group there were lesions in 5 of 8 rats and in 4 instances these were bilateral. Invasion of blood vessels by neoplastic cells, as described by Gillman *et al.*(9) in spontaneous pheochromocytomas of the rat, was not observed.

Discussion. Analysis of the results of chronic administration of pituitary growth hormone to adult male rats indicates that growth response was similar to, but less striking than, that of females so treated. Growth response of the older of the 2 age groups tested (14 months old at onset) was inferior to that of the younger group (6 months old at onset), particularly in the later part of the experiment. This may have been due to a number of factors, such as aging, debility due to neoplasms, and recurrent infections. In the earlier studies on females injected with growth hormone, pheochromocytomas frequently occurred. There was no evidence of a prolonged hypertension in those animals, as judged by weight of the heart and histology of the arterioles. In the present study no significant elevations of blood pressure were observed in experimental rats subsequently shown to have pheochromocytomas. However, the possibility of transient episodes of hypertension cannot be excluded.

Summary. The long term administration of pituitary growth hormone to adult male rats of the Long-Evans strain resulted in progressive growth in body weight and length. Many neoplasms developed in the rats during this prolonged period, but the incidence in treated rats was not significantly different from that in controls, with the exception of tumors of the adrenal medulla. Pheochromocytomas occurred in 9 of 16 growth hormone injected rats and in none of the controls.

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Received July 19, 1956. P.S.E.B.M., 1956, v93.

Influence of Acetylcholine on Human Pulmonary Circulation under Normal and Hypoxic Conditions.* (22668)

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Short periods of induced hypoxia characteristically produce a moderate increase in the pressure in the pulmonary artery in normal man(1). In general, the rise in pressure is greater than might be expected to be due to the increase in cardiac output which occurs. Moreover, the possibility that the augmented pressure results from generalized systemic vasoconstriction seems unlikely, since the pulmonary wedge pressure does not change(2,3) the brachial artery pressure remains constant, and the "central blood volume" shows no consistent alteration(3,4). From these data it may be inferred that the

* This investigation was supported by a research grant (PHS Grant H-833(C)) from the National Heart Institute of the National Institutes of Health, Public Health Service.

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rise in pulmonary artery pressure is probably produced by active constriction of the vessels of the lungs.

Previous experiments(5.6) have led us to believe that acetylcholine has a vasodilating action in the human pulmonary circulation. In diseased states there is evidence that this action is to some extent dependent on the pre-existing tone in the vessels of the lungs(5.7). The effect of acetylcholine might, therefore, be expected to be more evident when the tone in these vessels has been increased by hypoxia.

The present study was undertaken to investigate the effect of acetylcholine on normal pulmonary circulation, and to determine whether the effect was modified by the presence of hypoxia. The purpose of this note is to report the results obtained in studies performed on 6 normal subjects.

Methods. The procedure combined catheterization of the pulmonary artery and cannulation of the brachial artery. Each subject breathed through a mouthpiece for 2 periods of 20 minutes separated by a rest of 15 minutes. During one period, the subject breathed ambient air and, during the other, a mixture of 12% oxygen in nitrogen. Between the 11th and 15th minute of each period, acetylcholine was infused through the catheter into the main pulmonary artery at a rate of 0.5 mg min. Measurements of the pressure in the pulmonary and brachial arteries and of the cardiac output were made before and during the infusion of acetylcholine.

While the subject breathed ambient air, the cardiac output was estimated by the Fick principle. Under hypoxia, the Stewart-Hamilton dye-dilution technic was considered preferable since insufficient time was allowed for the establishment of the "steady state" required for the application of the direct Fick method. The dye output was obtained by injecting Evans blue dye into the main pulmonary artery while inscribing a dilution curve from the brachial artery by means of a Colson densitometer.

