volved auriculotemporal area in the one case tested showed sensitization to intradermal acetylcholine; further sensitization developed after section of the ninth nerve. This development of denervation-sensitization is strong evidence that ninth nerve fibers had been supplying the sweat glands; the syndrome, therefore, in all probability was due to misdirection of regenerated parasympathetic salivary fibers into the sweat glands. This resulted in a dual nerve supply to the sweat glands, similar to that of normal salivary glands. Besides sensitivity to intradermally injected acetylcholine and, presumably, to the parasympathetic humoral effector, there was an enhanced response of the sweat glands to parenteral injection of pilocarpine and to systemic heating, the latter probably depending upon a combination of residual sympathetic innervation and sensitization to the humoral effector.

Sensitization of sweat glands, associated with establishment of a parasympathetic nerve supply, stands in sharp contrast with the complete failure of sweat glands to show sensitization after acute or chronic sympathetic denervation. It is hypothesized, therefore, that establishment of a true parasympathetic nerve supply to sweat glands is associated, perhaps in causal relationship, with appearance of sensitization to a variety of stimuli and that the substance released by parasympathetic salivary nerve endings differs chemically from the cholinergic substance released by cholinergic sympathetic nerve endings.

2020 E. 93rd St. (6) (Dr. Gardner).

SUCCESSFUL HOMOTRANSPLANTATION OF THE HUMAN KIDNEY BETWEEN IDENTICAL TWINS

John P. Merrill, M.D., Joseph E. Murray, M.D., J. Hartwell Harrison, M.D.,
and Warren R. Guild, M.D., Boston

This report documents the successful transplantation of a human kidney from one identical twin to another. The function of the homograft remains excellent 12 months after the operative procedure. Previous attempts at renal homotransplantation, both clinically and experimentally, have been unsuccessful with one exception. In dizygotic cattle twins, a kidney transplant has survived and functioned for at least nine months. Success in this instance, however, presumably resulted from the production of an acquired mutual tolerance to each other's tissues by the mingling of fraternal protein in the common placental circulation.1 Transplantation of the kidney in dogs and other animals rarely maintains function for more than a 10-to-14-day period in spite of vigorous attempts to modify the presumed antibody response that results in rejection of the homograft. Similarly, permanent function has not been maintained in a human renal homograft,2 although in one such instance adequate renal function in a transplanted kidney has persisted for five and a half months.3 The ultimate cause for rejection in such cases is in all probability differences in individual tissue specificity. Since, however, skin homografts between identical human twins have survived permanently,4 it might be expected that renal homotransplantation might also be successful when performed between identical twins. The following case history describes such an event.

REPORT OF A CASE

A 24-year-old, white, single male was apparently in excellent health until 14 months before his first admission to the Peter Bent Brigham Hospital. Except for scarlet fever at age 5 without apparent complications, the history was noncontributory. A few months prior to his discharge from military service, the patient noticed some puffiness about the eyes on awakening in the morning, and on a routine physical examination some elevation of blood pressure was noted. During a five-month study period while at the Boston Public Health Service Hospital, he remained essentially asymptomatic except for epistaxis. Physical examination was negative except for a consistently elevated blood pressure averaging 170/100 mm. Hg. Pertinent laboratory findings included persistent 2 to 3+ proteinuria. The urinary specific gravity was fixed at 1.010 and microscopic hematuria

* A patient whose illness had begun with edema and hypertension was found to have suffered extreme atrophy of both kidneys. Because of the steady worsening of the condition and the appearance of uremia with other unfavorable prognostic signs, transplantation of one kidney from the patient's healthy identical twin brother was undertaken.

Preparations included collection of evidence of monozygosity and experimental transplantation of a skin graft from the twin. During the transfer of the healthy kidney it was totally ischemic for 82 minutes. Evidence of functional activity in the transplanted kidney was obtained.

The hypertension persisted until the patient's diseased kidneys were both removed. The homograft has survived for 11 months, and the marked clinical improvement in the patient has included disappearance of the signs of malignant hypertension.

From the medical and surgical services of the Peter Bent Brigham Hospital and Harvard Medical School.

This study was supported in part by grants from the John A. Hartford Foundation, Inc.; the Medical Research and Development Board, Office of the Surgeon General, Department of the Army; the U. S. Public Health Service; and the American Heart Association.

