Pituitary Response of Cryptorchid Dogs to LH-RH-Analogue before and after Sexual Maturation

Eiichi KAWAKAMI, Sumito HIRAYAMA, Toshihiko TSUTSUI, and Akira OGASA

Department of Reproduction, Nippon Veterinary and Animal Science University, 1-7-1 Kyonan-cho, Musashino-shi, Tokyo 180, Japan

(Received 29 May 1992/Accepted 8 October 1992)

ABSTRACT. An LH-RH-analogue (LH-RH-A) was administered to 5 cryptorchid (CR) and 6 normal beagles before and after sexual maturation and peripheral plasma LH and testosterone levels were measured. The response patterns of plasma LH levels for LH-RH-A in the CR dogs were similar to those in the normal dogs before and after sexual maturation. It was therefore demonstrated there is little difference in pituitary response to LH-RH-A in the CR and normal dogs. However, the maximum levels of testosterone in the CR dogs were generally lower than those in the normal dogs.—**KEY WORDS:** cryptorchidism, dog, LH-RH-analogue.

J. Vet. Med. Sci. 55(1): 147–148, 1993

The authors have reported that the peripheral plasma LH and testosterone (T) levels of adult congenitally cryptorchid dogs are lower than in normal dogs [5]. There have not, however, been any reports on pituitary response to LH-RH in cryptorchid (CR) dogs. In this study, an LH-RH-analogue (LH-RH-A) was administered to CR dogs before and after sexual maturation and the levels of peripheral plasma LH and T were examined. The pituitary response to LH-RH-A and testicular response to LH of CR dogs were assessed on the basis of the results.

Five congenitally CR beagles (two twenty-week-old and three one-year-old) and six normal beagles (three twenty-week-old and three one-year-old) were used in this study. They were found in the beagles linebred at the authors' laboratory. Cryptorchidism was unilateral in all of the CR dogs, except one twenty-week-old dog in which it was bilateral. The LH-RH-A used in this study was Fertirelin acetate, [Des-Gly-NH₂¹⁰, Pro-ethylamide⁹]-LH-RH (Conceral supplied by Takeda Chemical Industries, Japan). All of the CR and normal dogs received an intramuscular injection of 25 µg of LH-RH-A. Peripheral plasma was collected from a superficial leg vein at 10 or 30 min intervals until 180 min after the injection. Plasma LH was

measured by means of a double-antibody radioimmunoassay in accordance with the procedure reported by Nett *et al.* [7], except that radioactivated porcine LH (LER-778) and anti-porcine LH serum were utilized as previously reported [5]. Plasma T levels were determined by the radioimmunoassay method described by Makino *et al.* [6].

The peripheral plasma LH and T levels of the twenty-week-old normal and CR dogs are shown in Fig. 1. The LH peak occurred 10 min after the injection of LH-RH-A in all three normal dogs. Plasma T in the normal dogs reached the maximum either 90 or 120 min after the injection. The LH peak of all of the CR dogs occurred 20 min after the injection. There was no difference between LH peaks in the normal and CR dogs injected with LH-RH-A. The plasma T levels of all CR dogs reached the maximum at 90 min after the injection, but were lower than in the normal dogs. The peripheral plasma LH and T levels of the one-year-old normal and CR dogs are shown in Fig. 2. The LH peak occurred 20 min after the injection of LH-RH-A in all three normal dogs. LH levels returned to the base line 60 or 90 min after the injection. Plasma T levels in the normal dogs reached the maximum either 60 or 90 min after the injection. In the normal dogs the

Fig. 1. Peripheral plasma LH and testosterone levels in three normal (left) and two cryptorchid (right) twenty-week-old dogs after injection of 25 µg of LH-RH-A.

Fig. 2. Peripheral plasma LH and testosterone levels in three normal (left) and two cryptorchid (right) one-year-old dogs after injection of 25 μ g of LH-RH-A.

maximum LH and T were obviously higher than at twenty weeks of age. The LH levels of the CR dogs peaked 10–30 min after the injection. There was no difference between the LH peak of the normal and the CR dogs. The levels in the CR dogs subsequently fell to base line by 120 or 150 min after the injection. The basal levels of the CR dogs were obviously lower than those of the normal dogs. Plasma T levels in the CR dogs reached the maximum either 60 or 90 min after the injection. Although the maximum levels were obviously higher than at twenty weeks of age, the levels in the CR dogs were generally lower than in the normal dogs both 60 and 90 min.

Jones *et al.* [4] reported that the plasma LH and T levels of normal adult dogs peaked 15 min and 40 min, respectively, after an intravenous injection of LH-RH. Their results are similar to those obtained in the present study. The levels of LH peak of both the normal and CR dogs were higher after sexual maturation than before sexual maturation because of increased pituitary responsiveness to LH-RH-A. The response patterns of plasma LH levels for LH-RH-A in the CR dogs were similar to those in normal dogs. It has been demonstrated there is little difference in pituitary response to LH-RH before and after sexual maturation in the normal and CR dogs. Because of the lower basal levels of LH in CR dogs than in normal dogs, however, it is presumed that the LH-RH secretory function of the hypothalamus of CR dogs is for some reason inferior to that of normal dogs. It has been hypothesized that insufficient gonadotropin secretion by the pituitary gland is responsible for cryptorchidism [3, 8].

Thus it would seem that the pituitary function of infant CR dogs should be examined in a future study.

It was known that T synthesis by the Leydig cells of the testis is known to be suppressed by high temperature [1, 2]. Hence, it is suspected that the rise is less than in normal dogs because of a lower Leydig cell response to LH in the CR testis. Moreover, plasma T levels of bilaterally CR dogs were lower than that of unilaterally CR dogs at all times during the experiment before sexual maturation. This suggests that the T secretory function of CR testes is inferior to that of normal testes.

REFERENCES

- Bergh, A., Berg, A. Å., Damber, J. E., Hammar, M., and Selstam, G. 1984. *Acta Endocrinol.* 107: 556–562.
- Farrer, J. H., Sikka, S. C., Xie, H. W., Constantinide, D., and Rajfer, J. 1985. *Fertil. Steril.* 44: 125–132.
- Hutson, J. M. and Donahoe, P. K. 1986. *Endocrine Reviews* 7: 270–283.
- Jones, G. E., Baker, K., Fahmy, D. R., and Boyns, A. R. 1976. *J. Endocrinol.* 68: 469–474.
- Kawakami, E., Tsutsui, T., and Ogasa, A. 1989. *Jpn. J. Vet. Sci.* 52: 179–181.
- Makino, T., Inano, K., Yoshida, T., Den, N., Takagi, S., and Kanbegawa, A. 1973. *Clin. Endocrinol. (Tokyo)* 21: 867–873 (in Japanese).
- Nett, T. M., Akbar, A. M., Phemister, R. D., Holst, P. A., Reichert, L. E. Jr., and Niswender, G. D. 1975. *Proc. Soc. Exp. Biol. Med.* 148: 134–139.
- Radhakrishnan, J., Morikawa, Y., Donahoe, P. K., and Hendren, W. H. 1979. *Invest. Urol.* 16: 365–368.