injected with 40 mg developed, in 2–3 mo, weights exceeding 40 g.

Blood glucose levels before and after growth hormone treatment are given in Table 1. The dosage used was 2 mg/day for 3 consecutive days before the determination. It is readily seen that goldthioglucose obese animals do not show hyperglycemia as do the hereditarily obese hyperglycemic animals. The fact that this difference does not simply reflect strain idiosyncracies is demonstrated by the fact that the littermates of *ob ob* mice[4] made obese by goldthioglucose show blood glucose levels in the normal range. Similarly, goldthioglucose obese animals, whether Swiss or littermates of *ob ob* mice, do not exhibit any increase in blood glucose when treated with growth hormone, as do mice with the hereditary obese-hyperglycemic syndrome. In the latter, the response is so quantifiable that it has been made the basis of a method of determination (*5*).

Finally, it was found that subcutaneous injection of one unit of insulin was enough to cause hypoglycemic convulsions in all groups of Swiss mice (including the goldthioglucose obese animals), in nonobese littermates of *ob ob* mice, and in littermates of *ob ob* mice made obese by goldthioglucose treatment. By contrast, as reported previously (*2*), the blood glucose of animals with the obese hyperglycemic syndrome is totally unaffected by one unit of insulin, hardly affected by 20 units of insulin, and the animals can survive injection of even larger doses.

The profound metabolic difference between the two types of obesity is all the more striking in that the siblings, whether genetically obese or chemically obese, are completely identical in external appearance. Although fat deposition in all cases is dependent on positive caloric balance, these findings illustrate the fact that the existence of such a positive balance is not per se an explanation of obesity. It is simply a restatement of the first law of thermodynamics. In all cases, the real problem is to find the primary causes of this relative hyperphagia. Obesity appears to be a common end-result of syndromes of profoundly diversified etiologies (*1*).

[4] By *ob ob* mice is meant the animals in which the hereditary syndrome is present.

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Experiences with Transplantation of the Lung

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Relatively few studies pertinent to the transplantation of lung tissue have been made. Several workers have described (*1–3*) various types of autogenous and homologous lung transplants and have demonstrated the technical feasibility of such procedures.

Our initial experiences (*4*) were concerned with transplantation of the entire left lung from one dog to another. The left lung was removed from a donor animal by transecting the left auricle, the left main pulmonary artery, and the left bronchus. Left pneumonectomy was then done in the recipient animal. The donor lung was grafted to the recipient animal by anastomosing the left auricle, the left main pulmonary artery, and the main stem bronchus. Following these anastomoses, the grafted lung expanded, blood flowed throughout it, and to all gross inspection it appeared normal.

In a control group of 10 such homologous lung grafts the animals survived from 1 to 12 days with death usually resulting from pneumonia. Microscopic examination of the transplanted lung tissue at autopsy suggested that the changes were due to tissue incompatibility. In another group of 3 dogs wherein the donor and recipient animals were littermates, the survival period ranged from 13 to 30 days, significantly longer than the control group. In an attempt to alter the systemic antigen-antibody response, other groups of 3 dogs each were given benadryl (150 to 200 mg daily), cortisone (35 to 45 mg daily), or total body radiation (400 r) during their immediate postoperative course. Those animals receiving benadryl or total body radiation showed no appreciable difference in survival from the control dogs. Those to which cortisone was administered survived longer, from 12 to 18 days.

Subsequently we became interested in the role of the spleen and its possible effect in the viability of lung transplants. That splenectomy may reduce an animal's ability to produce antibodies has previously been demonstrated (*5*). In a group of 5 dogs splenectomy was done concomitantly with homologous left lung transplant. In another group of 6 dogs (the re-

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TABLE 1. Survival in homologous lung transplants.

Group	Number of dogs	Days of survival
Control	10	1, 2, 2, 3, 3, 4, 7, 11, 11, 12
Donor and recipient littermates	3	13, 20, 30
Benadryl	3	5, 6, 6
Cortisone	3	4, 12, 14
Radiation	3	4, 7, 8
Splenectomy at time of transplant	5	2, 4, 7, 7, 8
Splenectomy preceding transplant	6	2, 5, 5, 7, 8, 9

cipient animals) splenectomy was done 11 to 16 days prior to the left lung transplant. In neither group was the survival time enhanced by splenectomy.

