High-Intensity Intermittent Exercise: Methodological and Physiological Aspects

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High-intensity intermittent exercise (HIIE) has been applied in competitive sports for more than 100 years. In the last decades, interval studies revealed a multitude of beneficial effects in various subjects despite a large variety of exercise prescriptions. Therefore, one could assume that an accurate prescription of HIIE is not relevant. However, the manipulation of HIIE variables (peak workload and peak-workload duration, mean workload, intensity and duration of recovery, number of intervals) directly affects the acute physiological responses during exercise leading to specific medium- and long-term training adaptations. The diversity of intermittent-exercise regimens applied in different studies may suggest that the acute physiological mechanisms during HIIE forced by particular exercise prescriptions are not clear in detail or not taken into consideration. A standardized and consistent approach to the prescription and classification of HIIE is still missing. An optimal and individual setting of the HIIE variables requires the consideration of the physiological responses elicited by the HIIE regimen. In this regard, particularly the intensities and durations of the peak-workload phases are highly relevant since these variables are primarily responsible for the metabolic processes during HIIE in the working muscle (e.g., lactate metabolism). In addition, the way of prescribing exercise intensity also markedly influences acute metabolic and cardiorespiratory responses. Turn-point or threshold models are suggested to be more appropriate and accurate to prescribe HIIE intensity than using percentages of maximal heart rate or maximal oxygen uptake.

Keywords: interval exercise, HIIE prescription, acute responses, lactate steady state, turn-point concept

Interval exercise is a discontinuous mode of endurance exercise that is characterized by relatively short bouts of high-intensity workloads interspersed by periods of rest or low-intensity activity. The rationale behind interval-training programs is that the total accumulated time of vigorous exercise is higher than could be achieved during a single bout of continuous exercise at the same intensity until exhaustion. Therefore, high-intensity interval training is supposed to yield greater training effects on endurance performance, particularly in highly trained athletes, but nowadays it is also successfully applied in the rehabilitation of different kinds of chronic diseases. A multitude of investigations have revealed a wide range of beneficial adaptations after high-intensity intermittent exercise (HIIE) in various study populations. However, the interpretation of these training effects is difficult if the acute physiological responses during exercise are unknown or not indicated as is the case in many published HIIE studies. Achieved training adaptations are the consequence of a disturbance of homeostasis during exercise (acute responses). The extent of these acute physiological responses is strongly influenced by the specific prescription of the single exercise components such as intensity and duration of peak workload and recovery phases. To date there is still a lack of knowledge regarding the detailed acute physiological responses induced by various interval-exercise regimens, and a consistent systematic approach to the prescription of intermittent exercise is still missing. It appears to be highly important to understand, clarify, and standardize the methodology of interval exercise to optimize training effects and concurrently minimize health risks, particularly in patients suffering from different kinds of diseases. Therefore, this review focuses on the methodological aspects of interval training and on the acute physiological mechanisms caused by specific intermittent-exercise prescriptions.

Historical Review

As reviewed by Billat, interval training has been applied in competitive sports since the beginning of the 20th century. Middle- and long-distance runners such as the Finnish athletes Hannes Kolehmainen (3-fold Olympic champion in Stockholm 1912) and Pavoo Nurmi (9-fold Olympic champion between 1920 and 1928) used interval training to train at velocities close to their specific competition velocity. In addition, cross-country running and skiing at different intensities (uphill, downhill, and in the flat), called “fartlek,” was included in the training process of various endurance sports. The most famous athlete to popularize interval training was Emil Zatopek, the Czechoslovakian runner and triple Olympic.
gold medalist in 1952, who used short interval training including up to 100 × 400-m runs interspersed by 200-m recovery runs per day.6 At that time, the trainers used specific competition velocities from 800 to 5000 m for the prescription of interval intensity, not taking into account any physiological parameters.6

The first description of interval training in a scientific journal was made by Reindell and Roskamm in 1959.7 In 1960, the Swedish research group of Irma and Per Olaf Astrand8 and Erik Hohwü Christensen9 published studies on interval training, focusing on the acute responses of lactate (La), oxygen uptake (VO2), and heart rate (HR) during interval exercise of different work durations and during continuous exercise. In their review in 1976, Saltin et al10 pointed out the low blood La levels and the small oscillations of VO2 between peak-workload and recovery phases during short intervals (intervals with short work duration), whereas long intervals (intervals with long work durations) yielded high La levels and marked differences between the VO2 values of work and recovery phases.

