developed emotional disturbances with a moderate degree of depression. One other woman, age fifty-two, developed the same symptoms after three months of therapy. Cortisone was discontinued despite improvement in asthma of a marked degree in both. These two cases called attention to the reported possible mental effects of cortisone.[28] One male, age thirty-eight, developed a definite blurring of vision to the extent that here also therapy was interrupted though no objective findings were noted on ophthalmoscopic examination. This, to me, was of great interest in view of reported increased coagulability after cortisone and ACTH.[25,27] Knisely and Black[29,30] studied the peripheral circulation of the bulbar conjuctiva of human beings using reflected light and a binocular stereoscopic microscope. With similar technique Burrage[17] observed the vessels and circulating blood of the bulbar conjuctiva of six patients before cortisone and at weekly intervals during therapy. He noted clumping of cellular elements of the blood which never cleared during therapy and at times these clumps seemed to plug the ends of small arterioles intermittently.

An attempt was made at the end of three weeks to stop treatment to see if a remission could be obtained. If none occurred dosage was maintained to give adequate relief. The dosage varied from 12.5 mg. to 75 mg. daily and varied in different patients and in the same patient at different times.

At the end of two years of experience with oral cortisone the picture is not quite as impressive as it was at the beginning though still in the type of patient treated significant. In evaluating

TABLE VIII

STATUS

| | | |
|---|---|---|
| Remission (with specific therapy) | | 8 |
| Stopped therapy after asthma improvement | | 6 |
| cannot afford it | 3 | |
| depression | 2 | |
| blurring of vision | 1 | |
| Improved on Cortisone (plus specific therapy) (50 to 75 mg. daily) | | 2 |
| Improved on intermittent course of Cortisone | | 3 |
| Improved with Cortisone plus ACTH (gel) (plus specific therapy) | | 8 |
| Relapse with ACTH plus Cortisone plus specific therapy | | 3 |

this result it is well to recall that many asthmatics have prolonged remissions for no apparent reason. I must also emphasize that more specific treatment was also searched for, instituted, or maintained at the same time. It also must be stressed, however, that these were desperate cases that had not previously responded at all to usual therapy.

We obtained a remission in eight patients with no apparent relation to total dosage or duration of treatment. I wish to call particular attention to the three asthmatics who stopped treatment in spite of apparent improvement because they could not afford it. It is certainly no medical triumph to add a flat pocketbook to an unimproved asthma. The expense involved not only of the hormones that have and still are becoming less costly but of constant medical supervision must be explained before starting therapy. Two patients are satisfactorily controlled with 50 to 75 mg. of cortisone daily when other allergic measures are also adhered to. Three who could not afford continuous therapy and supervision and obtained more or less adequate remission for a period of three weeks to six months with a two to three week course of cortisone were given intermittent treatment with marked exacerbation of symptoms. Here, the usual procedure of initial large doses orally of 200 to 300 mg. in divided doses daily for two or three doses was given and then the dosage gradually reduced over a period of two to three weeks.

Eight patients did not get satisfactory improvement on the regime of cortisone alone and on maintainance doses of up to 75 mg. daily. Increased amounts gave grave side effects of increased hyper-adreno-corticism such as moon face, increased blood pressure, marked edema or increased weight in spite of the above mentioned precautions and procedures. In these eight, ACTH long acting (gel) form was given in addition to cortisone with the idea that by giving both cortisone and ACTH we might get the effect desired with smaller total dosage. We might also avoid the atrophy of the adrenals induced by cortisone or the hypertrophy of ACTH. While hypocorticoidism may normally be the most important stimulant for release of ACTH, it is not the only factor as ACTH production in stress is compatible with a high blood level of corticoids.[31]

Cortisone orally was maintained at 50 mg. daily. A dose of 40 to 60 mg. of ACTH gel intramuscularly was given the first day and daily doses thereafter in decreasing amounts until a satisfactory maintainance dose was attained. The dosage of both were varied to obtain the best clinical effect with smallest possible total amount. This varied in different patients and in the same patient at different times from 50 mg. of cortisone plus 10 mg. of ACTH daily to 12.5 mg. of cortisone daily and 5 mg. of ACTH every other day. The patients were taught to give their own injections as are diabetics in giving insulin. Attempts to stop this treatment were made at intervals but to the present with no success in these patients. The dosage is constantly varied with the clinical state. While it is difficult to determine definitely that the smaller dosage of the hormones are specifically beneficial, prolonging periods between therapy resulted in gradual exacerbation of symptoms. Further attempts to discontinue cortisone resulted in exacerbation of symptoms unless ACTH was increased in disproportionately higher dosage.