That an animal survives or expires following homologous lung transplantation is not proof of the viability, necrosis, or functional ability of the transplanted lung. To elucidate this further, homologous left lung transplantation was done in 5 dogs which were then immediately subjected to right pneumonectomy. In 3 animals death occurred either during the operation or within 25 minutes after its completion. However, the remaining 2 animals survived for 6 and 9 days, proving unequivocally the functional ability of the homologous lung during these periods.

Although the operative technique of transplantation of one entire lung has been demonstrated to be feasible, the limitations imposed by foreign protein implantation appear responsible for the present failure of these organs to survive. Further studies are in progress to investigate the antigen-antibody mechanism in these homologous lung transplants and possible methods of altering it. The survival of quick-frozen lung grafts after transplantation is being considered.

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A Comparison of the Total Protein and Albumin Content of the Blood Sera of Some Reptiles[1]

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Precipitin studies in the systematic serology of vertebrates are usually carried out with native sera as antigens. DeFalco (*1*) in a study of avian relationships, suggested that some discrepancies in the percentage relationships obtained might have been due to differences in albumin/globulin ratios of the serum antigens. Haurowitz (*2*) has stated that one cannot generalize about proportions of serum albumins and globulins on the basis of human A/G ratios.

Deutsch and Goodloe (*3*), in an electrophoretic survey of plasma from 20 species of animals, found species differences in mobility, amount, and number of protein components. Notwithstanding, the analytical data were relatively constant in a given species. Fowl, in particular, had larger amounts of protein with low mobility components (possibly globulins) than did the mammals.

Deutsch and McShan (*4*) later studied the blood serum proteins of lower animals. Reptiles and amphibians have a greater proportion of low mobility serum protein components (globulins) than do normal higher vertebrates. Deutsch and McShan called the electrophoresis patterns of snake sera "unique." Species specificity was shown by the fact that diamond-back and timber rattlesnake patterns are easily distinguishable. Gleason and Friedberg (*5*) also detected a preponderance of low mobility components in the serum of a turtle.

In the present study, the total protein content and the albumin fraction of the native blood sera of a number of snakes and turtles were determined by the biuret method of Gornall *et al.* (*6*), the results were spot-checked against Kjeldahl analysis. Blood specimens were collected from the snakes by decapitation, whereas cardiac puncture was effective in obtaining blood from the turtles. Sera were obtained by standard serological procedures and became the property of the Serological Museum of Rutgers University. Data collected during this study are presented in Tables 1 and 2.

TABLE 1. Snake serum proteins (g/100 ml of serum).

Species	Total protein	Albumin
Crotalus r. ruber (1)*	5.80	2.60
C. adamanteus (1)	2.75	0.11
C. v. viridis (7)	2.79	1.57
C. v. oreganus (1)	3.86	1.88
C. h. horridus (3)	2.50	1.08
Agkistrodon piscivorus (4)	4.52	1.20
Naja n. naja (1)	4.28	2.34
Lapemis curtus (1)	2.88	1.40
Natrix s. sipedon (1)	4.65	2.00
Lampropeltis getulus californiae (1)	4.10	2.20
Pituophis c. catenifer (1)	5.35	3.53
Coluber c. constrictor (1)	5.65	3.00
Thamnophis s. sirtalis (1)	3.20	1.80

* Number of specimens used shown in parentheses.

TABLE 2. Turtle serum proteins (g/100 ml of serum).

Species	Total protein	Albumin
Chelydra serpentina (1)*	5.00	1.60
C. serpentina (1)	2.60	0.65
C. serpentina (1)	5.04	0.90
Chrysemys elegans (1)	2.43	0.50
Clemmys marmorata (1)	3.20	1.40
Testudo spp. (Aldabra) (1)	2.74	0.50
Dermochelys coriacea (1)	3.70	1.42
Caretta caretta (1)	2.22	0.65

* Number of specimens used shown in parentheses.

Statistical analysis of the data[4] revealed that for

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