HEART RATE TRAINING

A SPECIAL REPORT FROM

PEAK PERFORMANCE

The research newsletter on stamina, strength and fitness

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From the editor

In a romantic novel, the heart flutters, hearts are frozen and hearts are warmed, hearts are stolen and hearts are broken. In training and sport, the heart is somewhat more practical, but unfortunately sometimes just as vulnerable.

This mini-report has two aims. The first is to help explain to coaches and athletes how heart rate monitoring can assist or detract from improved performance. The second is to highlight a worry that exists even amongst super-fit athletes; cardiac failure.

The first two chapters are concerned with measuring heart rate to enhance training. The first chapter focuses on heart rate variability, where every second counts. The second chapter analyzes the strengths and limitations of heart rate training, looking at every aspect of training, from VO_2max to fat burning.

The final chapter in the trio is probably the most important. It addresses the Sudden Cardiac Death phenomenon, which has tragically taken the lives of athletes approaching or during their prime. Not only does it explain the causes, but also provides preventative ideas and methods.

The heart is a wonderful thing, our engine that drives us on, so it's worth taking care of. I hope this mini-report proves beneficial.

Sam Bordiss
Editor

Heart rate variability – what is it and how can it be used to enhance athletic performance?

Heart rate monitors provide important feedback about the intensity of exercise, but can't measure the cumulative fatigue of workouts or the subsequent training effects. However, new innovations using heart rate variability data mean that's about to change.

Introduction

In practice, it's difficult to assess accurately the effect of training on the body. How do you fix your training load? How well is your body adapting to the training? Is there any accumulated fatigue and how much rest do you need for recovery? Other questions that you need to ask are – how do I know I am getting the right training effect? Have I improved? Am I over- or undertraining?

At rest your body system is in balance. To achieve a training effect, you need to disturb this balance by putting the body under an adaptive stress to which it can react. This stress is known as training and your body's reaction to training is called a training effect.

Traditionally, training zones have been established from fixed formulae. You may be familiar with some of them: using percentage of maximum heart rate or heart rate reserve, percentage of estimated maximum oxygen uptake (VO_2max) or estimated VO_2max reserve, lactate thresholds or a combination of these variables. Heart rate during training gives information on the momentary intensity of exercise but does not take into account the cumulative effect of exercise duration.

Recent research has focused on the use of heart rate variability (HRV) to assess training load, training adaptation and cumulated fatigue[1] and there are now some commercially available products to assist the serious trainer in using HRV to improve athletic performance.

'HRV data can indicate the impact of fatigue due to prior exercise sessions, hydration levels, stress and even the degree of performance anxiety, nervousness or other external stressful influences'

Polar's OwnIndex fitness test monitors resting heart rate (RHR) plus HRV to provide an indication of oxygen uptake. The use of HRV measurement by Polar has been further developed with the introduction of the OwnOptimizer feature. This is an overtraining test, which evaluates individual heart rate response to exercise using HRV and enables the user to optimise their training loads and recovery times. Suunto has products that use HRV to assess training load and accumulated fatigue (for a scientifically balanced view of HRV the reader is referred to an excellent review paper *Heart Rate Variability in Athletes*[2]).

Whilst innumerable studies have been published concerning training in general, relatively few studies are available on HRV and its application to athletes. Most studies involve small numbers of participants, which diminishes the power of the statistics, although since the review paper, significant progress has been made in the practical use of HRV to monitor fatigue accumulation during exercise.

What is heart rate variability?

Measurement of the beat-to-beat interval of the heart clearly shows that heart rate is not constant but alters from beat to beat. This is known as heart rate variability (HRV). At rest this beat-to-beat interval fluctuates with the breathing cycle – it speeds up during inhalation and slows down during exhalation.

This variation is due to the attenuation of the para-sympathetic activity to the heart during inhalation. Heart rate is regulated predominately by the autonomic nervous system (ANS). The ANS describes the nerves that are concerned with regulation of bodily functions and these nerves function without consciousness or volition; the autonomic nerves comprise sympathetic and parasympathetic nerves – sympathetic nerves

excite the heart, increasing heart rate, and parasympathetic nerves reduce heart rate.

During exercise, heart rate is regulated by increased sympathetic activity and reduced parasympathetic activity, causing the heart rate to rise. The relative roles of the two activities depend on the exercise intensity. A 1989 study was the first to test this hypothesis, with the data supporting a progressive withdrawal of parasympathetic activity during exercise[3]. A number of subsequent studies have concluded that HRV is a valid technique for non-invasive measurement of parasympathetic activity during exercise.

Measurement of HRV involves analysis of the R-R (beat-to-beat) intervals, with the simplest approach calculating the mean R-R interval. By accurately measuring the time interval between heartbeats, the detected variation can be used to measure the psychological and physiological stress and fatigue on the body during training. Generally speaking, the more relaxed and unloaded (free from fatigue) the body is, the more variable the time between heartbeats.

An important reality that all athletes and coaches should recognise is that incomplete recovery times will produce significant fatigue. In short, there is a cardiovascular (sympathetic and parasympathetic) form of fatigue that HRV can detect[5].

HRV is measured in milliseconds. During exercise HRV is reduced as heart rate rises. When the body is under a training load, HRV becomes more uniform. This data can be used to calculate information about the body during exercise to a high degree of accuracy.