## HAY FEVER

Because of the usually dramatic but usually short duration and purely blocking effects of cortisone in allergic manifestations, it was long felt, as I intimated in my first report in 1951,[13] that a self limited allergy would respond ideally to cortisone. As the symptoms of hay fever are usually limited to the duration of the pollinating season, after which the antigen is apparently no longer active, the use of the hormone could also be used for a short period of time. Haddon Carryer[32] initially treated three patients with hay fever and asthma with cortisone and reported relief of the symptoms of hay fever though less rapid than those of the asthma. Gelfand[33] treated two patients with severe ragweed hay fever with 50 mg. of cortisone on the days of high pollen counts with marked improvement in symptoms. Schwartz et al.[34] reports on the treatment of 25 patients with ragweed pollenosis with hyposensitization and added oral cortisone with good results in twenty-one. Hyposensitization was continued due to the feeling that it was a more specific and safe method of treatment and had more lasting benefit especially as regards asthma. Others have reported on the effects of ACTH and cortisone in hay fever.[35,36] As pointed out by McCombs[37] "exactly what place pollen hyposensitization will have in the management of seasonal bronchial asthma when hormone therapy has been thoroughly evaluated cannot be ascertained as yet". While a great majority of patients obtain very satisfactory results at present, by pre-seasonal or perennial specific injection therapy, the considerable number that do not should logically be given a trial on hormone therapy.

A total of thirty-six patients with severe ragweed hay fever were treated under the same restrictions as the cases of asthma with same indications, contra indications and precautions. The ages varied from 18 to 56; twelve males and twenty-four females; the period of previous hyposensitization varied from two to fifteen years and the duration of symptoms from two to thirty years. In spite of previous treatments with varying degree of relief especially of the asthma, these people still had marked difficulty in spite of all available means. With start of symptoms, cortisone therapy was begun with 50 mg. orally four times a day for one day, then 25 mg. four times a day until relief was obtained. The dosage was then gradually reduced to a maintainance dose of 12.5 to 75 mg. daily usually 50 to 75 mg. a day depending on degree of symptoms. No serious side effects were noted beyond occasional nausea, headache or abdominal fullness. Relief was obtained in one to ninety-six hours after treatment was started. Symptoms recurred in a brief period of a few hours to a few days with discontinuance of cortisone. Good results were obtained in ten, excellent results in twenty-two. Four patients had no marked effect from the dosage used.

TABLE IX

ORAL CORTISONE WITH HYPOSENSITIZATION IN HAY FEVER

| | |
|---|---|
| No. of patients | 36 |
| Ages | 18 to 56 |
| Males | 12 |
| Females | 24 |
| Previous treatment by hyposensitization | 2 to 15 years |
| Duration of Symptoms | 2 to 30 years |
| Good Results | 10 |
| Excellent Results | 22 |
| Poor Results | 4 |

In eight hay fever patients with definite pollen sensitivity by clinical history and skin tests, in a condition where correlation is usually excellent, reactivity as noted by scratch or intradermal skin tests persisted with no change even after prolonged treatment. The effect must be beyond the antigen antibody stage. This observation, confirming the results of Loveless and others[1,38,39] indicate, of course, that the fundamental therapeutic effect does not affect the mechanism of sensitization but may change temporarily the underlying pattern of tissue response. It

emphasized also the need for continued study in these patients of the stimuli that call forth the abnormal response.

## COMMENT AND SUMMARY

ACTH and cortisone in my opinion represent an important advance in the treatment of severe allergic states. While satisfactory symptomatic relief is almost the rule at the start, the real problem is maintainance of relief after the initial period with no serious effects. While I have kept two patients with severe asthma on cortisone and eight asthmatics on cortisone and ACTH (gel) for two years almost continuously with no apparent serious effect, it must be emphasized that they have all been kept under constant and careful supervision with continuous attempts to stop medication or find a more specific therapy. Moreover relatively small doses appear to be effective as compared with that required in most other serious conditions requiring hormone therapy.[33, 36,37] Severe hay fever patients treated with cortisone in addition to hyposensitization had worthwhile relief under similar conditions and precautions.

