TABLE II.
Hemorrhagic Changes in Adrenal Cortex Following Trauma, Intraperitoneal Injections of Glucose, and Glucose Injections Plus Trauma.

Trauma only			Glucose*		Glucose plus trauma†		
Dog No.	Survival after trauma	Hemorrhages in cortex	Dog No.	Hemorrhages in cortex	Dog No.	Survival after trauma	Hemorrhages in cortex
20	5 hrs.	mild	26	congestion	31	3 hrs.	severe
21	6 "	none	27	"	32	6 "	"
22	8 "	severe	28	mild	33	5 "	"
23	5 "	mild	29	congestion	34	9½ "	"
24	11 "	"	30	"	35	4 "	"
25	4 "	slight			36	1 "	mild

* Glucose left 5 hrs. in peritoneal cavity.
† Glucose left 3 hrs. in peritoneal cavity.

In addition to the lipoid and hemorrhagic changes mentioned, evidence of nuclear vacuolization, particularly in the fascicular zone, and the presence of large numbers of mononuclear phagocytes were noted in the adrenals of the shocked dogs.

The data indicate that following trauma and the onset of secondary shock the adrenal cortex is subjected to severe functional strains leading to a marked depletion of lipoid, gross hemorrhages into the gland and vacuolization of cells. These changes are indicative of marked reduction in the functional efficiency of the cortex in this syndrome.

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The Endometrial Mole.

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It is well known since the classical experiments of Loeb that the uterine mucosa is sensitized by the corpus luteum hormone in such a manner that it reacts to traumatization with the formation of a deciduoma, a tumor closely resembling in its structure the maternal part of the placenta. It has been noted, however, that while deciduomata form readily during the first part of gestation, they will not form during the later stages of pregnancy although active corpora lutea are present in the ovary. We have repeated and confirmed these observations on rats, placing silk threads into the uterus as a

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local irritant, and we found that deciduomata may only be produced if the threads are placed between the fifth and eighth day of gestation, while later on in pregnancy the trauma does not call forth any local reaction. This, however, does not mean that the uterus is insensitive to trauma during the second half of gestation, for in another group of animals in which we slit the uterus open on one side, instead of merely irritating it with thread, the mucosa showed a very marked and rather curious reaction here described briefly.

In 12 rats one horn of the uterus was slit open between the 12th and 13th days of gestation while the other horn with its gestation sacs was not touched. Biopsy specimens taken 2 days later showed that the uterine mucosa of these rats is much thicker on the traumatized side than usual, and histologically this thickening proves to be due to a hydrops of the stroma mucosae. Five days later the mucosa is enormously enlarged and transformed into a translucent gelatinous tumor with a more or less irregular surface. Histologically this tissue consists of hydropic connective tissue derived from the stroma mucosae, and—as Figs. 1 and 2 show—it resembles

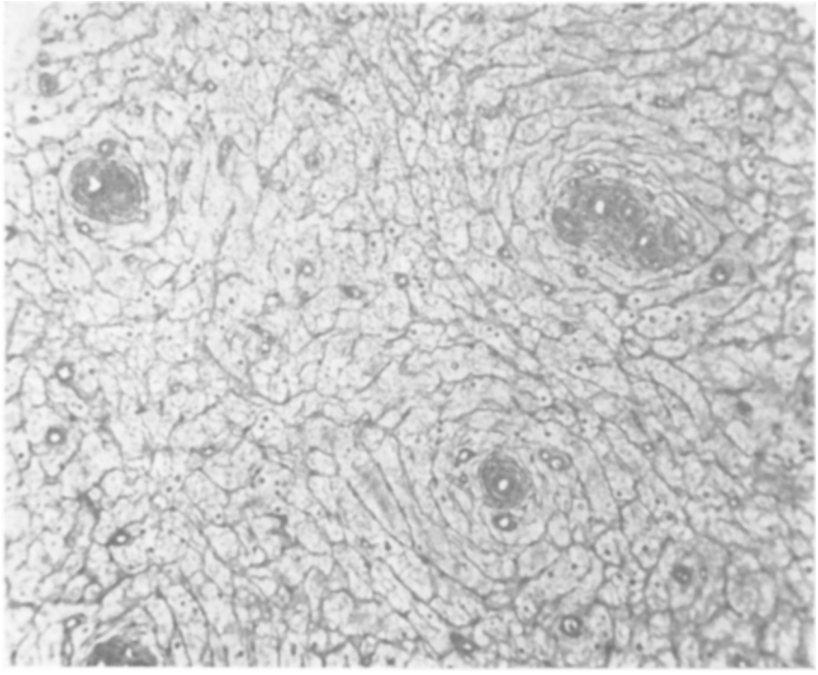

FIG. 1.

Low magnification of an endometrial mole showing the hydropic stroma and a few tubular glands in transverse section.

