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the simulated results with the authors of the models and a group of outstanding thermal physiologists who offer constructive criticism and suggestions for improving the models.



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AN EVALUATION OF HUMAN THERMAL MODELS

BY

EUGENE H. WISSLER

**A report based on a
workshop held at**

THE UNIVERSITY OF TEXAS AT AUSTIN

**Approved for public release;
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**Sponsored by the U. S. Air Force
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Part A



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INTRODUCTION

This report is based on a workshop held at The University of Texas at Austin in December, 1982 under the sponsorship of the Air Force Office of Scientific Research. The purpose of the workshop was to evaluate available mathematical models which could be used to simulate human thermal behavior under various conditions. The general plan involved the following four activities: (1) obtain copies of the mathematical models, install them on computers located at The University of Texas, and verify that they were operating correctly, (2) collect sets of data suitable for testing mathematical models and enter them into a machine readable data base, (3) use the models to simulate the conditions represented by the experimental data, and (4) discuss the simulated results with the authors of the models and a group of outstanding thermal physiologists who could offer constructive criticism and suggestions for improving the models. This could never have been accomplished without the willing cooperation of those who agreed to provide models and data and to participate in the workshop. Special recognition is due Dr. S. A. Nunneley of the U.S. Air Force School of Aerospace Medicine in San Antonio for her encouragement and support. Others who participated in the project are listed below.

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SUMMARY OF PHYSIOLOGICAL CONCEPTS

Thermal physiology in its entirety is a rather complex subject and no attempt will be made to include a complete review in this report. Instead, a brief outline will be presented for each of the principal areas: cardiovascular response to thermal stress and exercise, sweating, and shivering. A list of selected references is presented for each area, and readers who want to delve more deeply into any of these subjects should consult those references.

Cardiovascular Response to Thermal Stress and Exercise.

Undoubtedly the most complex physiological response is provided by the cardiovascular system, which must transport both chemical substances and heat throughout the body. Its capacity for doing so is limited by cardiac function, and thermoregulatory requirements for blood flow must be balanced against metabolic requirements. This becomes most apparent during exercise in a warm environment when there is a need to increase simultaneously blood flow to the skin for thermoregulatory purposes and to working muscles for oxygen transport purposes. These conflicting demands are met by reducing the flow-rate to regions which can tolerate a reduction, such as the spleen and inactive muscle.

An excellent summary of the concepts introduced above is presented in Rowell's paper published several years ago in Physiological Reviews [56]. Later summaries of results obtained by the University of Washington group are contained in papers published by Rowell and Wyss [57] and Brengelmann [8]. An excellent description of cardiovascular regulation has been presented also by Nadel [51], whose views on certain points are somewhat different from those of the Washington group, although there is general agreement on major points. These are summarized below.

1. At tissue temperatures below 38.0 to 38.5°C, the perfusion rate of muscle is determined by the metabolic demand for oxygen and the intensity of vasoconstrictor outflow. At higher temperatures, vasodilation may occur in muscle.
2. Vasodilation owing to heat stress is largely confined to the skin.
3. Increasing the temperature of central thermal receptors 1°C elicits 10 to 20 times the increase in skin blood flow caused by a

1°C increase in mean skin temperature.

4. Cutaneous vascular smooth muscle responds directly to heat and cold by vasodilating and vasoconstricting, respectively. These vessels show a unique local response to temperature, which tends to favor protection of cutaneous areas from thermal injury sometimes in opposition to the response required for central thermal regulation.

5. Local responses to heat differ from reflex responses; they develop slowly over many minutes and the intensity of vasodilation is smaller. Even the highest skin temperatures seen in hot environments (36 to 38°C) are insufficient to cause major increases in skin blood flow owing to direct local effects.

6. Most of the increase in skin blood flow during heating is caused by active vasodilation, rather than by decreased cutaneous sympathetic vasoconstrictor tone, which is influenced by skin temperature.

7. The only factor known to influence the vasodilator outflow is changing central temperature.

8. Vasoconstrictor outflow affects the perfusion rate of skin, muscle, and visceral organs.

9. Vasoconstrictor outflow is affected by several factors including central and cutaneous temperatures, intensity of exercise, posture, and central blood volume; these factors are not necessarily independent.

10. A fall in blood pressure causes cutaneous vasoconstriction under all conditions studied by Rowell.

11. Above approximately 50 percent of $\dot{V}_{O_2, \max}$, there is no further increase in cardiac output to provide additional blood flow to skin, and incremental skin blood flow must be provided by redistribution of cardiac output.

12. Sympathetic vasoconstrictor activity to skin is increased during exercise, but not in proportion to relative $\dot{V}_{O_2}$ or heart rate, as it is to splanchnic regions.

13. Exercise and upright posture both cause decreased forearm blood flow compared to supine rest at any given esophageal temperature.

14. In a hot environment, splanchnic vasoconstriction is augmented at any given $\dot{V}_{O_2}$ so that visceral organs receive less blood flow, thereby making more blood flow available to skin.

15. Exercise causes hand and forearm veins to constrict. A small increase in skin temperature causes reduced venous tone during exercise.

16. Cardiovascular responses are affected by plasma volume; skin blood flow is reduced in hypovolemic individuals.

The items listed above only serve to illustrate the complexity of cardiovascular response to exercise and thermal stress. To gain a full appreciation for the current state of understanding of this subject one must study the original literature carefully. Although the general features of the subject are well defined, many details remain obscure. One of the most important concerns the effect of exercise per se on cutaneous blood flow. Brengelmann's review [8] of reported experimental data led him to conclude that exercise tends to inhibit the skin blood flow response to increasing temperature, but the magnitude of the attenuation is independent of the intensity of exercise. While this conclusion is somewhat difficult to accept, it is also difficult to refute. Since exercise inevitably causes an increase in bodily temperatures, it is very difficult to separate the effect of exercise from the effect of changing temperature. Furthermore, there may be a dynamic component involved which could be quite important during exercise because steady states are seldom realized, but the effect of rate of change of temperature has received little attention. Hence, this area still presents many challenges to both experimentalists and modellers.

Sweating. The principal mechanism available to man to transfer heat to the environment is evaporation of sweat; at dry bulb temperatures above 35°C, it is the only mechanism. Hence, sweating must receive careful attention in the development of a mathematical model for human thermal regulation. Again, there is agreement about the general features of sweating, but various investigators disagree about some of the details.

That the rate of sweating depends on hypothalamic temperature, skin temperature, and the rate of change of skin temperature seems to be accepted by all investigators. Whether rate of change of hypo-

thalamic temperature affects the sweat rate appears not to have been considered. It seems to be reasonably well established that the rate of change of skin temperature is important when the skin temperature is decreasing, but there is some disagreement about the importance of positive rates of change. The prevalent point of view a few years ago was that negative rates of change inhibit sweat production, but positive rates have no effect [48]. However, recent studies by Libert [41-43] indicate that positive rates do enhance the rate of sweating if they are large enough. There is also disagreement about the relative thermal sensitivity of various segments of the skin. Nadel, et al. [50] and Cranshaw, et al. [12] have described locally different thermal sensitivities, while investigators from Vogt's group seemed to find relatively uniform sensitivities for various large areas of the body.

The concept of a threshold for sweating seems to be generally accepted, although there are differences of opinion about the precise nature of the conditions that must be satisfied to initiate sweating. Recently Libert [40] proposed that a special condition lying above the minimum condition necessary to sustain sweat production has to be satisfied before sweating can be initiated, but once started sweating can continue even though the initiation condition is no longer satisfied. Threshold conditions also exist for the enhancement or inhibition of sweating owing to changing skin temperature. Libert, et al. [42] report that the magnitude of the threshold is larger for increasing skin temperature than for decreasing temperature.

Afferent signals from central and peripheral thermal receptors are usually defined in terms of the difference between a given temperature and its corresponding set-point. Various relationships between efferent and afferent signals have been proposed; the two most commonly proposed forms involve either linear or multiplicative combinations of the central and peripheral signals. Recently, Libert, et al. [43] proposed that the form of the relationship changes depending on conditions, with the multiplicative relation being descriptive of sweating under warm conditions and the additive relationship applying to hot conditions. Although there have been

many attempts to define a relationship that describes sweating under a variety of conditions, a generally accepted relationship remains to be developed. Nevertheless, there is general agreement that a unit increase in central temperature elicits approximately ten times the change in sweat rate caused by the same change in mean skin temperature. There also seems to be agreement that the efferent signal resulting from integration of central and peripheral afferent signals is modulated locally as a function of local skin temperature [49].

A question which remains unresolved involves the effect of skin wettedness on rate of sweat secretion. Skin wettedness is defined as the ratio of the actual evaporation rate to the maximum possible evaporation rate computed as the product of a mass transfer coefficient and the difference between the vapor pressure of water evaluated at the mean skin temperature and the partial pressure of water in the environment. It is well known that the rate of sweat secretion diminishes slowly when profuse sweating is maintained for hours. Two factors appear to contribute to the decrease--one is fatigue of the sweat glands and the other is swelling of the keratin ring at the opening of the sweat duct. The second factor is usually referred to as hydromyosis. In addition to these factors, it has been reported that accumulation of sweat on the skin inhibits secretion of sweat through some undefined mechanism. Support for this notion is provided by the fact that local sweat rates measured using a capsule which maintains a dry surface are invariably higher than mean whole-body sweat rates. Observations involving sweat rates and temperatures with and without a fan to facilitate evaporation have also been interpreted in terms of inhibition of sweating by accumulated sweat. Alternative explanations for some of these observations can be developed using the fact that conditions are not uniform over the entire body [39]. The local rate of sweat secretion, the skin temperature which determines the vapor pressure of water on the skin, and the mass transfer coefficient all vary with location. When there is excess sweat secretion on some areas and insufficient secretion on others, one has the paradoxical situation that sweat drips from the body even though the total rate of evaporation is less than maximal. Furthermore, regional variations in skin temperature

certainly exist, and there is no reason to believe that a "mean skin temperature" computed somewhat arbitrarily can be used to evaluate the mean maximal escaping tendency of water from the skin. The subject of mass transfer from the skin is considerably more complex than many physiologists realize, and care should be exercised to establish that relevant physical factors are properly considered before conclusions are drawn about physiological phenomena. Until some of these questions are answered, those who develop thermal models will be faced with a dilemma which can only be resolved according to their personal preferences.

Shivering and insulation.¹ The physiological basis of increased heat production in the cold is still open to dispute in detail, but the main points are reasonably certain. There is longstanding evidence that nearly all cold-induced thermogenesis in the human adult is brought about by shivering and other forms of muscle activity. Speck, for example, showed in 1883 that the increase of O_2 consumption in the cold could be abolished by voluntary relaxation, and Johnson [36] showed in 1963 that it could be abolished by neuromuscular block. There have been many suggestions that metabolism of brown fat contributes to the human adult's response to cold, as it does to the newborn infant's. The existing evidence is compatible with brown fat or some similar mechanism being able to generate up to about a 20% increase in metabolic rate in the cold, but technical problems still leave the reality of this increase uncertain [37]. There have also been suggestions that increased thyroid activity can increase heat production in the cold. Production of thyroid hormones does increase on exposure to cold, but so does destruction of the

¹This section was written by W. R. Keatinge.

hormones, and circulating levels do not normally seem to increase during long exposures to cold. There is interesting evidence of an exception to this in that people intermittently exposed to severe cold stress, such as the Korean Ama and active skin divers [8], may show thyroid-induced heat production of around 40% when they are in the warm. Such increases are, however, not only slow to develop but also small in size compared to the sustained 300% increases in heat production which can be produced by shivering. Although exposure to cold causes circulating levels of norepinephrine, and possibly of epinephrine, to rise a little above their usual, very low resting levels, this seems to be of little importance and to represent largely an overflow into the circulation of some of the norepinephrine released from vasoconstrictor nerves. The overall picture, therefore, remains that shivering and cold-induced thermogenesis can be taken as virtually synonymous during acute exposures of human adults to cold, although there are interesting possibilities of limited alternative sources of thermogenesis associated with some types of cold exposure.

As regards internal body insulation, it is clear that subcutaneous fat thickness is a major determinant of whole body insulation in the cold. However, insulation is also dependent on other factors, including cutaneous blood flow, countercurrent exchange in the limb blood vessels, and on activity of limb muscles and the muscle blood flow associated with this.

Much less is known about the precise operation of the control mechanisms for these responses, which is crucial to modelling theory.

It is clear that the hypothalamus is the main regulator, and that it acts on the basis of sensory information both from cutaneous receptors, and from deep receptors which are mainly in the anterior hypothalamus. As regards the type of interaction between these sources, current evidence indicates that, to a first approximation, shivering is brought about by a graded reflex to cutaneous receptors with the amplitude of the reflex modulated by the level of deep temperature; in the absence of a cold skin, low hypothalamic temperature does not cause shivering, and the simplest evidence of this is the fact that people with low body temperature cease shivering as soon as their skin is rewarmed. Dynamic elements in the response of the receptors provide a major additional complication, and information about the role of different skin areas in thermal sensory input is very limited. Comprehensive mathematical models of heat production and regional heat exchanges in the cold [9, 32, 74], therefore, call for considerable assumptions about such variables, and usually require considerable retrospective empirical adjustment. We have accordingly recently concentrated on direct experimental predictions for people in the specific and theoretically more manageable situation of a steady-state of heat exchange in water at the lowest temperature at which they are able to stabilize their body temperature [11, 33]. This is a situation of particular practical importance as well as being theoretically convenient, and metabolic rate, total and regional heat losses of individuals of given fat thickness and body build can be obtained for this state. The data can then be used for predictions of similar limiting external conditions with few additional assumptions. They show that whole body

insulation and, to a lesser degree, regional insulations, in this situation can be predicted with considerable accuracy from an individual's subcutaneous fat thickness, particularly if height and weight are also used as predictors. Metabolic rate is, however, less accurately predicted. Inherent errors in the ability to predict an individual's ability to maintain body temperature in the face of a given degree of environmental cold stress are consequently substantial, particularly if spatial patterns of skin temperature vary. Very precise predictions of limiting external thermal conditions for an individual of given body build, even for steady states of heat exchange, would therefore require more information, and more understanding of the factors that vary individual metabolic response than we possess at present. Until that is available, we may have to accept all such predictions as primarily group mean predictions, which will inevitably show wide variation when applied to individuals.

Thermal Limits.² Based on laboratory studies involving exposure to heat and cold, plus real experience where measurements are sparse, human thermal tolerance is definable in terms of:

- performance limits,
- voluntary tolerance,
- pain and local injury,
- loss of consciousness,
- and death.

End-points of this sort presuppose that thermoregulatory responses have failed, so that the heat or cold exposures are noncompensable. But the major physiological responses of vasoconstriction, vasodila-

²This section was written by Paul Webb.

tion, increased cardiac output, shivering and sweating act to prolong the duration of nearly any exposure before a limit is reached. In modeling human thermal response, it is important to place limits on what really can be endured.

Real situations are of many varieties, and each sort tends to emphasize a particular definition of tolerance. When one considers telegraph operators in tropical climates, or divers who weld pipe or place explosive charges, or pilots who overheat in impermeable protective clothing, one thinks of critical performance. Pain and local injury are important to workers in steel mills, glass factories and kilns, as well as to outdoorsmen and military personnel in severe cold weather, especially in wind. When people fall accidentally into cold oceans, lakes, and rivers, when there are prolonged spells of hot, humid weather, and when we ask soldiers to march in chemical-biological protective clothing or workers to clean chemical storage tanks, the paramount problem may be loss of consciousness. The ultimate limit, death, can come from any of those limits being exceeded--that is, a diver with impaired judgment can make a fatal mistake, an overheated pilot can fail to pull out of his bombing run, and people can die of burns, hypothermia, drowning, and heat stroke.

Performance Limits. Predicting where performance begins to change, or has degraded badly, is vital in diving, aviation, and many industrial and military situations. We put people in cold and hot environments to do something important. Maintenance of strength and dexterity, skilled performance of learned tasks, vigilance, and good judgment are expected and sometimes critical. Even small errors can threaten life or mission. Sometimes performance is less critical, as in marching or just surviving; here the performance limit is

virtually the physiological limit. In war scenarios the military is fond of predicting casualty rates due to cold and heat.

Degradation in performance arises either from distraction and discomfort, or from ill-defined neurophysiological changes which are probably related to storage or loss of body heat content, both regionally (numb fingers) and generally (hypo- and hyperthermia).

Despite the practical importance of understanding performance limits in heat and cold, too little has been done, perhaps because physiologists don't like to measure performance and because psychologists don't appreciate the niceties of physiological measurement.

Voluntary Tolerance. Subjects of experimental exposures to heat and cold reach limits beyond which they are unwilling to go. With experience, this limit stretches a bit, but everyone will cry "uncle" at some point, and in many cases this is predictable. In exposures where sweating or shivering have compensated more or less well, the limit seems to be set by the cumulative cost of thermoregulation, hence fatigue.

In cold, after shivering has become heavy and continuous, another hour is physically exhausting.

In heat, heavy sweating and a high cardiac output over many hours produce fatigue. Three to four hours of heat with heart rate above 120 beats/min with no physical work, are about the limit.

Pain and Local Injury. An ouch-point is definable in heat exposure. Threshold pain from surface heating starts at a skin temperature of 42-44°C, but varies between and within individuals. When skin temperature reaches 45°C, pain is invariably intolerable. If T_{sk} is 45°C for several minutes, thermal injury begins.

When T_{sk} is 45°C and higher, the severity of burn damage increases as the product of temperature and time.

Cold fingers and toes are painful, perhaps from intense vasoconstriction, followed by numbness as chilling continues. Prolonged chilling without freezing causes the vascular injuries labelled trench foot and immersion foot.

When subcutaneous tissue freezes, there is the thermal injury called frostbite.

Loss of Consciousness. This limit in both heat and cold is

determined by storage (destorage) of body heat. There is evidence that the rate of storage is important, as well as its absolute quantity.

As a rough approximation, storage of 150 kcal in a 70 kg man at rest is a voluntary tolerance limit, and 200 kcal is likely to produce unconsciousness. Physical activity, e.g. running or marching while carrying a load, causes tolerable storage (rise in rectal temperature) in proportion to the metabolic cost of the activity. Two major modifiers are body size and rate of heat storage. A small person tolerates less storage (because it produces higher internal temperatures?) and a rapid rate of storage is less tolerable than a slow rate--for the same absolute storage level.

The storage limit in heat is defined by a condition of "impending heat stroke," in which the subject becomes quite restless, performs less and less well, hyperventilates, is vasodilated, has a high pulse rate and cardiac output, has a high and rising T_{re} , may show circumoral pallor, may begin to reduce his sweating, and, if not removed from the heat, becomes unconscious (but is easily aroused by cooling if treated immediately).

In cold, about 200 kcal of heat loss approximately defines voluntary tolerance, and unconsciousness probably comes at a 300-400 kcal of loss. Modifiers are body fat content, body size, and rate of heat loss. A person of 70-75 kg rapidly cooled by 200 kcal in 30 mins may be unconscious and, in open water, drown, while cooling to the same level over two hours produces a voluntary tolerance limit, but the person will still be conscious. The same man can lose 300 kcal over 6-8 hours and be only mildly uncomfortable. The puzzling thing is that core temperatures are quite different as well: 34°C in the first example, 35.5 in the second, and 36.5 in the third.

For completeness we should mention heat syncope, or heat prostration. It is a loss of consciousness like fainting when inexperienced people are exposed to heat, or work in heat, but there is only small storage and little rise in body temperature.

Death. Death from general cooling is clinical hypothermia, and the result of sizeable heat loss, which is unmeasured but probably

in excess of 500 kcal for a 70 kg man. Clinical data is rectal temperature, and T_{re} relates to the probability of ventricular fibrillation and death. The likelihood of fibrillation is small at $T_{re} = 32^{\circ}\text{C}$, noticeable at 30°C , and 50% probable at 27°C .

Death from heat stroke is a result of large heat storage and high internal temperature. As a guess, heat stroke should occur at storages above 300 kcal. The borderline of lethality in T_{re} is usually given as 42°C , although cancer patients have been heated to and held at that level for many hours without peril.

High or low body temperatures per se are not lethal, but cardiac irritability, altered cell functions, and tissue hypoxia cause death.

Healthy young men and women can withstand greater thermal stress and storage (heat loss) than old people, malnourished people, or the sick and injured.

We don't put all this in models, but there should be some model states which cause "TILT."

THERMAL MODELS

A theoretical model summarizes one's understanding of some natural phenomenon. In the physical sciences, such models are invariably quantitative, but that is not necessarily the case in the life sciences. However, thermal models are generally based on physical principles and, therefore, tend to be quantitative; all of the predictive models evaluated in this study are quantitative. Even so, they differ as to purpose and have different structures.

Regardless of its final form, every human thermal model starts with the first law of thermodynamics. Also incorporated into the model is information derived from the physics of heat and mass transfer, generally expressed as relationships which define thermal and mass fluxes at the skin in terms of appropriate thermal and diffusive driving forces. A principal difference between various models is the extent to which they attempt to incorporate detailed descriptions of physiological phenomena. Such descriptions invariably take the form of empirical statements derived from experimental data. Some models minimize the inclusion of such information and, instead, emphasize the use of data which are of direct interest, such as the rectal

Table 1. Characteristics of Human Thermal Models

Author Thermal Stress	Goldman Heat	Goldman Cold	Stolwijk Heat or Cold	Gordon Cold (or Heat)	Wissler Heat or Cold
Type	Empirical	Physiological	Physiological	Physiological	Physiological
Computed Quantities Temperatures	T_{re}	T_{re} , $\bar{T}_{sk}$, $\bar{T}_{sc}$	T_c , T_m , T_{sk} for head, trunk and extremities	154 temperatures de- fine profiles in 14 elements. Depends on T_{hc} , $\bar{T}_{sk}$ and local T_{sk}	225 temperatures de- fine profiles in 15 elements. Depends on T_{hc} , $\bar{T}_{sk}$, $\bar{T}_{sk}$, and local T_{sk} .
Sweat rate	Implicit	Not included	Depends on T_{hc} , $\bar{T}_{sk}$ and local T_{sk}	Depends on T_{hc} , $\bar{T}_{sk}$ and q_{sk}	Depends on T_{hc} , $\bar{T}_{sk}$, $\bar{T}_{sk}$, and $\bar{T}_{sk}$.
Shivering rate	Not included	Depends on T_{re} and $\bar{T}_{sk}$	Depends on T_{hc} and $\bar{T}_{sk}$		
Cardiovascular	HR	Not included	Local bloodflow rates depend on M , T_{hc} , $\bar{T}_{sk}$	Local bloodflow rates depend on M , T_{hc} , $\bar{T}_{sk}$ $\bar{T}_{sk}$ and $\bar{T}_{sk}$.	Local bloodflow rate depend on M , C , O_2 , T_{hc} and $\bar{T}_{sk}$.
Specification of Subject	Wt and SA	Wt, SA, and BF	Wt and SF	Wt and SF	Wt and SF
Environmental Specifications	Air (not cold): T_{db} , T_{wb} , and V_e	Water: T_{db} and h	Arbitrary air-water combinations: T_{db} , T_{wb} and h .	Air: T_{db} , T_{wb} and h Numerically unstable in water	Arbitrary air-water combinations: T_{db} , T_{wb} and h
Exercise	Specify arbitrary work/rest profiles	Rest (P)	Specify arbitrary work/rest profiles	Specify arbitrary work/rest profiles	Specify arbitrary work/rest profiles
Posture	Upright (P)	Upright (P)	Upright (P)	Upright (P)	Upright (P)
Hydration	Normal (P)	Normal (P)	Normal (P)	Normal (P)	Normal (P)
Acclimation	Specify days of exposure to heat	None (P)	None (P)	None (P)	None (P)
Clothing	Specify whole-body clo and i_m/clo	Specify thermal resistance	Specify regional clo and i_m/clo	Specify regional clo and i_m/clo	Specify regional clo and i_m/clo ; model LCC
Validation	Well validated for various work rates in the heat	Not validated for predictive use	Validated for exer- cise in a warm environment	Validated for rest in cold air	Validated for a wide variety of conditions
Computational Requirements	Short execution time on a small computer	Short execution time on a small computer	Short execution time on a small computer	Moderate execution time on a large com- puter. Tedious data preparation.	Long execution time on a large computer

temperature and heart rate. Other models attempt to include a considerable amount of physiological data of the kind referred to in the previous section. The need to incorporate physiological information into the model is greater when dealing with cold stress than with heat stress because large temperature differences do not develop under warm conditions, and it is relatively easy to define the thermal state of the body.

When they can be used, highly empirical models offer the advantages of simplicity and economy; in many cases, they can be programmed of small hand-held calculators. On the other hand, models which attempt to describe the underlying physiology of thermoregulation tend to be complex and require considerable computation power for execution. However, if one can get the physiology right, the resulting model is capable of describing human thermal behavior under a variety of conditions, and one does not have to be quite so concerned about the applicability of the model to a particular set of circumstances. Both kinds of models are evaluated in this study. In the following section, each model will be described briefly before its capacity for simulating various responses is tested against a set of carefully selected experimental data.

A brief overview of various models is presented in Table 1, where the principal characteristics of each model are summarized. In this table, the term, "specifiable," is used to identify parameters which the user specifies in order to define the particular situation being simulated. The notation, "(P)," is used to denote conditions that are assumed to exist, such as upright posture. When a quantity is characterized as "implicitly only," the implication is that a derived quantity which depends on the primary quantity is employed in the model, but the primary quantity does not appear explicitly. For example, time constants employed in Goldman's "heat" model depend on the intensity of exercise because transport of heat within the body is facilitated by enhanced blood flow during exercise, but local perfusion rates are not defined explicitly in this model. Hence, cardiovascular responses are said to appear implicitly in this model.

Ralph Goldman's Models. The principal proponent of a highly

empirical approach to model development is Ralph Goldman, who for many years was a civilian scientist at the U.S. Army Research Institute of Environmental Medicine in Natick, Massachusetts. Dr. Goldman's views on human thermal modelling are summarized in the following section (through the first paragraph on page 20) which he prepared.

Modeling for the Clothed Man

R. F. Goldman, Ph.D.

It is reasonably well established, in theory, that modeling for the clothed man should be no different than the typical modeling of a laboratory subject wearing shorts and footwear; in fact, the clothed man model [22, 27] should reduce, in the simplest case, to treat the unclothed case. However, there are two major differences. First, analytic physical heat transfer relationships using Reynolds, Grashof and Prandtl numbers have been quite usefully applied for the unclad man [75]; these are not, at present if ever, applicable to a dynamic, continuously changing boundary layer which consists of multiple layers of conducting and wicking filaments, assembled into fibres, assembled as meshes with varied pore dimensions and intersections with varying contact pressure, transferring heat by convection, conduction, radiation and evaporation at, and away from, the skin surface [7, 27, 63]. A combination of empirically determined factors coupled, whenever possible, with the rigorous physical relationships is necessary; e.g. the slope of the wet bulb temperature lines on the psychrometric chart (2.0°C) was used empirically by Woodcock in developing the permeability index theory, but the value was subsequently changed to the Lewis number (2.2°C) as Goldman began applying the theory to practice [25]. Second, perhaps because models for the clothed man are meant to be applied across the spectrum of real world activities, environments and worker populations [21, 62, 31, 23, 52, 26, 10, 59], such models are subjected to much more testing and evaluation than is typical of the "laboratory" models.

It seems equally clear that, at present, no single model can cover the entire range of temperatures and activities to which a clothed

*The work reported was carried out by the author, and members of his Division at the U.S. Army Research Institute of Environmental Medicine, Natick, Massachusetts.

workman can be subjected: indeed, heat exhaustion can be a problem for an arctic worker, while death from hypothermia can occur at an air temperature of 20°C. At present, four or five models seem necessary for the real world, clothed man:

- 1) A "comfort" model, covering typical indoor clothing and office or laboratory level activities that deals with the environmental range of perhaps 10 to 30°C. This model, with clothing at the typical shorts and footwear level, is closest to most of the models being discussed currently [21, 25, 26, 27, 28].
- 2) A "heat model, involving heavy sweating and the nature of the interference of clothing with evaporative heat transfer. In this model, acclimatization [19], dehydration, solar [54, 5, 6] or other radiant load and worker heat production [35, 16, 62, 24], coupled with the clothing permeability index ratio (I_m/clo) [7] are the dominant factors. The environmental ambient range of application generally is above 30°C but with heavy or impermeable clothing can go down to 5°C, and even lower with hard work [17, 18, 61].
- 3) An "extremity cooling" model, involving the thermal response of fingers, toes, ears, etc. and their modulation by the appropriate coverings (gloves, boots, etc.). In this model, circulatory heat input is key, surface area and tissue mass feature prominently in the time constant of the thermal response, and air motion/wind velocity dominate, with insulation. The temperature range of application is from 10°C to -100°C [69, 45, 46, 23, 70].
- 4) A "cold" model, involving heat production [35, 16], subcutaneous fat and body mass per unit surface area. Clothing insulation and air motion/body motion (shivering or activity) and wind are dominant factors. At present, this model has been most advanced for application to immersion in water at 5 to 30°C, but should be readily used for exposure at air temperatures from 0° to 20°C at rest, and to -70°C with hard work or if the extremities are prevented from being the limiting feature by use of auxiliary heated handwear and footwear [66, 67].

It is possible to define a necessary and sufficient set of input variables, write an executive program to examine the overall heat balance and automatically select which of the four proceeding models should be used [22]. However, it seems unreasonable to assume that a single model will serve for cases of: "comfort" - vasomotor temperature regulation; "heat" - sweat regulation; "cold" - shivering regulation; and "extremity cooling" - peripheral vasoconstrictive and cold induced vasodilation (CIVD) regulation.

There are varying opinions on the extent and nature of the tailoring of any model to fit individual subjects. Much of the laboratory modeling has been carried out a posteriori, fitting the data from a given individual, or group of (usually fairly homogeneous) subjects; the extent to which such models have been extrapolated far exceeds the extent to which they have been tested (a-priori or a-posteriori) against other subjects, environments or work levels. It seems clear that the greatest intra and inter individual variability will occur in the comfort models since intra- and inter-individual variability tends to disappear as we move to more severe thermal stresses. In one large series of heat studies (5 series of 10 day exposures of 10 subjects per day), correlations of tolerance time or physiological measures with the environmental forcing function (T_a , RH and W.V.) exceeded 0.95 [20]. Clearly adjustments for height, weight, surface area, body fat and morphology, and hydration level and acclimatization are necessary; whether adjustments for age, gender or racial stock are necessary is debatable. Our studies [2, 14, 60, 59], and others, suggest that age and gender may be simply treated as secondary variables related to physical fitness (VO_{2max}), which is a key factor in modeling where competition for cardiac output between muscle and skin is important.

There are several unresolved problem areas for these models. First, large variability and disagreement between the comfort model and actual comfort votes of subject populations may or may not be able to be resolved since it appears that about 1/3 of the population is relatively insensitive to mild thermal discomfort and 1/3 is hypersensitive; incorporation of psychological attributes (introversion versus extraversion, etc.) may be necessary [3]. Second, the dynamic changes in clothing insulation and permeability with wearer motion,

characterized to date by a difficulty to determine "pumping coefficient" [27] needs study; availability of "walking" copper manikins in the near future should help resolve this problem, which is key to both the heat and cold models. Third, the relationship between body heat content and the onset and degree of peripheral (extremity) vasoconstriction, and the incidence and magnitude of Cold Induced Vaso-Dilation are essential for extending the present, worst case assumption (full vasoconstriction and no CIVD) model for extremity cooling [18] to real world responses. Finally, a major unresolved problem area is that of the prediction of task performance [31]; couched in such terminology as "arousal," "motivation" and "expectation" must be some common, rational ground for predicting an individual's total task performance but at present we are, at best, able to estimate degradation for only a few of the steps involved in the total complex of performance of real world tasks.

Two of Goldman's models were evaluated in this study; these were referred to in his manuscript as the "heat" and "cold" models, and they are described in the following sections.

The "heat" model developed by Givoni and Goldman [16] has been described in a 1972 paper. In that paper, they describe a method for computing rectal temperature for various combinations of work rate, environmental conditions, and clothing. A companion paper [18] discusses a similar model for predicting heart rate. Since both papers are readily available and provide a rather complete description of the model, only a brief summary will be presented in this report. A 1975 report is also very helpful in understanding this model [4].

The thermal model defines an equilibrium rectal temperature which depends on metabolic rate, environmental conditions, clothing, and state of acclimatization of the individual. For a given set of conditions, the model also defines characteristic delay times and time constants which govern the transition from a specified initial temperature to the final equilibrium temperature. In the case of exercise or recovery from exercise, an exponential function is used to define the change; while a double exponential expression is used to describe resting changes.

Useful relationships for computing metabolic and heat transfer rates for various conditions have been derived empirically by the group at Natick and incorporated into the model. For example, an expression developed by Givoni and Goldman [16] is used to predict metabolic rate for various combinations of total mass, speed of walking, and grade; allowance for differences in terrain is also included in the model, although it is not mentioned in the 1972 paper [17]. The combined rate of heat transfer between skin and the environment owing to convection and radiation is computed using the thermal resistance of the garment (in clo) and an estimated value of the mean skin temperature; 35°C is recommended for a nude subject and 36°C for a clothed, fully sweat-wetted subject. Another important factor in this model is the maximum-evaporative capacity of the subject, which is defined in terms of the resistance to vapor transport of the garment (i_m/clo) and the difference in partial pressure of water on the skin and in the environment.

The equilibrium rectal temperature is assumed to increase linearly with both the net rate of metabolic heat production and the rate of sensible heat transfer to the subject. It also increases exponentially with the difference between the required and maximum allowable rates of evaporative heat transfer, where the required rate is simply the sum of the net metabolic rate and the rate of sensible heat transfer to the skin. A 1973 paper by Givoni and Goldman [19] discusses the effect of heat acclimatization on rectal temperature, which is also incorporated into the model.

If the rectal temperature is different from the equilibrium temperature, change occurs in a manner that depends on whether the subject is resting, working, or recovering from work. When environmental conditions or the metabolic rate change abruptly, it is assumed that there is an initial time lag before the response in rectal temperature begins. During the dead-time, rectal temperature is computed by extrapolation from the interval immediately preceeding the change. No prescription is given for handling continuously varying changes, such as a linearly increasing or decreasing environmental temperature.

The duration of the time lag and the time constant for change depend on particular circumstances. For a resting subject, the time lag is one-half hour and the transition is described by a double exponential function. During periods of work, the initial time lag

is inversely proportional to the metabolic rate, and the transition occurs exponentially with a time constant that decreases as $\Delta T_{re} = T_{re,f} - T_{re,i}$ increases. During recovery from exercise, both the time delay and time constant depend on the cooling power of the environment, which is defined as a weighted sum of the sensible and maximum evaporative rates of heat transfer from skin to the environment. The time delay decreases exponentially and the time constant increases exponentially with increasing cooling power; the transition is also described by an exponential function.

During the initial delay time for a resting or working person, rectal temperature follows the curve established at the end of the preceeding interval. For recovery, the rate of increase during the initial delay time is one-half of the rate of rise at the end of the preceeding work period.

The brief summary provided in this report should serve as an introduction to the description presented in much greater detail in the papers published by Givoni and Goldman.

Unfortunately, those who refer to the principal paper [17] will find that it contains several important expressions which require interpretation. Eqns. 1, 9, 11, 16, and 17 contain expressions of the form $e \exp z$, which is conventionally written as $\exp(z)$ or e^z . Similarly, the expression $0.1 \exp 0.4^{(t-0.5)}$ in Eqn. 10 means 0.1^u , in which $u = 0.4^{(t-0.5)}$. The term, H_{r+c} , in Eqn. 1 has the same meaning as $(R+C)$, which is used in the paragraph above Eqn. 1 and in Eqns. 5 and 6. The units of $(R+C)$ and E_{max} in Eqns. 5 and 7 are watts, not watts/°C and watts/mmHg, as given. The vapor pressure of water is expressed in mmHg in Eqn. 7. Readers who can obtain copies of the 1975 report [4] will find it somewhat easier to understand than the 1972 paper. As part of the workshop, a FORTRAN computer program was written and checked against sample problems presented in the 1975 report; this program is available from Dr. Wissler upon request.

The "cold" model developed by Strong and Goldman was described in a manuscript [67] made available prior to publication in the open literature. Therefore, this model will be described in somewhat greater detail than others. Portions of this section are excerpted from the manuscript.

This is a multi-compartment model in which heat transfer occurs through N compartments; N is arbitrarily large, but the set must include core, subcutaneous fat, and skin compartments. External thermal protective layers (clothing, air and/or water) may be included in the model by adding compartments to the three mentioned above. Energy balances equate the rate of change of thermal energy content for each compartment to the net rate of heat transfer into the compartment plus the rate at which heat is generated by sources acting within the compartment. If the mean temperature of the i^{th} compartment is T_i , the energy balance for the compartment can be written as follows:

$$m_i c_i \dot{T}_i = A_i h_{ij} (T_j - T_i) + Q_i$$

in which m_i = the mass of the compartment, c_i = the specific heat, A_i = the surface area, h_{ij} = the thermal conductance between compartments i and j , and Q_i = the rate of heat generation owing to all sources. Strong and Goldman specified Q_i to comprise the metabolic heat production which is operative only in the core compartment; they assumed that Q_i can be expressed as a linear function of the core and skin temperatures.

The physical parameters, m_i , A_i , and c_i , are defined in terms of the body mass (M), surface area (SA), mean skinfold thickness (SF), and percent body fat (BF) of the subject. Since explicit equations were not included in the manuscript provided for the workshop, an ad hoc solution was constructed by treating the height as an adjustable parameter, together with the outer radius and composition of the core compartment. Accordingly, these parameters were defined as follows:

$$1/\bar{\rho} = BF/\rho_{\text{fat}} + (1.0-BF)/\rho_{\text{aq}}$$

$$VOL = M/\bar{\rho}$$

$$R_3 = VOL/SA$$

$$H = SA/(2.0 R_3)$$

$$R_2 = R_3 - 0.0015$$

$$R_1 = R_3 - SF/2.0$$

in which $\bar{\rho}$, ρ_{fat} , and ρ_{aq} are the mean density and densities of fat

and aqueous tissue, respectively; VOL is the body volume; H is the height of the cylinders; and R_i is the outer radius of compartment, i. All dimensions are given in meters. Since it is assumed that the density of skin is ρ_{aq} and of the subcutaneous layer is ρ_{fat} , the masses of fat and aqueous tissue in the core compartment can be computed as follows:

$$m_{aq,1} = M(1.0-BF) - m_3$$

$$m_{fat,1} = M BF - m_2$$

in which m_i is the mass of compartment, i, and the subscripts, aq and fat, identify aqueous and fat components, respectively.

Definition of the model is completed by specifying the three conductances and the metabolic rate. Strong and Goldman defined the conductance between subcutaneous fat and skin in terms of the mean skinfold thickness, as follows:

$$h_{23} = 0.352/(SF-0.003).$$

The other two conductances and the relationship defining Q_1 were chosen to obtain good agreement between corresponding computed and measured values for a particular subject. While this is acceptable when the model is used to interpret experimental data, these values must be defined a priori when the model is used for predictive purposes. Inspection of the values included in Strong and Goldman's manuscript indicated that the following values were reasonable for h_{12} and h_{34} for nude subjects immersed in cold water.

$$h_{12} = 15 \text{ Kcal}/(\text{hr} \cdot \text{m}^2 \cdot ^\circ\text{C})$$

$$h_{34} = 150 \text{ Kcal}/(\text{hr} \cdot \text{m}^2 \cdot ^\circ\text{C})$$

Strong and Goldman also developed three regression equations for predicting metabolic rate as a function of rectal and mean skin temperatures for three different morphological types defined as follows: small-lean ($M < 70$ Kgm and $BF < 0.12$), average ($70 < M < 90$ Kgm and $0.12 < BF < 0.19$), and heavy-fat ($90 \text{ Kgm} < M$ and $0.19 < BF$).

For small-lean,

$$Q_1 = 998.0 - 17.0 T_3 - 9.5 T_1 \quad (r=0.66)$$

For average,

$$Q_1 = 540.9 - 13.0 T_3 - 0.4 T_1 \quad (r=0.75)$$

For heavy-fat,

$$Q_1 = 467.0 - 4.0 T_3 + 18.8 T_1 \quad (r=0.44)$$

In the preceding equations, Q_1 is expressed in Kcal/hr and T_1 (rectal temperature) and T_3 (mean skin temperature) are expressed in °C. These equations were used to evaluate the metabolic rate when this model was used to simulate various responses during the model evaluation phase of the workshop.

Since the set of differential equations defined by Strong and Goldman is linear, an analytical solution can be obtained without difficulty. However, the program written for use during the workshop employed a fourth-order Runge-Kutta numerical method to compute temperatures. This was done to provide the capability of analyzing problems in which the water temperature varies arbitrarily during the exposure without becoming involved in a complicated analytical exercise. The analytical and numerical methods are both capable of providing sufficiently accurate results when properly executed.

Common Characteristics of Models Developed by Stolwijk, Wissler, and Gordon. These three models attempt to describe human thermoregulation in more fundamental terms than the Goldman "heat" model discussed previously. In each of these models, the effect of heat transfer by conduction and convection is evaluated by subdividing the body into geometric regions representing various tissues and writing a thermal energy balance for each region.

Although they are similar, the models differ somewhat in the specific manner of subdivision and the way in which conductive heat transfer is computed. Stolwijk uses six major elements to represent the head, trunk, arms, hands, legs, and feet. Wissler further subdivides the trunk into two regions representing the thorax and abdomen, and subdivides each extremity into three longitudinal sections, although the hands and feet are not identified explicitly. Gordon's representation is similar to Stolwijk's, but the trunk is subdivided into upper and lower regions and the head is subdivided into four sections, representing the cranium, forehead, face, and neck.

In each of the major geometric elements described above, the models define subregions which represent various layers of tissue, such as skin, subcutaneous fat, muscle, bone, and viscera. Stolwijk uses four subregions in each element to represent core, muscle, fat

and skin, while Wissler and Gordon use a variable number of layers depending on the region. Stolwijk evaluates conductive heat transfer in terms of conductances which relate the rate of heat transfer between two adjacent regions to the difference in regional temperatures. Wissler and Gordon both use finite difference techniques to solve the heat conduction equation with appropriate property values assigned to each region. Hence, Stolwijk computes only a mean temperature for each subregion, while Wissler and Gordon compute temperatures at a sufficient number of nodal points to obtain an accurate temperature profile in each region.

The three models are essentially identical in their treatment of metabolic heat generation, perfusion and the boundary conditions. Heat generation in each region is assigned a value appropriate to the composition of the region, level and type of work, and intensity of shivering. During rest, heat generation occurs predominately in the brain and abdominal viscera; but during exercise, additional heat generation in active muscles of the arms and legs predominates. As a first approximation, the distribution of blood flow is similar to that of heat generation. During rest, most of the cardiac output goes to the brain and abdominal viscera; and during exercise, a large fraction of the additional cardiac output goes to active muscles. The remainder flows to the skin for thermoregulatory purposes. In addition to tissue regions, the models include a blood volume; regional heat transfer between blood and tissue is computed assuming that blood has a characteristic arterial temperature when it enters a capillary bed and the local tissue temperature when it leaves. Heat transfer between skin and the surroundings occurs by convection, radiation, and evaporation, which are treated in a similar manner by all three models.

In addition to regional heat balances, which account for temperature changes owing to variable energy content, the models contain thermoregulatory control equations that define the physiological responses necessary to maintain human bodily temperatures within an acceptable range. All three models are influenced to a considerable extent by the 1966 paper by Stolwijk and Hardy [64], which represented the first attempt to construct physiological control equations using

feedback control concepts. Stolwijk's model is still used relatively unchanged from the form that evolved between 1966 and 1971 under NASA sponsorship, although the models of Gordon and Wissler now include thermoregulatory control concepts somewhat different from those proposed originally by Stolwijk and Hardy. In the following sections, each model's treatment of physiological factors will be discussed briefly.

Stolwijk's Physiological Control Equations. The thermoregulatory control system is divided into three parts consisting of sensors which generate afferent signals containing information about the thermal state of the system, an integrating system which interprets these signals and generates appropriate effector signals, and the peripheral system which modifies the effector commands according to local cutaneous conditions and produces appropriate action.

Thermoreceptors are distributed throughout the skin and in the brain. The model recognizes that different skin areas contain different densities of thermoreceptors and that both warm and cold receptors are present in a particular site. In all models, the location of thermoreceptors must coincide with the locations of appropriate computed temperatures. Stolwijk uses head core temperature to represent the hypothalamic temperature and skin temperatures to define the state of cutaneous sensors. At each site, thermoneutrality is defined in terms of a local reference (setpoint) temperature. When the temperature is above the reference temperature, warm receptors fire and cold receptors are silent; otherwise, cold receptors fire and warm receptors are silent. Although it is not included in this model, there is also evidence that rate sensitivity can be demonstrated for thermoreceptors.

At various times, it has been proposed that the magnitude of the effector command is proportional either to a weighted sum of the afferent signals, with warm and cold receptors producing signals of opposite sign, or to the product of the central and peripheral signals. In a recent version of his model [65], Stolwijk uses linear relationships to define the effector commands for sweating, cutaneous vasodilation and vasoconstriction, and a product relationship for shivering. Furthermore, the sweating effector command is modified peripherally through an exponential relationship which doubles the rate of sweat

production for each 4°C increase in local skin temperature [48].

Bloodflow is variable in muscle and tissue compartments. In muscle, the incremental bloodflow rate (in liters/hr) is set equal to the incremental metabolic rate (in watts), both measured relative to the resting rates. Bloodflow in skin is determined by the strength of the effector commands for vasodilation and vasoconstriction; a prorated share of the enhanced bloodflow to skin is added to the resting flowrate in each element, and the sum is divided by a term which increases linearly with increasing vasoconstrictor outflow. Since the ratio of central to peripheral influence on vasodilation is 8:1, while the corresponding ratio for vasoconstriction is 1:1, the model permits vasodilator and vasoconstrictor outflows to occur simultaneously.

This is a relatively simple model, both in its treatment of heat transfer within the body and the description of cardiovascular responses to thermal stress and exercise. Consequently, the model is not expensive to use and can even be run on a microprocessor. It yields quite satisfactory results under certain conditions and was used extensively to simulate astronaut performance during Apollo missions. However, one would expect that there are conditions under which the model is not adequate; these limitations will be discussed when the model is evaluated in a subsequent section of this report.

Specific Features of Gordon's Model. This model was designed to simulate human thermal behavior during exposure to cold air. Although it contains Stolwijk's equations for vasodilation and sweating, the published description [29] of this model makes no mention of its response to conditions of heat stress. Prof. Gordon confirmed during the workshop that attention had been focused on cold stress when the model was developed.

Since this model is described in considerable detail in a readily available paper [29], only a brief description will be included in this report. Fourteen major cylindrical or spherical segments are used to represent the head, forehead, face, neck, thorax, abdomen, arms (2), hands (2), legs (2), and feet (2). These segments were chosen for their distinct thermophysical properties, anatomy, physiological characteristics, and/or control system characteristics.

Each segment consists of several concentric shells which represent anatomical structures found at different depths within the segment. Each tissue shell is subdivided further by superimposing a grid on it, and a finite-difference technique is used to derive an energy balance for the tissue around each node. All thermophysical and physiological properties are assumed to be constant throughout a shell. The blood flowrate and metabolic rate are separated into two parts: a basal part (including the Q_{10} effect) and a regulated part which is defined by the exercise level and thermoregulatory requirements.

The circulatory system is modeled as blood which flows outward from a central pool into major body segments, after passing through countercurrent heat exchanges. Blood flows from the arterial pool of a given segment into capillaries where equilibrium is established with adjacent tissue. Drainage from a capillary bed empties into a venous pool, from which it returns to the central blood pool after passing again through a countercurrent heat exchanger. The arterial blood temperature is simply the cup-mixing temperature of the various venous streams.

Heat loss through the respiratory tract is assumed to be proportional to the metabolic rate; only evaporative losses are evaluated in the model. An attempt is made to distribute the respiratory heat loss realistically, as follows: 10 percent is removed from the neck muscle band, 70 percent from the outer facial muscle band, and 20 percent from the inner facial muscle band.

Thermophysical properties and basal physiological values for each anatomical region were determined by Gordon without reference to other models. The distribution of material was determined from measurements made on scaled drawings in Eycleshymer and Shoemaker [15]. Values assigned to basal blood flowrate in various regions were also determined with considerable care using data from many sources.

A unique feature of Gordon's model is his inclusion of heat flux through the skin in the set of values sensed by thermal receptors, which also sense head core temperature and skin temperatures. Motivation for assuming that thermoreceptors are responsive to heat

flux is provided by the desire to generate a prompt response to sudden changes in environmental conditions. The existence of such receptors has been debated for several decades, but a definitive answer remains to be obtained.

Following Stolwijk's control strategy, Gordon defines the afferent signal from each receptor to be the difference between the instantaneous value of temperature or flux and a reference value chosen so that the afferent signal vanishes under thermoneutral conditions. Individual afferent signals are assigned a weight proportional to the density of receptors in the region and summed to form effector signals for sweating, shivering and vasomotor action. A detailed discussion of the equations used for each physiological response is presented in Gordon's paper [29].

Wissler's Physiological Control Equations. Since an effort has been made to incorporate many of the phenomena discussed earlier into this model, it is rather complex. However, only a brief description of the model will be included in this report; those desiring additional information should refer to a recently published paper [71].

As originally developed, the thermal model did not contain physiological control, but was only concerned with the physical (passive) aspects of heat transfer in the human [72, 73]. The first physiological control equations incorporated into this model were based on the feedback control scheme described by Stolwijk and Hardy [64]. A few years later, a manuscript prepared at the Pierce Foundation by Mitchell [44] and the pulmonary model developed by Grodins, Buell and Barb [30] provided stimulation to add material balances for oxygen, carbon dioxide and lactate to the model. This made it possible to deal with blood perfusion in more physiological terms than was previously possible. Other aspects of the control system changed also as new experimental results published by various investigators were incorporated into the model.

The general arrangement of thermoreceptors, an integrating center, and the peripheral effector system described by Stolwijk are used also in Wissler's model. However, he assumes that thermoreceptors respond to rate of change of temperature, as well as temperature itself. A unique aspect of his controller is that the time derivative

of temperature is fed into an integrator of the form,

$$\dot{S} = \beta (\dot{T} - S) \quad (4)$$

in which S = afferent signal generated by the changing temperature, $\dot{T}$. An alternative form obtained by solving the differential equation is

$$S(t) = \int_{-\infty}^t \beta \dot{T}(\tau) e^{-\beta(t-\tau)} d\tau \quad (5)$$

or, after integration by parts,

$$S(t) = \beta [T(t) - \int_{-\infty}^t T(\tau) e^{-\beta(t-\tau)} d\tau] \quad (6)$$

Hence, the signal produced by these rate sensitive thermoreceptors is proportional to the difference between the instantaneous temperature and an exponentially decreasing weighted mean of previous temperatures. This proves to be a useful concept under certain conditions.

Wissler's treatment of skin bloodflow is similar to that of Stolwijk. Thermally induced vasodilation occurs in response to elevation of hypothalamic or skin temperature. Vasodilator outflow varies linearly with temperature, and following the suggestion of Rowell and Wyss [57], the ratio of central to peripheral influence on vasodilation in skin is set at 20:1. The model also includes a term which depends on the rate of change of central temperature, as modified by the integrator mentioned previously, although the contribution from this factor is relatively small. Vasodilator outflow is defined as an incremental blood flow distributed throughout the skin. However, the actual flow-rate is affected also by vasoconstrictor outflow, which is influenced both by an individual's thermal state and central blood volume. In computing the thermally mediated vasoconstrictor efferent signal, equal weight is assigned to central and cutaneous temperature changes; in effect, this means that changes in skin temperature, which are much larger than changes in hypothalamic temperature, are dominant. The effect on skin bloodflow of lowered central blood volume owing to vasodilation in active muscle is taken into consideration by adding a term proportional to work rate to the thermally generated vasoconstrictor term. When computing blood flowrates to organs other than skin, a third vasoconstrictor component is added in proportion to the strength of the vasodilator outflow; this is intended to allow for the

effect of reduced central blood volume owing to vasodilation in skin. Hence, this model directs cardiac output to abdominal viscera, muscle, or skin in proportion to each tissue's relative need, while also making allowance for thermoregulatory requirements.

Wissler's model also accounts for venomotor action which directs venous blood in the extremities either to superficial veins under hot conditions, or to deep veins under cold conditions. This has the effect of allowing countercurrent heat exchange between arterial and venous streams when it is necessary to conserve heat, while not preventing convective transport of heat from core to skin in a warm environment.

The control function for sweating is very similar to the one used by Stolwijk, in that a central drive, which depends linearly on central and cutaneous afferent signals, is modified peripherally as a function of local skin temperature. Various experimental studies have shown that the gain assigned to the central receptor should be an order of magnitude larger than the gain of cutaneous receptors. A minor difference between the two models is that Wissler includes a contribution to central drive owing to changing central temperature, as interpreted by the integrator described previously.

The controller used by Wissler for shivering is considerably more complex than the one used by Stolwijk, although they may produce comparable metabolic rates under quasi-steady-state conditions. Wissler's controller consists of three parts which account for the contributions of rates of change of skin and central temperatures and the integrated effect of lowered skin and central temperature. During a period of rapidly decreasing skin temperature, such as the initial period of cold immersion, the time derivative of skin temperature is fed into the integrator described by Eqn. 4 to produce a transient increase in shivering intensity which persists for approximately five minutes. The rate of change of central temperature is assumed to be sensed directly; it is of significance during special circumstances, such as the ingestion of cold substances or mild exercise following prolonged exposure of extremities to cold. The integrated effect of lowered skin and central temperatures produces a forcing function which drives an integrator similar to the one mentioned previously.

Over a broad range of conditions, the forcing function is approximately equal to the product function described by Hayward, et al. [34], and, therefore, the action of this controller differs significantly from theirs only during periods of rapidly changing temperature.

A unique feature of the shivering controller used in this model is the explicit recognition that intense shivering cannot be maintained indefinitely. Although fatigue is a complex phenomenon, depletion of muscle glycogen appears to be a contributing factor in many cases, and the limiting condition employed in this model is based on glycogen depletion. This feature of the model assumes great significance when one attempts to predict survival times for conditions requiring prolonged intense shivering to maintain acceptable bodily temperature.

For completeness, the material balances mentioned earlier will also be described briefly. At each of the nodes where temperature is computed, balances are written for oxygen, carbon dioxide, and lactate. The rate of accumulation of each chemical species in tissue is set equal to the sum of the rate of production (consumption is negative) by metabolic reactions and the rate of transport into the region by blood perfusing capillaries of the region. In the case of highly diffusible substances, such as oxygen and carbon dioxide, it is assumed that quasi-equilibrium exists between capillary blood and adjacent tissues. Allowance for longitudinal variation along capillaries is made by dividing them into four sections and writing balances for each section. In the case of lactic acid, equilibrium is not assumed to exist between blood and tissue; instead, transport across the capillary wall occurs at a rate proportional to the difference in concentration.

The local metabolic rate, as specified by the intensity and kind of work, determines the local rate of production of ATP, which may occur either aerobically or anaerobically, depending on circumstances. If the oxygen supply is adequate, carbohydrates (represented in the model by glucose) are oxidized completely to carbon dioxide and water; but when the oxygen tension falls below a critical level, ATP is produced together with lactic acid in the anaerobic cycle. Lactate produced in active muscles diffuses into blood and is distributed

throughout other aqueous tissues. Since accumulation of lactate in submaximal exercise has been observed to occur primarily during the first few minutes when cardiac output is still increasing, and to reach a plateau during sustained exercise, the local perfusion rate in muscle is assumed to increase as lactate concentration increases. It also increases as the end-capillary oxygen tension decreases.

The model also includes material balances for oxygen, carbon dioxide and lactate in each arterial and venous pool. Oxygen and carbon dioxide are exchanged with the environment through the lungs, and lactate is converted into glycogen in the liver.

Adding material balances to a thermal model increases the computational requirements considerably. Since there are three components to be computed at four points along each capillary, the amount of data to be stored is much greater than what is required by the thermal model alone. Although that is becoming an insignificant problem with modern computers, choosing the correct set of equations for either aerobic or anaerobic conditions and computing concentrations in tissue and blood are time consuming operations which preclude running this program on a personal computer. However, it does run very well on a VAX and has been run on an HP-9000 desktop computer, so the computational requirements are not as imposing today as they were two years ago.

EXPERIMENTAL STUDIES

Since all of the theoretical models described in the preceding section only approximate the very complex human thermal system, experimental data are required to evaluate free parameters and establish the validity of the model. Therefore, the existence of a good set of experimental data is essential to model development. One objective of this project was to assemble such a set of data. Studies which were chosen for inclusion had to satisfy the following criteria: (1) The set of values measured had to be sufficiently complete to eliminate arbitrary adjustment of parameters. For example, a measure of subcutaneous fat thickness, such as mean skinfold thickness, was required for those studies involving exposure to cold. (2) Conditions had to involve sufficient stress to cause a significant response while not overloading the thermoregulatory system. An example of conditions

which are of limited value is provided by heavy work in a hot environment because a relatively small fraction of the heat generated metabolically is transferred to the environment, and all models respond in essentially the same way. (3) The study had to be conducted by an experienced investigator using accurate procedures. Data which have been assembled are summarized in the following table.

Table 2. Source of Experimental Data

INVESTIGATOR: A. Påsche, et al.

Norwegian Underwater Technology Center
N-5034 Ytre Laksevåg, (Bergen) Norway

REFERENCE: A. Påsche, et al.

Deep Ex 81, Project IV, Thermal Studies.
Subproject: Thermal Model.

• Preliminary Report No. 20-82, Feb. 15, 1982

ENVIRONMENT:

Helium-oxygen at two pressures--41 and 51 Bars-- $p_{O_2} \approx 0.5$ Ata;
 $T_{db} \approx 28^\circ\text{C}$

EXERCISE:

Subjects were seated quietly.

MEASURED VALUES:

T_{re} , 15 skin temperatures, HR, thermal flux measured on the
arm, chest, thigh and front calf.

NUMBER OF SUBJECTS:

Three subjects were exposed to both pressures.

PHYSICAL CHARACTERISTICS:

Subject	Ht <u>cm</u>	Wt <u>Kgm</u>	SA <u>m²</u>	SFT <u>mm</u>
1	185	85	2.09	19.5
2	177	74	1.91	13.0
3	178	78	1.96	9.5

INVESTIGATOR: J. J. Vogt, et al.

Centre d'Etudes Bioclimatiques, Centre National de la
Recherche Scientifique, 67087 Strasbourg-Cedex, France

REFERENCES: (1) Libert, J.P., et al, "Central and peripheral inputs in sweating regulation during thermal transients." J. Appl. Physiol., 52: 1147-1152 (1982); (2) Libert, J.P., et al., "Effect of rate of change in skin temperature on sweating rate." J. Appl. Physiol., 47: 306-311 (1979); (3) Libert, J.P., et al., "Sweating response in man during transient rises of air temperature," J. Appl. Physiol., 44: 284-290 (1978).

ENVIRONMENT:

Air of variable temperature--either a step change or saw tooth variation of T_{db} .

EXERCISE:

Subjects reclined on a mesh support.

MEASURED VALUES: T_{re} , T_{es} , 15 skin temperatures, HR, sweat and evaporation rates

NUMBER OF SUBJECTS:

Three subjects participated in one run each and two subjects participated in two runs each.

PHYSICAL CHARACTERISTICS:

Subject	Ht <u>cm</u>	Wt <u>Kgm</u>	SA <u>m²</u>	SFT <u>mm</u>
LP	177	60	1.75	6.5
KG	170	63	1.72	NA
OM	170	63	1.72	NA
OC	176	83	2.00	11.7
LA	172	71.4	1.84	NA

INVESTIGATOR: W. R. Keatinge
The London Hospital Medical College, Turner Street,
London E1 2AD

REFERENCE: None

ENVIRONMENT: Immersion to the neck in water at 29°C for
3.75 hours

EXERCISE:

Subjects sat quietly on an underwater chair.

MEASURED VALUES: T_{re} , T_{ac} , M, Ht, Wt, SFT

NUMBER OF SUBJECTS:

Three subjects exposed once each

PHYSICAL CHARACTERISTICS:

Subject	Ht <u>cm</u>	Wt <u>Kgm</u>	SA <u>m²</u>	SFT* <u>mm</u>
1 (m)	176	67.8	1.84	10.8
2 (f)	166.5	68.5	1.78	21.2
3 (f)	165.5	61.2	1.68	17.4

*Mean skinfold thickness was computed as $2 \times \text{mean fat thickness} + 2.4 \text{ mm}$

INVESTIGATOR: Paul Webb and Lorne Kuehn
 Webb Associates
 Yellow Springs, Ohio

REFERENCE: The Effect of Cooling Rate Upon the Tolerance and Thermal Responses of Mildly Hypothermic Men, pp. 161-162 in Preprints for the 1979 Aerospace Medical Association Meeting

ENVIRONMENT: Immersion to the neck in a bath calorimeter with water temperatures of 18 and 24°C. Time of exposure was 50 to 60 minutes.

MEASURED VALUES: T_{re} , T_{ac} , M, HR, Ht, Wt, BF, SFT,
 Rate of heat transfer from skin to the bath

NUMBER OF SUBJECTS:

Four subjects exposed twice to both bath temperatures

PHYSICAL CHARACTERISTICS:

Subject	Ht cm	Wt Kgm	SA m ²	BF %	SFT mm
PW	171.6	76.8	1.90	23.2	18.1
JA	182.5	73.5	1.95	16.9	9.4
JR	169.7	69.1	1.80	9.0	6.0
ST	187.5	95.4	2.22	23.2	18.7

INVESTIGATOR: B. Saltin, A. P. Gagge, and J.A.J. Stolwijk,
Pierce Foundation, Yale University, New Haven, Conn.

REFERENCE: Saltin, B., et al., "Body temperatures and sweating during thermal transients caused by exercise," J. Appl. Physiol., 28: 318-329 (1970).

ENVIRONMENT: Air at 10, 20, or 30°C.

EXERCISE: Subjects exercised for 30 minutes at approximately 25, 50, and 75% of $V_{O_{2,max}}$. The three exercise periods were separated by 30-minute rest periods.

MEASURED VALUES:

T_{re} , T_{es} , $\bar{T}_s$, M, evaporation rate

NUMBER OF SUBJECTS:

Three subjects participated in a total of 10 experiments

PHYSICAL CHARACTERISTICS:

Subject	Ht <u>cm</u>	Wt <u>Kgm</u>	SA <u>m²</u>
BC	183	79	2.03
BS	187	89	2.17
PM	189	84	2.09

To facilitate comparison between computed and measured values, a computer data base containing the experimental values was established [68]. A representative set of data is presented below.

WEDB
SUBJECT PW 1
18 DEG C WATER TEMPERATURE
1
13

TIME (MIN)
0.00 5.00 10.00 15.00 20.00 25.00 30.00 35.00 40.00 45.00 50.00
55.00 60.00 65.00 70.00 75.00 80.00 85.00 90.00 95.00 100.00 105.00
110.00 115.00 120.00 125.00 130.00 135.00 140.00 145.00 150.00 155.00

MEAN SKIN TEMPERATURE (DEGREES C)

35.88 36.00 36.09 27.30 29.40 31.21 32.94 34.24 35.36 35.88 35.88
36.21 36.14 35.81 35.35 34.75 34.72 34.62

RECTAL TEMPERATURE (DEGREES C)

36.85 36.70 36.85 36.85 36.85 36.85 36.65 36.70 36.65 36.60 36.60
36.50 36.35 36.06 35.92 35.78 35.94 35.81 35.73 35.69 35.63
35.60 35.63 35.67 35.73 35.78 35.86 35.96 36.03 36.15 36.23

TYMPANIC TEMPERATURE (DEGREES C)

35.75 35.75 35.70 35.60 35.60 35.70 35.60 35.45 35.30 35.20 35.10
35.00 34.90 35.03 35.12 35.23 35.30 35.38 35.47 35.56 35.59
35.63 35.72 35.81 35.91 36.00 36.09 36.20 36.25 36.28 36.33

ESOPHAGEAL TEMPERATURE (DEGREES C)

METABOLIC RATE (WATTS)

96.98 103.69 181.02 202.50 211.29
234.37 129.00 138.77 143.65 121.33 94.14 76.01 70.43 76.71
78.10 70.43 71.82 70.43 71.13 76.71 72.52 80.89 84.38 85.77

SKIN EVAPORATIVE HEAT LOSS RATE (WATTS)

RESPIRATORY HEAT LOSS RATE (WATTS)

3.00 3.49 6.07 6.83 7.18
7.81

TOTAL HEAT LOSS RATE (WATTS)

635.12 664.90 663.57 371.67 325.79
319.02 -177.1 -407.2 -431.6 -406.5 -431.6 -397.5 -119.2 -77.40
-120.6 -127.6 -71.13 -109.5 -53.69 155.50 169.45 185.79 60.67 142.95

HEART RATE (BEATS/MIN)

55.00 52.00 52.00 50.00 56.00 56.00 75.00 75.00 60.00 80.00 70.00
68.00 100.00

HEAD SKIN TEMPERATURE (DEGREES C)

CHEST SKIN TEMPERATURE (DEGREES C)

REAR CALF SKIN TEMPERATURE (DEGREES C)

ABDOMEN SKIN TEMPERATURE (DEGREES C)

LOWER ARM SKIN TEMPERATURE (DEGREES C)

WRIST SKIN TEMPERATURE (DEGREES C)

FRONT THIGH SKIN TEMPERATURE (DEGREES C)

FRONT CALF SKIN TEMPERATURE (DEGREES C)

To facilitate comparison of computed and measured data, graphs were prepared showing the measured values together with corresponding values computed using applicable models. A set of graphs covering the full range of conditions is presented at the end of this report. Unfortunately, spatial limitations preclude publishing all of the graphs generated for the workshop. Anyone who is interested in a particular unpublished set should contact Dr. Wissler.

Since the author of this report developed one of the models, it would be unfair to compare models and select the "best one." Indeed, none of the models is best suited for all applications. Under certain conditions, an empirical model which can be programmed on a hand-held calculator may be ideal; but under other conditions, it may be quite inadequate. Hence, attention will be focused on identifying conditions for which each model provides reasonable results, and readers will have to form their own judgments about the suitability of particular models for specific purposes.

A few general comments are relevant to many of the comparisons. The first concerns measurement and computation of the central temperature. There are four sites at which central temperature can be measured--the mouth, esophagus, auditory meatus, and rectum. In none of these sites does the temperature agree precisely with theoretically important values, such as the hypothalamic and aortic temperatures, which cannot be measured. As is often the case, there is an inverse relationship between ease of measurement and value of the result.

An oral temperature is easily obtained, but it is so subject to extraneous influences that it is almost never used. Rectal and aural temperatures, which are also easily measured, provide useful data under favorable conditions. However, they also have special characteristics which must be taken into consideration. In particular, the rectal temperature responds more slowly to passively induced changes than either the aural or esophageal temperatures; there are also differences during exercise, but the relationship between the three temperatures is different [13]. Temperature measured on or near the tympanic membrane responds rapidly to changing conditions, but this temperature tends to be influenced by environmental conditions whenever the ambient temperature lies outside of the comfort range. Even when

the external ear is insulated from the environment, bias can sometimes be detected in the aural temperature. Although the reason for such influence is not fully understood, the explanation probably involves countercurrent heat exchange between venous blood from facial skin and blood in the carotid artery. The esophageal temperature measured at the level of the heart appears to provide the best estimate of the arterial temperature, although it too can be influenced by environmental conditions if the ambient temperature is sufficiently low. This is especially true under hyperbaric conditions when the gas is very dense, but it does not appear to be a serious problem under usual conditions.

Of the three temperatures--rectal, aural, and esophageal--only the esophageal temperature is readily compared with values computed using one of the physically based models. All such models include a central blood temperature which can be compared with the esophageal temperature. On the other hand, the rectal and aural temperatures are influenced by blood and adjacent tissue temperatures in ways that are not well understood and, therefore, are not readily incorporated into any model. This presents a dilemma for both experimentalists and analysts.

Several additional comments will also help to interpret the results. One is that the initial state of an individual is never known with good accuracy, which means that there is always some uncertainty about the initial sensible heat content of the subject. During relatively short transients, such as those under consideration, computed temperatures can be shifted $\pm 0.5^{\circ}\text{C}$ by simply changing the initial temperatures in the arms and legs $\pm 1^{\circ}\text{C}$. However, this was not done; instead, the reader is asked to compare the shapes of the measured and computed curves during the later part of the observation. If the two temperatures follow parallel paths which differ by no more than a few tenths of a degree C, agreement can be considered to be satisfactory.

The reader should also note that different temperature scales were used on the graphs to obtain the best possible resolution. Before concluding that agreement between computed and measured values is either good or bad in a particular case, the reader must make

allowance for the temperature scale used to display that case.

For each simulation, the subject's physical characteristics and the environmental conditions reported by the investigator were used as input data for the model. Physical characteristics normally included the subject's weight, height, and mean skinfold thickness (for cold exposures). Since resting metabolic rates were not reported for any of the subjects, values were estimated making allowance for each subject's weight and the measured metabolic rate during the early stages of the experiment. When exercise was involved, measured metabolic rates were used to define the intensity. Environmental conditions included the kind of fluid (gas or water), fluid velocity, temperature, and humidity (for gases), and information about the radiant load. The same physiological parameters were used for each model for all conditions; no effort was made to improve agreement between computed and measured values by adjusting parameters in the thermoregulatory control equations.

Readers should make allowance for the fact that individual responses are being used to evaluate the models. Although this provides a very demanding test of the models, it also offers several advantages; one is that important individual characteristics, such as subcutaneous fat thickness in the case of cold exposure, are considered explicitly, and the other is that data can be used from various studies, even though they differ in important details. However, there are also disadvantages, including the fact that individual differences tend to be emphasized, and experimental artifacts reflect unfavorably on the models. These disadvantages can be overcome to some extent by comparing responses from related experiments in an effort to detect consistent differences between computed and measured values.

Each model will be discussed separately. An effort will be made to present generally important observations in an objective manner. Although the amount of detail provided will vary for the various models, this is a function of the author's familiarity with the models and should not be construed to represent special endorsement or criticism of any given model.

Goldman's Heat Model. This model was tested against the seven, constant temperature data sets of Saltin, et al. and the four, step-

change data sets of Libert, et al. Subjects performed exercise at 25, 50, and 75 percent of $\dot{V}_{O_{2, \max}}$ in the first series, and remained at rest in the second.