Results. Six satisfactory studies have been completed with similar results in each. The data from one of these are illustrated in Fig. 1. When the subjects breathed ambient air, acetylcholine caused a slight fall in

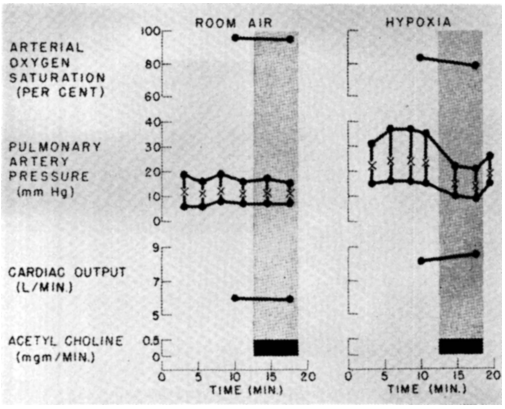

FIG. 1. Effect of acetylcholine on arterial oxygen saturation, pulmonary arterial pressure and cardiac output in one subject. The subject breathed room air for 20 min. and 12% oxygen for 20 min. Shaded areas represent time of infusion of acetylcholine. A period of 15 min. elapsed between the 2 experiments.

the pulmonary arterial pressure. This response was much more evident after hypoxia had raised the pressure in the pulmonary artery. The reduction in pressure during the infusion of the drug occurred despite the fact that the cardiac output had either remained constant or had slightly increased. The infusion caused no alteration in the heart rate, the brachial arterial pressure, the arterial oxygen saturation, or the ventilation. In 2 subjects, pulmonary wedge pressures were recorded and found not to change during the infusion of acetylcholine. Thus, since the pressure in the pulmonary artery had fallen, the gradient of pressure was decreased.

Discussion. In these studies, the cardiac output remained unchanged or increased during the infusion of acetylcholine; it never decreased. Moreover, the constancy of the wedge pressure measured in 2 cases suggests that the left atrial pressure was unaffected by the drug. Therefore, it is believed that the fall in pulmonary arterial pressure resulted from an active dilatation of the pulmonary vessels. The augmented effect observed during hypoxia suggests that the action of the drug is dependent on the pre-existing tone of the pulmonary vessels.

Summary. Acetylcholine infused into the main pulmonary artery caused a slight fall in pulmonary arterial pressure when the subject

breathed ambient air, but a greater fall in pressure after pulmonary hypertension had been produced by hypoxia. The fall in pressure was associated with either a constant or an increased cardiac output. The evidence suggests that acetylcholine causes vasodilatation in the lungs and that this action is largely dependent on the pre-existing tone of the pulmonary vessels.

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Received July 20, 1956. P.S.E.B.M., 1956, v93.

Relationship of Bat Salivary Gland Virus to St. Louis Encephalitis Group of Viruses.* (22669)

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The existence of rabies in bats in the United States was first suspected in 1951 when a woman in Big Spring, Texas died of rabies 3 weeks after being bitten by a bat. This experience was reported(1) after the first demonstration of rabies in bats in the United States when the virus was demonstrated in a Florida yellow bat (*Dasypterus floridanus*) which had attacked a child(2). Three months after this incident another case was reported in Pennsylvania where, without provocation, a woman was bitten on the arm by a bat (probably *Lasciurus cinereus*) from which the rabies virus was isolated(3,4). These and other experiences prompted several groups to initiate studies to assess the importance of these animals as reservoirs and carriers of the infection(5-10). The rabies virus has now been recovered from bats in widely scattered areas in the United States (Florida (2), Pennsylvania(3), Texas(6,7), California (10), and Montana(11).)

During the course of their studies on rabies in nonsanguivorous bats in Texas, Burns and Farinacci(8,12,13) recovered a number of viral agents in addition to the rabies virus from the salivary glands of encephalitic bats. Following preliminary tests(13) which suggested that these agents were antigenically related to the St. Louis encephalitis virus, one strain was referred to us for determination of its antigenic relationship to the Japanese (JE)-West Nile (WN)-St. Louis (SLE)-Murray Valley (MVE) encephalitis group(14-19). This report is concerned with the relationship of the bat salivary gland (SG) virus to the JE-WN-SLE-MVE group as established by cross complement-fixation tests using hyperimmune guinea pig sera.

Viruses. The bat salivary gland virus (SG) designated #1410-19 and in its 5th mouse brain passage was sent to us by Lt. Col. Kenneth F. Burns. The Nakayama strain of JE virus in the 41st mouse brain passage, a strain of MVE in the 10th passage, and the WN virus (J7259) in the 25th passage were obtained from the Viral and Rickettsial Disease Registry, American Type Culture Collection, Washington, D.C. The Hubbard strain of SLE was obtained from Dr. Isaac Rushman.

Antigens and antisera. The antigens and the antisera for the complement fixation tests and the grid type test using these materials are essentially the same as described by Pond and his associates(19) and by Hammon and Espana(20). Antisera against the JE, WN, SLE and MVE viruses were obtained from

* Aided by a grant from the Caruth Foundation.