HRV data can indicate the impact of fatigue due to prior exercise sessions, hydration levels, stress and even the degree of performance anxiety, nervousness or other external stressful influences. Studies have shown that it varies within individuals according to size of left ventricle (inherited trait), fitness level, exercise mode (endurance or static training) and skill (economy of exercise)[2]. Body position, temperature, humidity, altitude, state of mood, hormonal status, drugs and stimulants all have

an effect on heart rate and HRV[2], as do gender and age. The general conclusion is that all of the HRV parameters are higher in men but that this gender difference is confined to men and women below 40-50. There's also an age-related decrease in HRV, although for elderly athletes with a lifelong training history, this decline is reduced.

How can HRV be used to enhance athletic performance?

Well-timed rest is one of the most important factors of any training programme. The effect of training sessions can be negligible or even detrimental if insufficient rest and recovery is built in. HRV measurements demonstrate a significant and progressive decrease in parasympathetic activity during long-term heavy training, which is followed by an equally significant increase during rest. Sympathetic activity shows the opposite trend[4].

There is also evidence to suggest that, when recorded overnight, HRV seems to be a better tool than resting heart rate to assess accumulated fatigue and that HRV may be a valuable tool for optimising individual training profiles[4].

This cardiac autonomic imbalance suggests that HRV is a useful parameter to detect overtraining (a state of overstress caused by an imbalance between training/competition and recovery) and under-recovery in athletes.

Immediately after training, performance potential temporarily decreases, but it begins to rise during recovery. After a certain amount of time, performance rises above the pre-training level because the body is preparing to handle the next training load better than before.

If the body does not receive the next training load within a certain period of time any performance gain begins to slowly decrease. However, if the next high-intensity session is held before the body has recovered from the previous one, performance will remain lower than it would have been after full

recovery. Continuous hard training with insufficient recovery will slowly lead to lower performance and a long-term state of overtraining. When overtrained, even a long period of recovery may not be enough to return performance to the original level.

The body needs time for recovery after a single high-intensity session, or a hard training period of several days, or even after a low-intensity but long training session. Without rest, adaptation to the training load will not occur. In the worst case, training will lead to exhaustion and overtraining or under-recovery. Additional non-training stress factors and monotony of training may also contribute to overtraining syndrome.

EPOC

Until recently there were no useful methods of monitoring fatigue accumulation during training. Scientists have now demonstrated that excess post-exercise oxygen consumption (EPOC) can be predicted from HRV data recorded during exercise. Consequently, EPOC prediction may serve as a tool for monitoring fatigue accumulation during exercise[6].

'Scientists have demonstrated that excess post- exercise oxygen consumption can be predicted from HRV data during exercise. Consequently, EPOC prediction may serve as a tool for monitoring fatigue accumulation during exercise'

EPOC, simply defined, is the amount of oxygen your body needs to recover after a training session and is measured in millilitres of oxygen per kilogram of body weight (ml/kg). EPOC calculated from HRV data is therefore a measure of physiological training load and the accumulated cardiovascular fatigue.

EPOC is most useful to describe the stress caused to the body, especially to the respiratory and cardiovascular system, from endurance activities such as running, cycling, swimming and rowing. During exercise the body consumes more oxygen than at rest. The higher the intensity of training, the greater the fatigue and the more oxygen is consumed during and immediately after the training session. Simply put, a higher EPOC value means that the body is more physiologically tired.

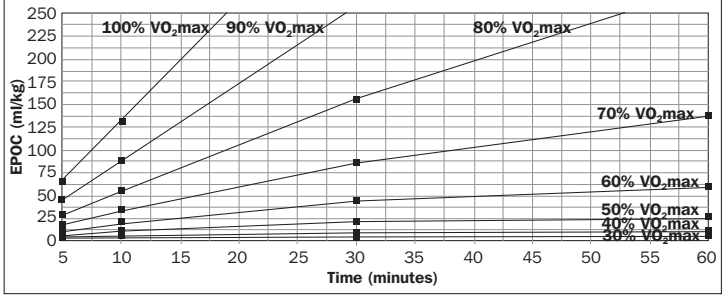
EPOC accumulates faster as training intensity increases but not necessarily when duration is increased, so low-intensity training may not result in a high EPOC value, even if the duration of the training is exceptionally long. With high-intensity training, high EPOC values can be reached even in a short period of time.

Without EPOC as a measure, the wrong conclusions may be drawn from a training session. You may believe that no improvement has occurred or performance has gone backwards, when in reality the difference is fatigue, and actual performance has improved.

At this point it is reasonable to ask the following question: if you can use heart rate as a measure of exercise intensity, why do you need HRV? The simple answer is that during two separate training sessions of equal status one may be harder on the body than the other, even when the heart rate is the same for both sessions. The difference is accumulated fatigue, which HRV can detect and convert into an EPOC value.

The body may appear to recover rapidly from a training session (short-term fatigue) but carry accumulated (long-term) fatigue from training session to training session. This long-term fatigue builds up over time and is one reason why periodised training programmes, which build in easier recovery weeks, are needed. So when comparing sessions using HRV and EPOC, the amount of accumulated fatigue affecting each session can be seen by the difference in EPOC value *(see figure 1)*.

Figure 1: The effect of training duration and intensity (%VO2max) on EPOC accumulation



Is there a tool to help athletes to use HRV and EPOC to optimise their training and recovery?

Suunto has developed a heart rate monitor (Suunto t6), which uses an athlete's unique physiological fingerprint to measure

training effect. It looks like a conventional heart rate monitor and uses HRV and EPOC to monitor the amount of stress that the body is experiencing to measure the cumulative fatigue (training effect) of each training session.