This model allows one to compute both rectal temperature and heart rate. The two sections of the program are independent, and only the thermal section is evaluated in this report. Skin temperature appears explicitly in this model as a specified quantity, rather than a computed quantity. Following guidelines presented in Berlin's report [4], a value of either 35 or 36°C was assigned to the skin temperature depending on the total thermal resistance of the garment and fluid boundary layer. Hence, one cannot expect good agreement between computed and measured skin temperatures. This model does not yield values for the rates of sweating, evaporation or sensible heat transfer, although a maximum (diffusion limited) evaporation rate is estimated.

Agreement between computed and measured rectal temperatures is generally quite good, although the model has a tendency to overestimate the increase in rectal temperature during heavy exercise in a hot environment. Even though this is a heat model, it seems to provide better results for the 10°C simulations than for the 30°C simulations. This is somewhat surprising because the principal defect in the way these simulations were conducted was that mean skin temperature was arbitrarily set at either 35 or 36°C, depending on the thermal resistance from skin to environment. Comparison of computed (assigned) mean skin temperatures with measured values indicates that such assignment is least valid at 10°C, where the best agreement between computed and measured rectal temperatures was obtained.

An additional simulation was performed for subject PM at 10°C to see what effect decreasing the mean skin temperature to 26°C would have on the computed rectal temperature. Somewhat surprisingly, the effect was to increase the rectal temperature by 0.3 to 0.4°C. Examination of the model equations reveals that decreasing the assumed skin temperature affects the equilibrium rectal temperature as follows: (1) the rate of heat transfer from environment to skin increases (the rate of transfer from skin to environment decreases); (2) the required sweat rate increases; (3) the maximum sweat rate decreases; and (4)

$T_{re,f}$ increases because of (1), (2), and (3). Since this model was apparently developed to describe human thermal behavior under conditions for which the mean skin temperature is approximately 36°C, it is not clear what should be expected when the model is used to simulate exposure to cold air.

Anyone interested in using this model should certainly read the papers published by Goldman and his associates, who tested the model under various conditions. In general, they obtained excellent agreement between computed and measured rectal temperatures when the mean response of sizeable groups of subjects was used. Of course, inter-individual variation causes greater differences when computed results are compared with measured values for an individual, as was done for the workshop. It seems clear that this model has value when used for the intended purpose. Stated in general terms, that purpose is to provide a prediction of rectal temperature changes occurring for various combinations of environmental conditions, clothing and exercise. Since only rectal temperature is predicted, evaluation of this model reduces to comparison of computed and measured values of a single variable, which is more nearly a problem in statistics than in physiology. The limited number of cases studied during the workshop does not allow one to make strong statements about this model; the original references are more helpful in this regard. However, results presented in this report do provide an additional indication of the kind of individual variation to be expected.

Goldman's Cold Model. This model was tested against the bath calorimeter data of Webb and Kuehn, and Keatinge's three runs involving immersion in 29°C water. The sixteen sets of data provided by Webb involved duplicate runs on each of four subjects immersed for one hour at two water temperatures, 18 and 24°C. Exercise was not a factor in any of these studies.

This model yields values for core and mean skin temperatures, metabolic rate, and rate of heat transfer to the environment. In the experimental studies, both tympanic and rectal temperatures were measured, but the computed core temperature is compared only with the rectal temperature, which is the procedure followed by Strong and Goldman in developing the model.

Inspection of the graphs of computed and measured rectal temperatures indicates that this model provided very good results in some cases and totally erroneous results in others. Agreement tended to be better for the leaner subjects (JR and JA of the Webb-Kuehn study) than for the fatter subjects (PW and ST). However, the most disturbing results were obtained for Subjects 2 and 3 of Keatinge's study, with the model predicting increasing rectal temperatures during immersion in 29°C water.

To some degree, the anomalous responses obtained for these two subjects is a consequence of the values assigned to the thermal conductances and the form chosen for the metabolic relationship. For example, using a value of 25.0 Kcal/(m².hr,°C) for the conductance from core to subcutaneous fat, instead of the "standard value" of 15.0, caused the core temperature to decrease rather rapidly when the heavy-fat metabolic relationship was used. Results of a small parametric study are summarized in Table .

Table 3. Summary of Results Computed Using Goldman's Cold Model for Keatinge's Subject 2 During Immersion in 29°C Water.

h_1 Kcal/(hr.m ² .°C)	h_3	Body Type	T_{re}^* °C	M^* watts
15	150	Average	37.9	162
15	150	Heavy-fat	37.4	132
20	150	Average	37.5	161
20	150	Heavy-fat	36.9	123
25	150	Average	37.3	161
25	150	Heavy-fat	36.7	117
15	250	Average	37.9	165
15	250	Heavy-fat	37.3	132

*Tabulated values of rectal temperature and metabolic rate were computed for 60 minutes of immersion.

Comparison of computed and measured metabolic rates indicates that reasonable values are generally obtained from the model. However, it is not clear how the metabolic rate should be computed for subjects who do not fit into one of the three classes defined for the model. As the results in Table 3 show, considerable variation in both temperature and metabolic rate occurs depending on whether one is classified as "average" or as "heavy-fat." Therefore, use of this model

for predictive purposes seems to be limited by lack of definitive rules for specifying the thermal conductance, h_1 , and the metabolic relationship.

Gordon's Model. Unfortunately, this model was not evaluated as thoroughly as the others because of the large effort required to prepare data for a particular run. As is the case with most transient-state models, this model uses a marching procedure starting with given initial conditions and taking a series of small steps to advance the solution over some period of time. In most models, successive steps are taken without calling for new data, unless there is a change in the imposed conditions, but Gordon's program calls for new data at the beginning of each time step. In the author's opinion, this feature seriously limits the usefulness of the model and should be changed.

It was also discovered during the evaluation that this model cannot be used to simulate immersion in water. An iterative procedure used to compute new temperatures at the end of each time interval fails to converge when heat transfer coefficients large enough to simulate immersion are used. Although this program was not used to simulate hyperbaric exposures, it is likely that convergence problems would also occur when one specifies the relatively large heat transfer coefficients that exist in hyperbaric heliox.

Since Gordon's model contains unique features, notably the inclusion of thermal flux in the set of afferent signals, not found in other models, it is unfortunate that a more thorough evaluation could not have been conducted. The capability of the model for simulating cold air exposures is discussed in Gordon's paper [29]. One warm air exposure (subject PM at 30°C in Saltin's study) was simulated, and the results were comparable to those obtained using other models.

Stolwijk's Model. This model was tested against all of the experimental data, even though it was developed primarily to describe human response to heat stress. Computed central blood temperature was compared with the measured esophageal or tympanic temperature, and trunk core temperature was compared with the rectal temperature.

Comparison of computed and measured values for the data reported

by Saltin, et al. indicates that this model yields good results when used to simulate exercise in a warm environment (30°C). Computed changes in central temperatures and sweat rates are in good agreement with observed values. Results computed for lower environmental temperatures appear to be somewhat less satisfactory, with a definite bias apparently present in results computed for exposure to 10°C air. As the ambient temperature decreases, computed central temperatures seem to decrease more than they should. Since the computed mean skin temperatures appear to be higher than the measured values, it is possible that the model does not properly account for vasomotor responses resulting from cold stress.

Comparison of computed values with the experimental data of Libert, et al. is also informative. Agreement is quite good during the first half of these 200-minute exposures to elevated environmental temperatures. During the second half, however, considerable difference between computed and measured deep temperatures develops because the model approaches equilibrium more rapidly than the human subjects. (It should be noted that the small step changes appearing in the graphs of esophageal and rectal temperature computed using Stolwijk's model are artifacts which were introduced by rounding data to the nearest 0.1°C; the computed curves should actually be smooth.) Agreement between computed and measured mean skin temperatures and rates of evaporation is also quite good.

A more detailed analysis of this model has been published by Stolwijk and Hardy [65]. In addition to describing the basis for the model and presenting the entire computer program, these authors discuss validation of the model using several of the test cases submitted to the workshop. That discussion used in conjunction with the results presented in this report should provide a rather accurate view of the model's ability to simulate human thermal responses to heat.

As noted previously, this model was designed to describe man's response to heat, although it includes cold responses owing to shivering and vasoconstriction. It has already been observed that predicted responses in 10°C air are not as satisfactory as at higher temperatures. Comparison of computed and measured values for immersion in cold water confirms that this model has limited capability for simulating cold

exposures. For most of the cold exposures simulated, central temperatures fall rapidly into the 36°C range with a corresponding increase in shivering metabolism. Equilibrium is rapidly established when the rate of metabolic heat production becomes large enough to balance the rate of heat loss to the environment. While this is qualitatively correct, it does not agree in several important respects with typical responses.

The Stolwijk model has been adapted by Montgomery [47] for analysis of cold immersion. A significant difference between the two models is Montgomery's use of 11 concentric layers in each element rather than the 4 layers used by Stolwijk. It seems quite likely that one source of error in Stolwijk's model is the poor definition of internal temperature profiles provided by only 4 temperatures [74]. Montgomery employed control equations for shivering and vasoconstriction which have the same form as those used by Stolwijk, although the numerical values of parameters may be different. In any event, those who want to use the Stolwijk model to simulate immersion in cold water should consult Montgomery's paper.

Wissler's Model. Since this model was developed to simulate human thermal response over a broad range of environmental conditions and work rates, it was tested against all of the experimental data. Computed central blood temperature was compared with the measured esophageal or tympanic temperature. A rectal temperature is computed in this model by assuming that the measurement site is located within an inert mass with thermal inertia which is influenced by the temperatures of various blood streams, including the arterial stream in the lower trunk, and the venous streams coming from superficial tissue in the lower trunk and the thighs.

With the exception of one subject (BS) this model yielded results generally in good agreement with the measured values of Saltin, et al. However, computed mean skin temperatures tended to be out of phase with measured values, especially at the highest work rate. In part, this may be attributable to increasing the convective heat transfer coefficient during exercise, which causes the skin temperature to decrease. Values specified by Saltin et al. [58] for the heat transfer coefficients were used for the simulations.

While it seems reasonable that increased convective heat transfer should occur during exercise, the magnitude of the increase on different sections of the body is open to question. Skin temperature is influenced also by skin blood flow, conduction of heat to the surface, and evaporation of sweat. The disparity between computed and measured skin temperatures can be attributed to a combination of these factors, but the exact origin of the problem has not been established.

As mentioned earlier, the responses of subject BS were not represented as well as those of other subjects. For example, during exercise at 75% of $\dot{V}_{O_{2,max}}$ (1350 watts) in 20°C air, the model predicted that the arterial temperature would increase to 39.6°C, which was considerably greater than the 38.4°C temperature actually attained by the esophageal temperature. There was a similar problem with the 30°C data. Since the metabolic rates for this subject were generally higher than for others, he was presumably quite fit. That fitness is a factor in thermal response is supported by many observations showing that intersubject variability is reduced when responses are correlated against the ratio, $\dot{V}_{O_2}/\dot{V}_{O_{2,max}}$, instead of against $\dot{V}_{O_2}$ alone. An unambiguous physiological basis for the effect of fitness on thermal response has not been established, and it has not been included explicitly in the model, but this is probably something that deserves additional attention.

Agreement between corresponding computed and measured values was quite good for the four cases studied by Libert, et al. The greatest disparity seems to be in rate of evaporation, with computed values tending to be slightly lower than corresponding measured values. However, the difference is not large enough to be of great concern.

The capacity of this model for predicting human thermal responses to cold was also evaluated. Agreement between computed and measured temperatures was generally quite good for three of the four subjects studied by Webb and Kuehn. However, Subject JR consistently responded in a manner rather different from the others, and his case will be discussed separately. For the three subjects, PW, JA, and ST, changes in computed rectal and arterial temperatures are generally

within $\pm 0.2^{\circ}\text{C}$ of measured rectal and tympanic temperatures. The bias that can occur in tympanic temperature is clearly apparent for several of these cases, where initial values below 36°C were measured. However, even in those cases, both computed temperatures tend to parallel the measured responses.

Agreement between computed and measured metabolic rates is not as good as between corresponding temperatures. Computed values seem to be consistently larger than the measured values. The greatest discrepancy usually occurs during the interval from 10 to 40 minutes, and seems to result from premature onset of shivering. During that interval, the discrepancy can be a very large fraction of the measured rate because the measured rate is still small. As the measured rate increases, the discrepancy decreases, and is usually about 25 percent at the end of the 60 minute test period.

Comparable discrepancies occur in the rate of heat transfer from the subject to the bath. Typically differences between computed and measured values are of order 50 watts for both metabolic rate and surface heat transfer rate. Since the surface heat transfer rates are much larger than the metabolic rates, the fractional error is smaller in the former case.

For the four subjects studied by Webb and Kuehn, it appears that the computed shivering and vasomotor responses to cold stress may occur a bit too rapidly. Computed increases in metabolic rate generally lead the measured values by 10 to 30 minutes, and computed heat transfer rates tend to decrease more rapidly than the measured rates. This could be due to weighting skin temperatures too heavily in the control equations, but additional experimental studies will be required before this question can be resolved.

The subject whose responses are not described well by this model is JR, who was very lean and apparently quite fit. Although not an athlete, he performed manual labor which contributed to developing good muscular structure.

This subject does not cool nearly as rapidly as the model predicts he should. Since the reported mean skinfold thickness for JR is only 6 mm, which is approximately a tenth percentile value, 7 mm was used in the model to see whether the anomalous responses

could be attributed to an error in skinfold measurement. The results shown in the graphs were computed using 7 mm for the mean skinfold thickness, and it is clear that the problem persists. This person appears to have the ability to limit heat loss to the water bath, even though he does not have a thick layer of subcutaneous fat. Presumably he does so by limiting blood flow to the extremities more effectively than the average person, which implies that his microcirculation supplies oxygen and removes metabolic products very efficiently. This appears to be consistent with recently reported observations that physically fit individuals tolerate immersion in cold water more readily than less fit subjects [55]. It has also been suggested that body type influences thermal responses, but a systematic study has not been conducted to define the relationship. Resolution of these questions will require additional experimental studies designed to demonstrate the effect of physical fitness and body type on response to cold.

Keatinge's study was similar to the Webb-Kuehn study except that his subjects were immersed for a longer time in warmer water--four hours in 29°C water--and heat transfer rates were not measured. Agreement between computed and measured temperatures was generally quite satisfactory, although there was a notable exception. The tympanic temperature of Subject 2 decreased from 37.2°C to 36.5°C, while the computed arterial temperature remained at 37.2°C. Since this subject was rather fat (mean skinfold thickness = 21.2 mm), such a large decrease in deep body temperature is not to be expected. Indeed, the final tympanic temperature for Subject 2 is very close to that of Subject 1 whose mean skinfold thickness is only 10.8 mm. Either the response of Subject 2 was anomalous or that particular measurement was in error, but it is impossible to choose between the two alternatives.

There is also some lack of agreement between computed and measured metabolic rates, especially for Subject 1 whose measured value was over 200 watts while the computed value remained around 130 watts. For all three subjects, computed values are lower than the measured values, which is just the opposite of the tendency noted in the Webb-Kuehn study. There is no obvious change in the

model that will simultaneously improve agreement between computed and measured metabolic rates for both sets of data.

HUMAN THERMAL MODEL: AEROSPACE APPLICATIONS³

As consultants on thermal stress and other aerospace environmental problems, our group receives inquiries on a variety of matters which could benefit from high-fidelity thermal modeling. Some of these problems are of interest not only to the USAF but also to other government agencies, and there is considerable interest in sharing knowledge and developing standard policies on an international basis. This paper will outline briefly our current areas of concern and point out where further basic research seems to be needed.

Requests for information on thermal stress range in importance from the relatively minor to the definitely major. We might be asked whether a given job involves sufficient heat exposure to justify special pay for a handful of civilian personnel. At the other end of the spectrum, a decision regarding thermal stress during military training might affect USAF procedures and equipment distribution worldwide.

Aerospace applications of the model can be divided into four categories, each involving different assumptions and trade-off analyses:

1) Routing training procedures. The theme here is "safety first," and one must allow a safety margin which protects the more susceptible individuals in a population exposed to stress. A classic example is the conservative use of WBGT to curtail hot-weather training of military recruits.

2) Combat scenario development. In this case commanders need realistic physiological input to their analysis of group performance under the severe demands of wartime. For USAF the key is "sortie generation," the ability to loft as many aircraft as possible in a given time. Consideration must be given both to the percentage of personnel who might become thermal casualties (normal distribution),

³This section was written by Dr. S. A. Nunneley.

and to the protection of certain individuals whose skills are critical to the task at hand.

3) Emergency protective measures. This covers decisions to alter procedures or provide equipment solely because of possible emergencies. An example is the regulation which mandates wear of anti-exposure suits aboard aircraft overflying water of a certain temperature, regardless of the fact that the suit can be a liability during normal flight.

4) Post-accident rescue and investigation. The issue here is usually whether survival is possible under severe conditions such as desert exposure or immersion in cold water. Whereas categories 1-3 involve consideration of either the most susceptible or the average individual, in this case interest focuses on the most resistant because the question is one of maximum time for rescue procedures.

To date most of these queries have been answered by "modeling" in the consultant's head, using knowledge of the literature and best-guess extrapolation to untested conditions. Good computerized thermal models should improve the validity of results. There are several types of application: (1) development of charts and other tools for use by non-specialist personnel in the field, (2) exploration of the effects of variation in factors such as clothing, work load, and personal thermal conditioning, (3) identification of critical areas where human experimentation is needed, (4) extrapolation to conditions which preclude human experimentation, and (5) on-line simulation of conditions during search-and-rescue efforts.

Table 4 shows the relative levels of USAF interest for various types of thermal stress. On the hot side, greatest concern focuses on problems of groundcrew members who must perform hard physical labor while wearing heavy protective clothing in hot climates. Modeling should be especially useful in the development of rational work-rest cycles, which will require an improved understanding of heat-stress recovery under various conditions. Heat stress is also a problem in the cockpit due to limited air conditioning, a great radiant load and required heavy clothing. While the situation is somewhat improved by the low metabolic rate typical of aircrew members in flight, there is great concern over the effects of even

moderate heat stress on performance of critical tasks. Artificial microclimate cooling is being considered for both aircrew and ground-crew applications, and modeling should contribute to the sizing of such systems.

Regarding cold stress, the greatest interest centers on the problem of immersion hypothermia following loss of an aircraft. Modeling should assist with selection of clothing ensembles which are acceptable in flight (minimal bulk and insulation) but which will provide the necessary protection in an emergency. Special attention needs to be paid to the designation of critical rates of cooling or hypothermia, the problem of shiver suppression or fatigue, and individual differences in cooling associated with skinfold thickness.

In summary, modeling should be useful for aerospace problems involving both heat and cold, for high and low work loads, and in cockpit, ground and water environments.

Table 4. USAF levels of interest in thermal stress.

	Heat -----	Cold -----
Cockpit	2	3
Ground Operations	1	3
Ground Emergency	3	3
Water Emergency	-	2

1=very high, 2=high, 3=moderate

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number) A workshop was held at The University of Texas at Austine in December 1982. The workshop evaluated available mathematical models which could be used to simulate human thermal behavior under various conditions. The program involved the following four activities: (1) obtain copies of the mathematical models, install them on computers located at The University of Texas, and verify that they were operating correctly, (2) collect sets of data suitable for testing mathematical models and enter them into a machine readable data base, (3) use the models to simulate the conditions represented by the experimental data, (4) discuss the		

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the simulated results with the authors of the models and a group of outstanding thermal physiologists who offer constructive criticism and suggestions for improving the models.

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AN EVALUATION OF HUMAN THERMAL MODELS

BY

EUGENE H. WISSLER

A report based on a
workshop held at

THE UNIVERSITY OF TEXAS AT AUSTIN

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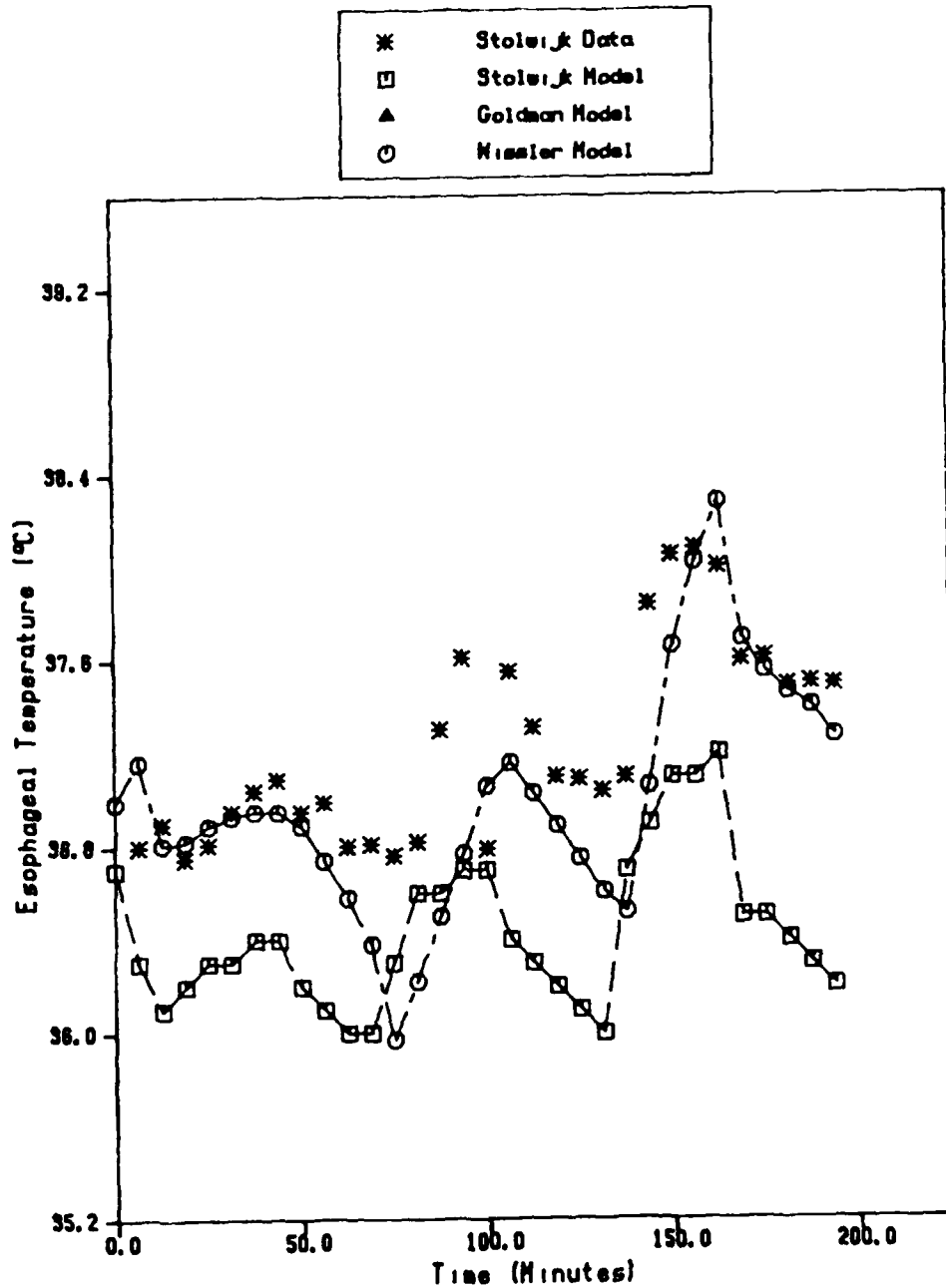
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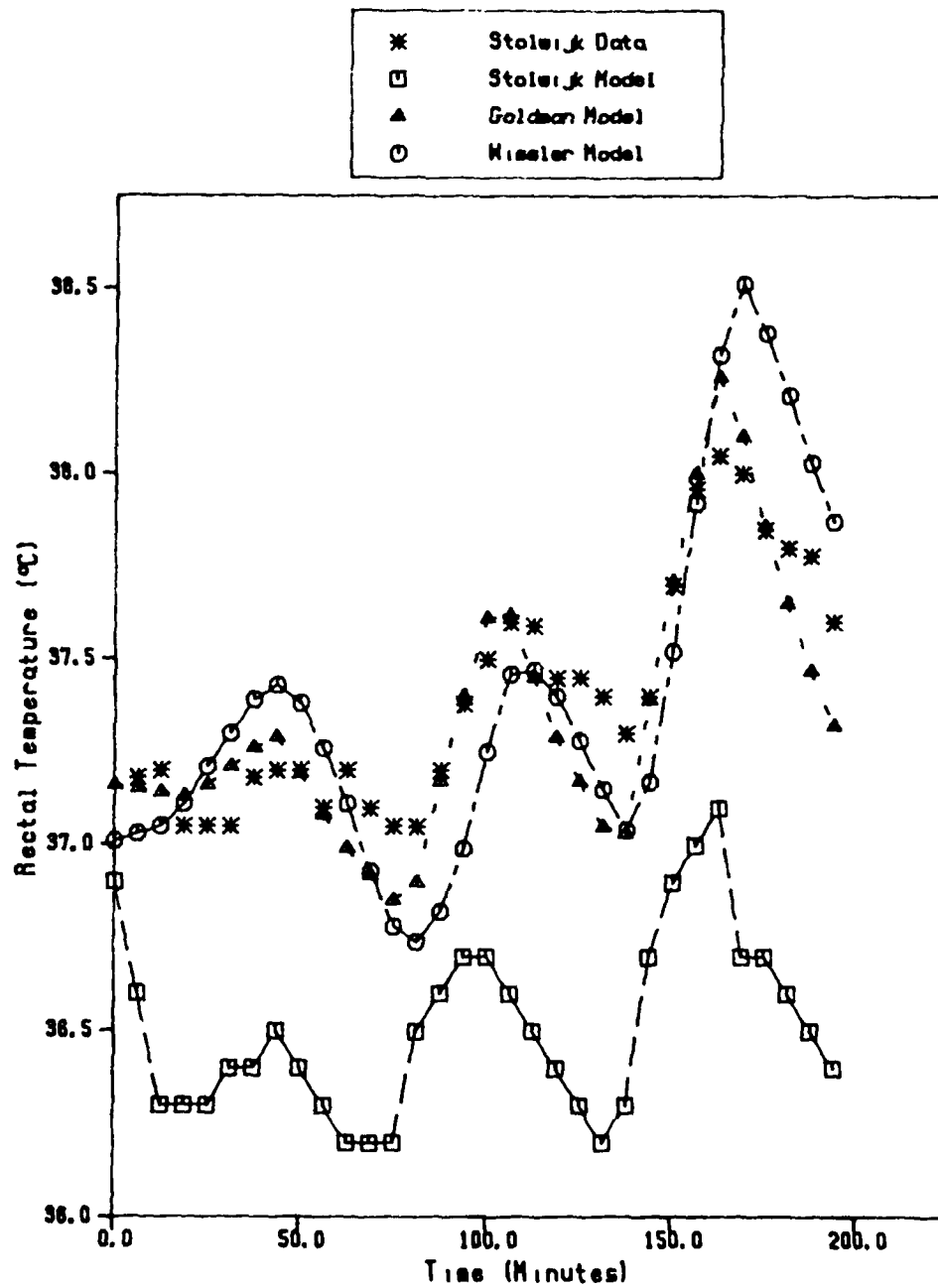
Table 5. Arrangement of Graphs Comparing
Computed Values with Experimental Data

<u>Investigator</u>	<u>Subject</u>	<u>Environment</u>	<u>Pages</u>
Saltin, et al.	PM	10°C Air	63 - 67
"	"	20°C "	68 - 72
"	"	30°C "	73 - 77
"	BC	10°C "	78 - 82
"	"	30°C "	83 - 87
"	BS	20°C "	88 - 92
"	"	30°C "	93 - 97
Libert, et al.	KG	38°C Air	98 - 101
"	"	42°C "	102 - 105
"	LA	38°C "	106 - 109
"	OM	42°C "	110 - 113
Webb & Kuehn	JA	18°C Water	114 - 117
"	"	" "	118 - 121
"	"	24°C "	122 - 125
"	"	" "	126 - 129
"	JR	18°C "	130 - 133
"	"	" "	134 - 137
"	"	24°C "	138 - 141
"	"	" "	142 - 145
"	PW	18°C "	146 - 149
"	"	" "	150 - 153
"	"	24°C "	154 - 157
"	"	" "	158 - 161
"	ST	18°C "	162 - 165
"	"	" "	166 - 169
"	"	24°C "	170 - 173
"	"	" "	174 - 177
Keatinge	1	29°C Water	178 - 180
"	2	" "	181 - 183
"	3	" "	184 - 186

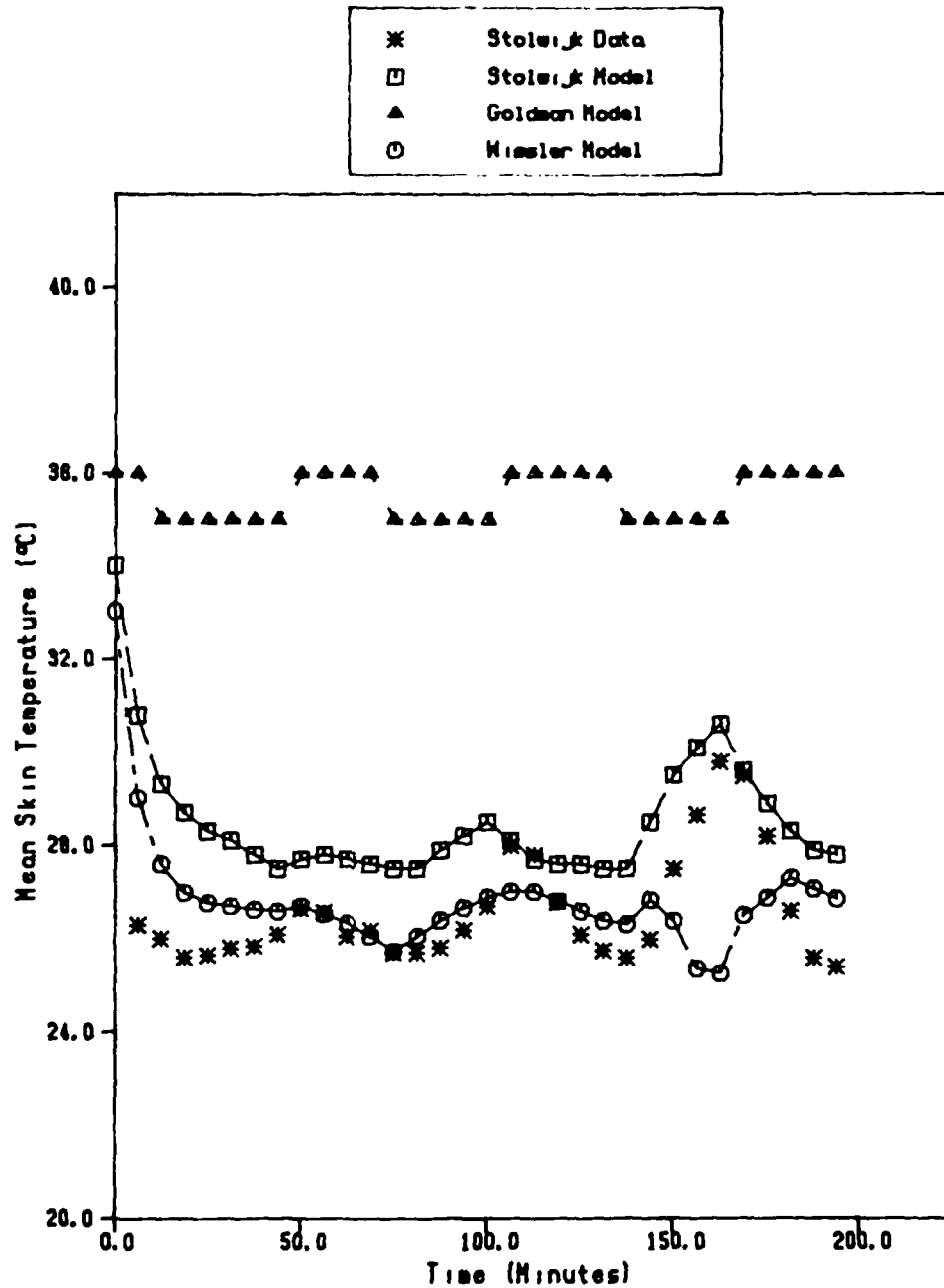
Esophageal Temperature
As a Function of Time
For Subject PM During Exercise Periods in 10 °C Air



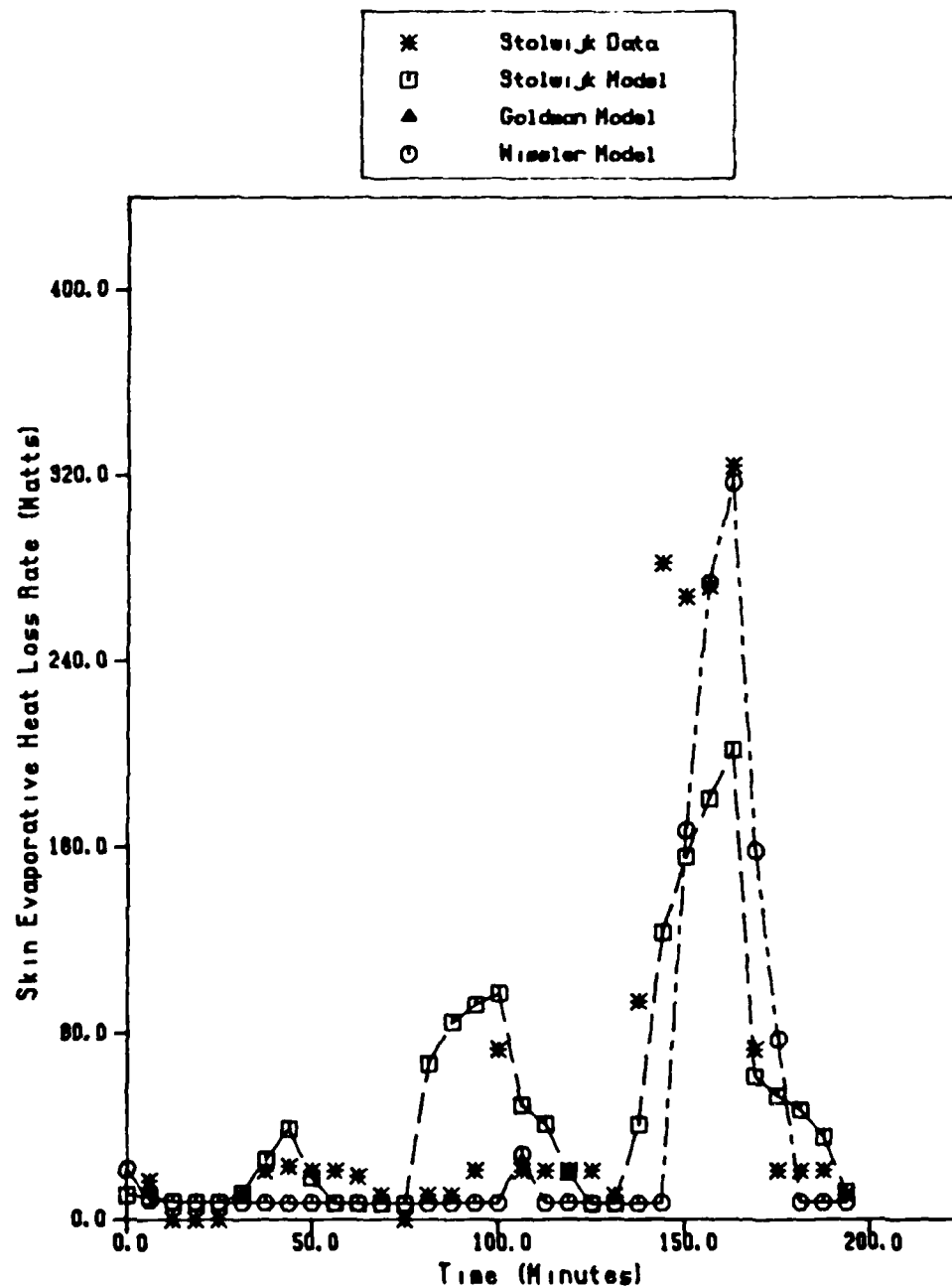
Rectal Temperature
As a Function of Time
For Subject PM During Exercise Periods in 10 °C Air



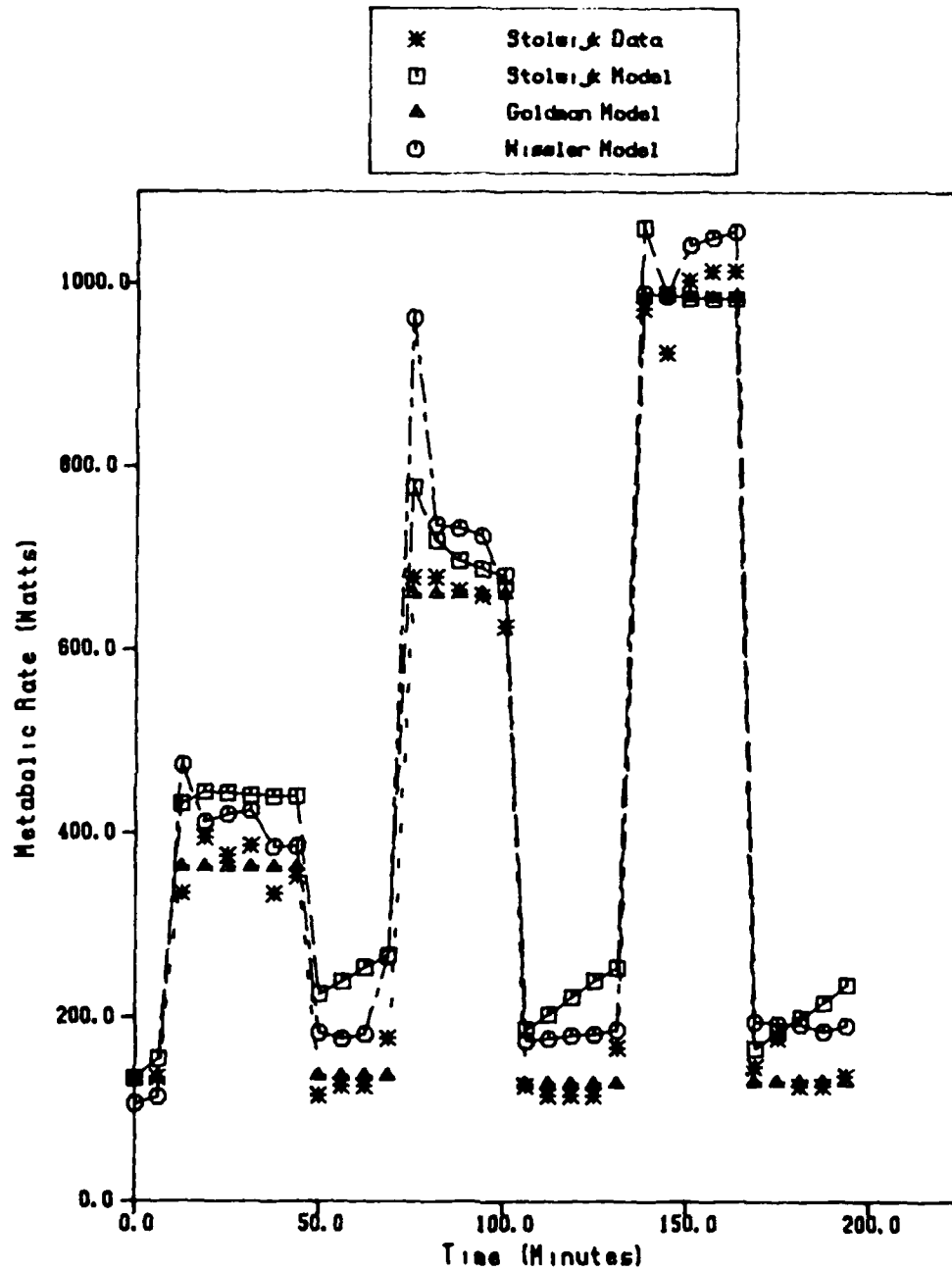
Mean Skin Temperature
As a Function of Time
For Subject PM During Exercise Periods in 10 °C Air



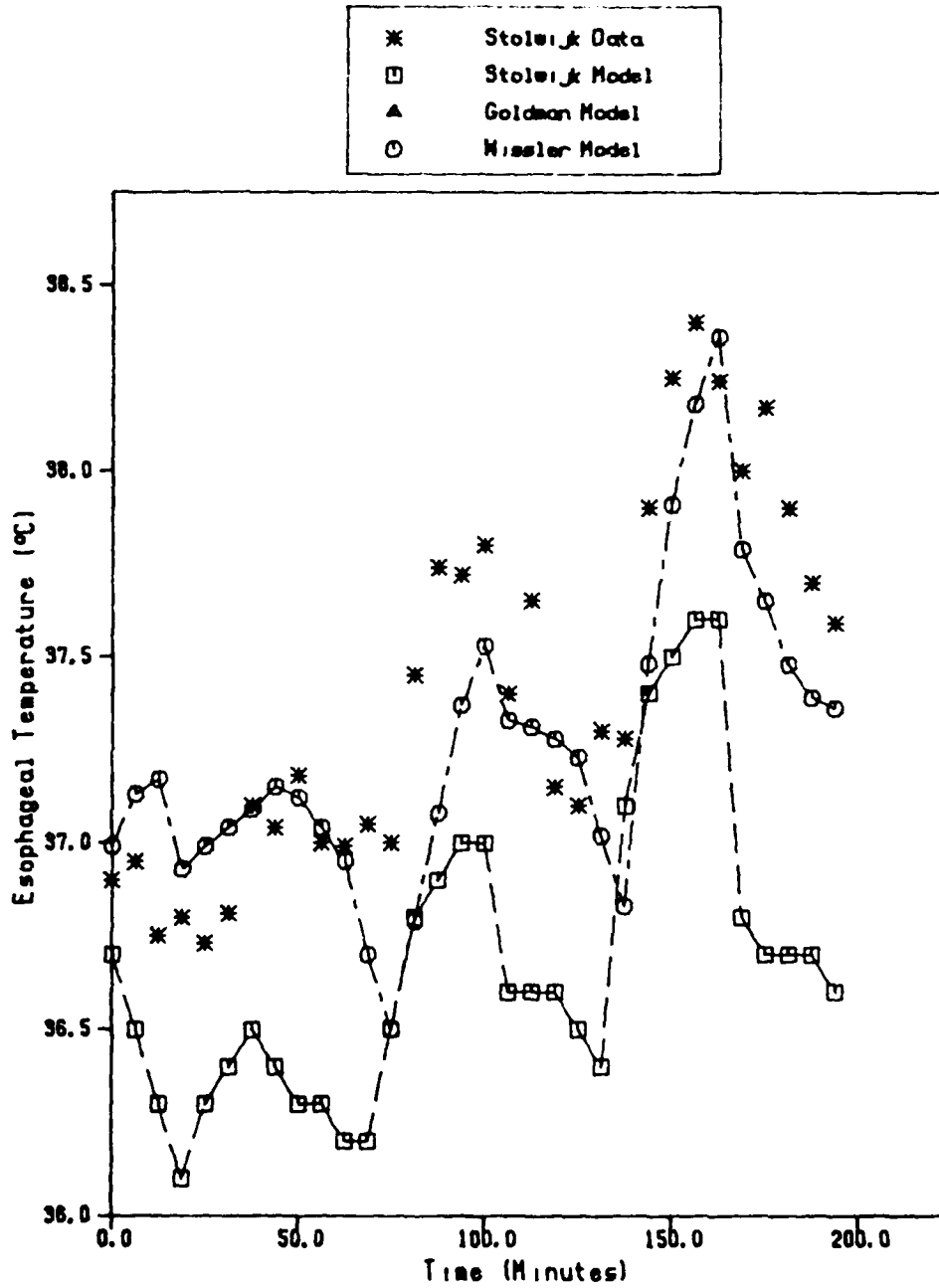
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject PM During Exercise Periods in 10 °C Air



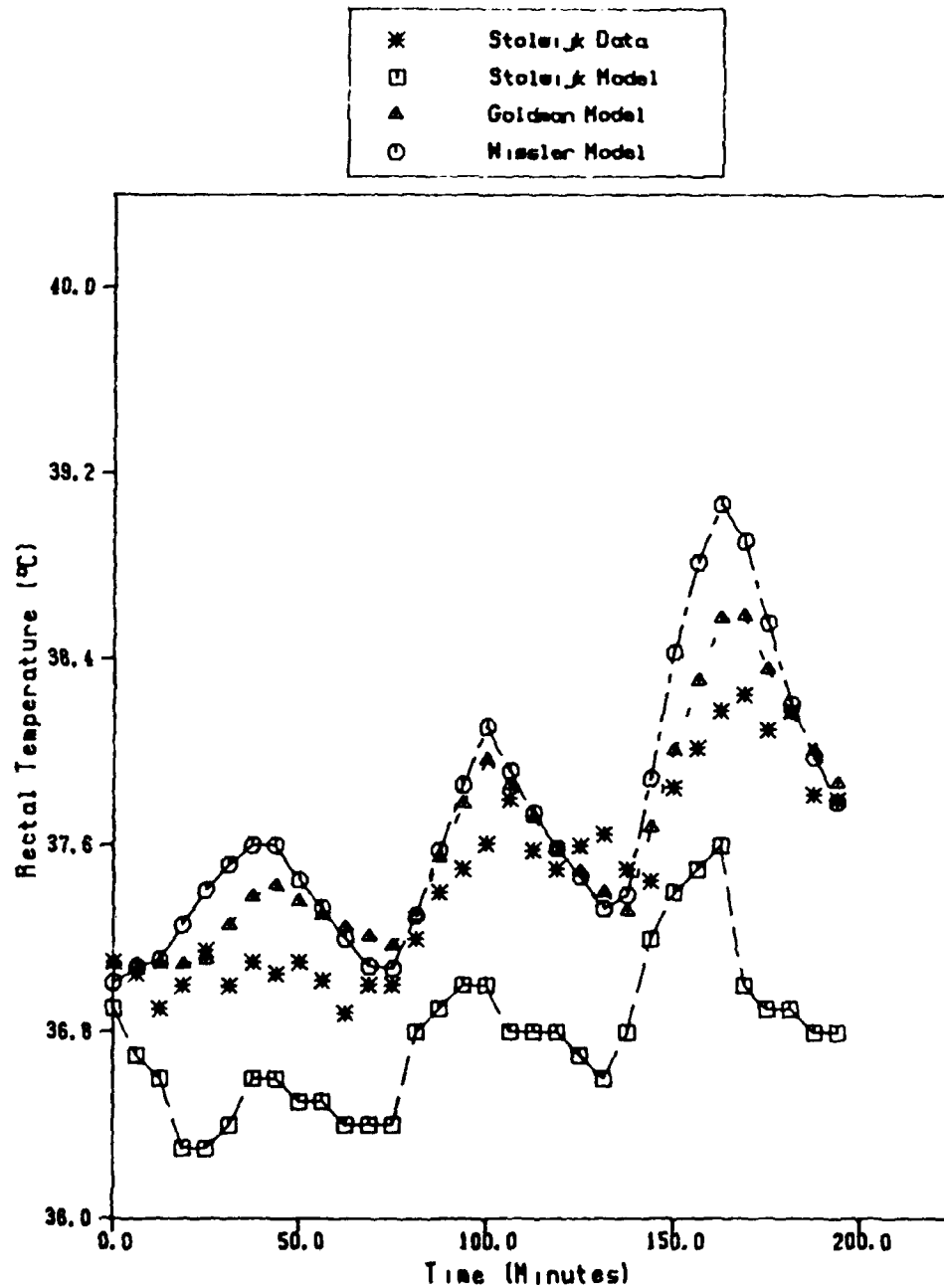
Metabolic Rate
As a Function of Time
For Subject PM During Exercise Periods in 10 °C Air



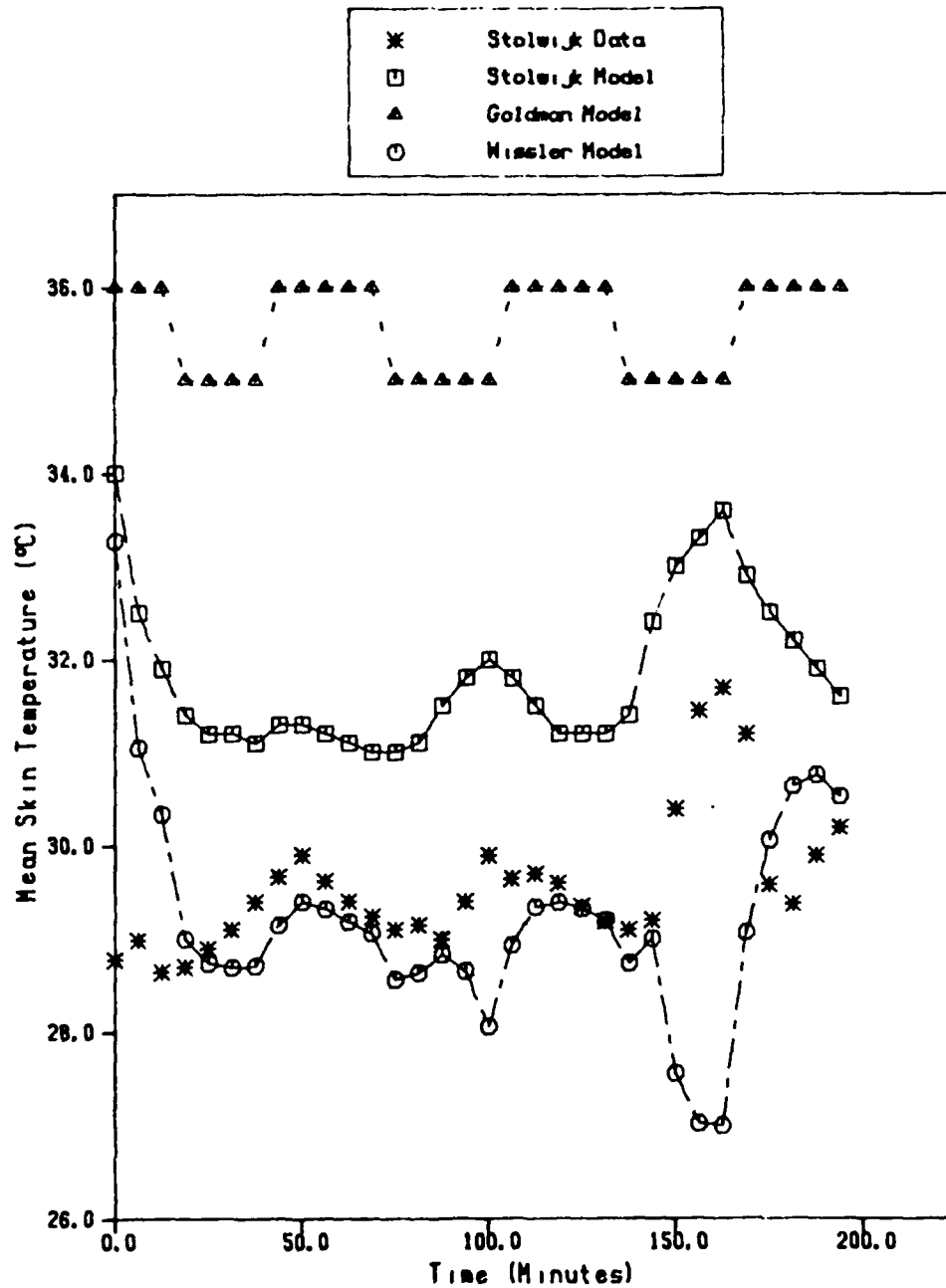
Esophageal Temperature
As a Function of Time
For Subject PM During Exercise Periods in 20 °C Air



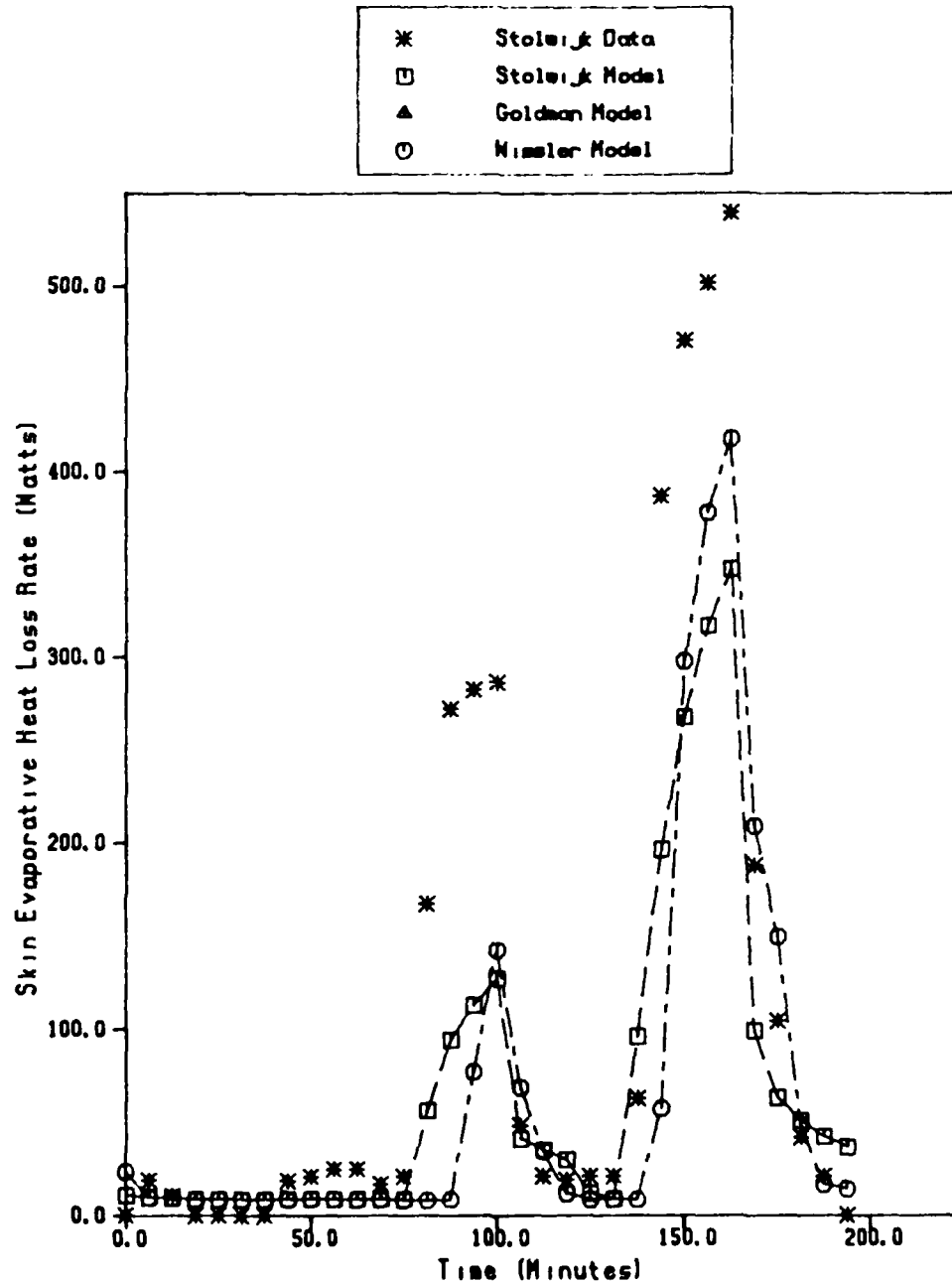
Rectal Temperature
As a Function of Time
For Subject PM During Exercise Periods in 20 °C Air



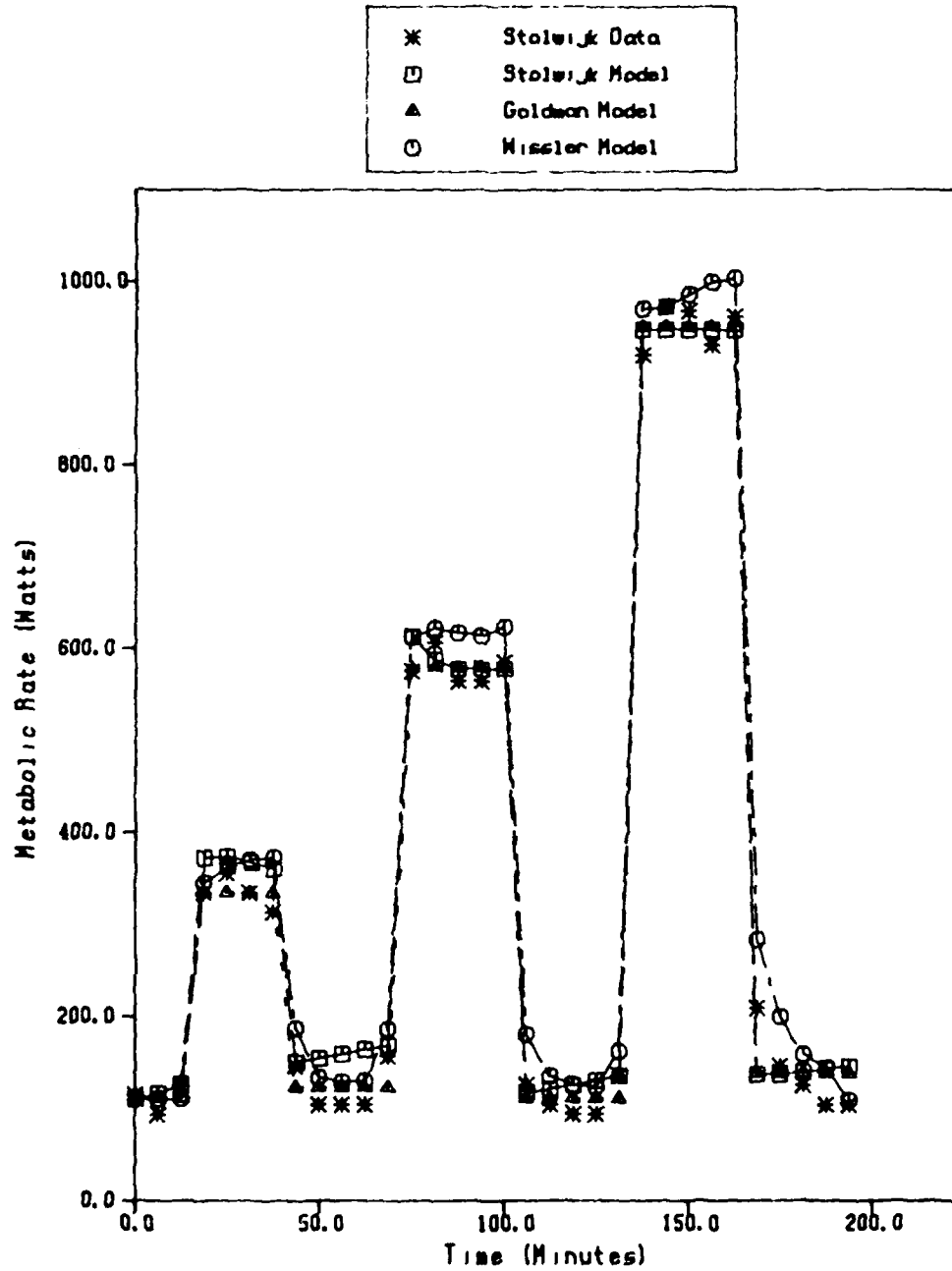
Mean Skin Temperature
As a Function of Time
For Subject PM During Exercise Periods in 20 °C Air



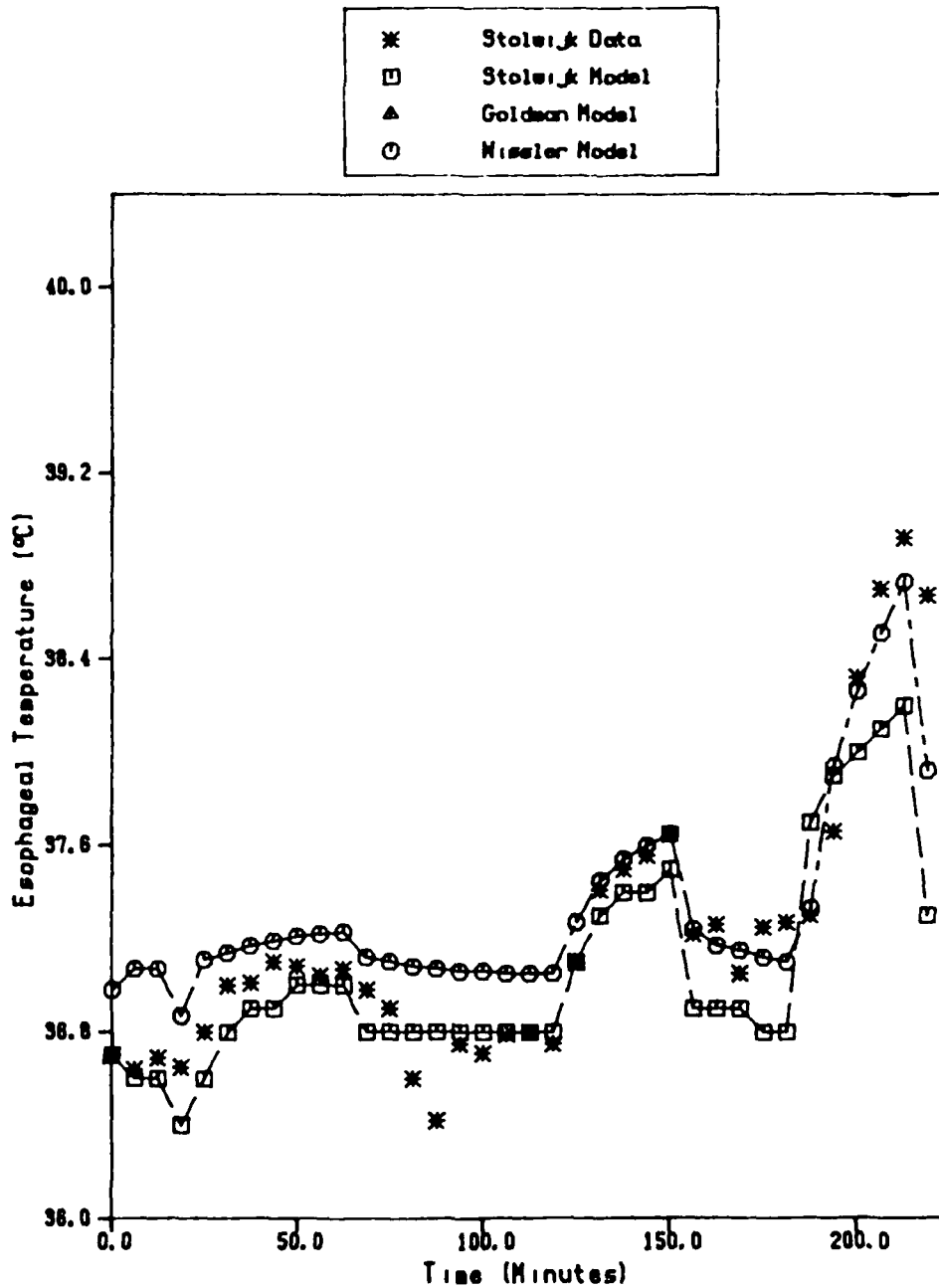
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject PM During Exercise Periods in 20 °C Air



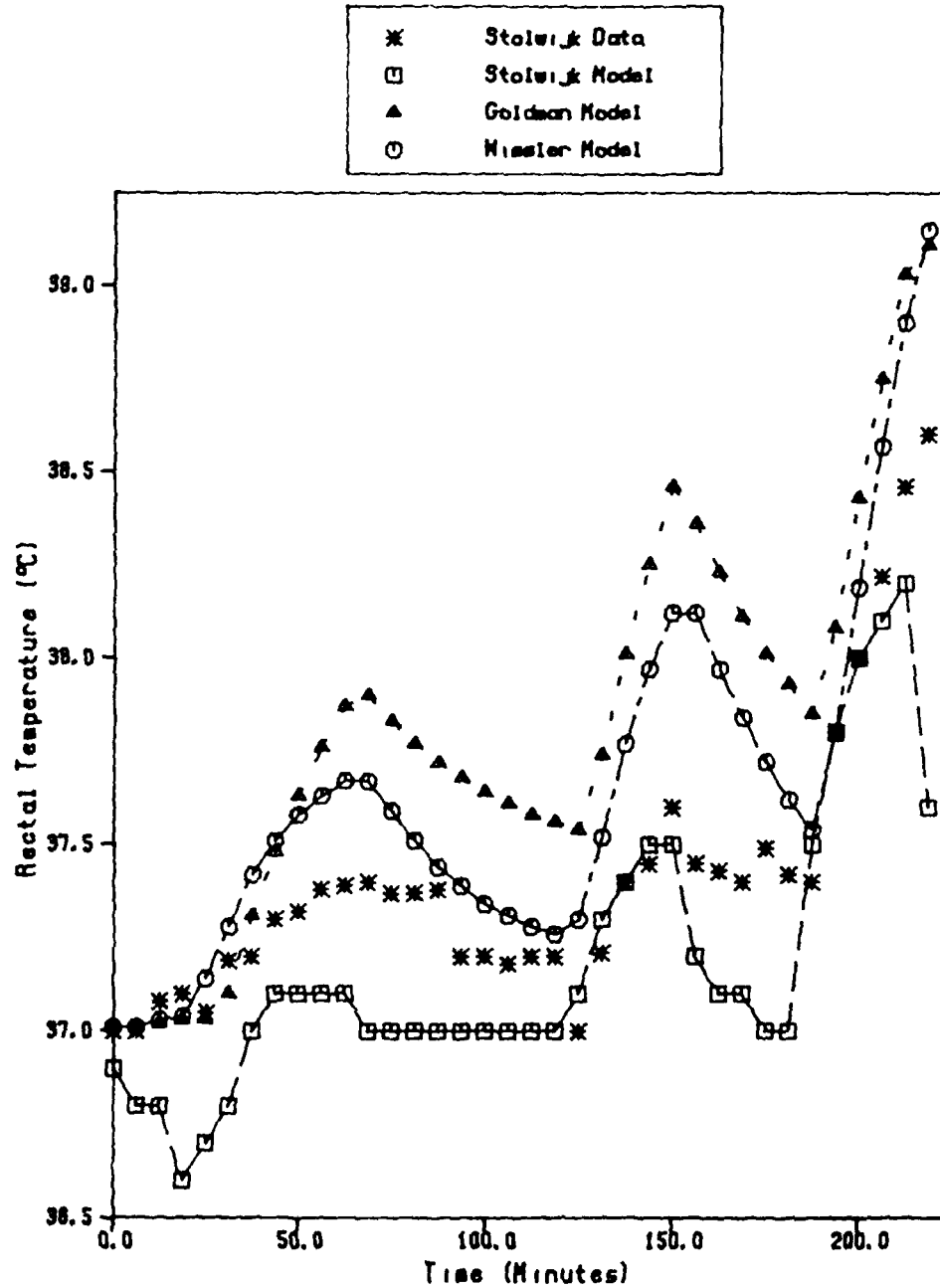
Metabolic Rate
As a Function of Time
For Subject PM During Exercise Periods in 20 °C Air



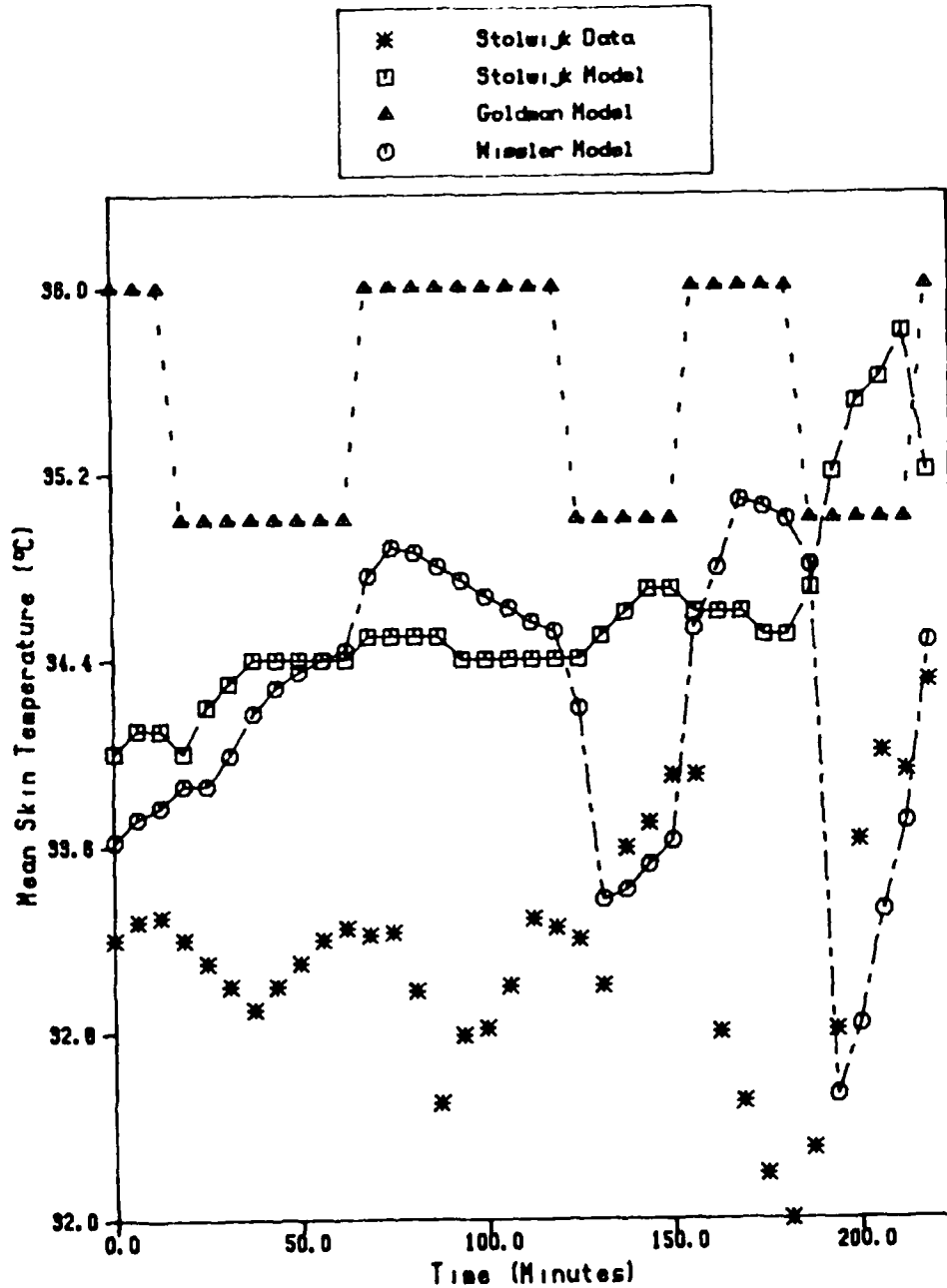
Esophageal Temperature
As a Function of Time
For Subject PM During Exercise Periods in 30 °C Air



