Kaufman, Iglauer, and Herwitz(9). As may be noted in Fig. 3, the Ia-Ja amplitude of the acceleration ballistocardiogram increased by an increment of 100% of control values; heart contractile force increased by approximately the same increment. The Ia-Ja time was reduced from .04 to .02 sec.; the total time of the heart contractile force curve decreased from .23 sec. to .14 sec. There was a 30 mm decrease in diastolic pressure and a 40 mm increase in systolic pressure. The heart rate increased from about 85 beats/min. to 220 beats/min. The increased Ia-Ja amplitude and contractile force along with shortening of the Ia-Ja time and the curve of contractile force change are consistent occurrences with this drug. The relation of these changes and those of arterial pressures and heart rate is being studied. A preliminary report(10) has been published describing changes produced by other sympathomimetic amines in the dog and human acceleration ballistocardiogram.

Summary. A method of obtaining direct-body displacement, velocity and acceleration ballistocardiograms of the dog has been described. Ballistocardiograms obtained by this method are similar to those of the human.

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Stability of Vesicular Stomatitis Virus at Varying Hydrogen Ion Concentrations. (21199)

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Various workers(1-5) have studied the stability of vesicular stomatitis virus at varying hydrogen ion concentrations. The detailed investigations of Galloway and Elford(5) have demonstrated the loss of viral infectivity at approximately pH 4.0 and its persistence at pH 9.6. Their criterion for survival of infective virus consisted of presence of characteristic lesions in the foot pads of guinea pigs inoculated intradermally with virus. No information is given on the stability of virus beyond pH 9.6 and no quantitative data are available concerning the actual number of infective viral particles surviving at any given hydrogen ion concentration. The lack of adequate quantitative data as well as the possible consequences associated with the common practice of disinfection of infected materials with sodium hydroxide emphasized the need for reexamination of this problem. The present studies utilizing more sensitive experimental hosts (mice and embryonated eggs as opposed to guinea pigs) in viral infectivity tests, report quantitative data on the survival of 2 strains of vesicular stomatitis virus over a wide range of pH.

Materials and methods. The viruses used in these studies were the New Jersey and the Indiana strains of vesicular stomatitis. These strains had histories of passages in various experimental hosts; the most recent passages were made in embryonated eggs via the al-

lantoic route. Infected allantoic fluid was used as a source of virus. The pH of the viral suspension was adjusted either by dilution (1:10) of virus-containing allantoic fluid in an appropriate buffer of desired pH or by addition of 0.5 N HCl or NaOH until the desired pH was achieved. These samples were left for 1 hour at room temperature (25°C), and the pH of the suspension was determined electrometrically at the beginning and at the end of the hour period. Determination of infectivity was made by a) intracerebral inoculation of 0.03 ml of serial dilutions into each of a group of 6 to 8 Namru(6) mice 4 to 6 weeks of age or b) suballantoic inoculation of 0.1 ml of serial dilutions into each of a group of eight 9-day embryonated eggs. All dilutions of virus were made in 2% peptone solution. Mice were observed daily for a period of 10 days and deaths were recorded. Inoculated eggs were examined daily for deaths over a period of 4 days. Whenever there was any doubt concerning the viral origin of death of embryos, the allantoic fluids were tested for specific antigen in the presence of vesicular stomatitis antiserum. The 50% mortality endpoint was calculated by the method of Reed and Muench(7).

Results. Table I summarizes the results obtained with mice and embryonated eggs of the effect of pH upon the stability of the infective component(s) of vesicular stomatitis virus. The data shown in the table are representative of the results obtained for both virus strains in a number of similar experiments.

The data indicate that the infectivity of both strains of viruses for mice is not markedly affected by acidic reactions until a pH value of 4.0 or less is employed. No infective virus was demonstrable after exposure of virus at pH 2.0 for 1 hour at room temperature. Virus appears extremely resistant to inactivation in alkaline environments. There was no great change in infective titer of the New Jersey strain up to pH 10.4 and only a slight decrease at pH 11.6. Marked destruction of viral infectivity occurred, however, upon exposure of virus to a pH value of 12.5. The results of infectivity tests in mice with the Indiana virus were quite similar, although this strain seemed to be slightly more susceptible to inactivation at alkaline reactions. It is evident from Table I that the results obtained with embryonated eggs closely parallel those with mice.

