DIAGNOSTIC AND THERAPEUTIC USE OF LH-RH

IN THE INFERTILE MAN

Luis Schwarzstein
G.E.F.E.R. (Grupo de Estudios en Fertilidad y Endocrinologia de Rosario)
Cordoba 1764, Rosario, Argentina

I would first like to acknowledge the organizers of this Symposium for their kind invitation. I am highly honoured by the opportunity given to me to join my efforts to those of colleagues working in the same field.

As an introduction, I should like to make a brief outline of the general knowledge which held the field of pathophysiologic interpretation and treatment of male infertility before the availability of LH-RH.

The cause of male infertility could be ascribed to a deficiency of gonadotrophic secretion (hypogonadotrophic hypogonadism) and urologic factors, as the most common etiology and also to specific testicular damage due to chromosomal, genetical, infectious and other causes, or several diseases or external factors, as well as clinical extragonadal diseases with alteration at the seminiferous tubular level.

However, it was not possible to find an explanation for several cases of oligospermia in the absence of evident modifications of the basal gonadotrophic levels. This last group, idiopathic normogonadotrophic oligospermia (I.N.O.) is still lacking a known etiology and its pathophysiology and treatment remain, up till now, as a gap in the knowledge of modern reproduction and endocrinology. I am not going to deal with immunological factors, since they form a vast and specific field in themselves.

Treatment of I.N.O. was based on the assumption that over-stimulation of the hypothalamo-hypophyseal-gonadal axis would lead to
an improvement of spermatogenesis. However, several patients treated with different treatment schemes, suitable for that purpose, failed to reach success. An approach to the prognosis of therapeutic possibilities was based on histological findings of the testis (Meinhard et al., 1973; Schwarzstein, 1974; Schwarzstein et al., 1975 a, b; Aafjes and Van Der Vijver, 1974). As a group, much better results are obtained in patients with less damage at the tubular level, as it can be seen in Figure 1 describing treatment of patients treated with HMG. These data could have been predicted since it is reasonable to expect a better therapeutical result among patients with less damage.

Fig. 1: Changes in sperm concentration during treatment with HMG in 12 oligospermic subjects.

Type I patients: Hypospermatogenesis or arrest up to the spermatid stage.

Type II patients: Arrest at spermatocyte I or spermatogonial stages.

However, there are several patients, without a very severe damage, in whom therapy is ineffective and on the other hand, in few pa-