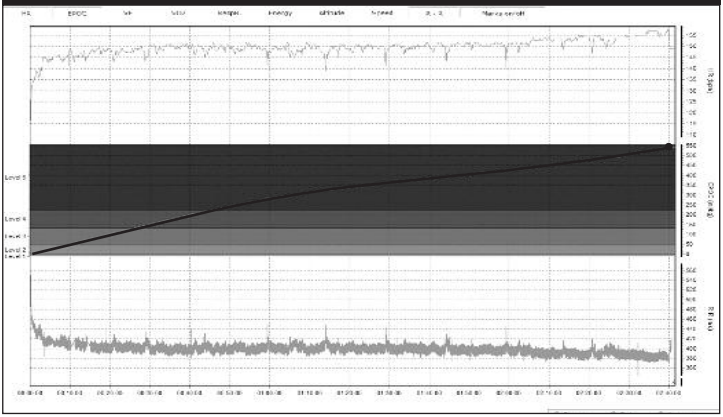
Based on accurate measurement of the time interval between heartbeats and the detected variation, the t6 software (run on a PC) calculates information about the performance of the body during training and displays the data in a user-friendly format for analysis. As the data is updated, the Suunto t6 becomes an increasingly precise tool for measuring training performance.

HRV and EPOC in action

HRV and EPOC work well with continuous (rather than interval) type training. At low intensity exercise (40-70% of VO_2max) there is a significant correlation between EPOC and blood lactate concentration. At maximal exercise the correlation is low, signifying that other factors such as body temperature and hormonal changes may influence EPOC, fatigue accumulation and recovery during high-intensity exercise[6].

In figure 2, a graph of EPOC data collected for a British record rowing marathon on the Concept 2 ergometer is shown:

Figure 2: A graph of EPOC data collected during British record rowing marathon



- The top trace is heart rate, which was kept below 90% of maximum with an even rowing pace;
- The middle trace is the EPOC (note that despite a level heart rate the graph continued to increase as fatigue accumulated);
- The bottom trace is the HRV, the R-R intervals (note the slow narrowing of the time interval between beats).

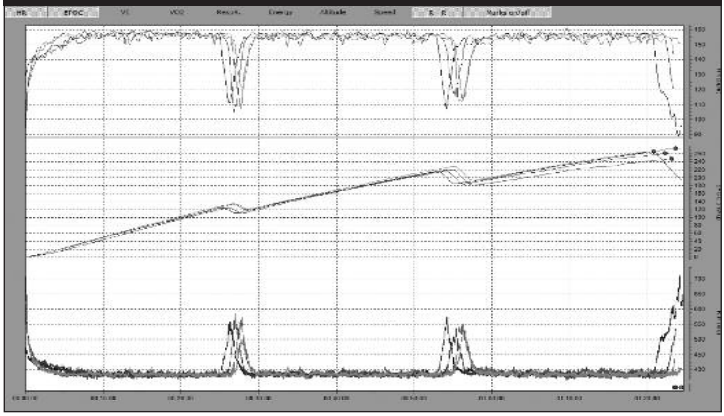
In this particular model there are five levels of EPOC. Recovery from each level is as follows:

- Levels 1 and 2 – 3 hours to 1 day
- Level 3 – 1-2 days
- Level 4 – 1-4 days
- Level 5 – 2-7 days

The exact amount of recovery required would depend on how long the training remained in a particular EPOC level. With this marathon the rower was into level 5 EPOC after 45 minutes and spent just under two hours at that level – recovery from this extended bout of exercise can be measured in weeks rather than days.

Figure 3 is an overlay graph of a rower doing an 18,000m row (split into 3 x 6,000m) on four separate occasions; two in week 1

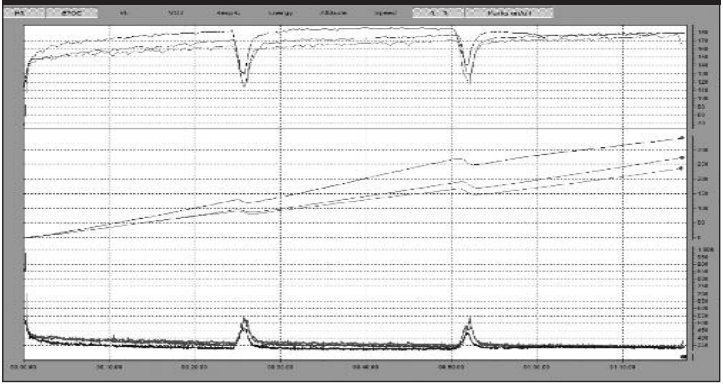
Figure 3: EPOC data acquired during 18,000m row (3 x 6,000m) on four separate occasions



of a periodised programme and two in week 4. Note the tight correlation of heart rate, EPOC and R-R interval, indicating that this athlete recovered well between sessions and that the training programme had the right balance of exercise intensity and rest.

Now let's look at figure 4 – an example of the same session (different athlete) giving different EPOC values each session and therefore requiring varying recovery periods. Here the periodisation wasn't right and the athlete was under-recovering and accumulating fatigue.

Figure 4: An example of different EPOC values, each session requiring varying recovery periods



Summary

The way in which the cardiovascular system responds to the stress of exercise continues to intrigue physiologists. Although some understanding of HRV and its application to athletes is becoming clearer, it is still almost an unexplored domain. The significant change is that there is now a commercial product available that athletes can begin to use to monitor this 'cardiovascular fatigue' to ensure that their training programmes include the right mix of duration, frequency, intensity, rest and recovery. HRV and EPOC can be used to monitor individual sessions, allowing the athlete and coach to react immediately to the output data by either amending subsequent training to

deal with accumulated fatigue or ensuring that the athlete has sufficient rest to make an adequate recovery.

