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Work of the Heart

Staphylococcal Products

Breast Cancer

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Staff Meeting Report

Studies on the Work of the Heart in a Hospital Environment*

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For the past 10 years this laboratory has been studying the muscular work and cardiac work required by: 1) activities performed by patients in the hospital, and 2) self-care activities that the cardiac patients may be expected to do at home. The cost of muscular work may be estimated by measuring the oxygen consumption during a period of activity. For an activity requiring constant muscular work over a prolonged period, at a level which does not exceed the ability of the cardiovascular system to supply the bodily needs, the average oxygen consumption for the period may be an adequate representation of the metabolism. But if the amount of muscular work fluctuates periodically or irregularly, the oxygen consumption will change in like manner. The more irregular the activity, the greater the variability in oxygen requirement. Whether or not this variability will be discerned depends upon the response time of the method of study used. If the oxygen intake is recorded continually and with sufficient rapidity, fluctuations will become apparent.

F. J. KOTTKE

METHODS

The supply of oxygen during work is governed by: (1) the respiratory oxygen intake; (2) the amount of oxygen extracted

*This report was given at the Staff Meeting of the University Hospitals on November 4, 1960.

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from the blood, or the arteriovenous oxygen difference; and (3) the oxygen debt. The ultimate supply of oxygen for metabolism depends upon the intake of oxygen through the respiratory system. Measurement of oxygen intake is an accurate method of assessing metabolic energy when the body is in a steady state of equilibrium. During short bouts of activity, however, the respiratory oxygen intake may be an unreliable index of the fluctuations in the use of energy by the muscles or the heart. The product of the arteriovenous oxygen difference and the volume of blood which flows to a working organ determines the oxygen supplied for metabolism. If one measures the oxygen difference between arterial blood and the mixed venous blood of the heart, the cardiac output can be estimated. Autonomic regulation of the vascular system influences the arteriovenous oxygen difference, which is found to fluctuate rather widely in different vascular beds even under apparently constant conditions. At the beginning of the work or during heavy work, the circulation is not increased rapidly enough to supply the demand for oxygen. This gives rise to an oxygen debt, which may be paid back as the work continues or in the rest period following work. In any attempt to estimate the work of the heart on the basis of the oxygen consumption, therefore, one must also consider the arteriovenous oxygen difference and the oxygen debt.

In these studies oxygen consumption has been estimated from analysis of the expired air. Through physical methods of gas analysis and electronic methods of computing and recording, respiratory gases were analyzed and recorded continuously with a time lag of only a few seconds. A Beckman paramagnetic oxygen analyzer provided a continual record of the concentration of oxygen in the expired air. The concentration of carbon dioxide in the expired air was measured by means of a Beckman infrared carbon dioxide analyzer. The volume of expired air was recorded electronically from a small direct-current generator activated by motion of the spirometer in which the expired air was collected. Repeated analysis has shown the oxygen content of the air in the laboratory to be 20.93 volumes per cent. Oxygen consumption during activity was calculated by means of an electronic computer from the difference in concentration of oxygen in the inspired air and in the expired air; this figure was corrected for carbon dioxide content, and multiplied by the respiratory volume. This method is rapid enough to record variations in oxygen consumption of only a few seconds' duration.

Cardiac function was monitored in a number of ways: Heart

rate was recorded continuously with a cardiotachometer which was triggered by the R-wave of the electrocardiogram to record the interval between R-waves as the heart rate per minute. Fluctuations in rate due to respiration, to changes in posture, or to work were clearly apparent. Electrocardiograms were recorded using two chest leads and a ground lead attached to the forehead. The first chest electrode was placed over the left second costosternal junction, and the second electrode was fastened over the fifth interspace in the left midclavicular line. This positioning minimizes muscular potentials during activity and produces an electrocardiographic tracing that is similar to a V_4 record.

Cardiac output cannot be measured accurately by methods that are simple enough to permit their routine clinical use. In the laboratory, cardiac output may be measured by the direct Fick method following cardiac catheterization, or by methods based on dye-dilution or foreign gas uptake.[1] All three methods give similar results.[2] Although there has been criticism[3] of the acetylene method[4] of estimating arteriovenous oxygen difference, studies by Asmussen and Nielsen[1,2] and by Chapman[5] show a high degree of correlation among the methods. Chapman found the acetylene method had a systematic error giving results approximately 24 per cent lower than these obtained by the direct Fick method. This difference was attributed to: an incorrect solubility factor for acetylene in blood, the recirculation of acetylene, and the increased oxygenation of the arterial blood during rebreathing. In work experiments with cardiac output values ranging as high as 22 liters per minute, the acetylene and dye-dilution methods were found to give identical results with smaller percentage standard deviation during work than at rest.[1] Both the direct Fick method and the acetylene method of Grollman are based on the Fick formula:

$$\text{Cardiac Output, liters per minute} = \frac{\text{oxygen consumption, cc. per minute}}{(O_2)_A - (O_2)_V, \text{ cc. per liter}}$$

Grollman[4] in 1932 reported a method of estimating arteriovenous oxygen difference from the rate of absorption of acetylene from a mixture of air, oxygen, and acetylene by the following formula:

$$(O_2)_A - (O_2)_V = \frac{(O_2)_{diff.} \times (C_2H_2)_{av.} \times (B - 48.1) \times 00921}{(C_2H_2)_{diff.}}$$

When the oxygen consumption had been measured during activity just prior to the estimate of arteriovenous oxygen difference,

cardiac output could be calculated. This method does not require an arterial or a venous puncture nor the use of a cardiac catheter; moreover, it allows the patient to carry on activity with no inherent danger. It entails, however, the disadvantage common to all methods of measuring oxygen consumption: The patient must wear some type of face mask and is restricted in his activities by breathing tubes and a mouthpiece, which may modify respiration or interfere with vision and head motion during activity. Moreover, the method is valid only if acetylene-free blood is completely saturated with acetylene as it passes through the lungs. An interval of 30 minutes must be allowed between determinations of arteriovenous oxygen difference by the acetylene method in order to allow for expiration of all acetylene which has been absorbed into the blood. For this reason, rapidly repeated determinations of cardiac output cannot be made, and thus one cannot obtain the kind of information about cardiac output or cardiac work that one can now obtain regarding metabolic work. The method is relatively simple to use, however, and it appears to give data similar to those yielded by the more complicated catheterization or dye methods, which restrict the subject in the activities we wish to study.

In this acetylene method, the patient rebreathes from a five liter anesthesia bag containing a mixture of oxygen, nitrogen, and acetylene. Gas samples are collected in glass vacuum tonometers at the end of the fourth and seventh breaths. The rebreathing time is 15 seconds or less. The blood pressure has been recorded during experiments by inserting a nylon catheter into a brachial artery and connecting the catheter to a Model P23D Statham strain gauge. The strain gauge is mounted on the chest so as to insure minimal change in the relationship between the strain gauge and the heart as the patient moves. Either the undamped pulse record or the mean blood pressure can be recorded continuously during the experiment. To prevent clotting, a constant injection pump injects a solution of heparin (20 mg./100 ml.) through the catheter at the rate of .07 cc. per minute.

Work of the left ventricle is estimated using the formula:

$$W = QR + \frac{wV^2}{2g}$$

Q=cardiac output in cc. per ventricular contraction
R=resistance (mean blood pressure)
w=weight of the blood pumped
V=velocity of ejected blood

FACTORS IN CARDIAC OUTPUT AND MYOCARDIAL WORK

The stresses engendered by various activities in the hospital and during rehabilitation have been studied in order that patients with cardiac diseases may be advised about the types of activities most likely to cause cardiac stress and possibly discomfort or injury. Factors influencing the demand on the myocardium during normal activity are: 1) posture, 2) muscular work, and 3) emotional influences.

Posture

The influence of body position on cardiac output and myocardial work is especially significant when minimal cardiac Output is desired. These effects of posture on cardiac output have been reported and denied as significant for many years.[6] Levine and Lown[7] have recommended that protecting the posture of the patient with myocardial disease be made part of clinical treatment, but this suggestion has not been generally accepted. This study on normal, healthy young men and women, and on men who have had myocardial infarction or coronary insufficiency with impairment of myocardial function, demonstrates the value of posture in decreasing the cardiac output (Table 1).

TABLE 1

EFFECT OF POSTURAL INFLUENCES ON CARDIAC OUTPUT AND METABOLISM

Posture	Cardiac Output % of Supine	Metabolism % of Supine
Sitting in easy chair, full support	86*	90*
Seated on straight-backed chair, relaxed	93	110
Seated on edge of bed with feet supported	95	110
Semi-reclining at 45° with knees up	110	98
Standing	92	120
Seated on high stool, forearms on table	—	120

*Male patients who had had a myocardial infarction

In these studies the reference position has been the supine, with the subject resting in bed in a fasting state and with emotional stimuli minimized. We have found that the position of

minimal cardiac work is that which obtains when the subject is sitting in a chair with full head, trunk, and arm support and with the feet flat on the floor. Sitting with legs dangling at the bedside or sitting in a straight back chair without head or upper extremity support requires greater cardiac output, but even this is less than that demanded when the subject is lying supine. The *only* sitting position which produces a cardiac output greater than that required when lying supine is a "semi-sitting" position in a bed with the backrest and the knee support elevated. The sitting position with the feet and legs dependent allows for the pooling of blood with a decrease in the venous return, and it thereby decreases the drive on the heart.

Studies on patients with myocardial disease indicate that cardiac patients respond to position as do normal individuals, except that their oxygen consumption *as well as* their cardiac output may be slightly less in the sitting position than in the recumbent position. This was true in a group of male patients who were able to carry on light activity, and who did not complain of orthopnea. It may be assumed that patients with orthopnea will experience even greater postural relief of cardiac output as well as oxygen consumption and that the sitting posture will be even more protective.

Muscular work

Continual recording of the oxygen consumption showed that patients did not remain at the basal level for long when in bed. Each small or large movement of the patient brought an increase in the oxygen consumption. Patients confined to bed move frequently; the unsupervised patient, especially, may reach, shift, turn, or even sit erect. All these activities require lifting of extremities or elevation of the body, and all increase the oxygen consumption. Even under the best of circumstances, the average oxygen consumption of the bedfast patient is somewhat above the basal, and it may fluctuate to levels far above the basal. In particular, the shift from the lying position to the sitting position requires an extremely high consumption of oxygen. Likewise, reaching to pick up an object at arm's length requires a great increase in oxygen consumption. Because these activities are performed frequently by bedfast patients, the assumed protective effect of "bed rest" is somewhat misleading. Bed rest may involve anything from the relatively low oxygen consumption attendant on complete minimization of muscular activity to a relatively high oxygen consumption as the patient turns, reaches, or shifts position.

In getting out of bed, the greatest amount of oxygen consumption, and probably the greatest cardiac work, is involved in shifting from the lying position to the sitting position in bed. Less energy is required to step down from the bed to a chair or to stand up from a chair when returning to bed. Thus the unsupervised patient who is restricted to bed may actually do more work if he is restricted to bed than if he is sitting. When a patient is seated in an armchair with adequate back and arm support and with a table before him to keep any necessary equipment within easy reach, he does less metabolic work than when he is lying in bed. The high peaks of energy expenditure are lower than when he is lying in bed, and since he has the benefit of venous pooling in his lower extremities, his cardiac output is lower than it would be if he were lying in bed.

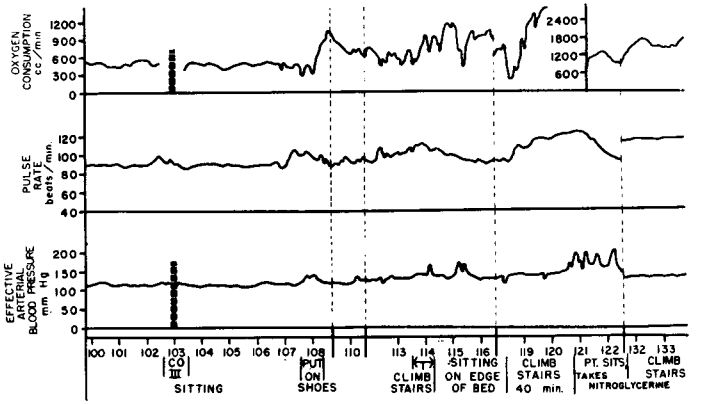

Fig. 1

Metabolic and cardiac responses of a 38 year old patient recovering from a myocardial infarction to the common stresses of putting on his shoes or climbing stairs. Stair climbing was over a double step with a seven inch riser. At 120 minutes, patient developed angina associated with bouts of increased blood pressure to 200 mm. Hg.

The vertical bar on the oxygen scale indicates cardiac output in liters per minute. The vertical bar on the blood pressure scale equals left ventricular power output in kg.-m. per minute.

Continual recording of oxygen consumption during simulated hospital activities showed that for short intervals the heart frequently has loads placed on it that are entirely unsupected.

As the patient lies in bed, his oxygen consumption—and presumably his cardiac output—shows significant fluctuations during shifting of position or movement of the extremities. Less shifting and less fluctuation in oxygen consumption occur when the patient is sittting quietly in an easy chair than when he is in bed. Whether the work rate of the left ventricle increases or decreases depends upon the change in the blood pressure under the circumstances. Scarcely more energy is required for the patient to stand when his motor-powered Franklin bed is raised to the vertical position than to lie prone. Transfer from bed to chair in this way does not greatly increase cardiac work.

Self-care activities such as bending over to put on shoes or other dressing activities, because they require bending and elevation, increase the rate of cardiac work (Fig. 1). Climbing stairs, one of the very heavy activities of daily living, quickly increases oxygen consumption and cardiac output.

