EFFECT OF UNILATERAL CASTRATION OF THE TESTIS IN ARTIFICIAL CRYPTORCHIDISM

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The right testis was fixed in the body cavity in adult male rats. Three weeks after this operation the intact left testis was removed. At the same time left-sided castration was performed on normal males. Fifteen days after unilateral castration compensatory hypertrophy of the testis in the normal males was virtually absent. Removal of the intact left testis of the cryptorchid rats led to a substantial increase in weight of the abdominal testis. Inhibition of spermatogenesis in the abdominal testis thus permits the development of its compensatory hypertrophy.

Compensatory hypertrophy of the testis after removal of the contralateral testis is the result of interaction between the gonadotropic function of the pituitary and the testis. It is based on correlations of the plus-minus type [1]. However, the power to undergo compensatory hypertrophy is lost in rats on the 23rd-24th day of life, which coincides with the time of activation of spermatogenesis [3]. Attempts to restore the power of compensatory hypertrophy in adult male rats by inhibiting spermatogenesis by injections of estradiol dipropionate have proved unsuccessful [2]. Inhibition of spermatogenesis was incomplete and no compensatory hypertrophy of the testis was obtained.

An investigation was accordingly carried out to determine whether unilateral castration of the adult rat can stimulate a testis artificially replaced in the body cavity. Such a testis is characterized by the absence of spermatogenesis and by destruction of the germinal epithelium.

EXPERIMENTAL METHOD

Male rats with a mean initial body weight of 217 g were divided into four groups. Group 1 consisted of animals undergoing left-sided castration. In the rats of group 2 a mock castration operation was performed. Group 3 consisted of rats in which the right testis was implanted through an incision in the scrotum into the body cavity where it was fixed. The left testis remained intact. Three weeks after the operation the intact left testis was removed from the animals of this group. The rats of group 4 underwent the same operation of formation of a fixed abdominal testis, but 21 days later a control mock castration operation was performed on them. The left-sided castration, like the control operations, was carried out on the same day. The rats were killed 15 days after castration (and the corresponding control operation) with chloroform. The weight of the right testis and the weight of the prostate gland were determined. The testes were fixed in Bouin's fluid and treated by histological methods. In every case the right testis was compared with the right testis of the corresponding control group.*

*The student G. A. Kurasova helped with the experiments.

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TABLE 1. Effect of Unilateral Castration on the Testis in Artificial Cryptorchidism

Group	Character of operation	No. of animals	Body wt. (in g)	Weight of right testis			Diameter of seminiferous tubules (in μ)	Weight of prostate		
				mg	mg/100 g body wt.	%		mg	mg/100 g body wt.	%
1	Left-sided castration	10	261,6	1311±58,3	501,1±26,0	106,8	286,2±2,52	477,1±36,0	182,4±18,3	98,7
					$P_{1-2}>0,05$					
2	Control operation	12	268,3	1259±25,7	469,2±15,3	100,0	289,1±5,46	486,0±26,8	181,1±7,44	100,0
3	Abdominal fixation of right testis and left-sided castration	15	257,0	472±25,0	183,7±9,63	126,7	155,3±6,44	514,9±22,3	200,3±9,22	128,0
				$P_{3-4}<0,02$	$P_{3-4}<0,02$			$P_{3-4}>0,05$	$P_{3-4}>0,05$	
4	Abdominal fixation of right testis and mock castration	11	263,5	382±23,0	145,0±11,2	100,0	163,7±5,74	412,5±48,5	156,5±20,5	100,0

EXPERIMENTAL RESULTS

It will be clear from Table 1 that left-sided castration of the normal males induced a very small increase (not statistically significant) in the size of the residual right testis. The weight of the absominal testes in both groups of cryptorchid rats was sharply reduced. The diameter of the seminiferous tubules was greatly reduced. In the control group of cryptorchids the wall of the seminiferous tubules in 58.3% of individuals consisted chiefly of Sertoli cells. Only in a few tubules were spermatogonia and isolated spermatocytes found. In 25% of animals, spermatogenesis was present in some of the seminiferous tubules, but it proceeded only as far as the spermatid stage. In one rat, spermatids were present in all the tubules. In two rats (16.6%), spermatozoa were present in the tubules.