Rectal Temperature
As a Function of Time
For Subject PM During Exercise Periods in 30 °C Air

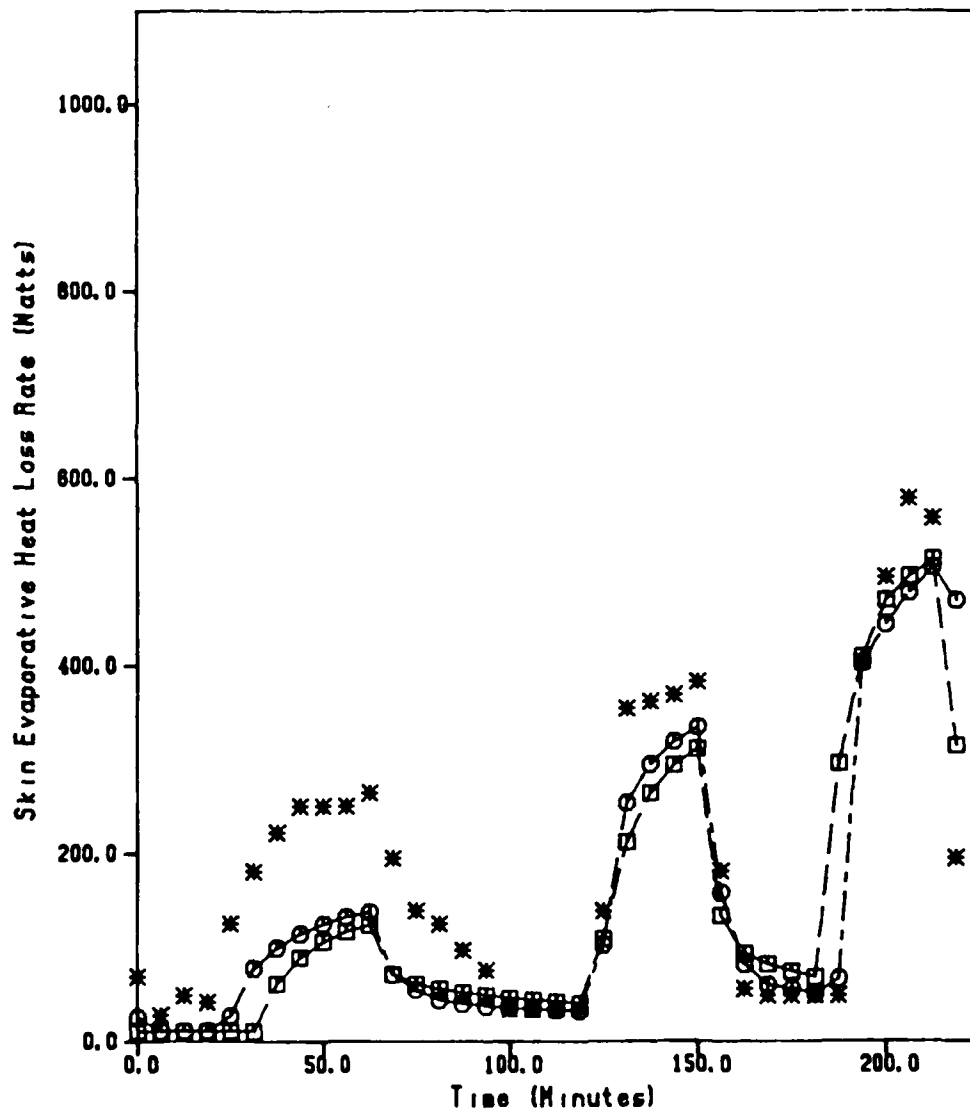


Mean Skin Temperature
As a Function of Time
For Subject PM During Exercise Periods in 30 °C Air

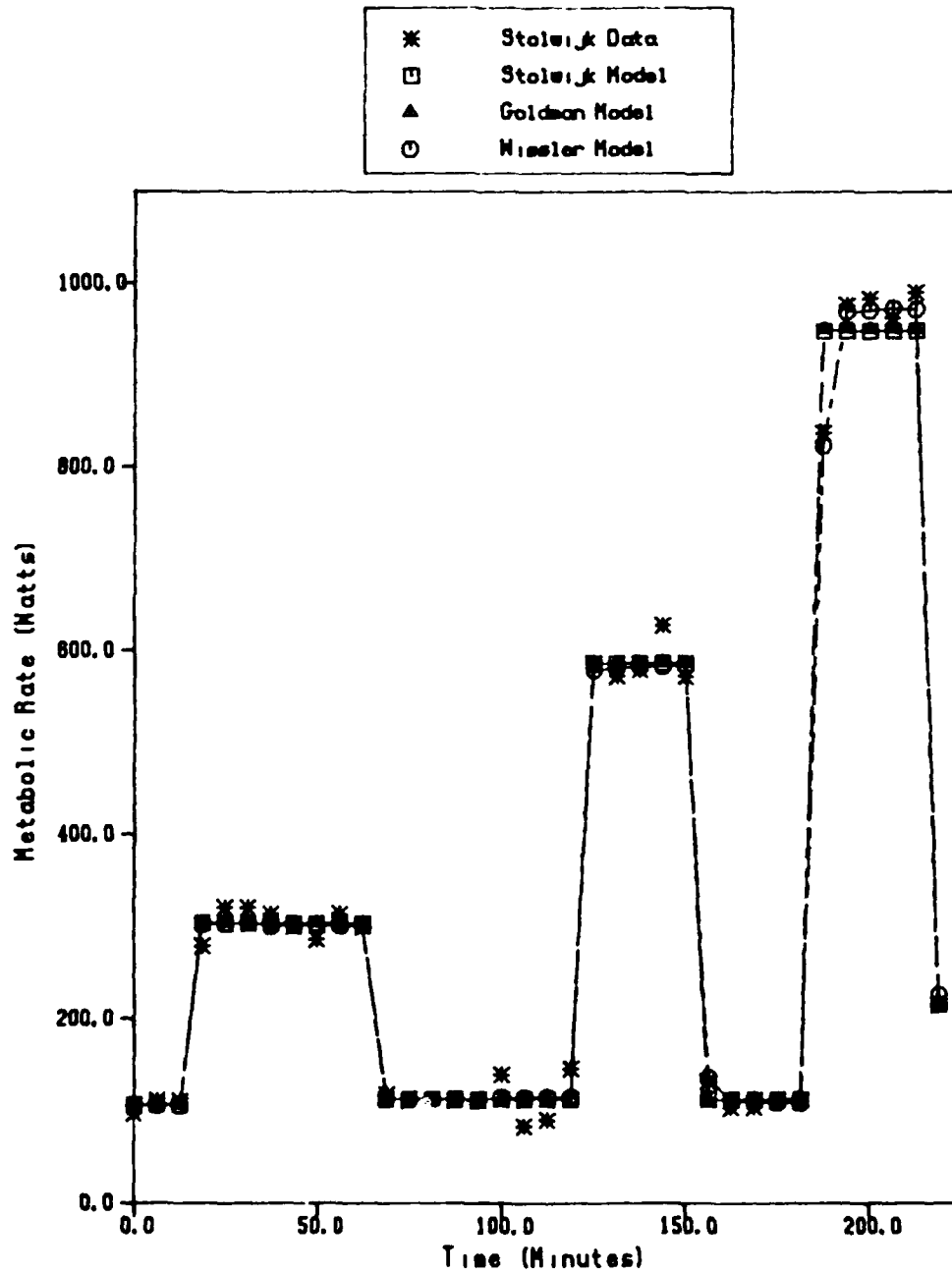


Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject PM During Exercise Periods in 30 °C Air

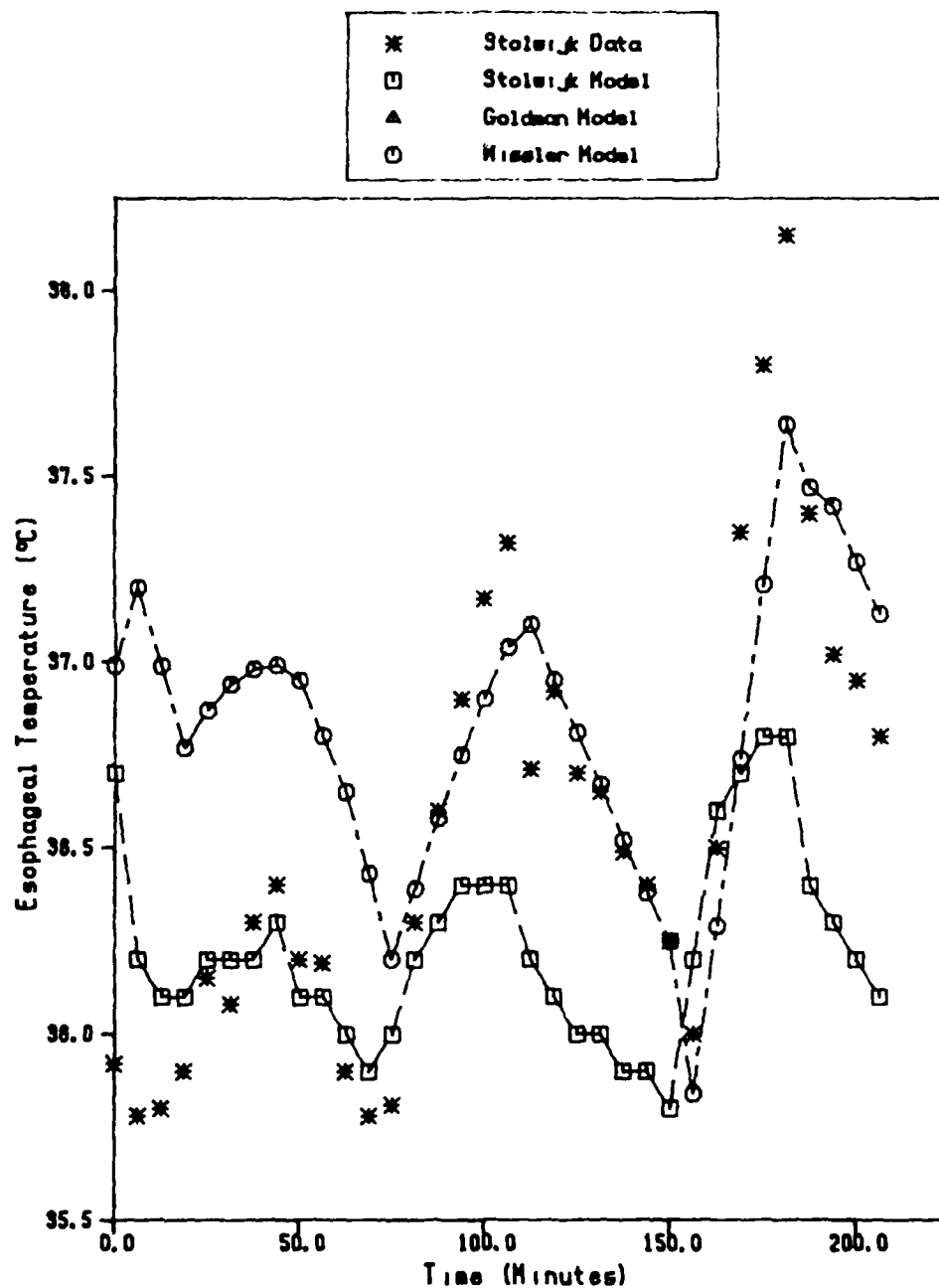
- | | |
|---|----------------|
| * | Stolwijk Data |
| □ | Stolwijk Model |
| ▲ | Goldman Model |
| ○ | Missler Model |



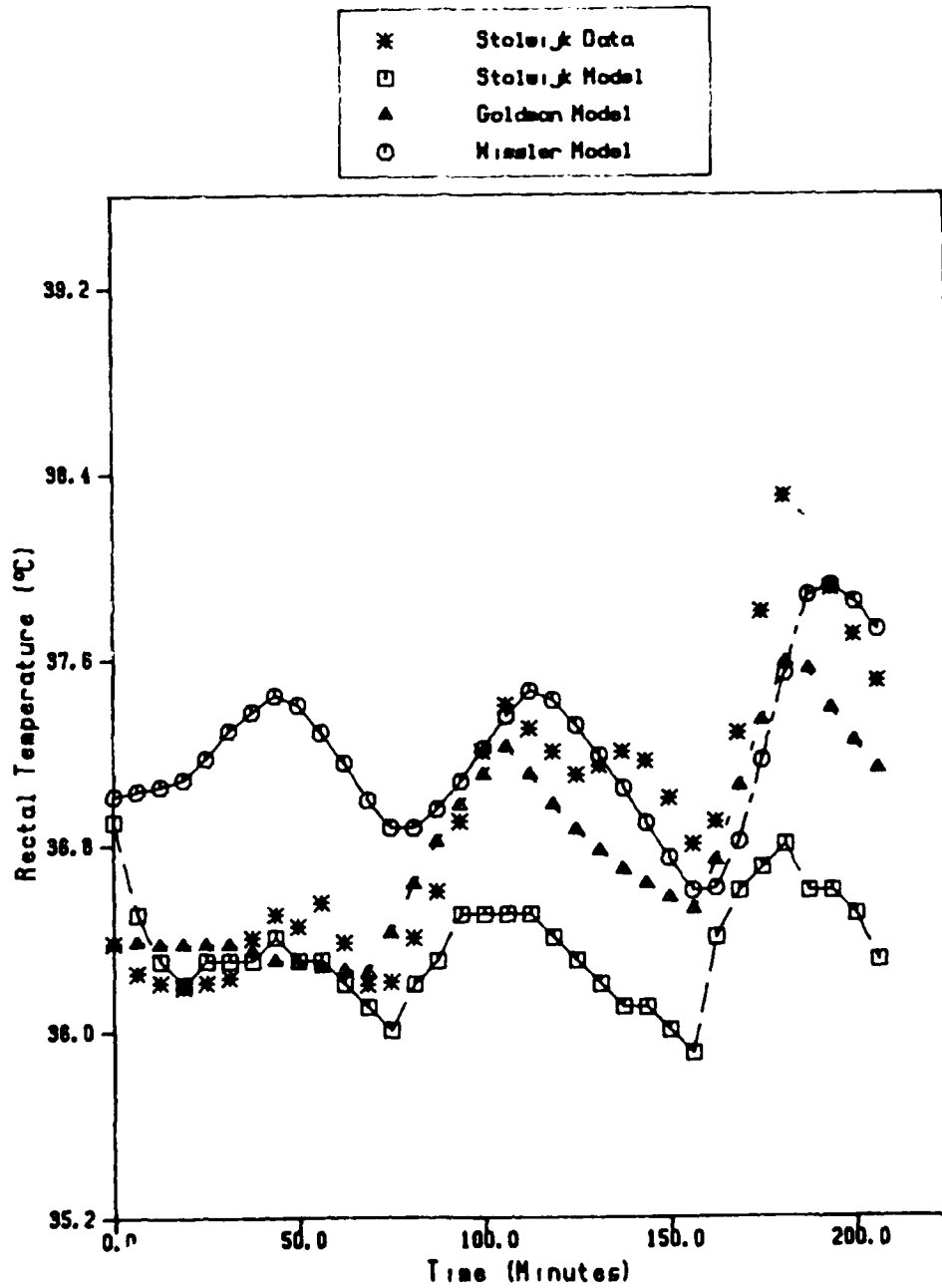
Metabolic Rate
As a Function of Time
For Subject PM During Exercise Periods in 30 °C Air



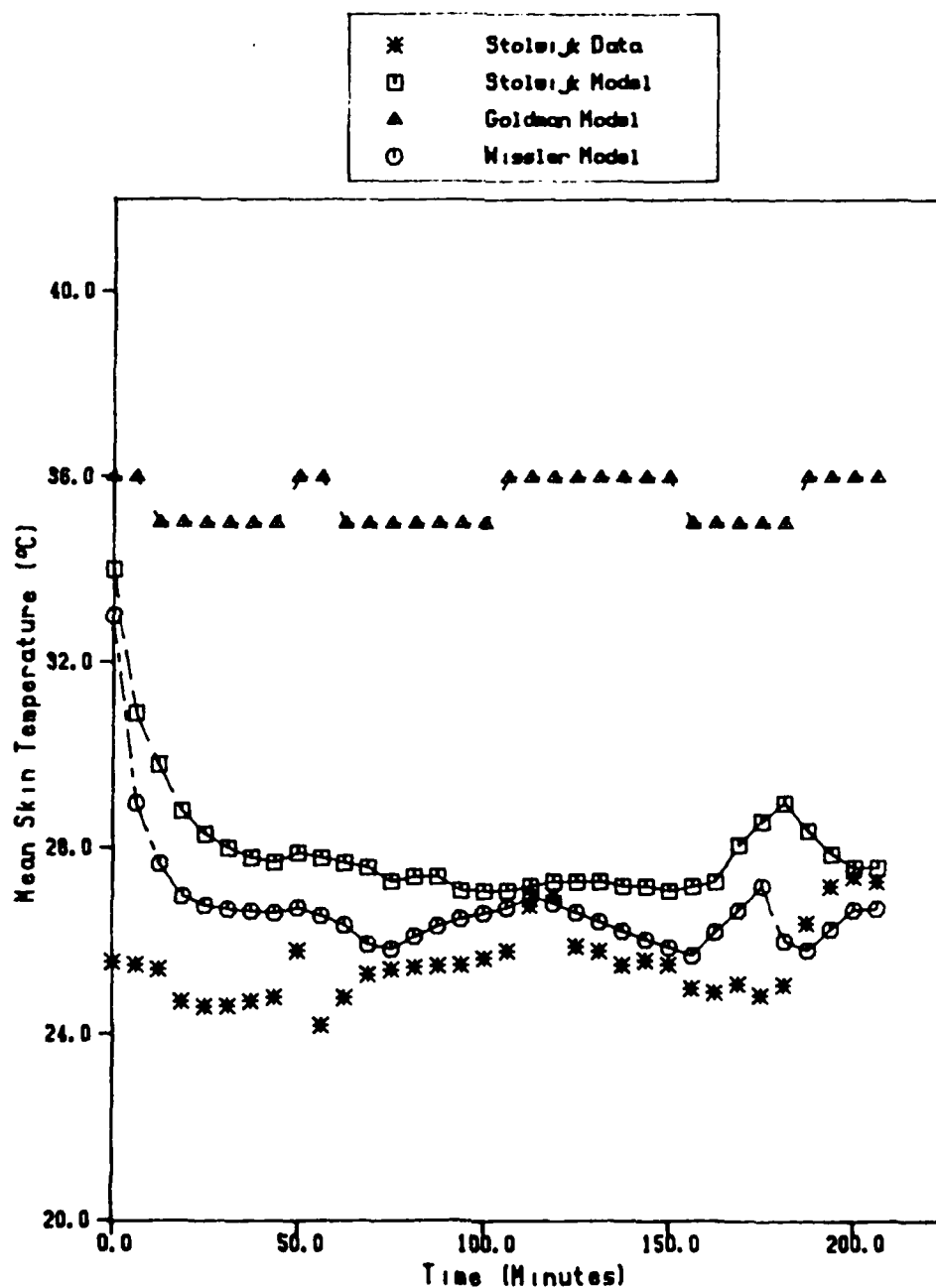
Esophageal Temperature
As a Function of Time
For Subject BC During Exercise Periods in 10 °C Air



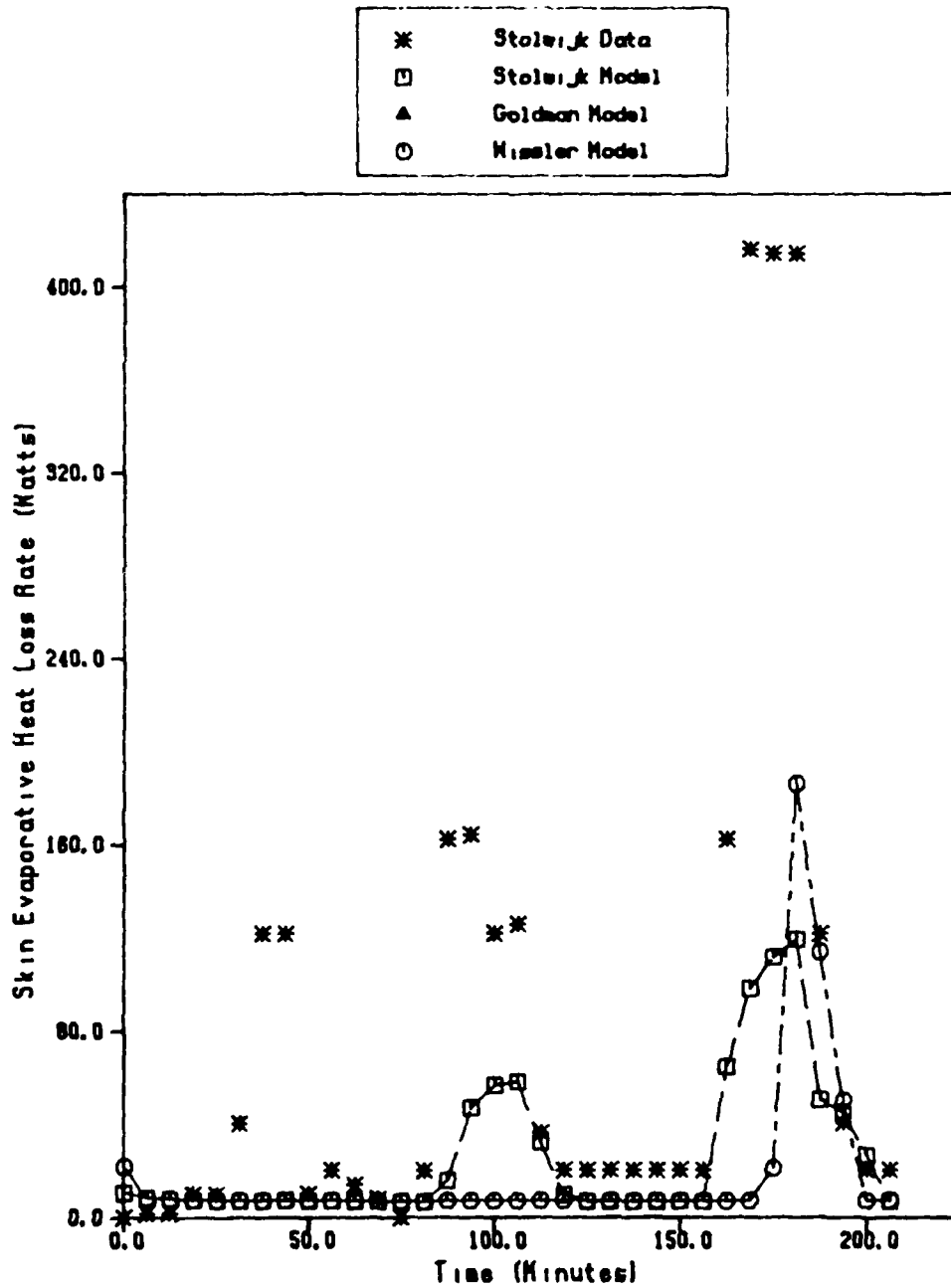
Rectal Temperature
As a Function of Time
For Subject BC During Exercise Periods in 10 °C Air



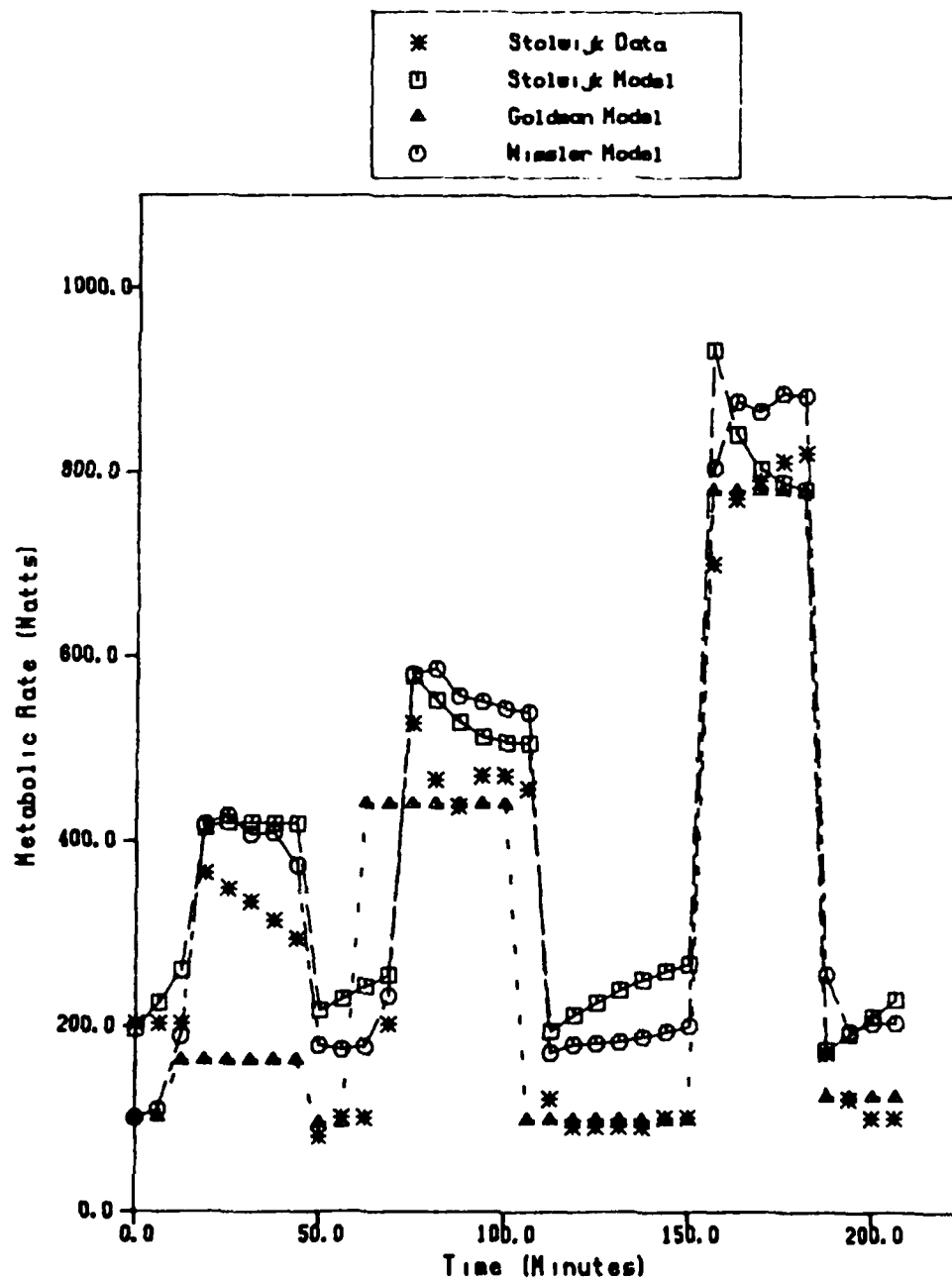
Mean Skin Temperature
As a Function of Time
For Subject BC During Exercise Periods in 10 °C Air



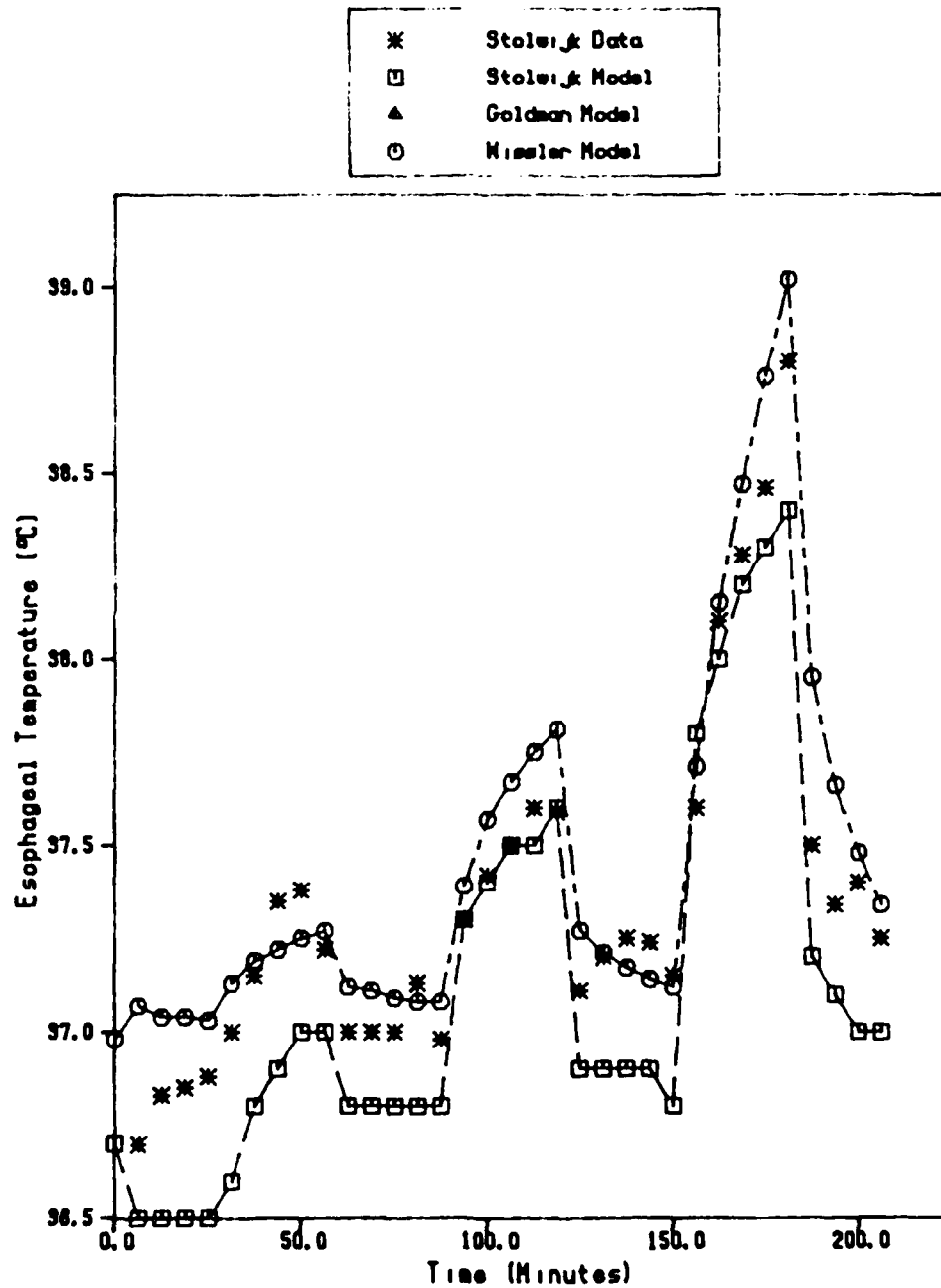
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject BC During Exercise Periods in 10 °C Air



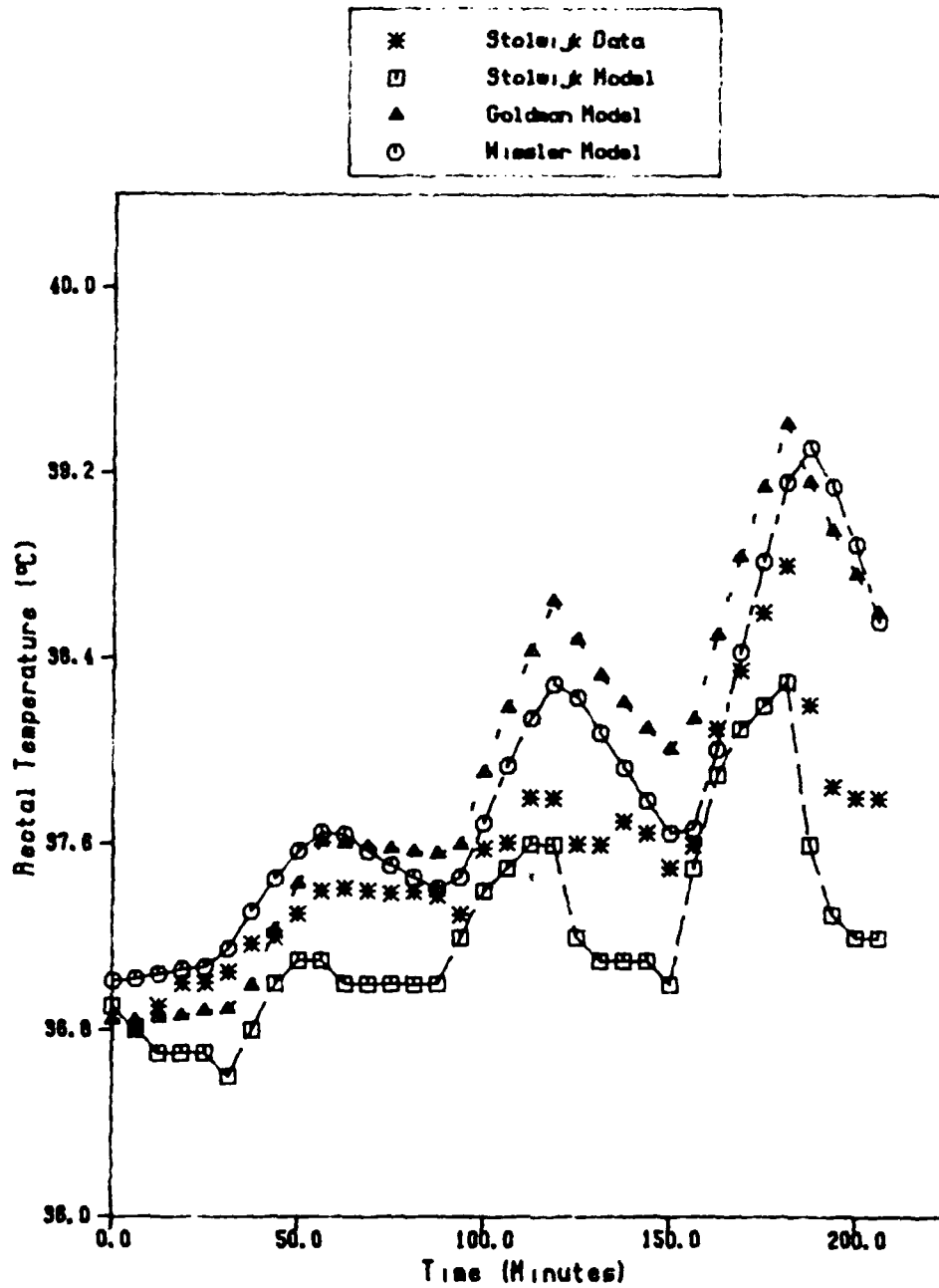
Metabolic Rate
As a Function of Time
For Subject BC During Exercise Periods in 10 °C Air



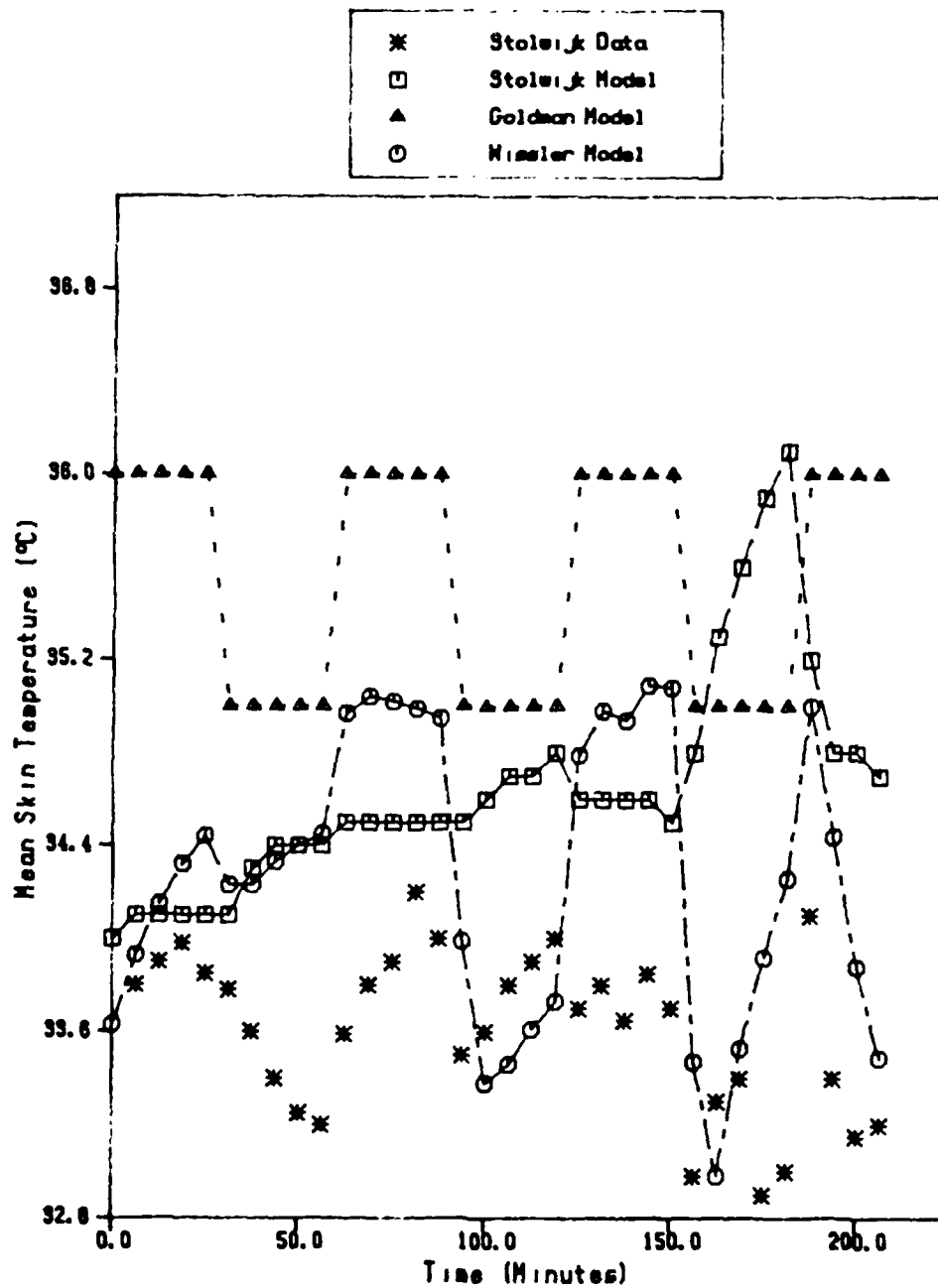
Esophageal Temperature
As a Function of Time
For Subject BC During Exercise Periods in 30 °C Air



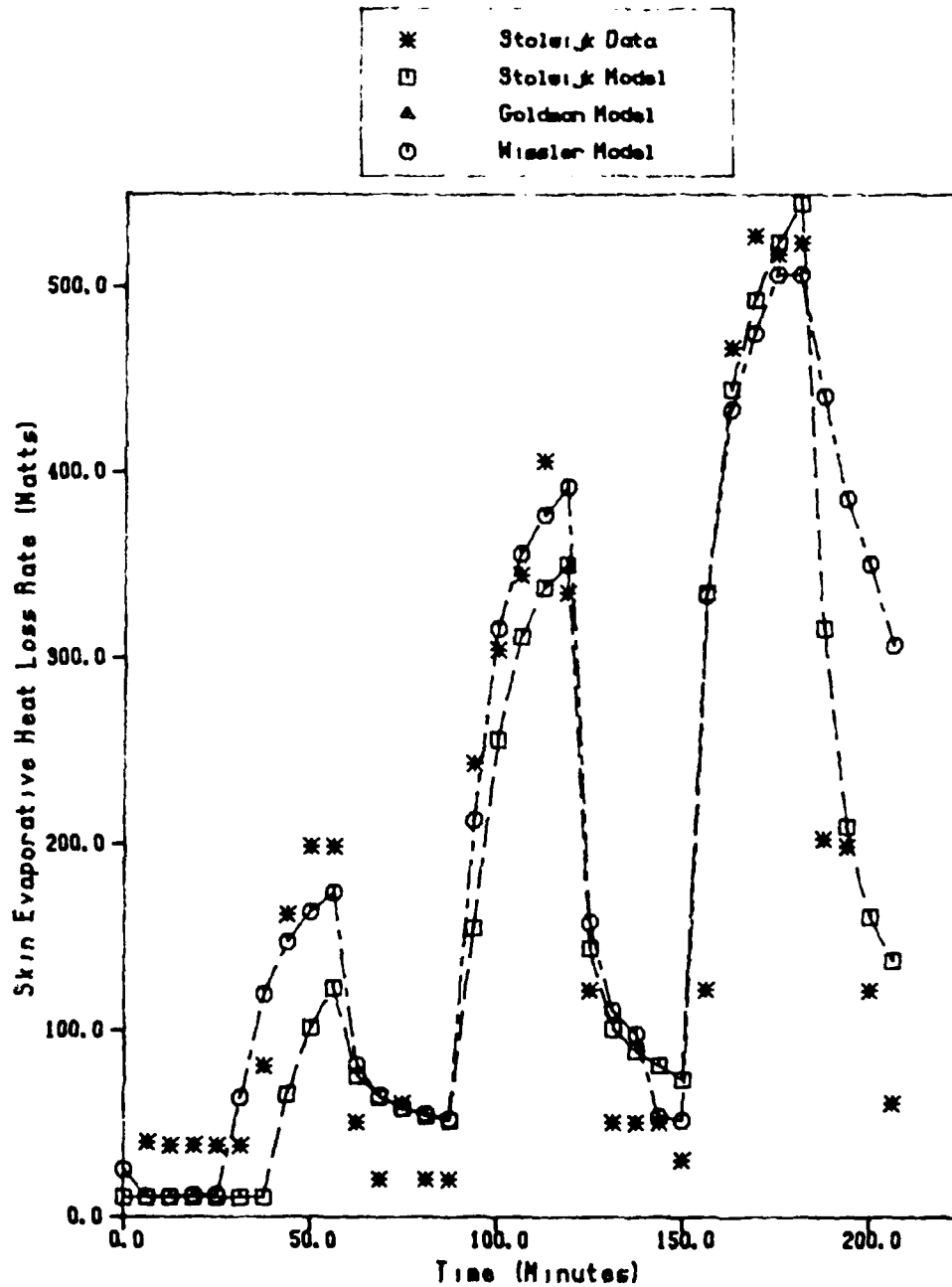
Rectal Temperature
As a Function of Time
For Subject BC During Exercise Periods in 30 °C Air



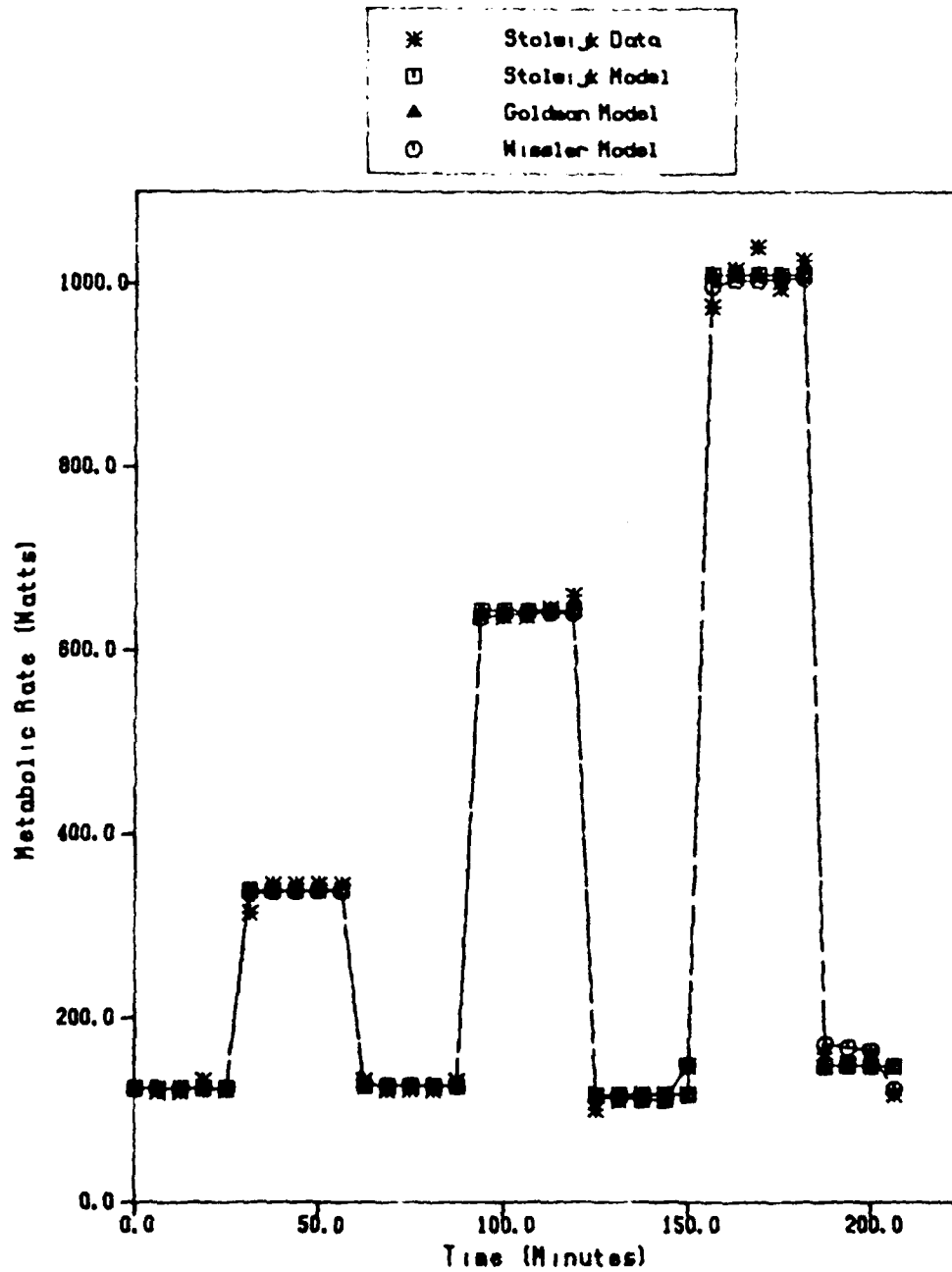
Mean Skin Temperature
As a Function of Time
For Subject BC During Exercise Periods in 30 °C Air



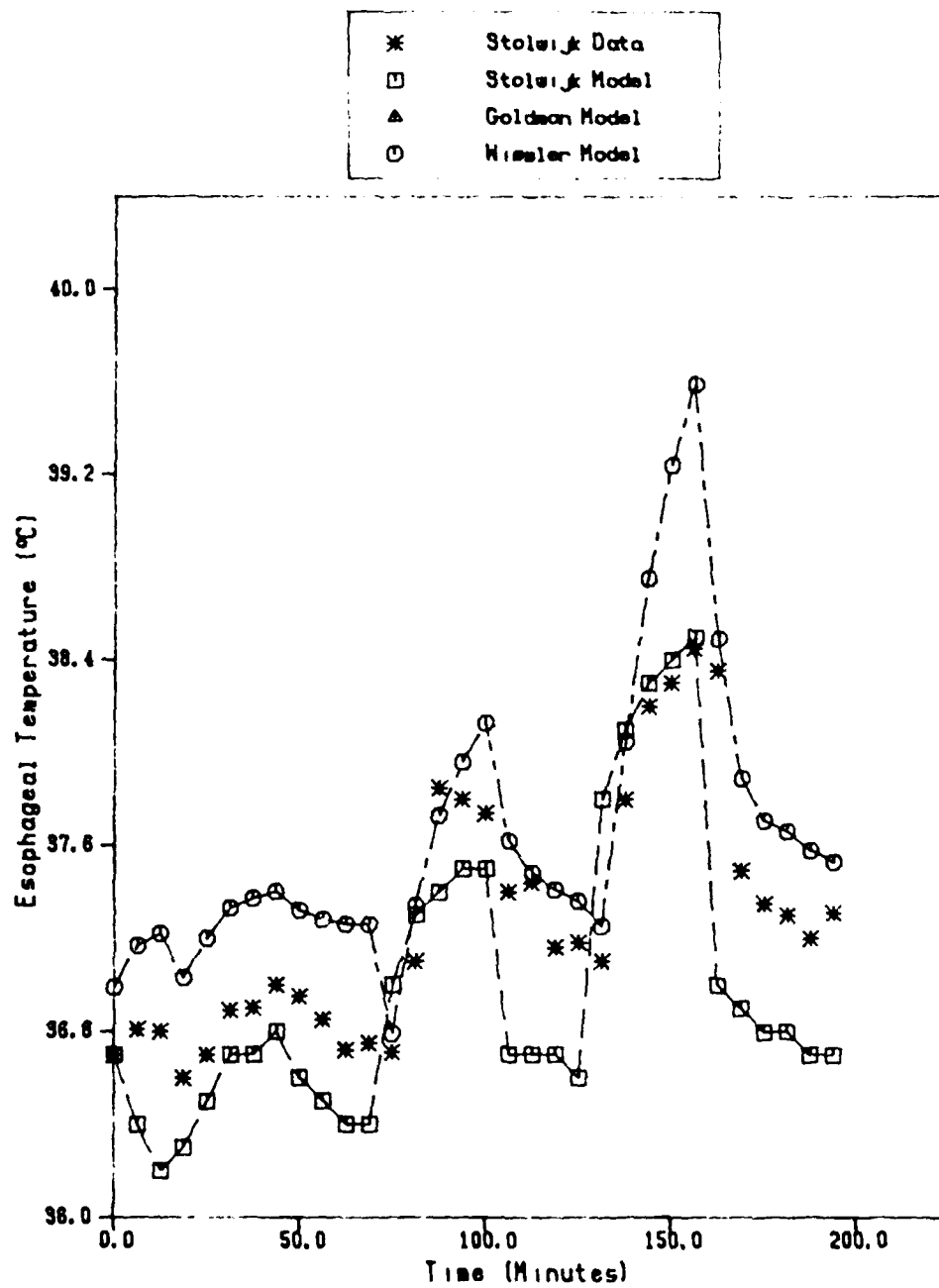
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject BC During Exercise Periods in 30 °C Air



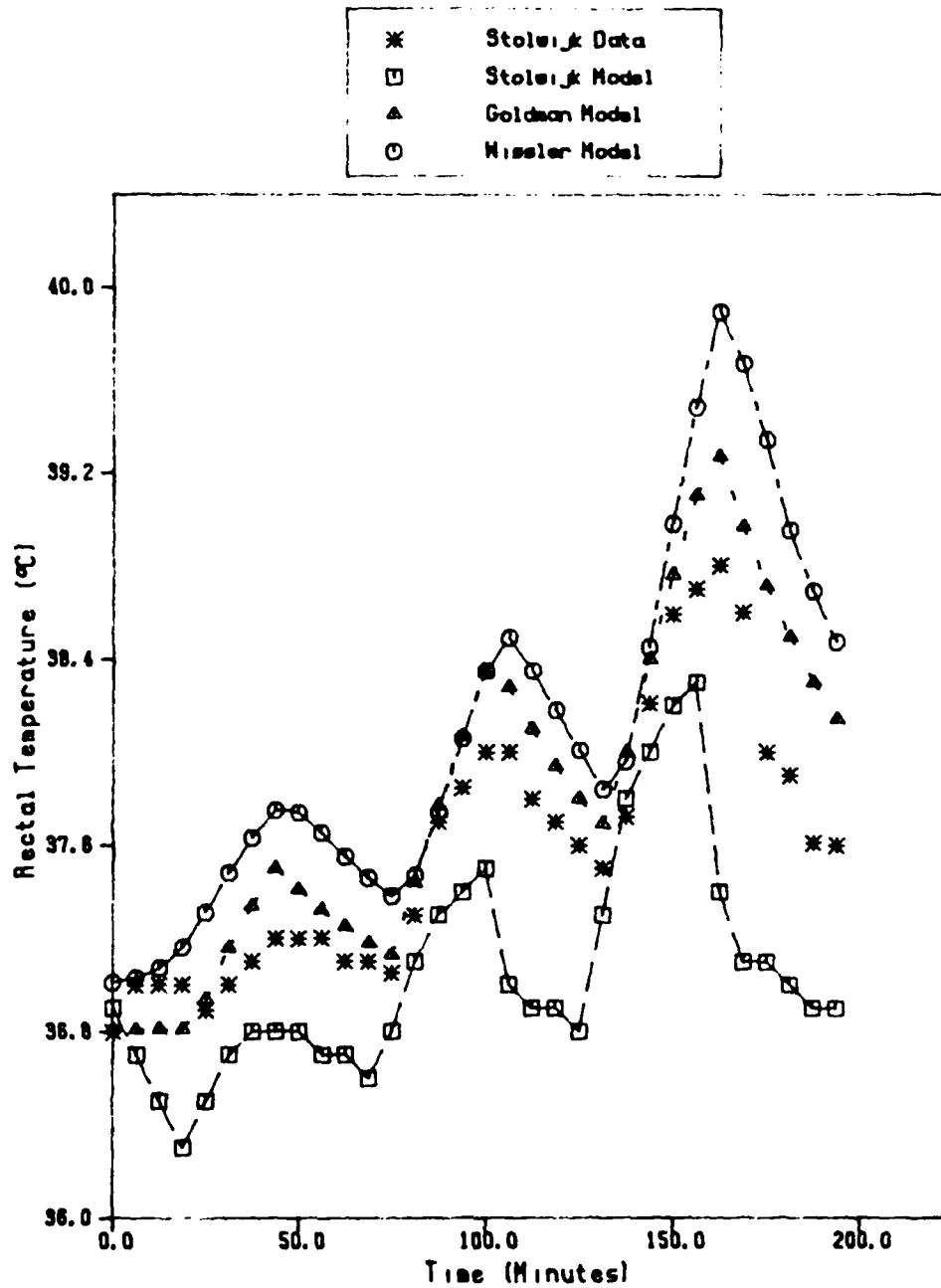
Metabolic Rate
As a Function of Time
For Subject BC During Exercise Periods in 30 °C Air



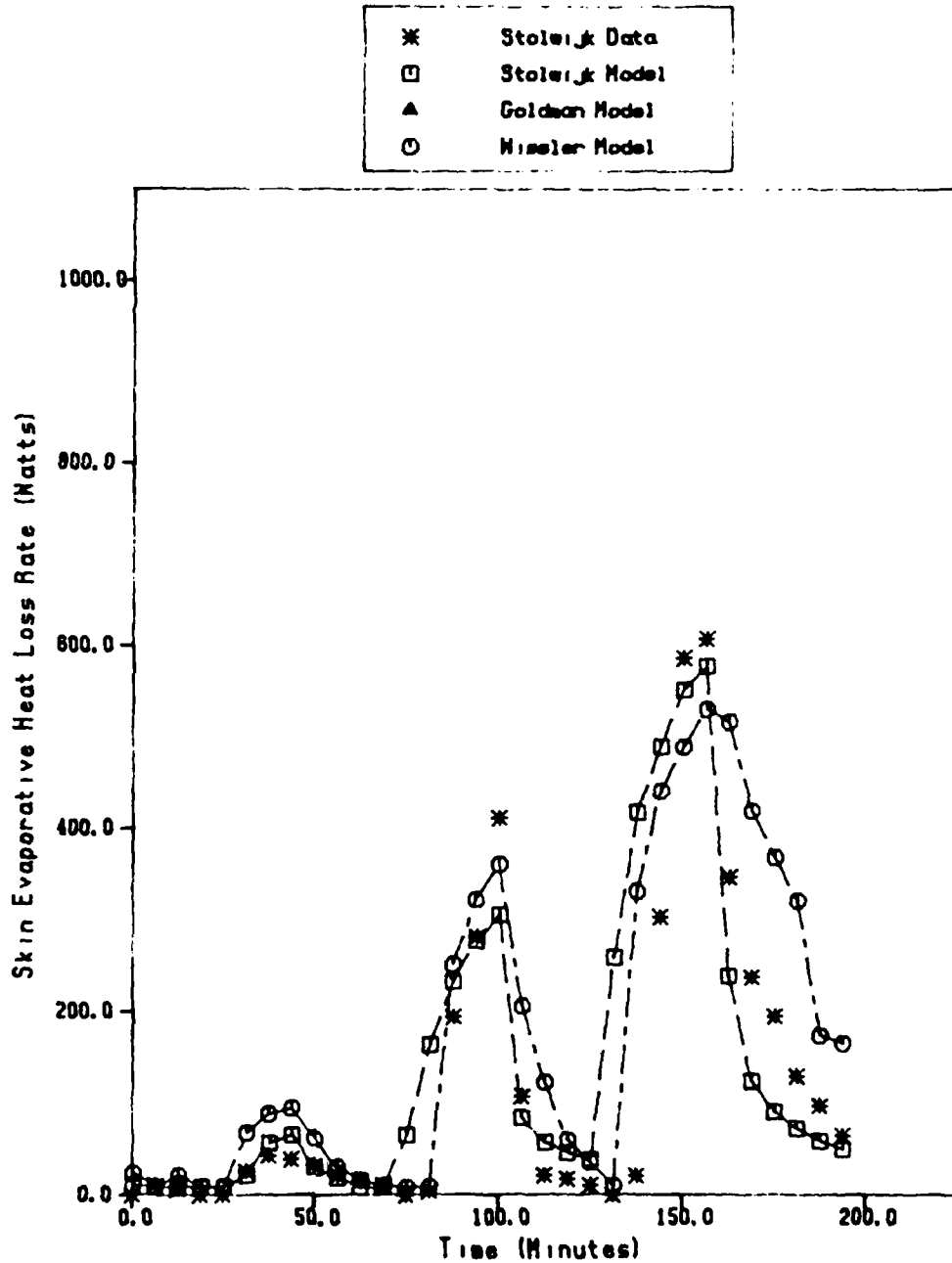
Esophageal Temperature
As a Function of Time
For Subject BS During Exercise Periods in 20 °C Air



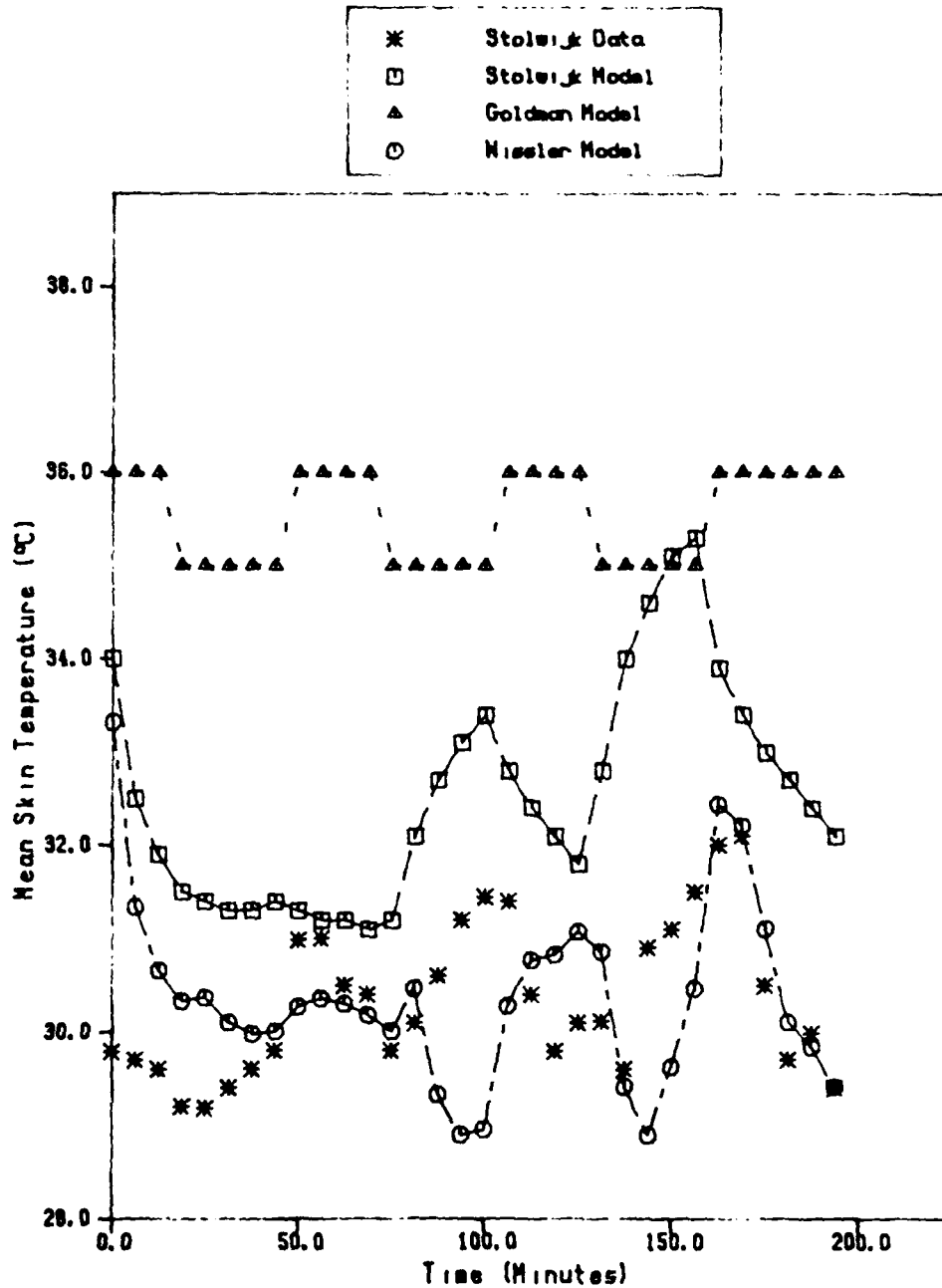
Rectal Temperature
As a Function of Time
For Subject BS During Exercise Periods in 20 °C Air



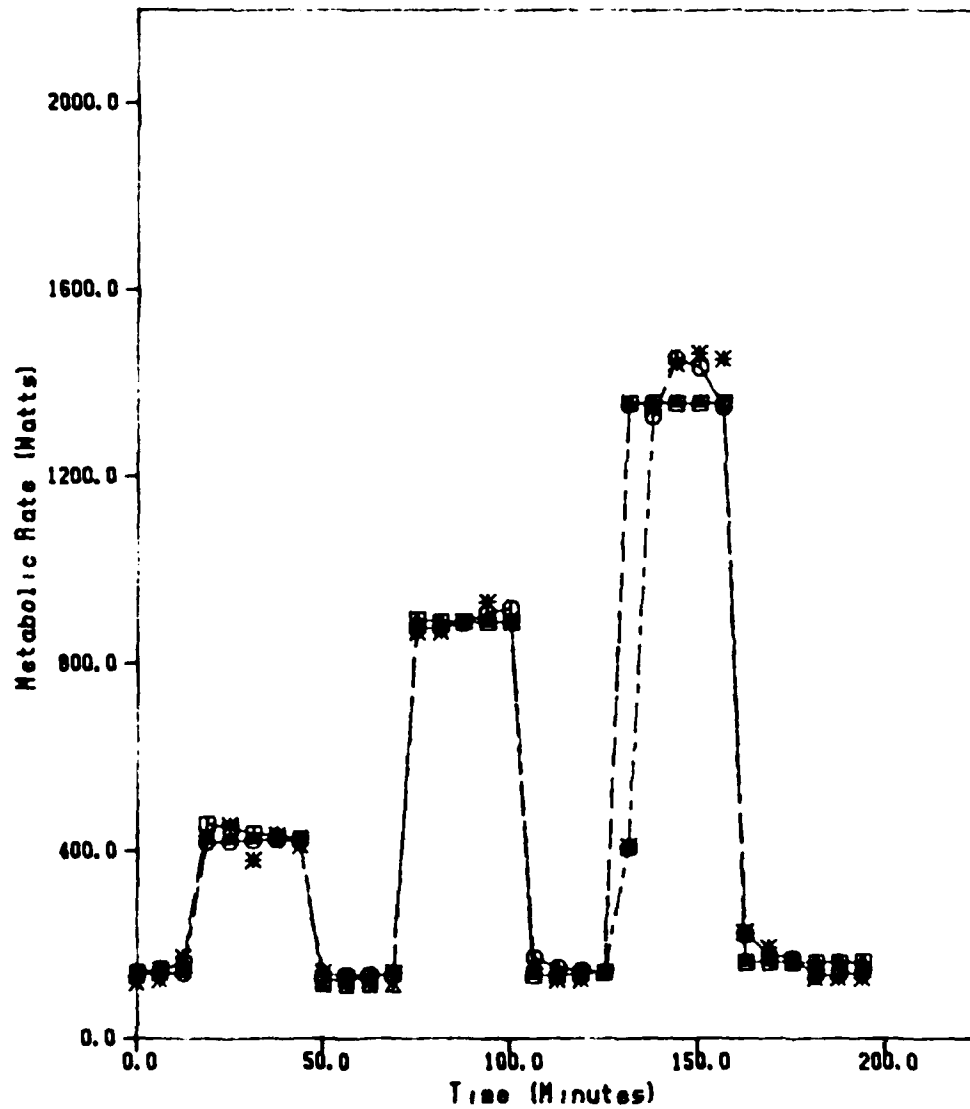
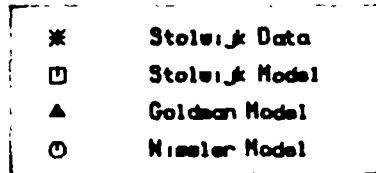
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject BS During Exercise Periods in 20 °C Air



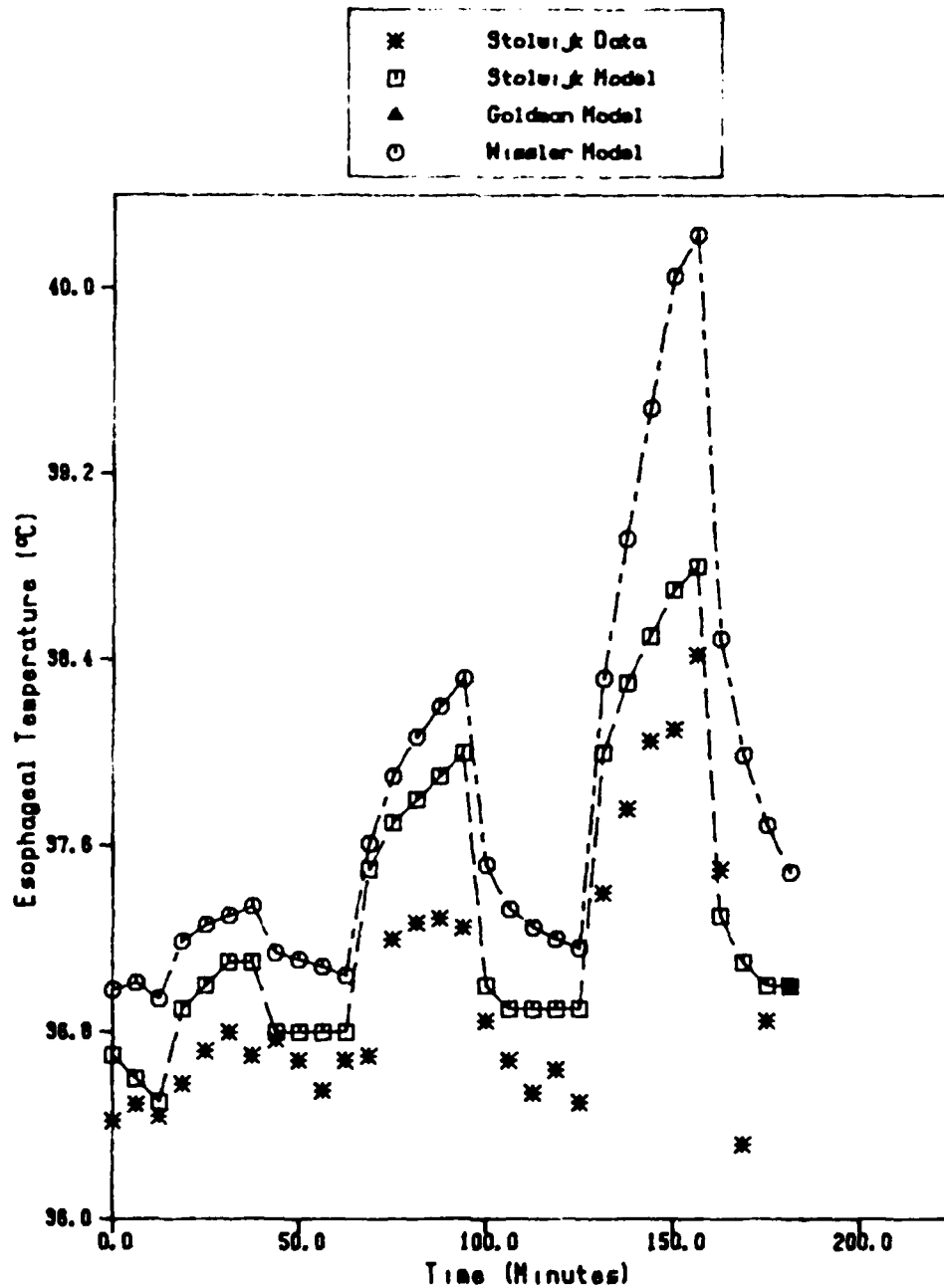
Mean Skin Temperature
As a Function of Time
For Subject BS During Exercise Periods in 20 °C Air



