HEAT INDUCED INHIBITION OF SPERMATOGENESIS IN MAN

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A reversible depression in spermatozoa output was reported by several authors who have induced an increase in body or scrotal temperature in man (Table 1). In most of the experiments, this rise was above the physiological temperature of the body. But there was a possibility, as strongly suggested by Robinson and Rock (1967), that a sustained yet relatively slight increase in the testis temperature might affect fertility in a way adequate to be used as a method of male fertility control.

From these experiments and with a group of men demanding male contraception, we invented a new method of testicular heating which uses the body as a heat source. The results presented here are general and concern work that was started in 1982; some results were in part reported (Mieusset et al., 1985, 1987a, 1987b). With this method testes were pushed up into the inguinal canal and kept there. In such a situation testes stay in a hotter surrounding, since the inguinal canal temperature is 1 to 1.5°C higher than the intrascrotal one (Kitayama, 1965). When testes are in the inguinal canal, their inferior pole is located at the superior part of the root of the penis. Testes were maintained daily during waking hours in such a situation by means of two techniques.

With the first technique (Fig. 1), support was ensured by briefs provided with an orifice through which the penis and scrotum were pushed outside. With this technique, testes were relatively free to travel due to the elasticity of the fabric, and tended to descend slightly, bringing their lower pole closer to the scrotal cavity. Because such movement was possible, we thought the rise in testicular temperature was liable to fluctuate.

In the second technique, a ring of soft material was either added to encircle the orifice of the briefs, or was worn alone. Testes were therefore exposed to a more constant increase in surrounding temperature.

Twenty-one unpaid volunteers (aged 27 to 35 years) were involved in this experiment: 13 with Technique 1 and 8 with Technique 2. The timing included a 4 to 6 month-baseline study, a 6 to 24 month-heating period and an 18 month-survey after heating. Every man was subjected to semen analyses with sperm count, motility and morphology, as well as clinical examinations.

RESULTS

The data of this study confirm the well known depression in spermatozoa output induced by an increase in testicular temperature (Fig. 2). But the results reported here

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Table 1. Induced "hyperthermia" in man

Authors	Number of Men	Heated Organ	Temperature	Exposure Time
MacLeod and Hotchkiss (1941)	6	Body	41 °C	45 min 1 day
Watanabe (1959)	18	Scrotum	44-46 °C	30 min/day 1-12 days
Procope (1965)	12	Body	+1 °C in rectal temp.	15 min/day 8 da/2 wk
Robinson and Rock (1967)	10	Scrotum	+0.8 °C in scrotal temp.	waking hrs 6-10 weeks
Robinson et al. (1968)	18	Scrotum	42.5 °C	30 min/day 14-28 days
French et al. (1973)	5	Scrotum	+2 °C	30 min/day 5 days
Brown-Woodman et al. (1983)	5	Body	+0.7 °C in rectal temp.	20 min 1 day

were obtained with a slight and sustained increase in testis temperature. The most important inhibitory effect is given by Technique 2, where it is greater than 97% after 3 months of heating. With this technique, the depression in the spermatozoa output appears sooner, is deeper and more constant than with Technique 1. For both techniques, recovery occurs within 6 to 8 months after the heating is stopped.

More important is the fact that heating the testis induced a depression not only in the amount but also in the quality of the spermatozoa output. This alteration in quality was observed in sperm motility as well as in sperm morphology.

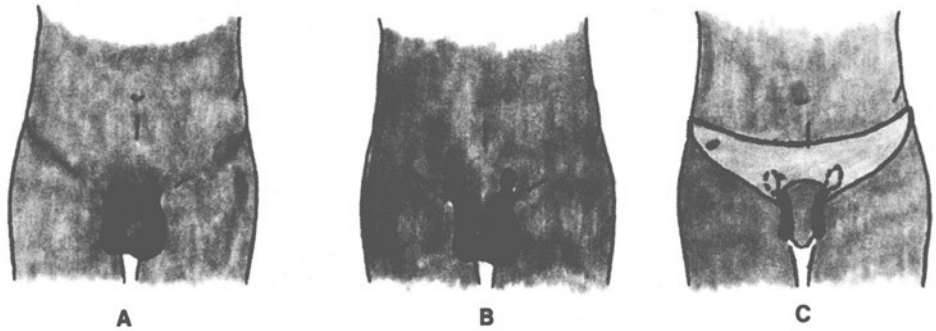

Figure 1. Draft of the first technique (T_1) used.
A. Testes in scrotal position.
B. Testes in low inguinal location, with an empty scrotum.
C. Testes maintained in the same position than in B by means of special briefs; the empty scrotum is outside of the briefs.