Eddie Fletcher

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Heart rate training limitations – don't be a slave to the rhythm!

Athletes have used heart rate training successfully for years. However, slavish adherence to heart rate training zones won't allow an athlete to reach his or her true potential. Here the strengths and limitations of heart rate monitoring and heart rate training are discussed.

Introduction

For centuries, heart rate monitoring consisted of placing an ear or a stethoscope on the chest. The heart's electrical activity was first recorded in the 19th century and American biophysicist Norman Holter invented a portable device for recording the heart's activity in 1961. The Holter monitor can record the heart's electrical activity for 24 hours or more, but it is too large for recording heart rate during exercise. Watch-like heart rate monitors were first developed in the 1980s, and since then most endurance athletes have used heart rate training[(1)]. It is claimed that today's heart rate monitors can perform all sorts of wonders, from predicting maximal oxygen uptake to detecting overtraining; in this article we'll examine the validity of these claims.

Prediction of VO_2max

Strengths: Some heart rate monitors can be used to predict VO_2max (maximal oxygen uptake). For example, it has been shown that the Polar S810 heart rate monitor can accurately predict VO_2max, which is a strong predictor of endurance performance and the best measure of health-related fitness. In different tests, most participants' estimated VO_2max scores

were within 5ml/kg/min of their directly measured scores[2]. The S810 is also highly reliable, yielding consistent results test after test[1]. What's more, S810-predicted VO_2max was favourably associated with cholesterol and blood pressure in a study of 3,820 men and women aged 25-64 years[3].

Limitations: In the example above, the Polar S810 predicts VO_2max from age, height, weight, gender, self-reported physical activity level, and resting heart rate measurements. Strict adherence to testing procedures is required because, as the manufacturer acknowledges, a number of factors influence resting heart function, including noise, temperature, time of day, diet, alcohol, smoking, exercise, and pharmacological stimulants. Be aware also that self-reported physical activity level is prone to error.

It is also noteworthy that the heart rate monitor penalises the 'fat fit' by expressing VO_2max relative to body weight. When we directly measure maximal oxygen uptake in our lab, we express the results relative to body weight and independent of body weight (millilitres of oxygen per kilogram of body weight per minute and litres of oxygen per minute, respectively). In our lab, for example, we might determine that a 110-kg middle-aged man has a VO_2max of 4 litres per minute, which is excellent compared to an untrained man of the same age. When this value is expressed relative to body weight, the same individual has a VO_2max of 36ml/kg/min, which is no better than an untrained man of the same age. Unfortunately, VO_2max is rarely expressed independent of weight outside of the laboratory.

Progress monitoring

Strengths: A reduction in heart rate for a given intensity is usually indicative of an improvement in fitness. In our experience, previously inactive individuals and those returning from a period of injury are often delighted to see a reduction in heart rate for a given intensity after only four weeks of aerobic training.

Limitations: A number of factors other than a change in fitness might explain why heart rates can differ from one test to another; for example: natural biological variation is such that

heart rate can vary by 2-4 beats/min from one day to the next; dehydration can increase heart rate by up to 7.5%; heat and humidity can increase heart rate by around 10 beats/min; and, altitude can increase heart rate by 10-20%, even with acclimatisation[4]. Trained individuals are unlikely to experience discernible reductions in submaximal heart rates and improvements in fitness are best identified from changes in the blood lactate response to exercise *(see 'determining training zones', p24)*.

Measuring exercise intensity

Strengths: In normal individuals, there is a linear relationship between heart rate and intensity during incremental exercise[5]. Therefore, exercise intensity can readily be expressed as a percentage of predicted or directly measured maximum heart rate. The near linear relationship between heart rate and oxygen uptake is such that exercise intensity can also be expressed as a percentage of VO_2max.

Limitations: Maximum heart rate is best determined in a graded exercise test, but a maximal exercise test can be inappropriate, especially in less well-trained individuals. Maximum heart rate can be predicted from the formula 220-age, from the formula 210 – (age x 0.65), or from the formula 207 – (age x 0.7); however, all predictions are subject to error[5,6].The variation in heart rate among the normal population is such that the predicted maximum heart rate of 95% of individuals of a given age will lie within a range of 40 beats/minute[5].

It is an oversimplification to suggest that there is a linear relationship between heart rate and oxygen uptake. For example, oxygen uptake often increases relatively more than heart rate during high-intensity exercise. The relationship between oxygen uptake and heart rate can be predicted more accurately when oxygen uptake is expressed as a percentage of heart rate reserve (HRR, the difference between maximum heart rate and resting heart rate)[7]. In fit people, for example HRR (beats/min) = 1.05VO_2 – 4.1. Don't concern yourself with these equations

however; the next section explains that lactate threshold should be used to determine exercise intensity, not VO_2 or heart rate.

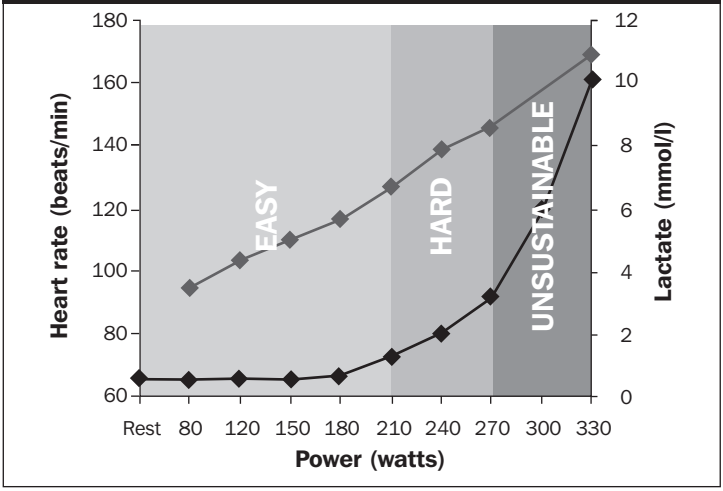
Determining training zones

Strengths: Heart rate monitoring allows individuals to train at the intensity recommended to improve aerobic fitness, which is 50-90 % of maximum heart-rate[8].