In a disease so frustrating to patient and as frequently to the doctor as perennial, intractable asthma or even severe unrelieved hay fever with their devastating effect on social and economic life, even the reassurance that ACTH and cortisone will relieve the more acute episodes of the conditions is very comforting. Contrary, the let-down after cessation of treatment, if mandatory, may be even more disappointing and devastating to some if no specific cause has been discovered meanwhile. By attention to more specific factors of usual allergic management coupled with the usual precautions, and on a regime of diet and medication as outlined, a considerable proportion of these unfortunates can be made more comfortable and returned to a useful place in society. Certainly, as pointed out by Kinsell,[27] the reaction of the profession to these hormones has swung from incredulity, to extreme euphoria then to the extreme alarm reaction. With the phase of maturity and stability we must now recognize that while no substitute for usual allergic regime and with all the dangers involved, and they are very real, under proper care and precautions, with a true understanding of their lack of specificity in the field of allergy, at least many patients are now productively active who had been in a semi-invalid or fully invalid state before the use of ACTH and cortisone.

## REFERENCES


1. Hansel, French K.
   Clinical allergy
   St. Louis, 1953. C. V. Mosby Co.

2. Feinberg, Samuel, M.
   Allergy in Practice
   Chicago Year Book Publishers. 1944

3. Selye, H.
   The Physiology and Pathology of Exposure to Stress.
   Montreal, Canada. Atta., Inc. 1950.

4. Rackemann, Francis M.
   Allergy.
   Arch. Int. Med. 77:6, 1946.

5. Tuft, L.
   Clinical Allergy.
   Phila. 1949 Lea & Febiger.

6. Waldbott, Geo. L.
   Nitrogen Mustard in the Treatment of Bronchial Asthma.
   Annals of Allergy 10:4, 1952.

7. Loveless, M. H.
   Immunological Studies of Pollinosis.
   J. Immunol. 38:25, 1940.

8. Dragstedt, C. A.
   The Significance of Histamine in Anaphylaxis and Allergy.
   Quat. Bul. N. W. Univ. Med. School 17:102, 1943.

9. Lewis, T.
   The Blood Vessels of the Human Skin and Their Responses.
   London. Shaw & Sons, Ltd. 1927.

10. Blumenthal, J. S.
The Antihistamine Drugs in the Treatment of Hay Fever in the Adult.
J. Lancet Vol. LXIX, No. 6 Page 215, June 1949.

11. Decourt, P.
Theory of "General Adaptation Syndrome"; Its unreality, Description and Refutation of Selyes' Concept.
Presse Medicale 60:1021 1952.

12. Blumenthal, J. S.
Pituitary Adrenocorticotropic Hormone (ACTH) in Asthma.
Minnesota Med. 33:797-798 1950.

13. Blumenthal, J. S.
Cortisone in Allergic Asthma.
J. Lancet, Vol. LXXI, No. 11, Page 473. Nov. 1951.

14. Segal, M. S. and Herschfus, A. J.
ACTH and Cortisone in the Management of the Hypersensitivities.
Ann. of Allergy 8:6, 1950.

15. Feinberg, S. M., Dannenberg, T. B., and Malkul, S.
ACTH and Cortisone in Allergic Manifestations of Allergy.
22:195, 1951.

16. Schwartz, E.
Oral Cortisone in Intractable Bronchial Asthma.
J. Allergy 22:1, 1951.

17. Burrage, W. S., and Irwin, J. W.
Maintainance Cortisone in Severe Bronchial Asthma.
J. Allergy 23:310, 1952.

18. Baldwin, H. S. and de Gara, P. F.
Hospital and Ambulatory Treatment of Asthma and Eczema with ACTH and Cortisone.
J. Allergy 23:15, 1952.

19. Arbesman, C. E., Schneider, M., Greene, D, and Osgood, H.
Intravenous ACTH and Oral Cortisone in the Treatment of Bronchial Asthma.
J. Allergy 23: 293, 1952.

20. Brown, E. A., Fox, L. A., Mobili, C., Norman, P. P., Norton, R. C., and Ruby, S.
The Use of ACTH in the Treatment of Ambulatory Asthmatic Patients.
Ann. Allergy 9:459, 1951.