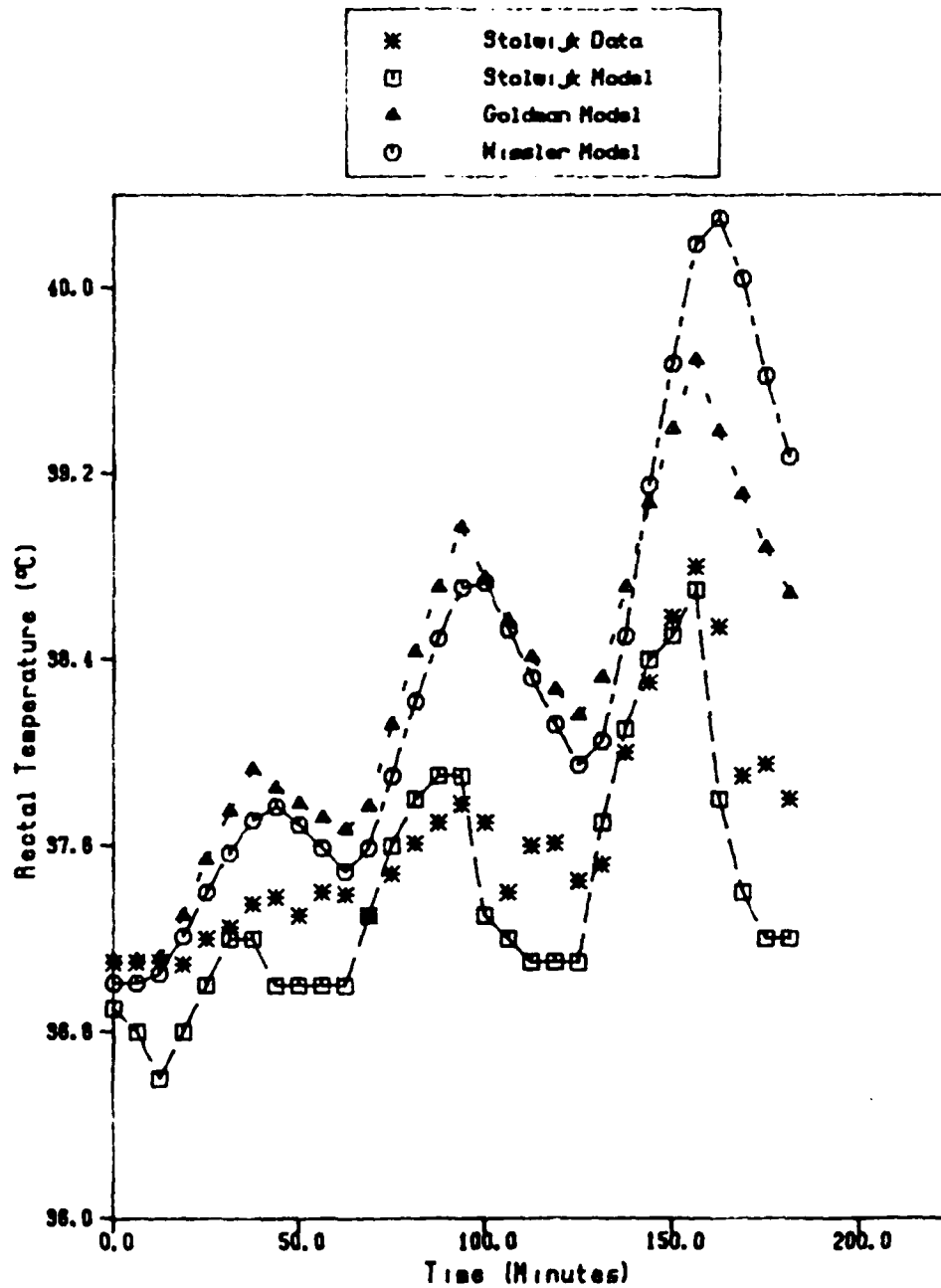
Metabolic Rate
As a Function of Time
For Subject BS During Exercise Periods in 20 °C Air



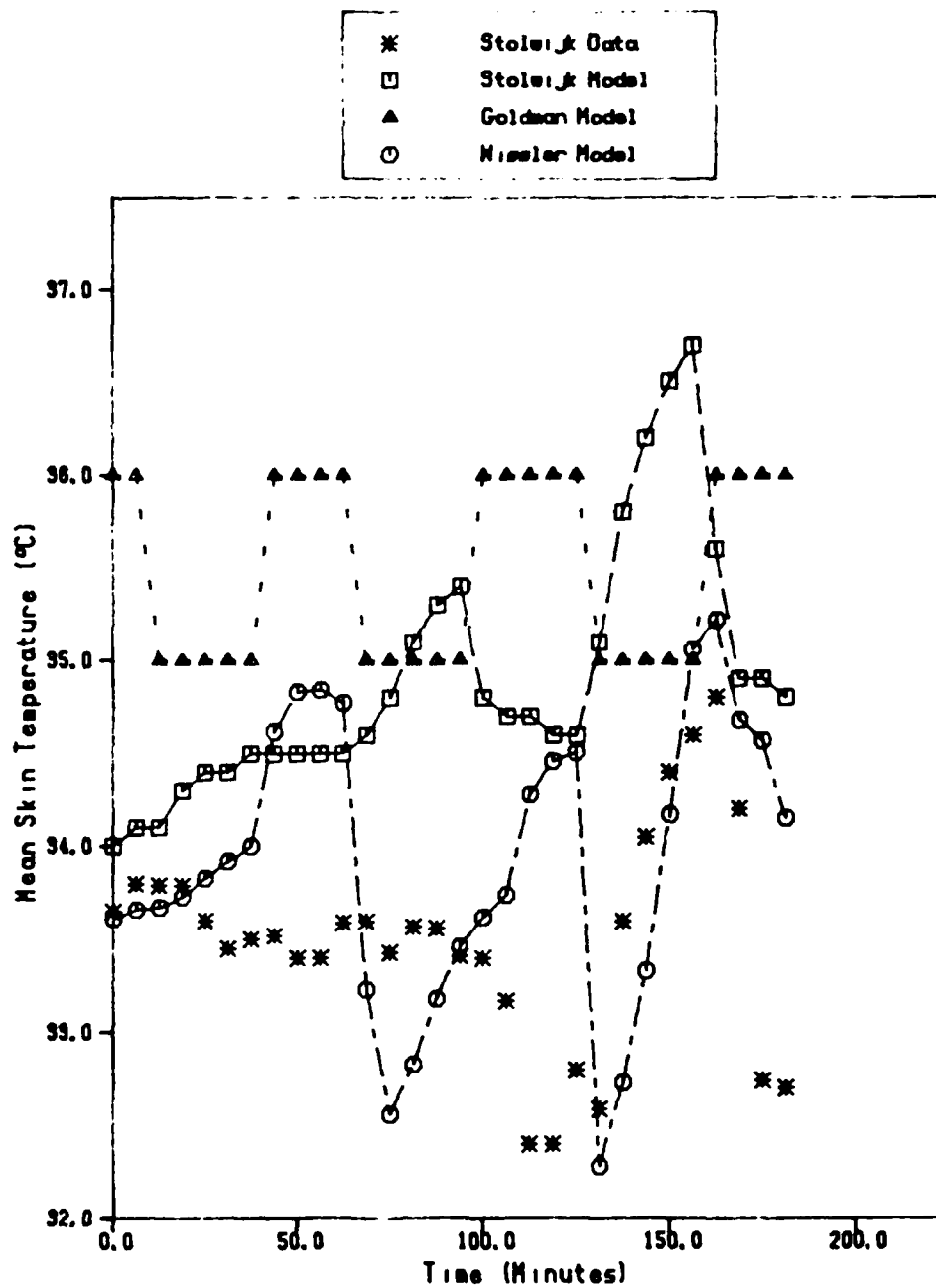
Esophageal Temperature
As a Function of Time
For Subject BS During Exercise Periods in 30 °C Air



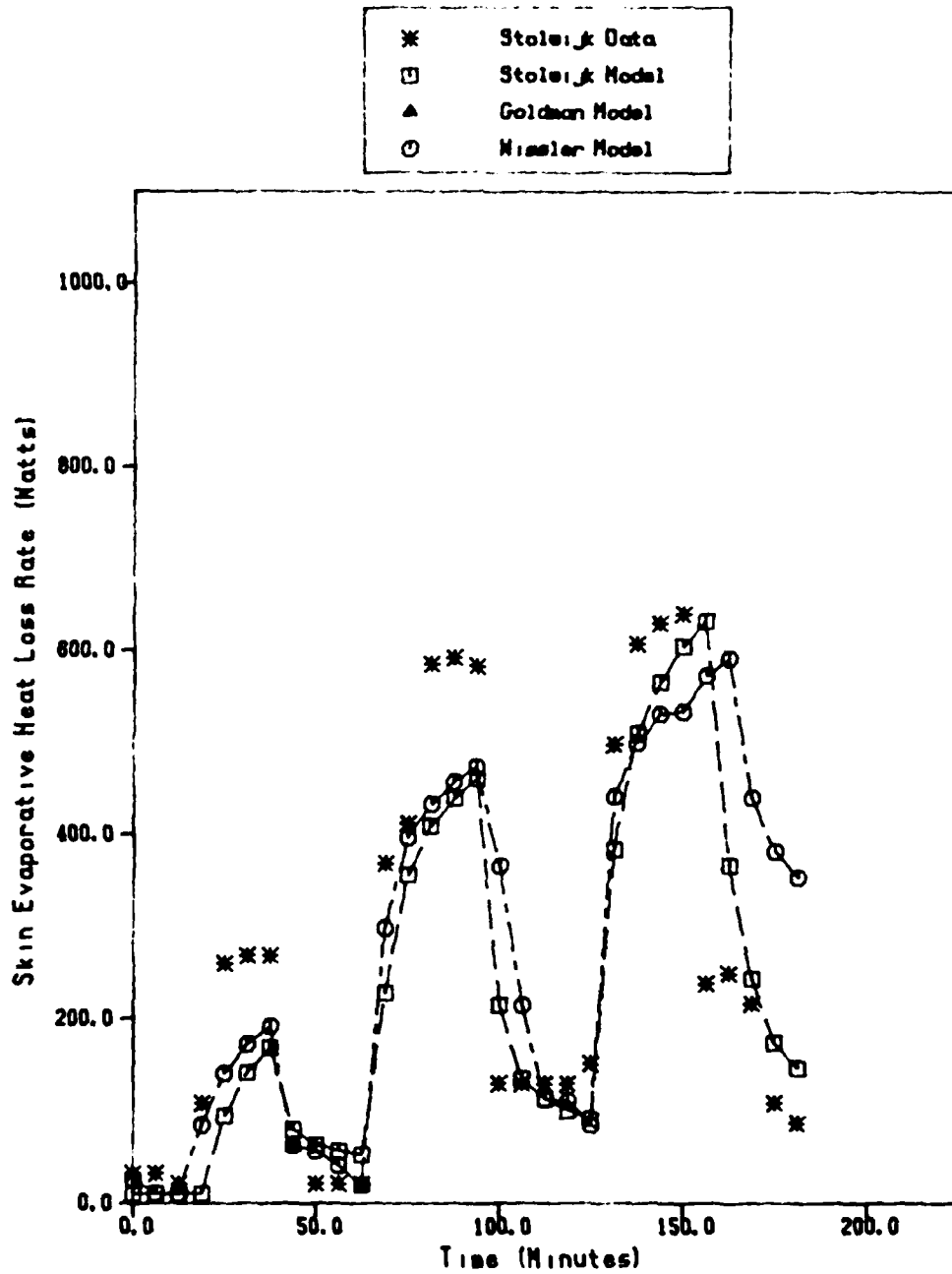
Rectal Temperature
As a Function of Time
For Subject BS During Exercise Periods in 30 °C Air



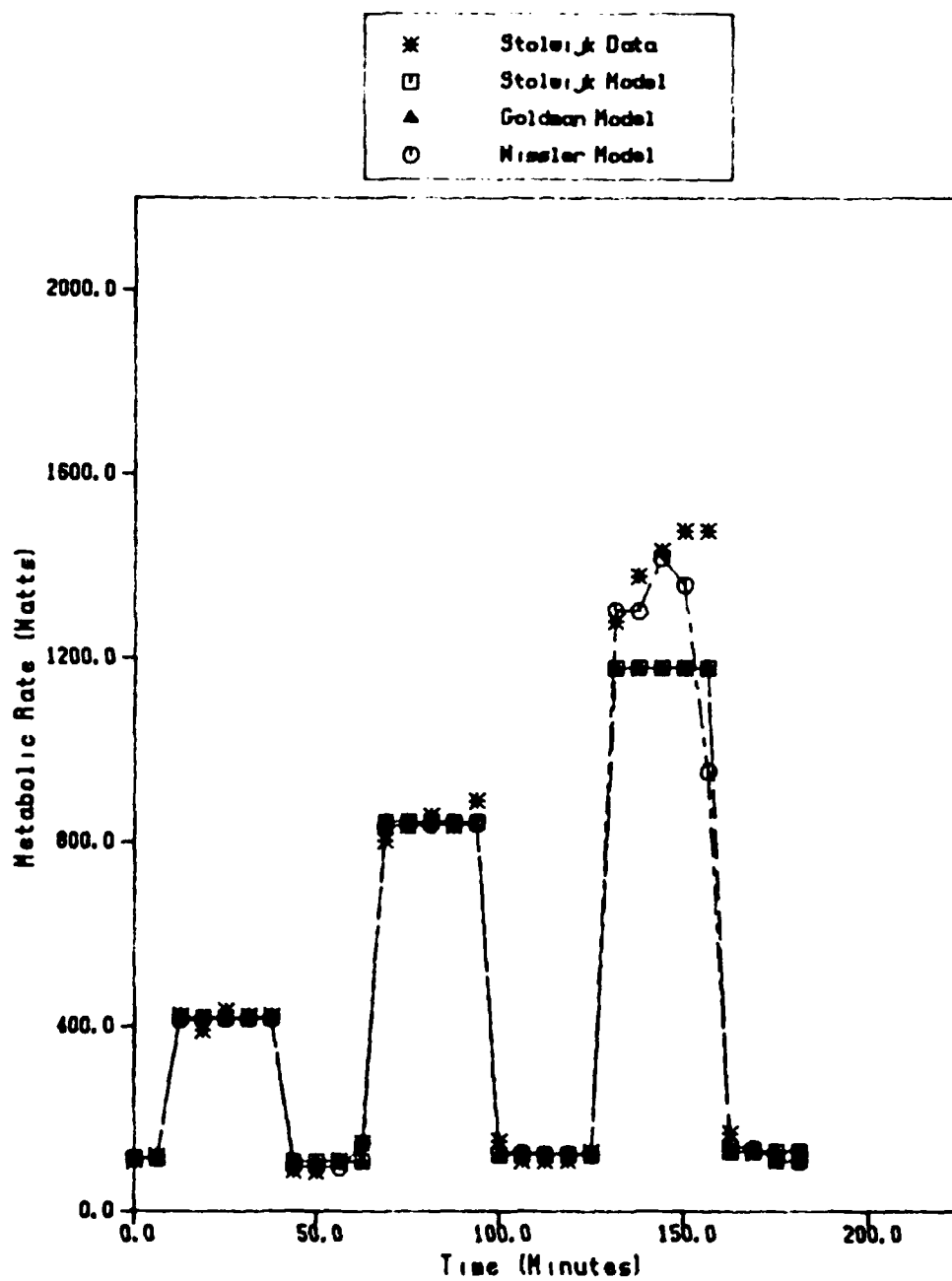
Mean Skin Temperature
As a Function of Time
For Subject BS During Exercise Periods in 30 °C Air



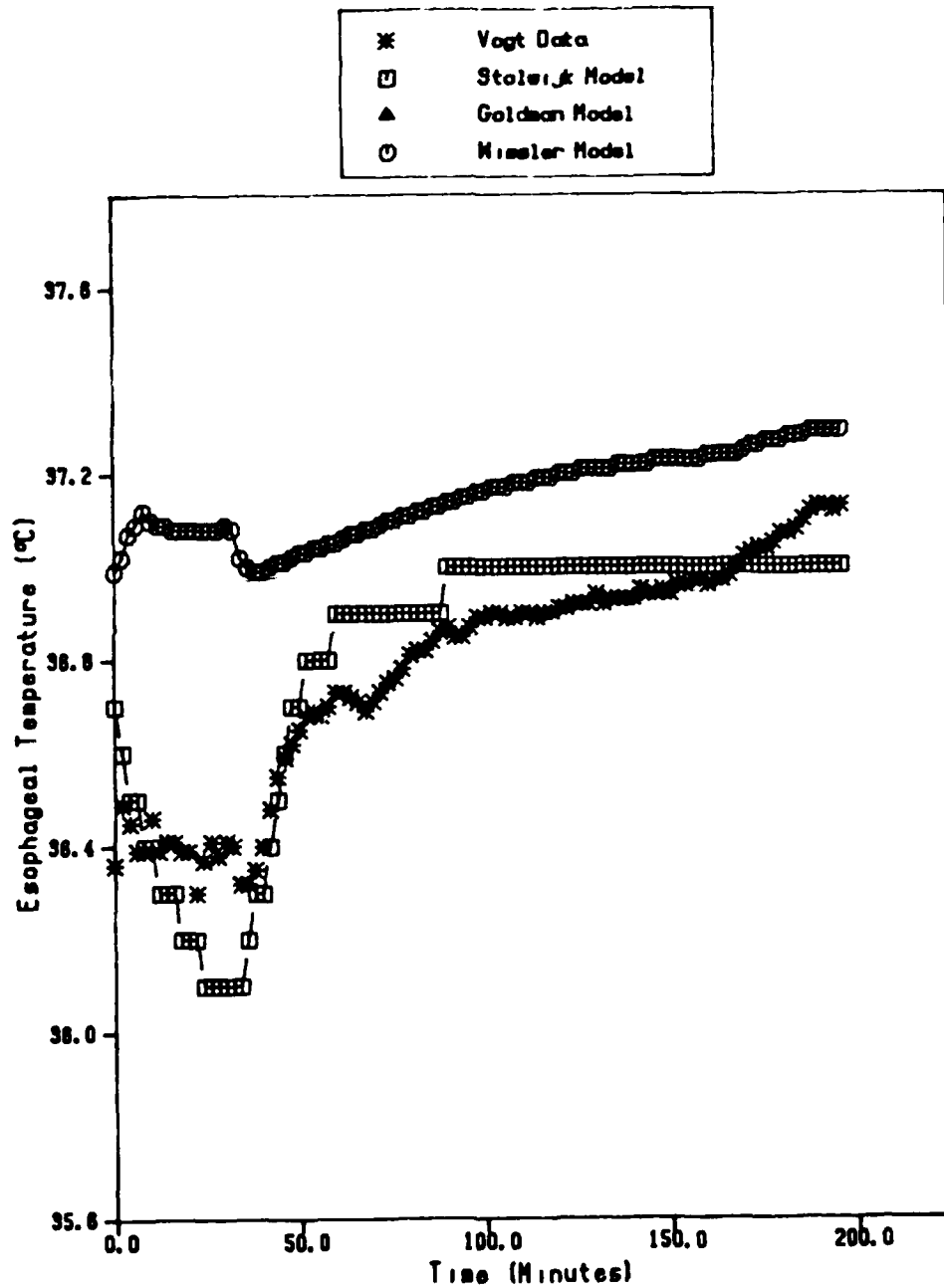
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject BS During Exercise Periods in 30 °C Air



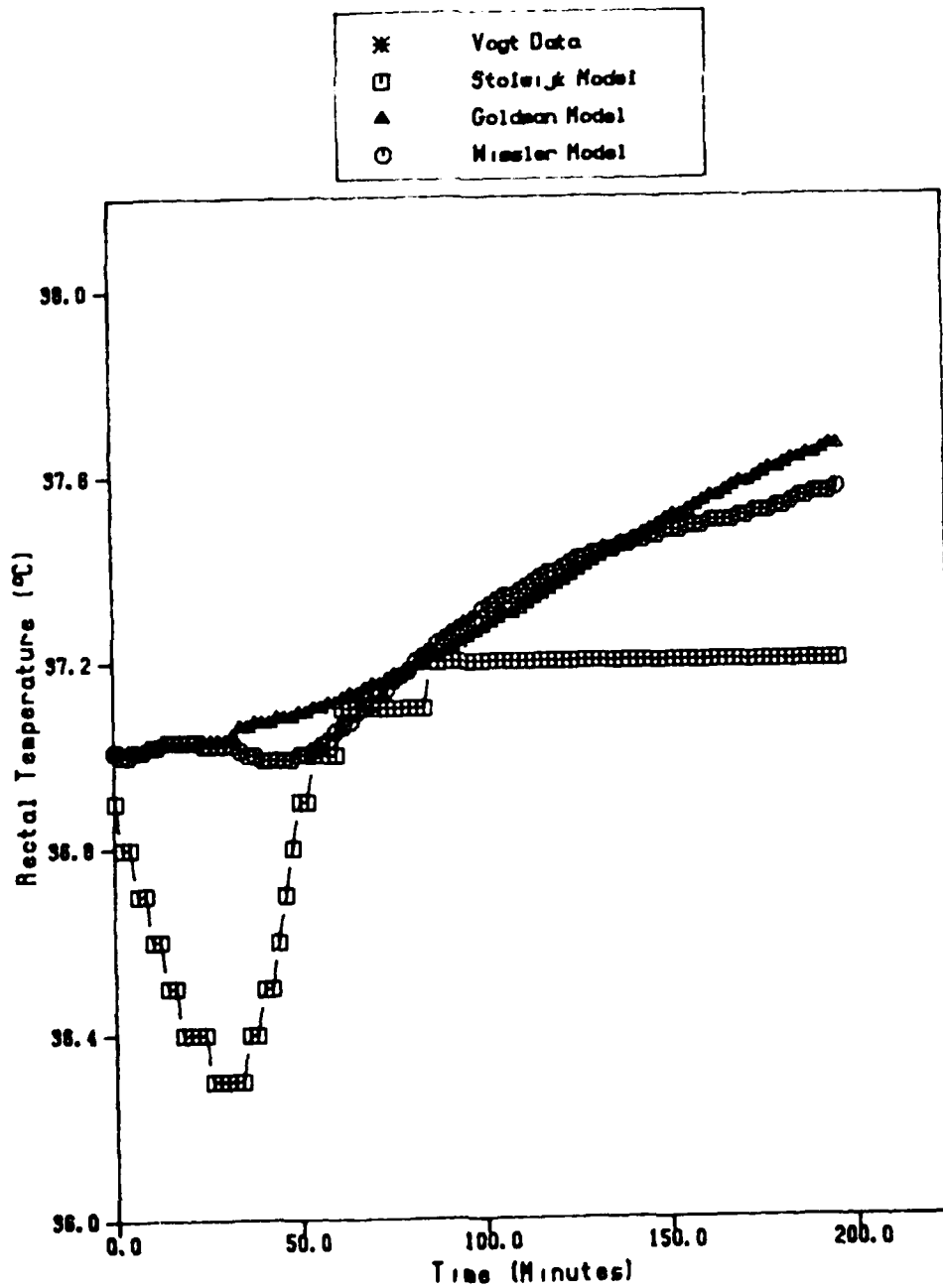
Metabolic Rate
As a Function of Time
For Subject BS During Exercise Periods in 30 °C Air



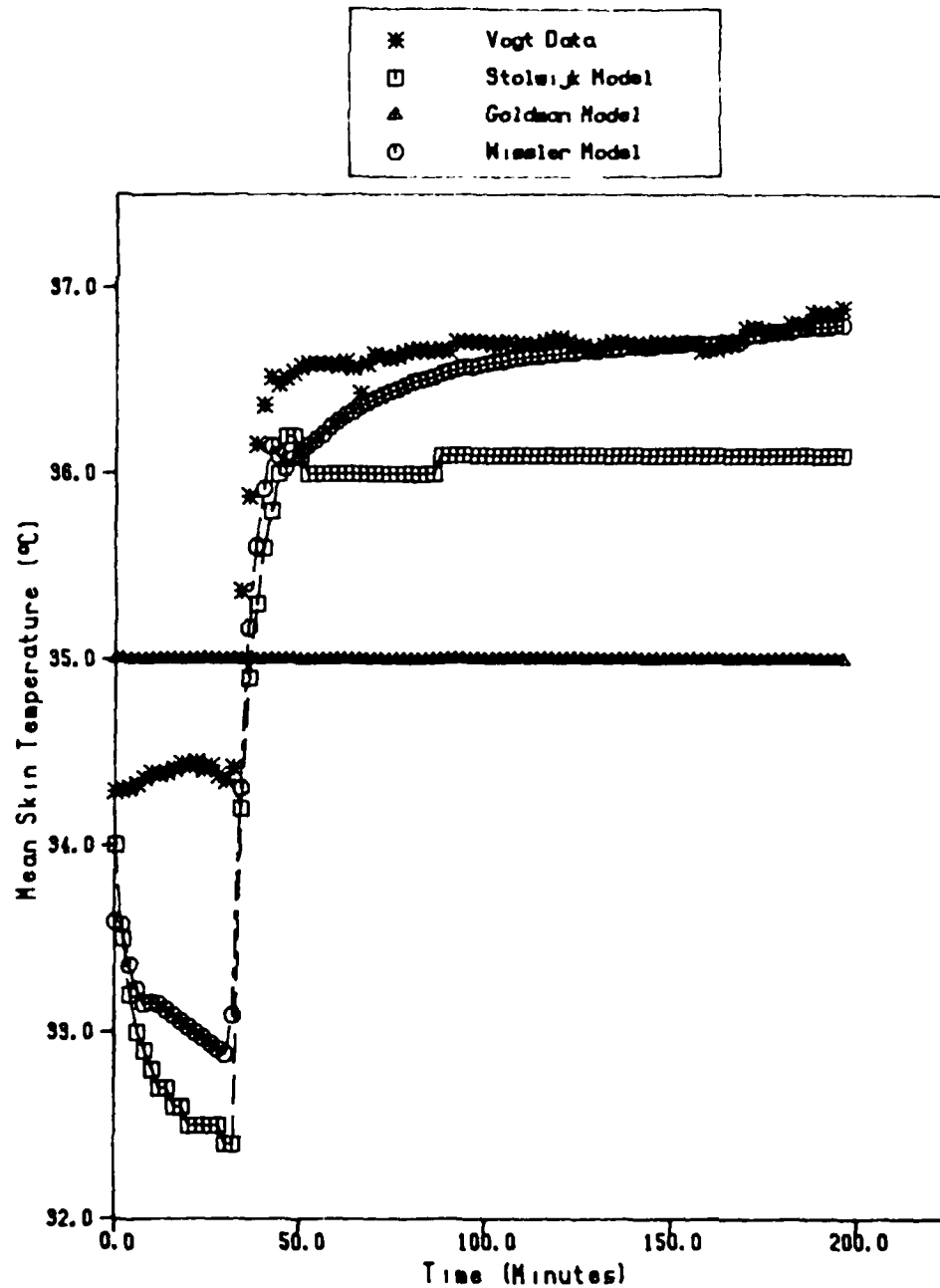
Esophageal Temperature
As a Function of Time
For Subject KG Step Change to 38°C (DP=35°C)



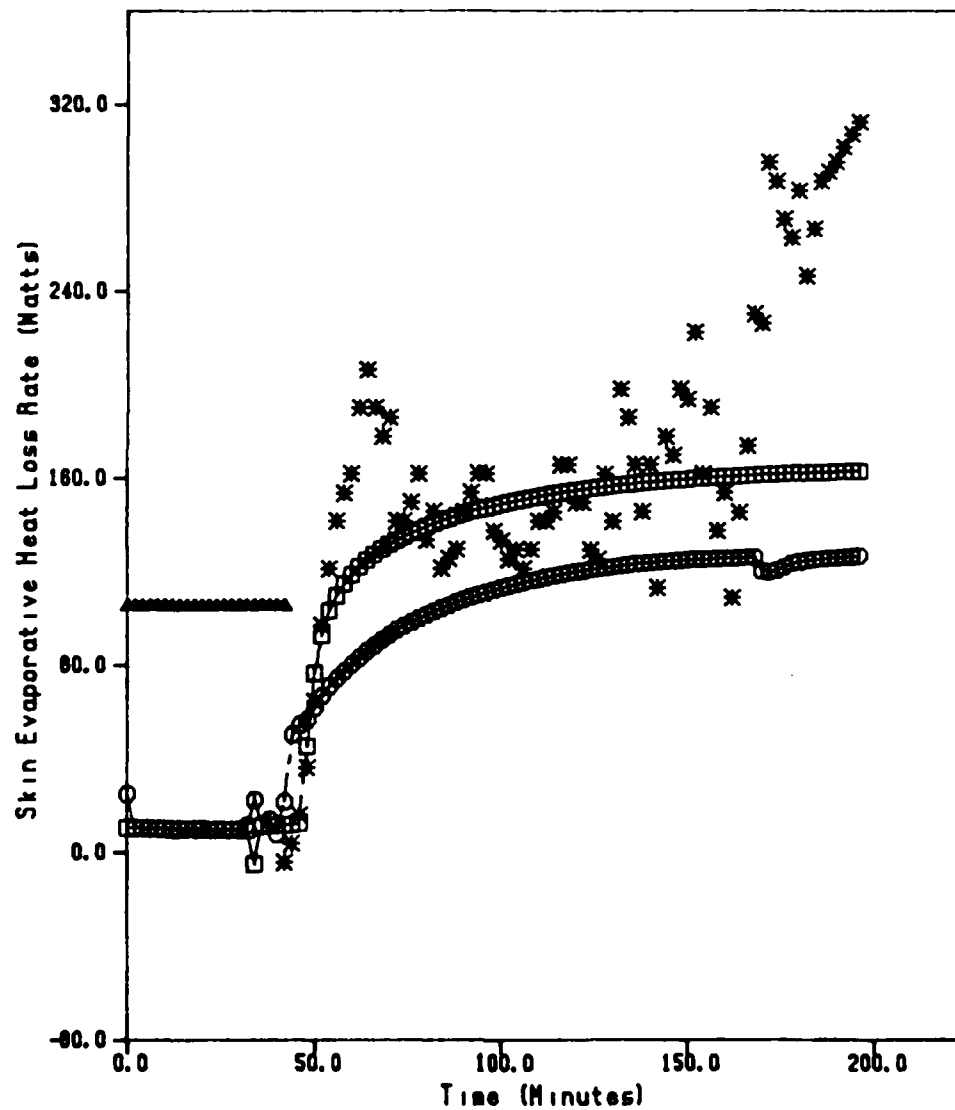
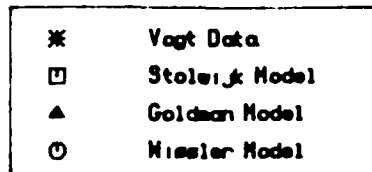
Rectal Temperature
As a Function of Time
For Subject KG Step Change to 38°C (DP=35°C)



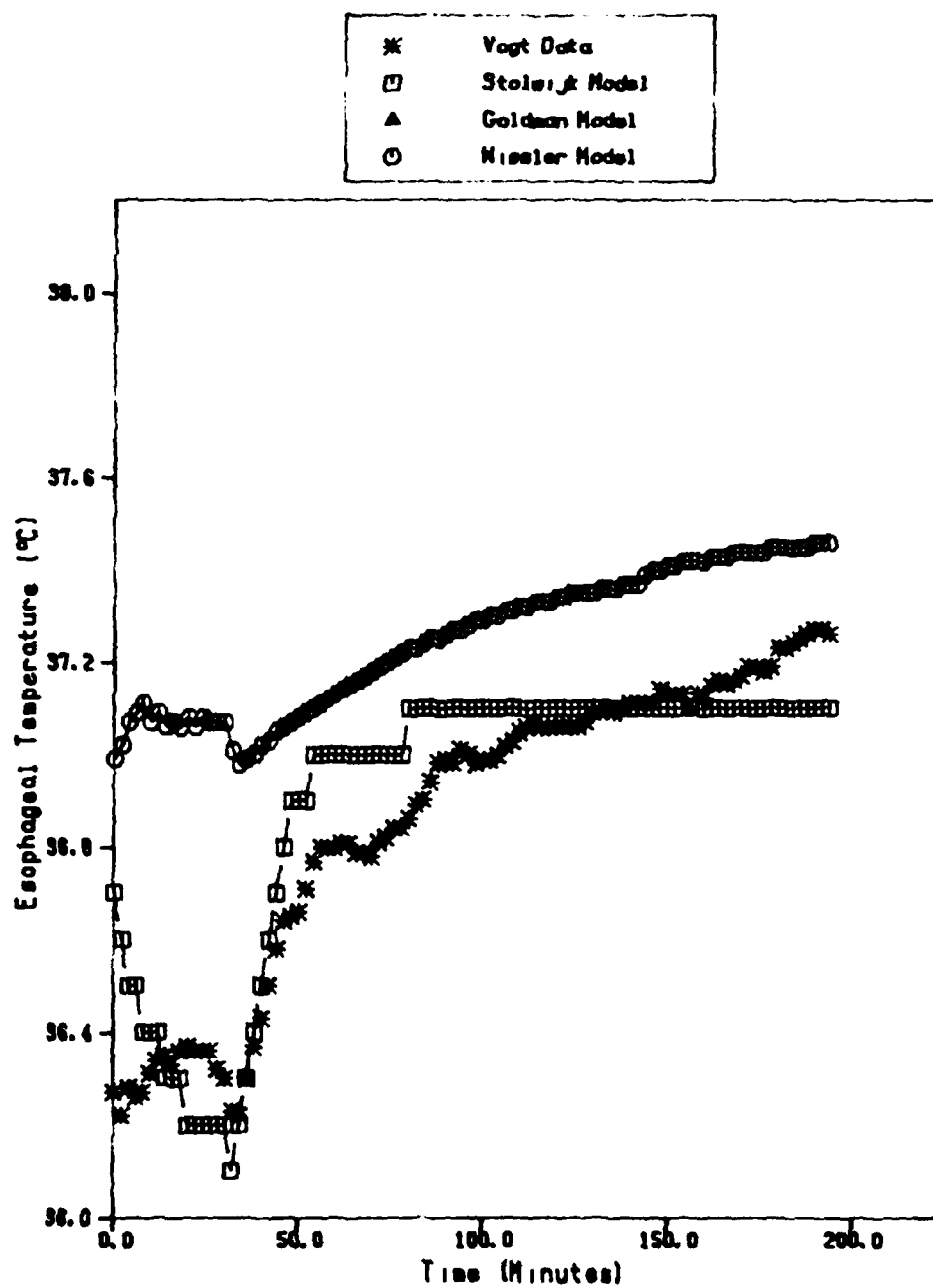
Mean Skin Temperature
As a Function of Time
For Subject KG Step Change to 38°C (DP=35°C)



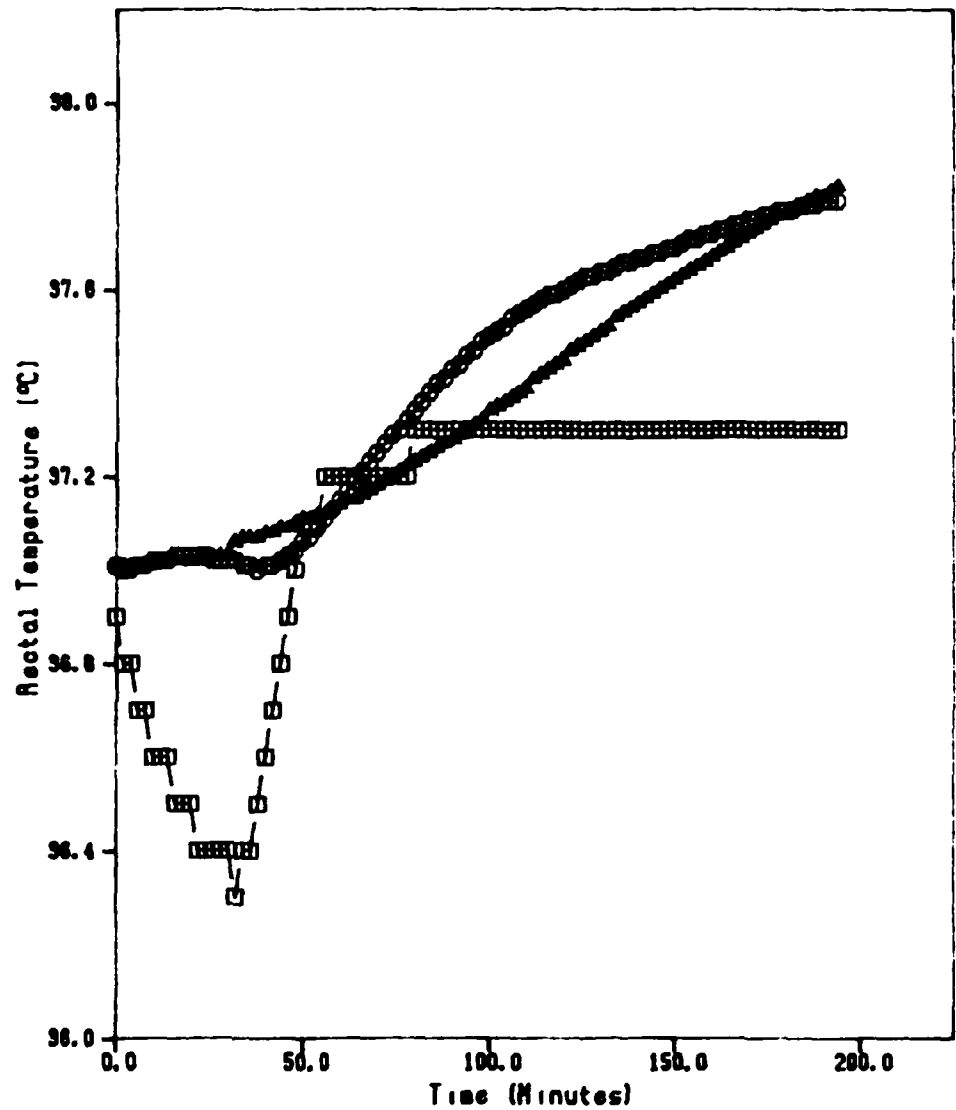
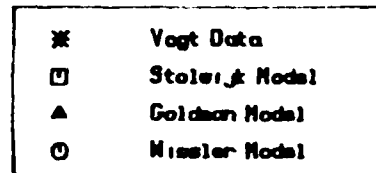
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject KG Step Change to 38°C (DP=35°C)



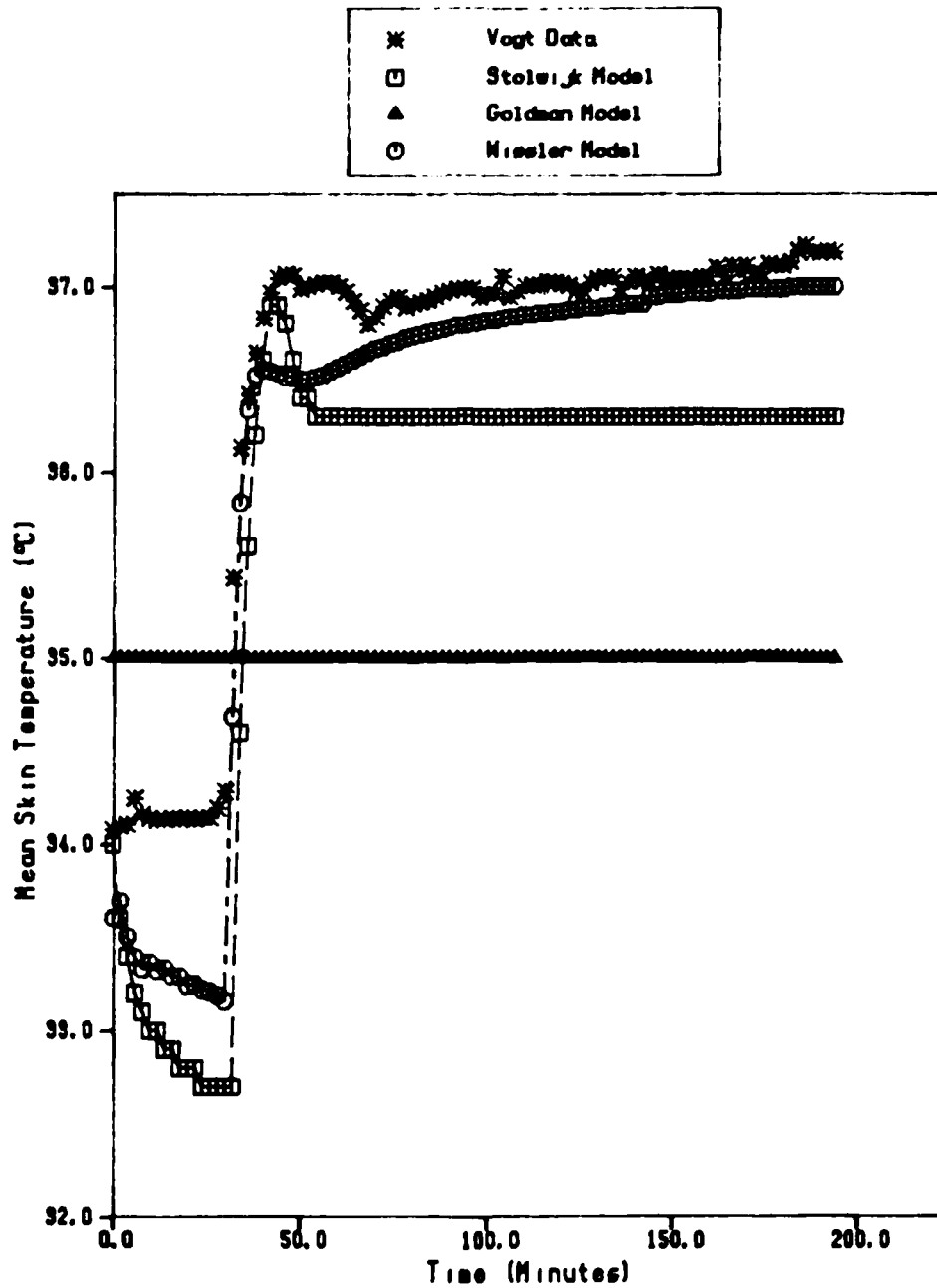
Esophageal Temperature
As a Function of Time
For Subject KG Step Change to 42°C (DP=28°C)



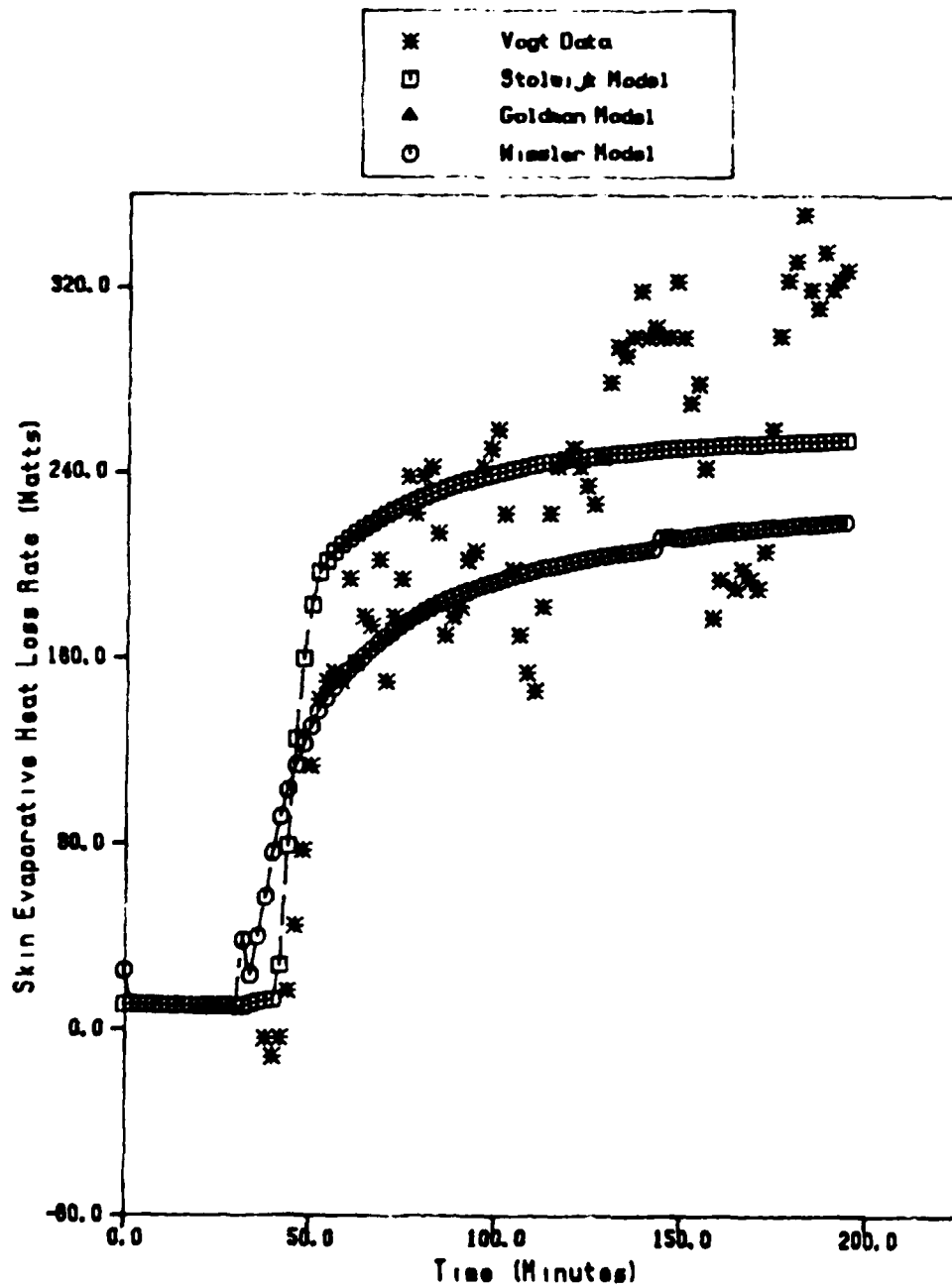
Rectal Temperature
As a Function of Time
For Subject KG Step Change to 42°C (DP=28°C)



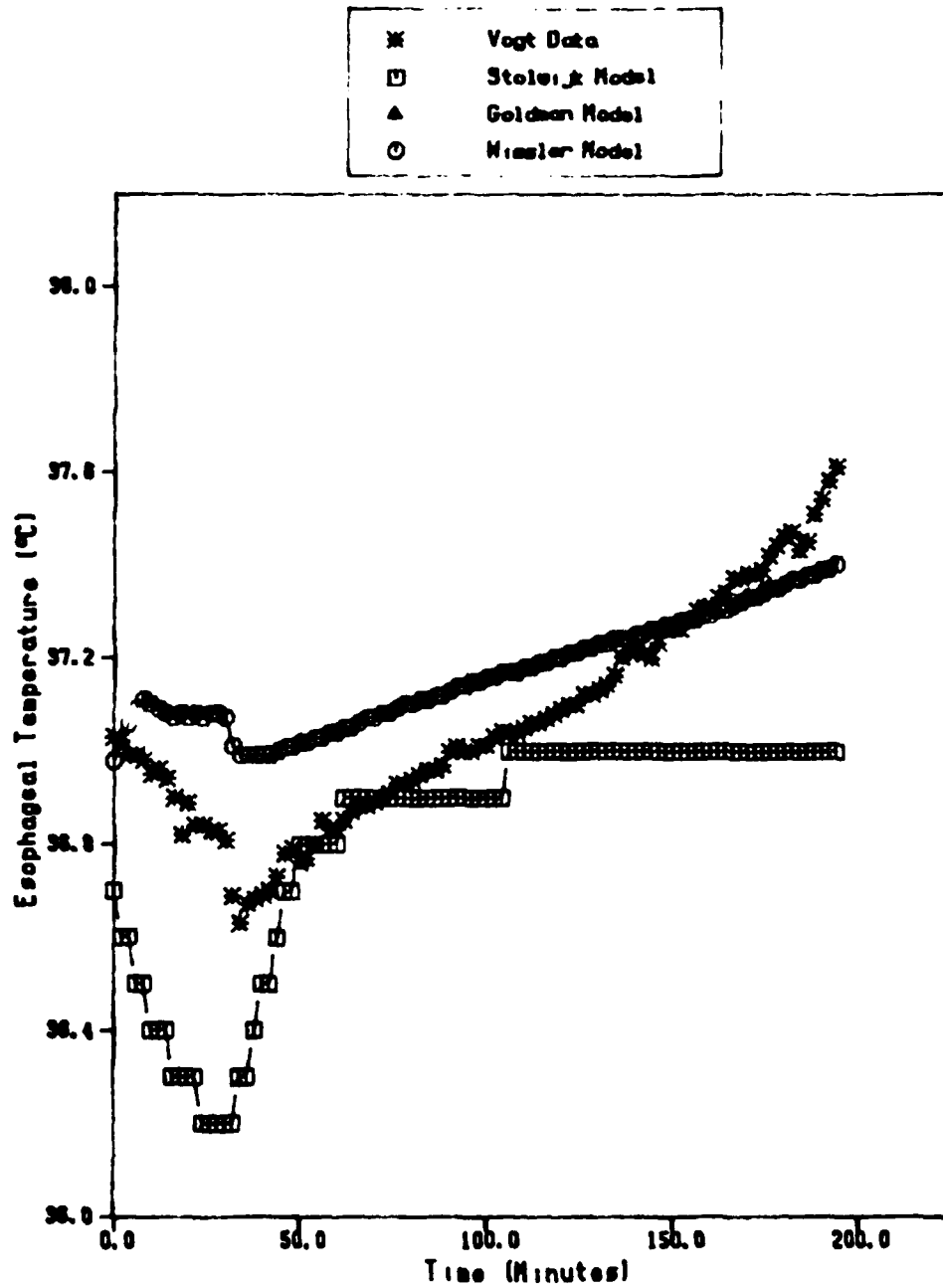
Mean Skin Temperature
As a Function of Time
For Subject KG Step Change to 42°C (OP=28°C)



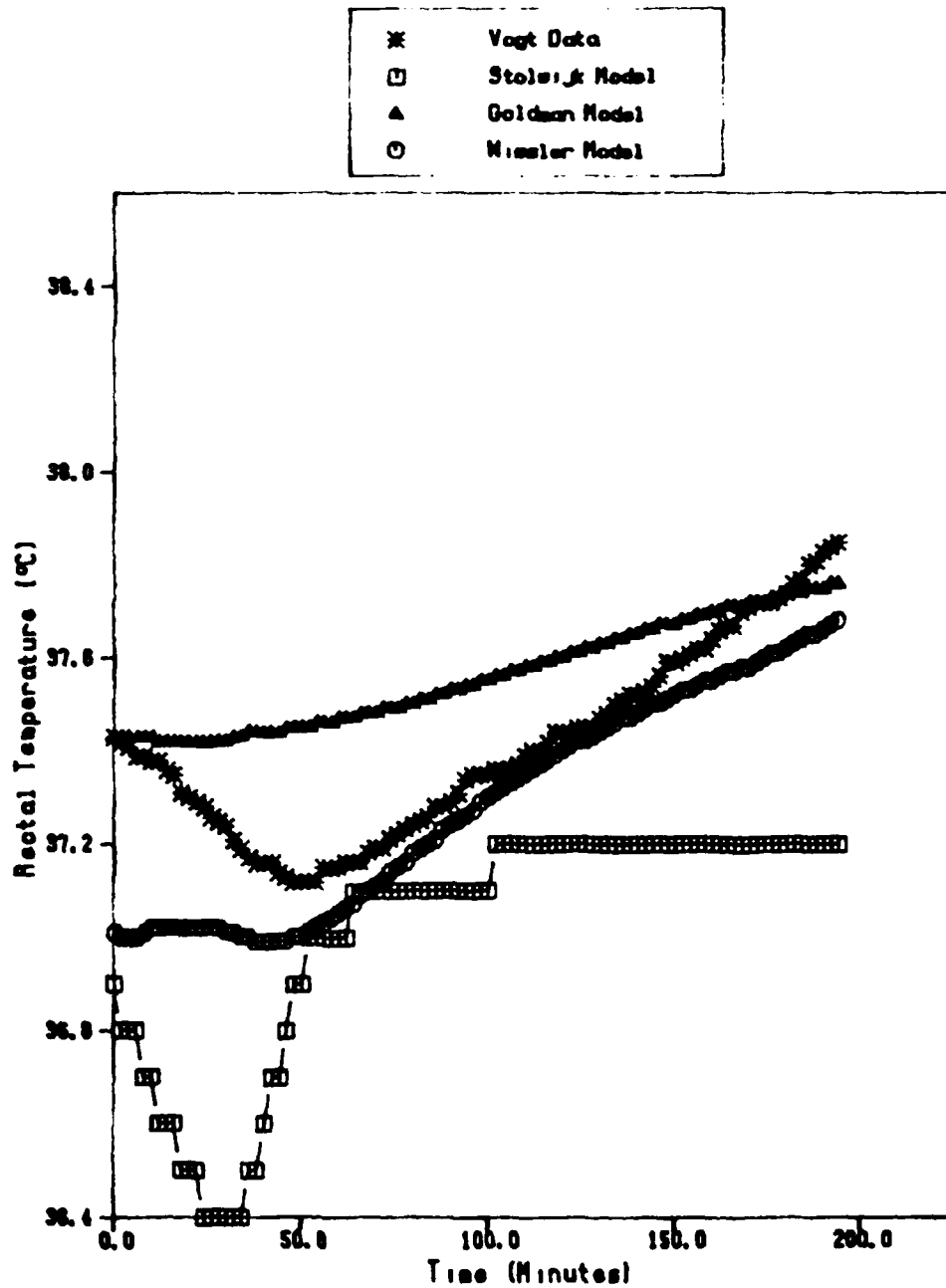
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject KG Step Change to 42°C (DP=28°C)



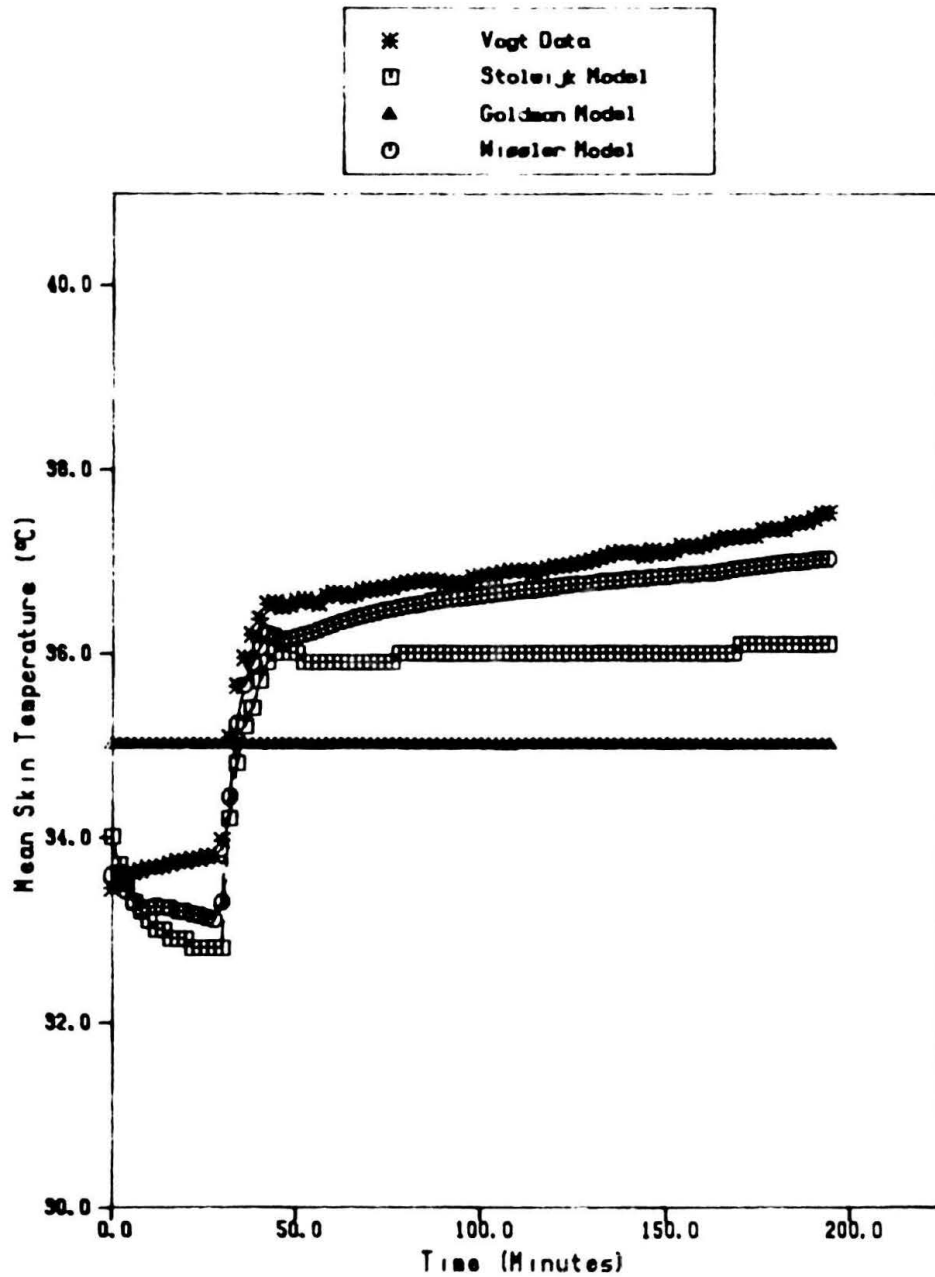
Esophageal Temperature
As a Function of Time
For Subject LA Step Change to 38°C (DP=35°C)



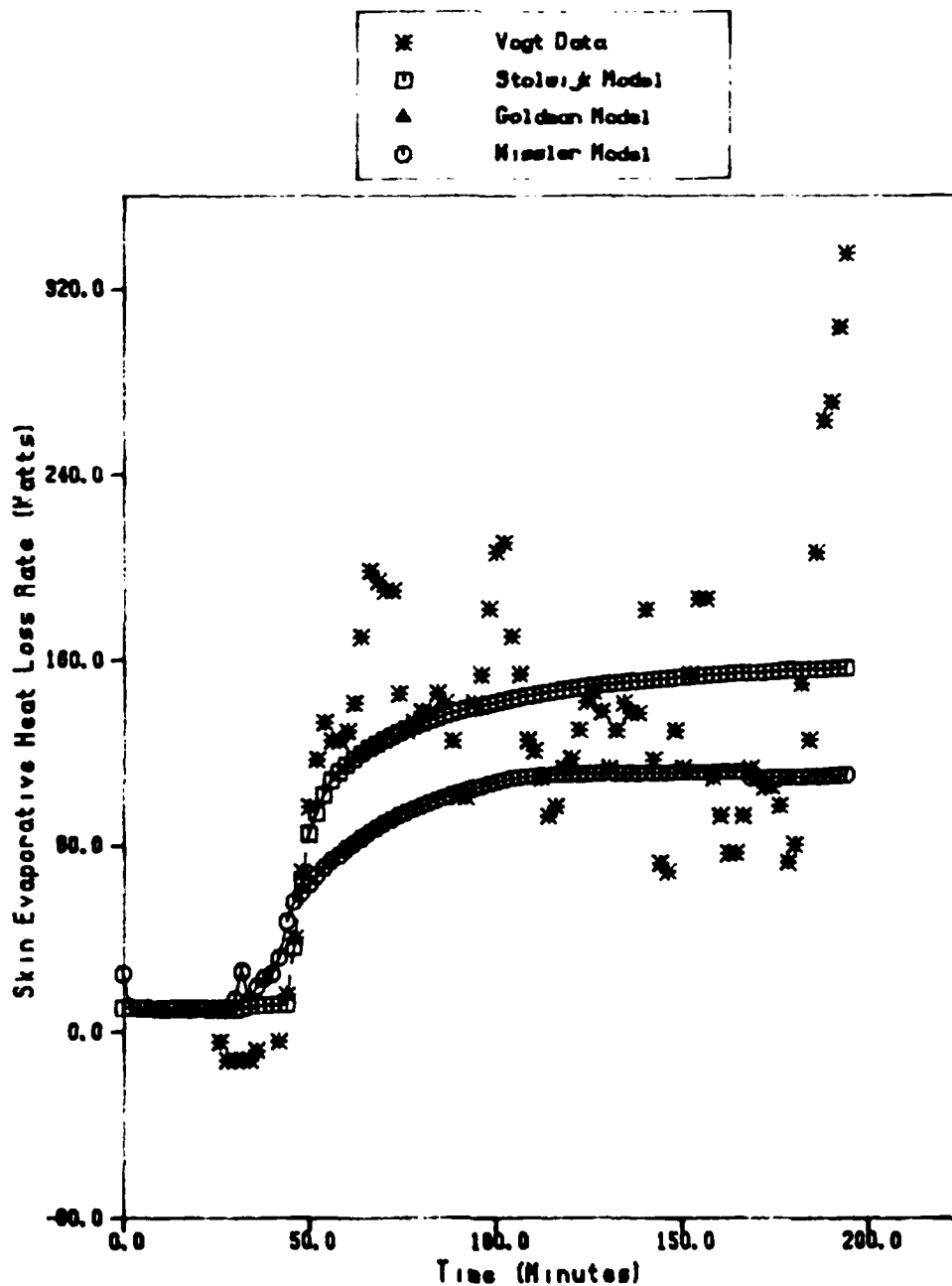
Rectal Temperature
As a Function of Time
For Subject LA Step Change to 38°C (DP=35°C)



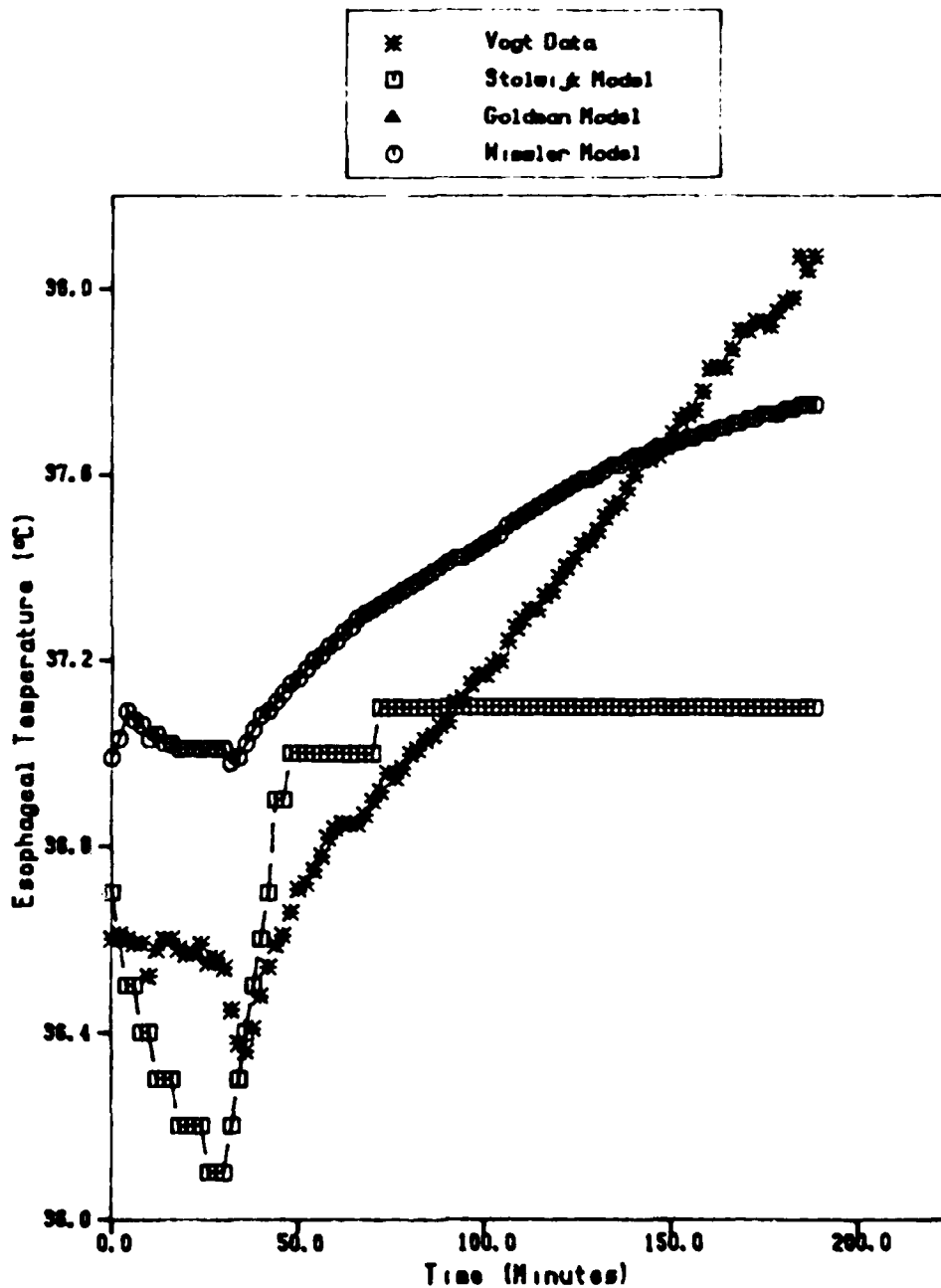
Mean Skin Temperature
As a Function of Time
For Subject LA Step Change to 38°C (DP=35°C)



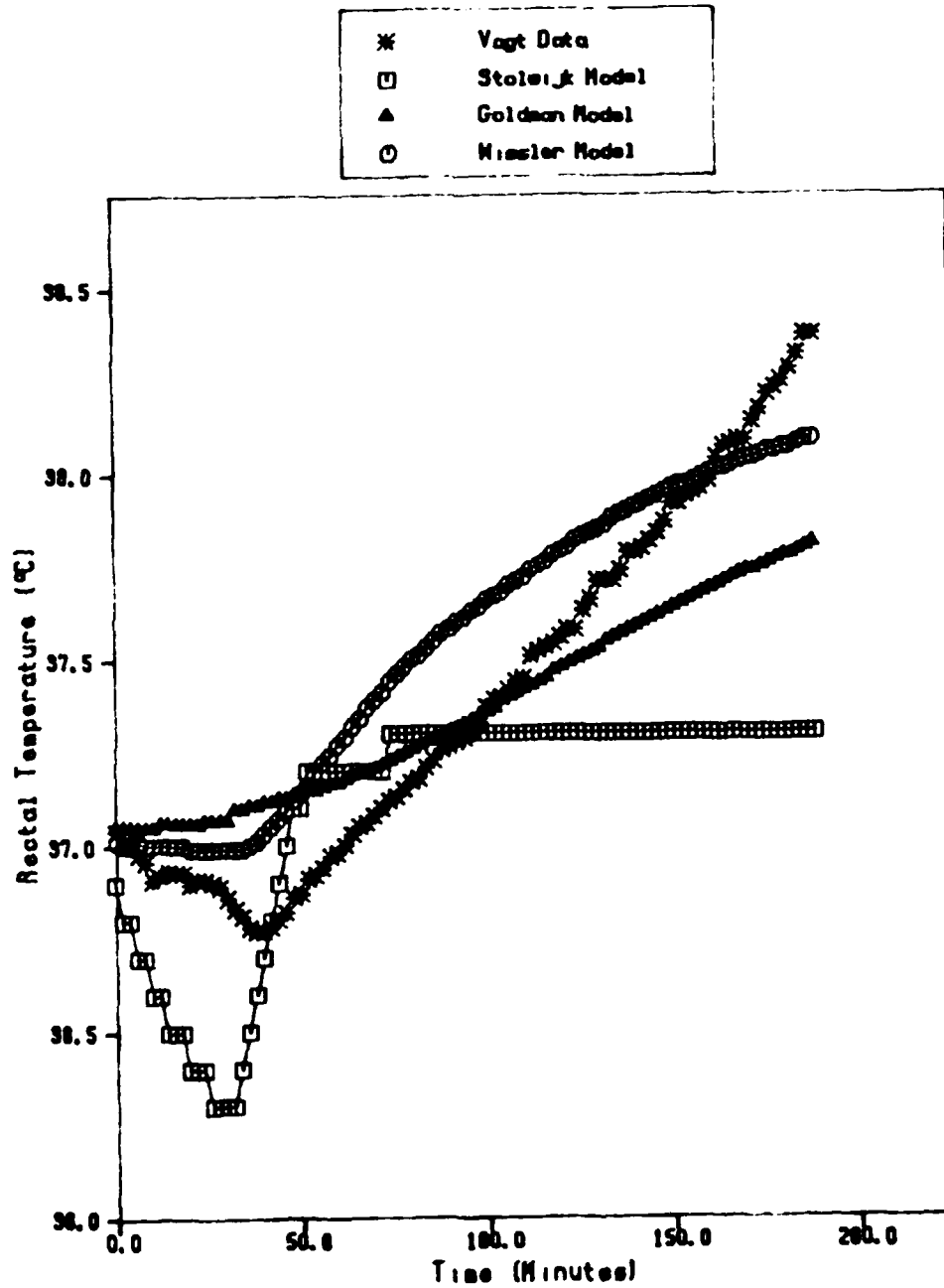
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject LA Step Change to 38°C (DP=35°C)



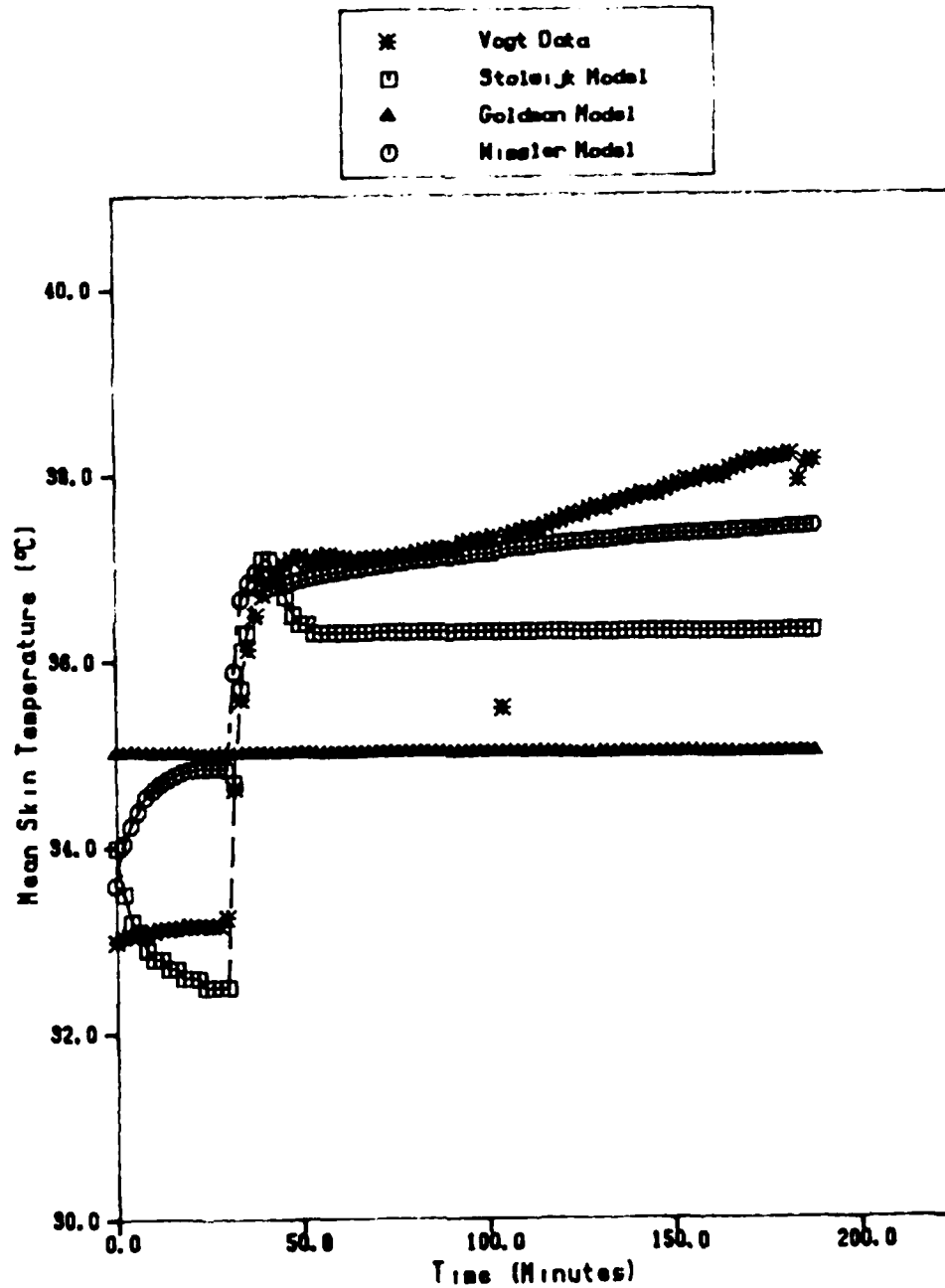
Esophageal Temperature
As a Function of Time
For Subject OM Step Change to 42°C (DP=35°C)