The aforementioned studies on interval training focused on exercise performance improvements in endurance athletes and used peak intensities during the velocity at maximal La steady state (max LaSS) and the minimal velocity eliciting maximal VO2max (vVO2max) or slightly above. However, during the last 20 years, interval training has increasingly been applied also in recreationally active individuals,3,11 as well as in team- and racket-sport players.12–14 As a consequence, higher peak workloads up to maximal efforts were used in HIIE training and testing. The so-called sprint interval training or repeated-sprint training including short bouts of maximal intensity came into the focus of exercise physiology research, and it was shown that repeated brief maximal sprints induced similar effects on the aerobic system in recreationally active individuals as traditional endurance training, but in a very time-efficient way (low-volume high-intensity interval training).3,15

In the last 10 years, interval exercise attained additional importance with respect to endurance training since aerobic high-intensity interval training was applied not only in healthy people but also in the rehabilitation of different kinds of chronic diseases.16,17

Components and Prescriptions of HIIE

In contrast to continuous exercise that only comprises the workload intensity and the total duration, intermittent exercise consists of 5 main components: peak workload intensity (Ppeak), peak workload duration (tpeak), recovery load (Prec), recovery duration (trec), and the mean load (Pmean), of which the result of the latter 4 can be calculated accordingly or set as a separate determinant.10 The number of intervals which means the total exercise duration is a further variable of HIIE prescription. In their recent review, Buchheit and Laursen14 also mentioned the number of series, the duration and intensities in recovery phases between the series and the exercise modality (eg, running vs cycling) as further determinants of interval exercise. However, they did not consider Pmean as a relevant variable.

Ppeak is usually set between the power output at the anaerobic threshold2 and “all-out” exercise, and tpeak ranges from a few seconds up to several minutes. The Ppeak phases are separated by periods of low- (or moderate-) intensity exercise or passive recovery with a trec that can be shorter than, equal to, or longer than tpeak. Notably, very sparse information is available concerning settings of the Pmean. Given the different potential combinations of Ppeak, tpeak, Prec, and trec, it is not surprising that there is a plurality of diverse prescriptions for intermittent exercise used in scientific studies and exercise training. For example, Trapp et al18 applied sprint exercise for 8 seconds during work phases and slow pedaling for 12 seconds during recovery phases, whereas Helgerud et al16 used (among others) 4-minute work phases at 90% to 95% HRmax and 3-minute recovery phases at 70% HRmax.

Despite this diversity of applied HIIE regimens, beneficial effects could be achieved in different HIIE studies and in various subjects (highly trained, as well as healthy sedentary and diseased, individuals). Therefore, one could assume that an accurate prescription of HIIE is not relevant. However, the isolated manipulation of each single variable presumably has a direct impact on acute metabolic and cardiopulmonary (and neuromuscular) responses during exercise.4 If 2 or more components are manipulated simultaneously, the impacts on the physiological responses are more complex and also more difficult to predict.14 These acute reactions lead to specific medium- and long-term training adaptations on the one hand, and on the other hand they might represent certain health risks, particularly in diseased persons. Therefore, the understanding of the acute physiological mechanisms provoked by the manipulation of (interval) exercise settings is of high relevance in exercise physiology research.

The variety of intermittent exercise regimens applied in different studies may suggest that the acute physiological responses during HIIE forced by particular exercise prescriptions are not clear in detail or not taken into consideration; a standardized and consistent approach to the prescription of HIIE is still missing.

Shortcomings in Denominations and Classifications of Intermittent Exercise and in the Prescription of Exercise Intensities

Denominations and Classifications

Besides the enormous variety in interval-exercise prescriptions, there is, not less important, also an uncontrolled growth of types and denominations of intermittent exercise in the literature: intermittent exercise, interval-type exercise, interval training, high-intensity interval training, aerobic high-intensity interval training, repeated-sprint exercise, sprint intervals, and low-volume high-intensity
interval training. A further problem is that the same terms are diversely used by different authors: Burgomaster et al.\textsuperscript{15} used the term low-volume high-intensity interval training for sprint (all-out) intervals of 30 seconds, whereas Currie et al.\textsuperscript{19} used the same term for intermittent exercise at 89% peak power output for 1 minute.

