(the 5th day after irradiation) the concentration of total and metabolically active corticosterone again increased in the blood of the irradiated animals (although much less so than 3 h after irradiation). Thyroid function at this period of the investigation was sharply inhibited. Against this background the time of onset of heat stroke and the period of survival of the hyperthermic animals were shorter than in rats exposed to heat during the latent period of radiation sickness; for practical purposes these indices did not differ from the control.

As was stated previously [5, 6], acute exposure to heat at all times of acute radiation sickness studied caused additional stimulation of the adrenals, although the free cortisone level showed no significant change. Thyroid function showed steadily increasing inhibition in response to hyperthermia during the development of radiation sickness.

Against the background of a sudden and considerable increase in the free corticosterone and thyroid hormone concentration in the blood, acute hyperthermia thus has its most unfavorable effect. The increased resistance of the irradiated animal to the action of a high ambient temperature was manifested only at a time of complete normalization of the initially raised indices of adrenal and thyroid function.

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PERMEABILITY OF THE PLACENTA IN RATS AFTER MASSIVE BLEEDING

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Intravenous injection of physiological saline, sigetin, and Premarin into rats on the 16th day of pregnancy had a marked effect on permeability of the placenta after blood loss.

KEY WORDS: Premarin; sigetin; permeability of the placental membrane; fluorometry.

The proper treatment of the posthemorrhagic syndrome during pregnancy requires knowledge of the effect of blood loss on the reactivity of the uterine vessels and the permeability of the placental membrane. The effect of physiological saline, sigetin,* and Premarin (increasing the intensity of the utero-placental blood flow [1]) on placental permeability for sodium fluorescein (which penetrates by simple diffusion [2, 3]) was studied in pregnant rats before and after blood loss.

*Dipotassium salt of disulfomeso-4,4-diphenylhexane.

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TABLE 1. Intensity of Fluorescence of Extracts of Embryonic Tissues after Injection of Sodium Florescein (% of standard)

Control	Number of rats	Number of fetuses	Number of measurements	Intensity of fluorescence	P
Intact					
Control	4	36	19	30,40±0,64	
Physiological saline	4	24	12	35,60±1,40	<0,001
Injection immediately after blood loss					
Control	6	48	24	32,25±1,06	
Physiological saline	12	98	49	43,42±0,80	<0,001
Sigetin	7	58	29	50,10±1,11	<0,001
Premarin	8	64	32	51,53±0,15	<0,001
Injection 24 h after blood loss					
Control	5	42	21	43,04±1,20	
Physiological saline	4	34	17	52,47±1,10	<0,001
Sigetin	6	50	25	79,40±0,20	<0,001
Premarin	6	50	25	75,20±0,21	<0,001

EXPERIMENTAL METHOD

Experiments were carried out on 71 Wistar rats on the 16th day of pregnancy. Bleeding from the jugular vein (20% of the total blood volume) was carried out over a period of 3-10 min either immediately before the experiments or on the 15th day of pregnancy. Sigetin or Premarin (10 mg of each), in a volume of 0.5 ml, or physiological saline in the same volume was injected into the femoral vein 20 min before the investigation. Sodium fluorescein was injected 10 min before removal of the fetuses.

The intensity of fluorescence of the fetal tissues was measured with the model FLYuM fluorometer. The results were expressed as percentages of the standard intensity [2].

EXPERIMENTAL RESULTS

The results are given in Table 1. Injection of 0.5 ml physiological saline into the femoral vein of intact animals caused a small but statistically significant increase in permeability of the placental membrane. Blood loss sharply increased this effect of physiological saline, evidently because of the increased sensitivity of the uterine vessels to an increase in the circulating blood volume. Intravenous injection of sigetin and Premarin into animals after blood loss (especially 24 h thereafter) also increased the permeability of the placenta very considerably. This increase was more marked than in intact animals [1, 2].

In pregnant animals blood loss thus increases the permeability of the placenta to sodium fluorescein and intensifies the response of the utero-placental circulation to physiological saline, sigetin, and Premarin.

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