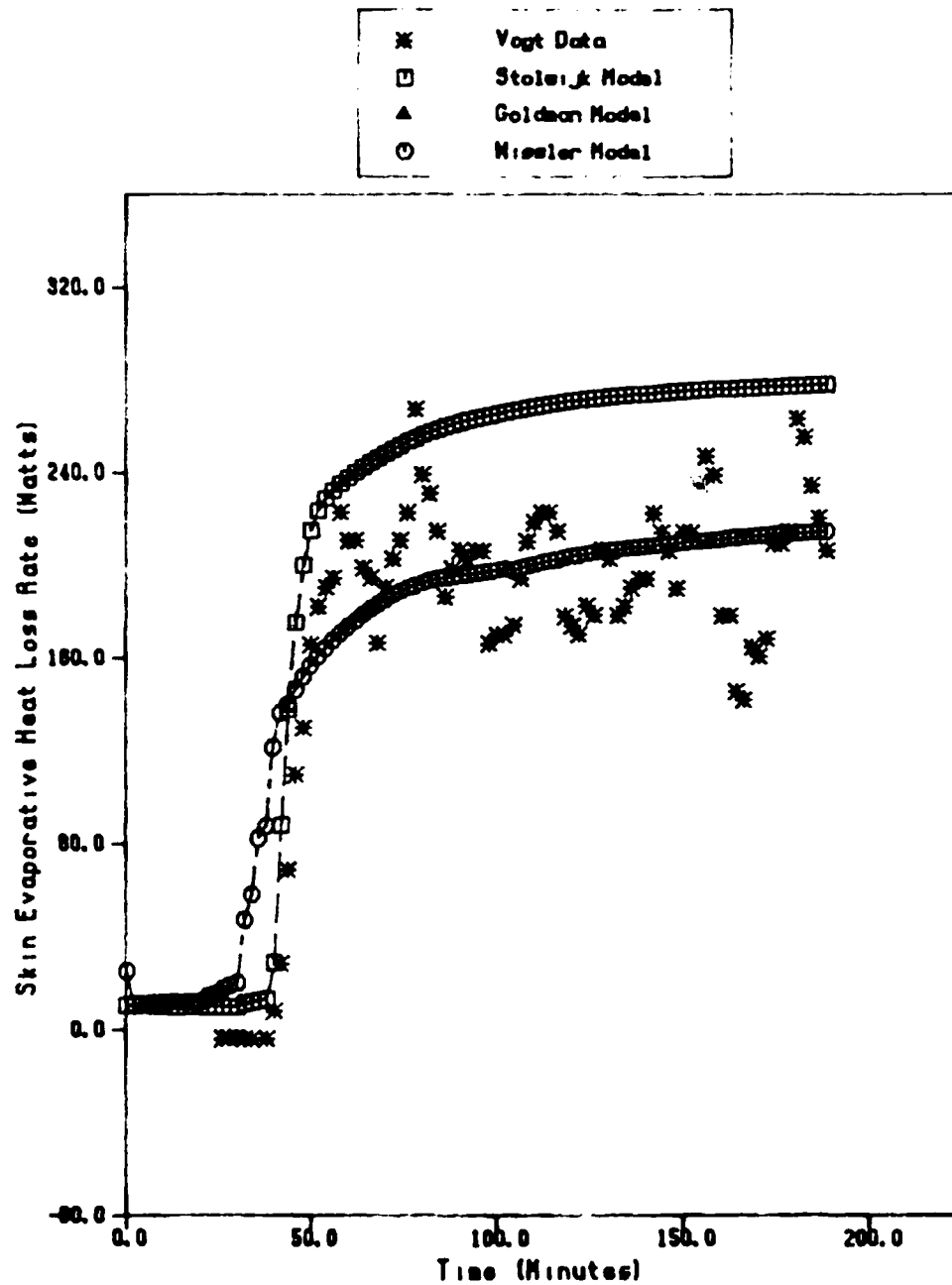
Rectal Temperature
As a Function of Time
For Subject OM Step Change to 42°C (DP=35°C)



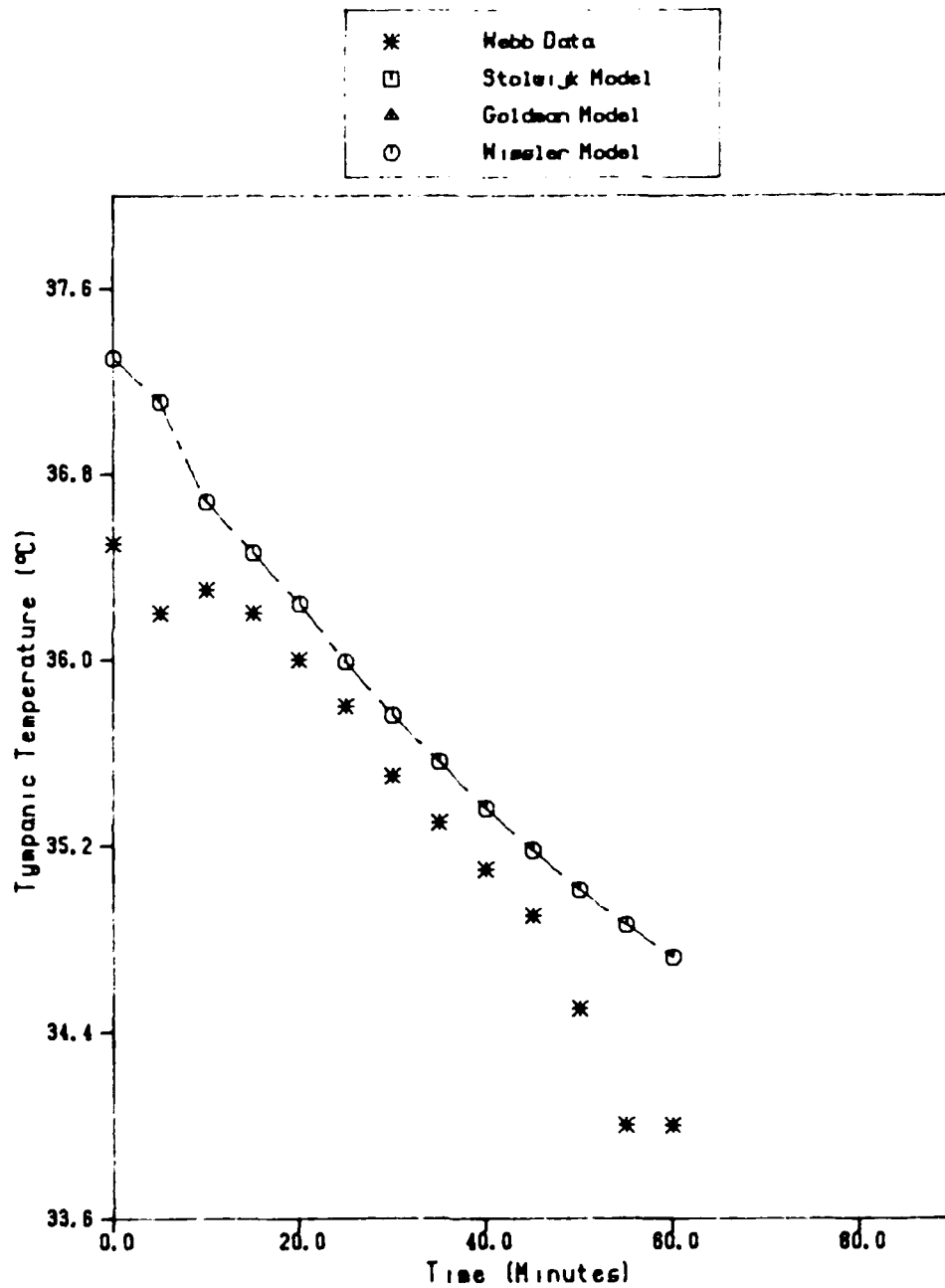
Mean Skin Temperature
As a Function of Time
For Subject OM Step Change to 42°C (DP=35°C)



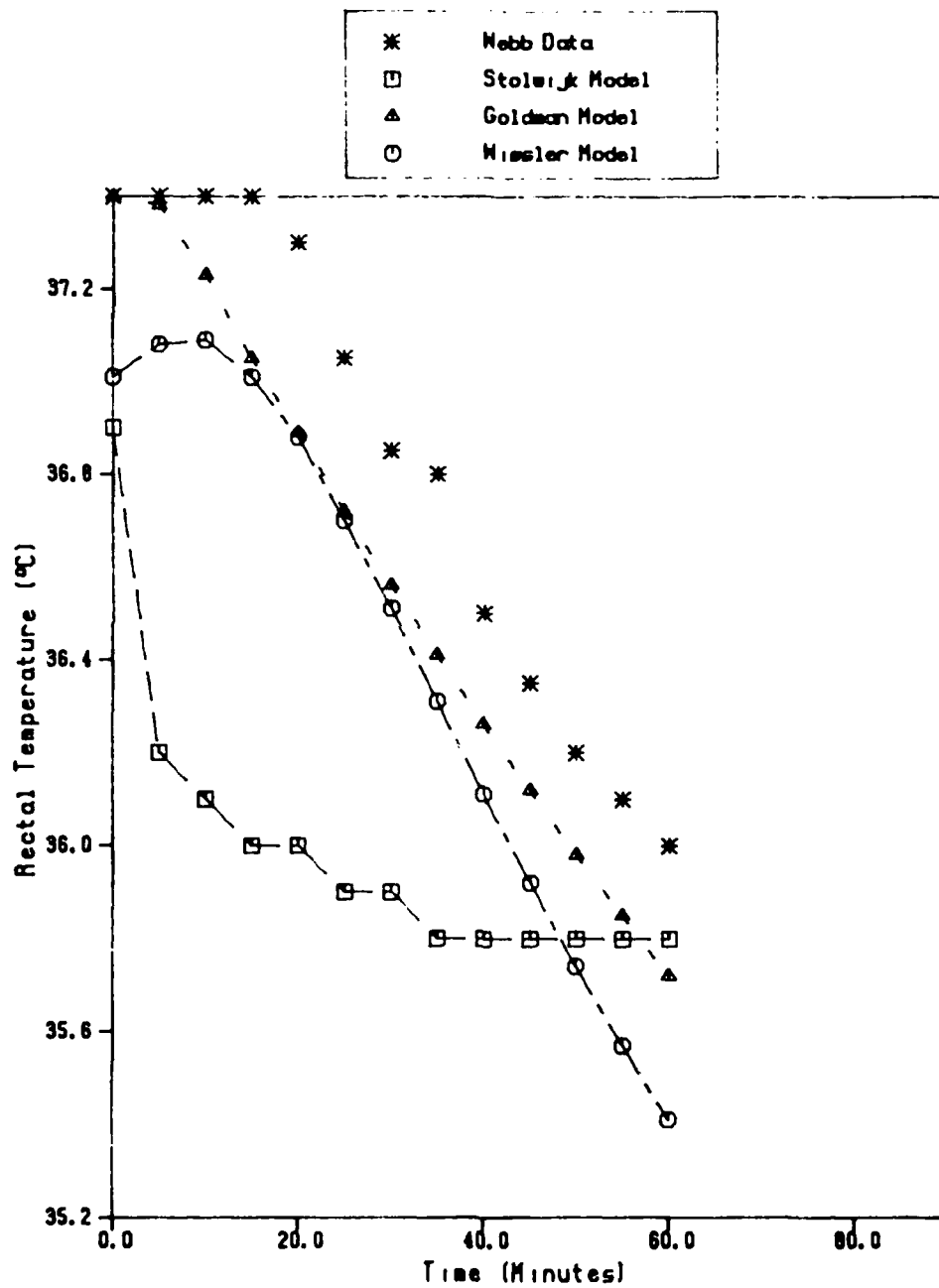
Skin Evaporative Heat Loss Rate
As a Function of Time
For Subject DM Step Change to 42°C (DP=35°C)



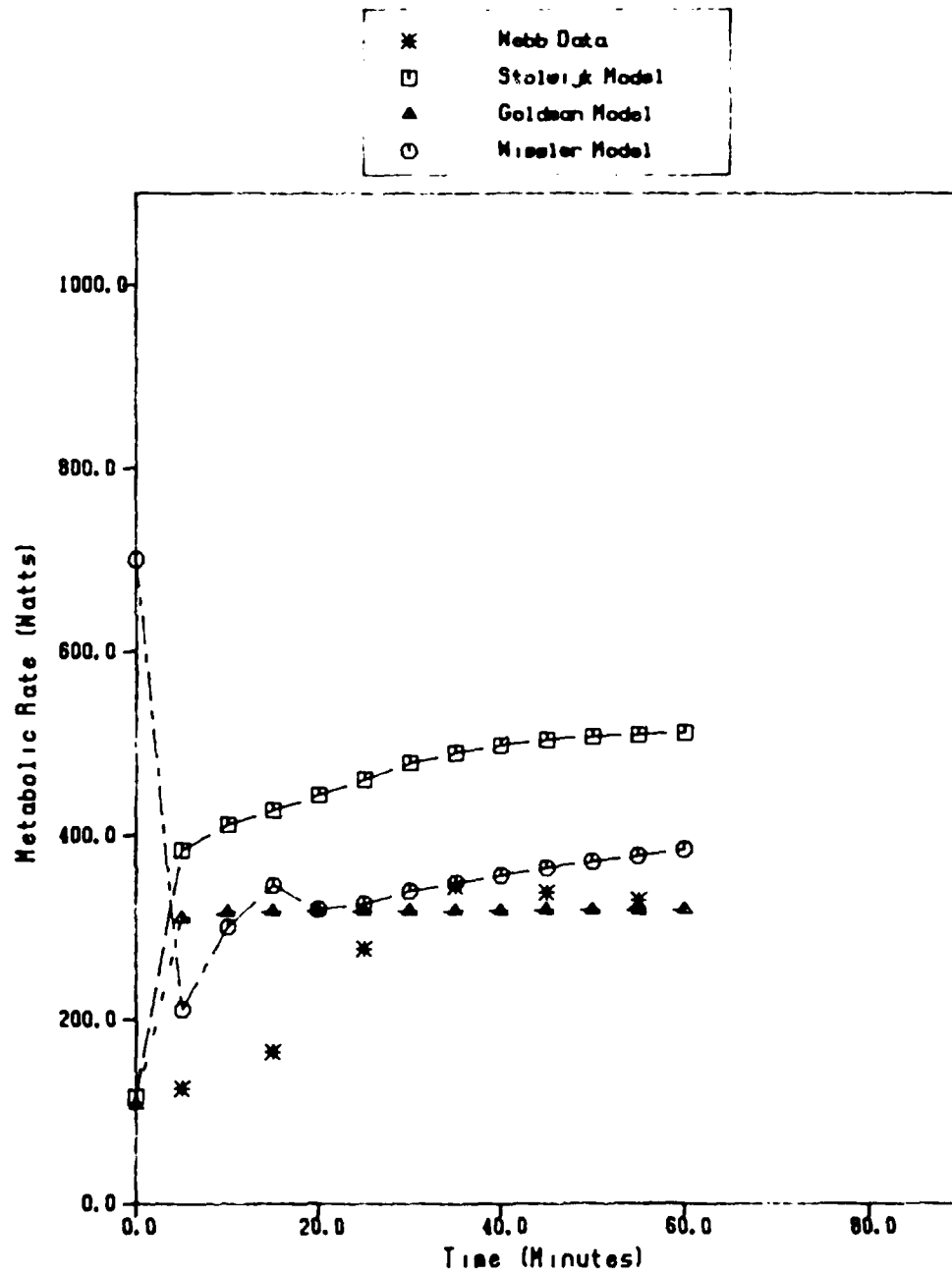
Tympanic Temperature
As a Function of Time
For Subject JA During an Immersion in 18 °C Water



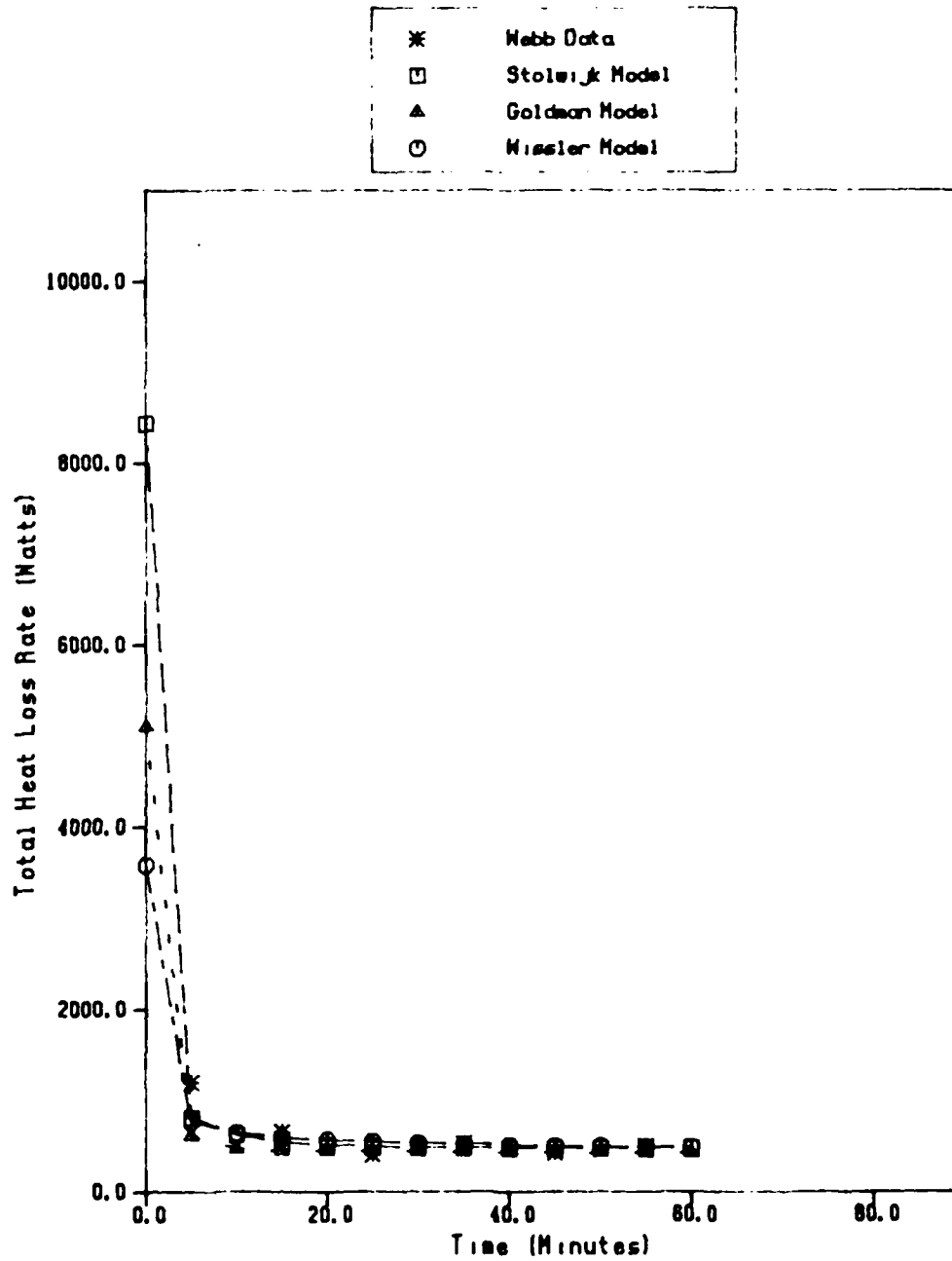
Rectal Temperature
As a Function of Time
For Subject JA During an Immersion in 18 °C Water



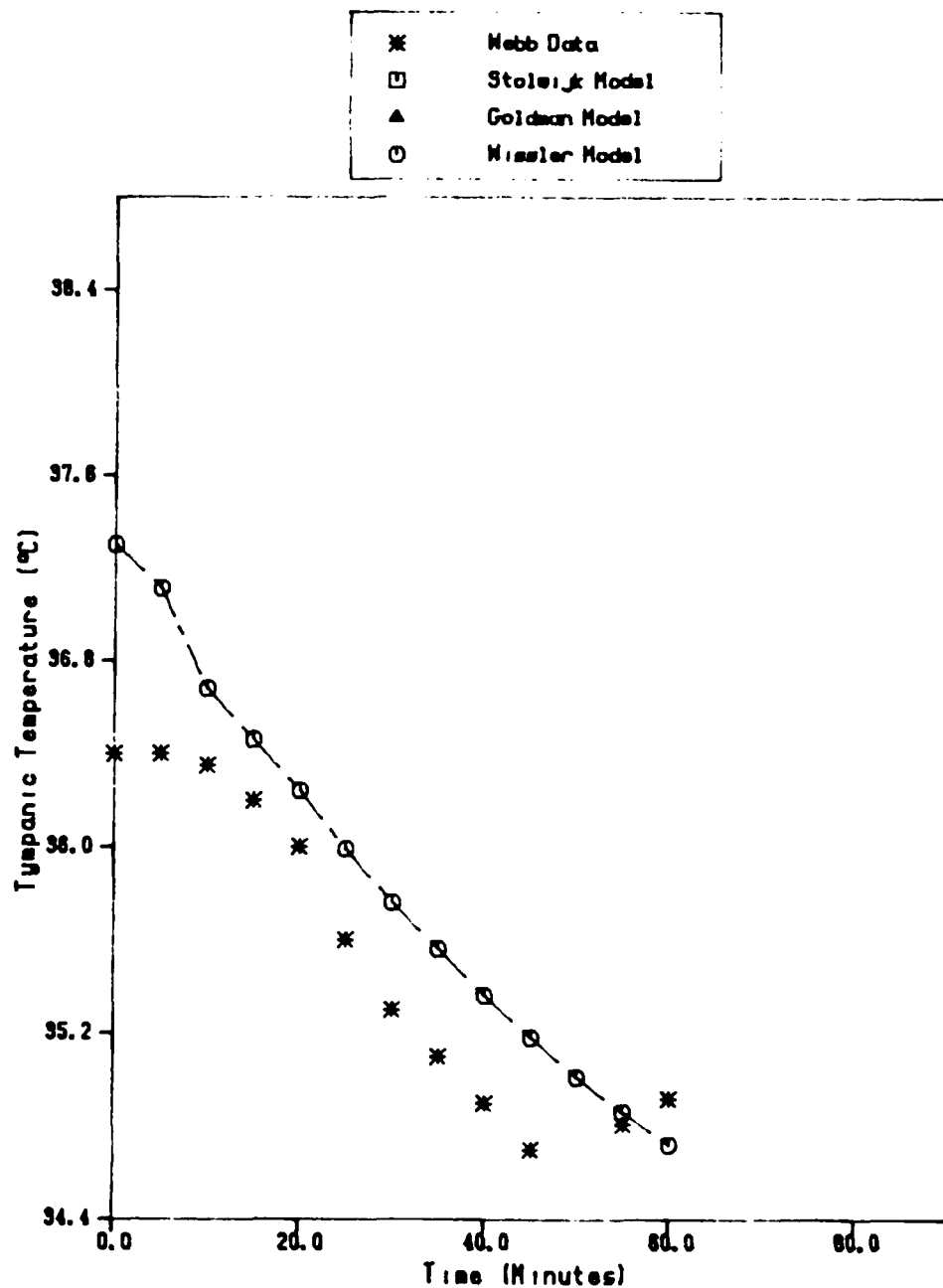
Metabolic Rate
As a Function of Time
For Subject JA During an Immersion in 18 °C Water



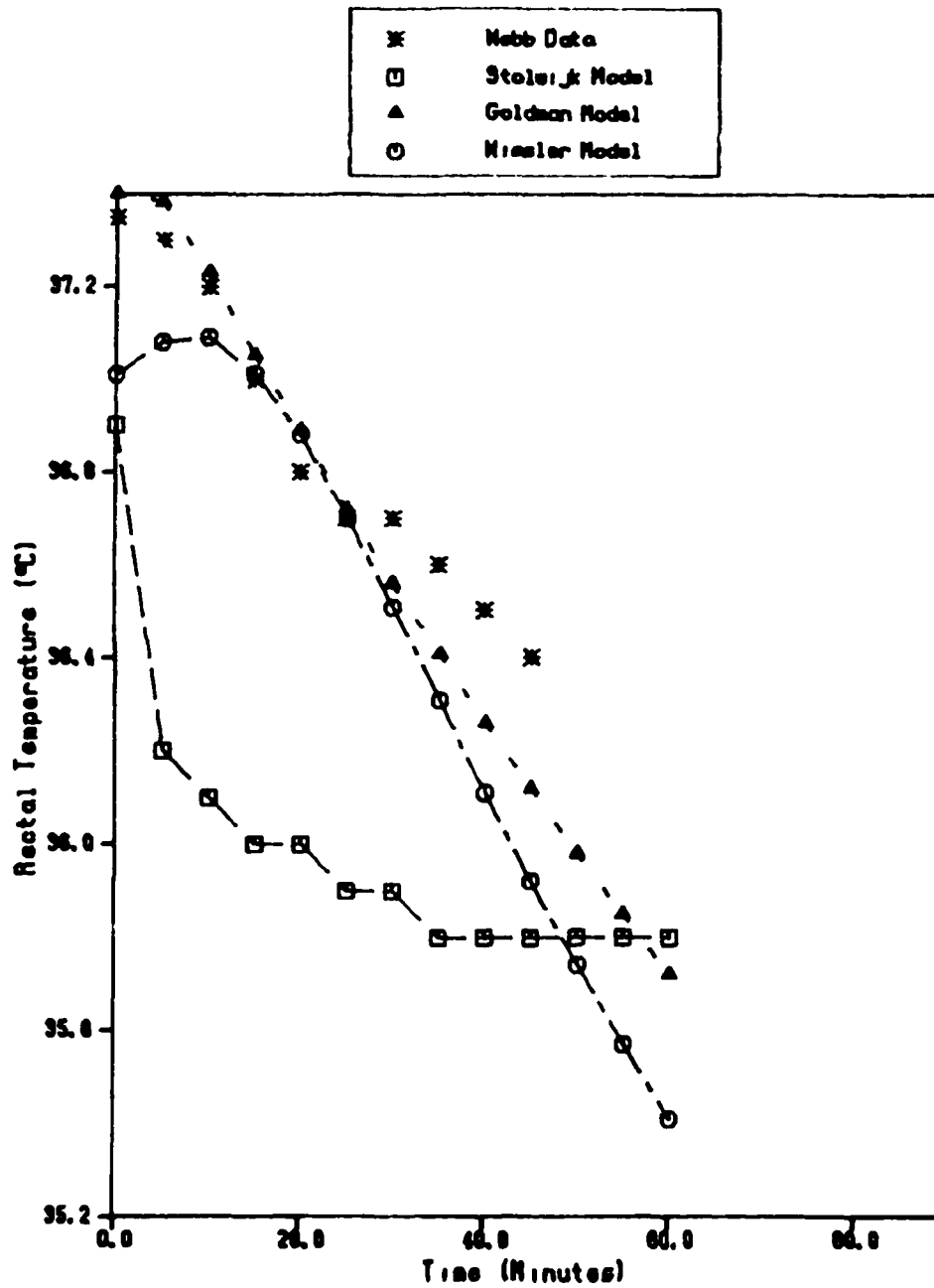
Total Heat Loss Rate
As a Function of Time
For Subject JA During an Immersion in 18 °C Water



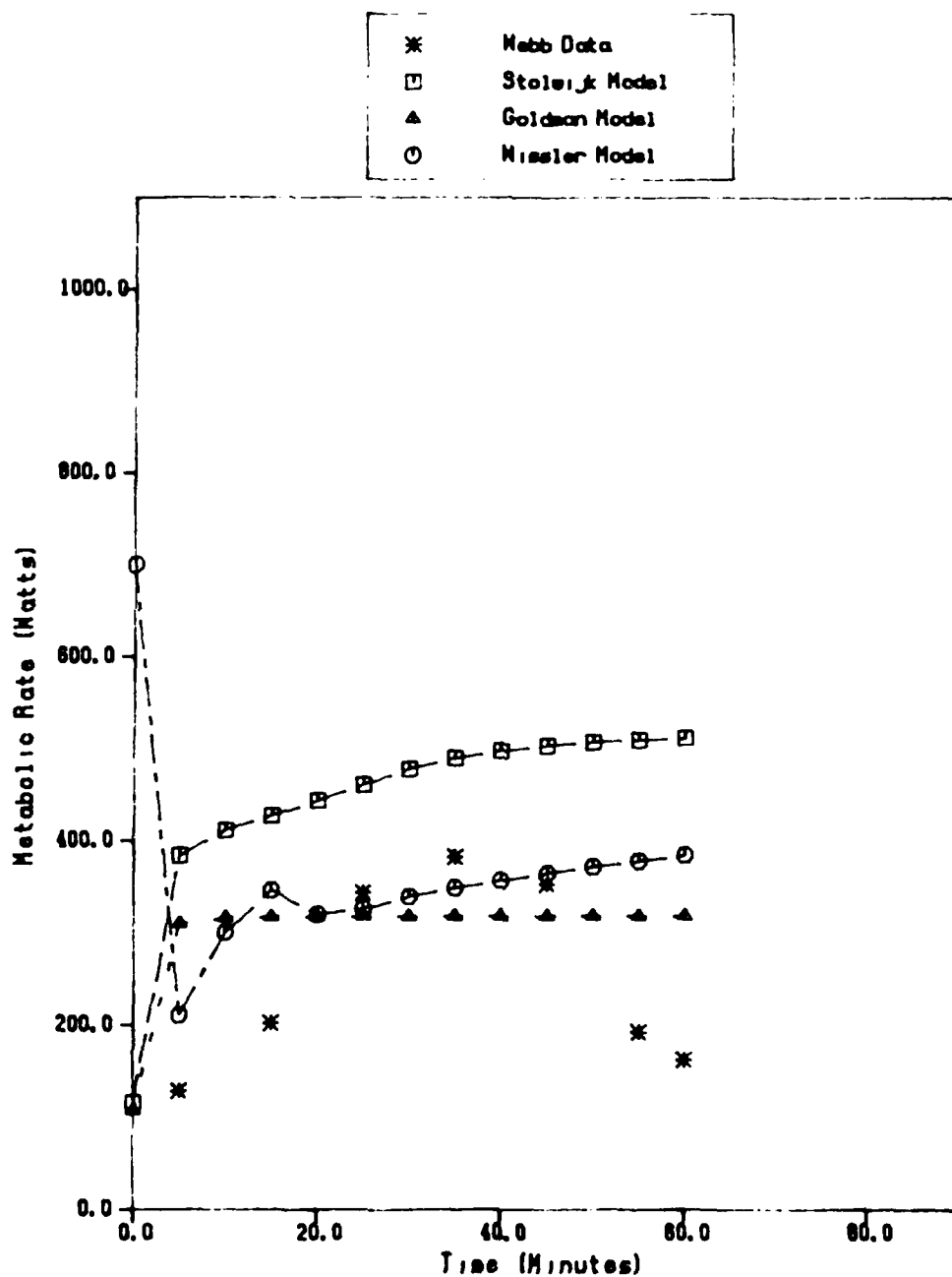
Tympanic Temperature
As a Function of Time
For Subject JA During an Immersion in 18 °C Water



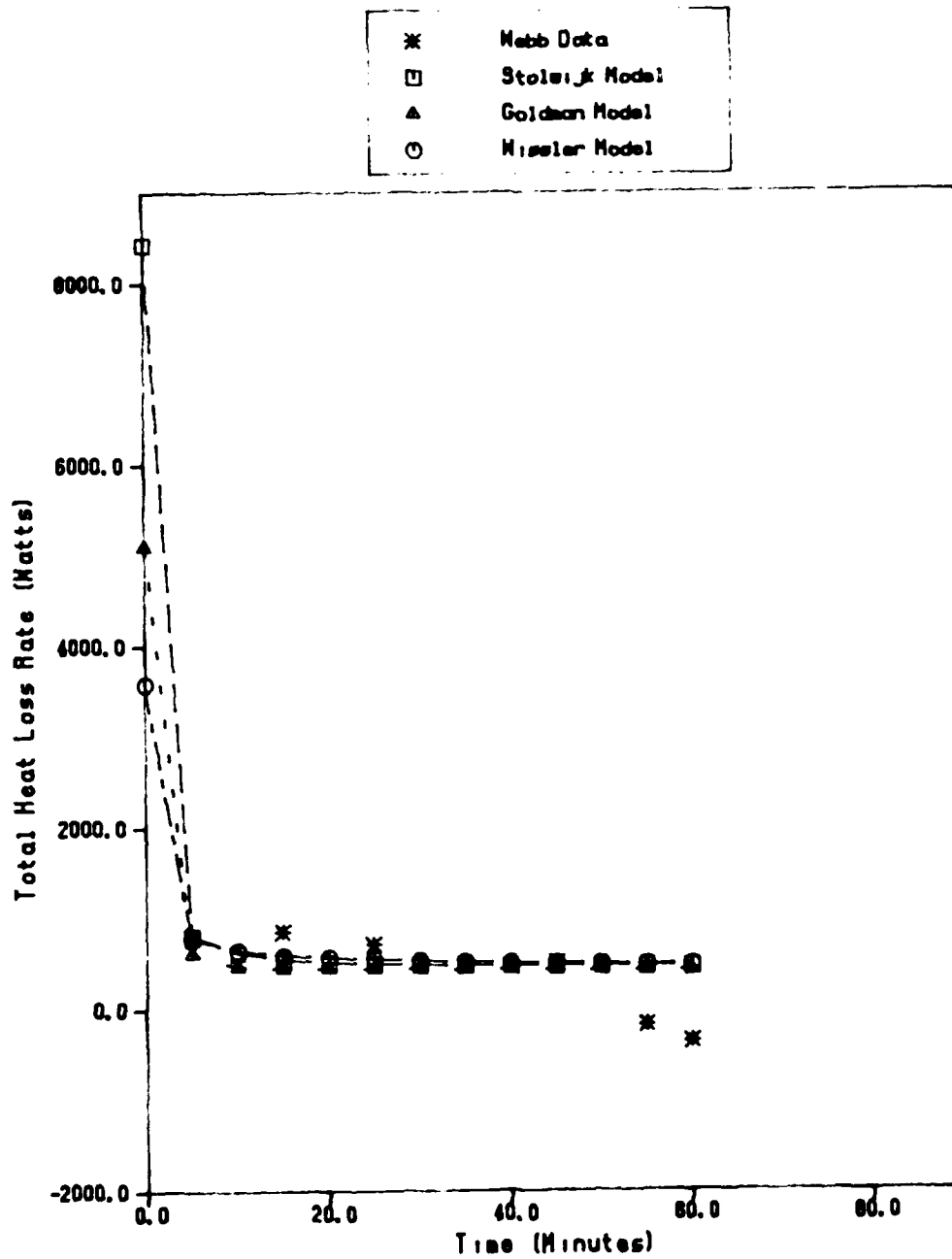
Rectal Temperature
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



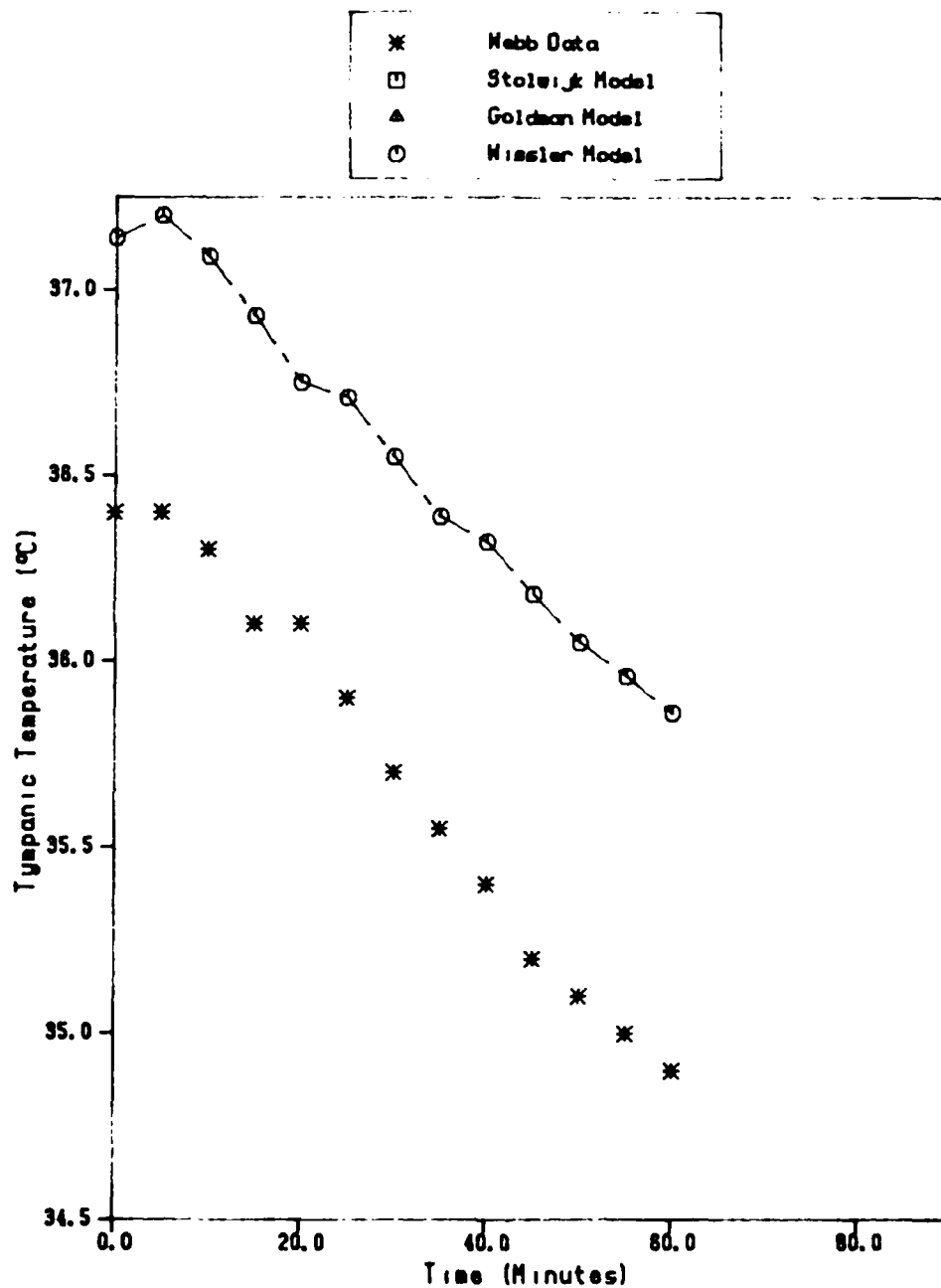
Metabolic Rate
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



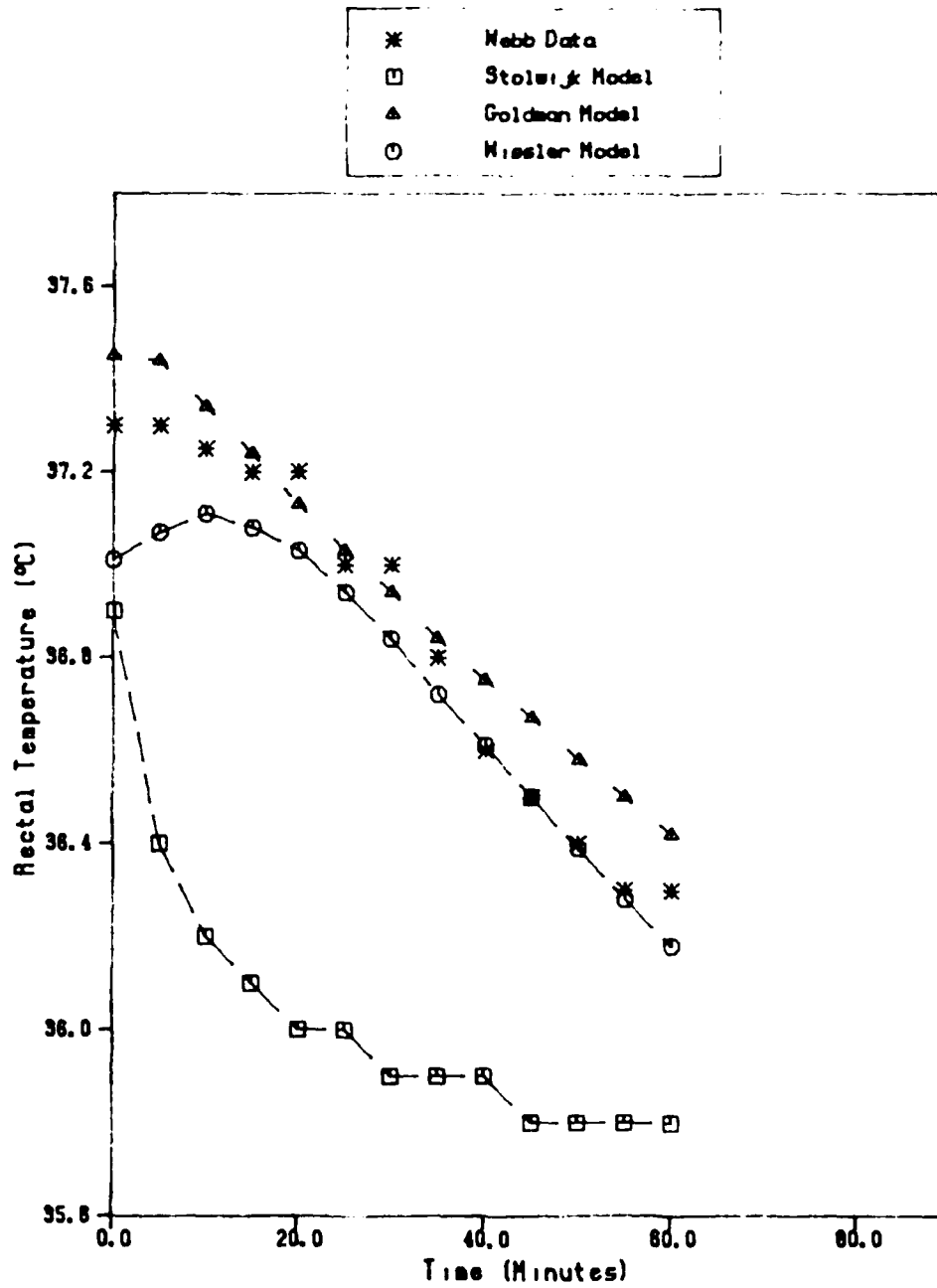
Total Heat Loss Rate
As a Function of Time
For Subject JA During an Immersion in 18 °C Water



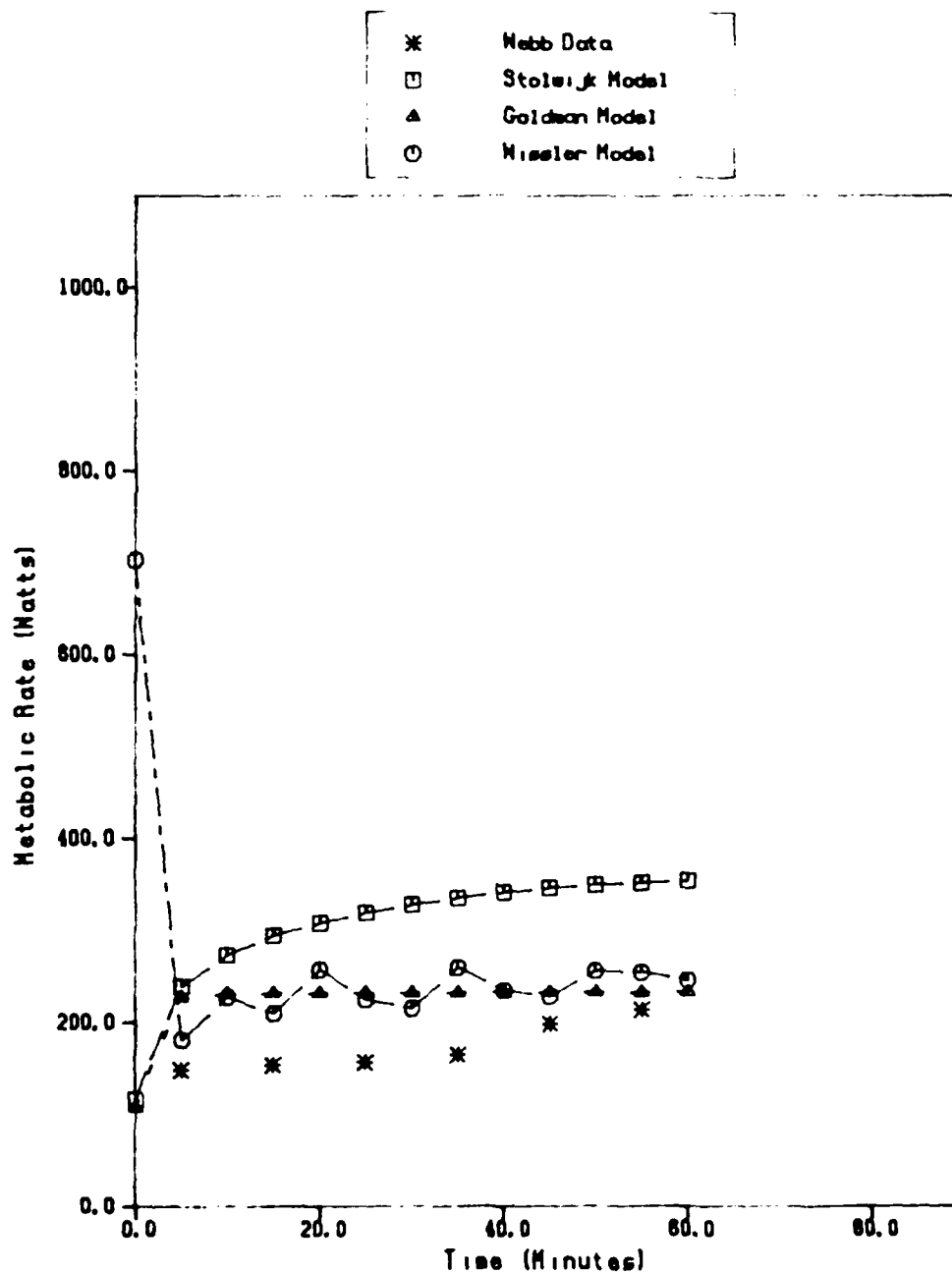
Tympanic Temperature
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



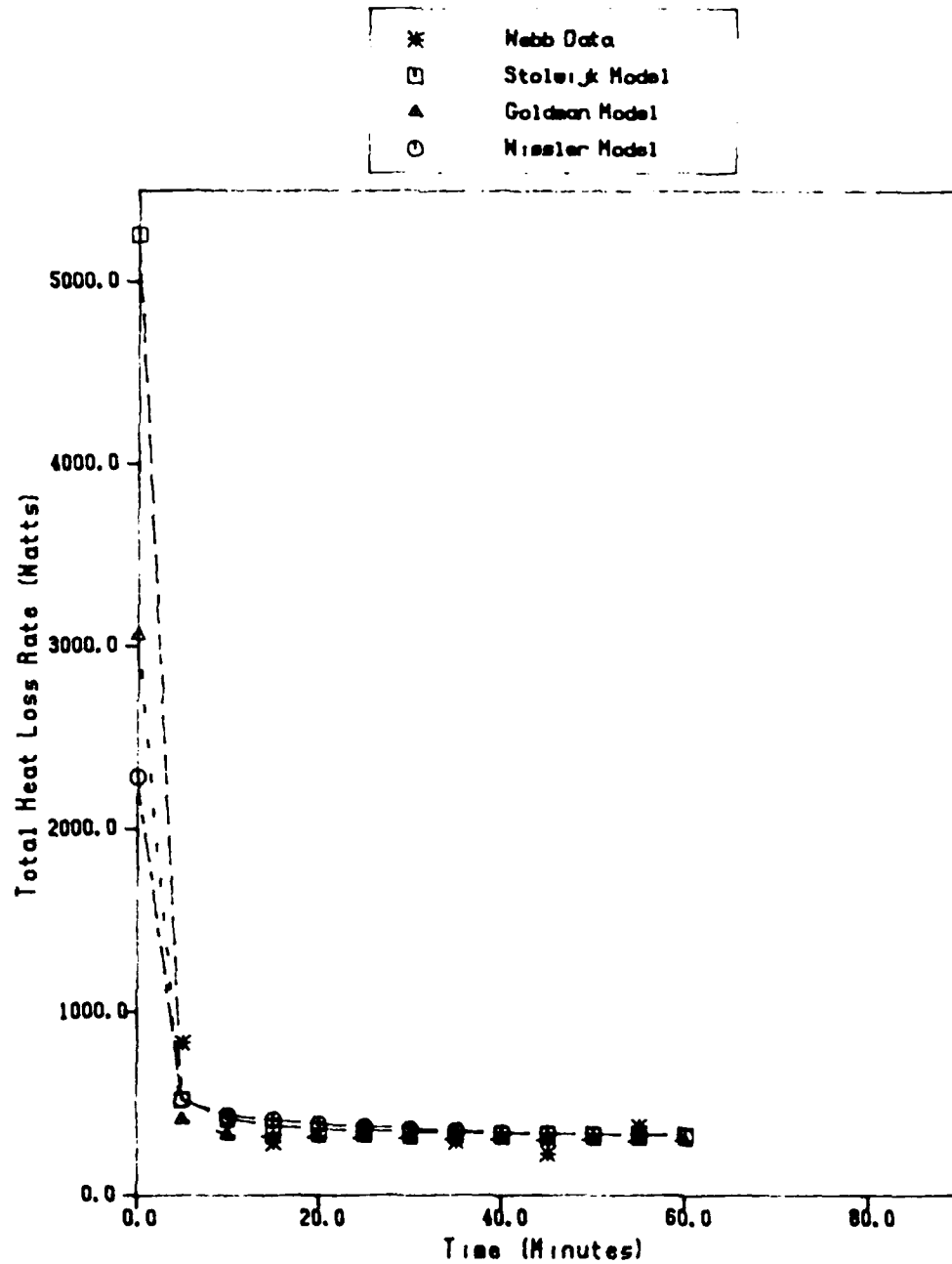
Rectal Temperature
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



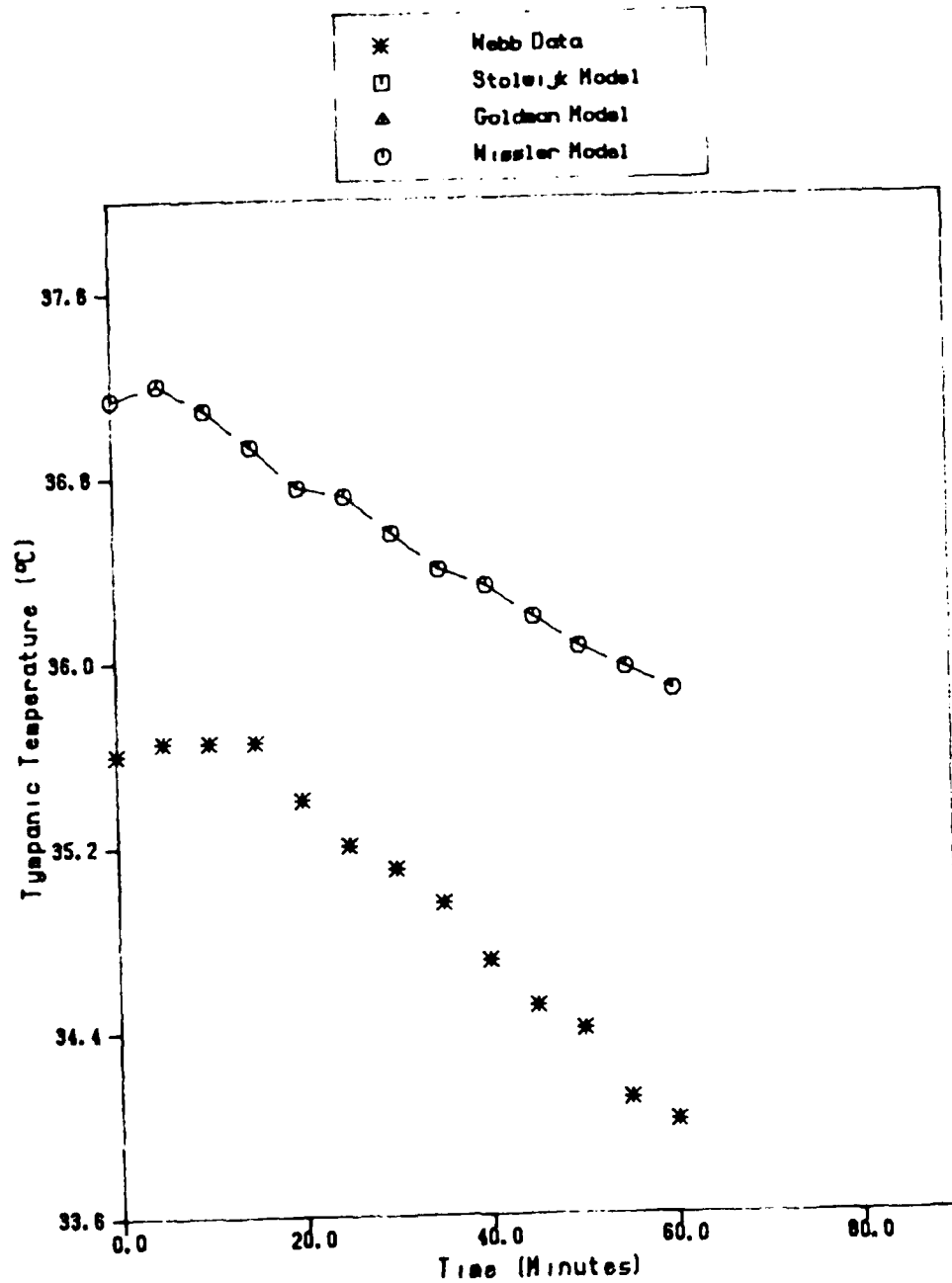
Metabolic Rate
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



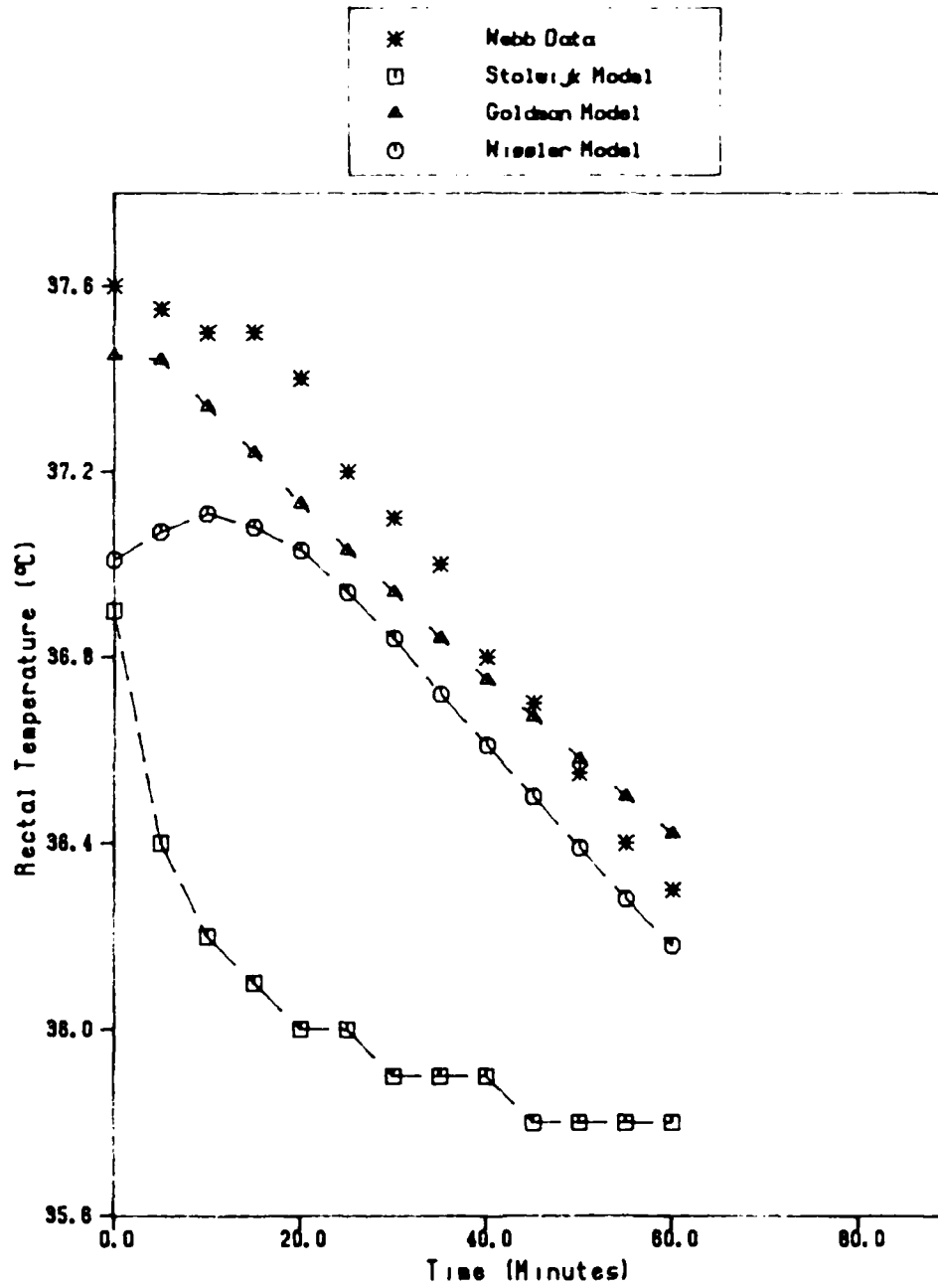
Total Heat Loss Rate
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



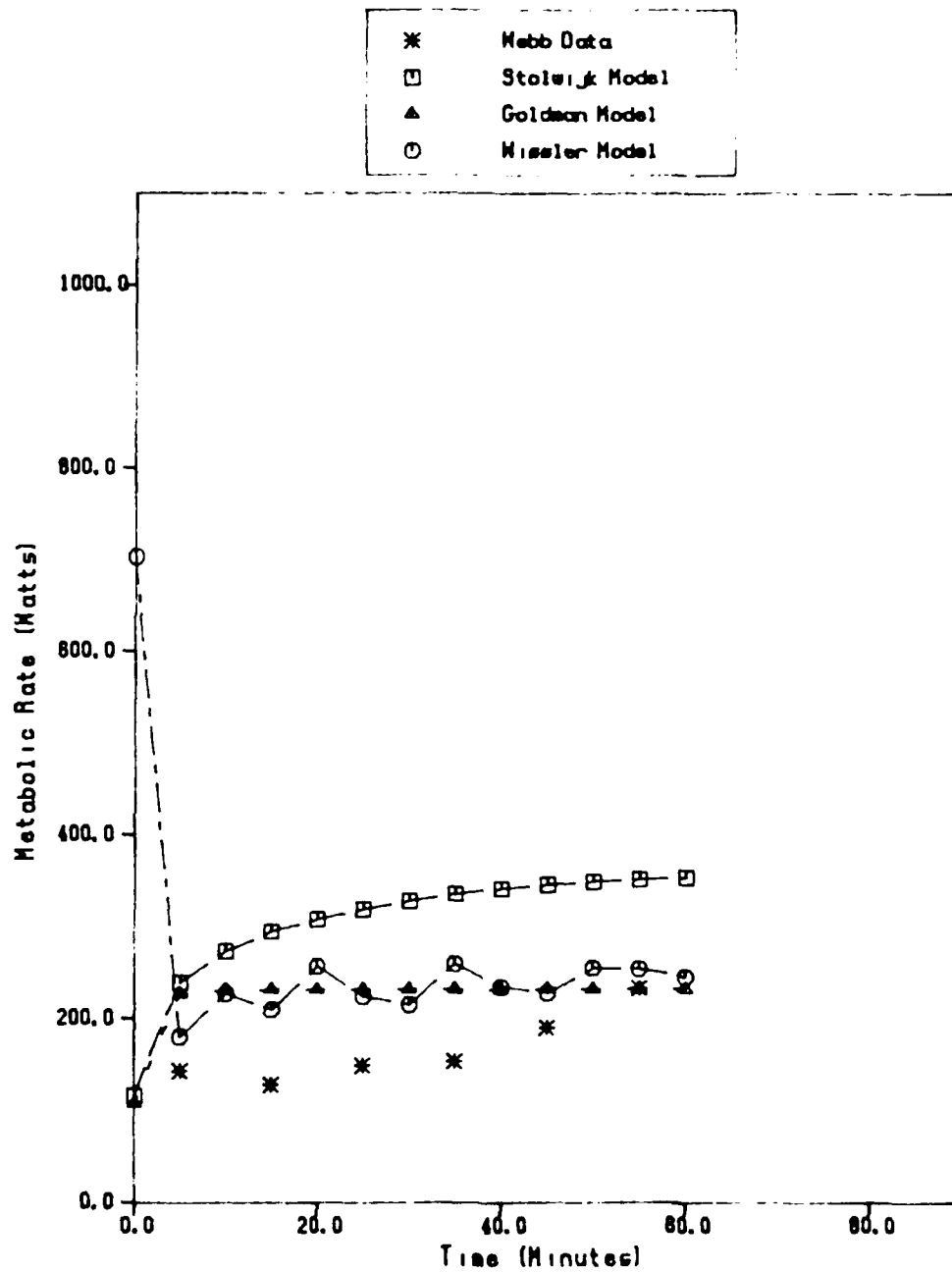
Tympanic Temperature
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



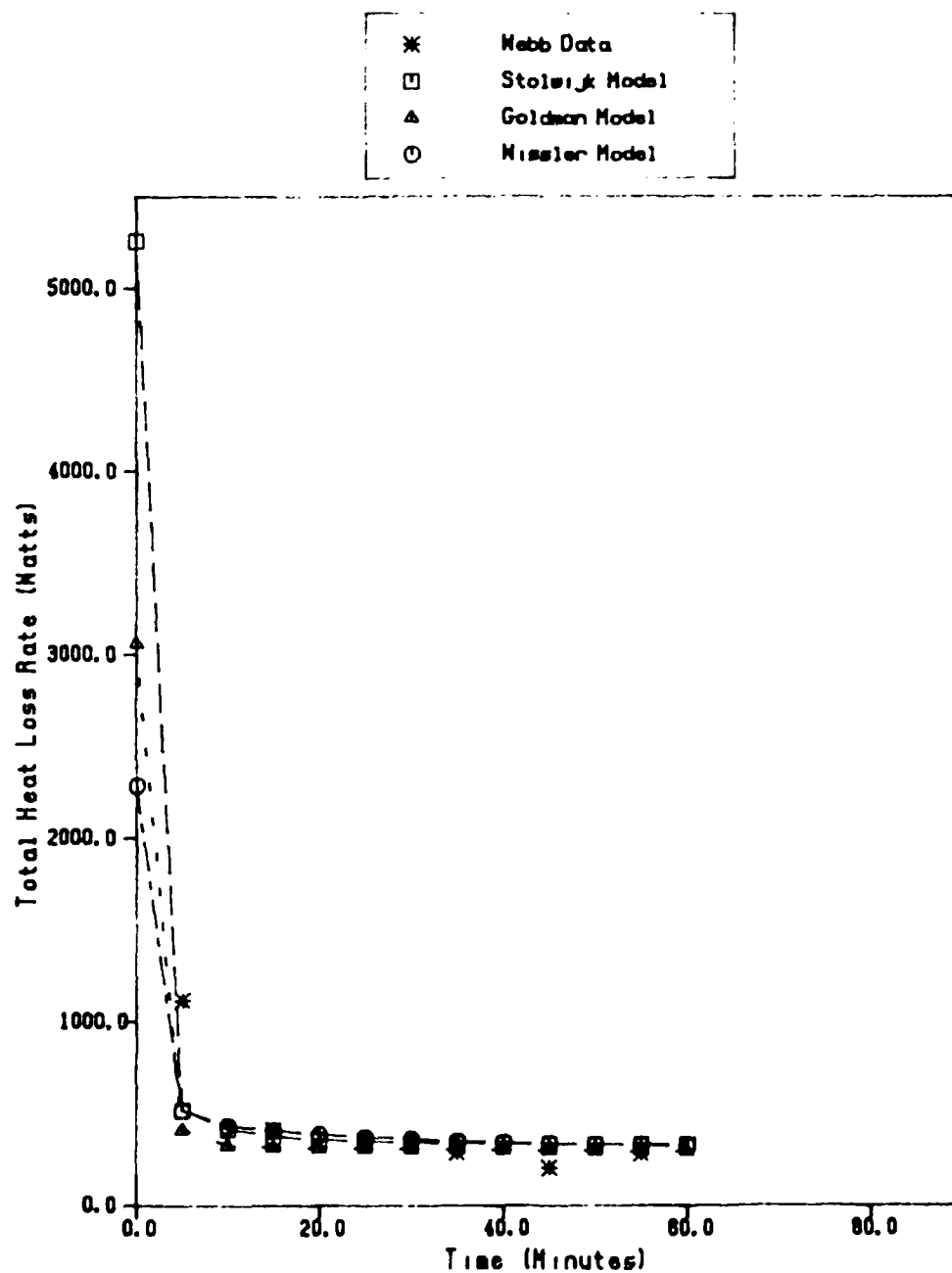
Rectal Temperature
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



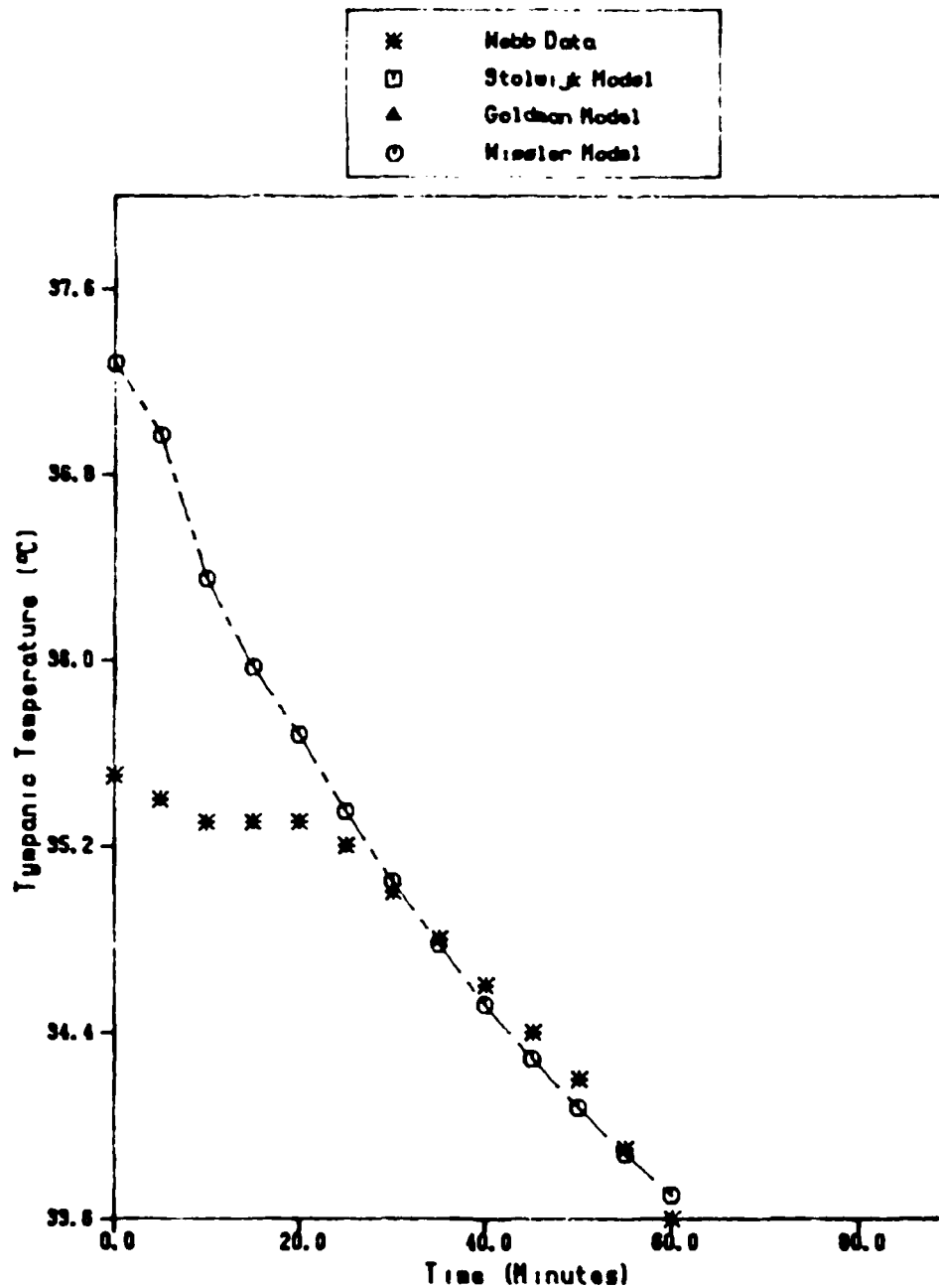
Metabolic Rate
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