More important, the actual acute physiological responses yielded by different interval regimens are not considered for the denomination and classification of intermittent exercise, except for the term aerobic HIIE. However, there is only rare information about how aerobic is defined. Billat\textsuperscript{6} defined aerobic interval training as training that elicits aerobic metabolism at a higher ratio than anaerobic metabolism. With respect to the La shuttle theory,\textsuperscript{20} we suggest to define the term aerobic as a balanced condition between La production in the working muscle and La elimination in the working muscle itself and in the system (through other organs such as brain, heart, liver, and resting skeletal muscles) resulting in a blood La steady state.

In German textbooks of training science,\textsuperscript{21} intermittent exercise is classified into 2 methods. One model is characterized by rest periods that are terminated prematurely, permitting just partial but not full recovery; this model is usually applied in HIIE studies (in German called \textit{Intervallmethode}). The other model includes considerably long rest periods allowing full recovery between the high-intensity exercise bouts (\textit{Wiederholungsmethode}). Both methods are divided into the intensity categories “extensive” and “intensive,” and these intensity categories are divided again into subcategories indicating the duration of peak workloads. Finally, a certain range of recovery durations is assigned to each subcategory.

However, physiological markers are also not sufficiently taken into consideration for this model of categorization of intermittent exercise. In addition, the allocation of the actual acute physiological responses to particular subcategories is partly incorrect due to the relatively wide ranges of both the intensity categories and the workload-duration categories.

Therefore, we recommend the application of the model shown in Figure 1 for the classification of intermittent exercise.

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**Figure 1** — Suggested model for the classification of intermittent exercise. Abbreviations: $P_{\text{LTP2}}$, second lactate turn point determined in an incremental exercise test; $P_{\text{peak}}$, peak workload during intermittent exercise; LaSS, blood lactate steady state.
Prescription of Exercise Intensity

The intensity of intermittent exercise referred to in German textbooks or applied in the first half of the 20th century is prescribed by means of percentages of race velocity. This mode of exercise-intensity setting is primarily suitable for endurance athletes. However, team- and racket-sport players, recreational athletes, and patients need different approaches to exercise-intensity settings.

In a multitude of methodological investigations and training-intervention studies, intermittent-exercise intensity is set by means of percentages of maximum HR (HR_{max}), HR reserve (HRR), maximum VO\(_2\) (VO\(_2\)_{max}), and VO\(_2\) reserve. However, prescribing exercise intensity via %HR\(_{max}\) (or HRR) and power outputs at fixed %HR\(_{max}\) (or HRR) results in considerable interindividual differences in relative P\(_{mean}\), P\(_{peak}\), and P\(_{rec}\). As a consequence, the cardiopulmonary and metabolic strain, as well as exercise stimuli, is substantially different across subjects, as well. This diversity is caused by different types of HR-performance curve. This fact was recently pointed out by Hofmann and Tschakert\(^{22}\) and previously shown by Hofmann et al\(^{23}\) and Wonisch et al\(^{24}\).

Also, the setting of intermittent-exercise intensity by means of %VO\(_2\)_{max} (or VO\(_2\) reserve) leads to substantial differences in physiological demands across subjects.\(^{25}\) Particularly in intervals with long peak-load durations, prescriptions of exercise intensities via %HR\(_{max}\) or %VO\(_2\)_{max} are not appropriate since the heterogeneity of acute physiological responses increases with the duration of peak-workload phases.

In addition, if exercise intensity is prescribed via %HR\(_{max}\), it is unclear how long it takes until each participant reaches his or her individual target HR, and no information is available about the power output and the metabolic situation until target HR is reached. Therefore, an accurate prescription of exercise intensity by means of %HR\(_{max}\)—also for intermittent exercise with short peak-load durations up to 30 seconds—is not possible.

Another shortcoming of actual intermittent-exercise prescriptions is that P\(_{mean}\) is only rarely considered despite the fact that it controls the acute cardiorespiratory responses. However, already Astrand et al\(^{8}\) mentioned the relevance of P\(_{mean}\) as they indicated that HR and VO\(_2\) values oscillate around the average power output.

