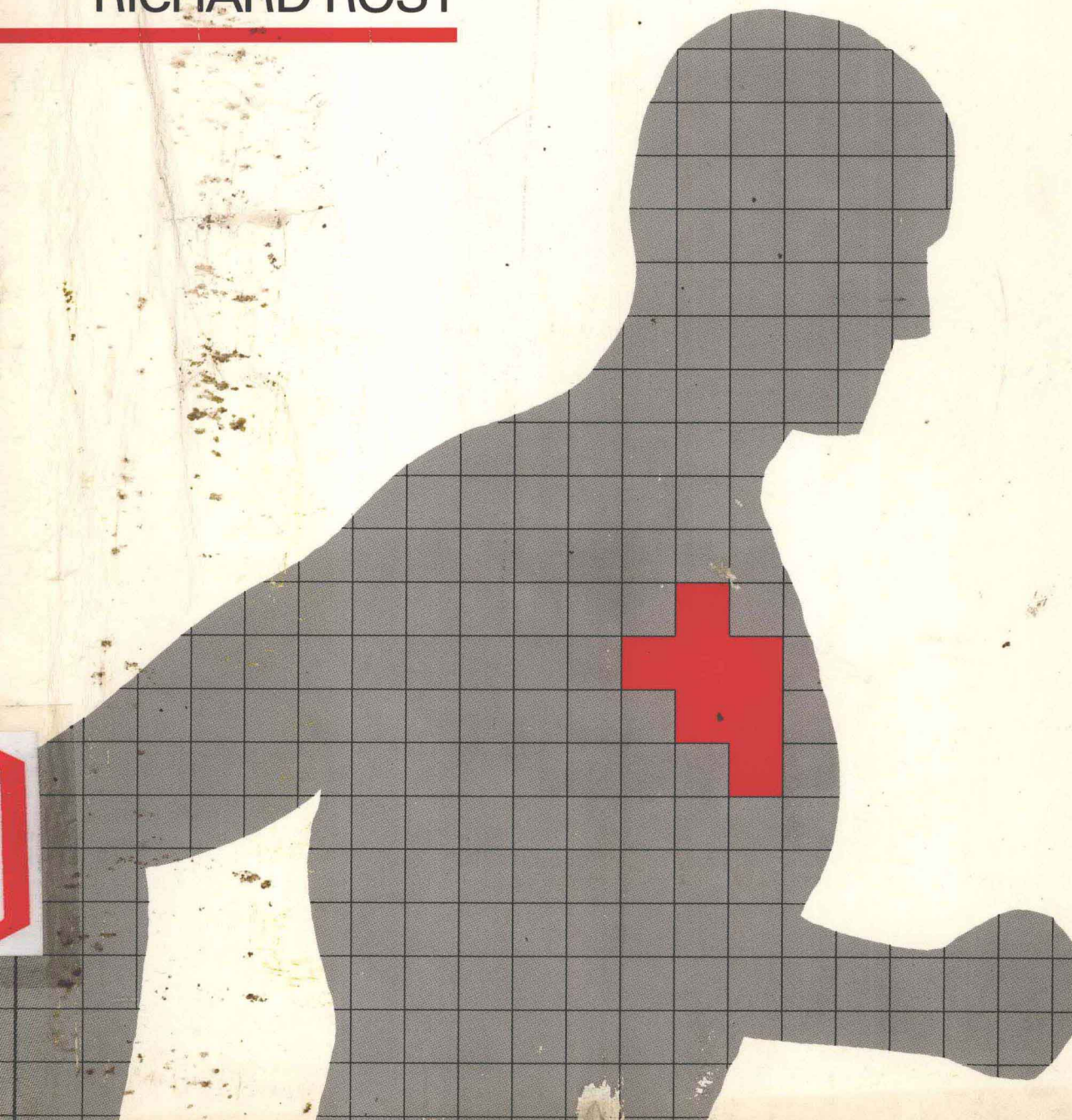
# ATHLETICS AND THE HEART

RICHARD ROST



# Athletics and the Heart

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YEAR BOOK MEDICAL PUBLISHERS, INC.
Chicago • London

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This book is an authorized translation from the German edition published and copyrighted 1984 by Perimed Fachbuch—Verlagsgesellschaft mbH, Erlangen, Germany. Title of the German edition: *Herz Und Sport.*

**Library of Congress Cataloging-in-Publication Data**

Rost, Richard.
Athletics and the heart.
Translation of: Herz und Sport.
Includes bibliographies and index.
1. Heart. 2. Sports—Physiological aspects.
3. Exercise—Physiological aspects. 4. Athletes—
Health and hygiene. I. Title. [DNLM: 1. Exertion.
2. Heart—physiology. 3. Sports. 4. Sports Medicine.
WG202 R839h]
RC1236.H43R6713 1986 612'.17 86-13179
ISBN 0-8151-7397-0

Sponsoring Editor: Linda A. Miller
Manager, Copyediting Services: Frances M. Perveiler
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Production Project Manager: Max Perez
Proofroom Supervisor: Shirley E. Taylor

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# 1

# Introduction

The relationships between the heart and athletics are manifold, fascinating, and of increasing practical significance for all those who wish to become involved in this area. Even the etymological derivation of the word "heart" is significant. Not all cardiologists are aware of the fact that the subject of their professional and scientific interest received its name from an athletic term by our forefathers. According to Boyadijan, the word "heart" has the same indogermanic root as the word "hart" (male deer). This could perhaps be freely translated as "jumper," so named by early man because the heart was "jumping" in the chest after heavy exertion.