Emotional Influence

Emotional tension increases cardiac output, blood pressure, and cardiac work in working situations and also within the hospital. It is hard to study this problem because of the difficulty of reproducing an emotional state consistently, so that values may be rechecked in one patient, or so that comparable studies can be made on a number of patients. In the hospital, fear adds greatly to the stress on the heart. Understandably, patients with cardiac disease have many fears. They are afraid because of their illness: They fear for their families, they fear for their jobs, they fear traumatic or painful procedures. They are upset by rounds, confusion, noise, and exposure to strangers. In a laboratory setting, where most of these stimuli are removed deliberately so as to provide a base line from which to study cardiac changes, it is difficult to establish a controllable emotional stimulus. Moreover, patients in the laboratory, surrounded by much complicated equipment, are inclined to accept the situation as safe and carefully controlled. When minor emotional tensions developed in the course of these experiments, the cardiac work was observed consistently to increase. Thus, for example, the cardiac output measured after an arterial catheter had been inserted was found to be slightly higher than that recorded prior to the arterial puncture. It appears likely that the injection of procaine® anesthesia used in this procedure, the draping, and the wearing of gloves all added to the emotional tension of the patient. Consequently, the cardiac output was observed to have increased approximately 25 per cent following

the arterial puncture. In 12 subjects the mean cardiac output before the arterial puncture was 3.50 ± 1.16 liters per minute, while the mean cardiac output immediately afterward was 4.80 ± 2.51 liters per minute.

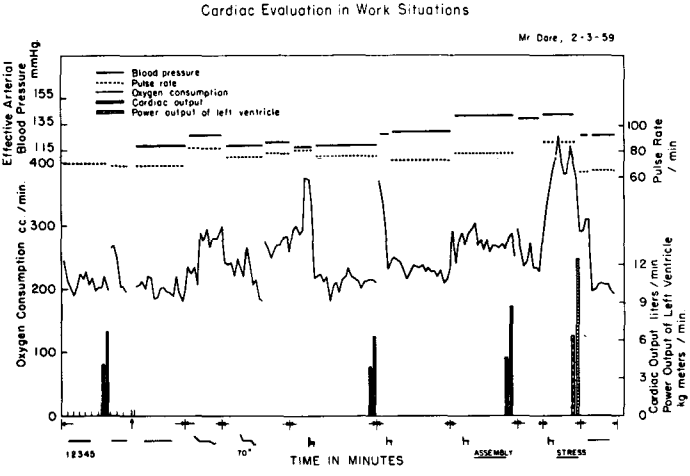

Fig. 2

Metabolic and cardiac responses of a 53 year old man to postural changes and to the stress of work. Diagrams at bottom of figure indicate position in bed, easy chair, or straight chair. Left ventricular power output increased above the supine level by 40 per cent when patient was carrying out a light assembly activity at his own rate; it increased by 100 per cent above the supine level when he was forced to hurry in the performance of the same activity.

Increased emotional tension in a vocational situation may also increase myocardial work due to an increase of muscular work or blood pressure or both. An experimental study on a patient recovering from a myocardial infarction showed the stressful effect of attempting to carry on a light task at a rate which was too rapid (Fig. 2). This patient had a lower cardiac output and left ventricular power output when sitting than when supine. When he carried on a light task of assembling electrical connectors at his own rate, his metabolism increased about 25 per cent, and his myocardial work rate increased 40 per cent. But when he was asked to work against a metronome set at a rate faster than his optimal working rate, his myocardial work rate increased 100 per cent above his rate during supine rest.

This type of stress in a vocational setting may not be easy to establish or to evaluate, but it may be of major importance in determining whether or not a patient may return safely to a particular job.

These studies indicate that the reasonable minimal level of cardiac output for a prolonged period when the patient is in bed or sitting in a chair is in the range not exceeding 125 per cent of the supine basal cardiac output. Only when activity is restricted to the hands with adequate arm support and with avoidance of reaching and lifting can cardiac output be restricted to this level. The light hand activities commonly used in occupational therapy increase cardiac output from 10 to 25 per cent (Table 2). There is a 5 to 10 per cent protective effect from sitting on a chair compared to lying or semi-reclining in bed while doing such activities. Moreover, these crafts relieve boredom and divert the patient's attention from worries about himself during the time that he must remain at minimal activity.

TABLE 2

ACTIVITIES PRODUCING A MINIMAL INCREASE IN CARDIAC OUTPUT

Activity	Cardiac Output % of Supine	Metabolism % of Supine
MINIMAL	less than 125	less than 150
Leather stamping, at table	112	130
Chip carving, at table	117	160
Leather tooling, at table	117	120
Leather tooling, semi-reclining in bed	125	110

This study was supported in part by Research Grant No. 349 from the Office of Vocational Rehabilitation, and Research Grant H-1290 from the National Heart Institute.

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Staff Meeting Report

An Unusual Staphylococcal Product and its Host Interactions*†

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Among unsolved bacteriologic curiosities, one of the most fascinating is the phenomenon described by Leon Muller in 1927.[1] He observed that if blood agar plates inoculated with staphylococci are incubated for relatively long periods of time (2 to 5 days), multiple discrete areas of proteolysis appear at some distance from the growing staphylococci. This phenomenon differs from ordinary hemolysis or proteolysis; its distinguishing feature—and one of its chief attractions as a biologic phenomenon—is the *discreteness* of the areas of clearing. A diagram of the Muller phenomenon showing a streak of staphylococci and the areas of clearing at a distance from the growing organisms is seen in Figure 1.

PAUL G. QUIE

At least three factors are necessary for the Muller phenomenon: a *bacterial* factor, a heat-labile *serum* factor, and a *suitable substrate*, such as red cells or hemoglobin. Since fibrin[2] and casein[3] may also be used as substrates, the phenomenon apparently involves a proteolytic system in some way. One must emphasize that there is no satellitism of staphylococcal colonies: *The areas of proteolysis are sterile.*

The sera from several mammalian species may provide the serum proteolytic factor, but the bacterial factor or activator has

*This report was given at the Staff Meeting of the University of Minnesota Hospitals on November 25, 1960.

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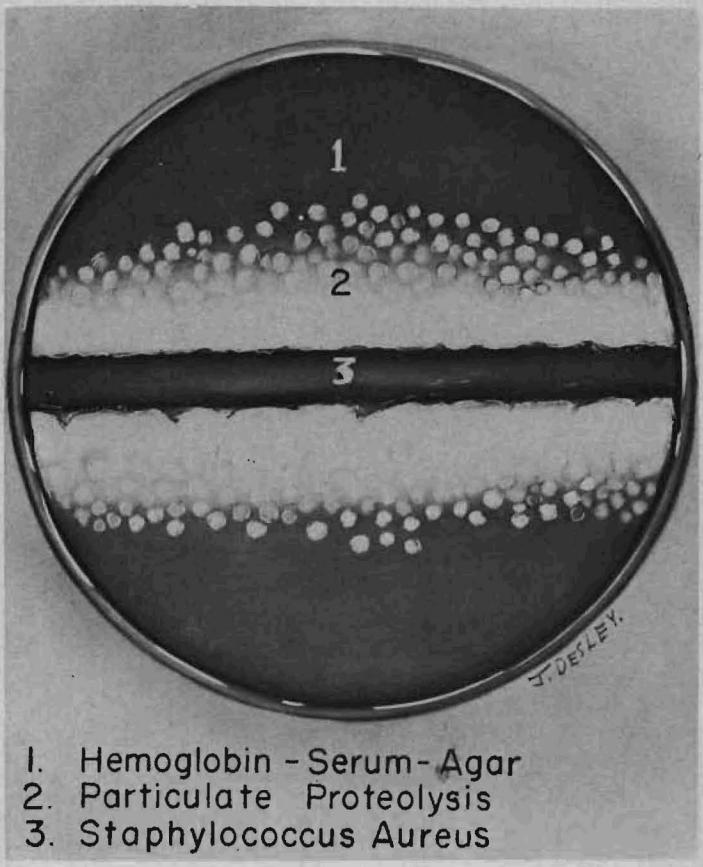

Fig. 1. The Muller phenomenon

seemed to be peculiar to coagulase-positive staphylococci. Approximately 90 per cent of coagulase-positive staphylococci from various sources produce the phenomenon, whereas coagulase-negative staphylococci do not produce it. Furthermore, the phenomenon has never been observed in relation to colonies of streptococci, including those strains which are known to be good streptokinase producers.

Although this phenomenon was described by Muller more than 30 years ago, investigation of its relevant staphylococcal and serum factors has awaited a satisfactory method for pro-

ducing the phenomenon in a sterile system. Elek[4] was able to extract the bacterial factor from agar on which staphylococci had been grown. We have obtained the staphylococcal Muller factor by the simpler method of growing the staphylococci in liquid dialysate media[5] with aeration.

Figure 2 shows the Muller phenomenon produced with a sterile staphylococcal supernate added to an agar plate containing rabbit serum and hemoglobin. The size and shape of the areas of particulate proteolysis are identical to those seen around the growing colony of staphylococci. Using a constant quantity of staphylococcal supernate, we observed that the number of areas of clearing is dependent upon the concentration of serum.

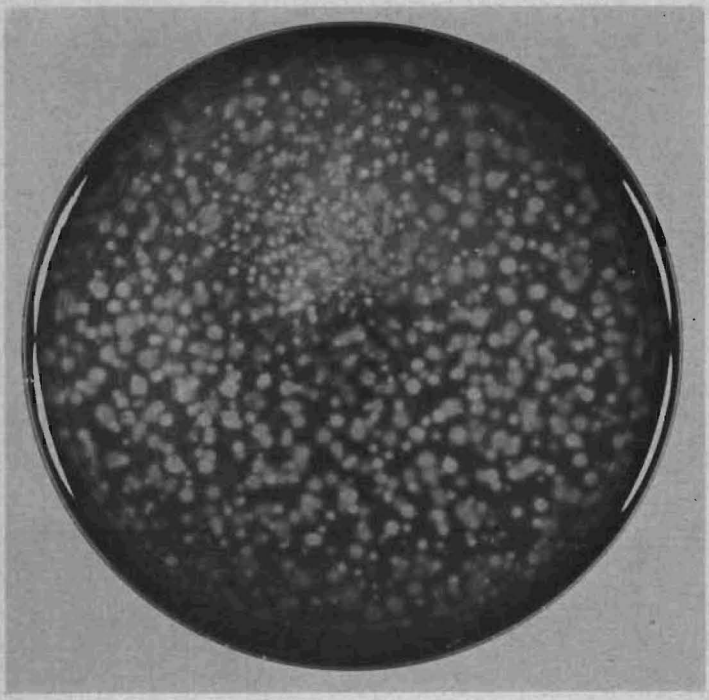

Fig. 2. Particulate proteolysis in a sterile system

In an attempt to purify the staphylococcal Muller factor, a concentrate of the crude staphylococcal supernate was pre-

pared and subjected to starch zone electrophoresis. By this technique, the Muller factor was separated from the other staphylococcal enzymes and unidentified protein constituents; this factor was not separated, however, from staphylokinase — a fact which suggests that these two staphylococcal products may be identical and also that the Muller phenomenon may be related in some way to the plasminogen-plasmin (fibrinolytic) system. Experiments in our laboratory indicate that the phenomenon can be produced by substituting "purified" plasminogen for whole serum. Moreover, the phenomenon was found to be inhibited by substances (epsilon-aminocaproic acid and soybean trypsin inhibitor) which inhibit the plasminogen-plasmin system. We have also produced the phenomenon with preparations of *streptokinase* using a double-diffusion technique in hemoglobin agar.

Although similar to the plasminogen-plasmin system in certain respects, the unusual discrete appearance of proteolysis in the Muller phenomenon remains unexplained. Other investigators have considered the discreteness of the phenomenon to be due to some particulate component of the serum. From ultrafiltration studies, Elek has postulated a particulate factor in serum of approximately 50 to 100 $\mu\mu$ size.[4] Preliminary studies in our laboratory, however, suggest that both the serum and the staphylococcal factor diffuse readily through agar and sediment slowly in the ultracentrifuge. Therefore, the explanation of the discrete appearance of the phenomenon may be more complex than is entailed in postulating a particulate factor in serum or in staphylococcal supernates.

Additional studies of the phenomenon in a cell-free system were greatly facilitated by the development of a technique for obtaining relatively large quantities of staphylococcal culture supernates containing the active Muller factor. Using the cell-free system, we found that the Muller phenomenon could readily be produced in plates containing rabbit serum, whereas it was rarely observed in plates containing human serum. This finding suggested the possibility that sera from normal adults contain an inhibitor of the staphylococcal Muller factor. The presence of such an inhibitor was confirmed when addition of human adult serum to a plate containing an active rabbit system was found to prevent the Muller phenomenon.

In order to determine whether or not the production of inhibitor could be induced in rabbits which normally lack the inhibitor, rabbits were immunized with concentrates of staphylococcal extracellular protein containing active Muller factor.

When added to plates containing staphylococcal supernate and normal rabbit serum, the sera from these immunized rabbits inhibited the Muller phenomenon. These sera from the immune rabbits were then subjected to starch zone electrophoresis, and we observed that the inhibitor of the staphylococcal Muller factor was present in the gamma globulin portion of the protein pattern; we also noted that the serum proteolytic factor was partially separated from the serum inhibitor (Fig. 3).

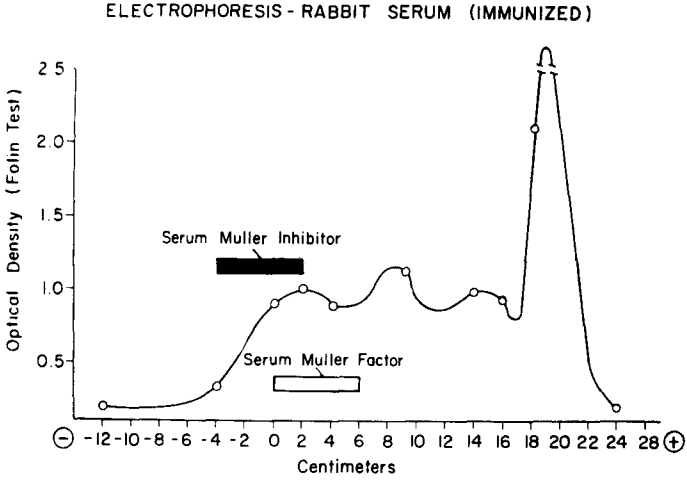

Fig. 3. Electrophoresis—rabbit serum (immunized)

The presence in human sera of an inhibitor of an extracellular product of staphylococci found only in coagulase-positive strains made it of interest to study the distribution and the level of inhibitor in human populations (Fig. 4). Our studies to date indicate that virtually all sera from "normal" adults show some inhibition of the Muller phenomenon. In children 2 to 14 years of age, the inhibitor levels vary widely. Sera from infants frequently failed to inhibit the Muller phenomenon, especially those from infants at the age of 4-7 months —an age when gamma globulin is at a low level. In contrast, the titers in cord sera were similar to those in sera from adults, suggesting placental transfer. Figure 5 shows the inhibitor levels in maternal and cord sera pairs. The inhibitor levels matched within 1 dilution in all of the cord-maternal pairs.