Suggestions

Since the aforementioned classifications of intermittent exercise do not consider the physiological reactions elicited by the particular interval regimen, we want to emphasize that exercise itself should be described in terms of the metabolic response it elicits.\(^{26}\)

It has been proven reasonable to prescribe exercise intensities for continuous exercise with respect to the first and second turn points for lactate (LTP\(_1\), LTP\(_2\)) or ventilation (VT\(_1\), VT\(_2\)) determined in a preliminary incremental exercise test (Figure 2).\(^{22,27}\) Therefore, we suggest that also for the prescription of intermittent exercise it is favorable to use submaximal thresholds or

![Figure 2 — Three-phase model of lactate (La) metabolism during an incremental exercise test.\(^{22}\) Abbreviations: LTP\(_1\), first lactate turn point determined in an incremental exercise test (IET); LTP\(_2\), second lactate turn point determined in IET; P\(_{max}\), maximal power output from IET; M, working muscle; S, system; P, lactate production; E, lactate elimination.](image-url)
Acute Physiological Responses to Manipulations of HIIE Variables

Creatine Phosphate, Oxymyoglobin

One of the most interesting questions in the context of intermittent exercise is the energy yield for ATP resynthesis obtained by anaerobic and/or aerobic metabolism. Important and fundamental findings concerning the acute metabolic (and cardiopulmonary) effects of intermittent exercise were already revealed more than 50 years ago by Irma and Per-Olaf Astrand, Bengt Saltin, Erik H. Christensen, and colleagues. They focused particularly on the physiological impact of the combination of $t_{\text{peak}}$ and $P_{\text{peak}}$.

In the initial phase of work and, therefore, also during the bulk of peak-workload phases of short intervals, respiration and oxygen transport by the blood to the working muscles does not reach the values that correspond to the actual oxygen demand due to delayed VO$_2$ kinetics. Therefore, the energy for ATP resynthesis must be obtained by means of intracellular stored oxygen and/or anaerobically. In this regard, the importance of oxymyoglobin as an intracellular oxygen store and of creatine phosphate (CrP), an energy-rich phosphagen, was emphasized by several authors since even high energy demands are met by these pathways in the first few seconds of work.$^{1,8,10,14}$ The stores of oxymyoglobin and CrP can be rapidly and fully restored during the recovery periods, unless $P_{\text{peak}}$ is too high or the recovery phases are too short or too intense.$^{14}$

Lactate Metabolism

The fundamental studies of Astrand et al.$^8$ revealed that long intervals of 2 or 3 minutes at intensities near the turn points,$^{14}$ as well as the maximal power output ($P_{\text{max}}$) from an incremental exercise test representing the individual aerobic-exercise performance. The 2 thresholds and 3 phases of metabolism, respectively, can also be detected during a series of continuous (Figure 3) and intermittent exercise.

Between rest and LTP$_1$ (phase 1), all La produced in the working muscles is aerobically eliminated in the working muscles; therefore, there is a balance of La production and elimination (La steady state) in the working muscle; blood La values remain at resting level. Between LTP$_1$ and LTP$_2$ (phase 2) La cannot be oxidized in the skeletal muscle entirely and has to be partly shifted into the blood, but it can be metabolized by other organs. Therefore, in this phase there are metabolically balanced (steady-state) conditions again, but now on a systemic level. Above LTP$_2$ (phase 3), more La is produced in the working muscles than can be muscually and systemically eliminated, resulting in a blood La accumulation (metabolically unbalanced conditions, no La steady state).$^{22}$

Concerning the mode of exercise-intensity prescription, we suggest using $\%P_{\text{LTP2}}$ as $P_{\text{mean}}$, $\%P_{\text{max}}$ as $P_{\text{peak}}$, and $\%P_{\text{LTP1}}$ as $P_{\text{rec}}$ and applying the equation $P_{\text{mean}} = (P_{\text{peak}} \times t_{\text{peak}} + P_{\text{rec}} \times t_{\text{rec}})/(t_{\text{peak}} + t_{\text{rec}})$ for intermittent exercise. In addition, we emphasize that $P_{\text{mean}}$ should not just be calculated as the result of $P_{\text{peak}}$, $t_{\text{peak}}$, $P_{\text{rec}}$, and $t_{\text{rec}}$ but consciously set to consider cardiorespiratory strain during intermittent exercise.

This new methodological approach enables the correlation of physiological responses and adaptations induced by interval-type exercise training to exercise intensities related to the 3 individual phases of energy supply. It is based on the turn-point concept,$^{22}$ which is strongly supported by the La shuttle theory.$^{20}$