Limitations: Too many athletes don't reach their true potential because they adhere to ill-conceived heart rate training zones. Untrained individuals will enjoy improvements in fitness at 50% of maximum heart rate. Trained individuals require more individualised exercise prescription.

Training zones are best determined from the blood lactate response to exercise. Figure 1 shows the blood lactate response to incremental exercise in a 46-year-old club cyclist. Notice that blood lactate does not increase above resting levels until power output reaches 210 watts. In this individual, exercise up to 210 watts is comfortable, sustainable, and ideal for long-duration or recovery training.

Figure 1: Heart rate and blood lactate responses to incremental cycling



As exercise intensity increases, the cyclist recruits more fast-twitch muscle fibres and produces more lactic acid. His body attempts to buffer lactic acid by combining it with carbonic acid, a weaker acid that splits in the lungs into water and exhalable CO2. From 210-270 watts, the cyclist's increased breathing is sufficient to expel CO_2 and buffer blood lactate. In this individual, exercise at 210-270 watts is difficult, but it will improve his ability to tolerate and dispose of blood lactate.

'This improvement in fitness would not have been detected if we had only used a heart rate monitor because his heart rate at each workload did not change over time'

At 270 watts, there is a rapid rise in blood lactate concentration. This is known as the 'lactate threshold'. The increase in blood pH (acidity) stimulates a dramatic increase in breathing (as the body attempts to expel CO_2) and exercise beyond the lactate threshold is not sustainable. Exercise beyond the lactate threshold is ideal for interval training, however. In order to improve endurance and 10-mile time trial performance in this individual, we might recommend three or four 5-minute bouts at 280 watts with 5-minute recovery bouts at 160 watts. In order to improve strength and sprinting speed, we might recommend ten 1-minute bouts at 330 watts with 3-minute recovery bouts at 160 watts.

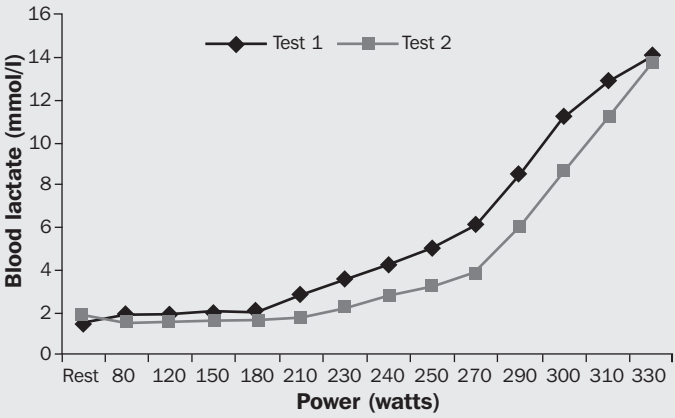
Lactate and heart rates

The data in figure 1 were derived from a cyclist who had a power meter on his road bike. Thus, he was able to train at the correct intensity outside of the lab. Subsequent visits to the lab showed that his blood lactate curve shifted to the right and his power output at lactate threshold increased to 290 watts *(see figure 2, overleaf)*. This improvement in fitness would not have been detected if we had only used a heart rate monitor because his heart rate at each workload did not change over time.

If the cyclist did not have a power meter, we would have anchored much of his training around the heart rate at the lactate threshold. We would also have advised him not to expect his heart rate to recover between intervals and to ignore increases in heart rate of 5-15% during an hour of steady state exercise[4].

In runners, we often prescribe exercise intensity around the speed at lactate threshold rather than the heart rate at lactate

Figure 2: Blood lactate responses to exercise before and after 12 weeks of training



threshold. In runners, cyclists and all athletes[4], it is important to stress that there is no predictable relationship between heart rate and lactate threshold. Lactate threshold tends to occur at around 90% of maximum heart rate in well-trained individuals, but it can occur at 50-90% of maximum heart rate[9].

When used in accordance with the author's instructions, Borg's[10] rating of perceived exertion (RPE) scale can also be used to determine exercise intensity outside of the lab because an RPE of 13-14 often occurs at the lactate threshold, regardless of gender, mode of exercise, and training status[11-13]. We recognise that many individuals will find RPE too subjective, and will be reluctant to train without a heart rate monitor. However, the running and cycling workouts in table 1 and table 2 are designed to show that it is possible to train effectively without a heart rate monitor (or a power meter).

