blood pressure changes. The EA-1 did not reverse the effects of epinephrine or prevent their appearance.

Comments. EA-1, in doses ranging as high as 2.5 mg/kilo, will cause disturbances of cardiac rhythm in the unanesthetized dog. Auricular paroxysmal tachycardia, auricular and ventricular premature beats are the most commonly observed disturbances. Therapeutic doses cause no such changes in the unanesthetized dog. However, in the heart sensitized by cyclopropane anesthesia, therapeutic doses can produce sufficient stimulation to cause disturbances in rhythm. These disturbances affect the auricles principally and appear to exert little effect on the ventricular automatic tissue.

Summary. In 5 dogs premedicated with morphine and scopolamine and anesthetized with cyclopropane, 2-methyl amino heptane (EA-1) caused sinus and auricular paroxysmal tachycardia, and ventricular premature beats. Ventricular paroxysmal tachycardia and auricular and ventricular fibrillations were not observed.

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Inhibition of the Deciduomal Response by Preexisting Deciduomata in the Mouse*†

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Loeb[1,2] discovered that surgical traumatization of the uterus during periods of luteal activity was followed by growth and differentiation of the endometrium into a structure which he called a deciduoma. In more recent years the observations of Krehbiel[3] and Atkinson[4] on the morphology and physiology of deciduomata have demonstrated that the reaction is the experimental equivalent of the naturally occurring maternal placenta.

It has been reported by Selye and McKeown[5] that deciduoma formation cannot be elicited after placentation in the rat. Moreover, these authors found that if the pregnant horn of the unilaterally pregnant animal is removed, the remaining non-pregnant horn will quickly regain its sensitivity to traumatization. Atkinson[6] demonstrated that this uterine desensitization to deciduoma formation is not due to changes in the estrogen-progestin balance in the pregnant mouse.

The phenomenon of uterine desensitization is difficult to analyze in the pregnant animal due to the complexity of the factors involved. However, it is possible to evaluate the role of the maternal placenta alone through the study of its experimental counterpart, the deciduoma. The present experiments were designed to determine whether the presence of deciduomata in one uterine horn would influence the responsiveness of the contralateral horn.

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2 Loeb, L., *J.A.M.A.*, 1908, **50**, 1897.

3 Krehbiel, R. H., *Physiol. Zool.*, 1937, **10**, 212.

4 Atkinson, W. B., *Anat. Rec.*, 1944, **88**, 271.

5 Selye, H., and McKeown, T., *Proc. Roy. Soc. London*, 1935, B **119**, 1.

6 Atkinson, W. B., *Endocrinology*, 1944, **35**, 193.

TABLE I.
Effect of Time and Manner of Uterine Traumatization on Deciduoma Formation in Ovariectomized Mice Receiving Progesterone (1.0 mg/day).

Group	No. of animals	Day of treatment traumatized	Manner of traumatization	No. of animals developing deciduomata
1	5	3rd	unilaterally	5
		7th	contralaterally	0
2	5	3rd	bilaterally	5
3	5	7th	"	5

Experimental. Fifteen young adult mice of the Swiss albino strain were bilaterally ovariectomized. Beginning 3 to 14 days after ovariectomy the animals were given daily subcutaneous injections of 1 mg of progesterone (Progestin). Crystalline progesterone dissolved in either sesame or peanut oil in concentrations of 10 and 20 mg/cc was used. The mice were divided into 3 groups of 5 animals apiece (Table I). In the first group, one horn of the uterus was traumatized on the 3rd day of hormone treatment by placing a longitudinal suture in its lumen. Four days later (7th day of treatment) the contralateral horn was similarly traumatized. Treatment was continued and the animals were then sacrificed on the 4th day following the second uterine operation. The uteri were bilaterally traumatized on the 3rd day of hormone treatment in the animals of the second group, and on the 7th day in the 3rd group. These animals were also sacrificed on the 4th day after traumatization. All the animals were killed with illuminating gas and were autopsied immediately.

Upon examination of the uteri of the animals in the first group in which traumatization of one uterine horn was delayed until 4 days after traumatization of the contralateral horn, deciduomata were found only in the horn first stimulated. On the other hand, deciduomata were found in both horns of the mice in the second and third groups in which the uteri were traumatized bilaterally during a single operation.

