function was demonstrably markedly deranged, presumably as a result of abnormalities induced by exclusion of bile and pancreatic secretion from the gut, the secretory function of the mucosa may also have been modified. Hence no conclusions as to quantity of cholesterol normally secreted by the mucosa can be drawn.

The sum of biliary and intestinal cholesterol secretions totals more than 2 g/day in normal persons under our conditions(1,2). If mucosal secretion normally were not greater than 400 mg/day, found in 2 of our subjects then there is more than 1.5 g/day of cholesterol normally secreted in the bile. This is a larger quantity than is usually estimated from studies carried out in bile fistula patients (6). Results obtained by the latter method may vary from normal, because of error in estimating the proportion of bile diverted into the fistula, because of partial exclusion of bile from the gut, and because studies were carried out in patients with diseases of the biliary tract.

Summary. In 3 patients with complete obstruction of biliary and pancreatic ducts cholesterol "secretion" by intestinal mucosa was 250-400 mg/day. Quantification of this function in either man or animals has not been reported previously.

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The Dwarf Mouse—An Animal with Secondary Myxedema.* (24891)

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It has been concluded that dwarfism in the house mouse is inherited as a recessive Mendelian characteristic dependent upon a single gene(1). Dwarfism results from hypofunction of the hypoplastic pituitary gland(2,3). It has been reported that there is an almost complete lack of eosinophil cells in the anterior pituitary, accompanied by deficiency in growth hormone production(2,3). Snell(4) first demonstrated that thyroid of the dwarf mouse was defective. Later(2,3) morphology of dwarf-mouse thyroid was investigated more thoroughly. Smith and MacDowell(2) and Kemp and Marx(3) found that thyroid tissue was interspersed with adipose and fibrous connective tissue which made it impossible to dissect and weigh the gland as a separate organ. Gland tissue present showed signs of delayed function. In addition, 30-40% lower basal metabolism occurred in dwarf mice as compared to normal mice of same age(5). A histological study of the thyroid from a few thyrotropin-stimulated dwarf mice showed no structural changes to indicate increased activity(3), but by daily transplants of fresh rat pituitaries into dwarf animals, small body size, sterility, and associated endocrine abnormalities, including thyroid changes of hereditary dwarfism, were corrected(2). All available data suggest secondary deficiency of the thyroid, but which pituitary hormone insufficiency is responsible has not been clarified. With modern technics, it is possible to obtain more exact information as to the cause of thy-

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roid disorder involved in the complex picture of dwarfism in mice. This report gives data that the dwarf mouse, besides other deficiencies, is in a state of secondary myxedema, dependent at least partly upon lack of thyrotropic hormone. This is of value in further investigations of pituitary cell morphology and in experiments concerned with effect of thyrotropin on connective tissue.

Material and methods. The 40 dwarf and 30 normal mice were from same litters and kept together in same room at 22°C. Dwarf animals were fed a special diet prepared from pellets given to normal mice. The pellets were ground and mixed with milk to make them easier to eat. Ten dwarf mice were killed by decapitation and blood from the wound was pooled for protein-bound iodine (PBI) determination.‡ A similar procedure was repeated with 10 other dwarf mice for a second PBI determination. Blood from 10 normal siblings obtained in same way, was analyzed for PBI content for control purposes. Ten intact dwarf mice and 10 dwarf mice treated with thyrotropin (Organon, 0.16 U.S.P./0.25 mg in 0.25 cc of water/animal) were injected subcutaneously with 10 μc of radioactive iodine I^{131}§ in 0.1 cc of carrier solution (0.01 μg I^{127}/μc I^{131}). Twenty-four hours later radioactivity was measured over neck and pelvic regions with EKCO‖ scintillation counter N 559 A used with auto-scaler N 530 and provided with special animal holder. The distance from animal to crystal was 50 mm. There was an aperture 21 mm in the chimney-like lead shield 30 mm thick. The same procedure was carried out with 10 control and 10 thyrotropin (Organon, 0.66 U.S.P./1 mg in 0.25 cc of water/animal) stimulated normal mice. Activity in each region was measured 3 times in each animal and averages used in calculating results.

Results. PBI values for dwarf mice (1.2 and 1.3 μg/100 ml of serum) compared with those obtained in blood from normal siblings (7.3 μg/100 ml of serum) showed a low pathological level.