The running workouts can readily be adapted for faster runners (and pace calculators are available online; for example: www.nemonisimors.com/anders/sports/paceCalculator.php). A runner wishing to run 10k in 39 minutes should decrease the duration of the 400m intervals to around 1:22 (with 1:10 rest intervals), decrease the duration of the 1,000m intervals to around 3:45 (with 2:45 rest intervals), and run at 15.4km/h for 15 minutes

Table 1: Using RPE to train effectively without a heart rate monitor

Day	Time	Intensity (RPE)	Distance*
Mon	60 min	Light (10-11)	10-11km
Tue	45 min	Hard (14-16)	6 x 400m in 1:37 (1:20 recovery between reps)†
Wed	Rest	-	-
Thurs	40 min	Light (10-11)	7km
Fri	60 min	Hard (14-16)	4 x 1000m in 4:15 (2:45 recovery between reps)†
Sat	Rest	-	-
Sun	45 min	Moderate (12-13)	15 min at 13.33km/h (4:40 per km, about 3.4km)‡

Table shows the first week of a four-week training programme designed for an individual aiming to run 10k in 45 minutes (time is min:sec). Do one of the following at the start of week two and week three: slightly increase the number of reps; slightly decrease the recovery time; or, slightly increase the speed. The fourth week (racing week) should be an easy one, including some intense but very short sessions. *Using a cycle and cycle computer, measure a safe path or a safe loop that you are used to running, and place a mark at 400m and at every kilometre. †Moderate and hard sessions should be accompanied by a 20-minute warm-up and a 10-minute cool-down. ‡The moderate session can be performed all year round in order to improve your ability to sustain race pace. The duration of the moderate session should be increased as fitness improves, for example: 20 or 25 minutes at 13.33km/h in this context.

Table 2: Cycling workouts without heart rate monitoring

Session	Objective	Format
Boardman Specials[16]	Improve 1-hour time trial performance	Twenty to sixty 10-second efforts at 1-hour average power with 20-second recovery intervals
All-out sprints[47]	Improve acceleration and ability to tolerate blood lactate	Eight to twelve 30-second all-out efforts with 4:40 recovery intervals
Eric Snider's weekly cadence [8] workout[1]	Cycle like Lance Armstrong to avoid fatiguing large gears!	Spin as fast as possible for 1 min in easy gear such as 39 x 19. Shift back to your usual pace gear for two min. Repeat 6-10 times.
CTS Time trial	Improve endurance and 10-mile time trial performance recovery intervals at 90 rpm	Three or four 5-minute sprints at around 110 rpm with 5-minute

Each session is best performed on a stationary cycle or turbo trainer, and should be accompanied by 1) a 10-minute warm-up with a 1-minute sprint at 03:00 and a 1-minute sprint at 05:00, and 2) a 6-10 minute cool-down. The CTS Time Trial is adapted from the excellent Carmichael Training Systems Train Right Video Series, which is available at www.wiggle.co.uk (each DVD includes a heart rate test that can be ignored – just go by perceived effort during the intervals).

(equivalent to 3:54 per km) during the weekly moderate session. The all-out, self-paced nature of the cycling workouts is such that they will automatically adjust to changes in fitness.

Fat burning

Strengths: None.

Limitations: Many so-called fitness professionals would have you believe that a 'fat-burning zone' exists at 60-70% of age-predicted maximum heart rate. This is nonsense. It is true that the rate of fat metabolism is greater during moderate-intensity exercise, but fuel utilisation cannot usually be measured outside of the lab. What's more, when fuel use has been measured in the lab, it has been found that the optimum intensity for fat burning is different in each individual, varying from 54-92% of maximum heart rate[14].

Some fitness professionals have purchased expensive gas analysers and are measuring fuel use outside of the lab. However, this still misses the point. Weight loss is induced by negative energy balance; you'll burn more calories during high-intensity exercise than during moderate-intensity 'fat-burning' exercise. For example, an 80-kg individual will burn around 11 calories per minute when jogging at 5mph (12 min/mile), around 16 Calories per minute when running at 7mph (8.5 min/mile), and around 22 Calories per minute when running at 10mph (6 min/mile). Advocates of the fat-burning zone also fail to realise that high-intensity training is more effective in improving one's ability to burn fat. This 'carbohydrate-sparing' effect does not accompany moderate-intensity training.

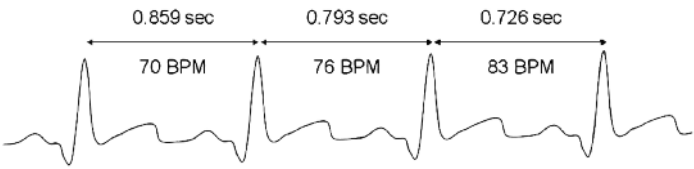
Preventing overreaching and overtraining

Strengths: Overreaching is characterised by signs and symptoms that last from a few days to two weeks, including fatigue, muscle soreness, insomnia and underperformance[4]. Overreaching is often utilised in a training cycle because 'supercompensation' may occur after an appropriate period of recovery[15]. It is thought that fatigue, performance decline, mood disturbance

and other symptoms are more severe in the overtrained state than in the overreached state[4]. An athlete may take months or years to recover from overtraining. Some top-of-the-range heart rate monitors can detect a 5-10 beat/min increase in resting heart rate and a decrease in resting heart rate variability *(see figure 3)*, which might be early signs of overreaching and overtraining[4].

Limitations: There is increasing evidence that heart rate variability is lower at rest in the overtrained state, but there is no diagnostic tool for overtraining; the condition is 'diagnosed' by excluding all other explanations for the decline in performance and mood[4]. Perform any overtraining test at least 24 hours after training because heart rate variability can be influenced by a prior exercise bout.

Figure 3: Heart rate variability



Heart rate variability describes the subtle variations in the intervals between consecutive heartbeats. Even when heart rate is stable, the time between two consecutive beats can vary considerably. At rest, heart rate variability is larger in aerobically trained individuals than in untrained individuals – probably as a result of healthy nervous activity in the brain's cardio-inhibitory centre. There is increasing evidence that heart rate variability is lower at rest in the overtrained state. Low heart rate variability is also a risk factor for cardiovascular disease.