The table and figure 1 have been omitted from THE JOURNAL and will be included in the authors' reprints.


Identical Twins, Surgery 1: 558, 1937.
and cylindruria with occasional red blood cell casts were found in all urine specimens. Blood urea nitrogen level ranged between 75 and 100 mg. per 100 cc. Hemoglobin level varied between 7 to 10 gm. per 100 cc. Phensulphonphthaline excretion was less than 1% in two hours, and an intravenous pyelogram revealed no dye excretion on either side. X-ray of the chest showed the lungs to be clear and the heart normal in size and shape. After seven transfusions of whole blood he was discharged improved. Five months later he was readmitted again to the Boston Public Health Service Hospital and appeared pale and chronically ill. The blood pressure now varied between 160/80 and 208/120 mm. Hg. The retinal vessels showed narrowing of the arterioles with changes in caliber and occasional arteriovenous compression. Hemoglobin was 67%. Red blood cells per high-power field on spun sediment. There were occasional granular and hyaline casts. The blood nitrogen was 40 mg. per 100 cc. The course was characterized by persistent nausea and vomiting and on the third hospital day he had a generalized convulsion. In the succeeding days he became increasings drowsy, disoriented, and irritable and had several convulsions. Since the patient had a twin brother, it was suggested by Dr. David C. Miller of the U. S. Public Health Service that the possibility of homotransplantation of a kidney should be considered. For the investigation of this possibility, he was transferred to the Peter Bent Brigham Hospital on Oct. 26, 1954.

On admission the initial blood pressure was 140/90 mm. Hg. The patient appeared thin, pale, drowsy, and extremely disoriented. The remainder of the physical examination and laboratory data were consistent with that outlined above. Urine culture grew out Escherichia coli and enterococci. The patient continued to be restless and unable to tolerate oral feedings and became overtly psychotic. On the fourth hospital day he was treated by external dialysis with the artificial kidney for a four-hour period. A good chemical response was obtained and 36 hours later the patient’s sensorium had cleared and he was cooperative and able to take diet and medications by mouth.

On the 15th hospital day, full-thickness skin grafts, 2.5 by 2.0 cm., were taken from the left leg. A control autograft was placed proximally and the homograft was placed 1 cm. distally, allowing a bridge of normal tissue to intervene between the two grafts. On the following day the patient was discharged feeling well on a diet containing 50 gm. of protein and no added salt. He was followed at weekly intervals in the outpatient clinic, continued to show hypertension, and gradually developed the manifestations of congestive heart failure for which he was digitalized with some improvement. On Dec. 12, 1954, however, he was readmitted to the Peter Bent Brigham Hospital because of marked increase in signs and symptoms of his congestive heart failure. Physical examination at this time revealed a blood pressure of 220/146 mm. Hg. There was + pitting edema of the lower legs up to the knees. Bilateral basal rales were present. The liver edge was tender and was palpated 4 cm. below the right costal margin. There was slight periportal edema and the optic fundi now showed a 2 diopter papilledema with exudates and hemorrhages. The heart was enlarged to the left with a loud diastolic gallop heard over the entire precordium. A chest film showed marked cardiomegaly in keeping with a large amount of fluid at the base of the right side of the chest. During the next three days, 350 cc. of turbid, amber fluid was removed from the right side of the chest and the patient received three units of packed red blood cells. He was started on therapy with parenterally given prototoxatrin. On this therapy there was marked clinical improvement.


On Dec. 16, 31 days after the original skin transplant, biopsy study of the homograft was done. In both gross and histological section the transplanted tissue appeared to have survived as normal skin. Because of this evidence of tissue compatibility and ancillary observations suggesting that the twins were monozygotic, on Dec. 23 a normal left kidney was removed from the healthy twin and transplanted to the patient. (Previous hospitalization had disclosed the absence of discoverable disease in the healthy twin and confirmed the presence of two normally functioning kidneys free of infection.)