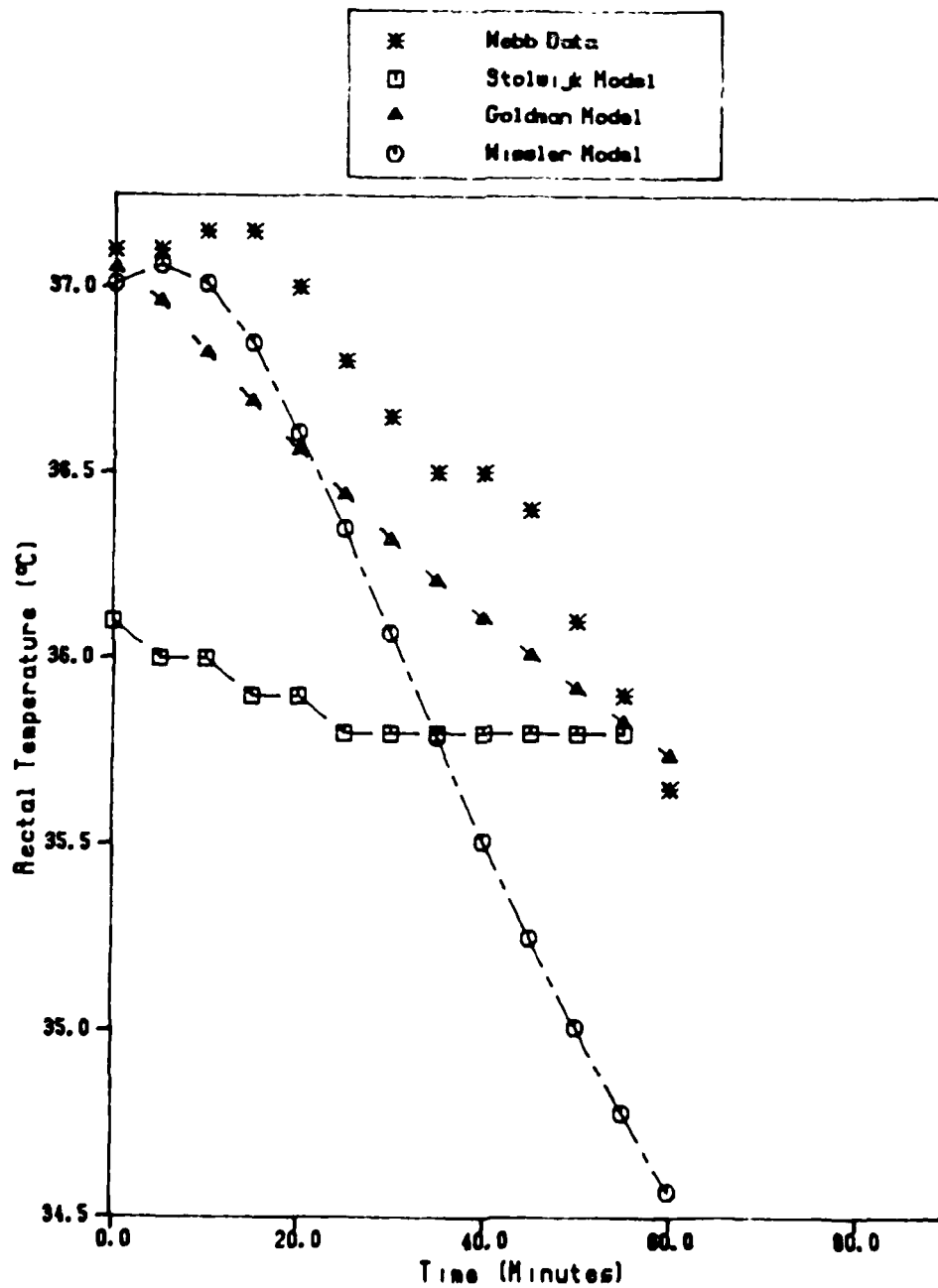
Total Heat Loss Rate
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



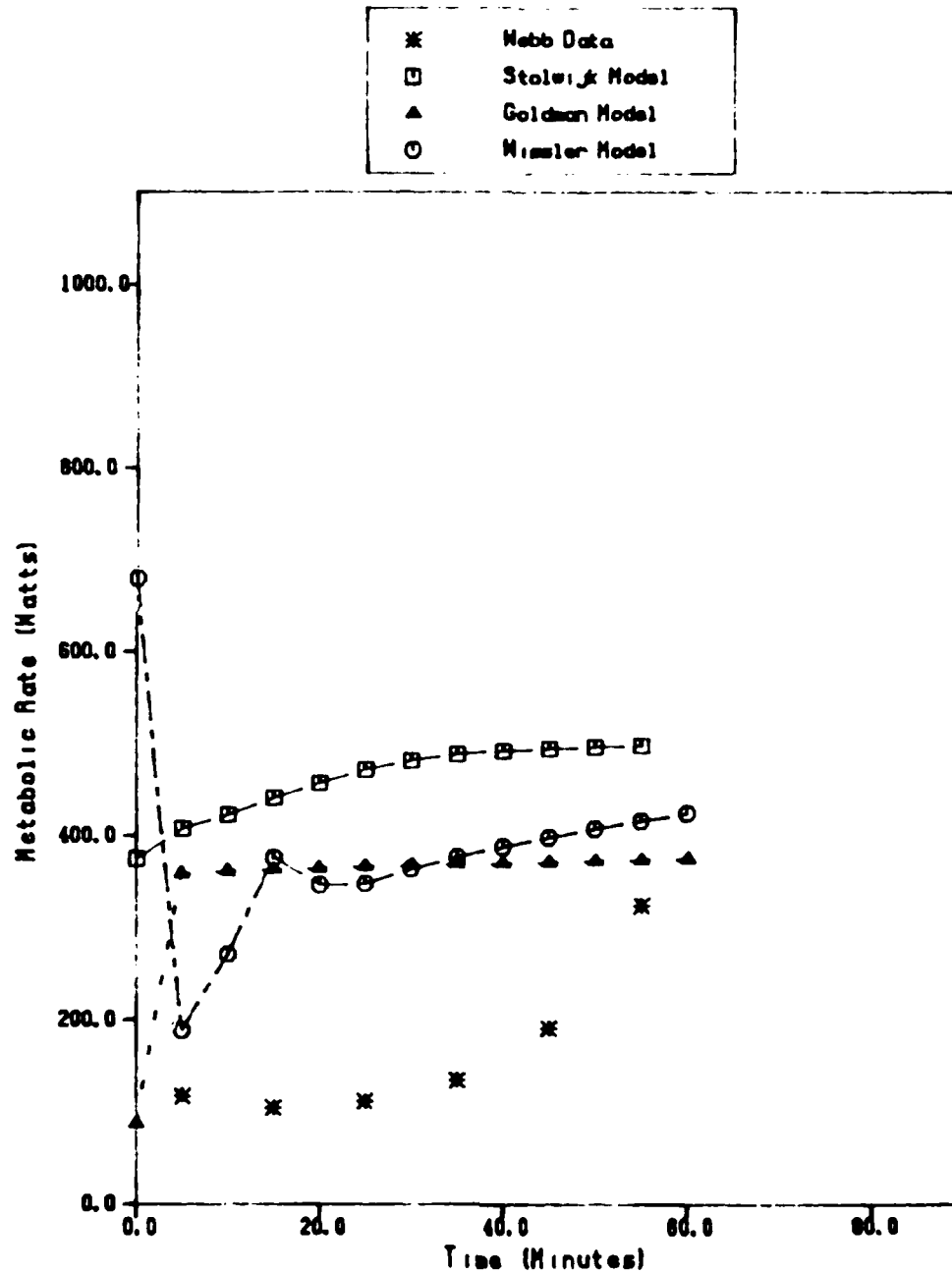
Tympanic Temperature
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



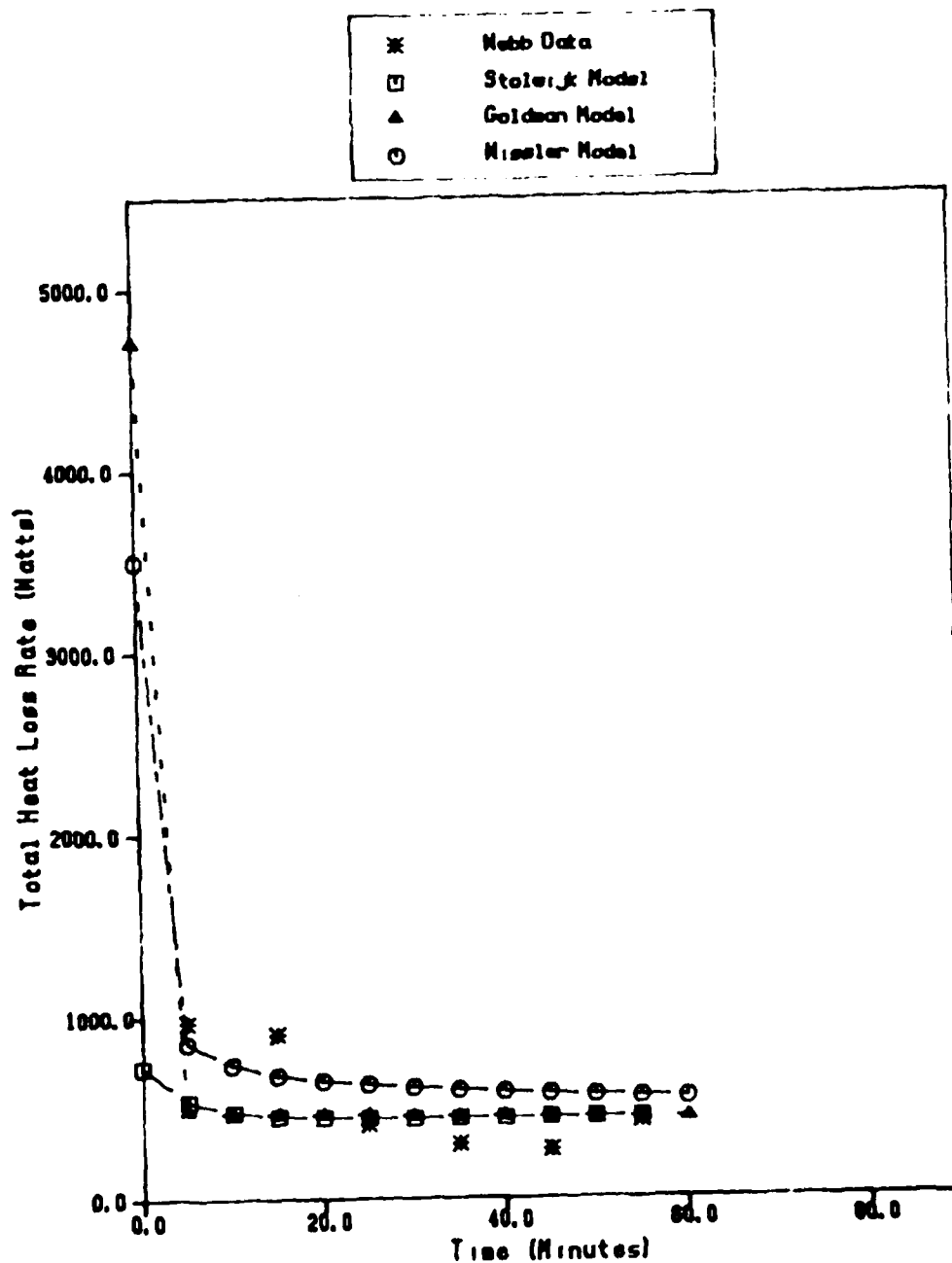
Rectal Temperature
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



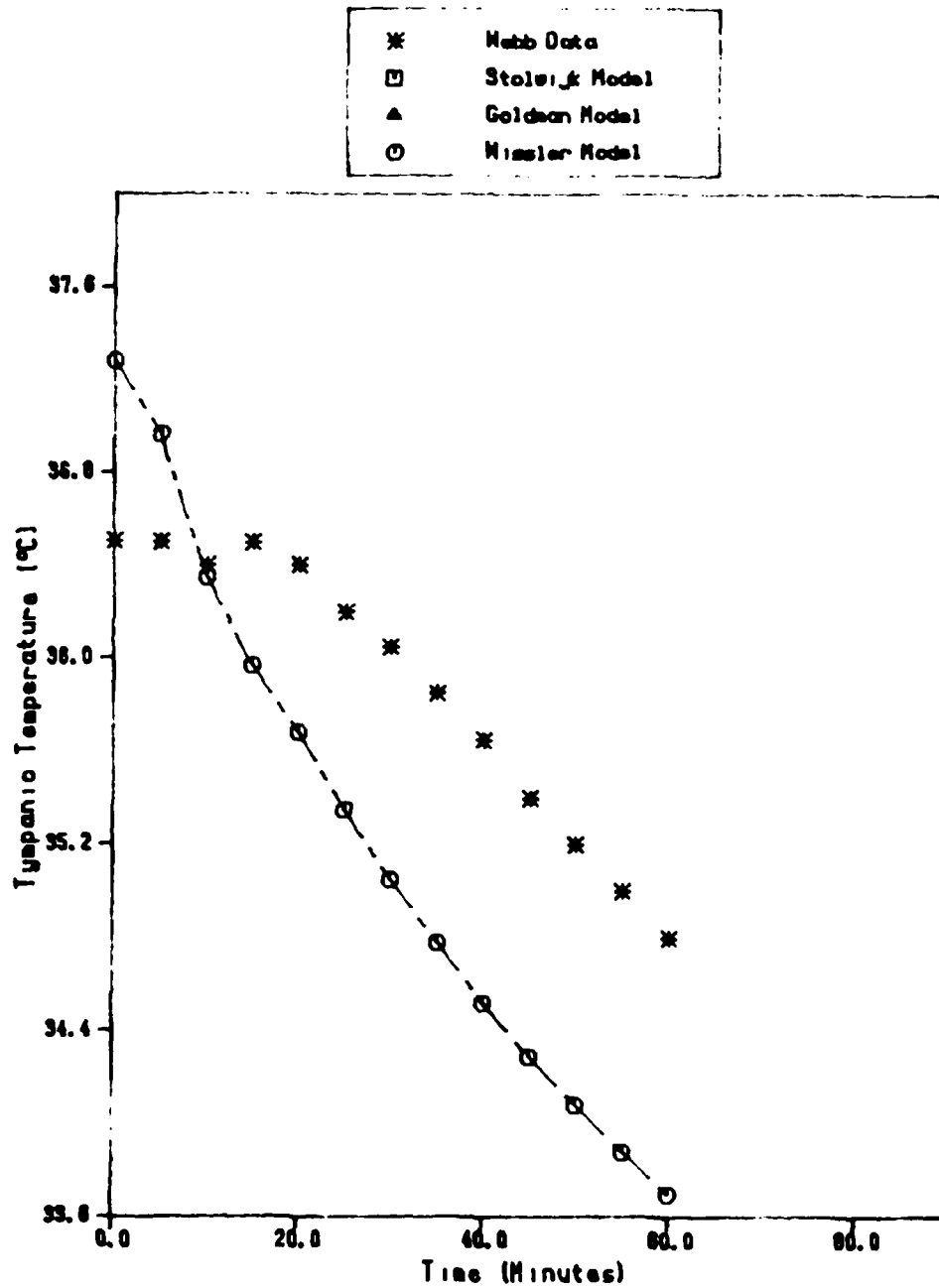
Metabolic Rate
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



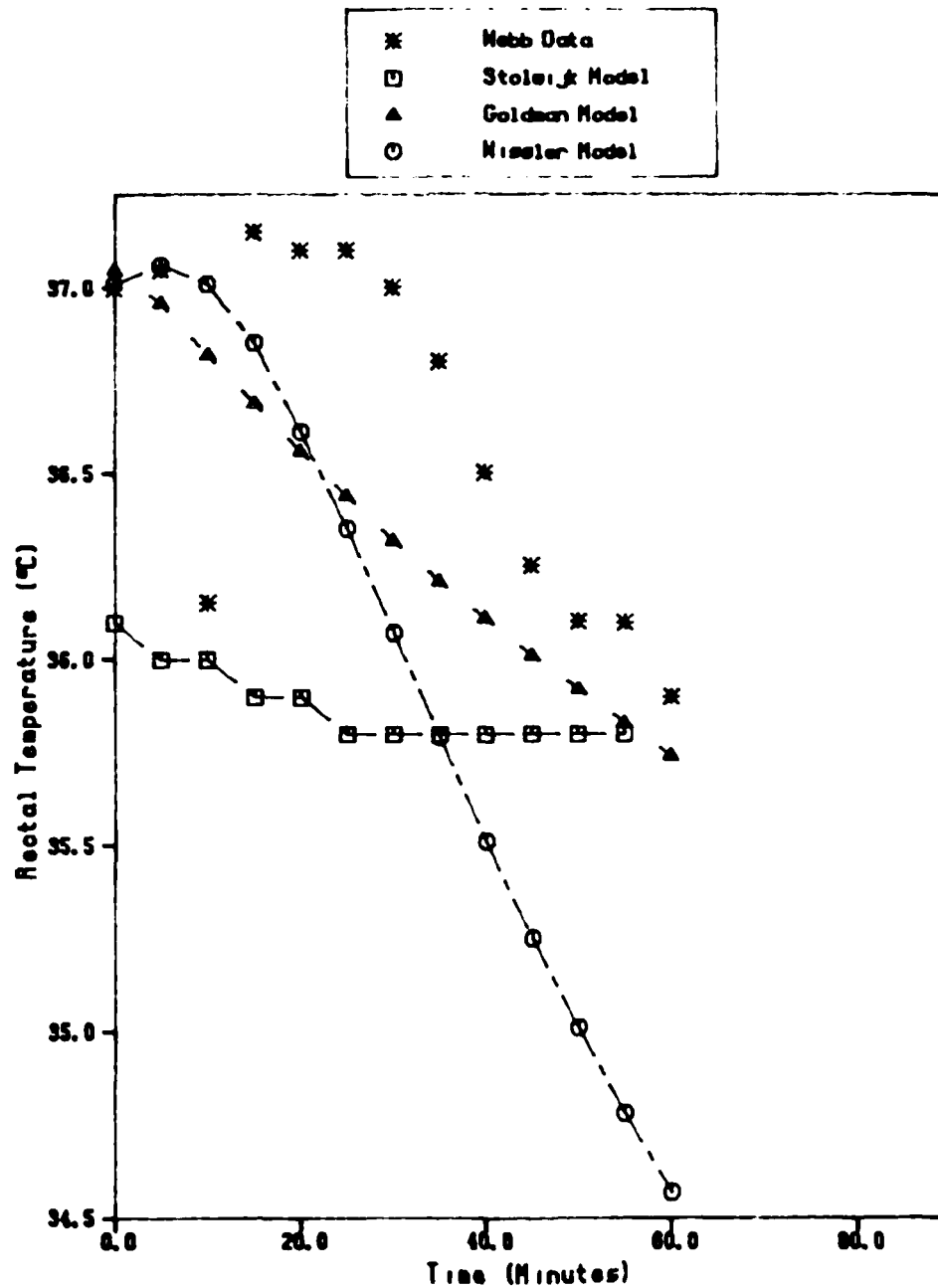
Total Heat Loss Rate
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



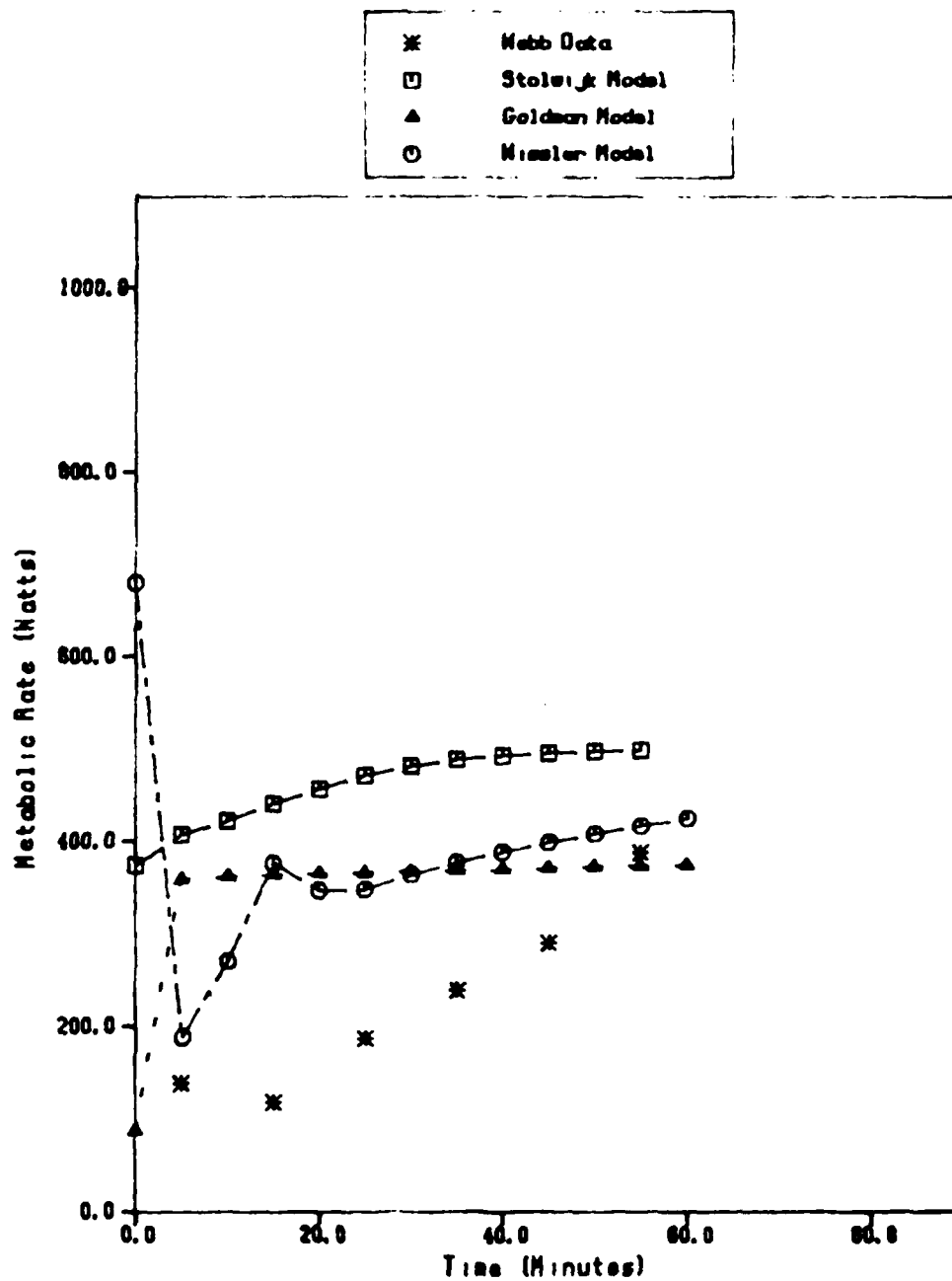
Tympanic Temperature
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



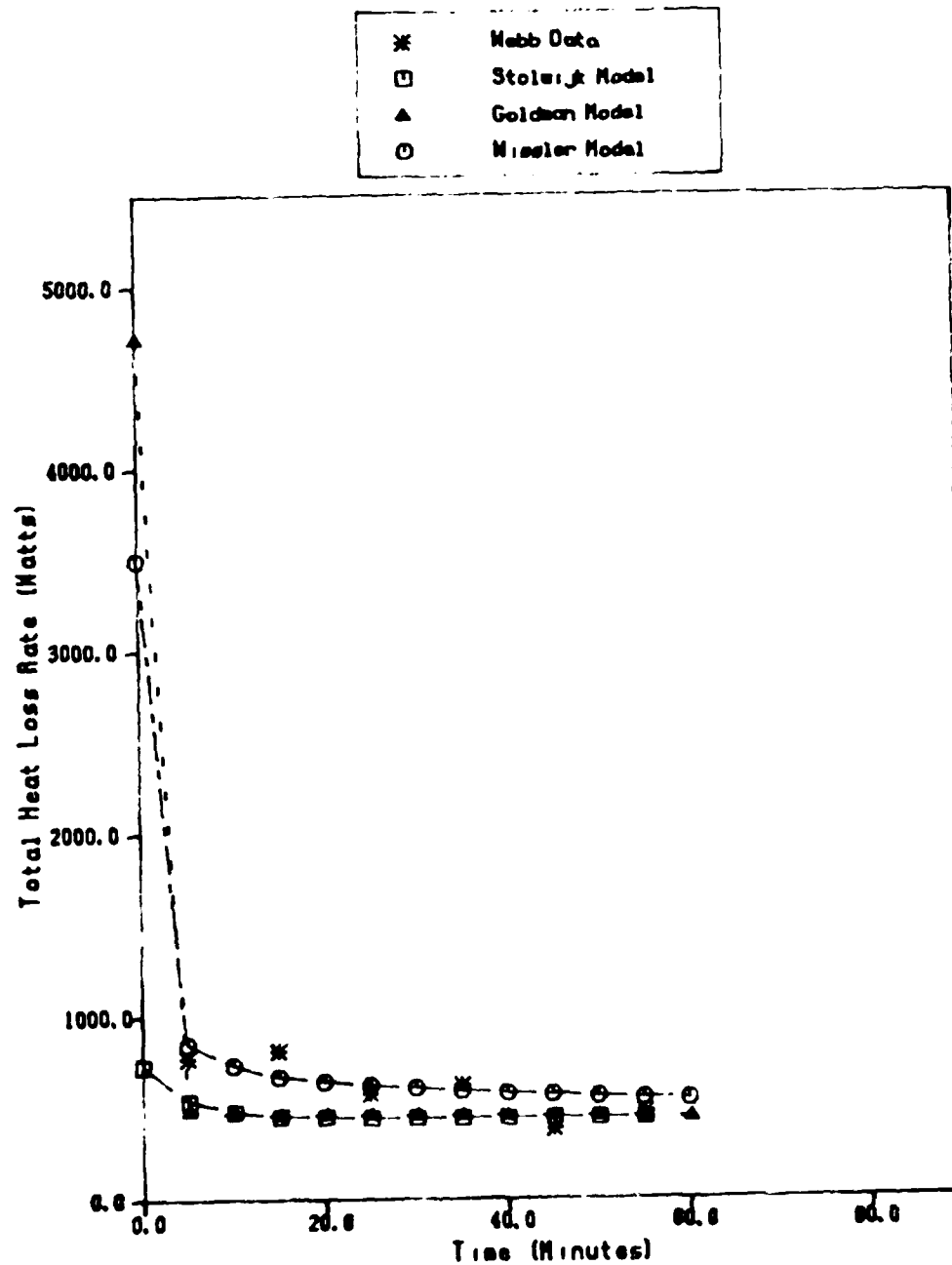
Rectal Temperature
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



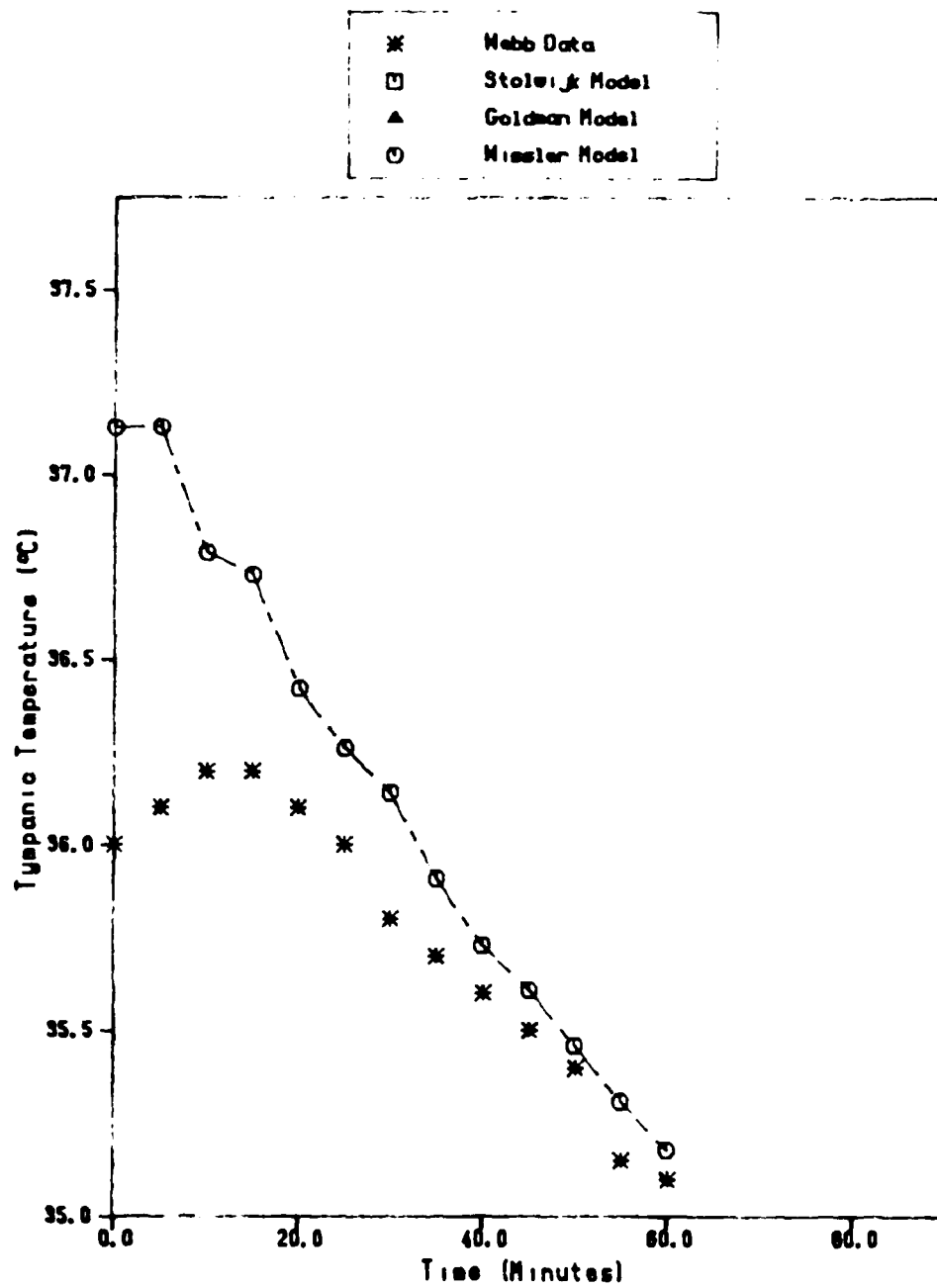
Metabolic Rate
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



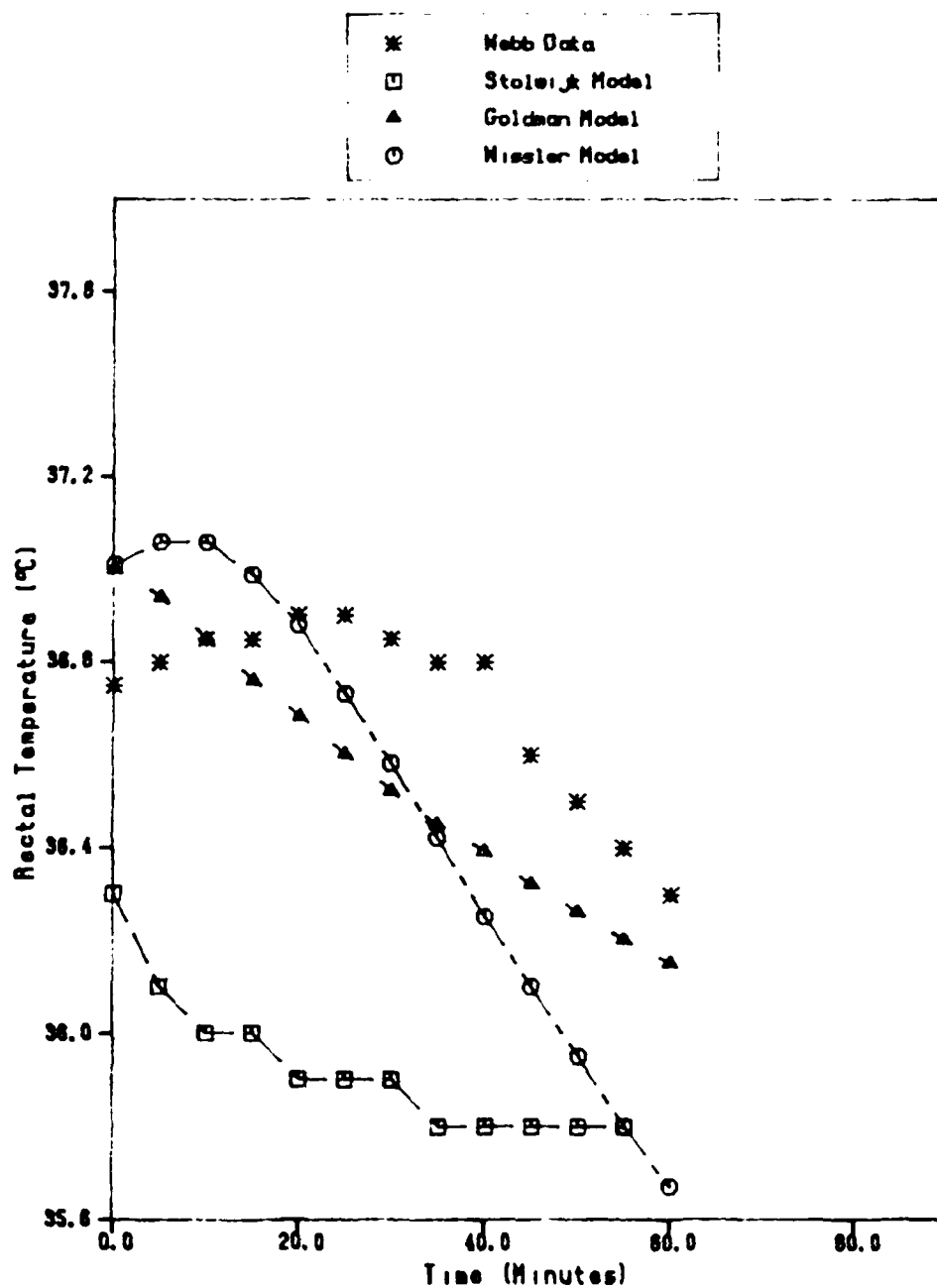
Total Heat Loss Rate
As a Function of Time
For Subject JR During an Immersion in 18 °C Water



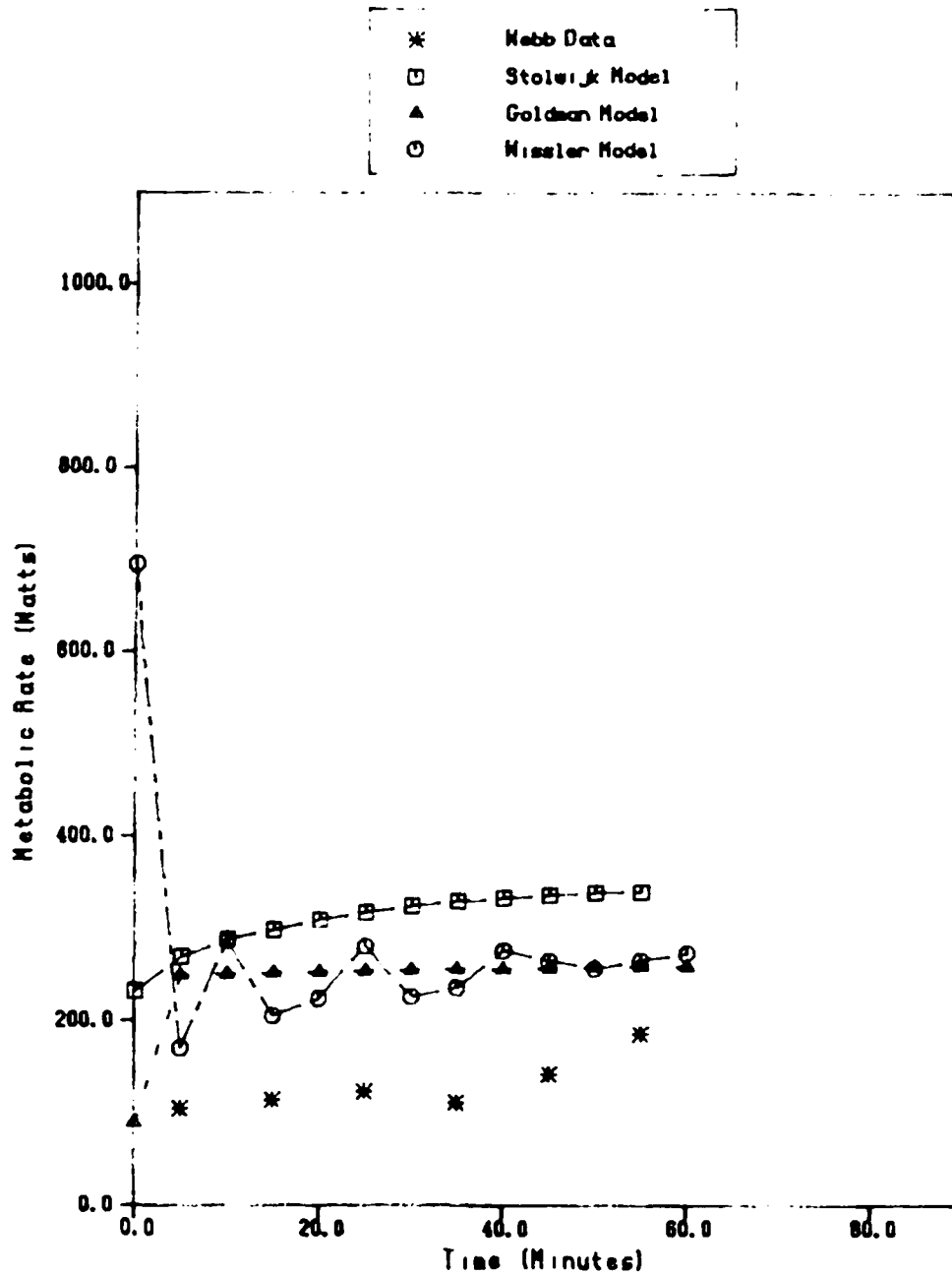
Tympanic Temperature
As a Function of Time
For Subject JR During an Immersion in 24 °C Water



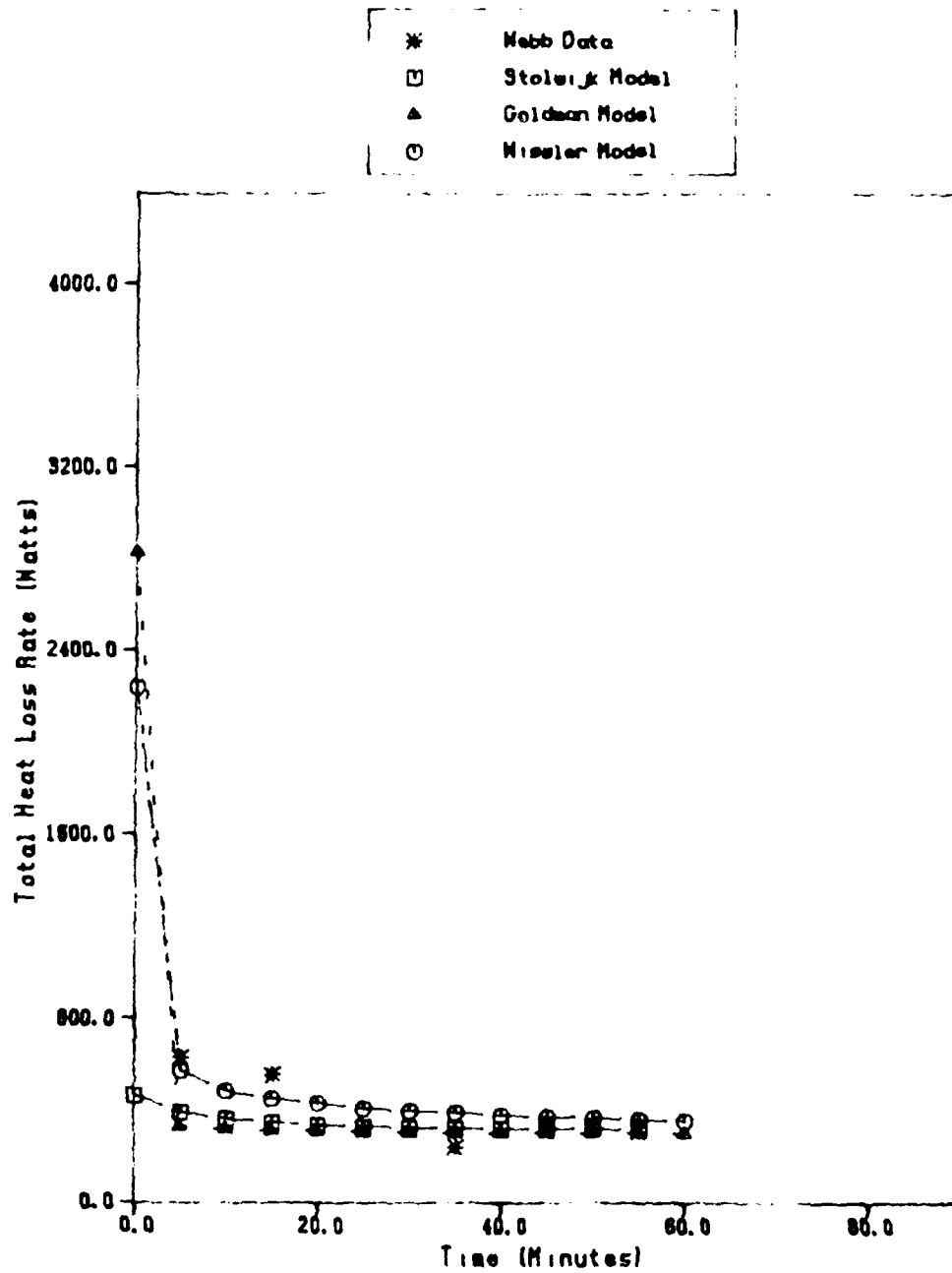
Rectal Temperature
As a Function of Time
For Subject JR During an Immersion in 24 °C Water



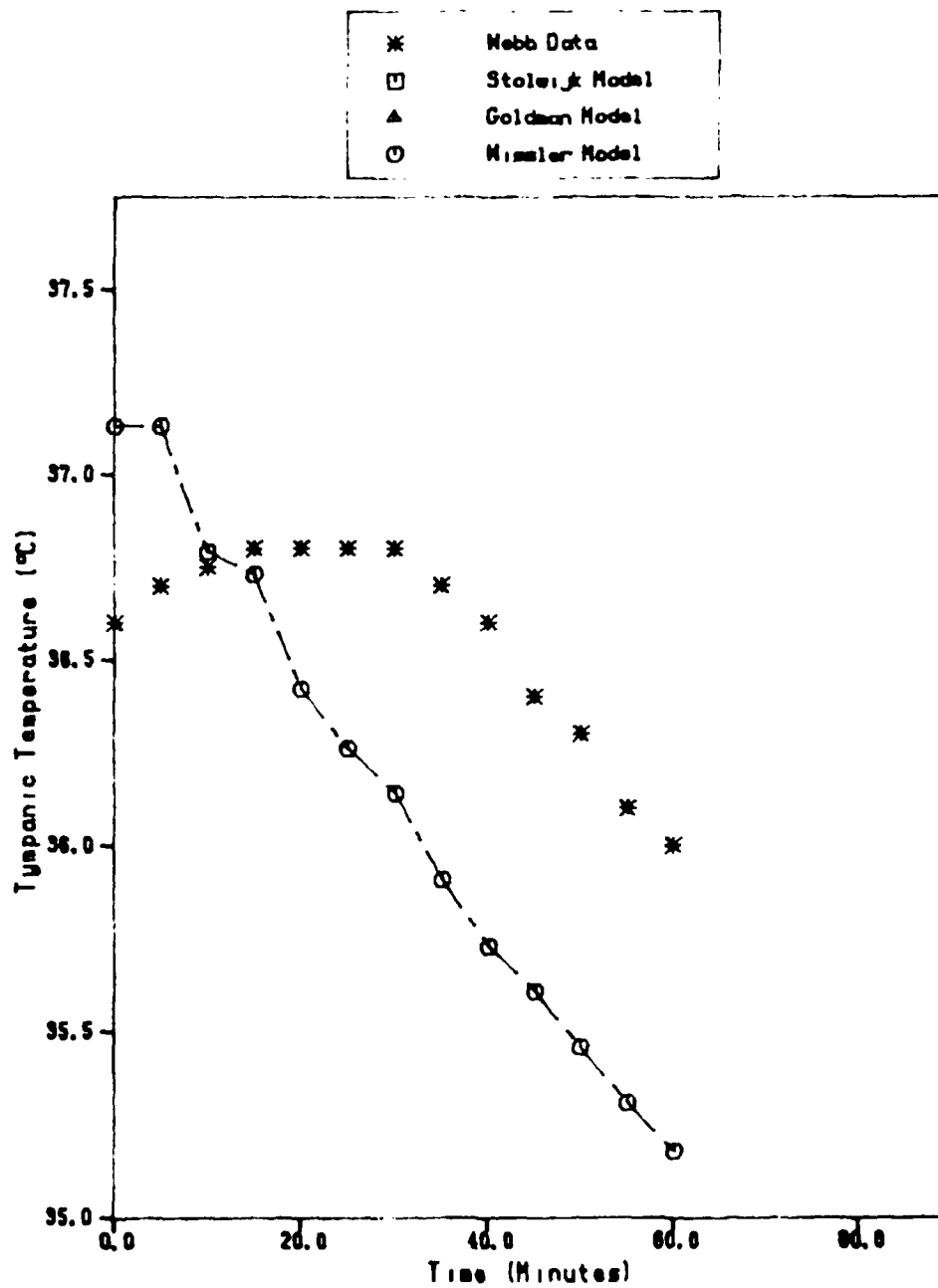
Metabolic Rate
As a Function of Time
For Subject JR During an Immersion in 24 °C Water



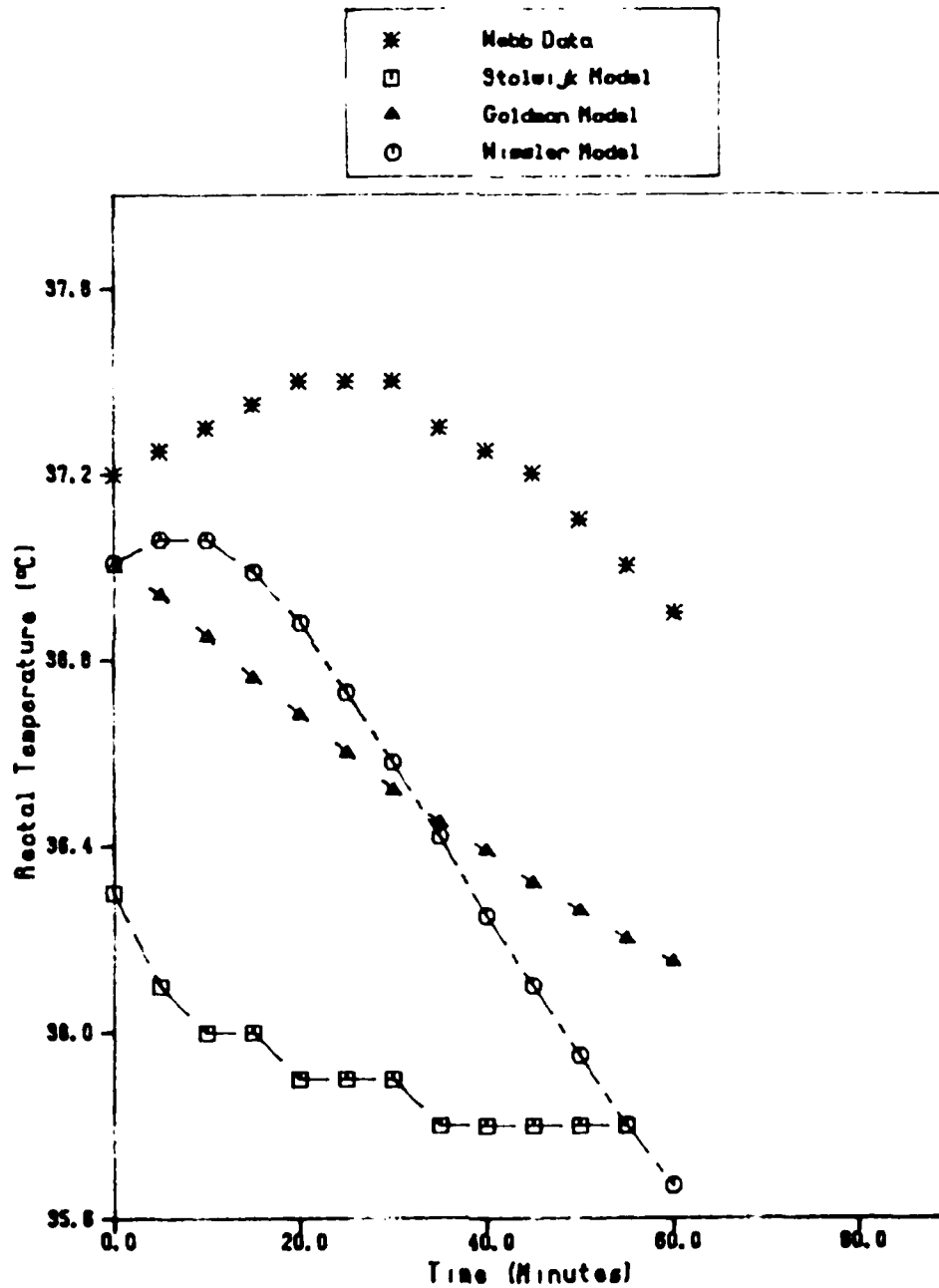
Total Heat Loss Rate
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



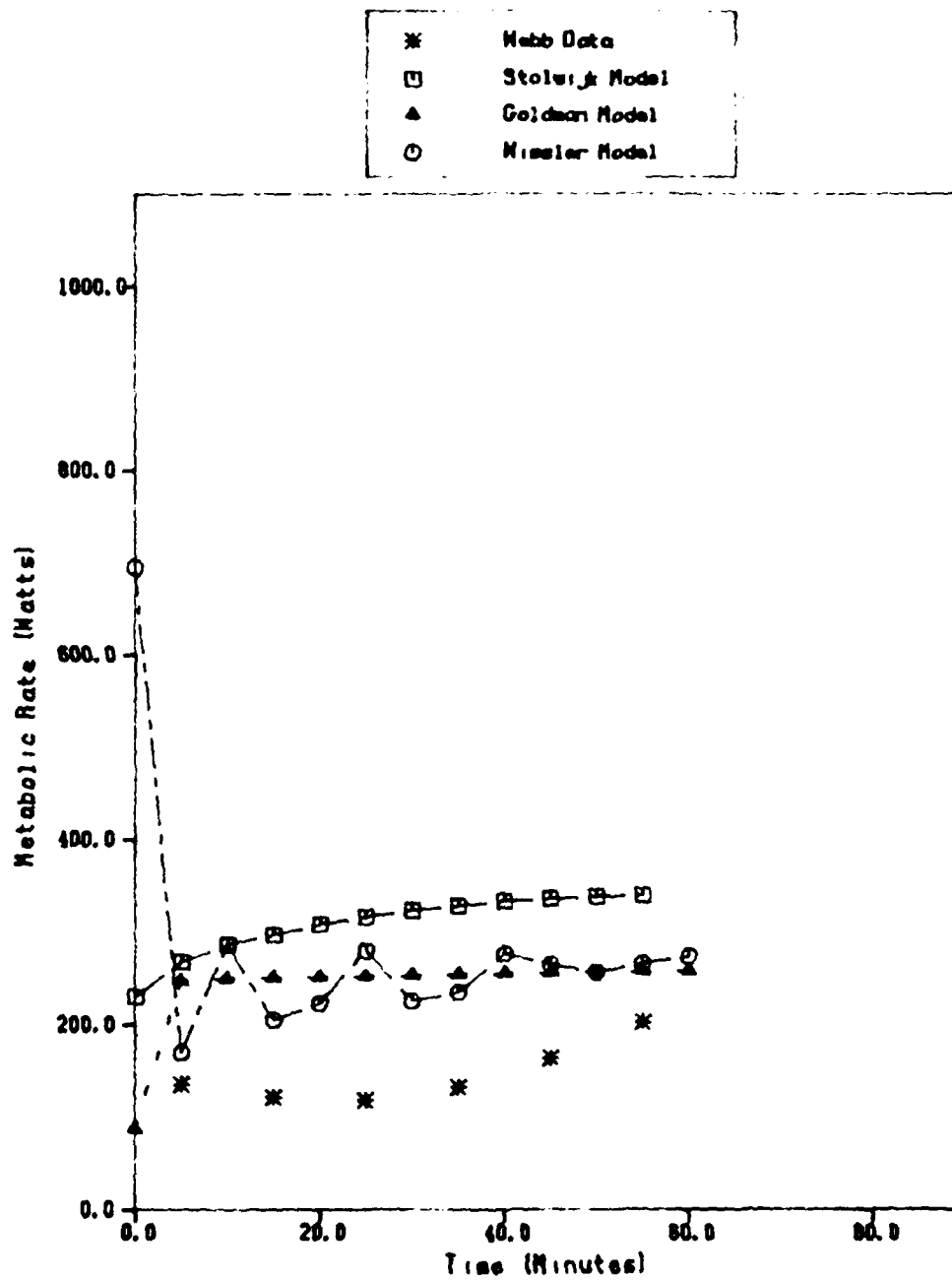
Tympanic Temperature
As a Function of Time
For Subject JR During an Immersion in 24 °C Water



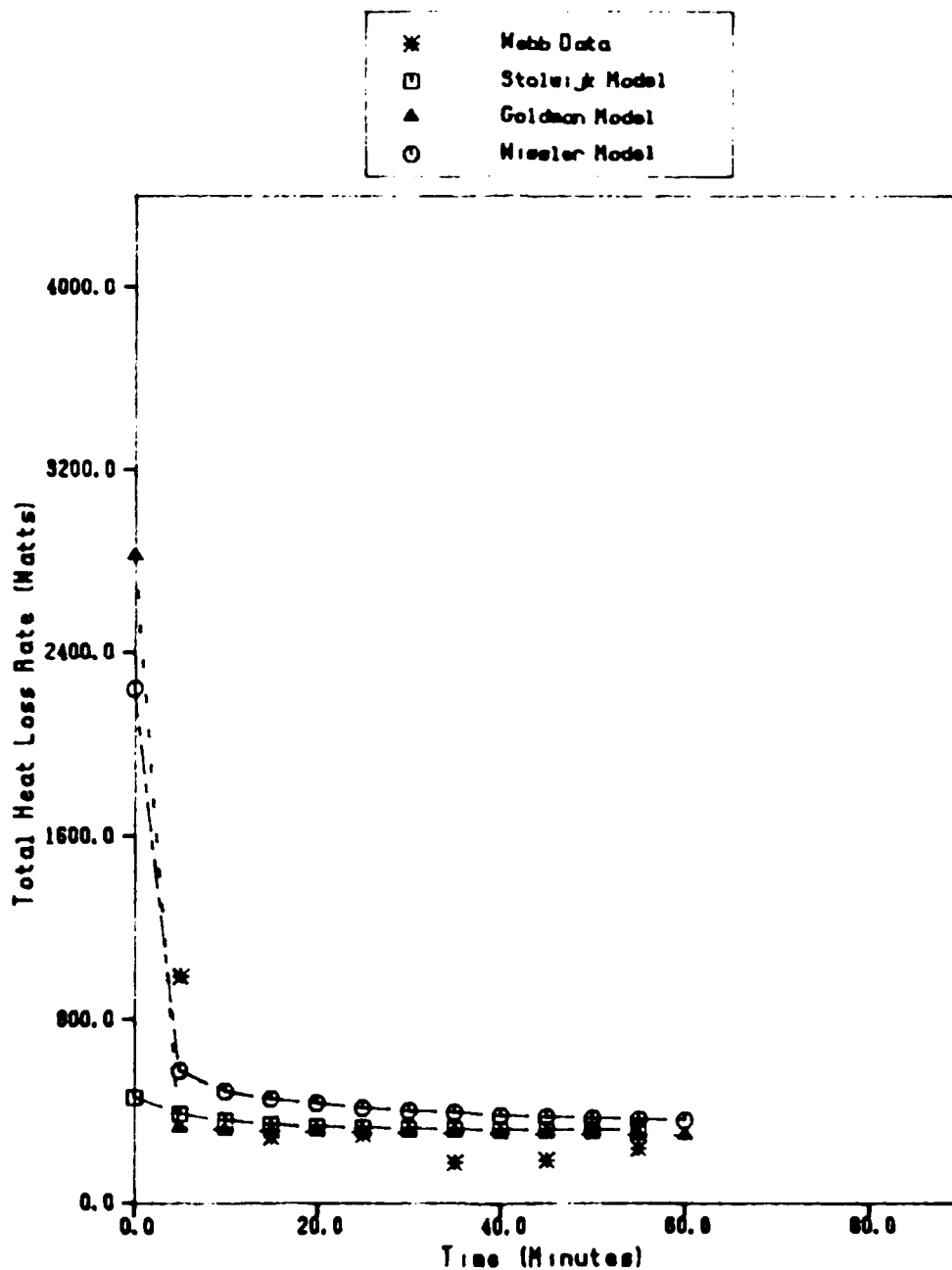
Rectal Temperature
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



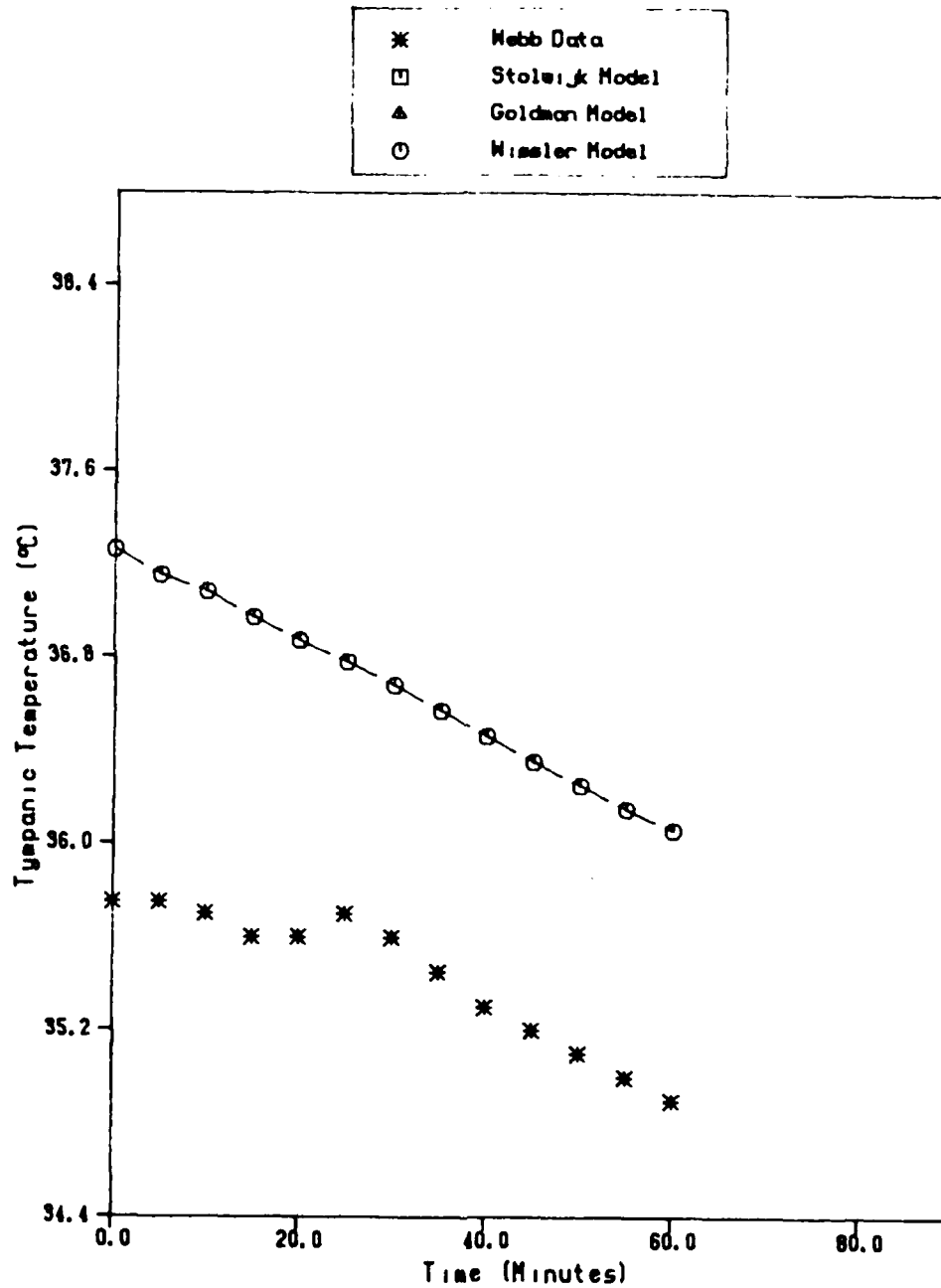
Metabolic Rate
As a Function of Time
For Subject JR During an Immersion in 24 °C Water



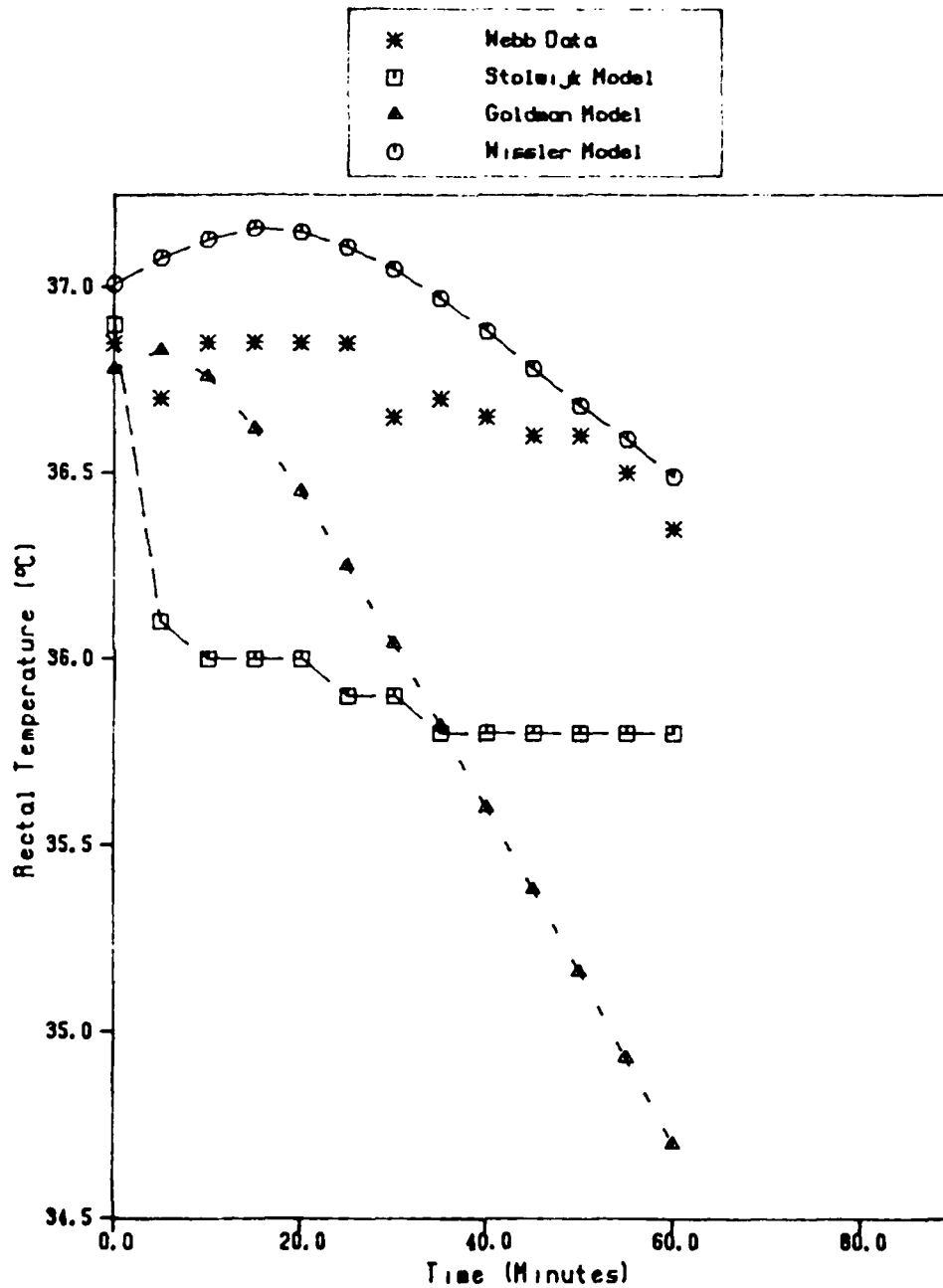
Total Heat Loss Rate
As a Function of Time
For Subject JA During an Immersion in 24 °C Water



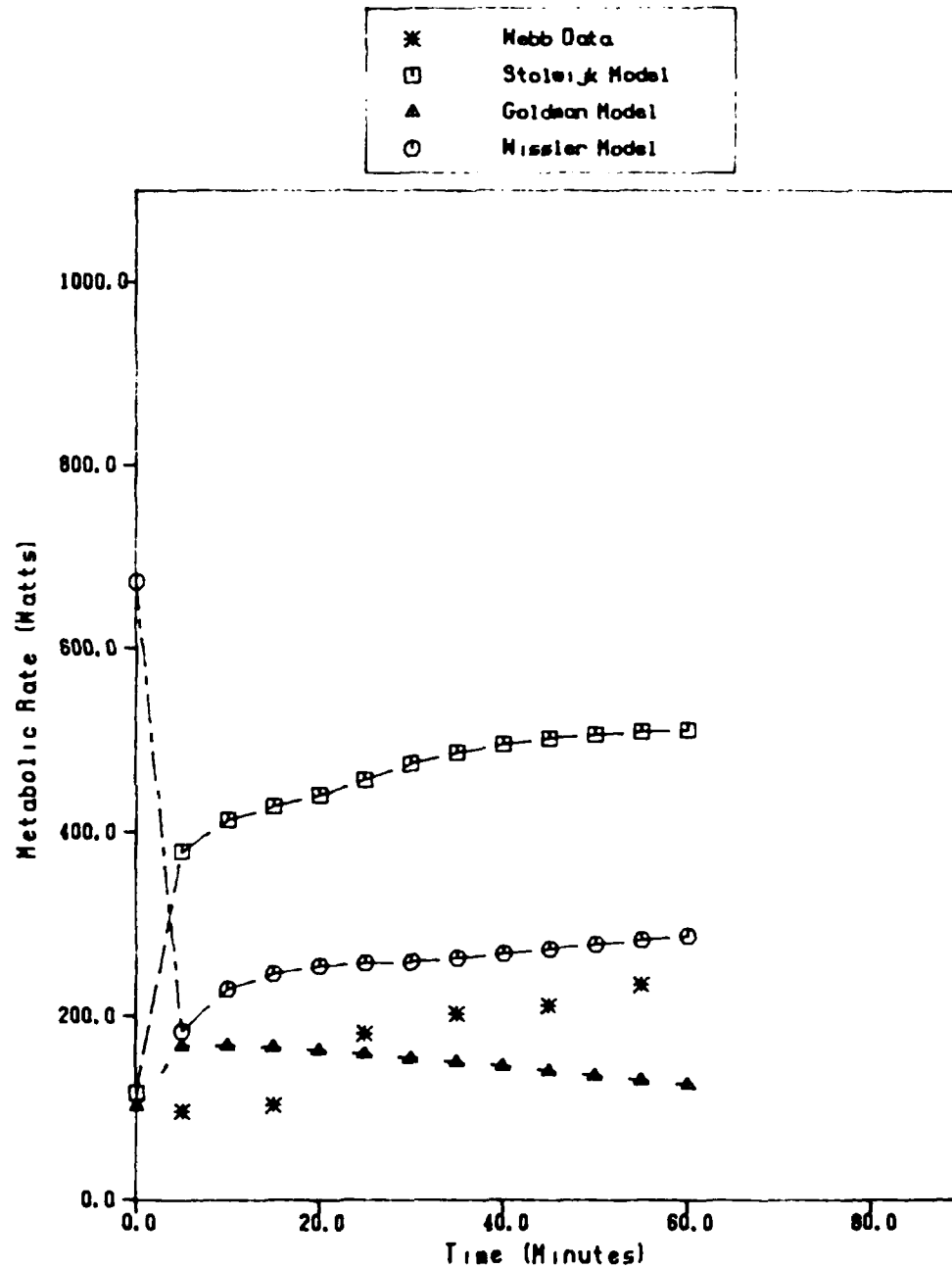
Tympanic Temperature
As a Function of Time
For Subject PW During an Immersion in 18 °C Water



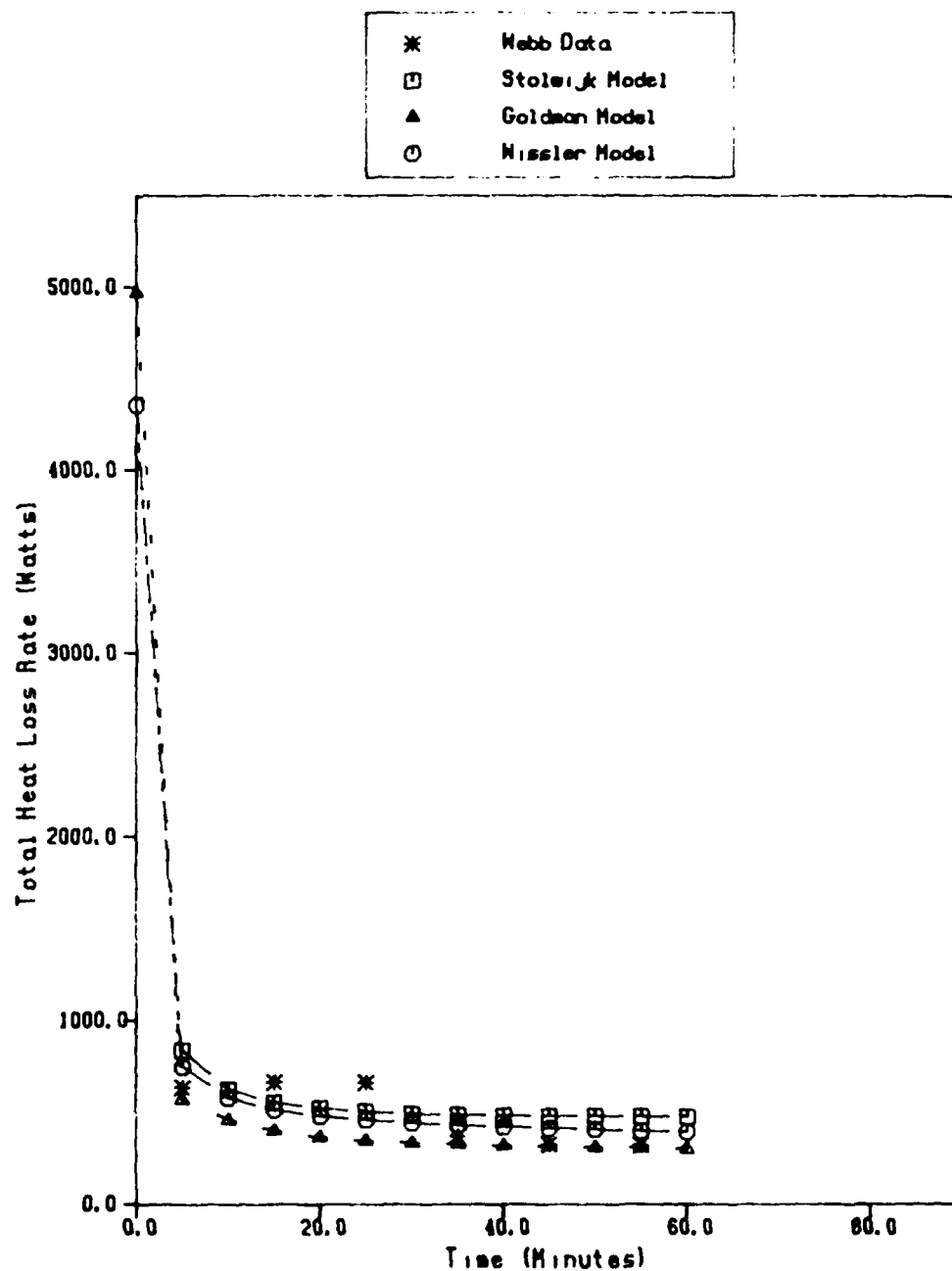
Rectal Temperature
As a Function of Time
For Subject PW During an Immersion in 18 °C Water