Summary

Heart rate monitors can be used to estimate VO_2max, to identify changes in fitness, and, possibly, to detect the early signs of overtraining. Heart rate training zones are meaningless unless they are identified from the blood lactate response to incremental exercise. Heart rate training is particularly inappropriate during interval training. Most well trained individuals don't need a heart rate monitor to know what constitutes an all-out one-minute interval, an all-out 5-minute interval, or an easy recovery interval!

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Why fit athletes suddenly drop dead, and how to stop it happening

Should there be screening programmes to prevent sudden cardiac death?

Introduction

Olympique Lyonnais Stadium, Lyon, 26 June 2003. In the 71st minute of the Confederation Cup semi-final between Cameroon and Colombia, the 28-year-old Cameroonian midfielder Marc-Vivien Foe collapsed. In the few minutes it took to stop play and administer treatment Foe suffered a cardiac arrest and died. An autopsy later confirmed the presence of hypertrophic obstructive cardiomyopathy (HOCM or HCM), a congenital condition that had been dormant until that evening.

The surprise caused by Foe's death was best summed up by a fan who wrote: 'I was and still am deeply saddened and shocked by the news of Marc's sudden death. How could this happen to such a young, fit and strong man like Marc? How could he just fall to the ground and die?'

Although exercise in young people usually brings a host of physical and psychological benefits, for a very small minority rigorous physical activity is a serious threat to life. Most deaths in young people are caused by 'inherited or congenital structural and functional abnormalities' of the heart that trigger cardiac arrhythmias and result in sudden cardiac death (SCD)[1].

Barry Maron, a US physician and leading researcher on SCD, has identified the most common causes of death in young people[2], and has been central to the publication of guidelines

for those with these conditions and their involvement in competitive sports[3,4] *(see table 1)*.

Table 1: Top causes of sudden death in young athletes and recommendations for participation in sport

Cause of death total SCD deaths)	Incidence (% of levels	Recommended participation
Hypertrophic Cardiomyopathy (HCM) and conditions highly suggestive of HCM	33.9	Should only participate in low intensity competitive sports (*eg* bowls)
Commotio cardis	19.9	None
Coronary artery anomalies	13.7	Should only participate in competitive sports six months after surgical correction provided investigations are normal
Myocarditis	5.2	Should be excluded from competitive sports for six months and only allowed to return provided investigations are normal
Ruptured aortic aneurysm	3.1	Those at risk of this condition may participate in moderate-intensity sports provided there is no family history of SCD and aortic root size is within safe limits

Hypertrophic obstructive cardiomyopathy (HOCM)

HOCM is the single largest cause of SCD in young athletes, occurring in 1 in 500 members of the general population. The condition is characterised by a marked thickening (hypertrophy) of the muscle cells (myocardium) of the heart's left ventricle. In 90% of cases, the wall separating the chambers of the heart is also involved.

This prevents blood from leaving the left ventricle and passing normally through the aorta. The combination of outflow obstruction and the thickening results in widespread electrical instability and can lead to a marked increase in ventricular arrhythmias and sudden cardiac death.

At present 3% of adults and 6% of children with HCM die each year. In the UK, HCM was brought to the public's attention by the death in 1992 of Daniel Yorath, a promising young footballer and son of Terry Yorath, the manager of the

Welsh football team at the time. Daniel's case highlights two important issues that can make diagnosing HCM difficult:

- A young professional footballer who had recently signed to Leeds United, Daniel was physically fit and in excellent health. Although HCM can present with symptoms of chest pain, breathlessness and loss of consciousness (which can sometimes lead to seizures), most youngsters are free from symptoms and lead normal, active lives.
- HCM can be inherited from affected parents, but in most cases there is no family history. In Daniel's family history there was no reason to suspect HCM – indeed, Terry Yorath had been a successful professional footballer for many years.

It is clear from Daniel Yorath's case that a thorough history is not enough to identify all cases of HCM. Further investigations usually involve a physical examination and an electrocardiogram (ECG) and cardiac echocardiography. Genetic tests can be done, but these are not widely available.

Commotio cardis

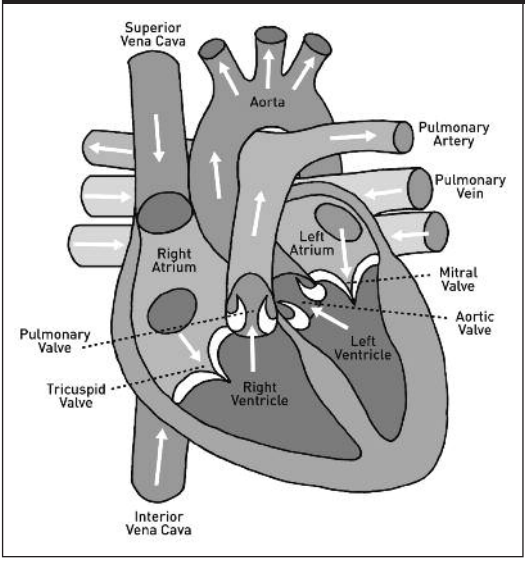
This is where sudden cardiac death is caused by a blunt, often innocent, non-penetrating blow to the chest. In a study published in 2002, 128 cases of commotio cardis had been identified among young athletes in the US over a five-year period. The study found that most victims were male (95%), aged under 18 (72%), and died immediately on the scene (82%)[5].