TABLE I. Effect of pH upon Infectivity of Vesicular Stomatitis Virus.

pH	Buffer-system	Virus titer* New Jersey strain Mice	Eggs	Indiana strain Mice	Eggs
7.2	Peptone broth	5.9	5.8	5.3	5.6
2.0	KCl-HCl	<1.0	<1.0	<1.0	<1.0
3.0	Citrate-HCl	2.3	2.5	1.0	2.3
4.0	Acetic acid-acetate	3.7	2.6	2.1	3.0
5.0	*Idem*	5.0	4.8	4.5	4.4
6.0	KH_2PO_4 - NaOH	5.5	5.7	4.7	5.2
7.0	*Idem*	5.8	5.8	5.3	5.7
8.0	"	6.3	6.0	5.2	4.7
8.9	H_3BO_3-KCl-NaOH	5.7	5.4	5.0	5.1
9.7	*Idem*	6.2	5.8	5.4	4.0
10.4	Glycine - NaOH	6.2	5.1	4.2	3.5
11.6	*Idem*	4.1	3.4	1.6	<1.0
2.0	pH adjusted with HCl	No virus	—	—	—
12.5	pH adjusted with NaOH	<1.2	—	—	—

* Expressed as log LD_{50} remaining.
— Indicates sample not tested.

Discussion and Summary. The present results are in conformity with the data of Galloway and Elford(5) concerning the instability of vesicular stomatitis virus at pH values of 4.0 or less. The use of more sensitive experimental hosts and determination of 50% mortality endpoints, however, showed that inactivation of virus was achieved only at pH 2.0. Extension of studies beyond the pH value of 9.6 reported by Galloway and Elford (5) revealed the striking resistance of virus to inactivation by alkaline environments. It would appear that the wide range of stability of vesicular stomatitis virus may render it a likely prospect for purification by means of the ion exchange resins.

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Sexual Development in Female Rats Treated with Cortisone.* (21200)

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An excess of endogenous adrenal oxysteroids as found in Cushing's Disease is associated with sexual dystrophy(1). Hench *et al.*(2) found that adolescents treated with cortisone suffer menstrual disturbances more frequently than adults. While cortisone treatment in women with regular menstrual cycles leads to irregularities, it may lead to establishment of menses in amenorrheic individuals(3). The observation of Sohval and Soffer(4) and of Maddock *et al.*(5) that patients on cortisone excrete excessive amounts of gonadotrophin suggests that these menstrual changes may be related in part to alterations in pituitary function and that cortisone may stimulate the production of gonadotrophin. Antopol(6) found that the ovary and accessory reproductive organs of immature mice treated with 1.25 mg of cortisone weighed less than normal. Byrnes and Shipley(7) found that Lipo-adrenal Cortex and Adrenal Cortex Extract inhibited gonadotrophin secretion in ovariectomized females in parabiosis with an immature female while daily doses of 0.5 or 2.0 mg of cortisone were ineffective. Fawcett and Deane(8) found no difference in water content or stroma development of uteri of ovariectomized rats treated with cortisone and/or estrogen. However, Szego(9) found a decreased hydration of uterus 4 hours after intravenous injection of estrogen to cortisone-treated rats, but no depression of wet weight. Talalay(10) reported that 2.5 mg of cortisone daily for 3 days partially prevented the increase in uterine weight produced by estrogen in ovariectomized rats. With administration of 0.5-5.0 mg of cortisone daily to young rats, Moore(11) found an increase in weight of the ovary accompanied by variable changes in uterine weight. It appears, therefore, that there is no agreement among investigators on the effects of cortisone on the female reproductive system and that only limited information is available on its effects on production and release of pituitary gonadotrophin.

The purpose of this investigation was to study the effects of a small dose of cortisone on the development of the reproductive system and on the gonadotrophic hormone content of the hypophysis of immature female rats.

Materials and methods. Four groups of 23-day-old female rats, 6 pairs to each group, were pair-fed with Rockland Milled Stock Diet. Another 4 groups of 23-day-old female rats, 8 pairs to each group, were fed *ad libitum*. One member of each pair was treated daily with 1 mg cortisone acetate† subcutaneously. At the end of 2 and 4 weeks of treatment as well as 3 weeks after cessation of treatment, 6 or 8 pairs of rats, respectively, were autopsied. Tissues were fixed in Bouin's solution or alcoholic-formalin and stained with hematoxylin and eosin or periodic acid-leucofuchsin, respectively. The pituitary of each animal was frozen individually for subsequent gonadotrophin assay. The uteri of 3 pairs of animals in each group were dried to constant weight at 110°C to determine their water content.

Results. Body weight gain was 22.8 to 48.0% less in the treated animals than in the controls. With cessation of treatment, the

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