The amazing range of performance that the heart is capable of can best be seen in athletics. The work load of the heart is quadrupled and the cardiac output is raised from 5–6 L/minute to 20–25 L/minute. The trained heart can raise its performance eightfold to 40 L/minute. In the resting state cardiac action has to function only at the lower one-fourth of its potential.

Such a functional point of view has wide implications in areas other than the heart of the healthy, active person. Athletics are increasingly recognized as a therapeutic modality for cardiac patients and as a prophylactic measure in the prevention of cardiocirculatory problems. This is shown in the enormous increase of ambulatory coronary activity centers. In the past 5 years, their number has risen from 80 to 800 in the German Federal Republic alone. Furthermore, the significant advances in cardiac surgery have placed into an entirely new perspective the importance of athletics in the management of cardiac patients. Coronary bypass surgery, valvular replacement, and other surgical interventions improve the performance potential of the failing heart, but can come to full fruition only when physiotherapy and rehabilitation improve the performance of the entire body.

The increasing importance of athletics in the prophylaxis of cardiovascular disease and in the treatment of the cardiac patient has been sufficiently recognized so that interest in this area is no longer limited to a small group of specialists as it was in the 1960s. For decades, the sports-cardiologists were interested primarily, and with few exceptions, in the peculiarities and evaluation of the athlete's heart. There were only a few cardiologists who recognized the importance of athletics for the sick heart. These discussions, which extend almost over a century, have resulted in a number of findings that today form the basis for athletic activities in the cardiac patient.

Clinicians have always regarded the large and efficient heart of the athlete with suspicion, and there are cardiologists who even today consciously or in-

stinctively consider the athlete's heart as a form of cardiomyopathy. Such a view is understandable when one looks at the characteristics of these hearts, e.g., the increase in size, the resting rate at or below 30 beats per minute, the appearance of unusual electrocardiograph (ECG) findings, such as functional third degree atrioventricular (AV) block, and reentry disturbances, which can simulate the findings seen in infarction. In spite of such findings, which raise a question about the range of physiologic adaptation of the heart, it must be emphasized that the decade-long discussions concerning the athlete's heart have proved only one thing: we are dealing with healthy and particularly effective hearts. The above-mentioned peculiarities of the athlete's heart and the importance of athletics for the cardiac patient make it necessary that the relationship between the heart and athletics become a major concern for the cardiologist. This need is not considered adequately in the standard textbooks on classical cardiology. The athlete's heart is either not mentioned at all or is largely discussed in a negative fashion. This is particularly true in the Anglo-American literature. In the more than 2,000-page textbook on cardiovascular medicine by Braunwald (1980), the words "athletics" or "athlete's heart" do not appear in the index. The ultimate in denial is reached by Friedberg (1972) who claims that the cardiac enlargement in the athlete may, among other interpretations, be due to overload on a heart previously damaged by syphilis.

Such gross misrepresentations cannot be found in the more recent German literature. This can certainly be attributed to Reindell (1960), who was an outstanding champion for the athlete's heart and who devoted the scholarly activities of a lifetime to the recognition that the athlete's heart was a positive adaptation phenomenon. Thanks to him and others, functionally oriented considerations of load and efficiency have been introduced into modern cardiology that prior to this time were limited to "resting phase" considerations. In consequence, stress ECG, ergometry, and pulmonary artery pressure measurements under stress can be considered as standard procedures.

In spite of this progress, even today the scientific and practical significance of the relationship of athletics and the heart gets all but short shrift in German textbooks on cardiology. It must be remembered, in this context, that the coronary activity clubs developed largely outside the mainstream of clinical cardiology. To date, clinical cardiology has not paid enough attention to the impact of physical stress on the healthy and sick heart. For the clinical cardiologist, the cardiac patient is still a person lying in bed whose functional ability can be precisely analysed in a resting state, and whose differential response to different stress (running, swimming, weight training) need not be considered outside the hospital.

This lack of involvement of the clinical cardiologist with the effects of physical activity on cardiac function illustrates one of the general problems of sports medicine. In spite of the considerable importance granted today by society to both competitive sports and to mass participation in athletics, it has been impossible so far to establish an independent department of sports medicine at any university medical school. The physician is frequently asked questions by a patient who wishes to engage in physical activity because of his heart; questions that the physician can only answer unsatisfactorily.

The patient who engages in athletics for the benefit of his heart usually does so to prevent cardiocirculatory problems. The motivation for this activity is provided by mortality statistics that show cardiovascular disease causing more than 50% of all deaths, and by advertising slogans such as, "Run away from your heart attack!" Unfortunately, patients are also aware of the opposing opinions in this

area, which are manifested by such contrary advice as, "Engage in sport *and* stay healthy" and "Engage in sport *or* stay healthy."

The motivation to write this monograph was provided by numerous colleagues who, during various postgraduate courses, expressed a wish to have a single volume in which they could review the relationships between athletics and the heart. Such a volume is currently not available in the literature. Since the last, and now classic, presentations of the Freiburg Group (Reindell, 1960) numerous new aspects of the athlete's heart and the general value of athletics to health have emerged. Even though the presentation of Reindell was considered to close the book on any further discussion of the athlete's heart, new noninvasive techniques, e.g., echocardiography, have brought new perspectives to bear on this issue. The newest developments in the area of athletics and cardiac patients were unforeseen 20 years ago.

For this reason it seems appropriate, once again, to determine the status of sports cardiology. To determine this status, it is necessary not only to review the scientific developments in this field, but also to realize the need to examine the relationships between athletics and the heart from a practical, medical point of view. An attempt will be made to answer the questions asked by patients from the practicing physician and which the latter usually passes on to the specialist in sports medicine. These answers will be approached from a scientific point of view.