While most (68%) cases occurred during organised sporting events such as baseball and ice hockey matches, the authors found that the rest had taken place in playgrounds, parks or gardens as a result of innocent blows to the chest. It has been suggested that improvements in protective equipment and better access to portable defibrillators would help to prevent this condition from occurring.

Coronary artery abnormalities

Normally the heart receives its blood supply from the right and left coronary arteries. These emerge from two distinct points in

Figure 1: The heart



the aorta, situated just above the aortic valve. Rarely, the left coronary artery can emerge from the site normally reserved for the right coronary artery. This tends to produce a pronounced kink at the origin of the artery, which during exercise leads to compression of the blood vessel between the aorta and the pulmonary vessels.

The result is a sudden drop in the blood supply to the left ventricle and the destruction of large segments of heart muscle. Although this condition can be cured with surgery, it is difficult to diagnose, often only identifiable with invasive procedures such as coronary angiography.

Myocarditis

Myocarditis is an inflammatory disease that causes destruction of the heart muscle wall, which can result in both heart failure and sudden cardiac death. Innocuous viral infections, with symptoms of flu or a common cold, are the most common cause of myocarditis. It is thought that up to 5% of those with an acute viral illness have some degree of myocarditis.

Although this tends to resolve quickly and leave no long-term effects, a small minority can either deteriorate rapidly or develop a more persistent condition (chronic active myocarditis) which may take months or even years to shake off. Both of these conditions are capable of triggering arrhythmias and causing SCD. It therefore makes sense for young athletes to avoid exercise during a viral illness and to seek medical attention when they experience cardiac symptoms such as chest pain, undue breathlessness or loss of consciousness.

Ruptured aortic aneurysm

The largest group of youngsters at risk from a ruptured aortic aneurysm are those with Marfan's syndrome. This distinctive condition is inherited directly from one parent and is associated with abnormalities in the formation of connective tissue. The result is a number of clinical features *(see box, below)* that can be identified on physical examination, and a high risk of developing abnormalities in the aorta. Provided that the dimensions of the aorta remain within normal limits, those with Marfan's Syndrome may participate safely in a wide range of competitive sports.

Features of Marfan's syndrome

- Tall stature with disproportionately long limbs
- Abnormally long and slender fingers and toes
- Joint laxity leading to an increased range of joint movement
- Spinal deformities such as scoliosis and kyphosis
- High-arched palate
- Visual disorders such as short sightedness and blindness

Other causes of SCD

A number of other diseases and conditions can be responsible for sudden death in young athletes. Although congenital abnormalities account for most deaths, other causes such as asthma and heat stroke can also be responsible. In recent years the increasing use of stimulants such as amphetamines and cocaine has been implicated, though without conclusive evidence. In approximately 2% of SCD no definitive cardiac cause can be found[1].

How to identify those at risk

In 1982 the Italian government brought in a Medical Protection of Athletic Activities Act, which ensured that all young athletes who wished to participate in organised sports activities underwent a regular screening assessment. The assessment had three components: a comprehensive medical history, a physical examination and an electrocardiogram.

The results are impressive: while levels of HCM in the population are similar to those in the United States, reports of sudden death from HCM are unheard of in Italy[6]. The American Heart Association has acknowledged this and has stated that: 'Some form of pre-participation cardiovascular screening for high-school and collegiate athletes is justifiable and compelling, based on ethical, legal and medical grounds[7].

So what should be done? The three components in the Italian screening programme seem an appropriate place to start:

Individual history

A small number of those at risk will reveal clues that may help to identify their condition. In the US, the Sudden Arrhythmia Death Syndrome Foundation recommends a nine-point questionnaire (aimed at parents) for the first stage of SCD screening *(see table 2, opposite)*.

Physical examination

This should be completed by a trained physician and should focus primarily on the cardiovascular system.

Electrocardiogram (ECG)

The 12-lead ECG is abnormal in 95% of those with HCM and often shows irregularities in other fatal coronary abnormalities. Unfortunately in a number of highly trained young athletes, abnormal recordings are common, such as slow heart beat or some forms of heart block which in older sedentary people would indicate a problem. Nevertheless, any abnormal readings should then be followed up.

The next step in the diagnostic process is echocardiography.

Table 2: The Sudden Arrhythmia Death Syndrome Foundation Questionnaire

	Yes	No
Has your child fainted or passed out *during* exercise, emotion or startle?		
Has your child fainted or passed out *after* exercise?		
Has your child had extreme fatigue associated with exercise (different from other children)?		
Has your child ever had unusual or extreme shortness of breathe during exercise?		
Has your child ever had discomfort, pain or pressure in his chest during exercise?		
Has your child ever been diagnosed with an unexplained seizure disorder?		
Are there any family members who had an unexpected, unexplained death before the age of 50 (including SIDS, car accident, drowning)?		
Are there any family members who died of heart problems before the age of 50?		
Are there any family members who have unexplained fainting or seizures?		

The echocardiogram provides a highly detailed view of structural abnormalities in the heart and is considered to be the 'gold standard' test for identifying HCM, aortic root disease and changes in left ventricle dysfunction which characterise severe cases of myocarditis.

Conclusion

It has proved incredibly difficult to adopt a standardised screening programme for sudden cardiac death. With the exception of Italy, no state-wide programme exists in western Europe to identify those at risk. Even in the United States, where the impetus for screening is greatest, only 17 states have adopted a strategy deemed 'adequate' by the American Heart Association.

In the US alone, four million youngsters participate in regular competitive exercise. This means that as many as 8,000 individuals may be living with HCM and putting their lives at risk every time they step on to a basketball court or a football pitch. Shouldn't something better be done?

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