The postoperative course of the donor was uneventful, and he was discharged on the 14th hospital day. The recipient tolerated the operative procedure well and, soon after the anesthesia was completed, clear urine was obtained from the transplant. Nine days after surgery the intravenous injection of sodium indigotindisulfonate (indigo carmine) showed prompt appearance in good concentration in the urine from the transplanted kidney and no excretion from the patient’s own kidneys. During the course of the following month, the homograft appeared to function well and began to hypertrophy. The patient was discharged from the hospital on the 37th postoperative day. He had gained 11 lb. (5 kg.) and was edema free. The blood urea nitrogen was 14 mg. per 100 cc. and the resting blood pressure, 120/60 mm. Hg. The chest was clear and the heart size normal. The serum carbon dioxide combining power was 25 mEq. per liter and the serum concentrations of sodium, chloride, and phosphate were all within normal limits. The phenolsulphonphthaline excretion was 18% in 15 minutes and 48% in 2 hours. Urinalysis at the time of discharge showed a trace of albumin, 4 to 6 white blood cells, and a rare blood cell per high-power field. Urine culture grew out Proteus vulgaris.

After discharge the patient’s appetite was good and he had no edema, dyspnea, or orthopnea. Blood pressures ranged from 130/80 to 160/88 mm. Hg. The optic disks were normal, and the retinal vessels became normal, although a few old scars persisted in the optic fundi. Because of continued mild bacilluria and pyuria, the patient was begun on methenamine mandelate (Mandelamine) therapy. An excretory urogram performed two months after the renal transplant and homograft showed prompt excretion of the injected dye in good concentration from the transplanted kidney but no detectable excretion by the two diseased kidneys.

Because of persistent mild hypertension, the patient was admitted to the Peter Bent Brigham Hospital for the third time three months after renal homotransplantation. At this time the initial 90/60 pressure was 152/90 mm. Hg. The patient appeared healthy and completely asymptomatic. There had been further gain in weight and muscle development. The palpable mass of the homograft in the right lower quadrant had hypertrophied to half again its original size. The electrocardiogram showed disappearance of the changes of left ventricular hypertrophy. The hematocrit was 48%, blood urea nitrogen 14 mg. per 100 cc., and carbon dioxide combining power 23 mEq. per liter. On the fifth hospital day a left nephrectomy was performed. The kidney weighed only 49 gm. and was covered by a markedly thickened capsule that was fibrosed and scarred. The cortex was markedly diminished in size and the microscopic section showed the majority of the glomeruli to be completely fibroos. The renal interstitium showed diffuse atrophy and a progressive disappearance of tubular elements and the appearance was that of diffuse advanced chronic glomerulonephritis. The patient was discharged 12 days after operation feeling entirely well. However, because of the persistence of mild pyuria and mild labile hypertension, he was readmitted for the fourth time on June 14, 1955, five and a half months after renal homotransplantation. On this seventh hospital day the patient underwent a successful right nephrectomy. The right kidney weighed only 29 gm. and showed the typical changes of advanced diffuse chronic glomerulonephritis with little functioning parenchyma remaining. On discharge the patient’s appetite was good; he had gained more weight and was essentially asymptomatic. At the present time his blood pressure is 125/70 mm. Hg. He weighs 52 lb. (11.3 kg.) more than his initial preoperative weight. He carries on unlimited activity and has no apparent physical disability. The urinary sediment is negative, although his 24-hour protein excretion is 4.5 gm.
COMMENT

The transplantation of functioning tissue from one individual to another of the same species has, with few exceptions, not been successfully accomplished to date. Successful transplantation has been occasionally reported in the case of embryonic thyroid, parathyroid, and in one instance adrenal tissue. The successful transplantation of bone and blood vessels depends not on their survival as living tissues but on their ability to act as bridges over which recipient tissue may grow. Because of its particular structure and the fact that it is frequently transplanted into an avascular field, corneal transplants in man, however, do survive as living tissues in a large percentage of cases. The immune response leading to the rejection of homografts is incompletely understood. Circulating cytotoxic antibodies cannot be measured in significant amounts. It is probable, however, that antibodies to donor tissue are formed by the recipient and that these are removed by the homograft so rapidly that they cannot be measured in the blood. The fact that an antigen-antibody reaction is responsible for the rejection of homografted tissue and that this response can be modified is suggested by work in which successful skin transplants were made after acquired tolerance to donor tissue resulting from the injection of donor cells into the embryonic recipient. This acquired tolerance by the embryo to foreign cells probably accounts also for the survival of renal homotransplants between dizygotic bovine twins. In the human the role of acquired antibodies is suggested by recent reports of the successful homotransplantation of skin to a recipient with agammaglobulinemia.