The following subject areas will be discussed in detail:

1. The *way the heart functions* under the different forms of physiologic stress. The wide variety of athletic activities makes it understandable that the responses of the cardiocirculatory system will differ considerably from each other. The physician who is familiar with these relationships can respond to the different signs and symptoms specifically on the basis of etiology.

2. The *adaptation responses* of the heart under physiologic stress. The athlete's heart will be described from the perspective of historical development and on the basis of its anatomic and clinical characteristics.

3. The *possible cardiac incidents* related to athletic activity. The most dramatic and affecting of these incidents is the sudden cardiac arrest. It is for this reason that particular medical attention must be directed toward the question of whether physiologic load can really lead to cardiac damage. The lay person usually accepts the health benefits of athletics as a matter of course but it is the duty of sports physician to determine the limits beyond which activity no longer contributes to health.

4. The value of athletics for the *cardiac patient* as a central issue in sports cardiology.

5. The necessary interaction of the therapeutic modalities, particularly the physical activity of the cardiac patient and the *pharmacologic management* of the same individual. While the relationship between these two therapeutic modalities has not been properly elucidated, it is desirable to highlight its particular significance.

It should be emphasized that athletics and medicine must work as partners in the care of cardiac patients. For that reason this volume is dedicated to the physician who is deeply concerned not only for the heart of the athletes but also for the heart of his other patients who engage in physical activity. This volume should be of particular benefit to athletic coaches and trainers who spend their life in this activity and whose duty it is to fit the trainees' performance into the framework established by the medical considerations.

# 2

# Heart Action Under Physical Exercise

> The human body is designed for muscular activity which can raise basal metabolism by a factor of 15 to 20.
>
> P. Astrand, 1974

The range of circulatory performance becomes manifest only under physical exercise. The different types of exercise result in correspondingly different cardiocirculatory responses. The diversity of these reactions is generally only poorly known.

In order to illustrate this diversity and these contradictions, some extreme variations will be presented as follows: It is generally assumed that exercise raises the blood pressure and the cardiac output. In the untrained person this rise can be four- to fivefold; in the trained person the rise can be as much as eightfold when compared to the resting volume. These changes can take place, but are certainly not inevitable. Under maximal effort of a certain type, e.g., under maximal power load, the cardiac output can drop to half of the resting value. Mean blood pressure can remain unchanged, e.g., running on a level surface. It can also drop under certain conditions of maximal effort, or it can rise by several thousand mm Hg, for instance, as in diving.

To be able to classify the various responses and to try to derive some uniformly applicable general laws concerning the various responses to physical effort, the two basic forms of muscular activity, isotonic and isometric contractions, will be taken as the starting point in the subsequent discussion. The shortening of the muscle fibers generally determines the dynamic work of the muscle, while the development of tension of the fibers determines the static holding work of the muscle. The two components of cardiac work, pressure and volume, will be discussed in the context of the above two forms of muscular activity. The data presented were partly generated by the author and partly derived from the literature. In as much as they are considered incomplete the reader is referred to an earlier, more complete presentation (Rost, 1979).

## THE WORK OF THE HEART UNDER A DYNAMIC LOAD

The dynamic load is characterized by a rhythmic contraction and relaxation of muscle. In the athletic world it can be found in running, cross-country skiing, bicycle riding, swimming, and rowing. This sequence was selected on purpose. In the first types of exercise, the muscle contraction must closely approximate the

physiologic nature of isotonia. In the subsequent types of exercise, an increasing force component becomes evident. Thus, in contrast to running, bicycle riding has a moderate, and rowing a pronounced, force component.

The dynamic contraction of the muscle may also manifest a certain, variable degree of tension development. This isometric muscle activity leads to a compression of extensive vascular fields, which clearly influences and affects the circulatory response, as discussed below. Let us start with a dynamic load, with minimal force development, as seen in running on level ground.

The *circulatory goal* is to transport more blood, i.e., to increase cardiac output. The extent of this rise can be determined accurately in a given individual, since it depends on the intensity of the performance, although it can be modified by such additional factors as body build, sex, age, and pathologic conditions.

## The Cardiac Output

To be able to determine the increased work, the heart has to perform for a given effort, it must be assumed as a basic premise, that identical efforts require identical energy outputs. The efficiency of muscle is neither age- nor sex-dependent and cannot be improved by training. The unit of measurement for bodily performance is the watt. Every increase of one watt requires an increase in oxygen transport of 12 ml/minute. The energy requirement thus increases linearly with the intensity of load (Fig 2–1).

On the other hand, the pumping action of the heart increases linearly with the energy utilization. The relationship between the increase in oxygen uptake and cardiac output can be expressed as the so-called "exercise factor." It corresponds to the required increase in cardiac output for every 100 ml increase in oxygen uptake and usually lies about 0.61. The precise relationship is expressed in the equation described by Holmgren (1956):