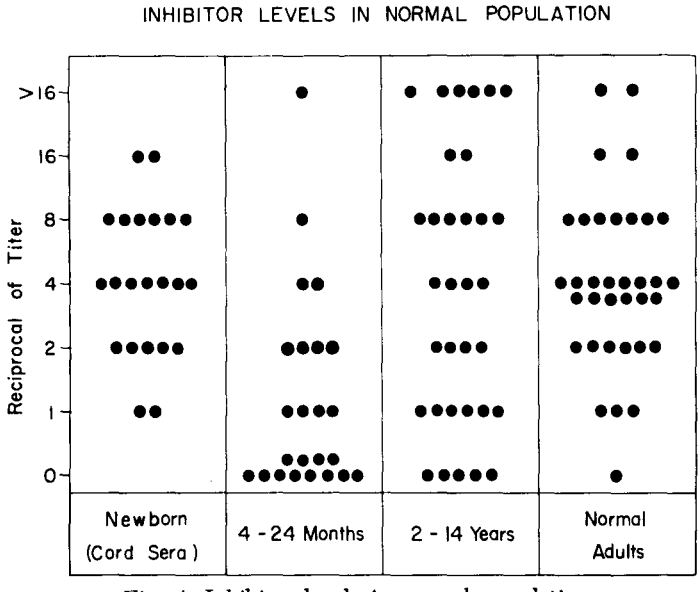

Fig. 4. Inhibitor levels in normal population

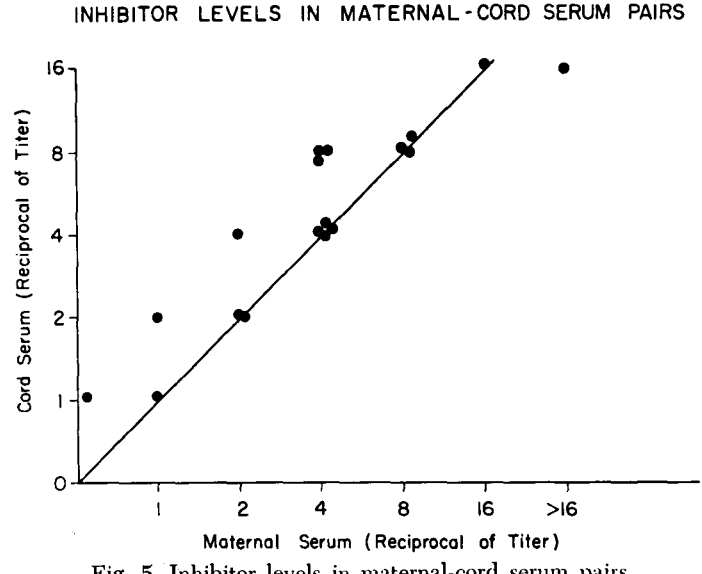

Fig. 5. Inhibitor levels in maternal-cord serum pairs

Interestingly, in 6 of the 18 pairs, the cord sera showed an inhibitor level that was 1 dilution higher. A similar tendancy for cord titers to be higher than maternal titers has been reported for several different antibodies.[6,7]

The likelihood that this inhibitor of the Muller phenomenon is an antibody is suggested by its appearance in the gamma globulin fraction of serum following immunization of rabbits; this likelihood is strengthened by its absence in the sera of patients with agammaglobulinemia. As shown in the last column of Figure 6, the sera of six children with this disease completely lacked this inhibitor.

The distribution of inhibitor levels in sera from patients with staphylococcal infections in general is seen in Figure 6. The levels in adult patients with staphylococcal lesions were similar to the levels found in healthy adults; furthermore, there was no relationship between the severity and duration of the infection and the level of inhibitor in the serum. The levels in sera of children who had recovered from staphylococcal disease were higher than most of the levels in sera from normal children of a similar age.

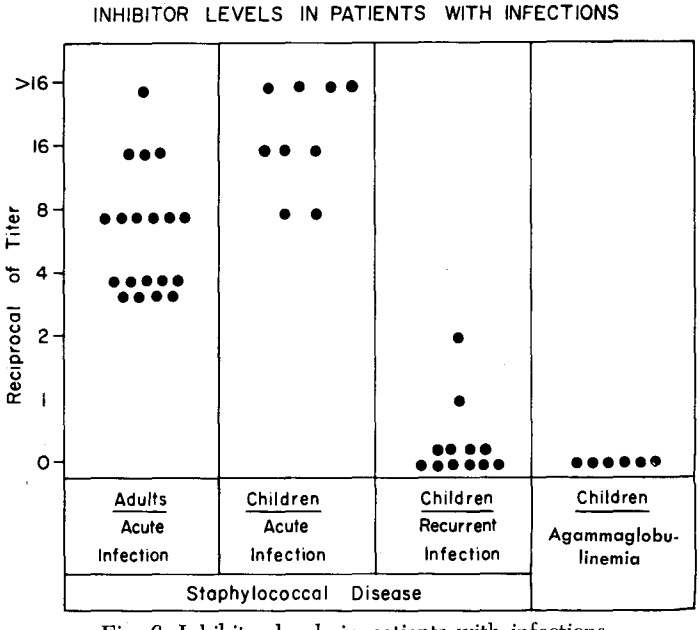

Fig. 6. Inhibitor levels in patients with infections

The sera from twelve children with recurrent infections from which staphylococci were repeatedly cultured, however, demonstrated either no inhibitor or low levels of inhibitor of the staphylococcal Muller factor. Seven of these children had recurring boils and superficial infections without systemic symptoms and were otherwise healthy children. The other five had severe debilitating staphylococcal disease. Upon thorough investigation of the 12 patients, no evidence was found for tuberculosis, cystic fibrosis, or fungal disease. The patients showed normal antibody responses to mumps and polio virus, typhoid vaccine, and diphtheria toxoid, and all the children with severe disease were found to have *hypergammaglobulinemia.* The reason for the lack of inhibitor response despite repeated infections with staphylococci is not known, but the early age at which certain of the patients were infected and the nearly continuous presence of infection suggest a specific immunologic unresponsiveness[8] to this staphylococcal product.

Summary

The curious bacteriologic phenomenon described by Muller in 1927 consists of multiple discrete areas of hemolysis or proteolysis which appear at some distance from colonies of staphylococci grown on agar containing serum and a suitable indicator (red cells, hemoglobin, other proteins). The bacterial factor has been obtained in supernates of staphylococcal cultures and has been partially purified by starch zone electrophoresis. By means of these preparations, the phenomenon has been produced in a sterile system. Since the phenomeon has been shown to require the presence of serum, the possible relationship of this phenomenon to the plasminogen-plasmin (fibrinolytic) system has been explored.

An inhibitor of the Muller phenomenon has been found in sera from normal human adults and from adults with staphylococcal infections. The inhibitor is heat stable and has many of the characteristics of antibody. This inhibitor is absent in normal rabbit sera, but it is developed in the sera of rabbits immunized with concentrates containing staphylococcal Muller factor. The inhibitor is found in the gamma globulin fraction of serum and is absent in sera from patients with agammaglobulinemia. The level of inhibitor was found to be similar in cord sera and maternal sera, but inhibitor was usually observed to be absent from sera of infants 4-7 months of age. In children with chronic staphylococcal infections, inhibitor levels were either zero or low, suggesting an immunologic unresponsiveness. The possible

role of this inhibitor in the pathogenesis of chronic recurrent staphylococcal infection deserves further study.

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Staff Meeting Report

Breast Cancer Morphology and Adrenal Corticosterone Elevation in C and D_8 Mice with Heterotopic Pituitary Isografts*†

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I. Introduction

For certain malignant disorders, hypophysectomies have been performed in human beings. Alternatively, and for the same purposes, one may attempt to modify pituitary function *in situ*. As far as possible, the latter approach aims at eliminating undesired pituitary effects without loss of other important functions of this gland; as yet this goal has not been realized.

ERHARD HAUS

The ultimate success of failure of attempts at modifying the function of the pituitary left *in situ* may depend upon the availability of specific physiopathologic information. Therefore, we can raise several related questions: How does the pituitary gland, so important for normal growth, contribute to malignancy? How many of its hormones are involved in carcinogenesis, and what is the nature of their detrimental interactions?

Among other approaches to these problems, we have used as our experimental basis mouse breast cancer induced by

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heterotopic isografts. The model is derived from the recognition that a pituitary functioning at a site removed from its normally adjacent controls predictably modifies breast cancer rate.[1–5]

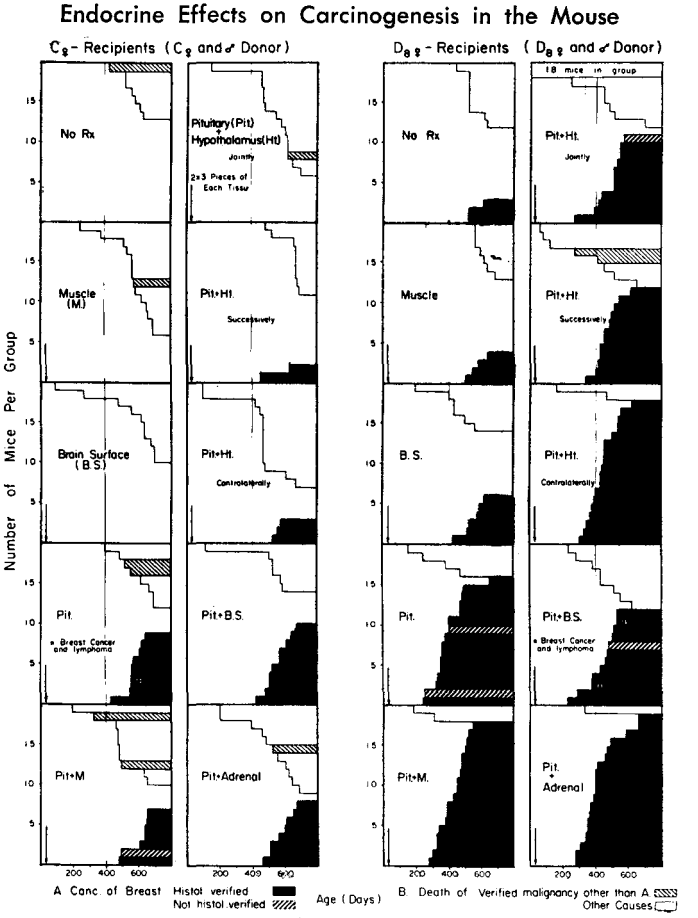

Fig. 1. Breast cancer incidence (black areas; staircase starting at bottom) and death from other causes (top) in each of several experimental subgroups from two inbred strains.

II. Cancer Model Used

Whether or not a mammary tumor agent (MTA)[6–10] can be demonstrated, the pituitary isograft induces or enhances mam-

mary carcinogenesis.[3,4] In an attempt to account for this effect Mühlbock and Boot suggested that heterotopic pituitaries secrete predominantly prolactin and possibly growth hormone.[3,4]

In this laboratory, the relation of pituitary function to breast cancer was explored by focusing upon the hypothalamus in one investigation,[5] and upon the adrenal in another.[11] Thus, in one strain of mice (C or Bagg albino), at least, breast cancer rate appears to be modified as a result of heterotopic pituitary isografting.[5] This modification is achieved by hypothalamus insertion at the time of pituitary implantation.

Moreover, in two strains of mice, C and D_8 (subline 8 of the Dilute Brown strain), adrenal involvement during pituitary isograft function also was ascertained.[11] Fluorometric estimations of corticosterone[12] in the adrenal itself were made at the anticipated peak time[13] of the circadian (circa-dies) adrenal cycle.[14] After two to six months, higher values were recorded in mice bearing pituitary isografts than in control mice given nonglandular tissues.[11]

The purpose of this progress report is: (1) to summarize some morphologic features of follow-up studies on the particular cancer model used, and (2) to amplify and to extend the work on adrenal corticosterone changes after pituitary isografting.

An analysis of the as yet unclarified hypothalamus effect is beyond our scope in this report. This problem and the higher breast cancer rate induced in mice by ectopic pituitaries from male donors (in comparison to pituitaries from female donors) will be the subject of a separate paper.[15]

Fig. 1 is included solely as an example of our current studies of pituitary-hypothalamic interactions in relation to breast cancer. This figure reveals a significant hypothalamus effect upon the cancer incidence of C mice bearing pituitary isografts but not upon that of similarly treated D_8 mice.

The data on C mice are given in the left of the figure. Breast cancers, shown as black areas, are numerous in those subgroups (each composed of 20 mice) that twice received three pituitaries by trocar into the axilla—the glands being given either alone or in combination with muscle, adrenal, or brain surface (two bottom rows of C mice data on the left).

A comparison of these bottom rows summarizing the cancer incidence of C mice bearing pituitary isografts with the three cells depicting control groups (above them on the left) leaves little doubt of the efficacy of heterotopic pituitary function in inducing mammary cancer in this stock. Thus, at 800 days of age, roughly half of about 80 animals receiving isografts had histologically verified breast cancer; after the same interval no breast tumor whatsoever was observed in about 60 control animals given no treatment, or given isografts only of a muscle or brain surface (Fig. 1).