21. White, A.
Role of the Adrenal Cortex in Immunity.
J. Allergy 21:273, 1950.

22. Sayers, G.
The Adrenal Cortex and Homeostasis.
Physiol. Rev. 30:241, 1950.

23. Sprague, R. G., Power, M. H., Mason, A. L.
Physiologic Effects of Cortisone and ACTH in Man.
J.A.M.A. 144:16, 1950.

24. Slocumb, C. H., Palley, H. F., Hench, P. S., and Kendall, E. C.
Effects of Cortisone and ACTH on Patients with Rheumatoid Arthritis.
Proc. Staff Meet. Mayo Clin. 25: 476, 1950.

25. Kinsell, L. W.
The Clinical Application of Pituitary adrenocorticotropic and Adrenal Steroid Hormones.
Ann. Int. Med. 35:615, 1951.

26. Blumenthal, J. S.
Cortisone in Allergic Migraine.
Minn. Med. Vol. 35 Pages 209-212, March, 1952.

27. Boling, Lenore, and Marger, Sheldon
Dietary Modification of the Metabolic and Clinical Effects of ACTH and Cortisone.
Annals of Int. Med. 37:5, 1952.

28. Clark, L. D., Bauer, W., and Cobb, S.
Preliminary Observations on Mental Disturbances Occurring in Patients Under Therapy with Cortisone and ACTH.
New Eng. J. Med. 246, 1952.

29. Knisely, M. H., and Bloch, E. H.
Demonstration of the Methods and Apparatus Currently Used in Making Microscopic Study of the Circulating Blood and Local Vessels of the Bulbar Conjunctiva of Living Unanasthelized Human Beings.
Anat. Rec. 100:768, 1948.

30. Knisely, M. H., Bloch, E. H., Eliot, T. S., and Warner, L.
Sludged Blood.
Science 106:431, 1947.

31. Fortier, C.
Limitation of the ACTH Regulating Effect of Corticoids.
Ann. J. Physiol. 165:466, 1951.

32. Carryer, H. M., Koelsche, G. A., Prickman, L. E., Maytum, C. K., Lake, C. F., and Williams, H. L.
Effects of Cortisone on Bronchial Asthma and Hay Fever Occurring in Subjects Sensitive to Ragweed Pollen.
Proc. St. Mayo Clinic 25:17, 1950.

33. Gelfand, M. L.
The Use of Small Maintainance Doses of Cortisone in the Treatment of Bronchial Asthma and Hay Fever.
Program 7th Annual Meet. Amer. Ac. of Allergy. N.Y.C. Feb., 1951.

34. Schwartz, E., Levin, L., Leibowitz, H., Reicher, J., Kelly, J., Wallman, M., and Feinblott, T.
Oral Cortisone Therapy in Ragweed Hay Fever.
J. Allergy 23:1, 1952.

35. Rappaport, B. Z.
ACTH in Ragweed Pollinosis.
J. Allergy 22:304, 1951.

36. Stier, R. A., Feinberg, S. M., Malkiel, S. M., Werle, M.
Cortisone in the Treatment of Patients with Ragweed Pollinosis.
J. Allergy 23:395, 1952.

37. McCombs, R. P.
Serial Courses of Corticotrophin or Cortisone on Chronic Bronchial Asthma.
New Eng. J. Med. 247:1, 1952.

38. Loveless, Mary
The Influence of ACTH in Sensitizing and Immunizing Antibodies of Inhalant Allergy.
Bull. N.Y. Ac. Med. 27:8, 1952.

39. Grob, D. Winkenwerder, W. L., Schoenrich, E. H.
The Effect of ACTH Administration on Skin, Conjunctivae and Nasal Mucous Membranes.
Bull. J. Hopkins Hosp. 90:301, 1952.

## II. MEDICAL SCHOOL NEWS

### Coming Events

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|---|---|
| March 2-4 | Continuation Course in Clinical Dietetics |
| March 9 | Special Lecture; "Studies of Hepatic Structure and Function", Dr. Allan L. Grafflin, Professor of Anatomy, Johns Hopkins University School of Medicine, Baltimore; Todd Amphitheater; 4:00 p.m. |
| March 26 | Special Lecture; "Trace Elements in Biochemistry and Medicine"; Dr. Burt L. Vallee; Peter Bent Brigham Hospital, Boston, Massachusetts; Owre Amphitheater; 4:00 p.m. |
| April 6-11 | Continuation Course in Proctology for General Physicians |
| April 16-18 | Continuation Course in Gynecology for Specialists |

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### Faculty News

Dr. Maurice Visscher, Professor and Head of the Department of Physiology, was elected Vice-President of the National Society for Medical Research at its annual meeting which was held in Chicago on February 8.