![Figure 3](image_url) — Blood lactate (La) during continuous exercise at intensities corresponding to phase I, phase II, and phase III. Phase I, range from rest to the first lactate turn point (LTP$_1$)—blood La values show a steady state at resting level due to a metabolic balance in the working muscle; phase II, range from LTP$_1$ to second lactate turn point (LTP$_2$)—blood La values are elevated but show a steady state on a systemic level; phase III, range from LTP$_2$ to maximal power output from incremental exercise test—no La steady state is reached.
power output at VO\textsubscript{2max} were extremely demanding, yielding high blood La levels of 16.6 mmol/L. In contrast, short intervals of 30 seconds or 1 minute at the same peak and mean workload induced low blood La levels of 2 mmol/L and were well tolerated for 1 hour. These data were surprising given the high peak workloads, but the results were supported by subsequent studies of Christensen et al.\textsuperscript{30} and Carlson and Pernow.\textsuperscript{28}

It should be kept in mind that the La concentration in the working muscle and in the blood reflects the concentration of hydrogen (H\textsuperscript{+}) ions at the particular site. The H\textsuperscript{+} concentration is expressed by means of the pH value and strongly influences the acid-base status. La and H\textsuperscript{+} ions are the result of the dissociation of lactic acid, which is produced through anaerobic glycolysis.\textsuperscript{29}

With respect to anaerobic metabolism during intermittent exercise, it is assumed that metabolic acidosis mainly takes place during the peak-workload phases.\textsuperscript{10} The rate of La production depends on P\textsubscript{peak}, the amount of La production primarily depends on the product of P\textsubscript{peak} × t\textsubscript{peak}, and the blood La concentration results from the balance between La production and elimination.\textsuperscript{20} Severe blood La accumulations can be evoked by exercise durations of several minutes at workloads between the power output at max LaSS (P\textsubscript{max,LaSS}) and P\textsubscript{max} from an incremental exercise test,\textsuperscript{8} but also by shorter exercise phases of 30 seconds or more at all-out intensity (eg, Wingate test).\textsuperscript{3} If such peak-workload periods are applied in intermittent exercise, it is supposed that LaSS cannot be reached. In contrast, intervals with short peak-load durations of about 20 or 30 seconds (or less) have been shown to be performed in LaSS conditions even if P\textsubscript{peak} is markedly above the power output at max LaSS (but below all-out exercise), although t\textsubscript{rec} is not longer than t\textsubscript{peak}.\textsuperscript{8–10} In this case, La is produced in such small amounts that the short recovery durations are sufficient to reach a balance between La production and elimination.\textsuperscript{10} This means that the setting of the peak-workload phases is a much more critical factor, whether blood La is accumulating or not, than the duration of recovery phases. This finding was already emphasized by Saltin et al.\textsuperscript{10} A multitude of combinations of t\textsubscript{peak} and P\textsubscript{peak} may lead to LaSS, but one should be aware that La and, of course, H\textsuperscript{+} concentrations (in muscle and blood) increase with t\textsubscript{peak} if P\textsubscript{peak} is set above the max LaSS.\textsuperscript{27,30,31} The higher P\textsubscript{peak} is, the shorter t\textsubscript{peak} has to be!

The advantage of a LaSS exercise is that it can be sustained for a longer total time, resulting in a longer accumulated training duration at high intensities than with non-steady-state exercise.

In addition, however, HIIE that evokes high levels of lactic acid production in the working muscle failing to reach LaSS during total exercise duration hormonally triggers specific responses\textsuperscript{32} and may elicit certain benefits, particularly for athletes, such as the improvement of La tolerance. In this regard, high intracellular lactic acid levels and high blood La levels are thought to lead to a restriction of glycolysis during subsequent work bouts due to a limitation of the enzyme phosphofructokinase,\textsuperscript{33,34} forcing the organism to a greater contribution of aerobic metabolism to ATP resynthesis despite workloads above max LaSS. This mechanism may be a reason for considerable improvements in VO\textsubscript{2max} after HIIE training with long peak-load durations, such as the frequently applied 4 × 4-minute HIIE protocol.\textsuperscript{16,17}

However, given the marked acute physiological responses during long intervals, the question arises as to whether this interval regimen is suitable for diseased individuals, as the health risks were shown to be higher.\textsuperscript{5}

Our working group compared long and short intervals matched by mean load and total exercise duration in healthy individuals and subjects undergoing cardiac rehabilitation. In both investigations, data showed significantly higher La values during long HIIE than with short HIIE. During long HIIE, the increase of net blood La, as well as the respiratory-exchange ratio, became smaller with each interval, suggesting a considerable lactic acid production during the first peak-workload phase that led to an inhibition of glycolysis in the following intervals (unpublished results). These data support the results of Von Duvillard et al.\textsuperscript{35} obtained in elite alpine skiers.