$$\text{Cardiac output (L/min)} = 7.03 + 0.0058 \times VO_2 \text{ (ml/min)}$$

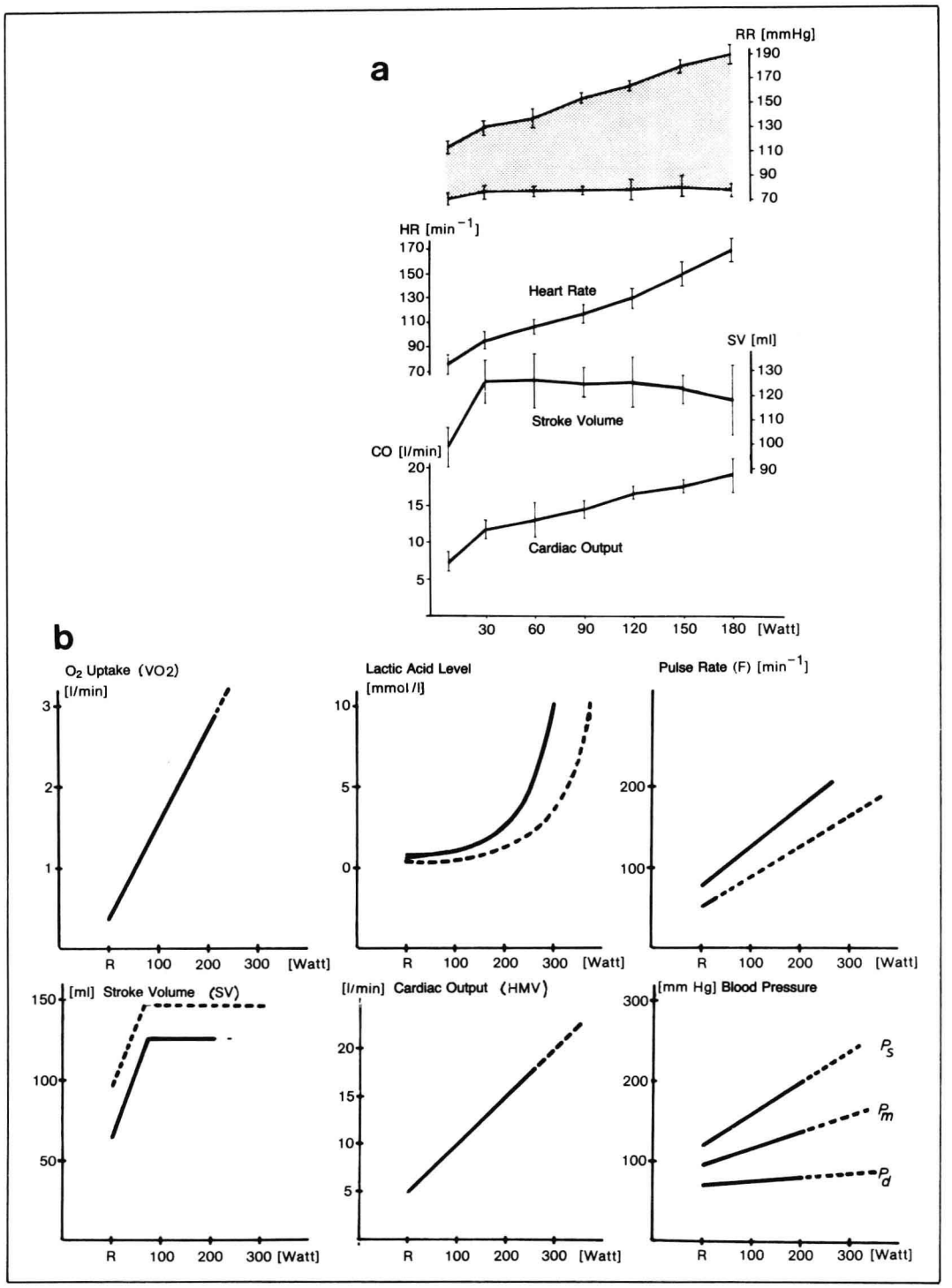
This means that for a performance of 100 watt, 1.21 L/minute additional oxygen is required. To deliver this amount, the heart must increase its output by 7.2 L. Starting with a resting output of 6 L this means that the minute volume must rise to approximately 13 L for a 100-watt performance. A 200-watt performance requires a cardiac output of 20 L. We can also estimate the output required on the basis of the speed of running. In level running the power cannot be expressed in watts, since according to the following formula, at least in theory:

$$\text{Power} = \text{Force} \times \text{distance/time}$$

Therefore, since no height has to be overcome, the power must equal zero. The required minute volume, however, can be derived from the required energy utilization. Oxygen uptake can be calculated for a given speed of running, by an equation according to Pugh (1970):

$$VO_2 \text{ (ml/min} \times \text{kg)} = 4.25 + 2.98 \times \text{speed (km/hr)}$$

The *regulation of cardiac output* on the basis of demand is one of the central topics of circulatory physiology under conditions of physical exercise. There are as many theories as there are investigators. In principle, all these theories can be divided into two groups. According to one theory, the heart as an active muscle regulates itself. If an increase in flow is required, the heart increases its output and this secondarily increases venous return. The contrary view claims that the heart is the "servant" of the circulation, and that the cardiac output is largely determined by the amount of venous return. Accordingly, cardiac performance is de-


a
RR [mmHg]
190
170
150
130
110
90
70
HR [min⁻¹]
170
150
130
110
90
70
Heart Rate
SV [ml]
130
120
110
100
90
Stroke Volume
CO [l/min]
20
15
10
5
Cardiac Output
30 60 90 120 150 180 [Watt]
b
O2 Uptake (VO2)
[l/min]
3
2
1
R 100 200 300 [Watt]
Lactic Acid Level
[mmol/l]
10
5
0
R 100 200 300 [Watt]
Pulse Rate (F) [min⁻¹]
200
100
R 100 200 300 [Watt]
[ml] Stroke Volume (SV)
150
100
50
R 100 200 300 [Watt]
[l/min] Cardiac Output (HMV)
20
15
10
5
R 100 200 300 [Watt]
[mm Hg] Blood Pressure
300
200
100
Ps
Pm
Pd
R 100 200 300 [Watt]

termined by the conditions in the periphery.

Such a division appears to be artificial. Factors that affect the cardiac output automatically alter venous return and vice versa. The control system of the circulation is a complex equilibrium, in which the metabolic requirements of all the organs participate, and that is affected by numerous humoral, nervous, and mechanical control mechanisms. The change in one factor leads retroactively to changes in other systems.

