EFFECT OF ADRENALIN ON HEAT-INDUCED DISTURBANCE OF SPERMATOGENIC FUNCTION IN RATS

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It is well known that spermatogenesis in man and most mammals can take place only within a strictly defined temperature range. If the environmental temperature of the testes rises, spermatogenic function is distrupted and sterility results. This is observed in artificial cryptorchidism, local heating of the scrotum, and also in general hyperthermia. However, the question of the mechanism of the action of heat on the male gonads still remains a matter for debate. There is evidence that 24 h after the operation of artificial cryptorchidism the glucose content in rabbit testicles falls by 12% [3]. During perfusion of rabbit testicles at a high temperature, glucose uptake from the solution was observed to decrease [4]. Sheep spermatozoa, incubated at 40.5°C, consumed 30% less glucose than when incubated at 34.5°C [6]. Immersion of the scrotum in water at a temperature of 38-41°C increased the oxygen comsumption of the testes in rats [5] and sheep [7]. The blood flow in the abdominal testicles of rats was reduced to 0.098 ml/min, compared with 0.325 ml/min in the scrotal testicle [2].

All these phenomena, observed as a result of exposure of the gonads to heat, are similar to the effect of adrenalin. To this it can be added that injections of adrenalin into rats for 2 weeks caused degeneration of the spermatogenic epithelium [1]. The question accordingly arose: Can adrenalin participate in heat damage to the testicles? It can be tentatively suggested that as a result of exposure to heat the reactivity of the testicles to adrenalin is increased. The investigation described below was undertaken to test this hypothesis.

EXPERIMENTAL METHOD

Experiments were carried out on male albino rats weighing 200-300 g. The animals were divided into four groups. Group 1 consisted of intact animals. The rats of group 2 received subcutaneous injections of adrenalin twice a day in a dose of 0.5 mg/kg for 5 days. The scrotum of the animals of group 3 was heated by water at a temperature of 41°C for 30 min, for which purpose the rats were placed in special slings. The rats of group 4 were subjected to heat in the same way, but 5 min before immersion of the scrotum in water, they were given an intraperitoneal injection of 0.5 mg/kg adrenalin, followed by a subcutaneous injection of the same dose of adrenalin once a day for 5 days. The animals were decapitated after 5 days. The two testes were weighed, after which the left testis was fixed in Carnoy's fluid and embedded in paraffin wax. Sections 5-7 μm thick were stained with hematoxylin and eosin. The

TABLE 1. Weight and Histological Structure of Testes in Rats Treated with Adrenalin, Exposed to Heat, and a Combination of Both (M ± m)

Group of rats	Number of rats	P	Weight of testes, mg%	Percentage of seminiferous tubules		
				with normal spermatogenesis	which had lost two generations of sex cells	which contained only spermatogonia and Sertoli cells
1	12	—	1163±30	86,8±1,7	—	—
2	10		1088±35	82,3±2,0	—	1,3±0,5
P		1,2	>0,05	>0,05		
3	12		845±46	9,7±1,7	17,8±2,3	1,5±0,5
P		1,3	<0,001	<0,001		
4	10		721±24	0,8±0,7	42,8±6,7	9,8±2,3
P		3,4	<0,05	<0,001	<0,01	<0,01

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TABLE 2. Effect of Adrenalin, Local Heating of the Scrotum, and a Combination of Both on Number of Spermatozoa and Their Activity (M ± m).

Group of rats	P	Number of spermatozoa in 1 mm³ physiological saline	Percentage of active spermatozoa
1	—	783±156	68,5±5,0
2		508±45	51,1±2,1
P	1,2	>0,05	<0,01
3		841±73	42,4±4,3
4		>0,05	<0,01
P	1,3	203±58	0,0
P	3,4	<0,001	

number of spermatozoa in the tail of the epididymis and their motility also were studied. For this purpose the tail of the epididymis was cut into two halves, which were placed in a bottle with 20 ml of physiological saline. After shaking of the bottle a sample was taken and the number of spermatozoa in 1 mm³ of the solution was determined in a Fuchs–Rosenthal counting chamber. The numerical results were subjected to statistical analysis.

EXPERIMENTAL RESULTS

As the data in Table 1 show, immersion of the rats' scrotum in water at a temperature of 41°C for 30 min led after 5 days to a decrease in weight of the testes. Injections of adrenalin for 5 days did not affect the weight of the testicles. However, combined exposure to heating and adrenalin led to a further fall in weight of the gonads. Injections of adrenalin did not affect the number of seminiferous tubules with normal spermatogenesis, but led to the appearance of a few tubules which contained only spermatogonia and Sertoli cells. Immersion of the scrotum in water at a temperature of 41°C for 30 min led after 5 days to a sharp decrease in the number of tubules with normal spermatogenesis. Combined exposure to heat and adrenalin caused an increase of almost 2.5 times in the number of tubules which had lost two generations of sex cells and to an increase of 6.5 times in the number of tubules which contained only spermatogonia and Sertoli cells.

As the data in Table 2 show, the number of spermatozoa removed from the tail of the epididymis decreased significantly only after combined exposure to heat and adrenalin. Injection of adrenalin alone, like heating alone, did not affect the number of spermatozoa. Nevertheless, these procedures both reduced percentage of active spermatozoa. With a combination of heating and injections of adrenalin, no active spermatozoa whatever were found.

Thus, although injection of adrenalin for 5 days into control rats was reflected neither in the weight of the testes nor in the number of tubules with normal spermatogenesis and had only a slight effect on spermatozoa from the tail of the epididymis, it began to exert a harmful effect on the gonads when they were also exposed to heat. During combined exposure to these factors, as the experiments showed, there was a further decrease in weight of the testes, with disappearance of active spermatozoa and a sharp reduction in the number of tubules with normal spermatogenesis. The impression is gained that the sensitivity of the testes to exogenous adrenalin was enhanced by exposure to heat. On this basis it can be postulated that the reactivity of the testicles to endogenous adrenalin may also be enhanced by a rise of temperature. Consequently, adrenalin can be regarded as an internal factor participating in heat damage to the male sex glands.

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