

A THERMOPHYSIOLOGICAL RATIONALE FOR ENDURANCE TRAINING FOR RACQUET ACTIVITIES

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ABSTRACT

Three male athletes performed incremental work: Basal Metabolic Rate (BMR), 100 W and 150 W in two levels of controlled environmental heat. Conditions inside an environmental chamber were preset at 25°C 40% RH and 30°C 50% RH being 22°C and 29°C on the Effective Temperature Scale. Expired air and six body temperatures — two invasive and four skin sites — were monitored. Core and mean body temperatures, calculated from these figures, correlated highly with expired air values for all the anthropometrically homogeneous group. Results were in agreement with unpublished data of Bundgaard, i.e. the higher the VO_2 max of the subject the smaller the range of expired air values and the smaller the increase in both mean and core body temperature during heat stress. Such thermophysiological reaction helps the athlete to prevent the onset of mild hyperthermia and the accompanying fatigue, independent of mitigating behavioural support.

The intermittent bursts of heavy physical activity required of the racquet athlete argue for a similar cardio-vascular training regimen. Data from this study suggest that such athletes would be wise to augment training schedules with prolonged cardio-vascular endurance work especially when the possibility of competing in high ambient temperature is foreseen.

Key Words: Training, Racquet Athletes, Cardio-Vascular Endurance, Mitigating Thermophysiological Reaction.

INTRODUCTION

The racquet sport player is often subjected to thermal stress from the operational environment. This stress is increased by the internal heat load produced by active muscle tissue. Together these factors serve to impose a considerable thermal strain upon the athlete during the intermittent bursts of heavy physical activity associated with racquet sports. On those occasions where heat transfer to the environment is of minimal value, the player may experience a considerable rise in body temperature.

Burton (1934) first described this process mathematically, through the expression:

$$M + W - R - C - E = S \quad (1)$$

which states that, heat produced through metabolic activity (M) and work (W), which is not lost to the environment through radiation (R), convection (C) or evaporation (E), results in some heat storage (S). This heat storage causes a rise in tissue temperature, with a concomitant rise in deep body temperature.

There is little evidence of mental performance impairment through such a rise in deep body temperature (Hancock, 1981). However, it has been suggested by Grether (1973) that many motor skills, vital in racquet sport performance (e.g. tracking), may suffer decrement as body temperature deviates from normal sedentary values. In addition, heat exhaustion may range from mild fatigue to complete collapse, dependent on exercise level and extant environmental conditions. Such fatigue from hyperthermia may be an additional physiological factor which limits performance efficiency. The experimental data reported here suggest a method of training which may help promote the ability to mitigate such debilitating heat storage. Specifically, this paper advances a thermophysiological rationale for cardio-vascular endurance training in the racquet sport athlete.

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METHOD

Procedure

Each subject, wearing only shorts and training shoes (clothing insulation value = 0.1 clo) was placed in an environmental chamber 4.72 x 2.92 x 3.10 m, a volume of 42.73 m³.

Subjects engaged in a monitored 12 min pre-exercise phase, an 18 min work phase at 100 W followed by an 18 min work phase at 150 W. Two experiments were performed. In Experiment 1, environmental conditions were set at 25°C, 40% RH, < 0.1 ms⁻¹ air velocity and in Experiment 2 conditions were set at 30°C, 50% RH, < 0.1 ms⁻¹ air velocity. These represent heat stress levels of 22°C and 29°C on the Effective Temperature Scale (Houghten and Yagloglou, 1923) which integrates the dry bulb, relative humidity and air velocity figures reported. Such conditions were monitored from the chamber control panel by a WBGT Index Meter (approximately the ET values) and manually inside the chamber by a whirling hygrometer, Kata and Globe thermometers. Subjects were tested after a three-hour period of food restraint. Informed consent was obtained prior to experimentation.

Subjects

Three subjects closely approximating "standard man" (Dubois and Dubois, 1915) were selected to form a relatively homogeneous group. Their details appear in Table I.

TABLE I

Details of subjects

		Subject		
		B.H.	S.T.	I.M.
Height	(cm)	170.0	169.0	166.7
Weight	(kg)	64.05	62.45	66.10
Age	(yr)	27.1	24.2	22.4

Each subject participated consistently in an English University Athletic Programme at an inter-university level at the time of testing.

Temperature Recordings

Six temperature probes were used to monitor body temperature throughout the experiments. Four skin probes were attached: one immediately below the right scapula and one on the sternum opposite the fourth intercostal space, and two on the limbs; one on the medial point of the right rectus femoris, and one on the medial point of the right biceps brachii. The rectal temperature probe was inserted to a depth of 10 cm. A tympanic probe was placed adjacent to the tympanic membrane and positioned to avoid painful contact during work phases and a 0.1°C lag correction factor was introduced (Piironen and Aikas, 1964). Readings were monitored every 2 min during pre-work phases and every 3 min during working phases on a six point electrical recording thermometer (Light Laboratories).

TABLE II

Increase in bodily heat storage: three derived measures

	Environmental Conditions					
	25°C 40% RH			30°C 50% RH		
	B.H.	Subjects S.T.	I.M.	B.H.	Subjects S.T.	I.M.
Increase T_R (°C)	0.3	0.7	0.5	0.5	0.8*	0.6
Range Exp Air (l/min)	19.4	35.0	28.6	15.1	27.9	27.1
Raw Data Correlation T_R vs Exp Air	0.70	0.93	0.85	0.59	0.84	0.84
Mean Body Core Temperature ¹ Increase (°C)	0.24	0.66	0.52	0.48	0.74	0.72
Mean Body Temperature ² Increase (°C)	0.30	0.62	0.65	0.83	1.04	0.88

*Raw Data Corrected for Rectal Probe Movement Artifact

¹ Mean Body Core Temperature Increase (MBCT_I) calculated by:
(Increase in Rectal Temperature x 4) + (Increase Tympanic Temperature)/5
i.e. MBCT_I = (IT_R x 4) + (IT_T)/5

² Mean Body Temperature Increase (MBT_I) calculated by:
0.64 (MBCT_I) + 0.36 (MBST_I) (Altered, after Burton, A.C. & Edholm, O. G., Man in a Cold Environment, Arnold, 1955).