Possible control mechanisms were identified in cortical reflexes, humeral impulses, chemoreceptors, and pressoreceptors. Cortical impulses do play a role in the adaptation to load conditions as shown in the *prestart reaction*. Runners show an appreciable increase in pulse rate and blood pressure even before the start, which provide an increased blood supply to the muscles. On the other hand, such a mechanism cannot provide a precise control under load conditions.

The theory of adaptation on the basis of *reflex self regulation* by Koch (1931) was previously widely accepted. According to this theory, the start of any physical exercise or increase of intensity leads to a dilatation of the vessels in the periphery and to a central fall in pressure. This should lead to a reflex increase in cardiac performance via the baroreceptors in the great vessels, e.g., in the carotid sinus. Direct arterial blood pressure measurement failed to show such a central drop in blood pressure at the onset of exercise. Even the *Bainbridge reflex* was considered to be responsible for the increase in cardiac performance through a reflex response to increased atrial filling.

Adaptation of the circulation to conditions of load can be readily explained on the basis of a peripheral muscular regulatory mechanism. Alam and Smirk (1937) have shown that regulation of the blood pressure during exercise could be effected by peripheral muscles via the chemoreceptors and mechanoreceptors. Stegeman (1974) developed a theory that based the regulation of circulation primarily on muscular chemoreceptors. If the metabolic conditions in the muscle

**FIG 2–1.**

Circulatory responses under dynamic load. **a,** hemodynamic measurements obtained in five volunteers in the supine position with increasing loads using the dye-dilution technique. While cardiac output and pulse rate increase in a linear fashion with increasing loads, the stroke volume increases initially but then remains level in spite of increasing loads. Blood pressure was measured by the indirect method in this study. The increase in diastolic pressure found by direct arterial pressure measurements (see Fig 2–3,B), cannot be seen in this study. **b,** the findings in the area of energy production and circulatory adaptation are compared between trained and nontrained volunteers in a schematic fashion. The *solid line* represents the values obtained in the nontrained. The *broken line* represents the comparable values in the trained persons. With increasing loads, there is a linear increase in energy production over oxygen consumption *(upper left)*. The trained athlete consumes the same amount of oxygen for the same amount of load, although he can reach load levels that are beyond the capacity of the nontrained person. The second important factor in energy utilization is the production of lactic acid. Initially no lactic acid is produced, but there is a sharp increase in lactic acid values at the two-third mark of maximal effort (aerobic-anaerobic threshold). This curve is shifted to the right in the trained athlete *(upper center)*. The rise in oxygen consumption is based on a corresponding increase in cardiac output *(lower center)*. Here again there is an identical cardiac output per load level for both the trained and nontrained person. The two components of cardiac output, i.e., the pulse rate and the stroke volume, are shown on the *upper right* and *lower left side*, respectively. Since the trained athlete has a larger stroke volume, he can get by with a lower pulse rate for an identical load intensity. The maximal pulse rate, which is about the same as in the nontrained person, is reached only at levels of load intensity that are beyond the capacity of the nontrained person. The blood pressure values are the same in the trained and nontrained at similar intensities of load.

deteriorate—the specific metabolic stimulus has not yet been identified—this results in an increased sympathetic drive of the heart and a corresponding increase in cardiocirculatory performance.

Even though the regulatory mechanism has not been fully elucidated, there is an adaptation of the heart to increased load that is based on an increased sympathetic tone, i.e., on an extracardiac mechanism.

The intracardiac reserves of the heart, the so-called *Starling mechanism*, are significant only in adaptations to momentary swings in demand. The fact that the Starling mechanism does not play a significant role in the adaptation of the heart to performance requirements can be seen from the observation that during exercise the end-diastolic ventricular filling changes just as little as the filling pressure.

The significance of the Starling mechanism on the athlete's heart will be discussed further in the section on "Function of the Athlete's Heart."

Of the two components of the cardiac output, heart rate and stroke volume, heart rate is the significant factor in the adaptation to performance. The anatomic configuration of the ventricle is the limiting factor in the ability to increase stroke volume. When the muscular pumping action begins to increase, there will be an increase in venous return and some increase in stroke volume. Further increase in activity, however, does not lead to further increase in stroke volume. An increase in inotropy under sympathetic stimulation leads to an increase in stroke volume. It can be shown by echocardiography that identical diastolic filling still allows an increase in systolic output (see Fig 3–15). While pulse rate, output, and oxygen consumption increase linearly with the intensity of load, the *stroke volume* behaves fundamentally differently, as shown in Figure 2–1.

The degree of increase in stroke volume depends on body position. Most of the existing studies were performed with the subject in the supine position. Under these circumstances the dye-dilution technique shows a 25% increase in stroke volume, while studies using the Fick principle show an increase of only 10%. Studies with the subject in the sitting position show an increase in stroke volume of 30% to 50%. In studies performed on a treadmill, i.e., with the subject in the standing position, Hanson (1965) and Wang (1960) have found an increase in the stroke volume of 100%. These differences can be attributed to the decreased resting stroke volume normally found in the standing position. This orthostatic effect is compensated for by the muscle pump.

According to some data in the literature, particularly the findings of Bevegard (1963), the muscle pump is insufficient to fully compensate for the *orthostatic effect*. According to these findings, the cardiac output in the sitting position is 1 to 2 L less than in the supine position, given the same load. Our own comparative studies could not reproduce these findings. In our studies, cardiac output was identical in the sitting and supine position during bicycle ergometry.

Interestingly, the highest stroke volume is attained not during the exercise but immediately afterwards. This is due to the fact that immediately following the cessation of exercise, the pulse rate drops very rapidly, while the venous return remains very high. In this situation, the already mentioned *Starling mechanism* plays an important stop-gap role. The increase in stroke volume can last for about 3 minutes beyond the maximal stress, and is dependent upon the intensity of the load.

