

Measurement of intrascrotal temperature in normal and subfertile men*

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Summary. Intrascrotal temperatures were measured bilaterally by a non-invasive method in 300 subfertile men (mean sperm count $21.4 \times 10^6/\text{ml}$) and 30 normospermic control men (mean sperm count $118.7 \times 10^6/\text{ml}$). The subfertile men had mean (s.d.) temperatures of 34.7°C (0.8) for the right and 34.8°C (0.7) for the left testis. The value for both testes of the control men was 33.4°C (0.6). The difference ($1.3\text{--}1.4^\circ\text{C}$) was significant ($P = 0.03$). An intrascrotal temperature of $> 34.1^\circ\text{C}$ was found in $> 83\%$ of subfertile men, regardless of clinical diagnosis. This method can therefore be used to survey large numbers of men. We suggest that small intrinsic temperature increases may interfere with the ability of the testis to accommodate to environmental temperature stresses and so lead to abnormal semen and subfertility.

Keywords: men; subfertility; testis; temperature; intrascrotal thermometry

Introduction

Extrinsic thermal stress to the scrotum has long been known to cause testis alterations and semen reflecting spermatogenic damage. That intrinsic elevation of scrotal temperature could play a role in men with subfertile semen has received much less attention. Davidson (1945) stated, without elaboration, that over half of his patients had a disturbance of testis temperature regulation. Robinson & Rock (1967) used scrotal/rectal temperature differential (SRD) to document differences in testis temperature between normal and oligospermic men. Their observation, not subjected to statistical evaluation, was that the mean SRD was 2.4°C in 36 normal men and 1.9°C in oligospermic men. Agger (1971) noted small elevations over normal men and concluded that a temperature increase could not be excluded in varicocele patients. Zorngiotti & MacLeod (1973) showed significant increase in intrascrotal temperature ($0.6\text{--}0.7^\circ\text{C}$) in a group of oligospermic varicocele patients over normospermic controls. Mieusset *et al.* (1987) demonstrated increases in mean scrotal temperature (right $+0.4$ and left $+0.5^\circ\text{C}$) in 150 infertile men compared with 37 fertile controls; these elevated values were above the 90th percentile for values of fertile men. Zorngiotti *et al.* (1980) devised a testicular hypothermia device which lowered temperature 2.0°C , demonstrating improvement in the semen of subfertile men and pregnancies in long-term infertile marriages.

A key factor in testis temperature study is thermometry. Thermistors require invasion into the testis making large scale studies difficult. Thermistors are not ideal for other reasons: e.g. use of anaesthesia (Waites, 1970) and evaporation of a liquid (skin preparation) applied to the scrotum will alter temperature (Zorngiotti *et al.*, 1980).

Methods which measure emissivity of the skin by infrared thermometry and thermography reflect the temperature of the underlying testis (Comhaire, 1986). Because of problems inherent in electronic devices as well as variations in skin emissivity, none of the instruments currently on the

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market and tried by us have the sensitivity ($\pm 0.1^\circ\text{C}$) needed to discriminate small differences. Similarly, liquid crystal thermometry is not sufficiently sensitive.

A non-invasive method for intrascrotal temperature was evolved by Zorngiotti & McLeod (1973). Readings are made by drawing the loose anterior scrotal skin about the bulb of a mercury laboratory thermometer placed against the scrotum overlying the anterior testis. A high degree of instrument accuracy is inherent since the thermometer measures absolute temperature (divisions = 0.05°C). Insertion of the thermometer within the scrotum during surgery and comparison with the external method yielded a mean difference of 0.1°C (Zorngiotti & MacLeod, 1973). Intrascrotal temperature reflects testicular temperature since the testis and epididymis constitute the largest thermal mass in the hemiscrotum. Factors which could alter the reading (e.g. scrotal skin temperature, scrotal skin circulation and heat coefficient of the glass and mercury prewarmed thermometer) are negligible. A printout of our calculations is available.

The aim of this study was to test the validity of the non-invasive testis temperature method and to evaluate the possible connection between low level intrinsic elevation of testis temperature and subfertility.

Materials and Methods

The 300 consecutive men (mean sperm count $21.4 \times 10^6/\text{ml}$) who presented for infertile marriage and abnormal semen (azoospermic men were excluded) had history, physical and other examinations including serum follicle-stimulating hormone (FSH) determination when appropriate. The 30 control men (normospermic; mean sperm count $118.7 \times 10^6/\text{ml}$) without proven fertility had examination of the genitalia only. All subjects were ambulatory and most were 20–40 years of age and, except for diabetes mellitus in some, were in good health. All had two semen analyses by independent laboratories specializing in fertility diagnosis. Semen was considered abnormal when the sperm count was $<20 \times 10^6/\text{ml}$, there were $<40\%$ actively motile spermatozoa, and there were $<60\%$ oval forms.

Intrascrotal temperature was estimated by A.W.Z. or by one of three Physician's Assistants, after an equilibration period. The subject was disrobed and laid supine for 6 min in an ambient temperature of $21\text{--}23^\circ\text{C}$. The calibrated mercury immersion thermometer was prewarmed to 36.0°C and quickly placed against the scrotum over the most prominent part of the anterior testis. The scrotal skin was then gently drawn about the bulb, 'immersing' it completely. The temperature was recorded when the falling mercury column reached equilibrium, usually in about 8 sec. The thermometer has a range of $32\text{--}40^\circ\text{C}$ with divisions of 0.05°C (Model No. C1148: Brooklyn Thermometer Company, Farmingdale, NY 11735, U.S.A.). Frequency distributions of all men were plotted and mean intrascrotal values for subfertile and control men were compared by Student's *t* test (unpaired).