The hypothalamus effect also stands out in the C mice data, particularly in those C mice given the pituitary and hypothalamus jointly (by the same trocar).

Our primary interest in the model of breast cancer discussed herein lies in this rather unexpected hypothalamus effect upon mammary carcinogenesis from pituitary isografts.[5] But apart from this effect, we felt it would be of physiopathologic interest to explore further the behavior

of adrenal corticosterone in mice bearing heterotopic pituitaries. These chemical studies will be discussed after a brief summary of the morphology of breast cancers such as those depicted as black areas in Figure 1.

TABLE 1

SOURCE AND DISTRIBUTION OF BREAST CANCERS EXAMINED

Strain	Sex	Total Number of Mice	No. of Mice with single Cancer	No. of Mice with multiple Cancer	Total Number of Cancers
C	F	303	42	16	81
D_S	F	339	112	37	212

III. HISTOPATHOLOGIC DATA

a) *Methodology.* For study of cancer morphology, the animals in most cases were killed before tumor size exceeded 2 cm. in diameter. Only an occasional animal died without a complete autopsy, which included, with the gross examination for metastases in the lung and elsewhere, the consistent removal for histology of all grossly altered tissues. In many instances, the pituitary and adrenals were also examined histologically.

Tissues were generally fixed in formalin; those pituitary glands which were removed for special study, however, were fixed in Zenker-Formalin or Susa, being stained either with Gomori's aldehyde fuchsin or with the staining method by Dickie.[16] For routine examination, all tissues, including the cancers reported herein, were stained with hematoxylin-eosin.

One block was taken from each tumor smaller than about a centimeter in diameter, while several blocks were cut from each larger tumor. Tumor type was evaluated by examining three to five sections from a given block. The variability encountered in cancer morphology was anticipated, since it was so often recorded by others for breast tumors arising in mice in the absence of pituitary isografts.[17–20]

In the present material, which consisted largely of cancers from mice bearing pituitary isografts, great variability was observed—both within tumors and among tumors. Nevertheless, certain distinctive morphologic features, which will be designated as "predominant components," did stand out clearly. Reference to such components must be qualified, of course, to the extent that we are dealing with a subjective and qualitative

estimate of the distribution within tumors of a given morphologic feature. By "predominant" we do not mean to imply that a given component necessarily occupied the major portion of the tumor areas examined.

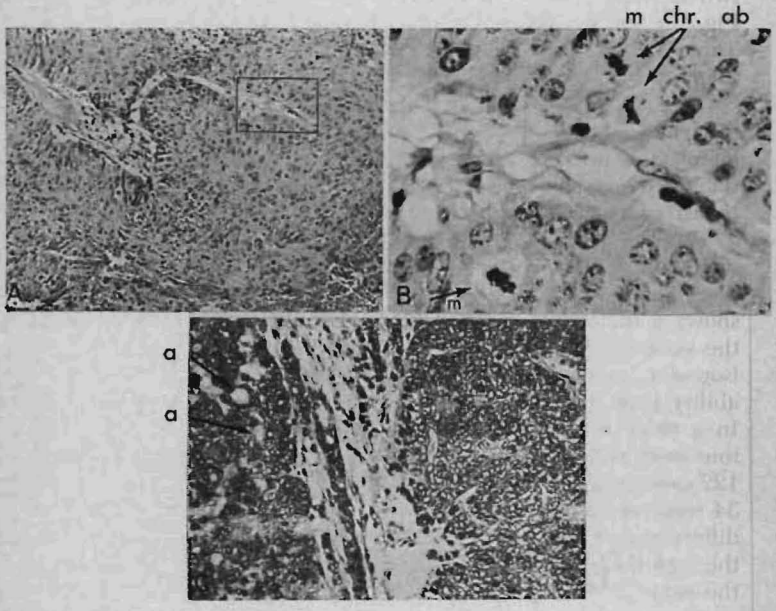

Fig. 2. Breast cancers—low degree of differentiation. (A) Undifferentiated compact tumor. Pleomorphic cells. No evidence of glandular structure. Many mitoses. Female C mouse, 18 months of age; Rx: 2 x 3 pituitary isografts into axilla, at about 5 weeks of age. H & E, × 80. (B) Higher magnification of A (× 425). Three metaphases (m). Chromosomal aberrations (chr. ab.). (C) Unequal low degree of differentiation (left) with sporadic structures resembling acini (a). Solid strands of cells, right. Female D_8 mouse, 13 months of age; Rx: 2 x 3 pituitary isografts into one axilla and 2 x 3 hypothalamic inserts into contralateral axilla, at about 5 weeks of age. H & E, × 150.

Furthermore, cancers with any of the predominant components were observed in mice bearing pituitary isografts as well as in control animals. A discussion of our material must be limited, therefore, to the proportion of the several morphologic types in certain treatment groups. Incidentally, it is this proportion of the morphologic forms of cancer which has been found

to vary so greatly in breast tumors from other distinctive groups of nonisograft-bearing mice, as Dunn[18,19] and Cloudman[20] authoritatively reported.

b) *Cancer morphology.* Materials examined here were from two separate studies involving several treatment subgroups (cf. Fig. 1) set up concomitantly. A total of 642 recipient mice, Table 1, were examined in these two experiments. Those mice dying before the appearance of the first tumor were omitted.

The breast cancers in both strains, D_8 and C, will first be discussed according to their degrees of differentiation. Morphologic types ranged from well-differentiated forms to undifferentiated tumors that were barely compatible with the diagnosis of breast cancer.

Figure 2 shows examples of less differentiated forms; these were encountered in both strains studied, as were the more differentiated forms, illustrated in Figure 3. Figure 4, in turn, shows a tumor embolus in the lung, as well as a metastasis in the same organ. The metastases found in mice bearing pituitary isografts, and the results of frequently performed transplantability tests give evidence of the malignancy of these cancers. In a total of 25 cancers arising spontaneously in 23 D_8 mice, four were undifferentiated (16 per cent). By comparison, among 122 cancers collected after pituitary isografting from 57 D_8 mice, 34 were undifferentiated (28 per cent). The percentage of undifferentiated cancers in D_8 mice given both the pituitary and the hypothalamus (total of 58 cancers from 25 mice) was about the same as that in animals given the pituitary alone (31 per cent).

Irrespective of treatment, in both strains the differentiated forms were more common than the undifferentiated ones, as may be seen from Figure 5. Moreover, as has been reported earlier for cancers arising in the absence of isografts, the preponderance of differentiated types is characteristic also of the breast cancer model obtained by heterotopic pituitary function.

Figure 5 presents, further, a breakdown of the proportion of "predominant components" among the differentiated cancers recorded in the two strains of mice studied. Squamous metaplasia was seen more often in C mice than in D_8 mice. The difference in the proportion of this component, tested by the Chi-square method was found to be significant below the 1 per cent level. A papillary component also predominated more frequently in cancers from C mice than in those from D_8 mice. This difference also was significant below the 1 per cent level when tested by Chi-square (Fig. 5).

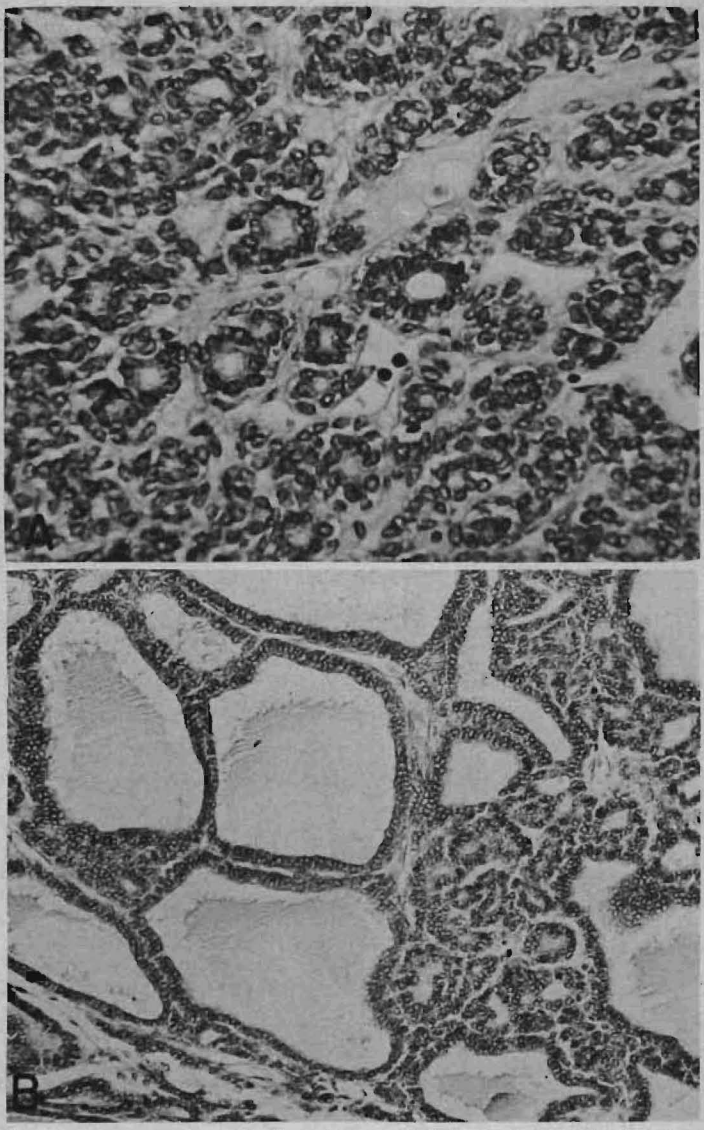

Fig. 3. Breast cancers–higher degree of differentiation. (A) Acinar adenocarcinoma; tumor reproducing mammary acini. Some signs of secretory activity. Relatively few mitoses. Female D_s mouse, 16 months of age; Rx: 8 x 3 adrenals into axillae during 2nd and 3rd months of life. H & E, × 320. (B) Cystic adenocarcinoma; cysts arising from dilatation of glandular lumina. Signs of secretory activity. Female D_s mouse, 20 months of age, 2 x 3 pituitary and adrenal isografts into axillae, at about 5 weeks of age. H & E, × 160.

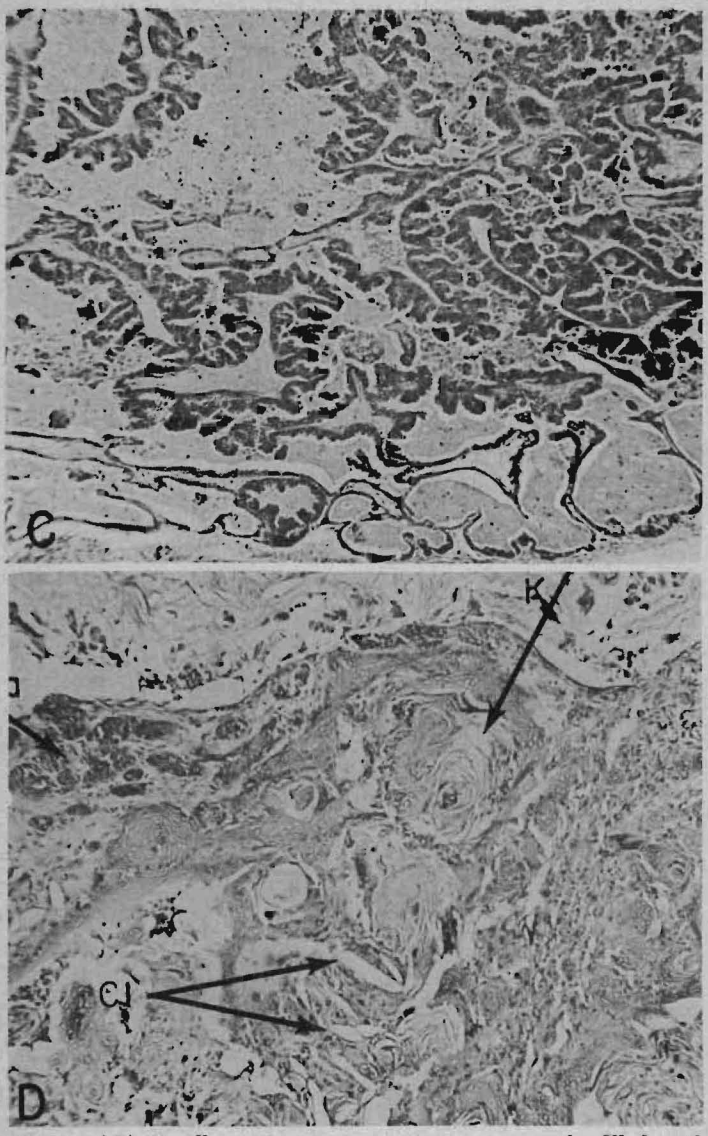

Fig. 3. (C) Papillary carcinoma; cystic spaces mostly filled with debris. Female D_8 mouse, 12 months of age; Rx: 2 x 3 pituitary isografts into one axilla and 2 x 3 hypothalamic inserts into contralateral axilla, at about 5 weeks of age. H & E, × 140. (D) Adenoacanthoma. Extensive squamous metaplasia and keratinization (k). Formation of epithelial pearls. Areas of necrosis and "cholesterol" clefts (Cl) are seen in lower center. Some areas of acinar structure (a), in upper left quadrant. Female C mouse, 16 months of age; Rx: 2 x 3 pituitary isografts into axilla at about 5 weeks of age. H & E, × 140.