These relationships are primarily of historic interest, since in the 1950s they served to establish the *short-term-interval-training* even for endurance athletes. The assumption was that the increase in

stroke volume was the determinant stimulus for the enlargement of the heart, and therefore, the shortest possible load-times and correspondingly frequent intervals during the same training period, allowed a large number of the so-called "reward pauses." This led to the practice that even long distance runners had to undergo short distance training and were running 20 consecutive 200 meter dashes. The fact that this assumption was ultimately found to be wrong is based on the finding that endurance training cannot be viewed from a hemodynamic perspective. It also ignored the effects of training on the metabolic mechanisms that have such decisive importance in the athlete conditioned for sustained effort.

In contrast to stroke volume, the *heart rate* has a decisive effect on the fine adaptation of cardiac performance to stress. While the stroke volume in the untrained person can rise from 80 ml in supine position and 60 ml in the standing position to only 100–120 ml under maximal exercise, the heart can triple its rate, from 70 beats per minute to 200 beats per minute under similar conditions. Thus, the *maximal cardiac output* can be raised from 20 L to 25 L in the untrained person. The *maximal heart rate* is determined biologically and is age dependent, but not sex or training dependent. As rule of thumb, the average value for maximal heart rate can be taken as 220/minute minus age. Thus in a 10-year-old child it will be 210 beats per minute and in a 40-year-old person, 180 beats per minute. The two standard deviations of this value is ± 20. In youngsters, therefore, a maximal value of 230 can be reached. Higher maximal rates, e.g., 250 and even 300, which are occasionally reported by athletes, are usually due to counting errors. We were never able to demonstrate such values by Holter monitoring. A threefold increase in pulse rate is necessarily conditional on a shortening of the contractile process. This is evident from an *increase in contractility*. Studies by Roskamm (1972) (see Fig 3–16) have shown that in the untrained person the maximal rate of pressure increase can be quintupled as a parameter of contractility.

In setting the cardiac output volume for a given exercise, the available circulating *blood volume* must increase its rate of flow. The blood volume reserves that, according to Wollheim (1931), can be mobilized in response to stress in animals from the splenic and hepatic reservoir, do not exist in man. In fact, the available blood volume decreases by about 5% under load conditions. This is the result of the appearance of hemoconcentration. *Hemoglobin values* can increase 1–2 gm% under stress, as consequence of the decrease in plasma volume. Several mechanisms participate in this occurrence, e.g., an increase in filtration pressure, as a consequence of increased blood pressure, and an increase in capillary permeability (see Ekelund, 1967; Kirsch, 1968).

In order to have a clearer understanding of the time factor in this acceleration of the blood volume, reference must be made to the *steady state* concept. In every discussion concerning the achievement of an optimal exercise in the context of ergometry, it is emphasized that the duration of the exercise must be at least 6 minutes per watt step increase. Only such a time span can reasonably assure the establishment of an appropriate equilibrium. It is in fact mandatory that such a time sequence be maintained in order to accelerate the sluggish blood mass from a circulating volume of 5 to 6 L/minute to the maximal or submaximal cardiac output. Our own studies have shown that the time-volume rate required for a 200-watt load could not be achieved in 3 minutes but could be achieved in 6 minutes. It remained constant thereafter, in spite of increased load-time. This time span of 5 to 6 minutes to establish the

steady state cannot be extrapolated to minor increases in load. If a performance of 25 watt is required, this means an increase in cardiac output from a 6 L/minute to 8 L/minute. This increase can be achieved in 1 to 2 minutes so that after this time span the required cardiac output is obtained.

If the time of exercise is extended once the steady state value has been achieved, the cardiac output remains generally constant. In hemodynamic studies, Ekelund (1967) found that with exercise times of up to 30 minutes there was a continuous increase in heart rate, but that the cardiac output remained stable once the steady state had been reached. This means that increasing exercise times are accompanied by a decrease in stroke volume. This relationship can be explained well by the regulatory mechanism postulated by Stegemann (1974). Increasing exercise time leads to a deterioration of the metabolic status in the muscle, which leads to an increased sympathetic drive, and thus to an increase in heart rate. Since the venous return does not increase appreciably, the stroke volume must decrease.

Finally it must be mentioned that the established cardiac output does depend on *age* and *sex*. With the exception of the known facts relating to the elderly, the data in the literature are poor. Astrand (1964) claimed that in the *female* the cardiac output was higher for a given load. He assumes that this is due to lower hemoglobin concentration. This hypothesis is not supported by other data, and Musshoff (1959) found no difference in exercise cardiac output between males and females. A lower hemoglobin value means a decrease in the maximal *arteriovenous oxygen gradient*. It does not, however, necessarily mean a decreased oxygen utilization at submaximal effort. Naturally females must produce a higher heart rate than males, given the same level of exercise, because of their generally smaller heart size and smaller stroke volume.

As far as the effect of age on exercise cardiac output is concerned, there are for obvious reasons, very few data in *children* based on invasive measurement techniques. According to the findings of Erikson (1971) the cardiac output in children is less than in adults for comparable loads. This finding needs to be substantiated by further studies. A decrease in cardiac output in the *elderly*, however, has been clearly demonstrated in conditions of equal loads. In this respect, the findings of Granath (1964) are particularly pertinent. The cardiac output and oxygen uptake is about 1 to 2 L/min less for similar loads than in the younger person. The peripheral oxygen utilization, however, is correspondingly increased. Since in the elderly, sclerotic changes lead to an increase in mean arterial pressure under similar loads, there is a shift in cardiac work during exercise from volume-work to pressure-work, even though the total cardiac work remains approximately the same. According to the studies of Gollwitzer-Meier (1937) this is an undesirable development since pressure work is much more detrimental for myocardial oxygen requirements than volume work.