Both the absolute and the relative weight of the residual abdominal testis was significantly increased in the rats from which the intact scrotal testis had been removed, i.e., compensatory hypertrophy took place. Although the weight of the prostate also was increased, the increase was not significant ($P > 0.05$). The histological structure of the testis in this group was similar to the structure of the abdominal testis in the control group. In 46% of the rats the wall of the tubules consisted chiefly of Sertoli cells. In some tubules a few spermatogonia and spermatocytes were seen. Spermatogenesis was present in 12% of the tubules in the abdominal testis of 38.5% of the animals and it proceeded as far as the spermatid stage. Spermatozoa were present in the seminiferous tubules of two rats.

Unfortunately, it was impossible to compare the weight of the testis directly with its structure. However, a calculation was carried out which enabled the compensatory hypertrophy of the abdominal testis to be compared to some extent with its structure. It was found, for instance, that 13% of rats with cryptorchidism did not respond to removal of the scrotal testis: the mean weight of the abdominal testis was actually less than the mean weight of the abdominal testis in the control group. On the other hand, spermatozoa were found in some tubules in the abdominal testis of 15% of the animals of the unilaterally castrated group. In 84% of rats of this group the weight of the abdominal testis exceeded that of the same testis of the control animals. In 84.5% of rats of the experimental group, spermatozoa were completely absent from the seminiferous tubules and spermatids were present in only some of them. The closeness of the percentages compared is apparently not accidental. Presumably when spermatozoa are present in the seminiferous tubules, compensatory hypertrophy of the abdominal testis becomes impossible. Consequently, depression of spermatogenesis to a degree in which spermatozoa are absent from the tubules and spermatids are present in only some of them makes the development of compensatory hypertrophy of the testis possible under corresponding conditions.

The absence of compensatory hypertrophy of the testis in adult rats could be connected with the action of the blood-testis barrier, preventing the factor produced by maturing sex cells from penetrating into the blood stream [2, 3]. If this hypothesis is accepted, the physiological role of the production of this factor, not penetrating into the blood stream, still awaits an explanation. There is some evidence that the factor produced by the sex cells does penetrate into the blood stream, for secretion of follicle-stimulating hormone is sharply reduced when spermatozoa appear in the testis [8]. In the writer's view, the only function of the blood-testis barrier is protection of the sex

cells against autoimmune reactions, and this barrier plays no part in the phenomenon of disappearance of the power of compensatory hypertrophy of the testis. In order to explain that phenomenon it must be assumed that active spermatogenesis, especially if reaching the stage of late spermatids and spermatozoa, is combined with the production of a substance maintaining the gonadotropic function of the pituitary and, in particular, the secretion of follicle-stimulating hormone [4-8], to which the name inhibin has recently been given. Unilateral castration of the male reduces the production of this factor by half. However, the hypothalamo-hypophyseal system does not undergo excitation despite the halving of inhibin and testosterone production. It is postulated that the reason is the high sensitivity of the hypothalamo-hypophyseal system to the inhibitory action of inhibin which continues to be formed by the remaining gonad. Under these conditions compensatory hypertrophy of the testis cannot arise. In the experiments in which the testis was fixed in the abdominal cavity, however, in most cases spermatogenesis either was completely absent or was severely inhibited, as a result of which that testis did not produce inhibin. Removal of the intact scrotal testis implied loss of the only source of inhibin and a decrease in androgen production. As a result the secretion of gonadotropins was increased, thereby stimulating the residual intra-abdominal testis. This stimulation occurred despite the fact that the intra-abdominally fixed testis is less reactive than the intact organ to the action of gonadotropins [5].

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