Although at the present time permanently successful renal homografts between humans cannot be performed because of this “antigen antibody like” reaction between donor tissue and recipient, skin homografts are known to survive between identical twins. Having established this fact, there were several experimental observations that made the success of a renal homograft seem likely if performed between identical twins and that justified the removal of a normal kidney from a healthy donor.

First, immunologic and genetic similarity accounts for the permanent survival of skin homografts between identical twins. Second, when skin or kidney homografts are carried out between antigenically dissimilar humans, the early function and the histological picture of rejection of each appears similar. Third, skin and kidney homografts possess a common antigen that can sensitize a recipient to a subsequent homograft of either tissue from the same donor. This further suggests that skin and kidney homografts behave similarly. Fourth, we have established to our own satisfaction that renal autografts have normal function indefinitely in animals.

This observation is important because, presupposing initial success of the transplant between antigenically similar (identical) twins, a second problem to be weighed was the permanence of such function. There were no reported instances of adequate functional studies in long-term surviving renal autografts.

It thus became imperative to establish beyond a reasonable doubt the fact that the twins were monozygotic (identical). Information that there was a common placenta was obtained from the hospital record of their birth. Blood samples from both twins were tested for all presently known reliable blood groups. These were found to be identical in both instances for each group (table). The two other siblings tested did not match. This was considered good but not indubitable evidence that the twins were identical. The twins and two other siblings were studied by Dr. Arthur G. Steinberg, geneticist of the Children’s Medical Center, Boston, who felt that, “On the basis of eight blood group systems plus the ability to taste phenylthiocarbamide, the sex similarity, and the a priori probability of dizegotic versus monozygotic twinning, the probability that these boys are identical twins is 0.985. Other data indicating that they are identical are 1) the presence of a single placenta, 2) both twins have the relatively rare Darwin’s tubercle on their ears while their sibs do not, 3) the twins have identical eye colors, including iris structure and pigment patterns, and their eyes are markedly different from their sibs’ eyes in color and in iris structure, 4) there are no data suggesting they are not identical. My conclusion is that the twins are identical.” Final decision to operate, however, was based on the most closely applicable evidence for antigenic similarity, that is the survival of transplanted skin between the two twins (fig. 1).

Evaluation of several factors determined the site for transplantation. The natural site for the homograft, the renal fossa, has two disadvantages. First, it requires simultaneous nephrectomy, thus increasing the magnitude of the operation. Secondly, it necessitates a uretero-ureteral anastomosis with the possibility of subsequent stricture formation because the length of the transplanted ureter vascularized by the renal pedicle is too short to reach the bladder. The upper thigh, the site of 13 previous homotransplants, was not used because it requires a skin ureterostomy with the possibility of subsequent ascending infection. In addition, it creates a problem in the collection of urine. The site selected, utilizing the iliac vessels for an anastomosis and placing the homograft retroperitoneally within the pelvis, allows implantation of the short ureteral segment directly into the bladder and places the kidney in its natural thermal environment. Furthermore, gravity drainage of the renal pelvis and ureter approaches normal physiological con-
ditions. The observations mentioned above, that renal autografts in animals maintain normal function indefinitely, appear important because of the previous opinion of other investigators that renal autografts so capable of survival soon develop impaired function manifested by abnormal renal dynamics and electrolyte excretion.\textsuperscript{15} We surmised that the permanently successful function ob-

served in our animal experiments resulted from the use of a recipient site that allows direct implantation of the ureter into the bladder, which has a normal thermal environment and which allows gravity drainage. This laboratory technique proved adaptable for use in man, provided that the left kidney was placed into the right iliac area or the right kidney into the left iliac fossa, thus reversing the normal anteroposterior relationship of the artery, vein, and ureter.