VO\textsubscript{2}

VO\textsubscript{2} is a metabolic parameter but is limited by the cardiopulmonary system that transports O\textsubscript{2} to the place of oxygen consumption. Since the HIIE-induced responses of cardiorespiratory parameters (eg, HR) are adapted to P\textsubscript{mean}, acute VO\textsubscript{2} during intermittent exercise is also based on P\textsubscript{mean} in this context, Astrand et al.\textsuperscript{30} indicated that the shorter the peak-workload duration is, the smaller is the oscillation of acute VO\textsubscript{2} values around the demand for P\textsubscript{mean}. These findings were supported by our own results (Figure 4[a]).\textsuperscript{36} It was shown that in short intervals of 30 seconds or 1 minute only 63% VO\textsubscript{2max} was reached.\textsuperscript{8} In contrast, Christensen et al.\textsuperscript{30} found out that during interval exercise with short high-intensity periods of 15 seconds at the minimum vVO\textsubscript{2max} and with passive recovery phases of 15 seconds, VO\textsubscript{2max} values were reached at the end of exercise despite low blood La levels. During HIIE with long peak-workload durations of 2 to 3 minutes at the same peak workload, the VO\textsubscript{2} oscillation increased, reaching maximal VO\textsubscript{2} values at the end of the peak-workload periods. These findings were supported by Seiler and Sjursen.\textsuperscript{37}

In addition to %VO\textsubscript{2max} values, the time spent near or at VO\textsubscript{2max} (t@VO\textsubscript{2max}) was reported to be a relevant criterion concerning peripheral muscular and cardiovascular effects of intermittent exercise.\textsuperscript{14} Therefore, the authors emphasized that an optimal HIIE stimulus is one where athletes maintain long periods of time above 90% of their VO\textsubscript{2max} and that long peak-load durations of 2 to 3 minutes enabled a longer accumulated t@VO\textsubscript{2max} than short HIIE. However, they primarily focused on the
training goals of endurance athletes and to a lesser extent on other athletes such as racket- and team-sport players or on patients. Already more than half a century ago, Astrand et al\(^8\) reported high training effects on circulation and respiration induced by long intervals, but they also pointed out the submaximal loading of the circulatory and respiratory organs during short intervals, which is of high practical and physiological interest in work physiology and rehabilitation. We additionally want to emphasize that intermittent exercise with a long \(t_{peak}\) may enable higher %\(\text{VO}_{2\text{max}}\) and \(t@\text{VO}_{2\text{max}}\) values, but at the same time long intervals may also lead to high \(L_a\) and low \(pH\) values.

Midgley and McNaughton\(^38\) suggested using short peak-load durations of 15 to 30 seconds and workload intensities between 90% and 105% \(v\text{VO}_{2\text{max}}\) to increase \(t@\text{VO}_{2\text{max}}\); however, they thereby suggest a \(P_{rec}\) between 50% \(v\text{VO}_{2\text{max}}\) and the \(L_a\)-threshold velocity and a preliminary 5- to 10-minute warm-up period performed 1 to 2 km/h below the \(L_a\)-threshold velocity to reach \(\text{VO}_{2\text{max}}\) values as quickly as possible during the subsequent work phases.

In the last decade, more investigations have revealed that even sprint-interval training with a short \(t_{peak}\) of 30 seconds\(^11,15,39\) or even less than 10 seconds\(^18\) yielded significant improvements in endurance performance and \(\text{VO}_{2\text{max}}\). Exercise intensity was reported to be the most important factor concerning training adaptations.\(^38\) Evidence suggests that improvements of peripheral muscular oxidative capacity and consequently of \(\text{VO}_{2\text{max}}\) and aerobic performance are related to exercise intensity.\(^40\) Bartlett et al\(^41\) and Gibala et al\(^3\) pointed out that exercise intensity is thought to be the key factor of activation of peroxisome proliferator-activated receptor \(\gamma\) coactivator-\(1\alpha\) (PGC-\(1\alpha\)), the master regulator of mitochondrial biogenesis in muscle, and of expression of PGC-\(1\alpha\) mRNA.\(^30\)

Therefore, the question arises as to whether either the high percentages of \(\text{VO}_{2}\) or the high peak power outputs cause the considerable aerobic effects after HIIE.

