diet than if it is not supplied.

Summary. The testes of male chickens kept on a diet deficient in vit. E for two months were much reduced in size, and the seminiferous tubules showed considerable degeneration. The combs, likewise, were smaller in the group deprived of vit. E. The assays of pituitary glands revealed that more gonad stimulating substance was produced by those of fowls which had received vit. E in their diet. The pituitary glands from the birds on the E-deficient diet had approximately one-half as many active basophilic cells as those from the birds with the E supplied.

Acknowledgement is made to Quaker Oats Co. for supplying ground oat hulls and to Distillation Products, and Ayerst, McKenna and Harrison for supplying the tocopherols.

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Received February 15, 1952. P.S.E.B.M., 1952, v79.

Pulmonary Infarction from Cardiac Catheterization.* (19407)

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The major complications that have been reported as a result of cardiac catheterization have been reviewed by Zimdahl(1) and have been of two general types: First are a variety of cardiac arrhythmias developing largely as a result of the introduction of the catheter into the right auricle and ventricle. These have consisted of ventricular premature beats, ventricular tachycardia, ventricular flutter, ventricular fibrillation, right bundle branch block, and heart block. Auricular premature beats, auricular flutter, paroxysmal auricular tachycardia, and auricular fibrillation also have occurred when the tip of the catheter has been in the right auricle. The second type of complication has been thrombosis occurring in the veins at the venotomy site and sometimes propagating centrally. In one reported case (2), in whom the catheter was introduced into the heart by way of a leg vein, a thrombosis propagating centrally to the heart resulted in death several weeks later.

We wish to report the production of pulmonary thrombosis and infarction in a series of patients with pulmonary congestion in whom a branch of the pulmonary artery has been occluded in order to record the pulmonary "capillary" pressure.

Methods. To measure the pulmonary "capillary" pressure, the catheter is introduced by the usual fashion into the pulmonary artery and then advanced further until it is wedged into one of the distal branches of this vessel(3). On the basis of about 100 ex-

* This work was supported by grants from the Life Insurance Medical Research Fund and the National Heart Institute, U. S. Public Health Service.
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periences with this procedure, it was reported (3) that no untoward reactions occurred. In March of 1950, however, the first of a series of pulmonary infarctions began to occur in patients with mitral stenosis and congestive heart failure following cardiac catheterization. At first, these infarctions occurred only sporadically, but later occurred with increasing frequency. Between March and November of 1950, only 4 such infarctions occurred, 2 in patients with congestive heart failure and 2 in patients with mitral stenosis. Between November, 1950 and May, 1951, pulmonary infarcts were produced in 15 of 27 patients with mitral stenosis. In no instance did death occur, but many of the patients had a stormy course for several days. Had it not been for the necessity of catheterization to determine the patients' operability, this procedure would hardly have been justified. There was a striking absence of this complication in patients with many types of congenital heart disease, pulmonary disease with cor pulmonale, various types of heart disease without pulmonary congestive phenomena, and liver disease.

Observations. Infarction was recognized in these cases by the occurrence of a rise of temperature, pulse, and respiration in all cases, associated with malaise, and often cough, signs of consolidation in the affected area, frank hemoptysis, blood-streaked sputum, pleuritic pain, friction rib, and a shadow in the lung by x-ray. With one exception, the infarct occurred in the area plugged by the catheter, from which it was concluded that this represented thrombosis *in situ* rather than embolism. In the one case in whom the infarct occurred at a distant site, it was presumed to have been on the basis of embolism from a right auricular thrombus. The symptoms and signs of infarction occurred as early as two and a half hours after wedging the pulmonary artery with the catheter and as late as 79 hours.