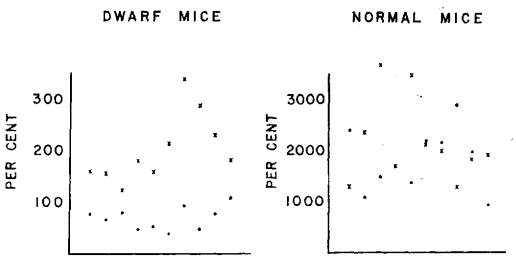

FIG. 1. Radiation from neck in % of radiation from pelvic region. ● Control animals. × Thyrotropin treated animals. (Each point represents individual animal.)

Results from radioactive iodine uptake test are shown in Fig. 1. Thyroids of untreated dwarf mice did not accumulate iodine. In 9 out of 10 animals, activity over pelvic region was higher than that over the neck. It is easily seen that thyrotropin stimulated the thyroid to increased uptake of iodine in all treated dwarf animals. Untreated mice took up about 10 times as much radioactive iodine as untreated dwarf mice. Normal animals also increased their uptake after thyrotropin stimulation, but response was not as uniform as in dwarf animals.

Conclusion. The histological picture(2,3), low basal metabolism(5), inability of thyroid to accumulate iodine, and low PBI values shown in present study, indicate a myxedematous state in dwarf mice. Daily rat pituitary transplantations into dwarf animals restore thyroid to normal(2), from which is concluded that a pituitary factor, or factors, is responsible for the poor state of the dwarf thyroid. Thyrotropin is able to stimulate the thyroid gland of dwarf mice, and sensitivity seems greater than in normal siblings. It may be concluded that there is a lack of both thyrotropin and of somatotropin in the dwarf mouse. These mice are in a state of secondary myxedema and an important etiological factor for this disorder is thyrotropic pituitary hormone deficiency.

Summary. 1) The level of protein-bound iodine in serum of dwarf mice, determined in 2 groups of 10 each (1.2 and 1.3 g/100 ml), was very low in comparison to the value (7.3 g/100 ml) obtained on serum of 10 normal

‡ Kindly carried out by Dr. Th. Friis at Frederiksberg Hospital, Copenhagen, Denmark.

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siblings. 2) Thyroids of untreated dwarf mice did not show accumulation of radioactive iodine (I^{131}), whereas injection of thyrotropin (0.16 U.S.P.) significantly stimulated the glands to iodine uptake. 3) From these data, and previous observations, it is concluded that the dwarf mouse is a secondary myxedematous animal because of deficient production or complete lack of thyrotropic pituitary hormone.

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Distemper and Measles Viruses. I. Lack of Immunogenic Crossing in Dogs and Chickens.* (24892)

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The demonstration of distemper antibodies in human serum and gamma globulin suggested at first an infection of humans with distemper virus at an early age, with possible clinical involvement of a respiratory nature (1,2). Although the presence of these antibodies in humans was confirmed(3) and their time of appearance in children studied(4), the question as to whether distemper virus *per se* was responsible for human illness has remained unanswered. More recently, a possible relationship between measles and distemper viruses was presented as another likely explanation for the appearance of distemper antibodies in humans(5,6,7). An investigation was therefore initiated in this laboratory to study the suggested similarity between the 2 viruses by a variety of laboratory methods. This communication reports on the search for measles and distemper antibodies in sera from puppies and chickens immunized with distemper virus, and in those of chickens which received multiple injections of measles virus.

Materials and methods. Virus strains. The canine distemper (CD) strain used for both immunization of puppies and chickens and for neutralization tests was the Lederle chick-embryo-adapted CD virus(8). Virus stock consisted of a 30% infected chorio-allantoic membrane (CAM) suspension which was maintained in frozen state. The 50% chick-embryo-infective titer (CED_{50}) was $10^{3.2}$-$10^{3.7}$ per ml. Puppies were challenged with a suspension of Snyder Hill strain CDV(9) prepared and stored as described earlier(10). A pool of Edmonston strain† measles virus (MV), having 50% tissue-culture, infective titer (TCD_{50}) of $10^{6.3}$/ml, was used for immunization of chickens. It represented a second monkey kidney passage of chick-embryo-tissue-culture adapted virus(11). Serum neutralization was carried out with a pool of the same MV strain derived from human-amnion tissue culture. This pool had a TCD_{50} titer of $10^{3.5}$-$10^{4.5}$/0.1 ml and was prepared in HeLa cells following 5 consecutive passages of the virus in this laboratory in an established human amnion cell line and one passage in HeLa. *Tissue culture.* HeLa cultures used for the propagation and titration of MV, and in the MV neutralization test, were grown in Eagle's solution(12) containing 20% calf serum. Just before inoculation, these cultures were renewed with the same solution containing 5% calf serum. *Control immune sera. CD.* A susceptible puppy was injected subcu-

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