With this background, a nephrectomy was begun on the donor simultaneously with the operation on the recipient in an adjacent operating room. Through a right lower quadrant incision, the retroperitoneal area was entered exposing the right iliac vessels in the recipient. The operation was begun at 8:15 a.m., and the vessels were prepared for the anastomoses by 9:50 a.m. The donor kidney was brought into the room at 9:53 a.m. At this time the common iliac artery was occluded for the duration of the anastomosis. An end-to-end anastomosis between the free end of the hypogastric artery and the renal artery was completed at 10:40 a.m., and an end-to-side anastomosis between the renal vein and the common iliac vein was finished at 11:15 a.m. Total ischemia of the donor kidney was one hour and 22 minutes. Both arterial and venous anastomoses were satisfactory, and the entire kidney became turgid and pink immediately on release of the arterial clamp. Therefore, a small artery in the renal pedicle that appeared to be an accessory renal artery was ligated rather than anastomosed. The last clamp was removed from the common iliac artery 10 minutes later, and pulsation was noted in the right foot immediately. At this point a suprapubic cystotomy was made in the medial and superior portion of the bladder and a small tunnel dissected in the submucosa. The ureter was let in through the muscular wall of the bladder and through the submucosal tunnel, and a mucosa-to-mucosa suture was carried out (fig. 2). A polyethylene ureteral catheter had been inserted in the ureter to the renal pelvis and carried out through the cystotomy at this time. The incisions in the bladder were then closed after a mushroom catheter had been inserted from a suprapubic site. At this time clear urine was flowing copiously from the ureteral catheter. The kidney now lay rather neatly in its new site except that it projected forward where the lower pole impinged upon the iliac crest. The kidney was fixed by sutures to prevent its rotation, and the overlying oblique muscles and fascia were sutured together over it. The total operating time was three hours and 30 minutes. The postoperative course was smooth, and the incision healed per primam. The ureteral catheter was removed on the ninth postoperative day after evidence of function had been confirmed by the prompt excretion of injected sodium indigotindisulfonate.

The patient's subsequent course was characterized by continued improvement. Renal function improved; and, as it did so, acidosis and nitrogen retention disappeared (fig. 3). There was a marked decrease in blood pres-

---


---

Fig. 2.—Schematic diagram of renal homograft in situ showing vascular anastomoses completed and ureter implanted in bladder. Renal artery end-to-end with hypogastric; renal vein end-to-side with common iliac; ureter mucosa-to-mucosa anastomosis with bladder.

Fig. 3.—Disappearance of azotemia and improvement in renal function after renal homotransplantation. There is a progressive decrease in blood urea nitrogen and an increase in serial creatinine clearances (shown by solid bars at the bottom of the diagram). In October, 1954, there was almost no discernible phenolsulfonphthalein excretion. On June 14, 1955, phenolsulfonphthalein excretion was normal. In August, 1955, filtration rate and renal plasma flow as measured by the clearances of inulin and p-aminobiphenyl were at near normal values.

---

sure, and with this decrease the evidence of cardiovascular disease disappeared (fig. 4). Marked cardiomegaly (fig. 5) disappeared, and the abnormalities in the electrocardiogram vanished. The significance of these changes by the addition of a normally functioning kidney in the presence of two badly damaged kidneys will be discussed in a future publication.\textsuperscript{16}
During the follow-up visits, however, the patient continued to show evidence of some elevation of blood pressure. This elevation was labile and in some instances directly related to the presence of the examining physician. A persistent tachycardia (pulse rate of 90 to 100 per minute) continued in spite of normal blood chemistry and hemoglobin values. The urine continued to show 6 to 8 white blood cells, a rare cast, and 1+ protein. Clean voided urine specimens grew out a variety of organisms, which included P. vulgaris and Esch. coli. In vitro sensitivity tests showed these organisms to be resistant to most of the antibiotics except chloramphenicol. Colony counts varied from 100 colonies per milliliter on a pour plate to 2,000 colonies. After a consideration of the foregoing facts, it was decided to remove the two damaged kidneys for the following reasons. 1. Intravenous pyelography and renal function tests had shown that the renal homograft was functioning well. 2. The data indicated almost total lack of function in the two diseased kidneys. 3. The possibility existed that infection of the graft might occur from the two remaining diseased kidneys. 4. Experimental evidence suggests that a nonfunctioning or infected kidney may ultimately interfere with the function of a normal kidney, particularly with regard to its role in preventing renal hypertension.

After the second nephrectomy the patient's blood pressure stabilized at lower, and almost normal, levels. Frequent urine examinations since that time have shown clearing of the evidence of urinary tract infection. Some proteinuria, however, persists. The renal function of the homograft as measured by the clearance of inulin and p-aminohippurate closely approximates that of its fellow, which remains in the donor twin. Intravenous urography shows prompt excretion of dye in good concentration (fig. 6). The ureter appears somewhat dilated and tortuous, but this appearance might be expected in view of the fact that it lacks innervation.