Age did not appear to be a factor, since infarction occurred in one patient who was only 14 years old. The company manufacturing the catheters‡ had not changed the surfacing lacquer(4). Three factors, however, appeared to be mainly concerned with the production of this complication in these patients. 1) Only in patients with pulmonary congestion did pulmonary infarction occur. Venous thrombosis in the legs and pulmonary infarction from embolism are particularly prone to occur in patients with congestive heart failure(5,6), although a definite abnormality of the clotting mechanism in these patients has never, to our knowledge, been established. This thrombophyllic tendency presumably varies considerably from patient to patient and represents fertile soil for the development of infarction. 2) The peak incidence of pulmonary infarction occurred in these patients at a time when the greatest effort was being made to obtain as good a "capillary" pressure tracing as possible. The catheter was left *in situ* for longer periods of time than previously, and the pressure was often checked in several different areas of the lung. Careful washing of the surface and lumen of the catheter with saline and attempts to shorten the period of wedging of the pulmonary artery to an irreducible minimum, however, did not prevent pulmonary infarction. Even full heparinization of the patients before wedging the catheter did not eliminate infarction. 3) During the period when infarctions were produced, the catheters were sterilized by soaking in a 1:1000 solution of mercurioxycyanide. In our original publication(3), they had been sterilized by autoclaving. On the reinstitution of sterilization by autoclaving in May, 1951, pulmonary infarction following catheterization abruptly ceased. There have been no infarcts in 20 consecutive patients with severe mitral stenosis or congestive heart failure studied since that time.

Other factors such as the length of time the catheter was left in place, variation in the roughness of the surface of the catheter and in the degree of traumatization of the vessel wall by the catheter, and the care with which the surface of the catheter was washed with saline before its introduction into the vein presumably played an aggravating role. There seems to be no doubt, however, that the *sine*

‡ U. S. Catheter and Instrument Company, Glens Falls, N. Y.

qua non in the production of infarction has been the chemical sterilization of the catheters.

Summary. 1. Pulmonary infarction has occurred in 19 patients with severe mitral stenosis or congestive heart failure 2½ to 79 hours following wedging of a cardiac catheter into a branch of the pulmonary artery for the measurement of pulmonary "capillary" pressure. 2. Over a period of 6 months, pulmonary infarction occurred in over half the patients studied with severe mitral stenosis. 3. In other patients without pulmonary congestion, pulmonary infarction did not occur. 4. When the catheters were sterilized by autoclaving instead of by soaking in mercurioxycyanide, pulmonary infarction did not occur in 20 consecutive patients with either severe mitral stenosis or congestive heart failure.

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Received February 15, 1952. P.S.E.B.M., 1952, v79.

Hemolysis in Hydrogen Peroxide of Erythrocytes of Premature Infants. Effect of Alpha-tocopherol.* (19408)

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György and Rose reported that administration of tocopherol prevented intravascular hemolysis and hemoglobinuria in vit. E deficient rats injected with alloxan(1). They found, also, that the red blood cells of rats deficient in tocopherol could be completely hemolyzed, while those of tocopherol-treated animals were never affected by incubation with dialuric acid or alloxantin, reduction products of alloxan(2). Since *in vitro* hemolysis was more marked with dialuric acid than with alloxantin, the authors suggested that dialuric acid might be the active moiety. Because Owens and Owens(3) had suggested that a dietary deficiency of vit. E might be responsible for retrolental fibroplasia, the erythrocytes of 14 premature infants, both with and without the disease, were tested for sensitivity to dialuric acid, using the procedure of Rose and György(4). The infants showed no evidence of E deficiency as measured by this test(5). Many premature infants do not absorb fat well, and Wright, Filer, and Mason (6) have shown that low serum tocopherol levels develop in these infants when fed diets designed to circumvent the handicap in fat absorption. Since the manifestations of E deficiency vary in different species, and in the same species at different ages(7), the possibility remained that E deficiency might exist in premature infants even though the dialuric acid test did not so indicate. The finding by György, Cogan, and Rose(8) that the red blood cells of newborn full term infants, resistant to dialuric acid, were hemolyzed by dilute solutions of hydrogen peroxide, and that this hemolysis could be prevented by alpha-tocopherol led to the present study of prematurely born infants. A significant degree of hemolysis has been found and this is consistently prevented by the feeding of alpha-tocopherol.

Subjects and methods. Observations were made in 23 premature infants, born either at the Colorado General Hospital, or admitted to its Premature Infant Center because of inadequate facilities for care at the place of birth. The infants' weights ranged from 745

* Aided by a Grant from the Association for the Aid of Crippled Children.

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