In this regard, the improvements in \(\text{VO}_{2\text{max}}\) revealed in sprint-interval studies were mainly due to adaptations of the muscular oxidative potential, such as increases in mitochondrial content and mitochondrial enzyme activity. Effects on cardiac function would need peak-load durations of at least 2 or 3 minutes.\(^14\) However, Helgerud et al\(^16\) revealed no significant differences in the increase of \(\text{VO}_{2\text{max}}\) and stroke volume (SV) between short HIIE with \(t_{peak}\) of 15 seconds and the \(4 \times 4\)-minute HIIE matched for total work.

The assumption that short intervals more likely can be sustained for a longer overall time than long HIIE is supported by respiratory-exchange-ratio values that were found to be higher during long HIIE than during short HIIE.\(^9\) This indicates a higher contribution of fat oxidation to energy yield during short HIIE than with long HIIE, but no explicit data could be found on this subject. However, Christmass et al\(^42\) compared the rates of substrate oxidation during 2 different HIIE regimens with a work-to-rest ratio of either 6:9 or 24:36 seconds (whereby even HIIE with a \(t_{peak}\) of 24 s is usually not characterized as a long interval). They showed a significantly higher fat and lower carbohydrate oxidation with concurrent lower \(L_a\) and pyruvate levels, as well as, surprisingly, higher \(\text{VO}_{2}\) values during the shorter intervals.

**Cardiac Output**

**HR.** Astrand et al\(^8\) emphasized that in HIIE with long \(t_{peak}\) of 3 minutes, the oscillations of HR values between the peak-workload phases (188 beats/min)
and recovery periods (118 beats/min) were shown to be great, reaching peak values. In contrast, mean- and peak-load-matched short intervals \( t_{\text{peak}} = 30 \) s led to much smaller HR oscillations (150–137 beats/min) and accordingly to HR values that were similar to mean-load-matched constant-load-exercise HR. This is remarkable given the fact that peak workload was twice as high in short intervals as in continuous exercise. However, the oscillation peak and minimum HR values around HR\(_{\text{mean}}\) were small during short HIIE because of the short \( t_{\text{peak}} \), and HR\(_{\text{mean}}\) (similarly to VO\(_2\)\(_{\text{mean}}\) as mentioned before) follows \( P_{\text{mean}} \). These results were supported by investigations of our own working group as shown in Figure 4(b).36

In this regard, it is important to point out that the acute HR during intermittent exercise does not necessarily give information concerning the peripheral-muscle metabolic strain. Relatively low HR values can be accompanied by extremely high lactate levels, nearly reaching maximal values. Therefore, lactate measures in interval exercise studies are highly relevant.

**SV.** Common opinion often prevails that during recovery phases of intermittent exercise, improvements in cardiac function are achieved through an increase of left-ventricular (LV) volume (a decreased peripheral resistance leads to an enhanced end-diastolic volume \([EDV]\) and \(SV\)).14,43 However, investigations of Pokan et al44 revealed that in sport students, the LVEDV decreased \([EDV]\) and SV).14,43 However, investigations of Pokan et al44 revealed that in sport students, the LVEDV decreased during recovery from incremental exercise. Since the SV remained unchanged, there was an increase in LV ejection fraction (the percentage of SV at LVEDV) immediately after exercise. It was pointed out by the authors that this enhancement of LV ejection fraction was not the result of an improved cardiac output but was caused by a reduction of the left ventricle along with an unchanged SV after the end of exercise. These findings are supported by Dawson et al.45

**Intensity and Duration of Recovery Phases**

Although both time and intensity of peak workloads are suggested to be primarily responsible for the extent of acute physiological responses to intermittent exercise, the applied intensity and duration of recovery phases are also relevant variables. On the one hand, the setting of recovery phases affects the VO\(_2\) values during recovery, influencing the time that is necessary to reach VO\(_2\)\(_{\text{max}}\) values during the following peak-workload phases. On the other hand, \( P_{\text{rec}} \) and \( t_{\text{rec}} \) affect the muscle metabolic recovery that is crucial to maximize the work capacity during the subsequent intervals.14 The muscle metabolic recovery was reported to be manipulated by several variables.

The decrease of blood H\(^+\) and La concentrations during recovery became greater with recovery duration.46 The optimal recovery intensity to reduce blood La is controversially discussed, but it is supposed to be between rest (passive recovery) and \( P_{\text{LTP1}} \) since at this range of intensities no “new” lactate is shifted from the muscle into the blood. However, Hermansen and Stensvold47 showed that blood La removal was faster when recovery intensity was higher (up to 65% VO\(_2\)\(_{\text{max}}\)), concluding that an active recovery facilitates La oxidation in the working muscles. Repeated-sprint performance, CrP resynthesis, and muscle oxygenation were reported to be lower when using active recovery compared with passive recovery.14 The resynthesis of CrP,14 as well as the restoration of the oxymyoglobin store in the working muscle,8 was shown to be impaired only if \( t_{\text{rec}} \) was too short. Seiler and Hellelid48 found out that moderately trained runners who were asked to self-select their recovery modality during HIT preferred a walking (low-intensity) recovery mode.