RESULTS

From the data three main parameters were used to assess body heat storage in each of the two experiments, Table II.

Firstly, the observed increase in rectal temperature which reflected heat storage in the trunk of the body. Secondly, a mean core temperature increase, derived from weighted values of the two core temperature measures. Thirdly, a mean body temperature increase which integrates figures from each of the temperature sites and provides an overall index of bodily heat storage. In each condition the following held true, the higher the VO_2 max of the subject, reflected in the expired air values, the lower the increase in the mean of the heat storage parameters. Conversely, the lower the VO_2 max the higher the rate of heat storage in the body. This follows the pattern of Bundgaard's unpublished results from Nielsen's laboratory (Nielsen, 1976) and finds further support from the work of Mostardi, Kubica, et al (1974).

Each subject in the present study was selected on the basis of similarity to a standardised model of the human being. The differences between heat storage are not thought to be due to anthropometric factors. Should subjects have differed in height, weight, surface area, or fat content, then temperatures reported may have been a reflection of such differences, although Bundgaard indicates a minimal importance for such factors. Rather, the subject who consistently engaged in cardio-vascular endurance training was able to resist the onset of debilitating heat storage in a markedly superior manner, in both experimental conditions.

DISCUSSION AND CONCLUSIONS

There are three main factors, associated with the physiological change induced by cardio-vascular overload training, which suggest an account of the data reported. Firstly, during submaximal exercise the endurance trained athlete, being habituated to work at maximal levels, may utilise blood flow more effectively by supplying blood preferentially to specifically active tissues. This would leave a relatively greater percentage of blood flow free to effect heat transfer away from the body core to the skin. With more blood available for surface transfer to the environment better regulation is achieved.

Secondly, there are changes in peripheral vascularisation and heart action associated with the physical overload produced by prolonged endurance training. Such a trained athlete may be able to perfuse vessels near the skin layer during exercise, in a superior manner. This more efficient movement of blood is in part dependent on the state of the vessels encountered. With endurance training comes increased usage and amenability to the passage of blood. The larger the peripheral surface area perfused, the greater the heat exchange and therefore the reduction of the storage of debilitating excess heat. Finally, the increase in resting blood volume associated with endurance training allows for prolonged sweating, with continued body fluid balance, during exercise in severe heat. This further increases the thermoregulatory efficiency of the endurance trained athlete.

In sedentary activities the human being accomplishes temperature regulation through behavioural action and vasomotor control. In the active racquet sport performer, the more powerful physiological effector system of sweating is employed to maintain thermal balance. However, this may prove insufficient on occasions where activity is undertaken in high ambient temperature. Although behavioural actions may ease discomfort, continuous usage over a long period is neither desirable nor sufficient to alleviate more than mild hyperthermic symptoms. The data from the very few subjects in the current study suggest that preparation of the athlete, through progressive increases in cardio-vascular loading, may enhance bodily mechanisms which act to prevent heat retention. However, further research is required to help establish the efficacy of such training, as a method of reducing performance limiting heat strain, in the active racquet sport athlete.

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BOOK REVIEW

Title: THE PHYSIOLOGY OF BONE. 3rd Edition
Author: Janet Vaughan
Publisher: Oxford University Press, 1981
 Price: £25.00

Dame Janet Vaughan, the inexhaustible doyenne of bone physiology has produced a 3rd edition of her book. This excellent, but expensive, little book is hard to classify. It contains virtually nothing of *direct* relevance to exercise physiology or to the clinical practice of sports medicine. On the other hand, it must surely be an essential "handbook" for anyone working with bone who, although not himself a biochemist or a metabolic physiologist, still takes an intelligent interest in the underlying physiology and biochemistry of his tissue.

"The Physiology of Bone" is essentially a guide to review articles, monographs and key publications. "The present volume is intended for readers who want a bird's-eye view of the subject and directions as to where they can find more detailed information on particular aspects." One of its strengths, therefore, is that it is one expert's personal synopsis of the subject. This is a welcome change from the usual multi-author textbook. The inevitable corollary, however, is that coverage of the subject is not comprehensive and the reader may feel that his own areas of special interest have received scant attention. As with the previous editions of her book, Dame Janet has excluded any detailed consideration of teeth or of haemopoietic marrow. It is perhaps unfortunate that she has also omitted the mechanical and structural aspects of bone. Her accounts of the "development", "structure", "growth", and "function" of "bone as a tissue" would have been of even greater value to the orthopaedic surgeon or the rheumatologist if they had included something more of her view of the relationship between them and the load-bearing function of the skeleton. Personally, I should have been interested to read Dame Janet's views of the reciprocal relationship which appears to exist sometimes between bone and muscle, e.g. the muscle weakness of osteomalacia and of Engelmann's disease or the susceptibility to fractures observed in muscular dystrophy. Other clinicians may well be surprised that there is so little about the pathophysiology of ectopic ossification.

These, however, are but niggling criticisms of an extremely useful guide to the world of bone metabolism. The thinking rheumatologist, orthopaedic surgeon or metabolic physician will almost certainly wish to queue alongside the biochemist specialising in other aspects of biochemistry in order to purchase this new edition to replace his well-thumbed copy of its predecessor.

Archie Young Jnr.