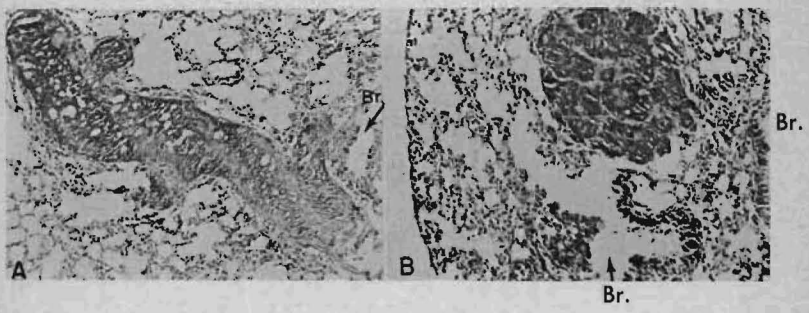

Fig. 4. (A) Tumor embolus, mostly necrotic, in medium-sized branch of pulmonary artery. No apparent infarction of surrounding lung tissue. Br. = Bronchus. Female D_8 mouse, 12 months of age; Rx: 2 x 3 pituitary isografts into axilla at about 5 weeks of age. H & E, × 65. (B) Lung metastasis. Br. = Bronchus. Same animal as in 4A. H & E, × 80.

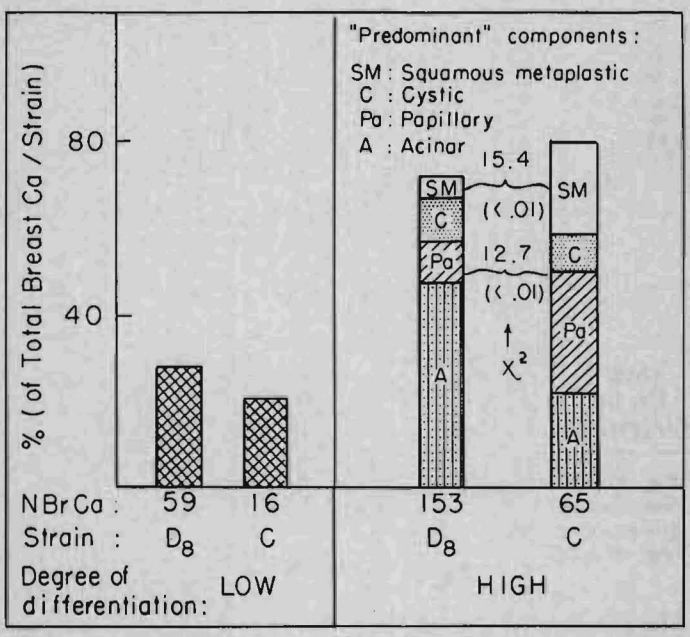

Fig. 5. Summary of some differences in the proportion of distinctive types of breast cancer in strains investigated, irrespective of treatment. P values in parentheses (see text).

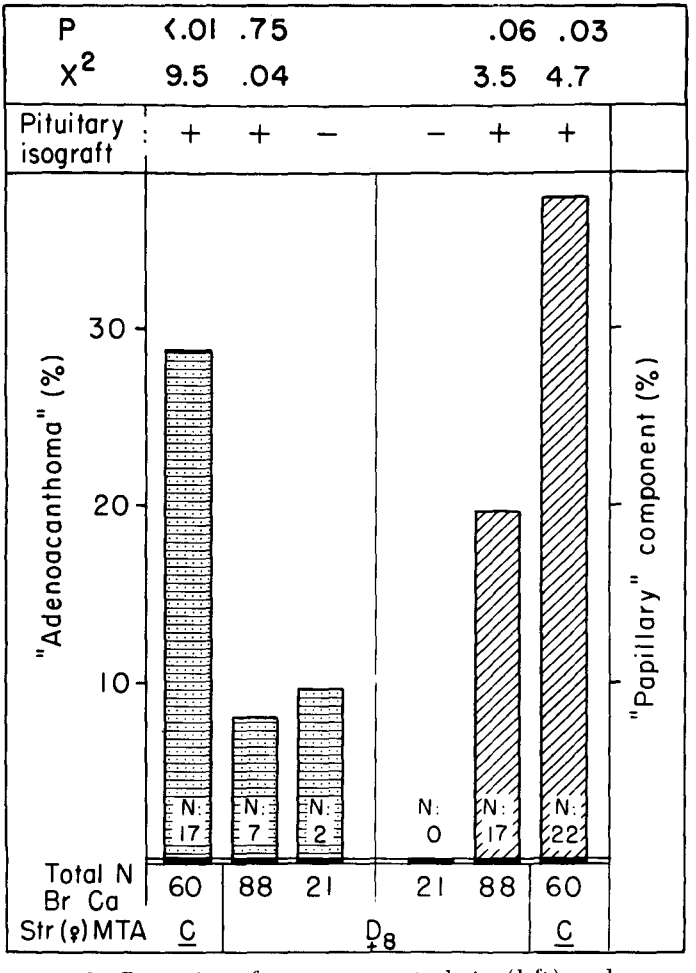

Fig. 6. Proportion of squamous metaplasia (left) and predominantly papillary carcinoma (right) in several treatment groups investigated. Chi-square tests of proportions in adjacent columns (top).

The presence or absence of MTA, in D_8 or C mice, respectively (*cf.* ref. 21), as well as strain differences, may underlie these findings—quite apart from any possible effects of the pituitary isograft. Indeed, the pituitary isograft seems to have no

effect (Fig. 6) upon the incidence of squamous metaplasia. Thus, whether or not the animals had heterotopic pituitaries, adenoacanthoma was much more infrequent among cancers from D_8 than from C mice.

Among D_8 mice, papillary carcinoma was found to occur more frequently in isograft-bearing animals than in control animals (Fig. 6). But the degree of generality of this result, and its significance, await further study on larger samples of tumors.

IV. Recovery of Implanted Pituitaries

The cancers discussed above were collected in two experiments involving axillary implants. Other studies underway in this laboratory involved implantations made subpectorally, into

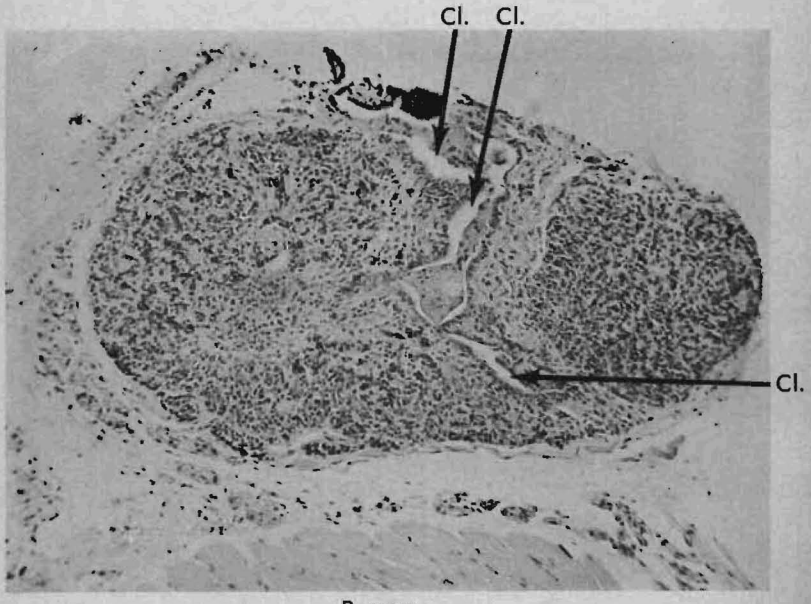

Fig. 7. Implanted pituitary recovered 19 months after isografting. Well vascularized gland. Chromophobe cells. Cleft-like formations (Cl) lined by cuboidal ciliated epithelium and filled with colloid material. Increased amount of connective tissue in center and to the left of clefts. P. musc. = pectoral muscle. Female C mouse; Rx: 2 x 3 pituitaries and 2 x 3 pieces of brain surface into axilla at about 5 week of age. H & E, × 140.

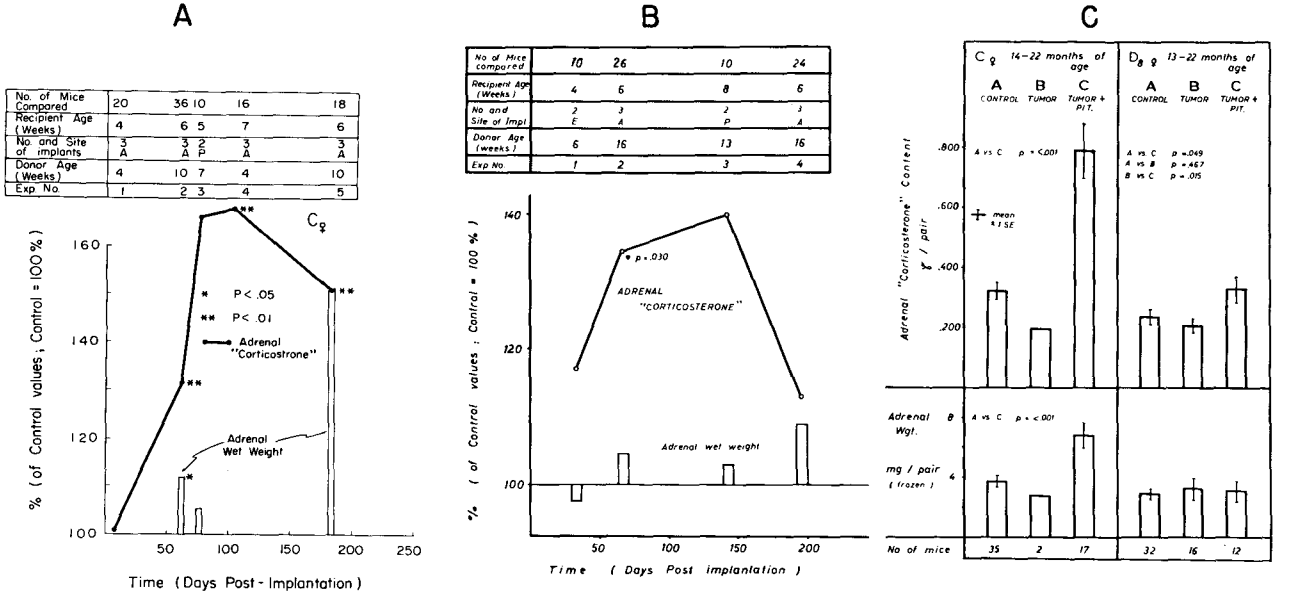

Fig. 8. Adrenal corticosterone elevation following ectopic pituitary isografting, in two stocks of mice, during two selected time periods (third to sixth month after isografting [A and B], and later [C] after appearance of overt breast cancer).

the ear or under the kidney capsule. These sites obviously compare favorably to the axilla in permitting easy recovery of the pituitary isograft.

Thus, in two experiments involving subpectoral implantations, most of the pituitary implants were recovered and examined histologically in serial sections. Most of the cells were found to be chromophobic, and a few chromophil cells were seen with the particular stains employed. On the average, the cells of the heterotopic graft appeared to be smaller than those of the pituitary *in situ*. Interestingly, some of the axillary implants also were recovered. One such ectopic pituitary was recovered about two weeks after implantation; it was well vascularized and showed occasional mitoses. At the other end of the time scale after axillary implantation, the pituitary shown in Figure 7, among several others, was recovered 19 months after grafting. Experiments are presently being directed at the question of how long heterotopic pituitary function must be exerted in order to enhance or induce breast cancer.

V. Adrenal Corticosterone and Heterotopic Pituitary Function

Our recent findings on adrenal corticosterone in mice bearing pituitary isografts as well as breast cancer are summarized in Figure 8c. These are aligned with data on the same variable obtained several months before the appearance of cancer (Figs. 8a and b). Some of the latter data (Fig. 8a) have been the subject of a recent communication[11] but for an appropriate overall evaluation all the procedures are summarized herein.

Female C and D_8 mice were used as recipients. Female mice of comparable genetic background were used as donors. Implantations were performed on animals kept under natural conditions of lighting; they were made during the afternoon or evening hours except for one experiment on each strain in which subgroups of recipients were given the treatments under study at four-hour intervals starting at 8 a.m. one day and ending at 4 a.m. the next. The grafting involved the insertion of three pituitaries per animal by trocar into the axilla (A), except for one experiment in which two pituitaries were inserted under the pectoral muscle (P) under ether anesthesia. Control groups of comparable mice were given pieces of femoral muscle, except for one experiment in which pieces of brain substance or hypothalamus were used. In experiments 1, 2, and 5 on C mice and in experiment 2 and 4 on D_8 mice, the animals were kept under conditions standarized for light-synchronized periodicity analysis[11] from grafting to killing. In the other experiments, the mice were housed under standardized conditions, with light from 6 a.m. to 6 p.m. and darkness from 6 p.m. to 6 a.m. for at least one week before they were killed. In experiments 3 and 4 on the C mice and experiments 1 and 3 on the D_8 mice, the animals were multiple-housed (5 to a side) in wooden boxes and were transferred to single cages for a standardization period of five days. In experiments 2 and 5 on C mice and in experiments 2 and 4 on D_8 mice the animals were multiple-housed from the time of implantation until they were killed.

Groups of pituitary implant and control animals were killed at several time points after grafting, as indicated in Figures 8a and b. In an additional experiment, animals without apparent tumors were compared with mice of corresponding age bearing a) spontaneous breast tumors or b) spontaneous breast tumors *and* pituitary isografts. This comparison was made in C and D_8 mice.

In C mice, however, the spontaneous cancer rate is extremely low less than 1 per cent. Accordingly, only two mice with breast cancer arising in the absence of pituitary isografts were available for comparison from the breeding and surplus stock in the laboratory. (Another study involving MTA injection in C mice, now underway, bears on a related problem which, however, is not identical.) The supply of D_8 mice with spontaneous breast tumors was large (Fig. 8c).

The mice bearing both breast cancer and pituitary isografts were taken from different studies, and hence they represent a heterogeneous age group. The control mice without breast tumor, however were so selected that they matched in age the corresponding animals in the tumor groups as far as possible.

All the animals used for the study summarized in Fig. 8c were multiple-housed under routine conditions in the same laboratory, until one week before being killed. During this last week, the animals were transferred to single cages, kept in periodicity rooms maintained at 24° C. with light from 6 a.m. to 6 p.m., alternating with darkness. Mice of the several experimental groups were killed in alternation between 3 p.m. and 5 p.m., the anticipated peak period of corticosterone rhythm.