Discussion. The current results demonstrate that in progesterone-treated ovariectomized mice deciduomata present in one uterine horn inhibit the development of a second set of deciduomata in the contralateral horn. This failure of secondary deciduomata to develop is not due to inadequate hormone treatment or to the loss of uterine sensitivity under prolonged treatment. This is shown by the bilateral development of deciduomata in animals traumatized on either the 3rd or 7th day of hormone treatment. These observations seem to agree with the suggestion of Selye and McKeown[5] that the placentae are responsible for the inhibition of deciduoma formation in the pregnant rat. Moreover, the present work eliminates the fetal portion of the placenta as an important factor in the desensitization of the uterus.

The current observations have a significant bearing on the problem of superfetation. Although the validity of this phenomenon has been questioned,[7] occasional cases of true superfetation probably occur.[8] In the normal course of events, however, the suppression of ovulation and mating during pregnancy prevents the implantation of a second set of ova in the already gravid uterus. The present work emphasizes a second and perhaps equally important factor, that of the suppression of the ability of the endometrium to differentiate into decidual tissue in the presence of pre-existing placentae.

It is well known that estrogen prevents the nidation of the egg and subsequent placentation.[9,10] It has also been shown that deciduoma formation is dependent on a favorable estrogen-progesterone balance, the

[7] Weichert, C. K., *Anat. Rec.*, 1942, **83**, 511.

[8] Littleford, R. A., and Gysin, H. M., *Anat. Rec.*, 1944, **89**, 507.

[9] Smith, M. G., *Bull. Johns Hopkins Hosp.*, 1926, **39**, 203.

[10] Parkes, A. S., Dodds, E. C., and Noble, R. L., *Brit. M. J.*, 1938, **2**, 557.

reaction being inhibited by increasing amounts of estrogen.[11] However, the possibility that increased production of estrogen might be responsible for uterine desensitization during pregnancy seems to have been eliminated by the observation that as much as 20 times the minimal effective dose of progesterone fails to restore sensitivity in the pregnant mouse.[6] The true nature of the mechanism of uterine desensitization remains obscure.

Summary. When traumatization of one horn of the uterus of progesterone-treated ovariectomized mice was delayed until 4 days after the traumatization of the contralateral horn, deciduomata developed only in the horn first stimulated. On the other hand, deciduomata developed in both horns when the uterus was traumatized bilaterally during a single operation. Since the deciduomal reaction is the experimental equivalent of the maternal placenta, these observations suggest that the maternal placenta plays an important role in the desensitization of the pregnant uterus to decidua formation. This is an important factor in the prevention of superfetation.

[11] Rothchild, I., Meyer, R. K., and Spielman, M. A., *Am. J. Physiol.*, 1940, **128**, 213.

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Radiosensitivity of Lymphocytes and Granulocytes *In Vitro* According to the Method of Unstained Cell Counts.*

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The method of unstained cell counts[1] is used in the present investigation to determine the effect of x-rays on the survival of cells in suspensions.

Bacteriologically sterile cellular suspensions were subjected to x-rays and incubated at 37°C. Eosin in Tyrode's solution was added to the radiated and to non-radiated suspensions and the numbers of unstained cells were determined. As in previous investigations, the cells resistant to staining with eosin were presumed to be viable. The suspensions were further studied by the preparation of smears on cover slips and staining with hematoxylin and eosin and by Wright's method.

Cellular suspensions derived from the thymus and spleen of rabbits were exposed to 1,000 roentgen units. No changes were observed in the number of unstained cells during the first 3 hours of incubation. Following this latent period, the unstained cell count decreased rapidly; after 24 hours, nearly all the cells were stainable with eosin. In control suspensions incubated for 24 hours most of the cells remained resistant to staining.

Exposure of thymic cells to 50r caused a slight but definite decrease in the number of eosin-resistant cells after 24 hours of incubation. Between 50 and 1000r, the greater the dosage, the greater was the decrease in the number of unstained cells after incubation.

Stained smears of thymic suspensions, both before and after incubation for 24 hours, had numerous lymphocytes which were normal in morphologic appearance. Degenerated, poorly stained cells were few in number in the smears of the fresh suspension and were moderate in number after incubation. In contrast, nearly all the lymphocytes in smears of radiated and incubated suspensions were

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[1] Schrek, R., *Am. J. Cancer*, 1936, **28**, 389; *Arch. Path.*, 1943, **35**, 857; *ibid.*, 1944, **37**, 319; PROC. SOC. EXP. BIOL. AND MED., 1943, **54**, 283; *ibid.*, 1944, **57**, 348.