Genital examination was made standing. Varicocele or fullness of the spermatic cord upon voluntary increase in abdominal pressure had to be palpated, otherwise varicocele was considered not to be present. Patients were classified as shown in Table 1.

Table 1. Mean temperature ($^\circ\text{C}$) of the right and left testes of normospermic and subfertile men

	No. of men	Mean temp. (s.d.)	
		Right	Left
Normospermic controls	30	33.4 (0.6)	33.4 (0.6)
All patients	300	34.7 (0.8)	34.8 (0.7)
Varicocele left	76 (25.3%)	34.7 (0.6)	34.8 (0.6)
Varicocele right	2 (0.7%)		
Varicocele bilateral	32 (10.7%)	34.7 (0.5)	34.8 (0.6)
Varicocelectomy failure	86 (28.7%)	34.7 (0.7)	34.7 (0.7)
No palpable varicocele (idiopathic)	86 (28.7%)	34.7 (0.7)	34.8 (0.8)
Cryptorchidism	10 (3.3%)	34.9 (0.6)	34.7 (0.7)
Other	8 (2.7%)		

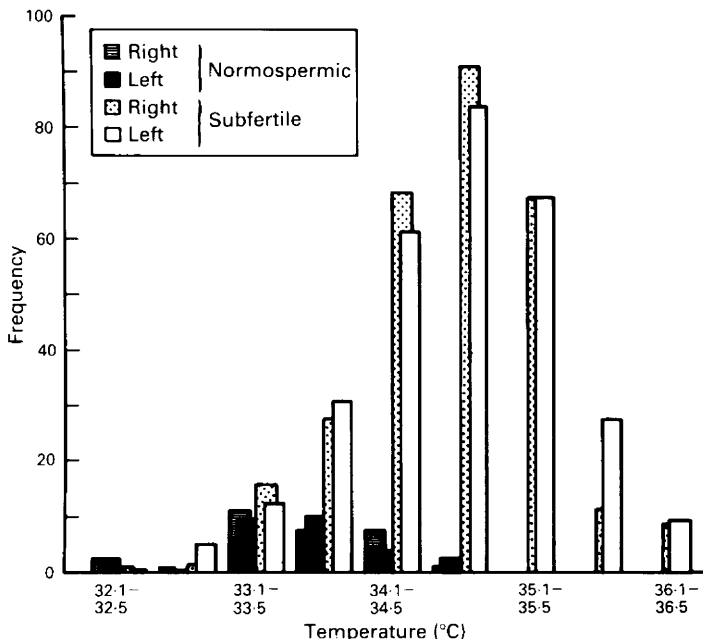


Fig. 1. Temperature/frequency distribution of 30 normospermic men and 300 subfertile men.

Results

The frequency distributions of right and left intrascrotal temperatures were symmetrical for the subfertile men but skewed to the right for the control men (Fig. 1). There were peaks at 34.6–35.0°C in the subfertile men and at 33.1–33.5°C in the normospermic controls, with overlap. Mean temperatures of the various types of men are given in Table 1, and all values for the subfertile men were significantly higher than those for the controls ($P < 0.03$). There were no temperature differences according to semen parameter (i.e. count, motility and morphology): 54% of the subfertile men (160/300) had counts $< 20 \times 10^6$ /ml while 78% (235/300) had motility and 85% (255/300) had morphology values below the minimum values for normal semen. FSH determinations for 122 of the subfertile men gave values of < 15.8 mi.u./ml (normal range) for 100 men and > 16.0 mi.u./ml (elevated) for 22 men. There were no differences in mean intrascrotal temperature between either FSH group.

Discussion

The difference in intrascrotal temperature means between the subfertile and normospermic men of 1.3–1.4°C parallels observations by Lazarus & Zorngiotti (1975) on men with fever not due to intrascrotal pathology. When rectal temperature reached 37.8°C, there was a precipitous decrease in the scrotal/rectal temperature differential of 1.5°C. This rise in intrascrotal temperature can be attributed to failure of the countercurrent heat exchange of the pampiniform plexus. The 1.5°C may represent the maximum rise in temperature possible from failure of thermoregulation.

We suggest that, based upon the mean ± 2 standard deviations, an intrascrotal temperature range of 32.2–34.6°C be considered normal for right and left testes and that values of 33.1–36.3 and 33.4–36.2°C be considered elevated for right and left testes respectively although there is some

overlap. Correcting for an absent testis in 11/300 of the subfertile men, we found that 246/291 (84.5%) and 237/298 (83.2%) had an intrascrotal temperature of $> 34.1^{\circ}\text{C}$ for the right and left testis, respectively. These findings suggest that increased intrascrotal temperature is prevalent in subfertile men regardless of clinical diagnosis and that internal spermatic vein reflux or stasis which affects countercurrent heat exchange in the pampiniform plexus may be a common mechanism for elevated temperature (Zorngiotti, 1981, 1982).

With regard to the validity of the thermometric method, the narrow, symmetrical peak obtained in the subfertile men suggests that the results are homogeneous. Measurement of intrascrotal temperature, as described, indirectly reflects testis temperature for the reasons stated above. It is our opinion that actual testis temperature is a major factor influencing spermatogenesis and sperm maturation.

We suggest that the semen of subfertile men is influenced by subtle thermal forces more than is generally believed; (1) there is an intrinsic thermoregulatory defect that causes elevation of intrascrotal temperature of $\sim +1.3^{\circ}\text{C}$; (2) these small elevations affect spermatogenesis and epididymal maturation as does extrinsic heat application; (3) the chronicity of heat exposure, implicit in intrinsic elevation, may explain the progressive nature of spermatogenic failure; and (4) unlike normal men, those with impaired thermoregulatory function are not able to compensate for extrinsic accidents which have been shown to elevate temperature such as climate, hot baths, hot working conditions and clothing (VanDemark & Free, 1970).

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