With the qualifications given as to procedures, Figures 8a, 8b, and 8c strongly suggest that heterotopic pituitary function results in elevation of adrenal corticosterone. Figures 8a and 8b, based largely on data published earlier,[11] demonstrate the ectopic pituitary effect during the third to sixth month after grafting. The new data in Figure 8c suggest that in both stocks studied, adrenal corticosterone also is elevated in mice with pituitary isografts when breast cancer has developed in these animals.

The content of adrenal corticosterone during the several months preceding the appearance of overt breast cancer awaits further investigation, as does the course of adrenal weight change after pituitary isografting. From these studies and others, however, the weight of the adrenal appears to be much less sensitive than hormone content of the adrenal as an index of functional changes. Actually, the effects of pituitary isografting on the mouse adrenal may have been missed by earlier students simply because weight, rather than hormone content, was evaluated.

The primary physiopathologic question arising from these studies is the extent of relationship between the changes in adrenal corticosterone level due to isografting and the induction or enhancement of breast cancers resulting therefrom. Although both effects are brought about by heterotopic pituitary function, they are not necessarily related.

Many other questions remain unanswered. How, for example, does the heterotopic pituitary modify adrenal corticosterone content? Hertz has reported the release of growth hormone from pituitary homografts in the rat,[22] and, more recently, the release of gonadotropin and adrenocorticotropin as well.[23]

Furthermore, the present study does not indicate clearly whether the isograft elevates adrenal corticosterone content directly by its possible ACTH output or only indirectly, i. e., via an ovarian-adrenal interaction. The former possibility cannot be ruled out, and it has gained support from the recent study by Hertz.[23] Nor need we reject the hypothesis that the ovary is involved in isograft effects upon the corticosterone content of mouse adrenal. This possibility, in fact, gains some indirect support from recent work in this laboratory, where we have detected a higher corticosterone content in female mice in comparison to males[11] and have observed that ovariectomy lowers the corticosterone content in adrenals of C-strain mice, as well as in several other stocks.[24]

Finally, another recent morphologic finding in this laboratory seems pertinent to the results here reported. Clusters of cells resembling ganglion cells were found in a recently studied series of subpectoral implants from mice of several treatment groups. Such cells were detected in areas adjacent to the recovered pituitary implant in five of fourteen mice given 2 pituitaries and 2 hypothalami, 2½ months earlier.

Comparable elements were not seen in the neighborhood of pituitaries recovered from 11 control mice, given brain surface instead of hypothalamus at the time of pituitary implantation over 10 weeks earlier. ($P < .04$). It is methodologically important to note that the examination for elements resembling ganglion cells was done in all instances on coded sections by the same person (E.H.), who had detected such cells during the earlier routine examination of some of the slides.

Subpectoral elements resembling ganglion cells, found 2½ months after hypothalamus-insertion and only in animals so treated, are of interest in several ways. Are such structures functional? If so, are they contributing to the "anti-cancer" effect associated in C female mice with the addition of the hypothalamus at the time of ectopic pituitary isografting (Ref. 5; cf. also Fig. 1)?

The possibility compatible with these findings that hypothalamic elements are not only morphologically demonstrable but physiologically active as well, at 10 weeks post-implantation, is under continued study.

Summary and Conclusions

Breast cancers induced or enhanced by heterotopic pituitary isografts in C and D_8 mice are more often differentiated than undifferentiated morphologically. Squamous metaplasia and papillary carcinoma predominate more often in tumors of C-strain mice than in those of D_8 mice.

Heterotopic pituitary isograft function results in clearly detectable adrenal involvement. Corticosterone contents of the adrenals from mice bearing such isografts are significantly higher than the corresponding values from control mice during the period from the third to the sixth month after isografting; this may also be the case in mice with overt breast cancer. The physiopathologic mechanisms involved await further study. Such information may contribute to the ultimate modification of pituitary function, so that undesired effects exerted by this gland are eliminated while as many of its important functions as feasible are saved.

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Medical School News

RESEARCH GRANTS AWARDED

Seven Minnesotans are sharing in awards totalling $1,869,027 which the U. S. Public Health Service has made available to 94 institutions for research in various aspects of aging. Research of direct and indirect application to aging is being supported in medicine, biology, psychology, and sociology. The grants are part of a continuing Public Health Service study into health-related characteristics of the aging process.

University of Minnesota scientists participating are Dr. Carlos Martinez and Dr. J. B. Aust, ($43,105—Studies on Tolerance for Homologous Normal and Neoplastic Tissues); Dr. John E. Anderson, ($15,110—Adult Adjustment and Early Experience and Behavior); Dr. A. B. Baker and Dr. Anthony Iannone, ($18,768—A Clinical Pathological Study of Degenerative Cerebroarterial Disease); and Dr. Ernst Simonson and Dr. Lee D. Cady, Jr., ($8,188—Electrocardiographic and Constitutional Correlation).

The U. S. Public Health Service has published a list of 150 long-term research projects concerned with viruses as a possible cause of cancer. More than $5.6 million in research funds has been made available through the National Cancer Institute.

Minnesotans sharing in the program include Dr. John J. Bittner, (The Genesis of Mammary Cancer in Mice); Dr. William D. Kelly, (Effect of Bone Marrow Suspensions on Hodgkin's Disease); Dr. William F. Scherer, (Cells and Nondestructive Viruses); and Dr. J. T. Syverton, (Role of Viruses in Human and Animal Neoplasms.) All are members of the faculty of the Medical School.

The University of Minnesota School of Public Health has been awarded $21,837.00 by U.S.P.H.S. for a project in graduate training. Twenty two other schools received similar grants last Fall to strengthen and expand graduate public health training with particular emphasis on improving curricula to meet the needs of changing and emerging public health programs.

Departmental News

RADIOLOGY

Dr. Richard G. Lester, Associate Professor of Radiology, has been named Professor and Head of the Department of Radiology at the Medical College of Virginia, Richmond, Va. He will assume his new post April 1, 1961 after completing nearly seven years on the faculty at the University of Minnesota.

RICHARD G. LESTER

Dr. Lester is a 1948 graduate of the Columbia University College of Physicians and Surgeons, receiving advanced medical training at Stanford University Hospital, and the U.S. Air Force. He joined the faculty of the University of Minnesota Medical School on July 1, 1954.

A member of the American College of Radiology, and certified by the American Board of Radiology, Dr. Lester is 35 years old, married, and has two children.

UROLOGY

Dr. C. D. Creevy, Professor of Surgery and Director of the Division, delivered the Belfield Memorial Lecture before the Chicago Urological Society on Nov. 30.

BACTERIOLOGY

Dr. William F. Scherer, Professor, has been named to the Board of Trustees of the American Type Culture Collection.

The third annual F. G. Novy Lecture at the University of Michigan was presented December 15, 1960 by Dr. Herman C. Lichstein, Professor of Bacteriology. Title of his lecture was "Physiological Control Mechanisms in the Bacterial Cell."

PHYSIOLOGY

Dr. Maurice B. Visscher, Professor and Head of the Department, lectured Nov. 17 on "Education Today for Medicine Tomorrow," at Albany Medical College of Union University, Albany, N. Y. He also delivered the "Lilly Lecture" Nov. 22 at the Eli Lilly Company, Indianapolis, Ind.

PROCTOLOGY

Dr. William C. Bernstein, Professor and Director of the Division, lectured at the postgraduate teaching course of the California Academy of General Practice, held Nov. 10-12, 1960 in Las Vegas, Nev.

UNIVERSITY HEALTH SERVICE

Prof. Richard G. Bond, public health engineer, is on a leave of absence for three months while accepting a travel fellowship from the World Health Organization. He is studying environmental sanitation conditions in India and the Far East, and will return to Minneapolis about Jan. 15, 1961. His mission is to help W.H.O. determine the kind of training which personnel from Far Eastern countries might profit from most when they come to the University's School of Public Health for graduate study.

RICHARD G. BOND

Dr. John Rutherford Anderson is joining the University Health Service medical staff Jan. 1, 1961 as a full time physician. He is a 1958 graduate of the University of Minnesota Medical School.

OTOLARYNGOLOGY

Dr. Robert E. Priest, Clinical Professor of Otolaryngology, is a Vice President of the American Laryngological, Rhinological, and Otological Society, and Chairman of its Middle Section which will meet in Minneapolis Jan. 27–28, 1961. The Department of Otolaryngology will sponsor a one-day Continuation Medical Education Course in Otolaryngology for Specialists on Jan. 26 at the University.

Dr. Victor Hildvard, who was a Fellow in Otolaryngology at Minnesota in 1954–57, has been appointed Head of the Department of Otolaryngology at the University of Colorado Medical School.

Dr. Joan Davison is currently doing graduate study in the Department of Biochemistry at the University of Minnesota, in preparation for research work related to the problems of otosclerosis. Dr. Davison completed a year of graduate study at Minnesota in 1960.

Dr. Albert Hohmann, who was a Fellow in Otolaryngology at Minnesota 1955–58, will join the Department in 1961. He is currently studying in the Otological Research Laboratory in Henry Ford Hospital, Detroit, under a fellowship from the National Institutes of Health.

PHYSICAL MEDICINE AND REHABILITATION

Dr. Frederic J. Kottke, Professor and Head of the Department, gave two lectures, "Rehabilitation of the Cardiac Patient," and "Concepts of Rehabilitation," at the University of Miami School of Medicine Dec. 12–16.

Mr. Clarence A. Sicard, Supervisor of Occupational Therapy, was official delegate of the University of Minnesota Rehabilitation Center to the annual meeting of the American Occupational Therapy Association Nov 14–18 in Los Angeles, Calif.

Dr. Glenn Gullickson and Dr. Romine E. Matthews were MRC delegates to the annual meeting of the Conference of Rehabilitation Centers and Facilities Dec. 2–6 in Berkeley, Calif. Dr. Gullickson is a member of the Board of Directors.

New appointees in the Department include Bertrum W. Griffis, vocational counselor; Elaine Davidson, research assistant; Karyne Quast, senior occupational therapist; Spencer L. Robnik, research assistant; Rita M. Getzel, and Sharon K. McGovern, physical therapists, and Karol K. Orr, occupational therapist.

JOHN H. MOE

ORTHOPEDIC SURGERY

Dr. John H. Moe, Clinical Professor and Director of the Division, spoke on "Non-Operative Treatment of Scoliosis" at the Southern Medical Association meetings in St. Louis, Mo., Oct. 31–Nov. 3, 1960.

SURGERY

Dr. Donald J. Ferguson became a Professor of Surgery Oct. 1, 1960 at the University of Chicago School of Medicine. He was Chief of Surgery at the Veterans Administration Hospital in Minneapolis since 1954, and is known for research in pulmonary arteriosclerosis. He worked extensively in use of the magnetic flow meter, an instrument to measure and record blood flow, especially in artery surgery.

Dr. Ferguson is a 1943 graduate of the University of Minnesota Medical School, and received a Ph.D. in surgery here in 1951. His undergraduate education was at Yale University.

His successor as Chief of Surgery at the Minneapolis V.A. Hospital is Dr. William D. Kelly, one of his former associates.

SURGERY

Dr. Richard C. Lillehei, Assistant Professor, and Dr. Robert L. Replogle, Surgery Intern, were in London, England Oct. 19–20, 1960, as invited guests to deliver papers before the International Federation of Surgical Colleges and Kindred Societies. Meeting at the headquarters of the Royal College of Surgeons, Dr. Lillehei spoke on "A Comparison of Whole Spleen Autografts, Shielded Spleen, and Whole Spleen Homogenates in the Treatment of Irradiation Sickness in Dogs." He is a Markle Scholar and a 1952 graduate of the University of Minnesota Medical School.

Dr. Replogle, a 1960 graduate of Harvard Medical School, spoke on "Renal Circulatory Response to Cardiopulmonary Bypass," a report on work done at Boston, Mass. Children's Hospital with his co-author, Dr. Robert E. Gross.

LABORATORY MEDICINE

Dr. Rex B. Conn, Jr., was appointed Director of the Division of Laboratory Medicine at the West Virginia University Medical School effective January 1, 1961. He will also hold the rank of Assistant Professor, Department of Medicine.

REX B. CONN, JR.

Dr. Conn has been an Instructor in Laboratory Medicine at Minnesota after joining the staff as a Fellow in 1957. He interned at Minnesota following graduation from Yale University Medical School in 1953, and received a Research Degree from Magdalene College, Oxford University in 1955. He was also Chief Resident physician at Minnesota for two years.

Dr. Conn is 33 years old. His family includes a wife and three children.

PEDIATRICS

Dr. Paul M. Ellwood, Clinical Assistant Professor, was installed recently as President of the Conference of Rehabilitation Centers and Facilities, a society representing 110 institutions in the U. S. and Canada. Dr. Ellwood is medical administrator of the Elizabeth Kenny Rehabilitation Institute, Minneapolis.

PHARMACOLOGY

Dr. Wilbur M. Benson, former professor in the department, has been appointed Director of Pharmacology in the Mead Johnson & Co. Research Division, Evansville, Ind. He is now occupying his new post.

Medical Foundation News

PROFESSORSHIPS APPROVED

The Board of Regents of the University of Minnesota has announced the appointment of Dr. Maurice B. Visscher as Distinguished Service Professor of Physiology, and of Dr. Owen H. Wangensteen as Distinguished Service Professor of Surgery.

The supplemental academic recognitions were recommended by President O. Meredith Wilson of the University, and approved by the Board of Regents following the bestowal of Minnesota Medical Foundation awards upon the medical educators on Sept. 26, 1960. Dr. Visscher is Professor and Head of the Department of Physiology and Dr. Wangensteen is Professor and Chairman of the Department of Surgery.