Several members of the Department of Surgery attended the recent meeting of the Society of University Surgeons which was held in St. Louis on February 12-14. Dr. C. Walton Lillehei, Associate Professor, and Dr. Richard L. Varco, Professor, presented a paper entitled, "Certain Physiologic, Pathological, and Surgical Features of Complete Transposition of the Great Vessels." Dr. Ivan D. Baronofsky reported on some work he has done in conjunction with Doctors J. F. Haddy, C. W. Borden, and A. L. Ferrin, "Cardiopulmonary Dynamics of Mitral Stenosis: A Clinical and Experimental Study.".

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### Publications of the Medical School Faculty

This issue of the Bulletin marks the introduction of a new feature, Publications of the Medical School Faculty. The Editor wishes to extend thanks to all of the members of the faculty who have sent either reprints or notifications of articles published. The initial response was very gratifying, and it is hoped that members of the faculty will continue to keep the Editor informed of publications. Today's listing covers only a small portion of the publications received to date. This listing will appear in the Bulletin at weekly intervals until it is up-to-date and at monthly intervals thereafter.

Baronofsky, I. D., Ferrin, A. L., and Adams, W. L.: Evaluation of Experimental Methods of Producing Functional Mitral Stenosis. Surgical Forum, Clinical Congress of the American College of Surgeons, 1951. Saunders, Phila., 1952, p. 206.

Baronofsky, I. D. and Varco, R. L.: Surgical Problems in Rheumatic Valvular Heart Disease. Rheumatic Fever, A Symposium. University of Minnesota Press, Minneapolis, 1952, p. 249.

Bittner, J. J.: The Genesis of Breast Cancer in Mice. Texas Reports on Biology and Medicine, 10: 160, 1952.

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Bittner, J. J.: Transfer of the Agent for Mammary Cancer in Mice by the Male. Cancer Research, 12: 387, 1952.

Bittner, J. J.: Tumor-inducing Properties of the Mammary Tumor Agent in Young and Adult Mice. Cancer Research, 12: 510, 1952.

Bittner, J. J.: Studies on the Inherited Susceptibility and Inherited Hormonal Influence in the Genesis of Mammary Cancer in Mice. Cancer Research, 12: 594, 1952.

Bosch, H. M.: Report on a Visit to Yugoslavia; W.H.O. Report, MH./59.52

Bosch, H. M.: Report on a Visit to Finland; W.H.O. Report, MH./69.52

Downey, H.: The Megaloblast-Normoblast Problem: A Cytologic Study. J. Lab. & Clin. Med., 39: 837, 1952.

Gliek, D. and Ochs, M. J.: Mucolytic Enzyme Systems XIX. Comparison of Hyaluronidase Inhibitor and Heparin Levels in Serum. Proc. Soc. Exp. Biol. Med., 81: 363, 1952.

Hilding, A. C.: Studies on the Otic Labyrinth I. On the Origin and Insertion of the Tectorial Membrane. Ann. Oto., Rhin., and Laryng., 61: 354, 1952.

Hilding, A. C.: Studies on the Otic Labyrinth II. A Theory on the Stimulation of the Organ of Corti by Sound Vibrations. Ibid. 61: 371, 1952.

Johnson, F. E. and Boyden, E. A.: The Effect of Double Vagotomy on the Motor Activity of the Human Gall Bladder. Surg., 32: 591, 1952.

Larsell, O.: The Morphogenesis and Adult Pattern of the Lobules and Fissures of the Cerebellum of the White Rat. J. Comp. Neur., 97: 281, 1952.

Madden, J. F.: Therapy of Leukoplakia Buccalis. Current Therapy, W. B. Saunders, Phila., 1952.

Madden, J. F. and Ravits, H. G.: Enzyme Debridement of Indolent Infected Cutaneous Ulcers. J.A.M.A., 149: 1616, 1952.

Michelson, H. E.: Inflammatory Nodose Lesions of the Lower Leg. A.M.A. Arch. Derm. & Syph., 66: 327, 1952.