An additional aspect is that if the aforementioned equation, \( P_{\text{mean}} = (P_{\text{peak}} \times t_{\text{peak}} + P_{\text{rec}} \times t_{\text{rec}})/(t_{\text{peak}} + t_{\text{rec}}) \), is applied for the prescription of HIIE, and if \( P_{\text{mean}} \), \( P_{\text{peak}} \), and \( t_{\text{peak}} \) are matched, a passive recovery mode entails a much shorter recovery duration than an active recovery mode, so the acute physiological responses (even the cardiorespiratory responses) might be markedly higher during HIIE with passive recovery.

**Practical Applications**

To prescribe HIIE intensity by means of %\( P_{\text{max}} \), \( P_{\text{LTP2}} \) (or \( P_{\text{VT2}} \)), and \( P_{\text{LTP1}} \) (or \( P_{\text{VT1}} \)) assessed in a preliminary incremental exercise test allows for an individual and very accurate setting of each single interval parameter. This is of high relevance in scientific studies, as well as in the training process of athletes and patients. However, the implementation of HIIE training in the field, particularly in groups, might be difficult since \( P_{\text{peak}} \) and \( P_{\text{rec}} \) are thought not to be controlled by HR but by power output (velocity or Watts). This problem can be solved if the velocities of work and rest phases are set by means of time per distance or controlled by a pacer.

In addition, this HIIE prescription mode based on the equation \( P_{\text{mean}} = (P_{\text{peak}} \times t_{\text{peak}} + P_{\text{rec}} \times t_{\text{rec}})/(t_{\text{peak}} + t_{\text{rec}}) \) has another interesting aspect. Through the manipulation of \( t_{\text{rec}} \), different mean workloads (\( P_{\text{mean}} \)) can be gained. Given a short \( t_{\text{peak}} \) (which is associated with a low La production), \( P_{\text{mean}} \) and cardiorespiratory strain can be substantially reduced by an extension of \( t_{\text{rec}} \), and therefore we suggest that this type of HIIE can be applied within high-volume training in the first preparatory period despite high peak workloads. This is particularly important for endurance athletes, who are reported to spend 70% to 80% of their total training volume in zone 1 (below 60% VO\(_2\)\(_{\text{max}}\) or 70% HR\(_{\text{max}}\)).49 In contrast, if \( t_{\text{rec}} \) is shortened (or \( t_{\text{peak}} \) is lengthened), \( P_{\text{mean}} \) (and cardiorespiratory strain) increases, and this interval type can be used directly before the competition period, depending on the structure of the specific sport.

By this means, the application of high intensities between \( P_{\text{LTP2}} \) and all-out exercise are of high relevance not only for endurance athletes50 but also more for
racket- or team-sport players, since repeated-sprint ability is thought to be main aspect of competition in this kind of sport.\textsuperscript{12}

Conclusions

There is a large variety in HIIE regimens applied in exercise training and scientific studies, and a standardized and consistent model for the prescription and classification of HIIE is still missing. In this regard, we suggest that turn-point or threshold models are the appropriate solution to achieve homogeneous exercise stimuli across subjects in contrast to the use of percent HR\textsubscript{max} and VO\textsubscript{2}\textsubscript{max}. In addition, the HIIE prescription mode based on the equation \( P_\text{mean} = \frac{P_{\text{peak}} \times t_{\text{peak}} + P_{\text{rec}} \times t_{\text{rec}}}{t_{\text{peak}} + t_{\text{rec}}} \) represents a systematic and consistent approach to standardize HIIE methodology. The prescription of intermittent exercise substantially affects the level of acute physiological responses and, later, the attainment of specific training adaptations. The physiological reactions induced by intermittent exercise, such as La production and elimination or \%VO\textsubscript{2}\textsubscript{max}, should be taken into account for the setting of the HIIE variables \( P_{\text{peak}}, t_{\text{peak}}, P_{\text{rec}}, t_{\text{rec}} \) and \( P_\text{mean} \) and for the classification and denomination of intermittent exercise.

References