The Medical Foundation named Drs. Visscher and Wangensteen recipients of its first Distinguished Service Awards for exceptional performance in teaching and research. Each award carried a prize of $5,000 annually until retirement from the faculty of the Medical School. Funds to finance the awards were supplied totally by the Modern Medicine Publications Foundation (for Dr. Visscher), and the Phillips Foundation (for Dr. Wangensteen), both of Minneapolis.

Expansion of the faculty awards program is planned by the Medical Foundation as rapidly as sponsors become available.

CHRISTMAS CONTRIBUTION

Receipt of a $1,500 contribution for heart disease and cancer research programs has been announced by the Minnesota Medical Foundation. The funds were donated by the Schwan Ice Cream Company of Marshall, Minn. in lieu of 1960 Christmas gifts to customers.

The Minnesota Medical Foundation extends Best Wishes for the Holiday Season to all of its Members and Friends.

Alumni News

CLASS OF 1959

The Minnesota Medical Foundation recently polled the 113 members of the Medical School's 1959 graduating class, asking information on their individual post-internship plans.

Results are interesting because of the keen interest shown in what types of practice our current graduates are tending to follow in medicine, and because the Medical Foundation maintains an address file through which recent graduates can locate their scattered classmates and friends.

All of the class responded and 59 were headed for advanced medical training as residents and fellows; thirty four were to begin the general practice of medicine (all but three in Minnesota), and twenty others were either in military service or occupied in some other type of practice.

The class list:

Raymond Albrecht is in general practice in St. Paul, Minn. His address is 1166 Randolph Ave.

Dale L. Anderson is in general practice in North St. Paul, Minn. His address is 147 7th Ave. N.W.

F. D. Anderson is taking a residency in obstetrics and gynecology at the Salt Lake County General Hospital, Salt Lake City, Utah.

Bradley E. Appelbaum is a resident in pediatrics at Illinois Research and Educational Hospital, Chicago, Ill.

Frank R. Arko is a general practitioner in association with the Mesaba Clinic, Hibbing, Minn.

Thomas R. Arlander is a resident in pathology at the University Medical Center, Department of Pathology, Jackson, Miss.

John W. Aughenbaugh is taking an obstetrics and gynecology residency in University of Minnesota Hospitals, and lives at 78 W. Wentworth Court, Minneapolis.

John D. Banovetz is a resident in surgery at the Minneapolis Veterans Administration Hospital.

Richard P. Bendel is engaged in the general practice of medicine at 8030 Minnetonka Blvd., St. Louis Park, Minn.

William E. Bernstein is a resident in psychiatry at the University of Colorado Medical Center, Denver, Colo.

Ronald N. Berry is a resident at the Mayo Clinic, Rochester, Minn. His address is 1115 Seventh Ave. SW, Rochester, Minn.

Phillip Bloom is at the Veterans Administration Hospital in Portland, Oregon taking a residency in internal medicine.

Ausma S. Blumentals is taking a residency in St. Paul. She lives at 973 Bradley Street, St. Paul, Minn.

William J. Broussard has entered military service. He can be reached c/o A. A. Broussard, Pecan Island Route, Box 12, Kaplan, Louisiana.

John B. Campbell is taaking a pediatric residency at Tripler General Hospital, Honolulu, Hawaii.

Carl E. Christenson is in the general practice of medicine at Clinton, Minn.

Wilfred A. Corson is taking a residency in Washington state. His address is 746 N. 148th Street, Seattle, Wash.

Robert T. Dale is taking a residency in Minneapolis. His address is 5544 Tenth Ave. S., Minneapolis, Minn.

Allan D. Davidson is in the general practice of medicine at Caledonia, Minn., in association with Dr. J. J. Ahlfs (Med. '22).

Michael W. Davis is a resident in surgery at Veterans Administration Hospital, Minneapolis, Minn.

Richard A. DeRemee is with the U.S. Army medical corps stationed at Ft. Myer, Va.

Thomas E. Dredge, Jr. said he planned to enter general practice but the location was uncertain.

David K. Drill is a resident in orthopedics at Minneapolis General Hospital, Minneapolis, Minn.

Philip L. Eckman is taking a research fellowship in the Department of Surgery, University of Illinois, Chicago, Ill.

Franklin Elevitch is a resident in pathology at the University of California Medical Center, San Francisco, Calif.

Carl G. Evers is taking a pathology residency at the University Medical Center, Jackson, Miss.

W. Daniel Flory and his wife, **Sonja K. M. Flory,** have settled at 5545 34th Ave. S., Minneapolis, Minn., while Dan undertakes a residency in internal medicine at the Minneapolis Veterans Administration Hospital. They are parents of their first child, Kathryn, born Sept. 6, 1960. Sonja plans to begin a part time practice in 1961.

Jerome C. Fluth was in general practice in Minenapolis last summer, and hoped to become a medical missionary in British Cameroons, West Africa, late in 1960.

Bradford E. Friedrich is taking a residency at Highland Alameda County Hospital, Oakland, Calif.

Arthur J. Gerdes is taking a residency at King County Hospital, Seattle, Wash. His address is 326 Ninth Ave., Seattle.

Benjie L. Goldfarb is a resident in internal medicine at Peter Bent Brigham Hospital, 721 Huntington Ave., Boston 15, Mass.

Lawrence M. Greenberg is taking a residency in pediatrics at the University of Minnesota Hospitals, Minneapolis, Minn.

Mark D. Hafermann is a medical fellow at the Mayo Foundation, Rochester, Minn.

Kenneth Halverson has begun the general practice of medicine at Chester, Mont.

Richard N. Harner is taking a residency in neurology at the University of Minnesota Hospitals, Minneapolis, Minn.

John M. Hendrickson is in general practice in Red Lake Falls, Minn. in association with Dr. Lester N. Dale. (Med. '43).

James P. Herberg is in general practice in Altoona, Pennsylvania. His address is 1303 Ninth Street.

John A. Hetzler is in general practice at Pine Island, Minn.

John A. Hiatt is in general practice in Minneapolis. His address is 2645 First Avenue S., Minneapolis 8, Minn.

Charlotte Weeks Hill is taking a residency in ophthalomology at the Veterans Administration Hospital, Milwaukee, Wis.

Leslie H. Hoium entered private practice in St. Paul. His address is 675 Stuber Court, Roseville, Minn.

Jack O. Hubbard has begun a residency in orthopedics at the Mayo Clinic, Rochester, Minn. His address is 2301 Elton Hills Drive, Rochester, Minn.

James Janecek is taking a residency in psychiatry at the University of Minnesota Hospitals. His address is 611 SE Delaware Street, Minneapolis, Minn.

Carl E. Johnson is taking a residency in psychiatry at the Veterans Administration Hospital, The Menninger Foundation, Topeka, Kansas.

Franklin L. Johnson has gone into the general practice of medicine in association with the Morgan Park Medical Clinic, Duluth, Minn.

Thomas A. Johnson is in military service and is attached to the U.S. Public Health Service Health Unit, Atomic Energy Commission, Germantown, Maryland.

Morton C. Kane is taking a residency in Minneapolis. He lives at 5453 Girard Ave. So., Minneapolis, Minn.

Helen M. Kelly is a resident physician at St. John's Hospital, St. Paul, Minn., and lives at 372 W. Moreland, St. Paul.

Stephen A. Kieffer is taking a residency in radiology at the University of Minnesota Hospitals, Minneapolis, Minn.

Lowell L. Kvam has entered general practice in St. Paul. His address is 1031 Payne Ave., St. Paul, Minn.

Miles I. Lane is engaged in the general practice of medicine with offices located at the Alla-Bar Shopping Center, White Bear Lake, Minn.

Russell H. Larsen has gone into general practice in Wheaton,

John S. Leighton has begun general practice in association with Dr. G. A. Schissel and Dr. J. J. Pattee at 5617 Hampshire Ave. N., Minneapolis, Minn.

Arthur T. Lindeland is taking a residency in internal medicine at the Minneapolis General Hospital, Minneapolis, Minn.

Thomas Litman is taking a residency in surgery at the Minneapolis Veterans Administration Hospital and can be reached at 5200 Clinton Ave. S., Minneapolis, Minn.

Everett W. Lovrien is a resident in pediatrics at Children's Hospital, Washington, D. C.

Douglass A. Lowe is a resident in psychiatry at the University of Minnesota Hospitals. His address is 4200 Oakdale Ave., Minneapolis 16, Minn.

Richard O. Lundborg is a Captain in the U.S. Army Medical Corps. He is with the Fourth Infantry Division, Fort Lewis, Wash.

Charles B. Lundquist has entered general practice. His address is 421 Ruby Drive, West St. Paul, Minn.

Charles J. Martell is now associated with Donner Laboratories, University of California, Berkeley, Calif.

Ray M. Martinson has entered general practice in association with the East Range Clinic, Virginia, Minn.

Daniel L. Maryland is taking a residency in ophthalmology at the Detroit Receiving Hospital, Detroit, Michigan.

Paul D. Mayer lives at 26 Sioux Valley Drive, Cherokee, Iowa. He is taking a residency in psychiatry.

Charles B. McCreary is taking a surgery residency at the St. Louis County Hospital, Clayton 5, Missouri.

Robert C. McGee is a resident in orthopedics at the Veterans Administration Hospital, Minneapolis, and lives at 5621 Elliot Ave. S., Minneapolis 17.

James A. McKinnon is a Captain in the U.S. Army Medical Corps. His address is 317 Tactical Hospital, A.P.O. New York, N. Y.

Richard A. Meland is also in the U.S. Army Medical Corps, stationed at Brooke General Hospital, Fort Sam Houston, San Antonio, Texas, where he is taking a residency in radiology.

Melvin E. Meyer is a medical officer in the U.S. Navy, stationed at the Navy Recruiting Station, Ashland, Kentucky.

Mark A. Muesing is located in Tyler, Minnesota, where he is in general practice.

Thomas F. Mulrooney is in the U.S. Navy, where he is general medical officer on the staff of a destroyer squadron. His address is c/o Destroyer Division 72, F.P.O., San Francisco, Calif.

Rodger K. Nelson is in military service.

Ronald J. Nelson, class president, is now at the Fuller Theological Seminary, 135 N. Oakland Ave., Pasadena, Calif., where he is training to become a medical missionary.

Thomas O. Nichols is in general practice in St. Paul. His address is 1115 York Ave., St. Paul 6, Minn.

David J. Nielsen is engaged in general practice of medicine in association with the Broadway Suburban Clinic, Minneapolis. His address is 3801 27th Ave. N., Minneapolis.

Vern Olmanson has joined his father, Dr. E. G. Olmanson, and brother, Dr. Donald Olmanson, in the St. Peter Clinic, St. Peter, Minn. Vern is in general practice.

Duane L. Orn has entered private practice in Rochester, Minn. He lives at 1605 7th Ave. N.W., Rochester.

Richard R. Oslund is taking a residency in radiology at St. Joseph Mercy Hospital, Pontiac, Michigan.

James J. Plorde is a resident in internal medicine at the University of Washington, Seattle, Washington. His address is 746 N. 148th Street, Seattle.

Robert L. Powers is in general practice in St. Paul, Minn., located at 1124 Larpenteur Ave.

William E. Prickman is taking a residency in ophthalomology at the Mayo Clinic. His address is 1011 Tenth Street SW, Rochester, Minn.

Barbara Meyer Puumala and her husband, **Ricard R. Puumala,** are both in general practice at Cloquet, Minn.

Gerald Ratinov is taking a residency in neurology at the University of Minnesota Hospitals.

John D. Riley is in the U.S. Navy, taking training at the School of Aviation Medicine, Pensacola, Fla. His mailing address is Stephen, Minn.

Lawrence Ringhofer is in the Navy, stationed at the U.S. Navy Recruiting Station, Indianapolis, Ind. His address is 3537 Lowry Road, Indianapolis.

Franklin D. Roller is taking a residency at the U.S. Public Health Service Hospital, San Francisco, Calif.

Homer H. Russ is in general practice at LeCenter, Minn.

Milton F. Sadd is taking a residency in radiology at the State University of Iowa Medical School, Iowa City, Iowa.

Robert L. Sadoff is taking a residency in psychiatry at the University of California at Los Angeles, Los Angeles 24, Calif.

Harry P. Santrizos is practicing industrial medicine as a physician with the Chrysler Corporation. He lives at 27740 Shagbark Drive, Southfield, Michigan.

Dean T. Schamber is a Captain in the U.S. Army Medical Corps, and is taking a residency in urology at Letterman Army Hospital, San Francisco, Calif.

William R. Schmalhorst has joined the Meeker Clinic, in Meeker, Colorado, as an associate in general practice.

Edward Seljeskog is taking a residency at Minneapolis General Hospital, Minneapolis, Minn.

Lee A. Simso is in military service, and can be reached c/o A. R. Simso, 5740 Eleventh Ave. S., Minneapolis, Minn.

George Skaff is taking a residency in California. His address is 319 Fulton Street, Redwood City, Calif.

Darline D. Smith is taking a residency in internal medicine at Highland Alameda County Hospital, Oakland, Calif.

William N. Spellacy is taking a residency in Minneapolis. His address is 711 Marigold Terrace, Minneapolis 21, Minn.

Herbert S. Strait has become a partner in a clinic at Waconia, Minn., where he is engaged in general practice.

Thomas O. Swallen is a resident in pathology at the University of Minnesota Hospitals. His address is 38 St. Mary's Ave. SE, Minneapolis, Minn.

Richard L. Swanson is a resident in ENT at the University of Oregon Hospital. His address is 9466 N. Woolsey Ave., Portland, Oregon.

Robert L. Telander is a resident in surgery at Minneapolis General Hospital.

Byron A. Teska is a staff physician at the University of Minnesota Health Service.

Gail Wesley Thompson is taking a residency in California, and lives at 416 S. 38th Street, Richmond, Calif.

James R. Thompson is taking a general surgery residency at the Duluth Clinic, Duluth, Minn.

Roy Toyama is engaged in research at the University of Minnesota Hospitals. He lives at 2419 Lincoln St. NE, Minneapolis, Minn.