### *Arterial Pressure*

The final comments of the previous section introduce one of the significant components of cardiac work under stress, namely arterial pressure, that will be discussed in detail below. The behavior of blood pressure is quite variable and can undergo rapid changes, as compared to the much more sluggish changes in cardiac output. These conditions could be elucidated only when direct arterial pressure measurements became available and when telemetry became a practical tool. In this relationship, the studies of Bachmann (1969, 1970) are particularly pertinent.

The comments concerning the usefulness of direct arterial pressure measurements, lead naturally into some remarks concerning the *indirect blood pressure measurement, or Riva-Rocci, method.* This latter method allows only a very limited understanding of the behavior of pressure under stress. On one hand the method is very slow, and stretches over a number of heart beats, so that rapid changes cannot be appreciated. On the other hand, this method is very unreliable in determining the diastolic pressure and thus makes the determination of mean pressure equally unreliable. Ergometric studies have shown that when diastolic pressure was measured by the indirect method, it frequently showed a decrease under increasing stress. In extreme cases, it is sometimes possible to observe the so-called *null phenomenon* when, usually after stress, the Korotkoff sounds were audible even though the cuff pressure had fallen to zero. Comparative, direct, and indirect measurements do not support such observations (Fig 2–2). According to direct measurements on the bicycle ergometer, increasing stress results in increasing diastolic pressure, although to a lesser degree than the systolic pressure.

The decrease in diastolic pressure, found frequently with indirect pressure measurements during bicycle ergometry, does not correspond to the true conditions existing in the vascular tree. The basis for the original observation was a problem of technique. To date, there is no generally accepted theory concerning the origin of the Korotkoff sounds or why these sounds disappear in the neighborhood of the diastolic pressure, or indeed what the precise criteria are for the measurement of diastolic pressure. The recommendations whether to use the decline in the sounds (phase IV) or the disappearance of the sounds (phase V) vary from expert to expert. Nevertheless, it can be stated that the origin of the Korotkoff sounds is related to intravascular flow and to the pressure of the cuff. Under stress, the intravascular flow is in-

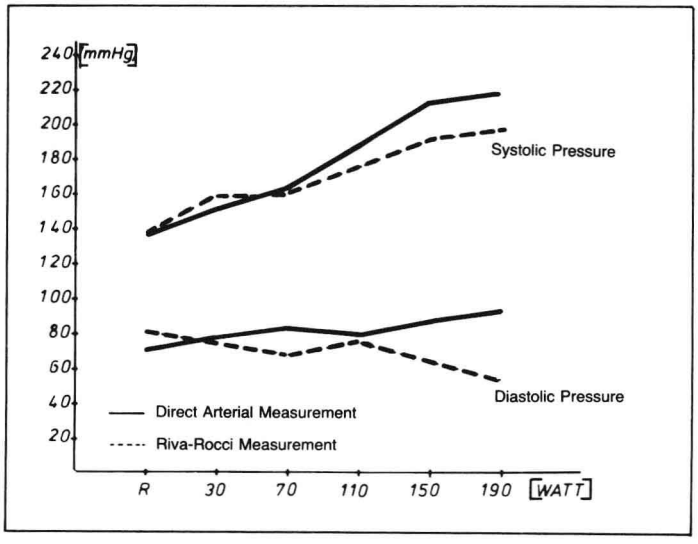
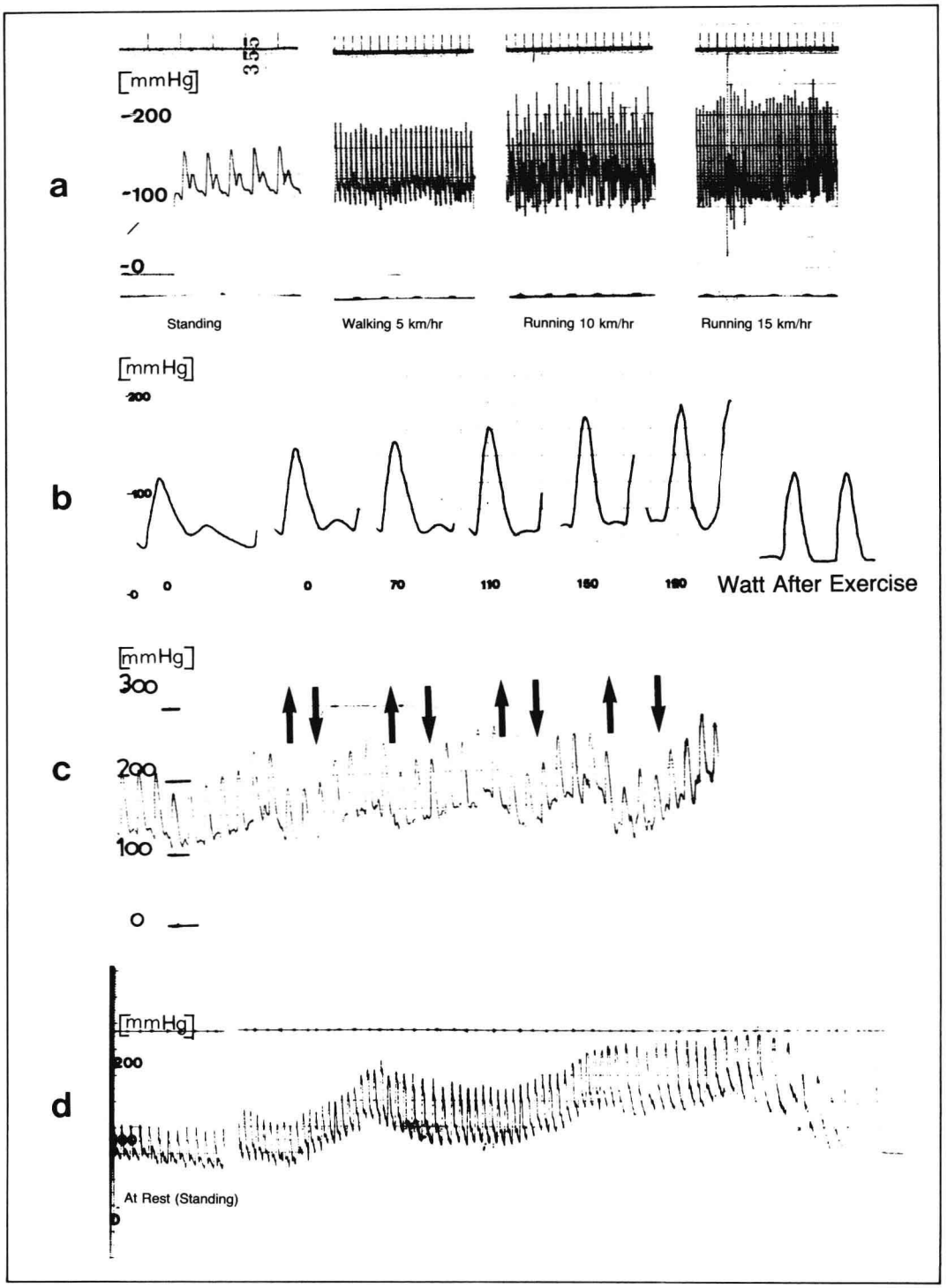