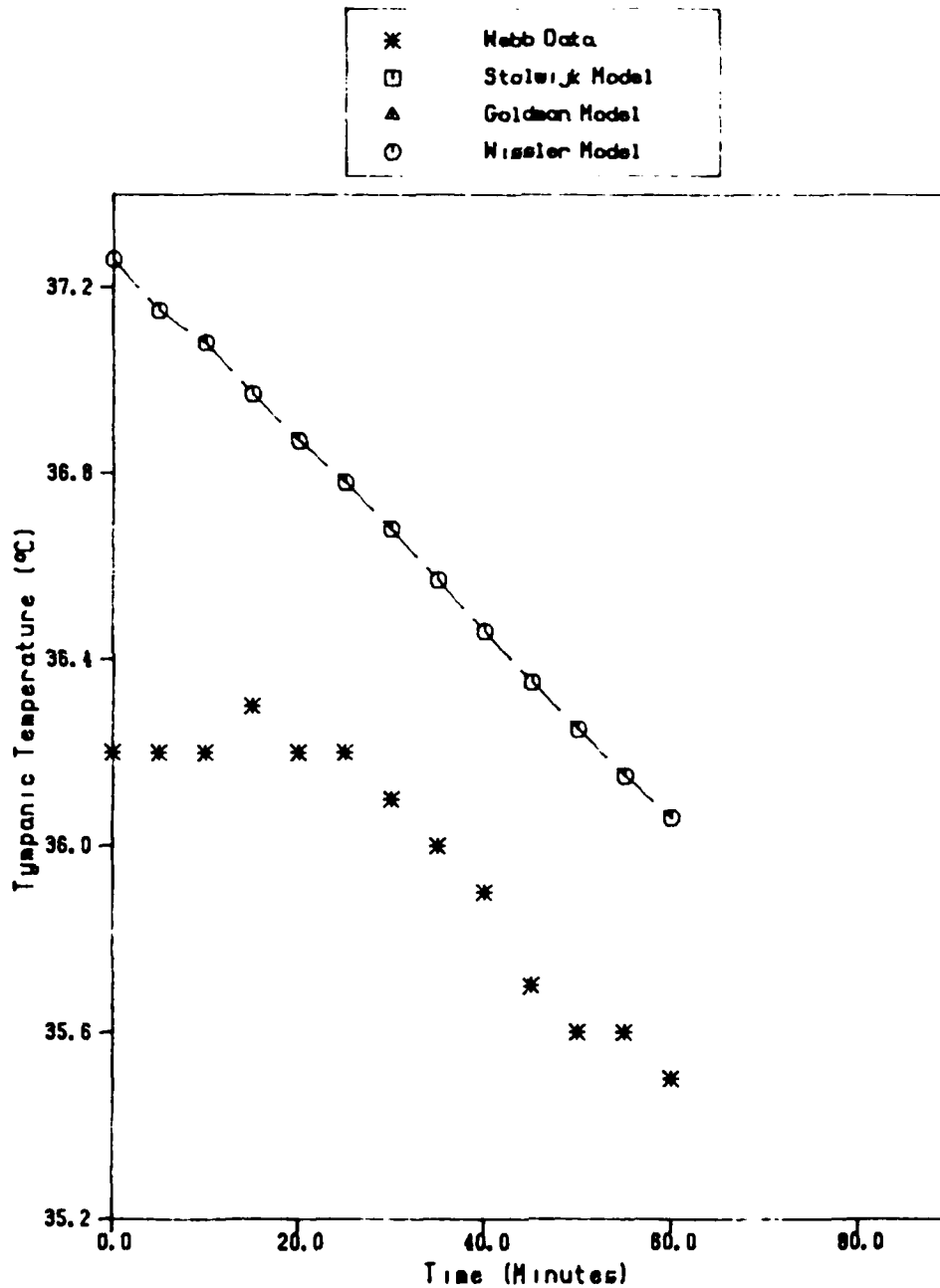
Metabolic Rate
As a Function of Time
For Subject PW During an Immersion in 18 °C Water



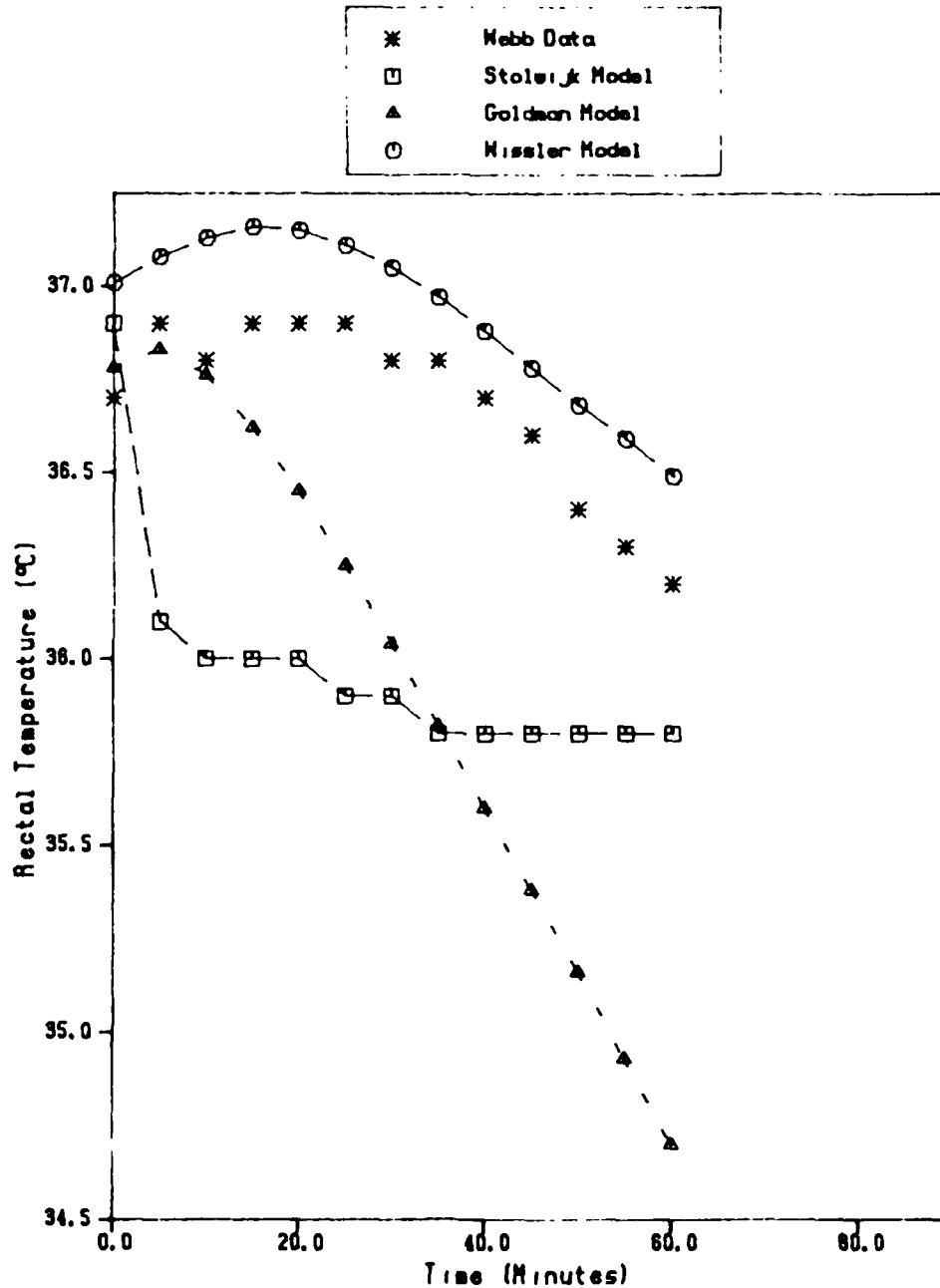
Total Heat Loss Rate
As a Function of Time
For Subject PW During an Immersion in 18 °C Water



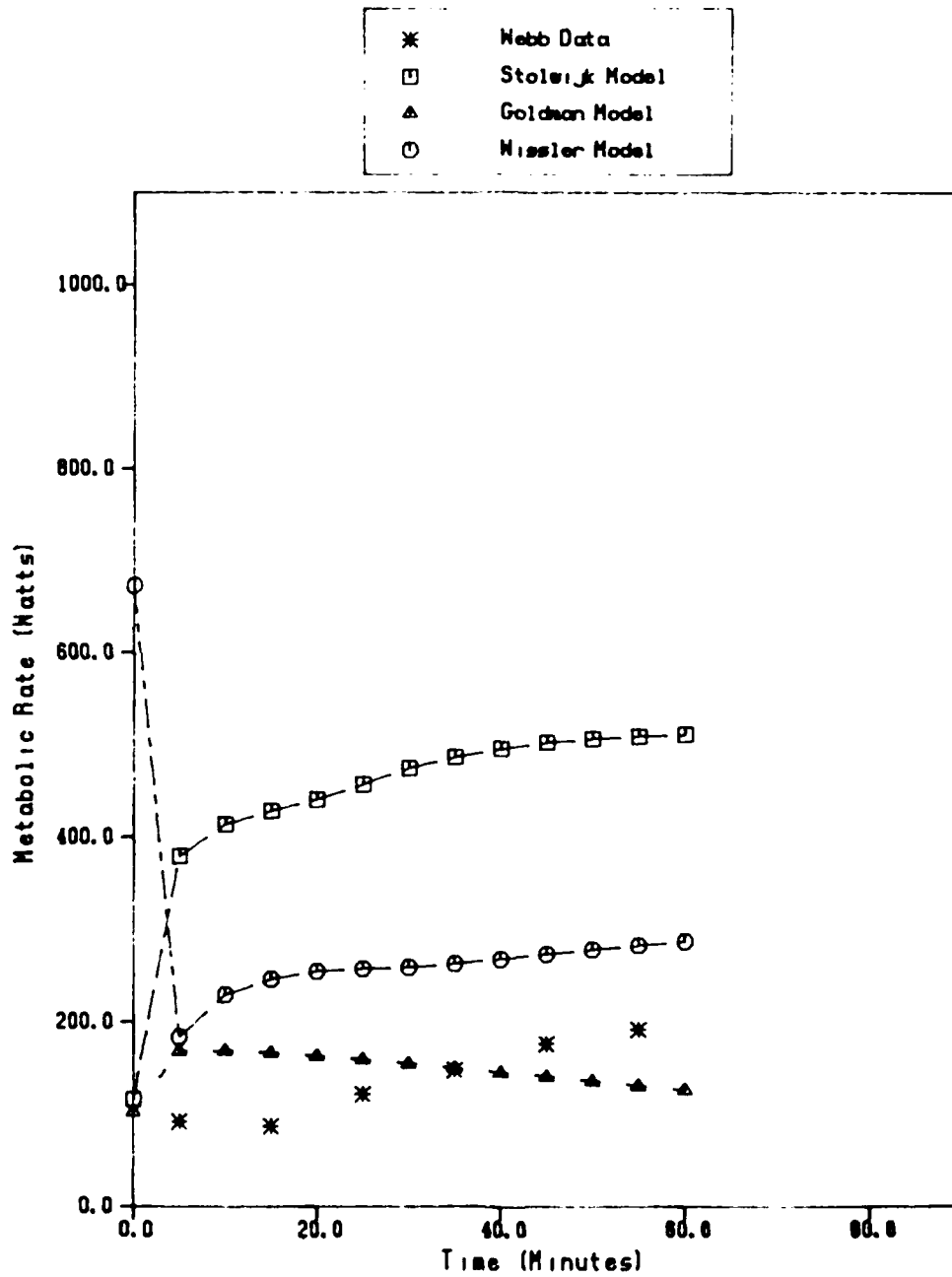
Tympanic Temperature
As a Function of Time
For Subject PW During an Immersion in 18 °C Water



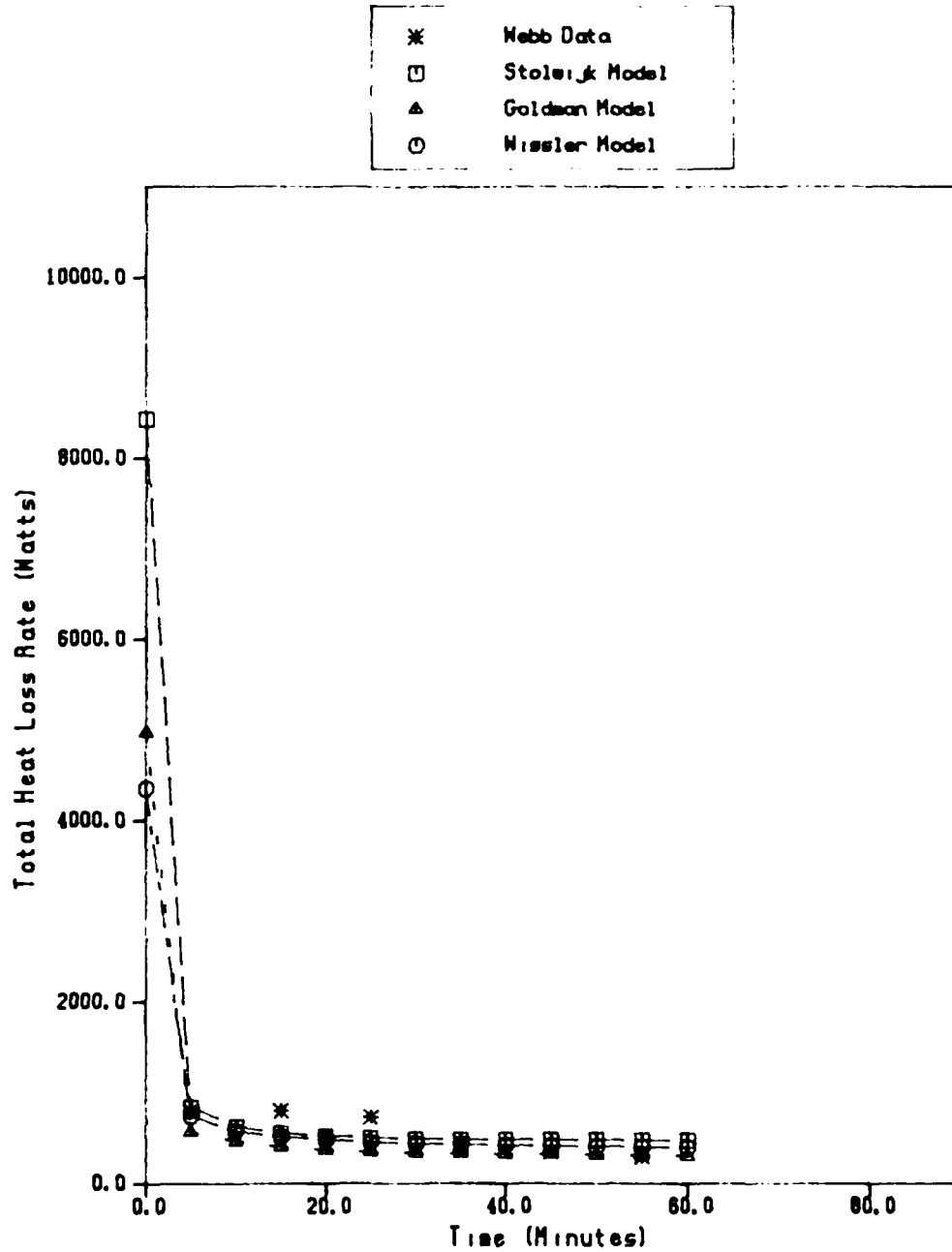
Rectal Temperature
As a Function of Time
For Subject PW During an Immersion in 18 °C Water



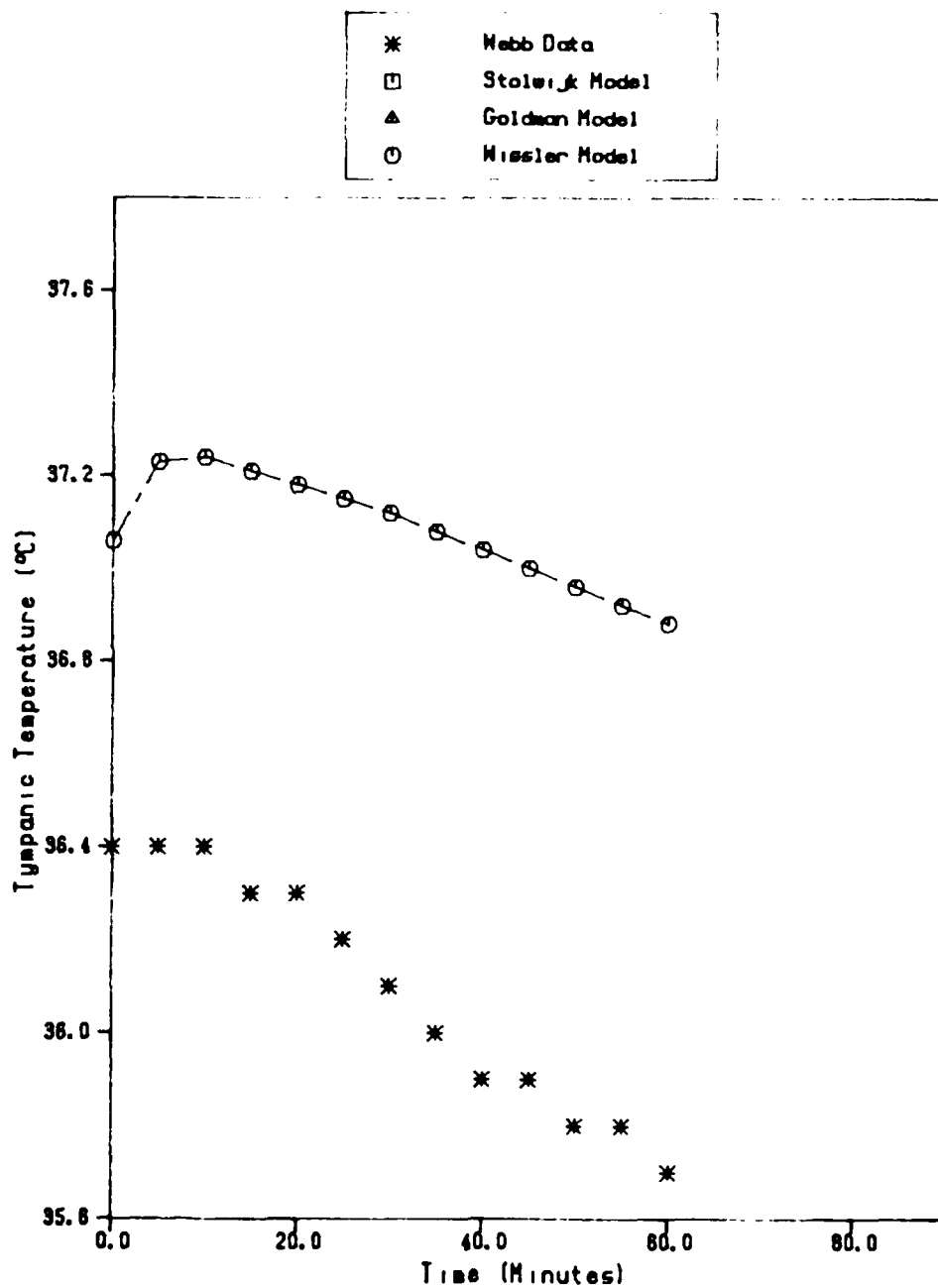
Metabolic Rate
As a Function of Time
For Subject PW During an Immersion in 18 °C Water



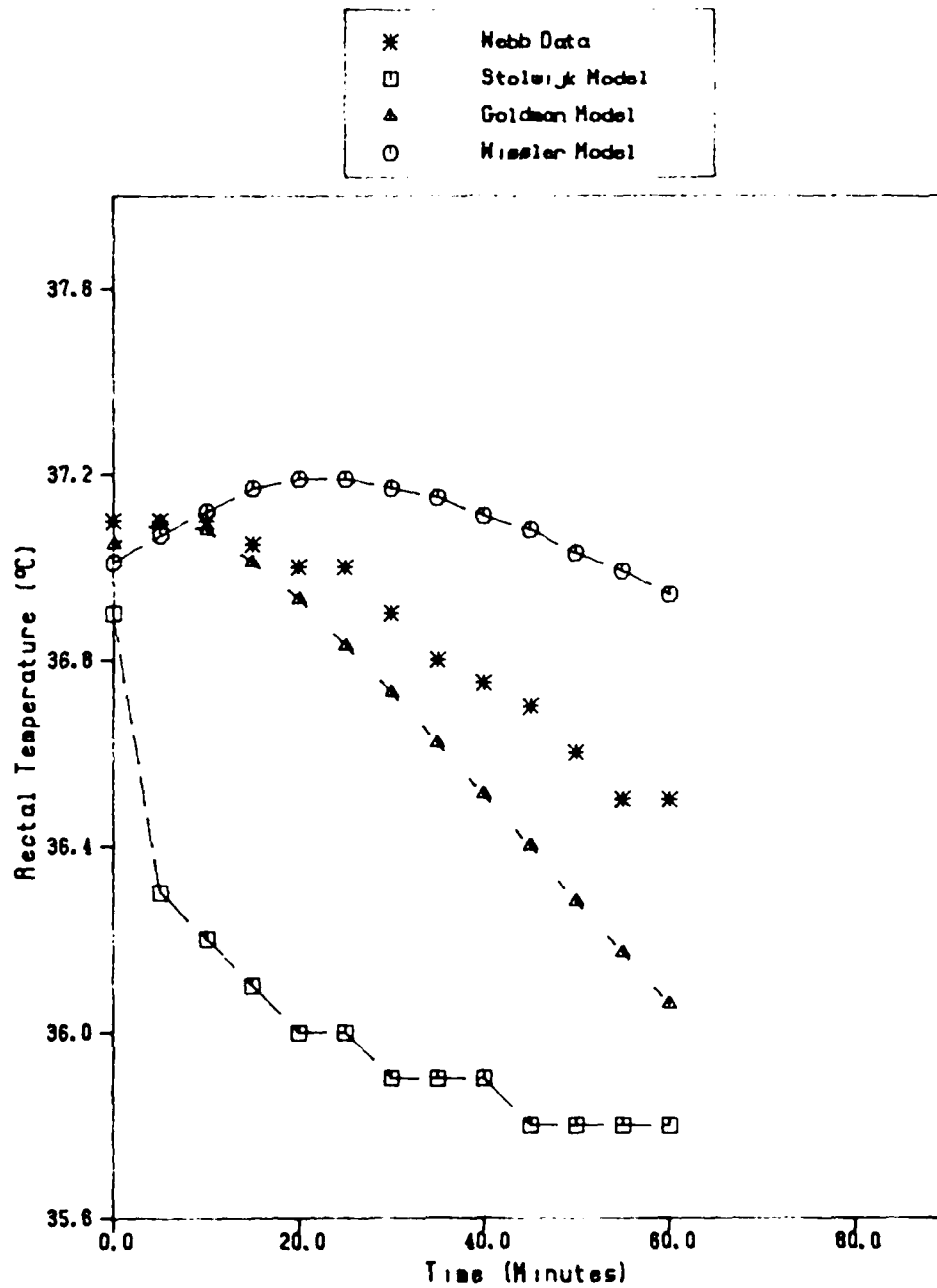
Total Heat Loss Rate
As a Function of Time
For Subject PW During an Immersion in 18 °C Water



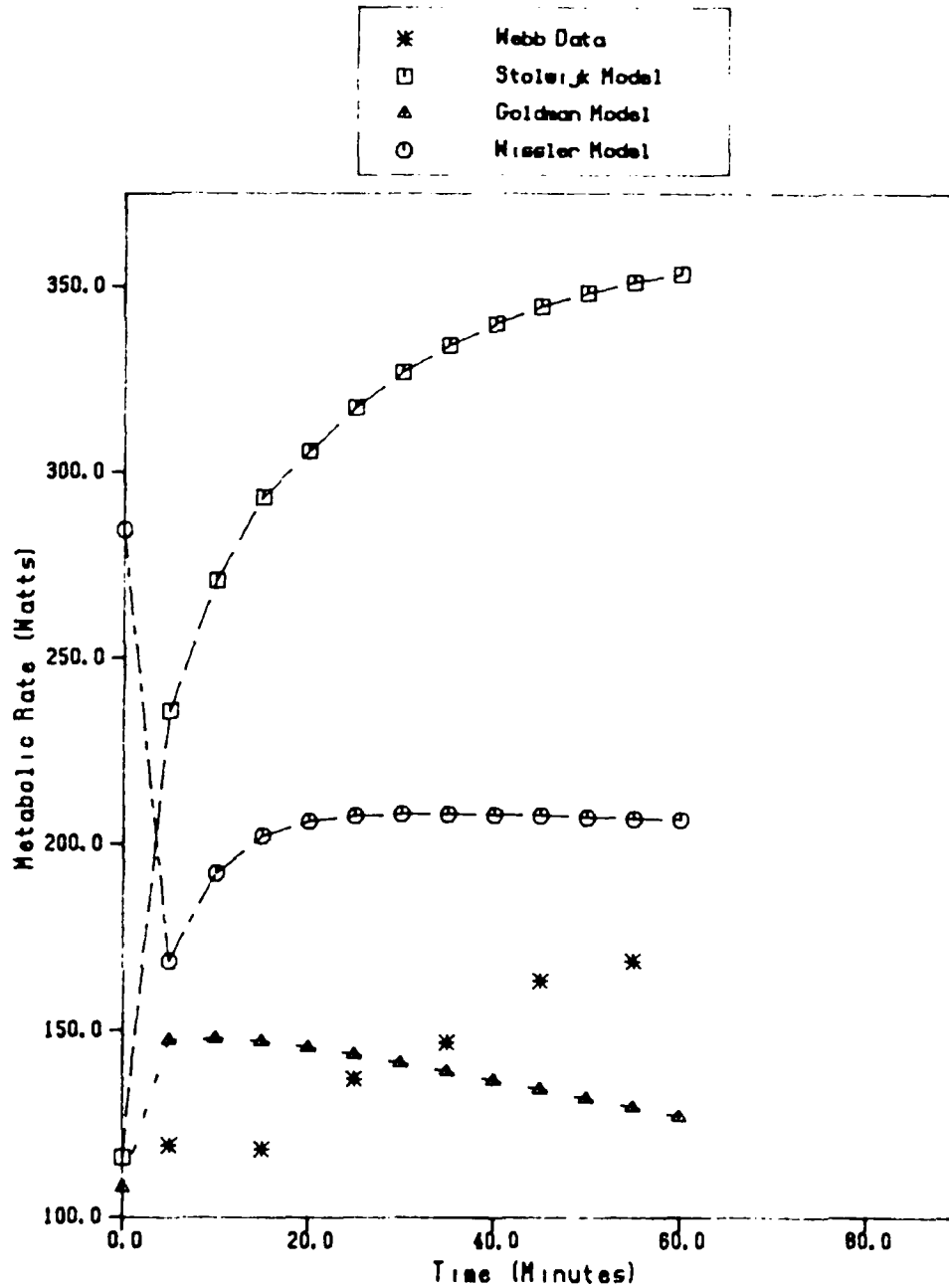
Tympanic Temperature
As a Function of Time
For Subject PW During an Immersion in 24 °C Water



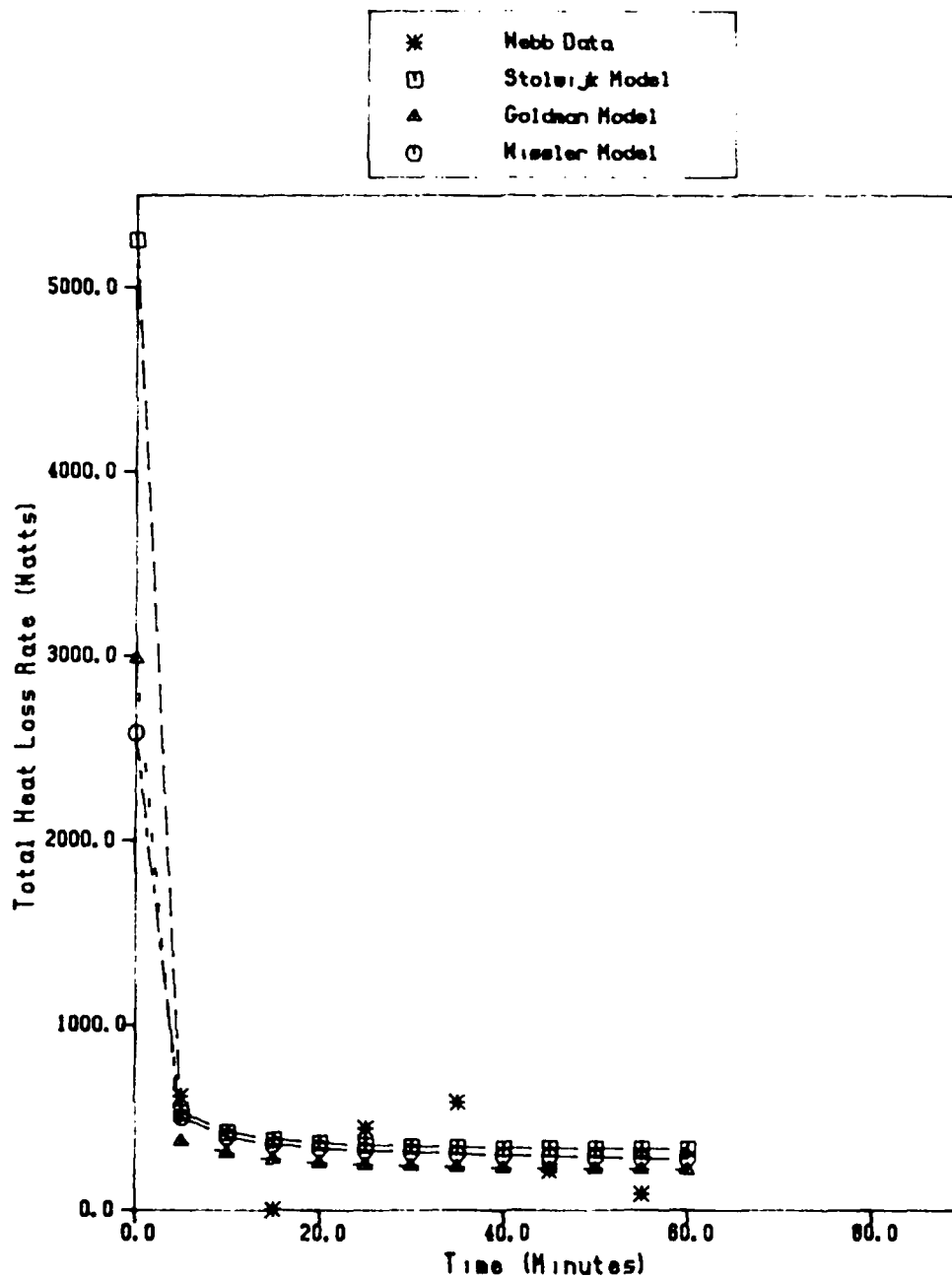
Rectal Temperature
As a Function of Time
For Subject PW During an Immersion in 24 °C Water



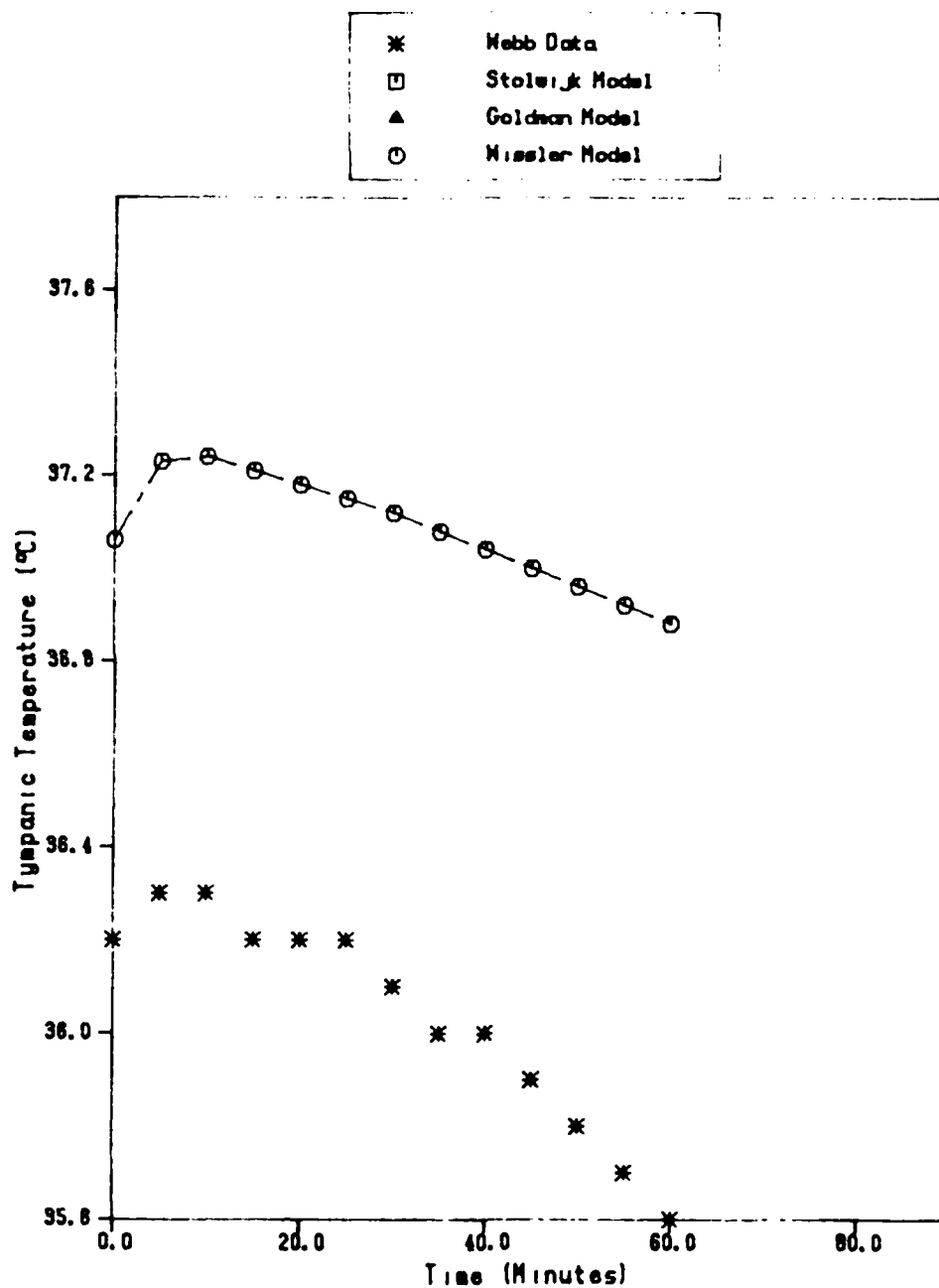
Metabolic Rate
As a Function of Time
For Subject PW During an Immersion in 24 °C Water



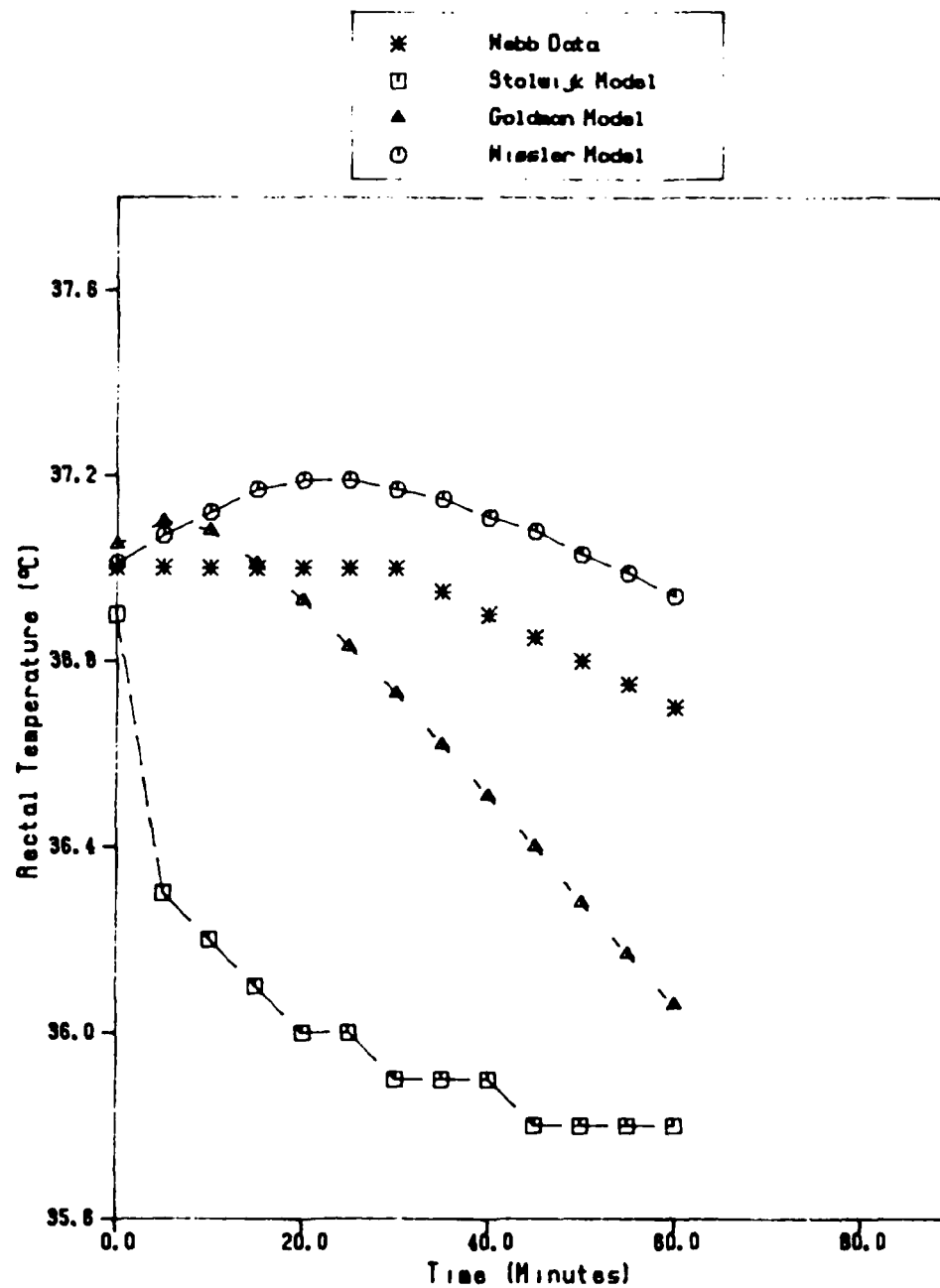
Total Heat Loss Rate
As a Function of Time
For Subject PW During an Immersion in 24 °C Water



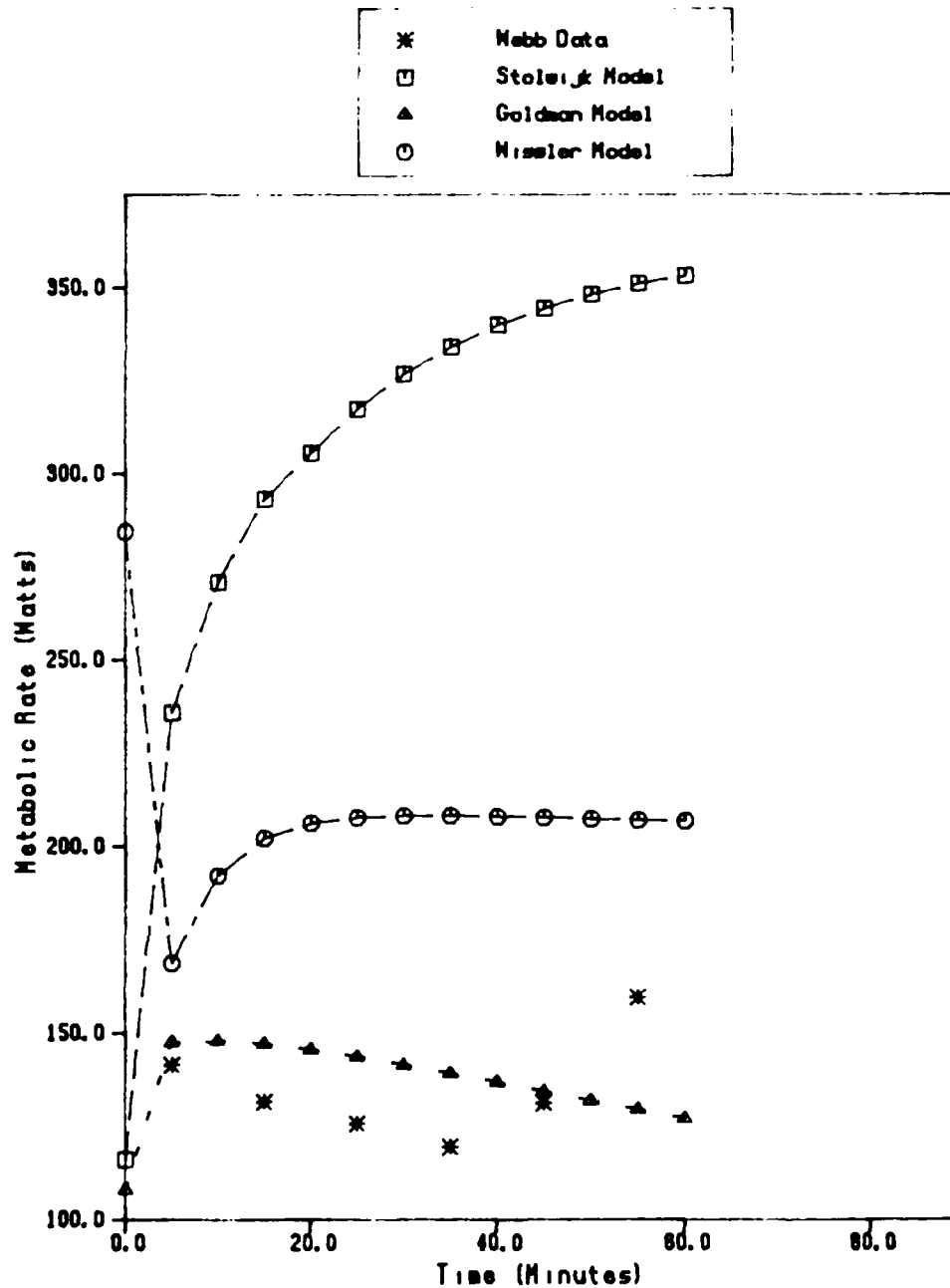
Tympanic Temperature
As a Function of Time
For Subject PW During an Immersion in 24 °C Water



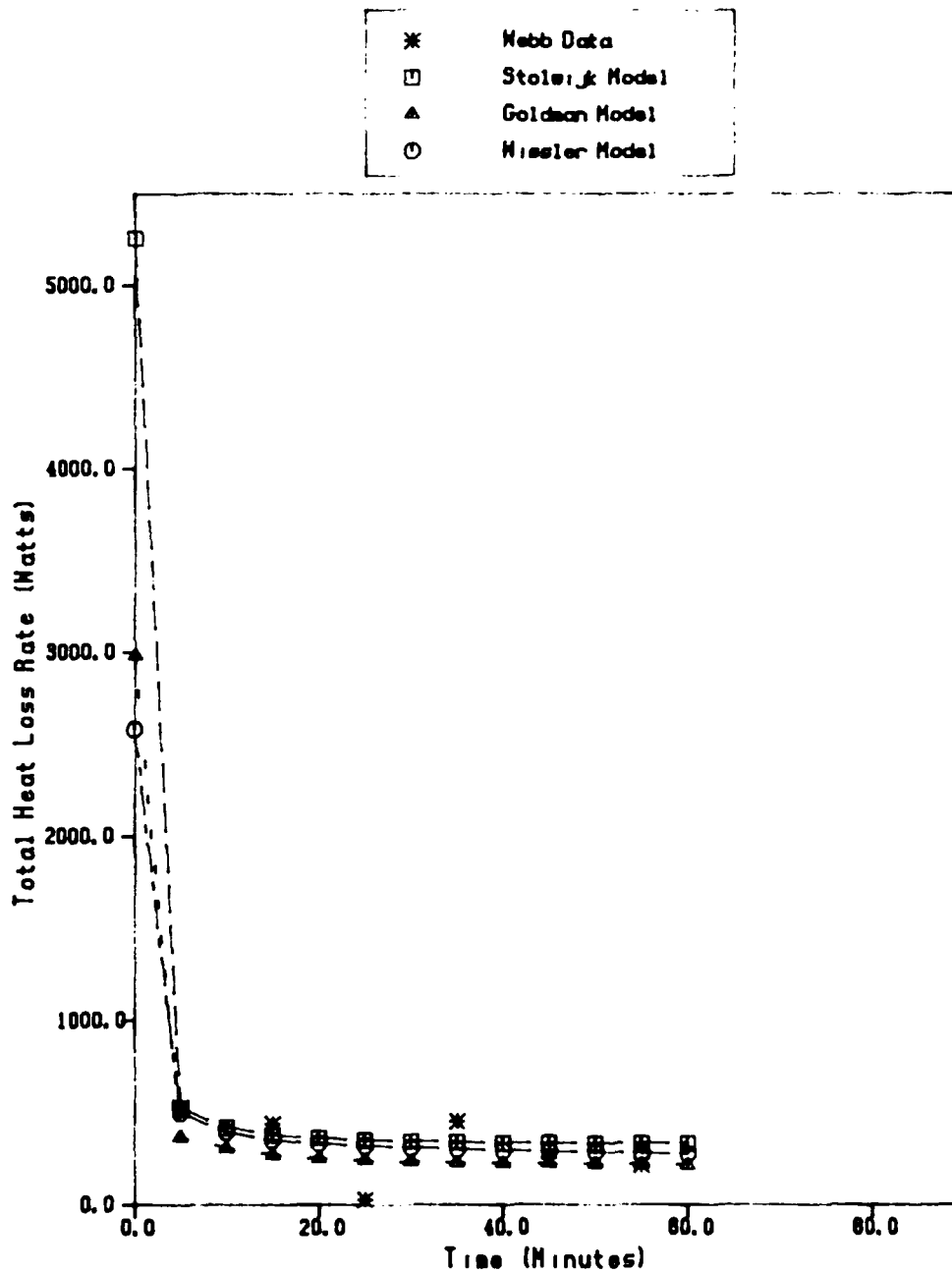
Rectal Temperature
As a Function of Time
For Subject PW During an Immersion in 24 °C Water



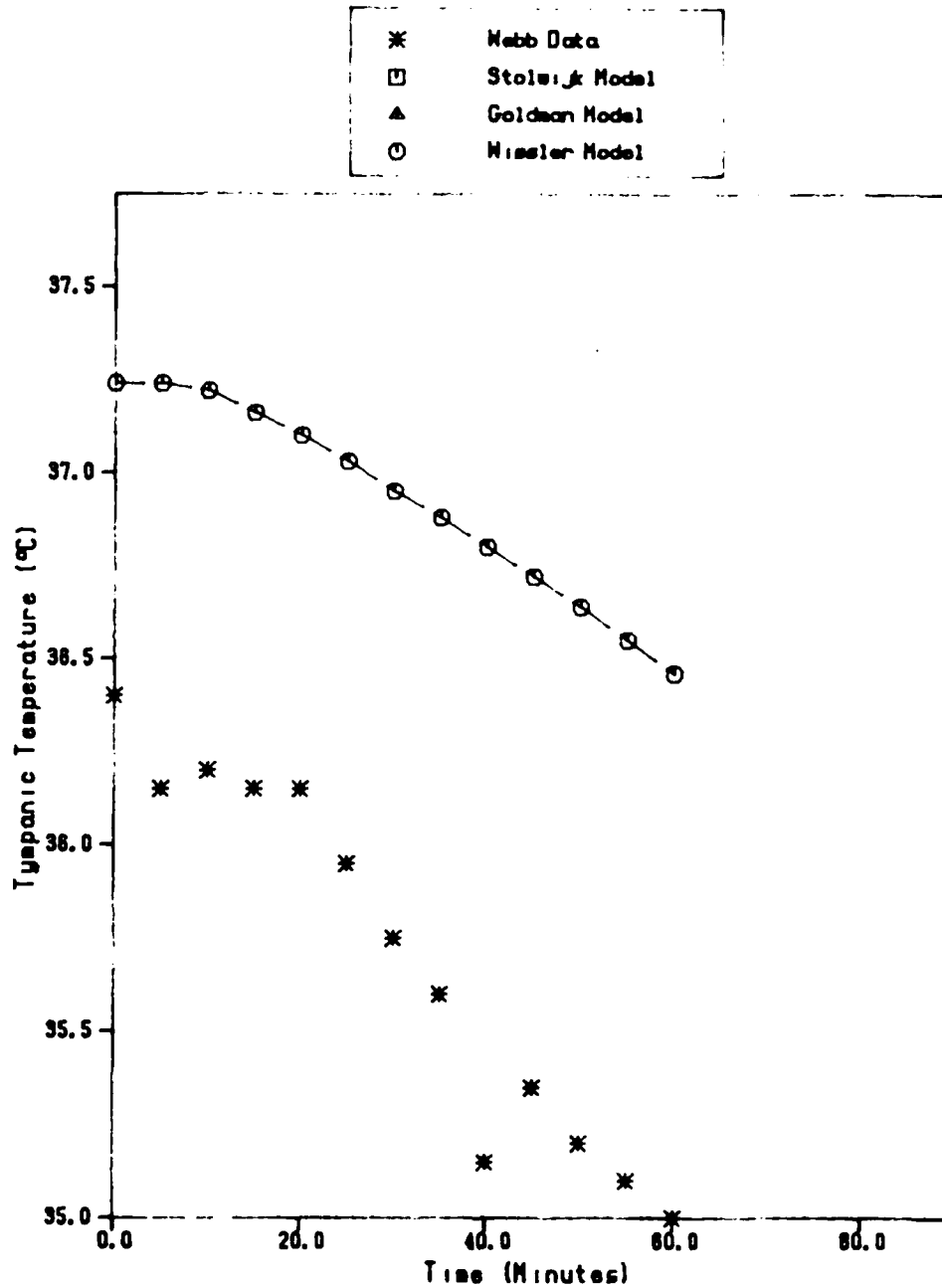
Metabolic Rate
As a Function of Time
For Subject PN During an Immersion in 24 °C Water



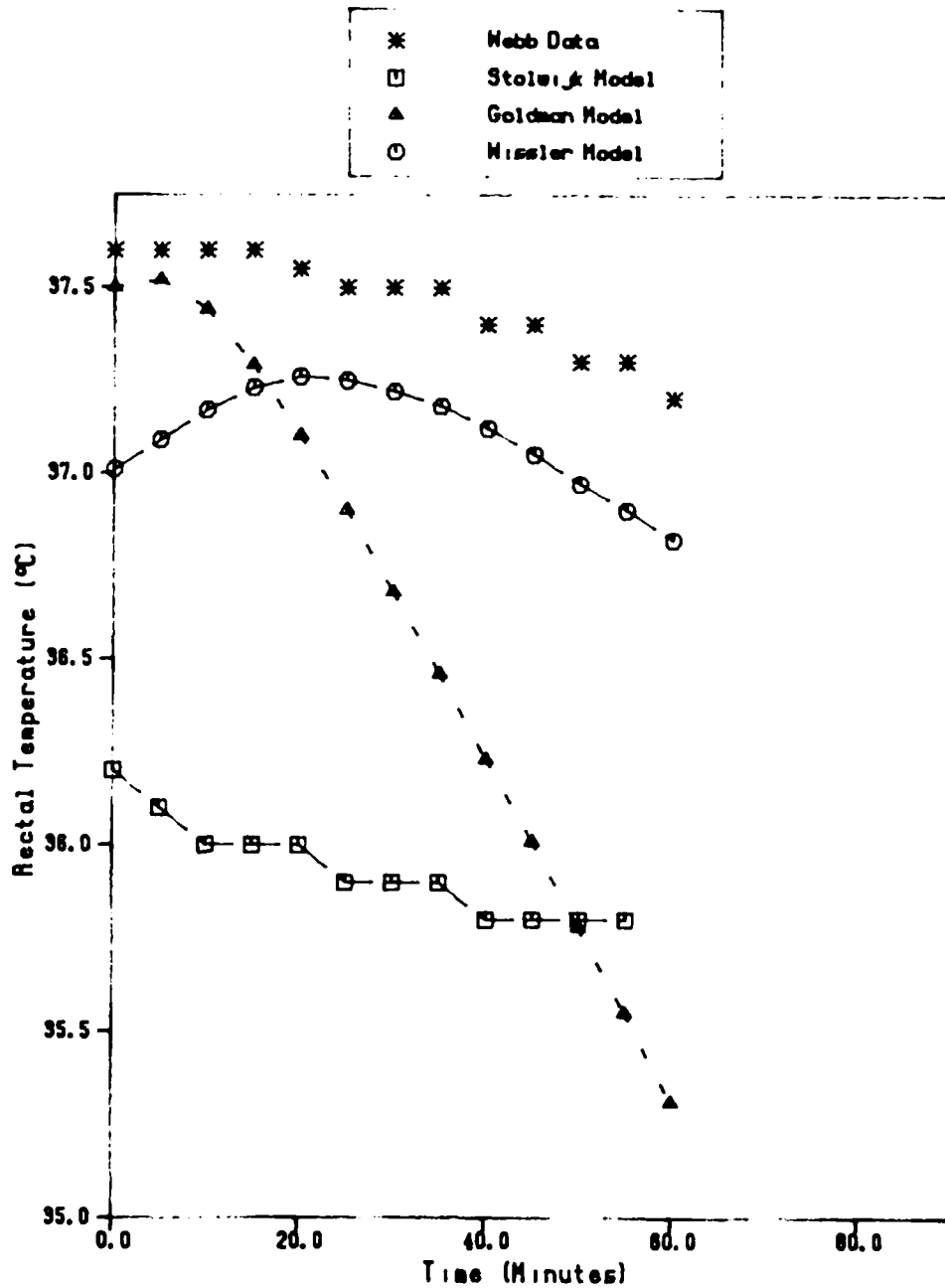
Total Heat Loss Rate
As a Function of Time
For Subject PW During an Immersion in 24 °C Water



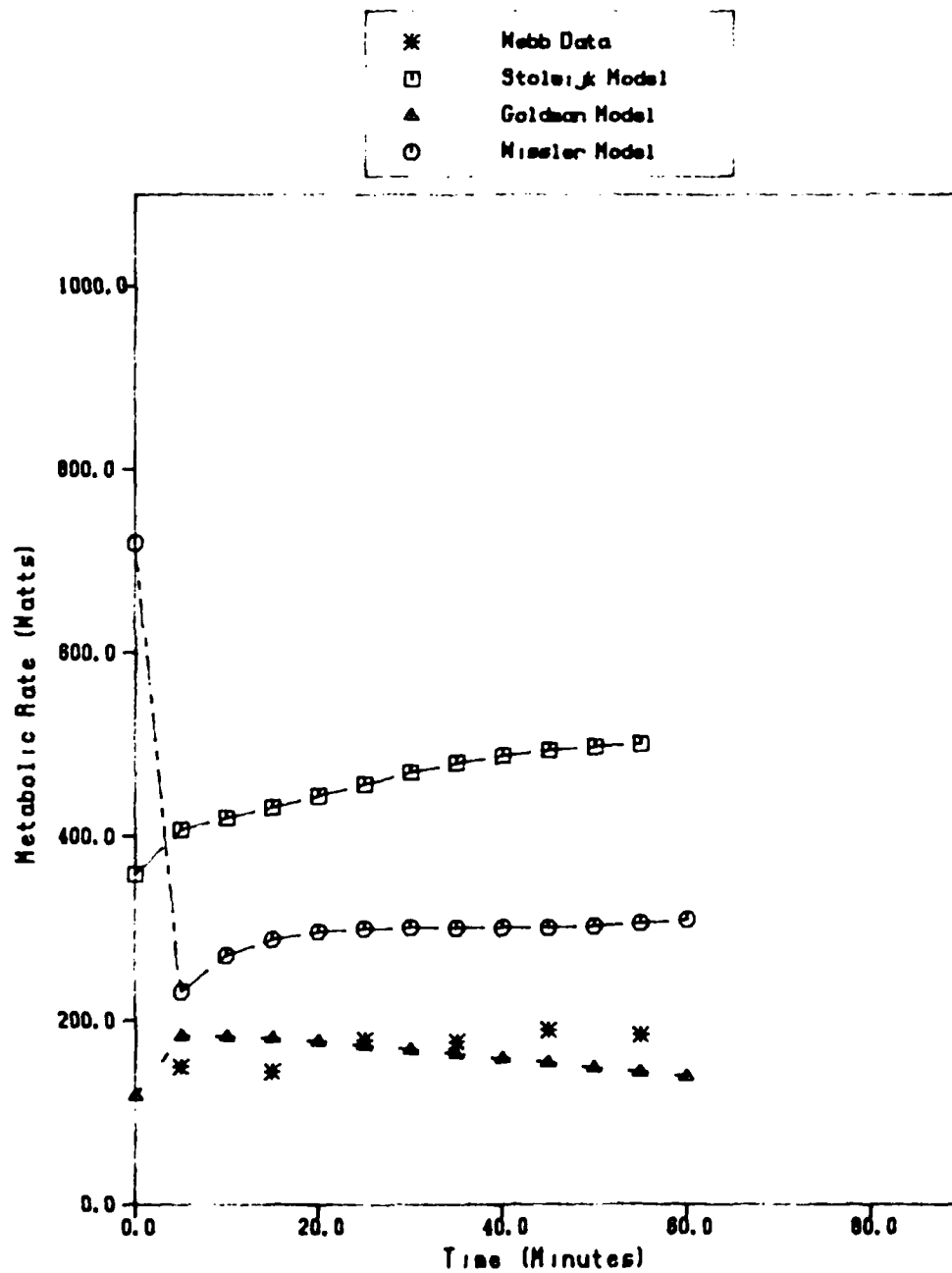
Tympanic Temperature
As a Function of Time
For Subject ST During an Immersion in 18 °C Water



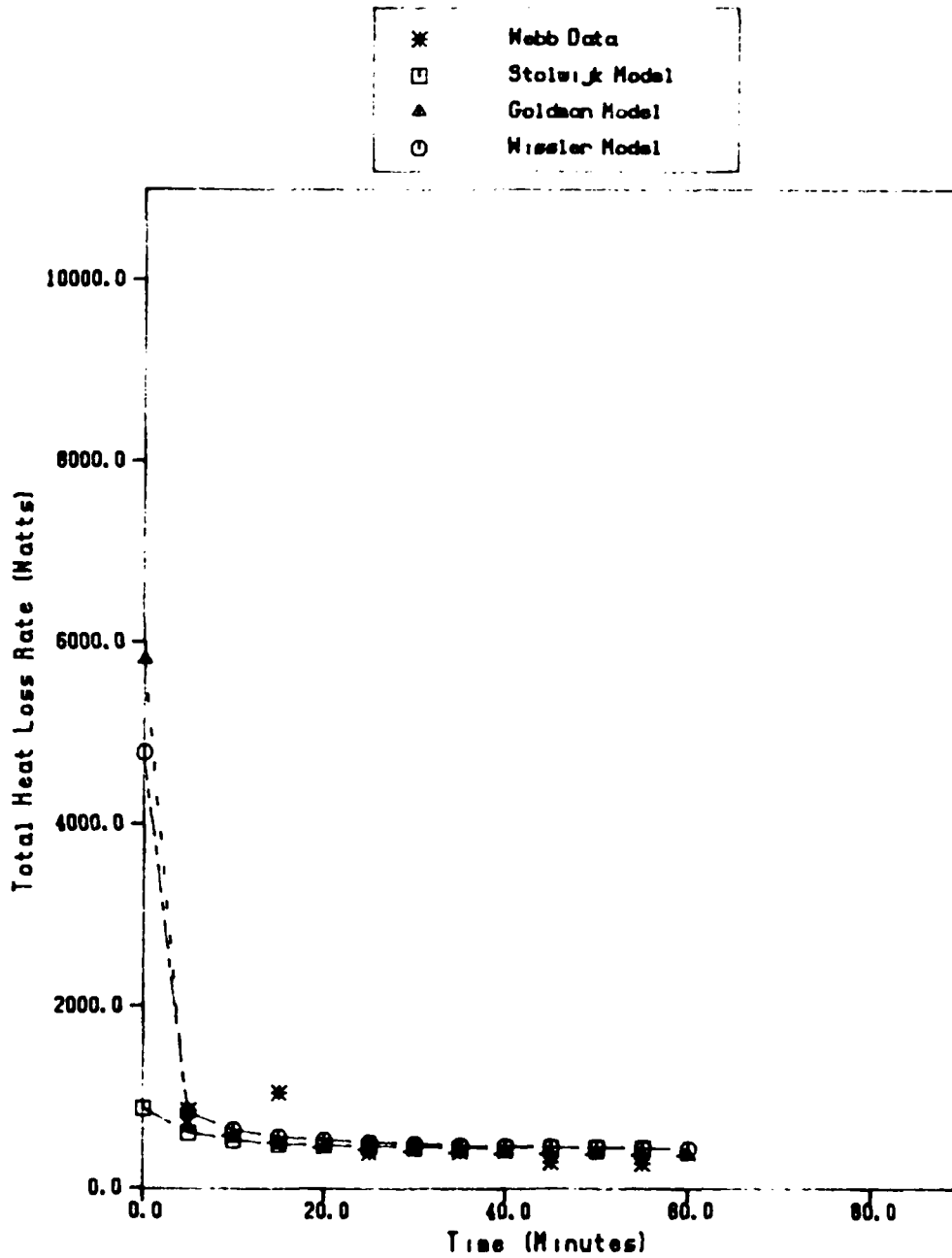
Rectal Temperature
As a Function of Time
For Subject ST During an Immersion in 18 °C Water



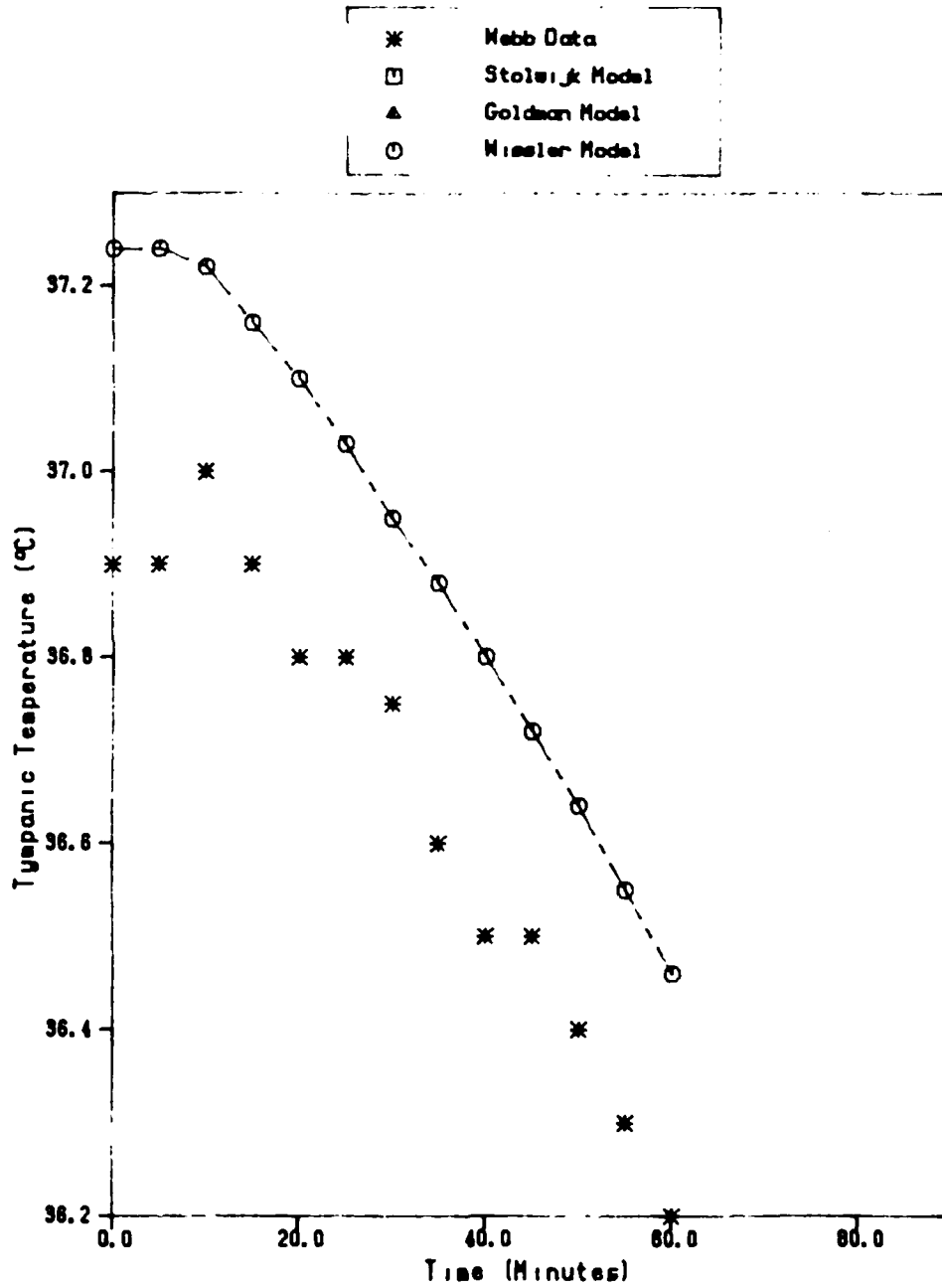
Metabolic Rate
As a Function of Time
For Subject ST During an Immersion in 18 °C Water



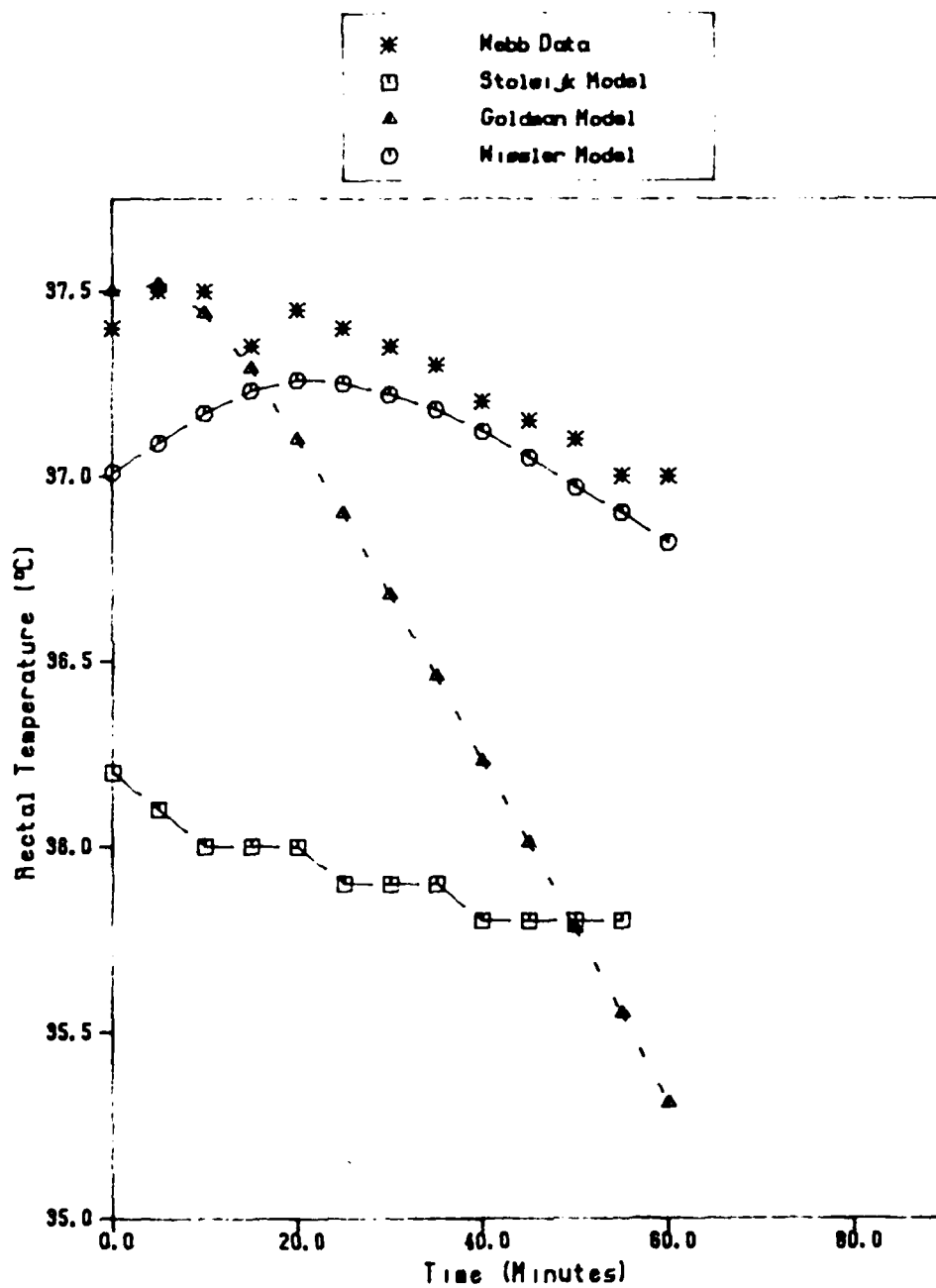
Total Heat Loss Rate
As a Function of Time
For Subject ST During an Immersion in 18 °C Water