Gerald F. Tuohy is taking a residency in anesthesiology at the Mayo Clinic. His address is 1442 Damon Court, Rochester, Minn.

Richard L. White is a resident in surgery at the Boston City Hospital, Boston, Massachusetts.

Alvin L. Wiens received a commission in the U.S. Public Health Service, and is engaged in general practice of medicine as the staff physician at the Federal Prison, Sandstone, Minn.

John A. Wilson is taking a residency in surgery at Minneapolis General Hospital, Minneapolis, Minn.

Robert A. Wymore is commissioned in the U.S. Public Health Service and is assigned in Portland, Oregon. His address is 7120 SE 66th Ave., Portland 6, Ore.

Elmer W. Ylitalo is taking a residency in general surgery at Brooke Army Medical Center, Ft. Sam Houston, Texas.

John F. Zachman is engaged in general practice of medicine at Melrose, Minn., with his father, Dr. A. H. Zachman.

Alumni Notes

◆ **1900**

W. H. Valentine, who has practiced medicine in Tracy, Minn., during his entire medical career spanning 60 years, recently celebrated his 85th birthday.

◆ **1909**

Henry W. Meyerding, Emeritus Professor of Surgery at the Mayo Foundation, spoke on orthopedic surgery, Sept. 26, before the Manitoba Medical Association in Winnipeg, Canada. He has just completed a term as president of the International College of Surgeons.

Alfred L. Vadheim, pioneer southwestern Minnesota physician, now resides in Brookings, S.D. He has completed fifty years of medical practice in the Tyler, Minn. area.

◆ **1920**

J. Arthur Myers participated in a Symposium on "An Orientation in Geriatric Management" in Philadelphia, Pa. on October 16. The Symposium was presented by five medical schools of the Philadelphia area and the county medical society. Dr. Myers also addressed the McLean County Medical Society in Bloomington, Ill., on November 15.

◆ **1921**

Burton C. Ford, and associates, **Drs. K. A. Peterson (Med. '42),** and **J. E. Eckdale (Med. '40)** of the Plaza Medical clinic in Marshall, Minn., announce the association of **Dr. Robert Phelps,** a recent graduate of the Nebraska Medical College in Omaha, Nebraska.

Ralph T. Knight of Minneapolis was named recipient of the 1960 Distinguished Service Award of the American Society of Anesthesiologists. He was president of the A.S.A. in 1953, also serving as president of the state group, and is often referred to as the "First Anesthesiologist in the State of Minnesota." Dr. Knight retired recently after 40 years of practice in his specialty. He is professor emeritus of Anesthesiology at the University of Minnesota Medical School, and is being honored by his colleagues who are now establishing the Ralph T. Knight Anesthesiology Research Laboratory in the new Bio-medical library at the University of Minnesota.

◆ **1922**

Dr. Gordon R. Kamman, St Paul, a founder of the Minnesota Medical Foundation, was in the Far East on a tour last summer. While in Bangkok, Thailand, he met and visited with **Dr. Thomas Dooley,** head of MEDICO, who practices in Laos.

Harold G. Reineke, professor of clinical radiology at the University of Cincinnati College of Medicine, was installed as president of the American Roentgen Ray Society at its 1960 annual meeting in Atlantic City, New Jersey.

◆ **1926**

Erling Ostergaard, formerly of Evansville, Minn., has begun pracice near Chinle, Ariz., among Indians at the Rock Point Mission there.

◆ **1927**

William C. Heiam is the new Chief of Staff at Cook Community Hospital, Cook, Minn. **Dr. Floyd J. Swenson (Med. '58)** is secretary.

◆ **1928**

John F. Briggs, St. Paul, was elected first vice president of the American College of Chest Physicians at its 26th annual meeting this year in Miami Beach, Fla.

◆ **1929**

Hubert L. Anderson, a Captain in the U.S. Navy Medical Corps, was recently transferred from Adak, Alaska, and is now on duty at the naval facility in Twentynine Palms, Calif.

E. G. Hubin, Sandstone, Minn. physician, has been appointed Coroner of Pine County.

♦ **1930**

C. H. Holmstrom and **Dr. E. E. Pumala (Med. '41)** are now occupying their new Warren Clinic building in Warren, Minn.

♦ **1931**

Ralph D. Hanover and **Dr. Charles B. Will (Med. '38),** partners in practice at International Falls, Minn., announce the addition of a new physician to their practice. He is **Dr. George M. Crow,** recently discharged from the U.S. Army Medical Corps.

♦ **1932**

Among new officers named recently by the Minnesota State Obstetrical and Gynecological Society were **Dr. Martin O. Wallace,** Duluth (**Med. '32**), vice president; and **Dr. Edgar G. Ingalls,** Minneapolis (**Med. '41**), Assistant Secretary-Treasurer. New President of the organization is **Dr. Charles H. McKenzie,** Minneapolis.

Jan H. Tillisch, consultant in internal medicine at the Mayo Clinic, was appointed to an 11-man Medical Advisory Council by the Federal Aviation Agency, Washington, D.C. All are prominent in field of aviation medicine, and will assist in developing, operating and coordinating FAA's medical program.

♦ **1934**

Irvin Kerlan of Washington, D.C. has been named in the Authors and Writers WHO'S WHO, recently published in London, England.

♦ **1935**

Charles W. Vandersluis has opened an office for the practice of medicine at Minneota, Minn. He formerly practiced in Bemidji, and Warren, Minn.

♦ **1936**

Harold G. Scheie has been named chairman of the Department of Ophthalmology at the University of Pennsylvania School of Medicine. He is a professor of ophthalmology at the Philadelphia school.

♦ **1937**

Lyle J. Hay was elected president of the Minnesota Division, American Cancer Society. He has been a member of the Board of Directors of the Division for ten years. He is director of the intern and resident training programs at St. Barnabas and Swedish Hospitals, Minneapolis, and is a clinical professor of Surgery at the University of Minnesota Medical School. **Dr. Philip F. Eckman,** Duluth (Med. '22) was named third vice president of the state cancer organization.

John T. Pewters, Minneapolis physician and surgeon, was named a member of the Minneapolis Board of Public Welfare. He is on the staff at Abbott Hospital, and was Chief of Staff there from 1957 to 1959.

◆ **1938**

Albert A. Bodaski is now practicing in Harmony, Minn., following a move from Tyler, Minn., where he practiced eight years.

◆ **1945**

Chester A. Anderson, Hector, Minn. physician, announces his association in practice with **Dr. James Rud,** recent graduate of University of Nebraska Medical School.

◆ **1946**

Dale Furnell, an orthopedic surgeon, has assumed the practice of **Dr. Donald L. Olson (Med '50),** Fargo, N.D. surgeon who died in December 1959. Dr. Furnell was most recently associated with the clinical research division of G. D. Searle & Co., Chicago. He practiced in St. Paul, Minn. from 1949 to 1954, spent two years in the U.S. Army Medical Corps, and then took special orthopedic training at Georgetown University and Northwestern University.

Robert V. Hodapp has rejoined the Lakeland Medical Center of Willmar, Minn. following completion of pediatric residency training at the University of Minnesota.

◆ **1949**

Eugene I. Saxon is a heart surgeon at the Cedars of Lebanon Hospital, Los Angeles, California.

◆ **1950**

Donald F. Holm is at the University of Minnesota Hospitals taking a residency in radiology. He practiced in Benson, Minn. for seven years in association with **Dr. Robert Nelson,** before starting his residency.

Roger S. Johnson is now a Fellow in Surgery at Ancker Hospital in St. Paul, Minnesota. He was formerly associated with the Minnetonka Clinic, Excelsior, Minn.

Gerald E. Nelson, Jr. is now an associate of **Dr. G. T. Schimelpfenig (Med. '28)** in the Stoughton Avenue Clinic in Chaska, Minn.

◆ **1952**

Louis A. Buie, Jr., Mayo Clinic, is recipient of the Howard K. Gray Travel Award for 1961 through the Mayo Foundation.

◆ **1953**

Carl R. Heinzerling, Chaska, Minn. physician, announces his association in practice with **Dr. Patrick J. Adams,** a recent

graduate of the Stritch Medical College of Loyola University in Chicago.

Joseph W. Teynor has opened an office in Minneapolis following completion of a fellowship in Otolaryngology at the Mayo Foundation, Rochester, Minn.

◆ **1955**

Reno W. Backus, medical director of the Nopeming, Minn. Sanatorium, has been appointed to the Medical Advisory and Nominating Committees of the Minnesota Tuberculosis and Health Association.

Kenneth H. Nelder was appointed a Fellow in Dermatology at the Mayo Foundation, Rochester, Minn.

James R. Schuft has joined **Dr. Robert M. Watson (Med. '43)** in practice at Morris, Minn. Dr. Schuft was formerly in general practice in California, following completion of two years duty with the U.S. Air Force.

◆ **1956**

Richard Engwall, Ivanhoe, Minn. and **Dr. Mark Muesing, (Med. '59),** formerly of Duluth, have joined **Dr. C. P. Johnson** in the practice of medicine at Tyler, Minn.

Roger Hallgren has established a practice in Belle Plaine, Minn., in association with **Dr. H. M. Juergens (Med. '20).** He interned at Milwaukee, Wis. County Hospital, and completed three years in the medical corps of the U.S. Air Force.

James R. Pluth has been appointed a Fellow in Surgery at the Mayo Foundation, Rochester, Minn.

Memorial Gifts

Memorial gifts to the Minnesota Medical Foundation have been received recently in memory of:

Mrs. Ella Lounberg
Minneapolis, Minn.

Dr. Calixto Torres
Bogota, Colombia

Memorial contributions are a practical means of honoring the memory of a friend or loved one, while helping the Minnesota Medical Foundation in the advancement of medical education and research. Appropriate acknowledgements are promptly sent to both donor and family of the deceased.

ALUMNI DEATHS

Dr. George Francis Drew (Med. 1900) passed away June 8, 1960 at Mercy Hospital, Devils Lake, N. D. Death was caused by secondary complication of diabetes. He was 85 years old, and had been Devils Lake health officer for many years.

Dr. James T. Larson (Med. '29) died September 9, 1960. He was 56 years old and had practiced 16 years in South St. Paul, Minnesota, where he was medical doctor for Swift and Co. He was a native of North Dakota and took his internship at Minneapolis General Hospital.

Dr. Daniel F. Pennie (Med. '14) died Oct. 3, 1960 in Duluth, Minn. He was 79 years old and a Life Member of the St. Louis County Medical Society.

Dr. Nordahl P. Peterson (Med. '28) died Nov. 15, 1960, following a cerebral hemorrhage suffered while witnessing a University of Minnesota football game three days earlier. He was 72 years old and a member of the state board of medical examiners. Prior to entering medical school he was a teacher and school superintendent in southern Minnesota for several years.

Dr. William G. Richards (Med. '04) died July 4, 1960 in Billings, Mont., of arteriosclerotic heart disease. He was a specialist certified by the American Board of Internal Medicine and fellow of the American College of Physicians. Death came at the age of 90.

Dr. N. Wells Stewart (Med. '26) died August 19, 1960, following a heart attack. He was 61 years old and practiced medicine in Lead, South Dakota.

Dr. Bernard Marlin Urenn (Med. '35) died June 26, 1960 in Fargo, N.D. He was 55 years old, and had served as an intern and resident at Minneapolis General Hospital. He was associated with St. John's and St. Luke's Hospitals in Fargo, and was a past president of the North Dakota Obstetrical Society.

Dr. Melvin Vik (Med. '24) died Sept 16, 1960 in Cambridge, Minn. He was assistant superintendent of the Cambridge State School and Hospital, and director of its physical medicine and rehabilitation program. Dr. Vik was a native of Clarkfield, Minn. He practiced in Onamia, Minn., and had also served on the staff at Anoka State Hospital. He was 63 years old at the time of his death.

Coming Events

University of Minnesota Medical School

TENTATIVE LIST OF CONTINUATION COURSES FOR PHYSICIANS
1960-1961

University of Minnesota
Center for Continuation Study

January 3-7 . . .	Introduction to Electrocardiography for General Physicians
January 26-28 . .	Otolaryngology for Specialists
February 6-8 . . .	Anesthesiology for Specialists
February 13-18 . .	Neurology for General Physicians and Internists
Feb. 27 March 1 . .	Pediatrics for General Physicians and Specialists
March 13-15 . . .	Allergy for General Physicians and Specialists
March 17-18 . . .	Trauma for General Physicians
April 17-19 . . .	Internal Medicine for Internists
April 20-22 . . .	Otolaryngology for General Physicians
May 1-3	Ophthalmology for Specialists
May 8-10	Gynecology for General Physicians
May 11-13 . . .	Surgery for Surgeons
May 15-19 . . .	Proctology for General Physicians
June 1-3	Office Psychotherapy for General Physicians
1960-61 all year . .	Cancer Detection for General Physicians

The University of Minnesota reserves the right to change this schedule without notification.

Courses are held at the Center for Continuation Study or the Mayo Memorial Auditorium on the campus of the University of Minnesota. Usual tuition fees are $30 for a two-day course, $50 for a three-day course, and $75 for a one-week course. These are subject to change under certain circumstances.

Specific announcements are sent out for each course to all members of the Minnesota State Medical Association and to any physicians who request information for a specific course, about six weeks to two months before the date of the course. For further information write to:

DIRECTOR
DEPT. OF CONTINUATION MEDICAL EDUCATION
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UNIVERSITY OF MINNESOTA
MINNEAPOLIS 14, MINNESOTA

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A Word About

Memorial Gifts

The Minnesota Medical Foundation welcomes your memorial contributions when an appropriate occasion arises. Memorial gifts serve the living and pay thoughtful tribute to the memory of a friend or relative.

The Foundation will promptly acknowledge your gifts to both the donor and the family of the deceased. The gift will help finance the Foundation's program for the advancement of medical education and research. The Medical School at the University of Minnesota will be the direct benefactor.

Gifts should be sent to the **Minnesota Medical Foundation, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14, Minn.**