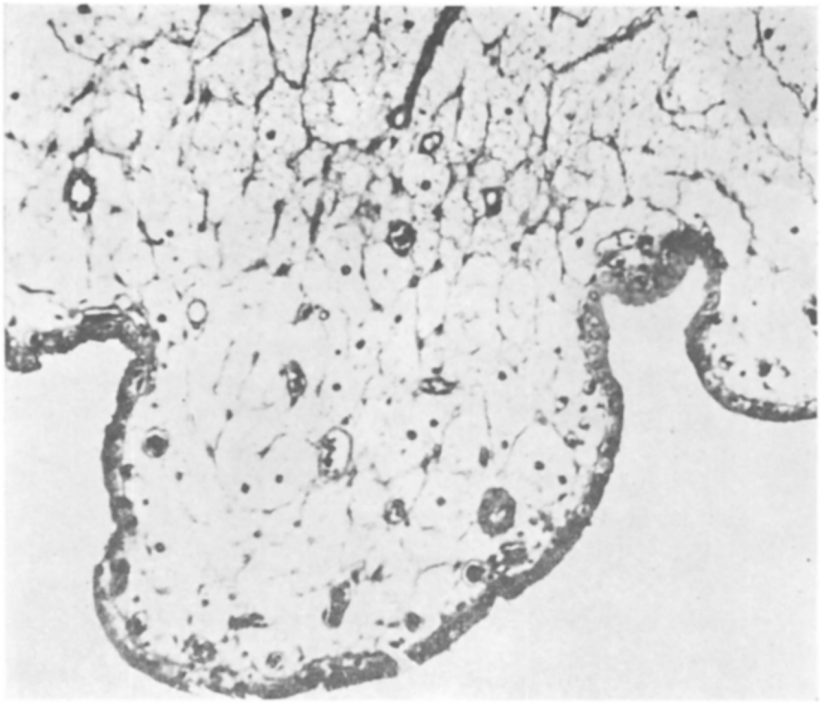

FIG. 2.
Endometrial mole at high magnification, showing the surface epithelium.

strikingly the connective tissue of a hydatidiform mole. The surface of the tumor is lined by the epithelium of the uterus. Since this tissue forms from the stroma of the endometrium and structurally resembles an hydatidiform mole we will refer to it in our discussion as the "endometrial mole" bearing in mind, of course, that there is no intimate relationship, other than the morphological similarity, between this structure and the hydatidiform mole, since the latter is derived from the foetal part of the placenta.

Since oestrin is known to inhibit deciduoma formation, we wished to see whether it would also interfere with the formation of these endometrial moles. In an experiment on 7 rats, the uterus was slit on one side between the 12th and 13th days of gestation and from then on the animals received 30 γ of oestrone, dissolved in corn oil, daily by subcutaneous injection. Biopsy on the 6th day after traumatization showed that far from being inhibited, the endometrial moles were larger in this group than in the previous one. These animals showed no vaginal oestrus. (The vaginal reaction was counteracted by the presence of the corpus luteum of gestation.)

It occurred to us that the ability of the uterus to form endometrial moles may be dependent on the simultaneous presence of oestrin and an active corpus luteum, and that the presence of oestrin, possibly formed by the placenta during the second half of gestation, may be responsible both for the formation of endometrial moles and for the impossibility to produce deciduomata at this period.

In order to subject this theory to experimental proof we treated a series of 4 lactating rats with 30 γ of oestrone daily beginning on the fifth day of lactation, at which date one horn of the uterus was slit, as in the previous experiments. Biopsy on the 5th day thereafter showed that large endometrial moles had formed under these circumstances but no deciduomata. Vaginal oestrus was not produced by this treatment owing to the presence of the corpus luteum of lactation. We might mention that control experiments showed that at this stage of lactation slitting of the uterus invariably leads to the formation of deciduomata in untreated animals. After this was established, we wanted to see how oestrin in itself would sensitize the mucosa of the uterus in the absence of corpus luteum hormone.

In 4 ovariectomized rats, treated with oestrone in the same manner as the animals of the previous series, the mucosa did not form large tumors after traumatism as it did in the experiments mentioned above, but microscopic examination showed marked hydrops of the mucosa. Since numerous control experiments have shown that no endometrial moles can be produced by trauma in the normally cyclic or ovariectomized female, it seems justified to conclude that the endometrial mole is a reaction to trauma dependent upon oestrin and particularly enhanced in the presence of an active corpus luteum. We think that this reaction is able to detect oestrin even though its vaginal effect be mashed by the simultaneous presence of an active corpus luteum. In other experiments we found that the daily administration of as large amounts as 2,000 to 3,000 γ of oestrone was unable to elicit the vaginal response in the presence of the corpora lutea of lactation or pregnancy, while as the present series shows, as small a dose as 30 γ produces unmistakable uterine reactions. The hypotheses that the corpus luteum hormone is responsible for the modification of the oestrin action, or that oestrone is transformed by the corpus luteum into a derivative with modified physiological effects, are equally compatible with our findings.

The formation of endometrial moles after trauma during the second half of gestation in the rat makes it very likely that oestrus-producing hormone is formed during pregnancy in the rodent.