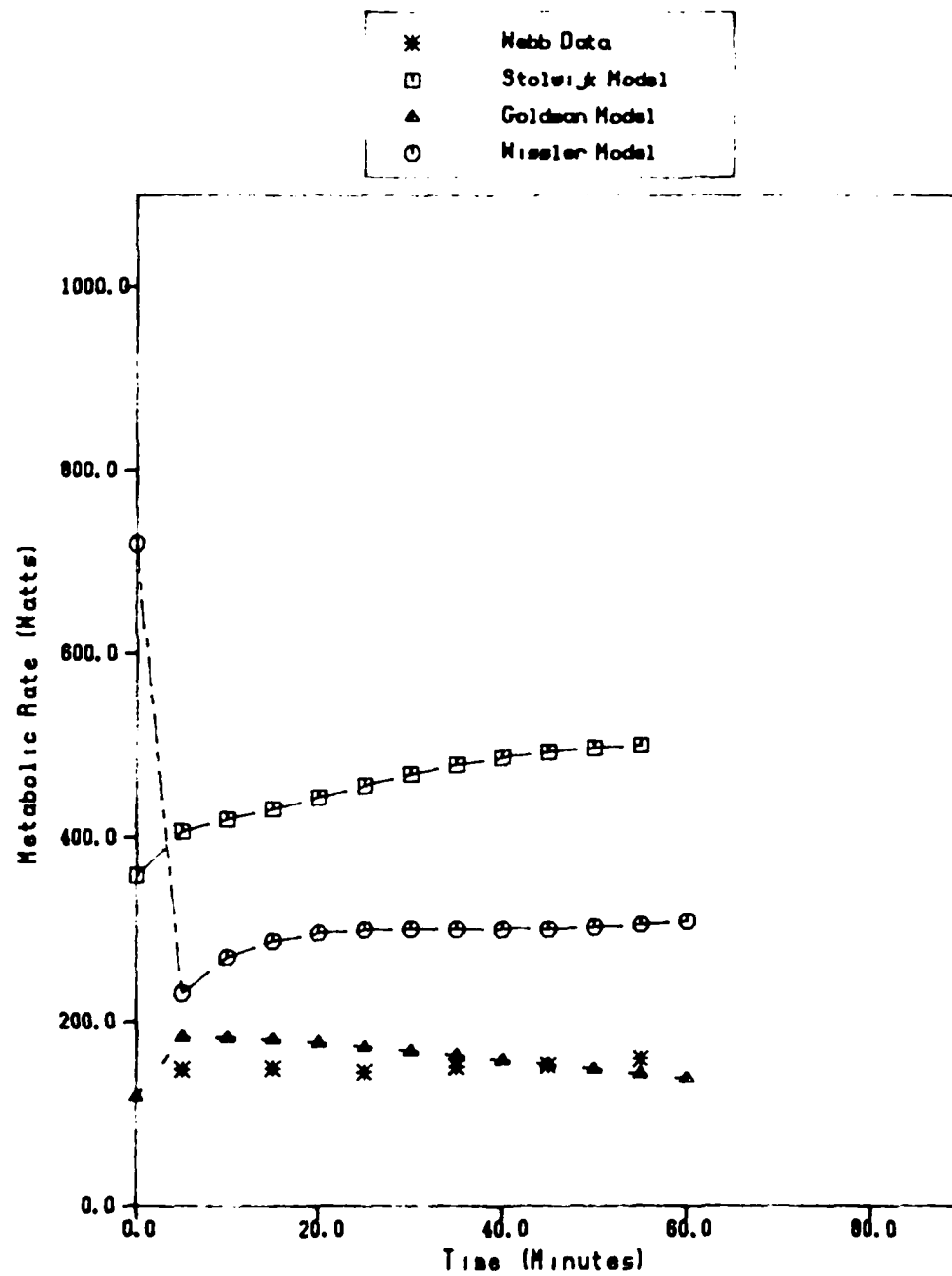
Tympanic Temperature
As a Function of Time
For Subject ST During an Immersion in 18 °C Water



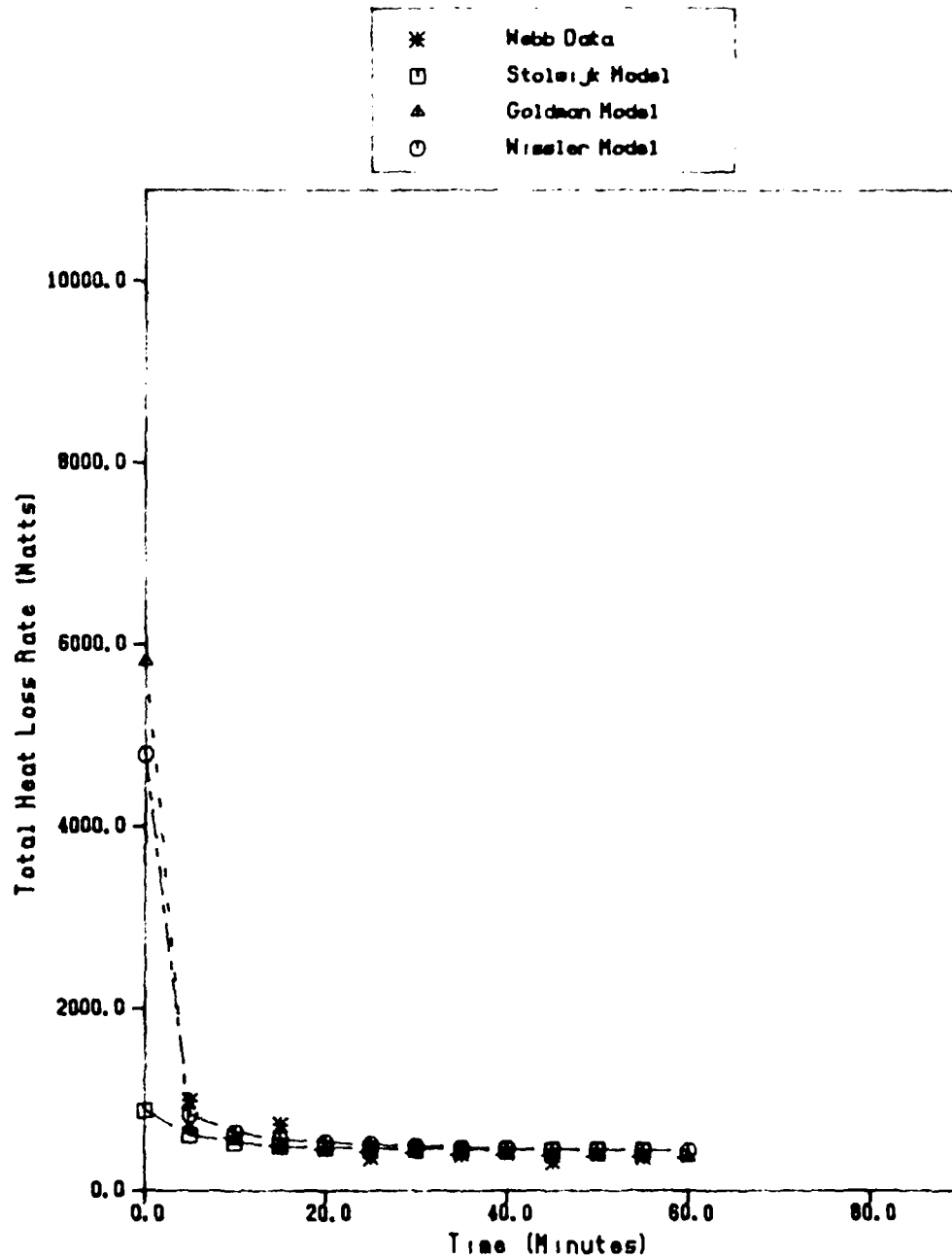
Rectal Temperature
As a Function of Time
For Subject ST During an Immersion in 18 °C Water



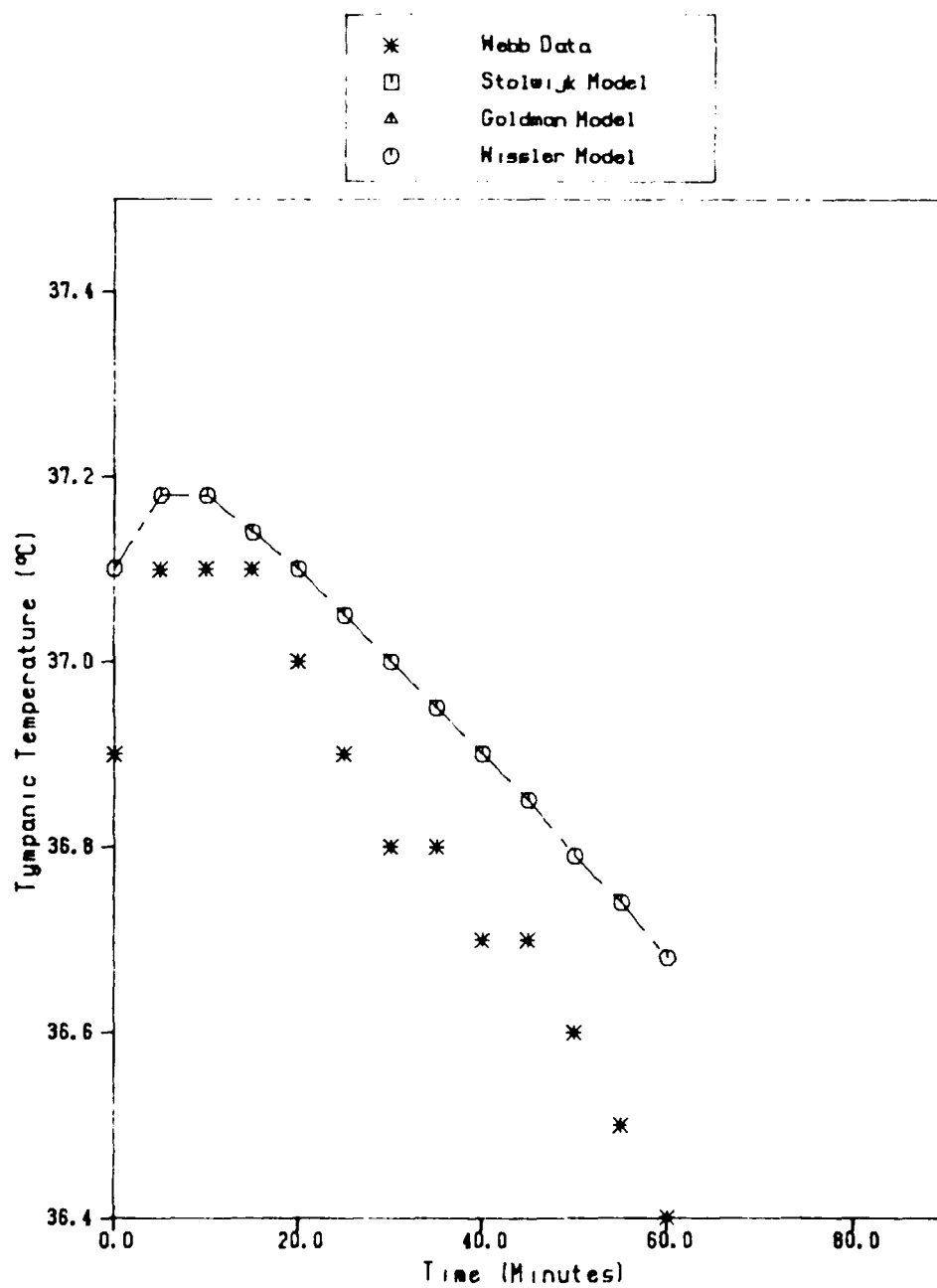
Metabolic Rate
As a Function of Time
For Subject ST During an Immersion in 18 °C Water



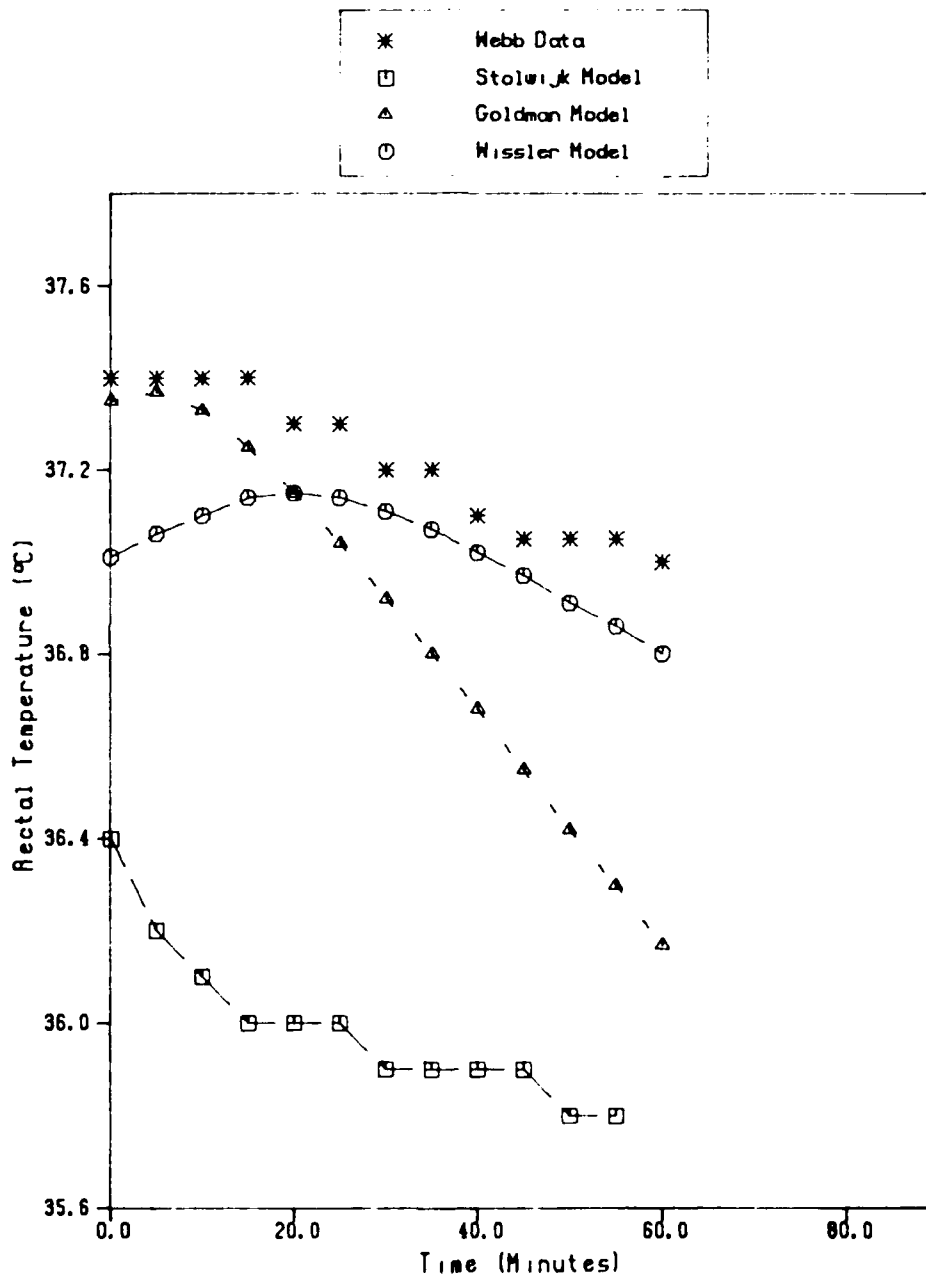
Total Heat Loss Rate
As a Function of Time
For Subject ST During an Immersion in 18 °C Water



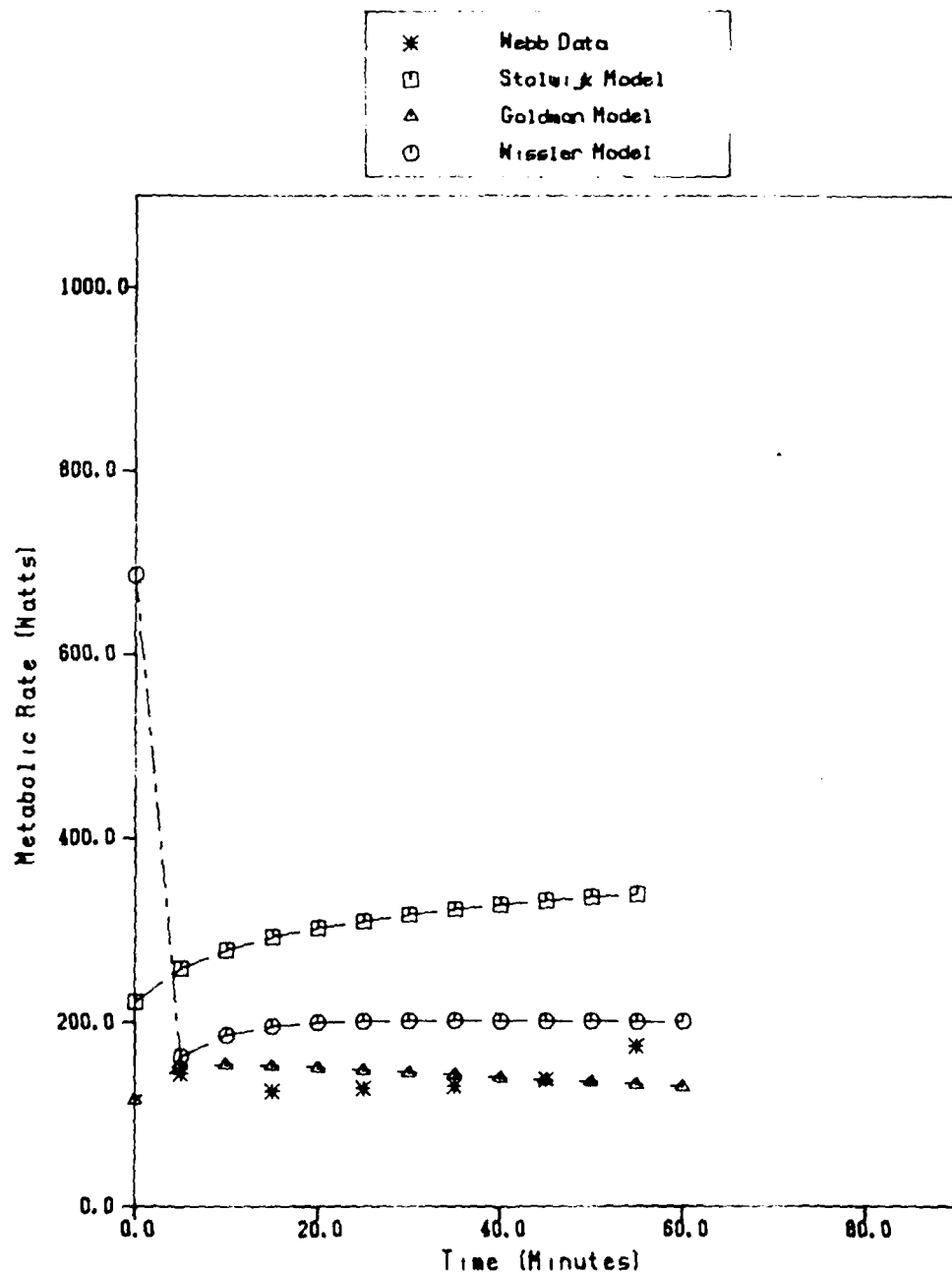
Tympanic Temperature
As a Function of Time
For Subject ST During an Immersion in 24 °C Water



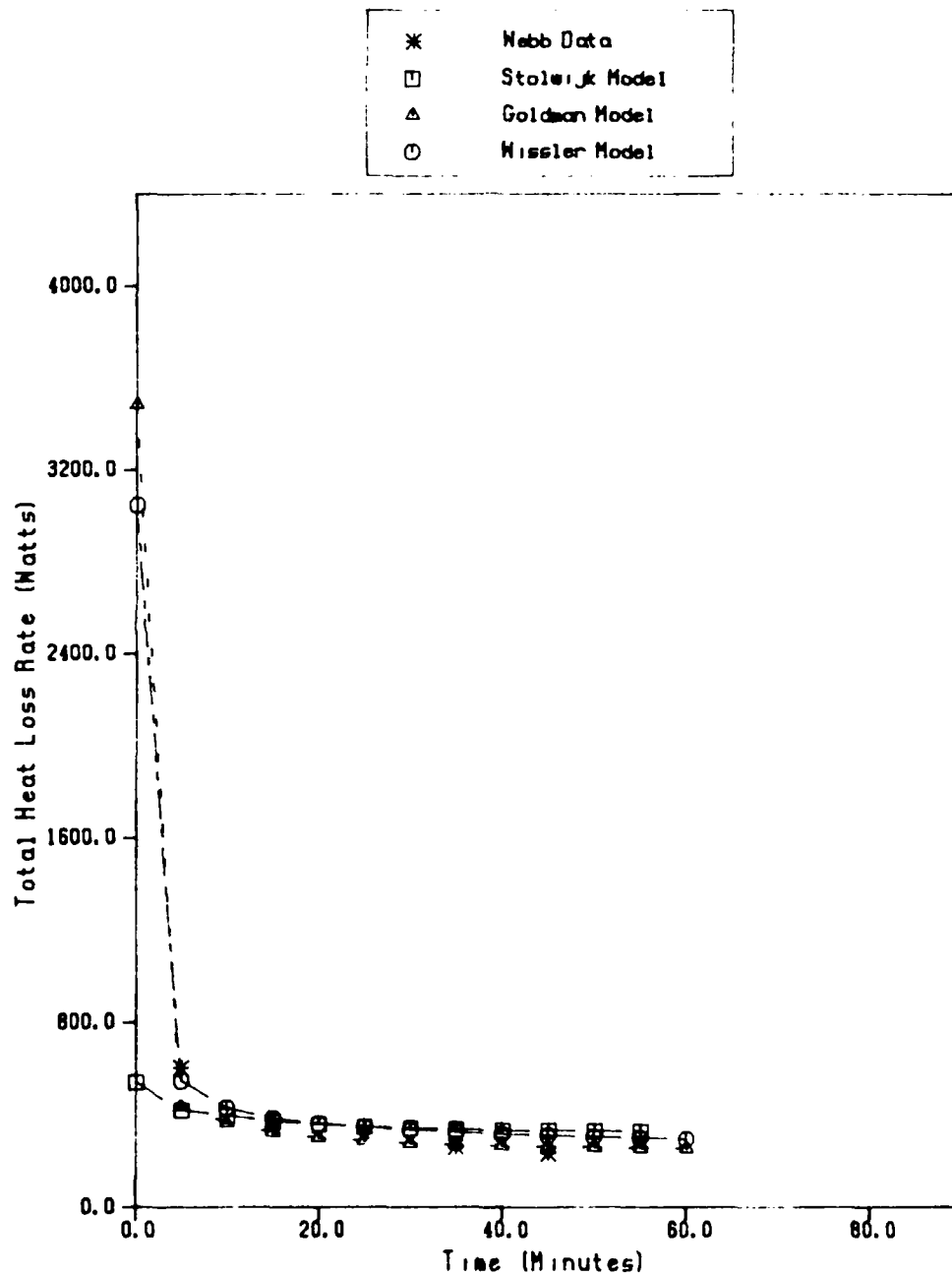
Rectal Temperature
As a Function of Time
For Subject ST During an Immersion in 24 °C Water



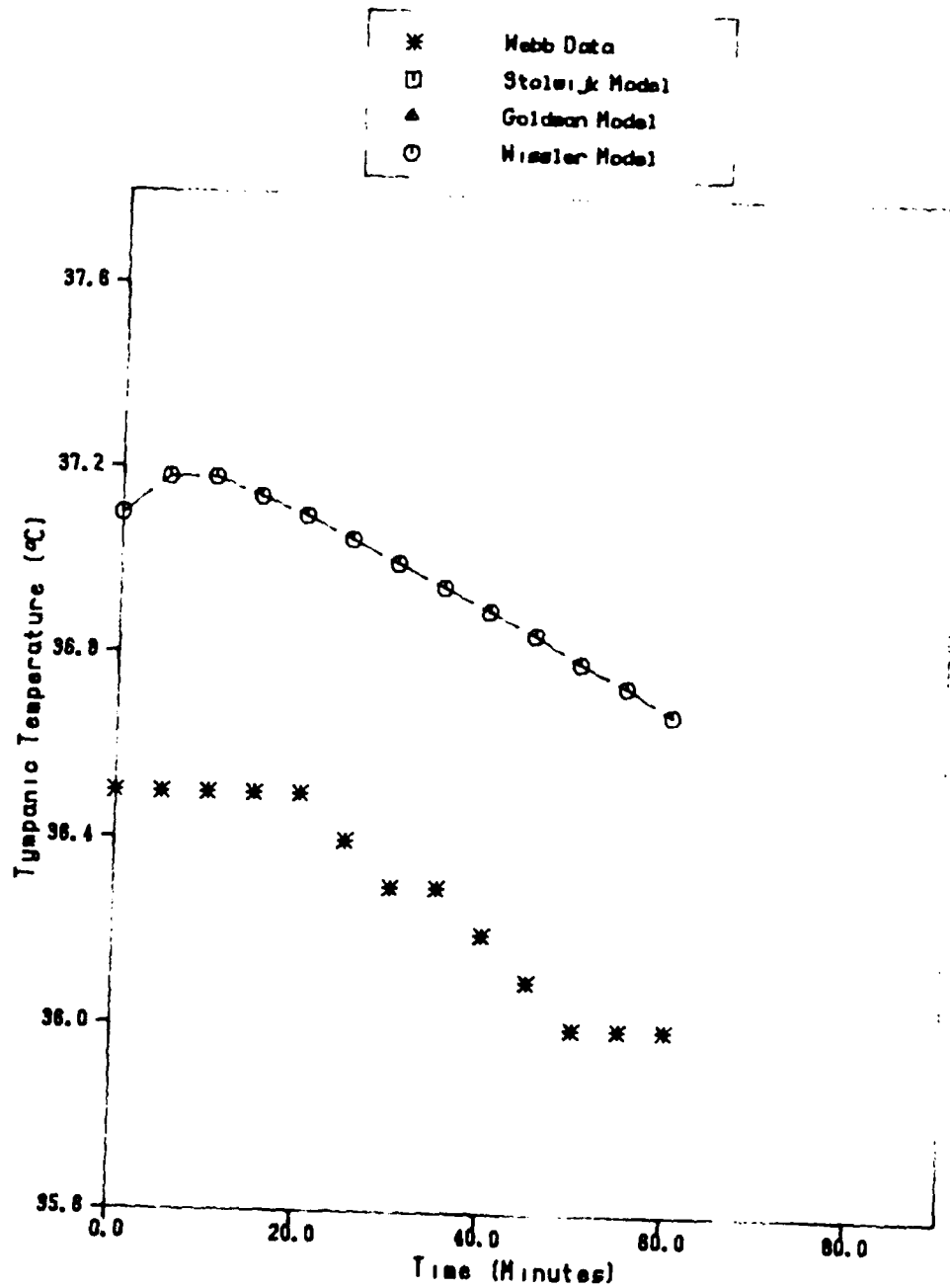
Metabolic Rate
As a Function of Time
For Subject ST During an Immersion in 24 °C Water



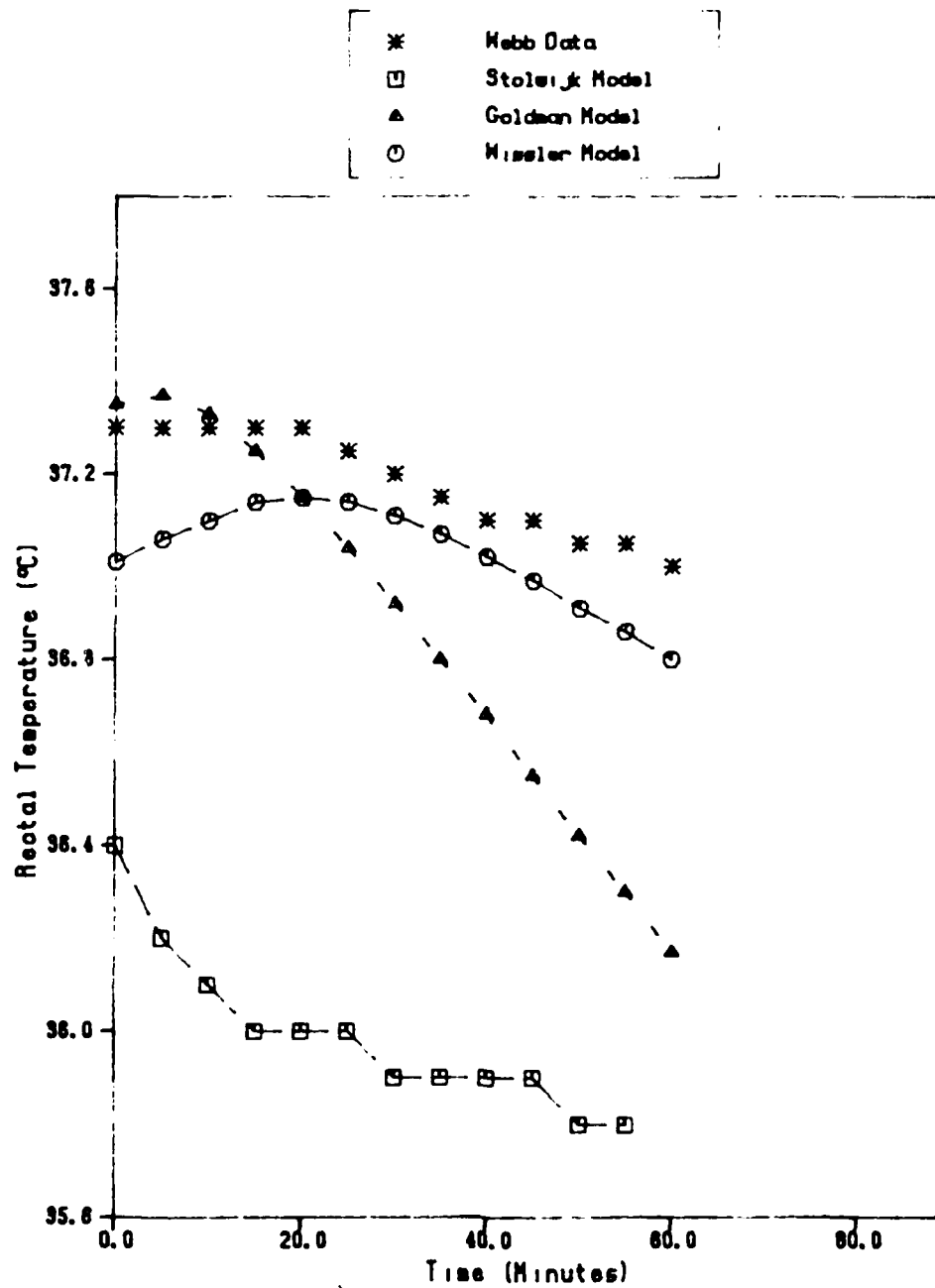
Total Heat Loss Rate
As a Function of Time
For Subject ST During an Immersion in 24 °C Water



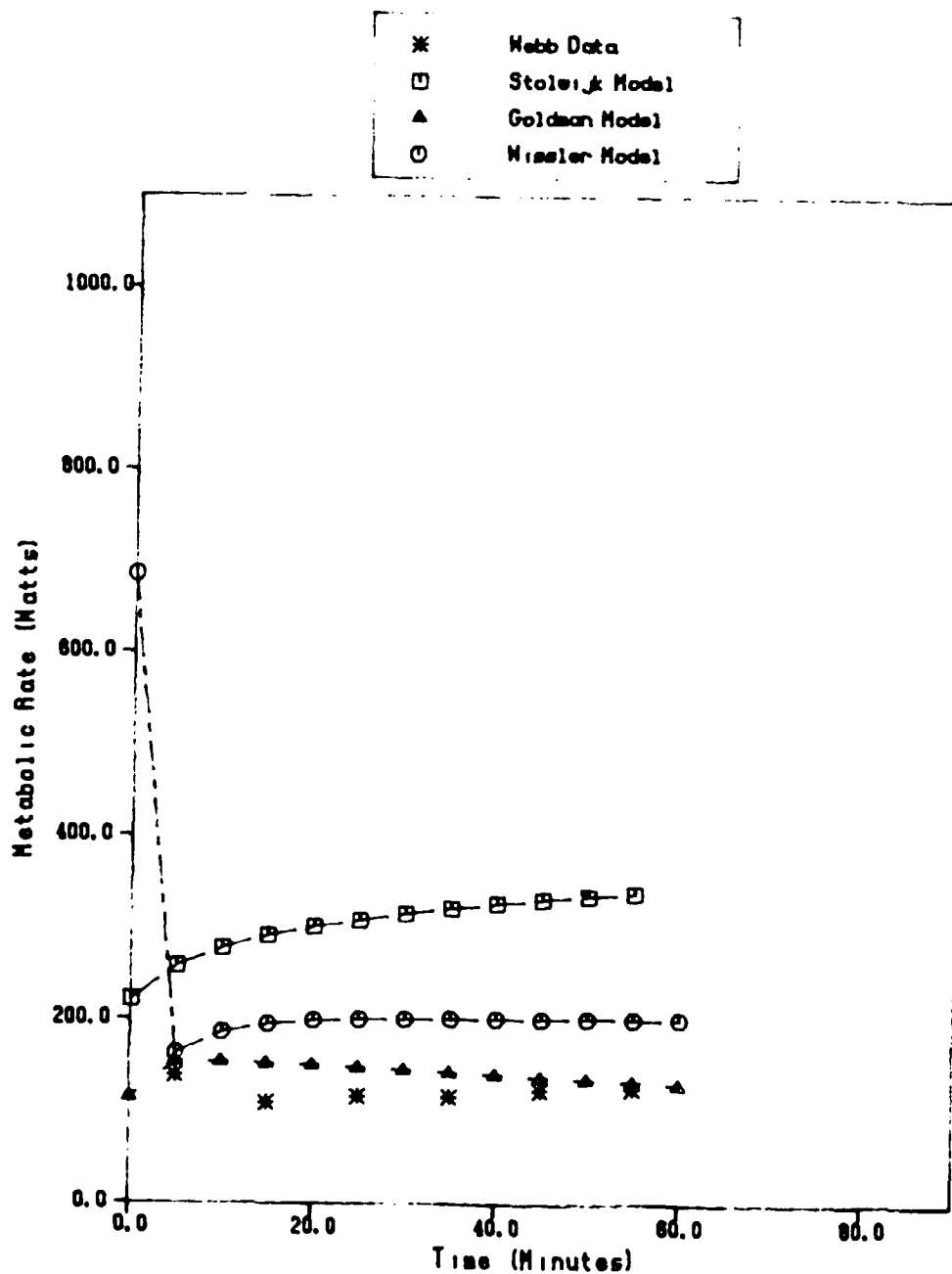
Tympanic Temperature
As a Function of Time
For Subject ST During an Immersion in 24 °C Water



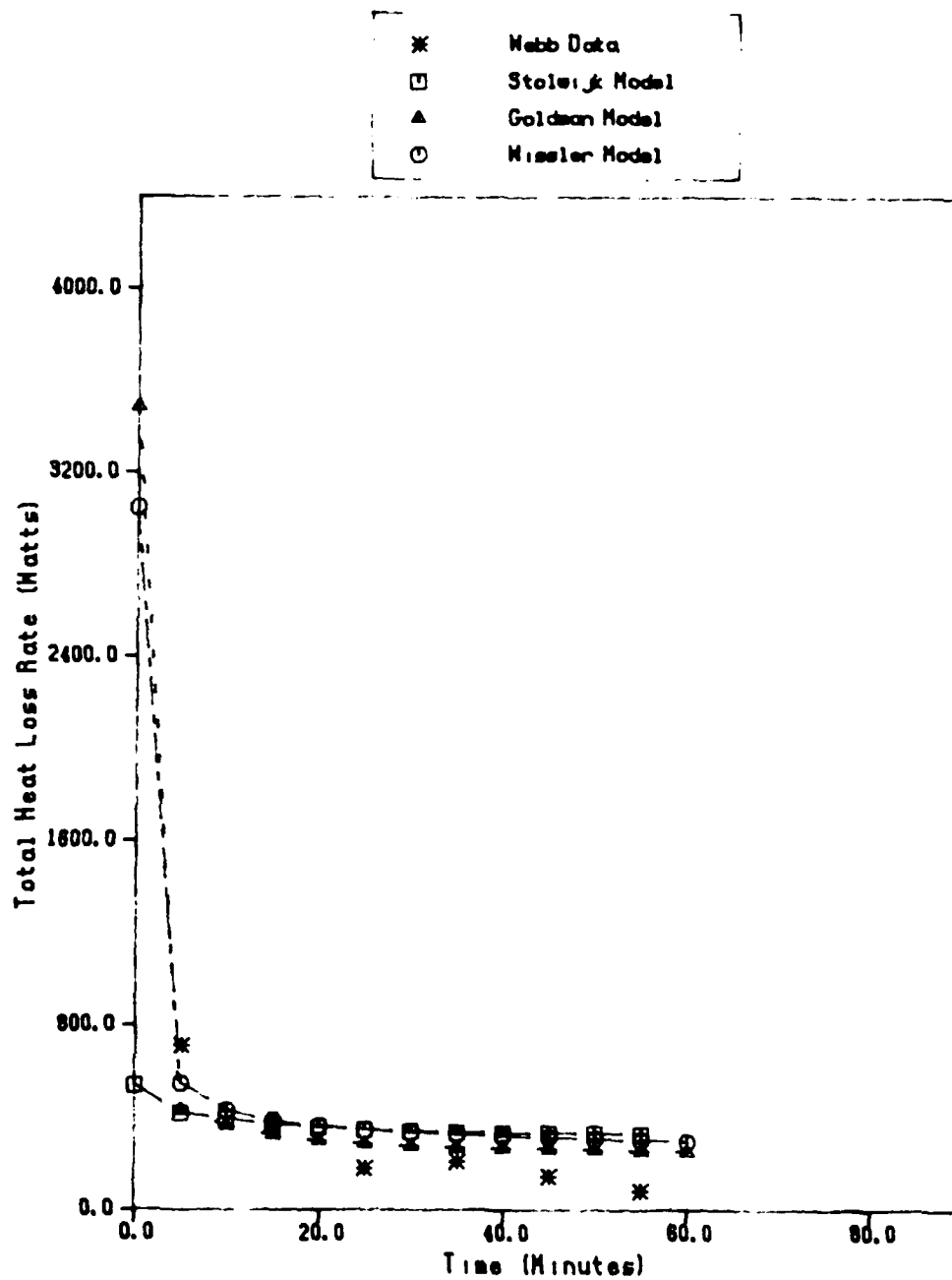
Rectal Temperature
As a Function of Time
For Subject ST During an Immersion in 24 °C Water



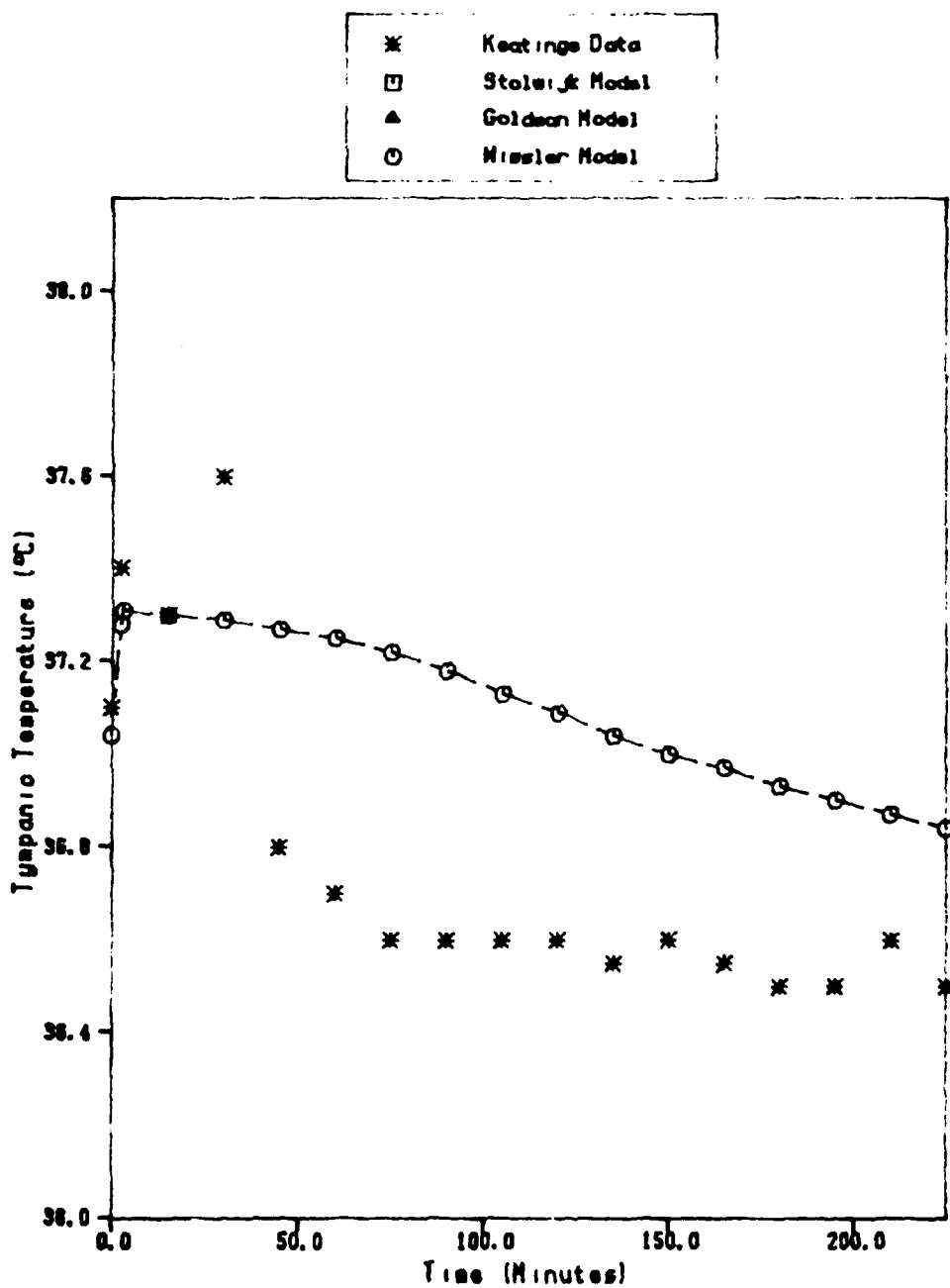
Metabolic Rate
As a Function of Time
For Subject ST During an Immersion in 24 °C Water



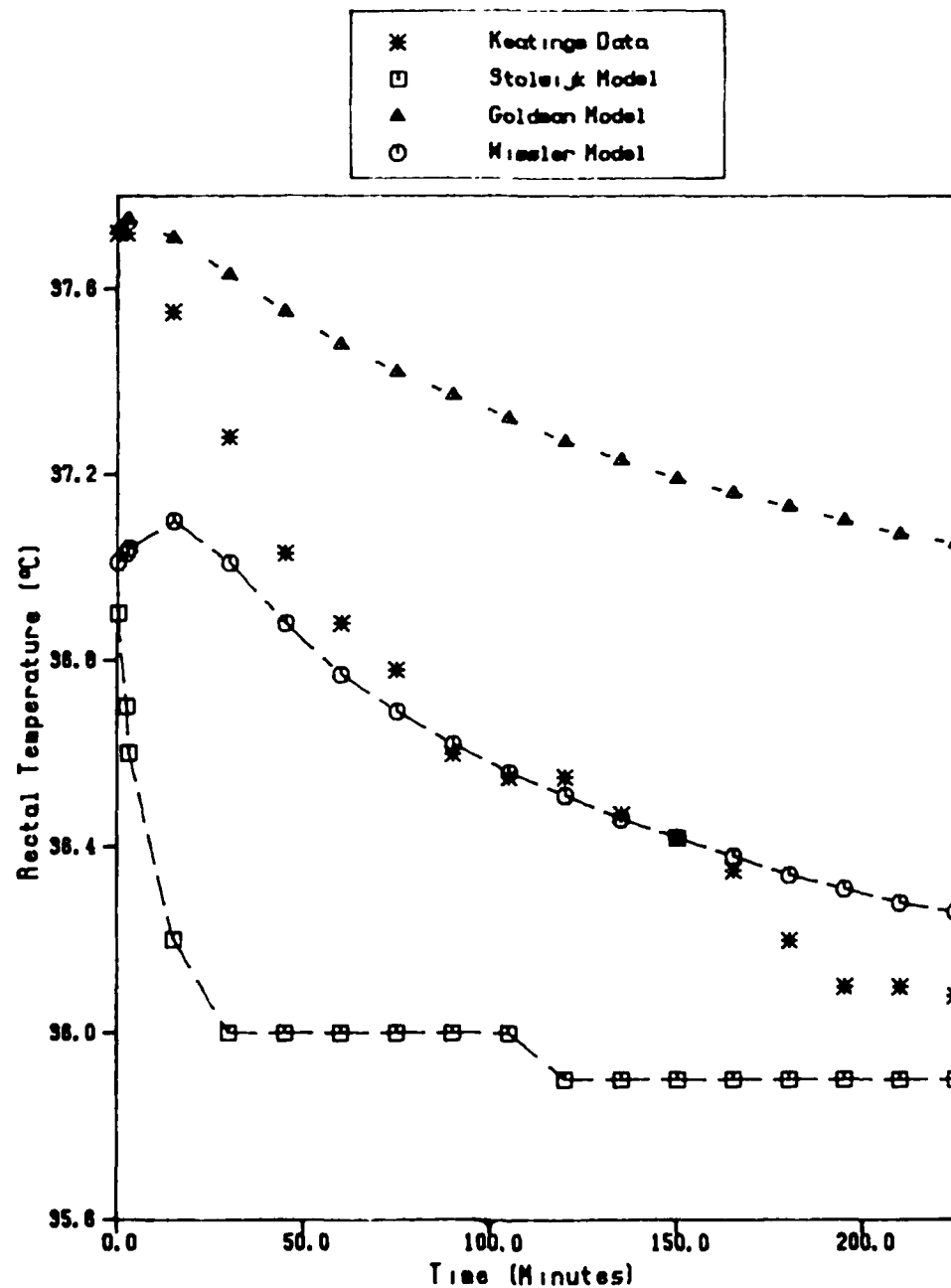
Total Heat Loss Rate
As a Function of Time
For Subject ST During an Immersion in 24 °C Water



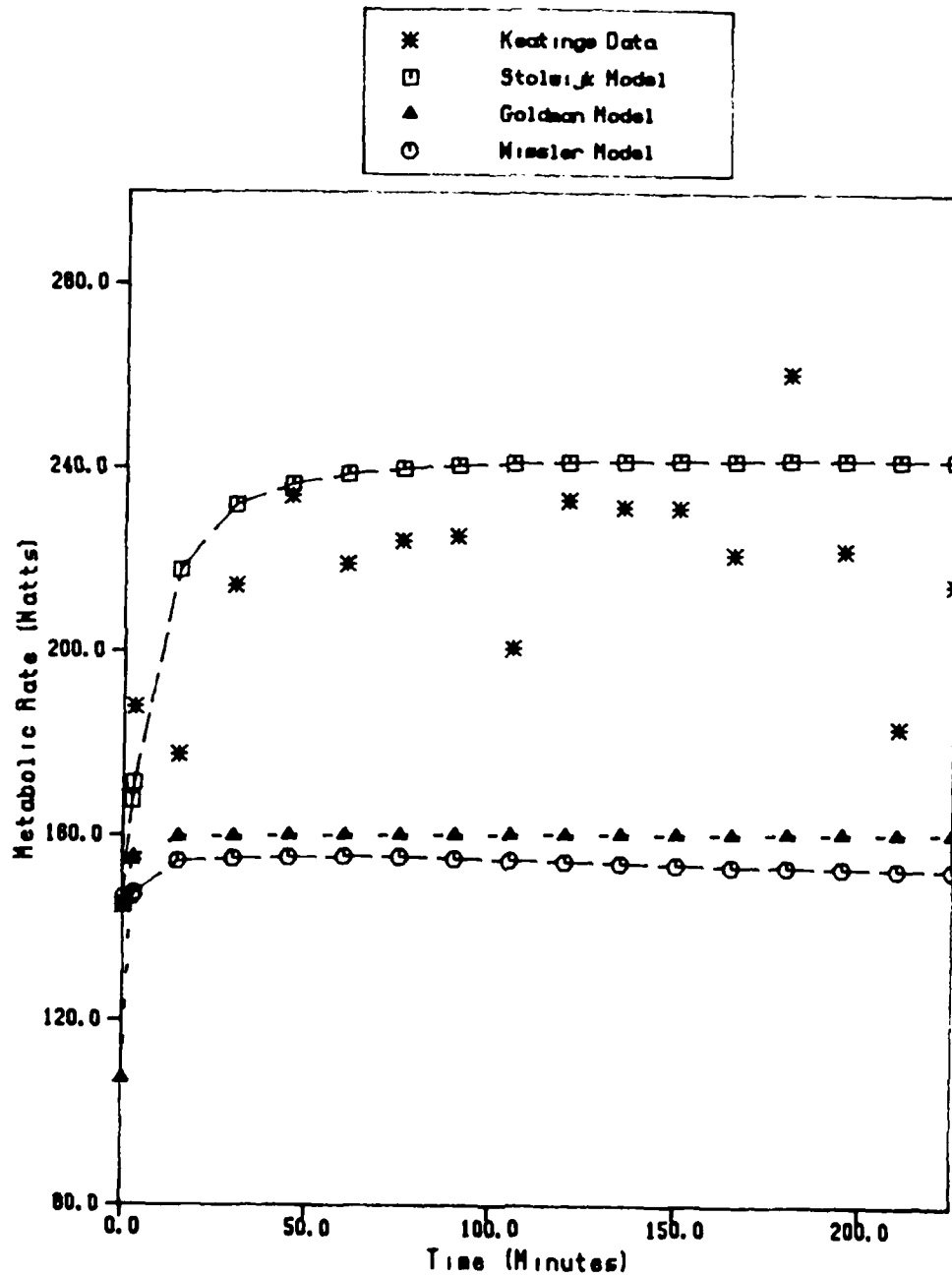
Tympanic Temperature
As a Function of Time
For Subject 1 Four Hour Immersion in 29°C Water



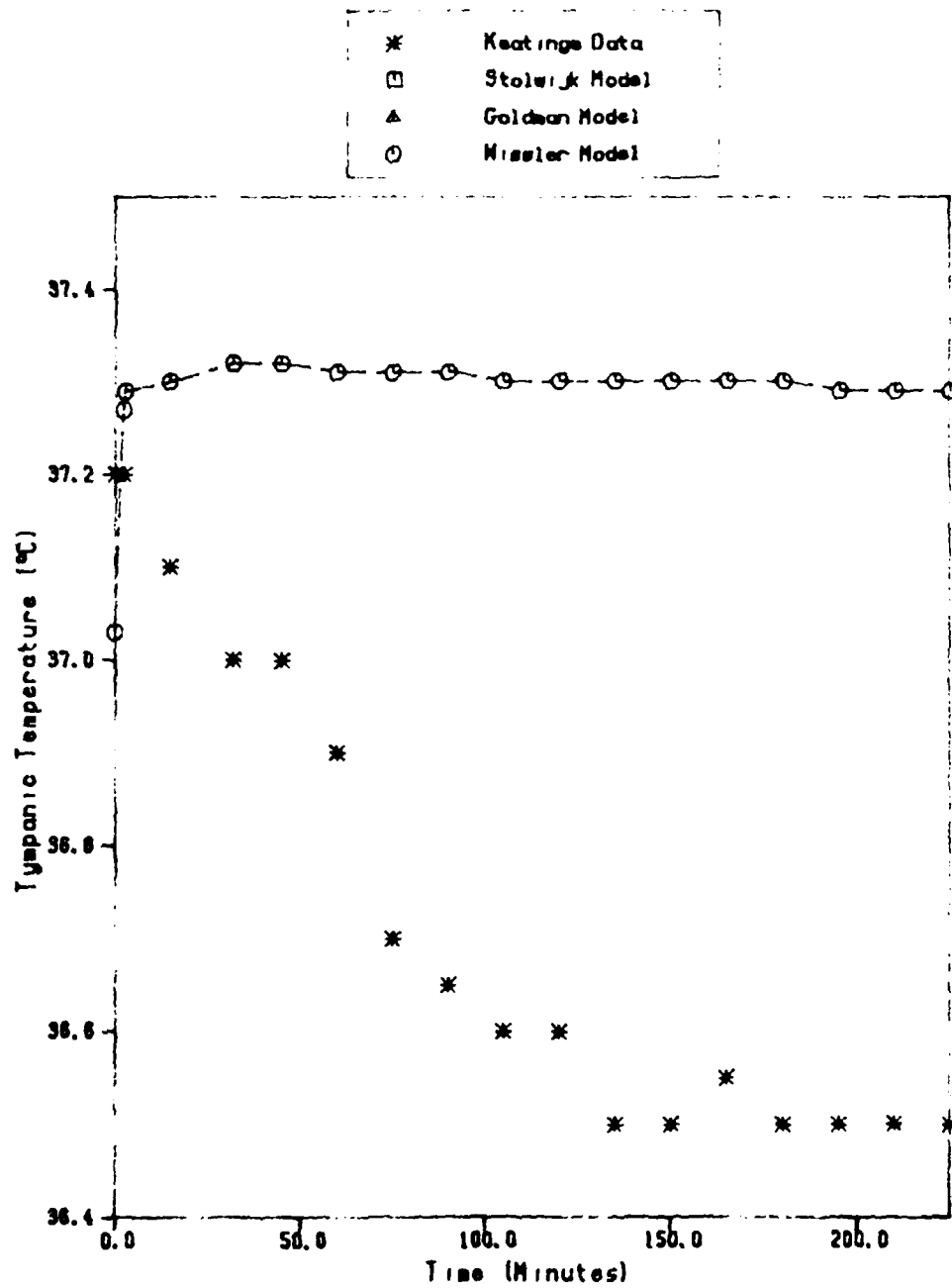
Rectal Temperature
As a Function of Time
For Subject 1 Four Hour Immersion in 29°C Water



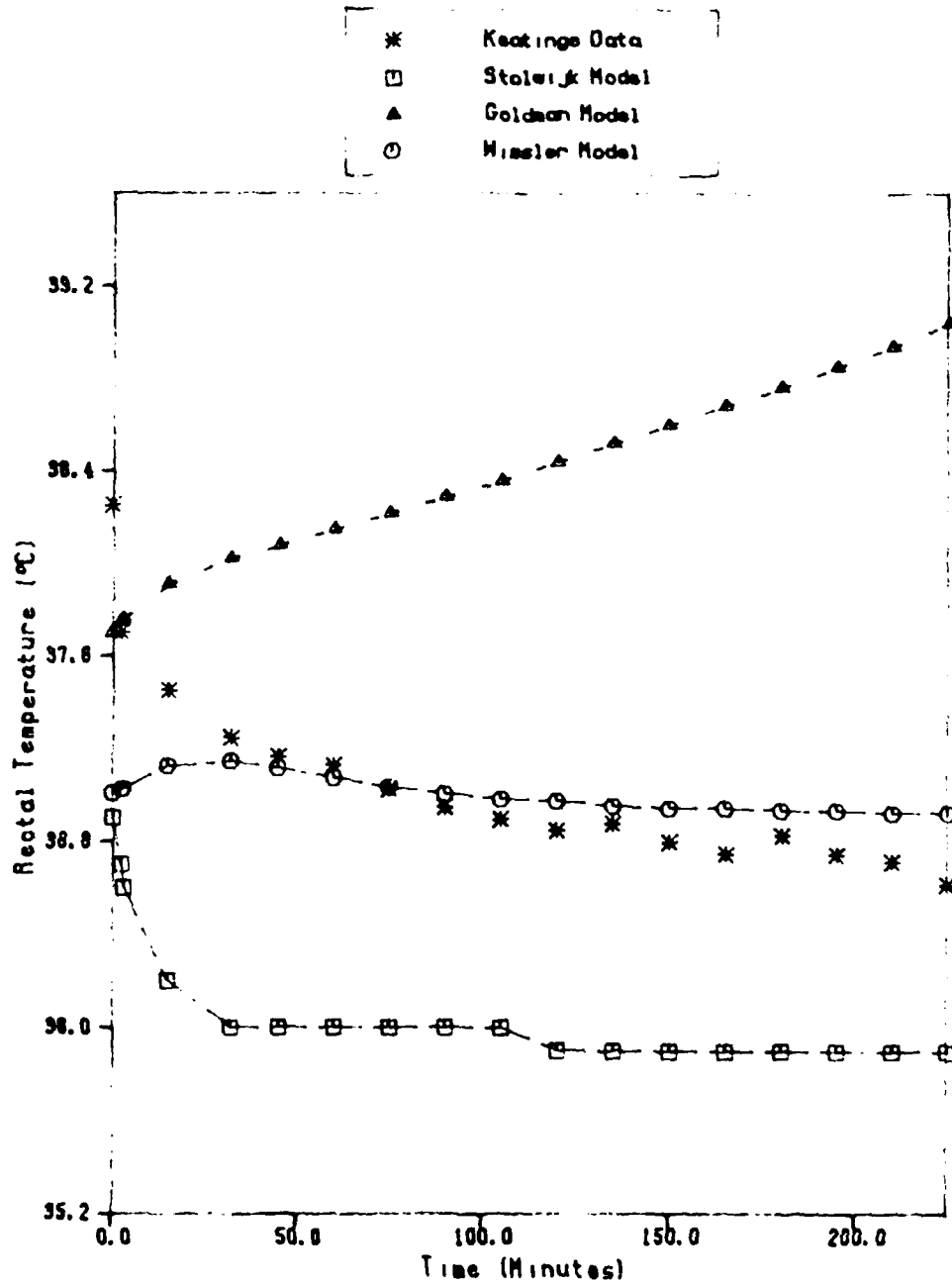
Metabolic Rate
As a Function of Time
For Subject 1 Four Hour Immersion in 29°C Water



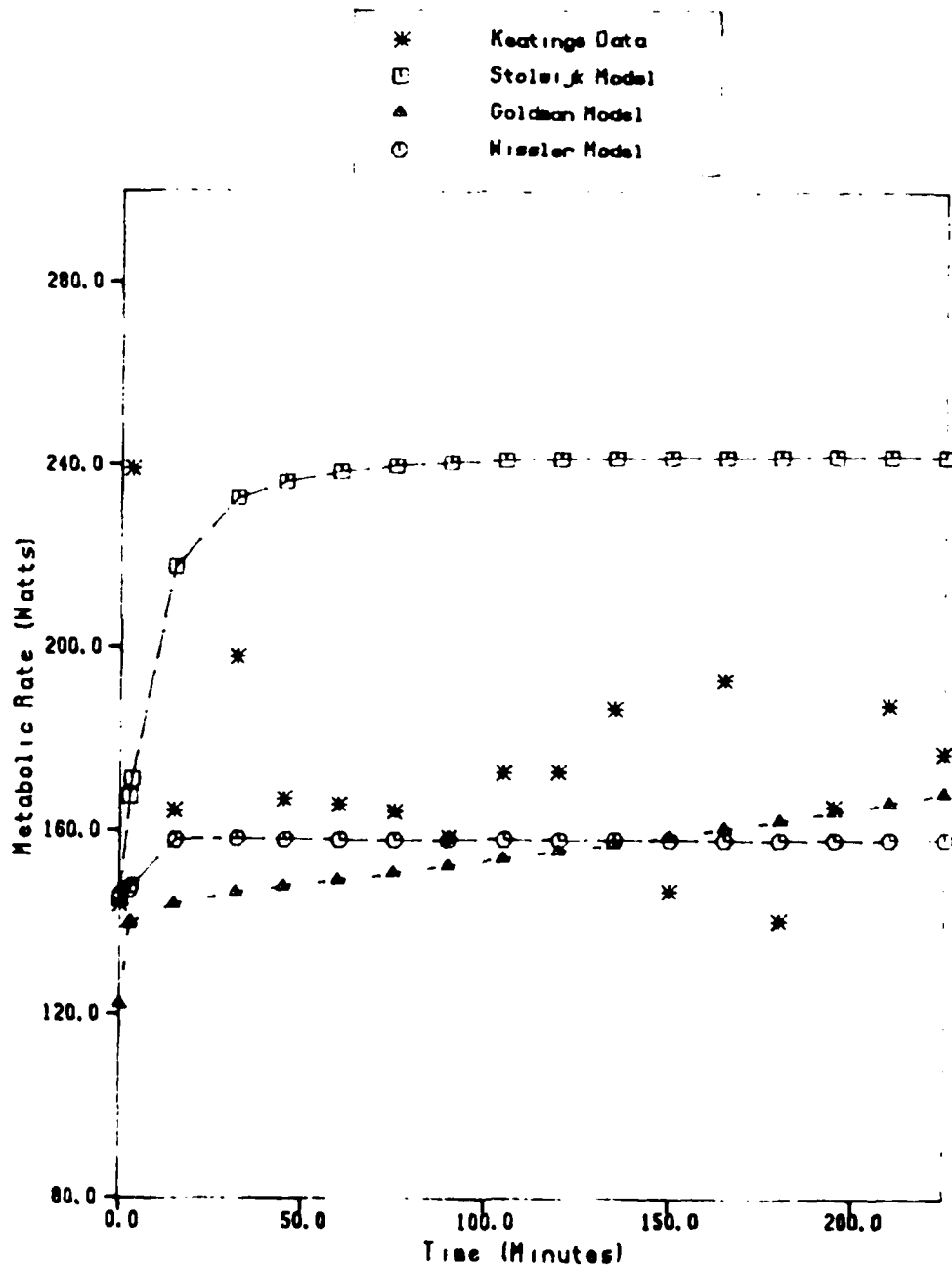
Tympanic Temperature
As a Function of Time
For Subject 2 Four Hour Immersion in 29°C Water



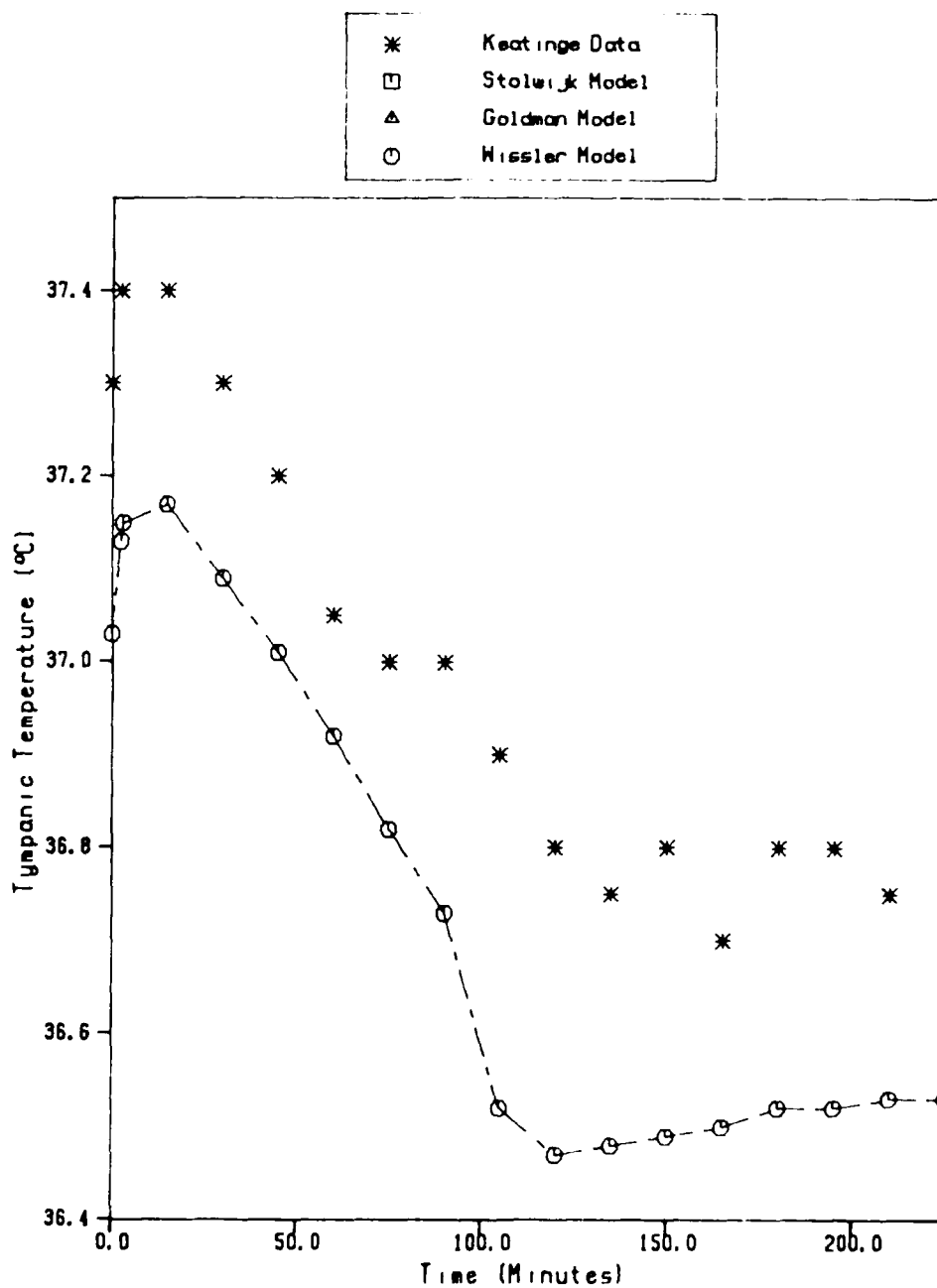
Rectal Temperature
As a Function of Time
For Subject 2 Four Hour Immersion in 29°C Water



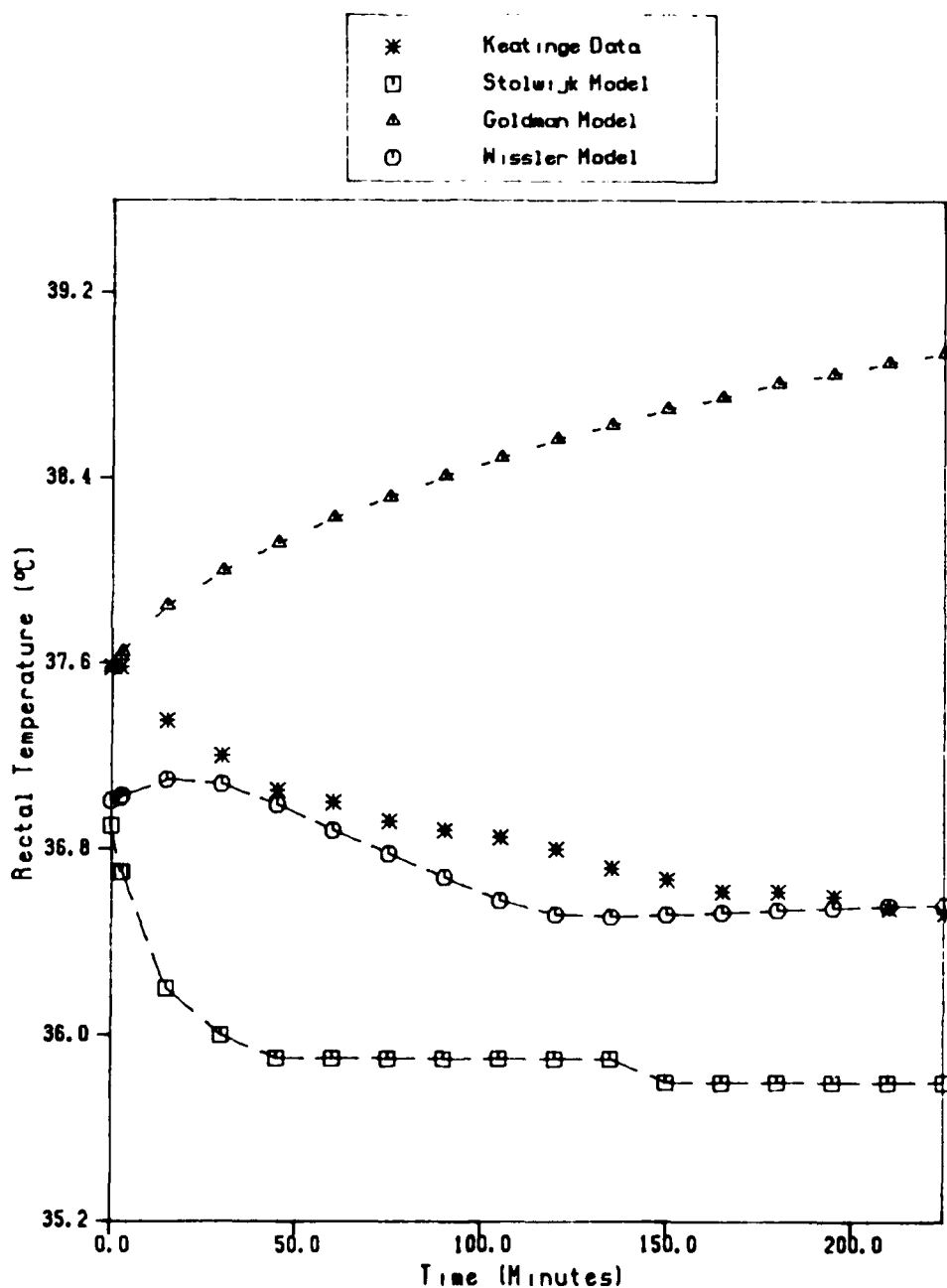
Metabolic Rate
As a Function of Time
For Subject 2 Four Hour Immersion in 29°C Water



Tympanic Temperature
As a Function of Time
For Subject 3 Four Hour Immersion in 29°C Water



Rectal Temperature
As a Function of Time
For Subject 3 Four Hour Immersion in 29°C Water



Metabolic Rate
As a Function of Time
For Subject 3 Four Hour Immersion in 29°C Water