**FIG 2–2.**
Comparison of arterial pressure obtained by direct and indirect measurement during ergometry. The systolic pressure is very similar by both techniques. On the other hand, the diastolic pressure shows an apparent decline with increasing loads when measured by the Riva-Rocci method. This finding cannot be substantiated by direct arterial measurement.


a
[mmHg]
-200
-100
-0
Standing
Walking 5 km/hr
Running 10 km/hr
Running 15 km/hr
b
[mmHg]
0
0
70
110
150
190
Watt After Exercise
c
[mmHg]
300
200
100
0
d
[mmHg]
200
At Rest (Standing)

creased to the point where the pressure of the stethoscope suffices to produce sounds, even though the cuff pressure has fallen to zero.

Because of the lack of reliability of the indirect method, *direct pressure measurements under stress* have revealed new and surprising results. Since an increase in pressure under physical exercise was first described by Zadek (1881), it has been considered self-evident that physical activity results in an increase in blood pressure. This does not seem to be obvious when looked at teleologically. If the goal of the circulation during exercise is to increase the cardiac output, the flow, and the amount of oxygen carried, this could be accomplished, according to Ohm's law, without increase in pressure and solely through a corresponding decrease in peripheral resistance. It does not seem apparent why the heart should have to increase its pressure-work substantially just to increase the amount of blood pumped.

From the point of view of circulatory economy, the most sensible solution would be if an increase in cardiac output were compensated for by a corresponding dilatation of the blood vessels. It will come as a surprise to those who are not particularly knowledgeable in circulatory dynamics during exercise that if the load is primarily due to isotonic muscle contractions, the above-described circulatory model is closely approximated. Thus, in running, the mean arterial pressure does not rise significantly, as proved by arterial pressure curves. It is shown in Figure 2–3 that during *running* with increasing speed, the diastolic pressure remains unchanged, while the systolic pressure rises. Figure 2–3 also shows that there is a simultaneous distortion of the pressure wave form. The dicrotic wave keeps sliding lower and lower. Since during running the shaking of the indwelling catheter makes it almost impossible to get artifact-free tracings, the changes in the pressure wave form have been substantiated during pedaling exercises in the supine position. Figure 2–3 shows in tracings obtained by this method that the dicrotic wave practically disappears with increasing stress and the arterial pressure curve becomes steeper.

Holmgren (1956) was the first one to point out this relationship. The change in the shape of the arterial curve is due to the decrease in peripheral resistance under stress and to the altered reflective responses in the periphery, which are responsible for the dicrotic wave. The *dicrotic phenomenon* is due to a superimposition of a "standing wave" on the pressure curve, which changes its maxi-

**FIG 2–3.**

Arterial pressure tracings under different types of dynamic exercises. **a,** arterial pressure while standing, walking, and running at two different speeds. The time markers show that the paper speed was reduced during running in order to capture more pressure waves. There is a speed-dependent increase in systolic pressure while the diastolic pressure remains unchanged. The dicrotic notches appear as a dark heavy line. This represents an increasing flattening of the dicrotic wave, seen better in **b. b,** bicycle ergometric exercises in the supine position. To illustrate the increasing steepness of the pressure wave better, a single wave is shown, obtained at a fast paper speed. A distinct rise in systolic pressure and a moderate rise in diastolic pressure can be seen. The dicrotic pressure wave gradually flattens and is totally flat immediately after the end of the exercise. **c,** arterial pressures during push-ups. At the beginning of the push *(arrow pointing down),* there is a definite increase in pressure. When the arms are extended *(arrow pointing up),* the muscles relax. The decreased effort leads to an immediate drop in pressure. **d,** arterial pressure during diving. The measurements were made in a volunteer at a depth of 1 m. In order to avoid any Valsalva effect, the volunteer was equipped with a respirator. There is an increase in arterial pressure that corresponds to the external hydrostatic pressure (about 75 mm Hg). There is a simultaneous decrease in heart rate that corresponds to the increase in pulse pressure.