The survival of the renal homograft for this period of time with continuing good function indicates the complete lack of a rejection response by the host and demonstrates that renal transplantation is a technically feasible procedure. It stresses further that, as indicated in previous studies, total anoxia of the kidney (in this case for a period of one hour and a half) does not mitigate against resumption of adequate function. The implications of the dramatic response in malignant hypertensive disease to the transplantation of a normal kidney should carry considerable weight in future thinking about the renal mechanism in human hypertension. Why one identical twin and not the other should develop glomerulonephritis and whether the kidney of the unaffected twin transplanted into the diseased recipient will be susceptible to further attacks is a question still to be answered. Unanswered also is the question of whether the transplanted kidney in its unusual position with a short and abnormally innervated ureter will escape eventual infection.
SUMMARY

Homotransplantation of a healthy kidney from one identical twin to another was performed. The homograft has survived for a 12-month period, and renal function is apparently normal despite the fact that both of the recipient’s diseased kidneys were removed. A striking sequel to the marked clinical improvement that was observed was the disappearance of the signs of malignant hypertension. Tissue transplantation including that of a functioning kidney appears to be a feasible procedure in identical twins, but to date successful permanently functioning homografts appear to be limited to such individuals.

721 Huntington Ave. (15) (Dr. Merrill).

---

LET’S SAVE THE GOOSE THAT LAYS THE GOLDEN EGGS

Robert Cutler, LL.D. (Hon.), Boston

Almost two centuries ago, Dr. Samuel Johnson, the great lexicographer wrote this in his broadsheet, the Idler: “Among those actions which the mind can most securely view with unabated pleasure, is that of having contributed to an hospital for the sick.” 1 It should be our objective, gentlemen of the House of Delegates, to make it possible for such unabated pleasure to be experienced by generous generations still unborn.

But the way of accomplishing this fine objective is beset with difficulties. Especially is this true in the case of university teaching hospitals supported by voluntary funds. Of course, all hospitals worthy of the name are teaching hospitals, whether they have 20 beds or 2,000 beds, and I know that every doctor worthy of that title is a teacher. But tonight, when I use the term “teaching hospital,” I am thinking and speaking only of university hospitals in which medical students receive their clinical training.

Let us get down to brass tacks. The fact is that our voluntarily supported hospitals are caught in the Big Squeeze. They are caught in that shrinking space between the upper millstone of collectible charges to patients and the nether millstone of skyrocketing operating costs. Sometimes it seems as if every hand were against the viability of voluntary hospitals. The legislature passes each year one or more laws that increase our burdens. The economic cycle pushes up the cost of services, of food, of supplies. And then our best friends, the doctors, keep on discovering and inventing new wonder-drugs, new mechanisms, new necessities for the saving of human life. Upon the university teaching hospitals of America a new burden has come crushing down since World War II. This burden, the child of increased research activities and increased costs related thereto, is what I want to talk about to you tonight.

COSTS OF RESEARCH

My own hospital, the Peter Bent Brigham, is a relatively small (280 bed) general hospital affiliated with the Harvard Medical School and forming an integral part of what we call the Harvard Medical Centre. The Brigham’s professional staff is shared with the Harvard Medical School. The Brigham Hospital has three functions: One is to care for the sick; the other two buttsress and support the first and suffuse with light the dark places. These other two are to teach those who will become the doctors and the nurses of tomorrow, and to advance by exact, inspired, and researching skill mankind’s store of medical knowledge.

As a trustee, I sometimes seem to alarm my great professors with my consciousness of the cost of research. But they really know better. Beyond any shadow of doubt, I march under the banner of inquiring research. In a university hospital, research is the pulsing arterial blood of life. It is the vital essence, as important to such a hospital’s place in the scheme of things as breath is to the nostrils of a living man. And I glory, as a lay brother, in the accomplishments of America’s teaching hospitals. To salute what they have achieved is not intended to, and does not in any way, derogate from the solid achievements of our other great hospitals. In this work, it is easy to find enough stars for everyone’s crown.

But I do say that the voluntarily supported university teaching institutions are, by their performance, a frontline in America’s care for the sick, today and tomorrow. Here are the geese that have been laying the golden eggs of medical discovery, and it is of primary consequence to the progress of medicine that these precious birds be assured in the years ahead of every opportunity for reproduction and for lying-in.

One of the difficulties confronting voluntarily supported medical schools and their affiliated hospitals to