Miller, F. A., Brown, E. B., Buckley, J. J., Van Bergen, F. H., and Varco, R. L.: Respiratory Acidosis: Its Relationship to Cardiac Function and Other Physiologic Mechanisms. Surg., 32: 171, 1952.

Myers, J. A.: The Ever-Continuing Search for Immunity in Tuberculosis. Post. Med., 12: Nos. 2, 3, & 5 (Aug., Sept., & Nov.), 1952.

Peyton, W. T., Moore, G. E., French, L. A., and Chou, S. N.: Localization of Intracranial Lesions by Radioactive Isotopes. J. Neurosurg. 9: 432, 1952.

Rice, C. O.: The Evaluation of Compensable Disability. Minn. Med., 35: 740, 1952.

Rice, C. O.: The Calculation of Industrial Disabilities of the Extremities. Charles Thomas, 1952.

Rice, C. O. and Strickler, J. H.: Parenteral Nutrition in Elderly Surgical Patients. Geriatrics, 7: 232, 1952.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

March 2 - 7, 1953

Monday, March 2

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.

11:30 - 12:30 Physical Medicine Staff Seminar; Venous Pressure in the Lower Extremities; Sarah Gault; Heart Hospital Auditorium.

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

12:30 - 1:30 Physiology Seminar; Relation of Inherited Hormonal Mechanism to the Development of Breast Cancer; John J. Bittner; 214 Millard Hall.

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

4:00 - Pediatric Seminar; Neurologic Complications in Infectious Mononucleosis; Wilmer Pew; Sixth Floor West, U. H.

4:00 - 5:30 Seminar on Fluid and Electrolyte Balance; Gerald T. Evans; Todd Amphitheater, U. H.

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

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

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.

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 2 (Cont.)

Ancker Hospital

8:30 - 10:00 Chest Disease Conference.

1:00 - 2:00 Medical Grand Rounds.

Veterans Administration Hospital

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

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

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

2:00 - Psychosomatic Rounds; Bldg. 5.

Tuesday, March 3

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.

5:00 - 6:00 X-ray Conference; Presentation of Cases from Mt. Sinai Hospital; Drs. Friedman and Zheutlin; Eustis Amphitheater, U. H.

Ancker Hospital

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

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

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

Minneapolis General Hospital

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

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.

Tuesday, March 3 (Cont.)

Veterans Administration Hospital

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

8:30 - Infectious Disease Rounds; Dr. Hall.

8:30 - Surgery Staff Seminar; Aneurysms of Aorta; George Werner; Medical Conference Room, Bldg. I.

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

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

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

1:00 - 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 Autopsy Conference; E. T. Bell and Donald Gleason; Conference Room, Bldg. I.

Wednesday, March 4

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 Staff; Todd Amphitheater, U. H.

12:30 - 1:30 Radioisotope Seminar; In Vivo Synthesis of Porphyrins and Hemes; S. Schwartz; 12 Owre Hall.

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

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

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

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

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

Ancker Hospital

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

2:00 - 4:00 Medical Ward Rounds;

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

Wednesday, March 4 (Cont.)

Minneapolis General Hospital

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

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

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

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

12:00 - Surgery Seminar; Dr. Zierold; Classroom.

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.

2:30 - 4:00 Psychosomatic Rounds; C. K. Aldrich; Conference Room, Bldg. I.

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

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

Thursday, March 5

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.

12:30 - Physiological Chemistry Seminar; Mode of Action of Antibacterial Drugs; James Jarvis; 214 Millard Hall.

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

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.

5:00 - 6:00 Radiology Seminar; Treatment of Lymph Node Tumors; Charles M. Nice; Eustis Amphitheater, U. H.

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

Ancker Hospital

4:00 - Medical-Pathological Conference; Auditorium.

## Thursday, March 5 (Cont.)

Minneapolis General Hospital

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

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

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

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.

## Friday, March 6

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; Aortography; William E. Price; 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.

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

## Friday, March 6 (Cont.)

Minneapolis General Hospital (Cont.)

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

## Saturday, March 7

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.

11:30 - Anatomy Seminar; The Effects of Testosterone upon the Primate Testis; David Seibel; 226 Institute of Anatomy.

Ancker Hospital

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

Minneapolis General Hospital

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

Veterans Administration Hospital

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

8:30 - 11:15 Hematology Rounds; Drs. Hagen, Goldish, and Aufderheide.
11:15 - 12:00 Morphology . . . . . Dr. Aufderheide.