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A Review of the Concept of the Heart Rate Deflection Point

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Contents

Abstract

The heart rate deflection point (HRDP) is a downward or upward change from the linear HR-work relationship evinced during progressive incremental exercise testing. The HRDP is reported to be coincident with the anaerobic threshold. In 1982, Conconi and colleagues suggested that this phenomenon could be used as a noninvasive method to assess the anaerobic threshold. These researchers developed a field test to assess the HRDP, which has become popularised as the 'Conconi test'. Concepts used to define and assess the anaerobic threshold as well as methodological procedures used to determine the HRDP are diverse in the literature and have contributed to controversy surrounding the HRDP concept. Although the HRDP may be assessed in either field or laboratory settings, the degree of HR deflection is highly dependent upon the type of protocol used. The validity

of HRDP to assess the anaerobic threshold is uncertain, although a high degree of relationship exists between HRDP and the second lactate turnpoint. The HRDP appears to be reliable when a positive identification is made; however, not all studies report 100% reproducibility. Although the physiological mechanisms explaining the HRDP are unresolved, a relationship exists between the degree and direction of HRDP and left ventricular function. The HRDP has potential to be used for training regulation purposes. Clinically, it may be incorporated to set exercise intensity parameters for cardiac rehabilitation.

The heart rate deflection point (HRDP)[1-8] is known in the literature as the 'deflection velocity',[9,10] 'heart rate break point',[11] 'slope variation point',[12] 'heart rate threshold'[13-19] or the 'heart rate turnpoint'.[20,21] It is characterised by a distinctive differentiation or 'deflection' in the linear HR-work relationship or heart rate performance curve (HRPC)[20-26] exhibited during progressive incremental exercise testing.

The HRDP normally evinces a decrease in the slope of the HR-work relationship (fig. 1) and is considered a 'regular' type of deflection.[22] This is visually manifest as a curvilinear response and is reported in the range of 88 to 94% of maximum HR.[3,5,11,16,22,28,29] In some instances, a regular HRDP may exhibit a phasic flattening of the HRPC followed by another linear rise with decreased slope.[12] However, inverse deflections or increases in HR slope have also been reported.[20,22,23,26,30] A study of 227 healthy young males showed that 7.9% of deflections were of an inverse nature.[22]

Fitness state does not appear to regulate the occurrence of HRDP. Both trained[2,9-11,15,27,28,31-35] and untrained individuals[11,13,14,17,23,24,33,35,36] have evinced the HRDP. HRDP has also been observed in individuals with cystic fibrosis[15,37] and heart disease,[20] and paraplegic athletes.[6] HRDP is not dependent upon age. Children,[38-43] adolescents,[39] university students[22] and middle-aged men[25,29] have all demonstrated evidence of this deflection phenomenon.

The intriguing aspect of the HRDP is the reported coincidence with the 'anaerobic threshold'.[44] The hypothesis that this HRDP alone can be used to assess the anaerobic threshold, in runners at least, was first suggested by Conconi et al.[9] Their research demonstrated that running speeds at the HR deflection point were highly related to running speeds at the anaerobic threshold. Conconi et al.[9] developed a simple field test by which to assess the HRDP, and this methodology has since been expanded to include other sporting activities in both field and laboratory settings.

The HRDP test is appealing since it is noninvasive. Additionally, the time necessary to conduct the test is relatively short (approximately 20 to 30 minutes) which means that it can be incorporated within or as part of a training session. This testing method is popular in Europe and is used to assess training programmes and evaluate endurance capacity.[45] Clinically, the HRDP may be used as an inexpensive, noninvasive marker by which to set exercise parameters for cardiac rehabilitation.[20,30]

However, the concept of the HRDP is somewhat controversial. Some individuals exhibit a completely linear HR response.[1,2,11,16,21,22,33,36,45] Furthermore, there is not complete agreement in the literature with respect to the HRDP and its relationship to the anaerobic threshold. The methods used to determine and calculate the HRDP and nomenclature for the anaerobic threshold concept may, in part, explain these discrepancies. Furthermore, the physiological mechanisms responsible for the deflection in HR are not fully understood.

1. Concept of the Anaerobic Threshold

Since the HRDP involves a testing method using noninvasive parameters to assess the anaerobic threshold, a discussion of this concept should help to set the definitions for the review.

The anaerobic threshold is accepted as a measurement of the ability to perform at an optimal intensity for prolonged periods of time[46] and is strongly related to endurance performance.[47-52] The anaerobic threshold was initially defined as a work intensity or level of oxygen consumption ($\dot{V}O_2$) preceding the onset of metabolic acidosis and the associated changes in gas exchange that take place.[44] The term 'anaerobic' is controversial and a matter of debate.[53,54] More conventional terminology designates this threshold as the lactate threshold (LT),[53] the workload beyond which blood lactate levels abruptly increase during progressive intensity testing.

The ventilatory threshold (VT) is reported to be coincident with the LT.[44] The VT is described as the workload beyond which there is an abrupt nonlinear increase in ventilation ($\dot{V}_E$) during progressive intensity testing.[53] This hyperventilation relative to oxygen consumption ($\dot{V}_E/\dot{V}O_2$) without a simultaneous increase in the ventilatory equivalent for CO_2 ($\dot{V}_E/\dot{V}CO_2$) also represents the VT.[55]

It is hypothesised that 3 phases of energy supply contribute to the work necessary to progress from low to high intensity exercise. These phases are identified by 2 transition or breakpoints.[56] Energy supply in the first phase is derived completely from aerobic metabolic processes. The first breakpoint, the beginning of phase II, is designated the 'aerobic threshold' and is characterised by an increase in blood lactate levels from around 2 mmol/L and a disproportionate increase in $\dot{V}_E/\dot{V}O_2$. Phase III is designated the 'anaerobic threshold' and is characterised by the second breakpoint, wherein there is an increase in blood lactate levels from around 4 mmol/L and a further increase in $\dot{V}_E/\dot{V}O_2$.[56]

The second breakpoint is relevant, for it is considered to be representative of the maximal lactate steady state (MLSS).[57] The MLSS may be defined as the highest constant workload in which lactate release into and removal from the blood is in equilibrium.[57]

2. Historical Development of the Heart Rate Deflection Point (HRDP)

2.1 Early Observations

In 1968, Brooke et al.,[58] and again in 1972 Brooke and Hamley,[59] observed that the HR response to incremental testing does not always indicate a strictly linear coupling. Their findings showed that amongst a group of racing cyclists, some individuals demonstrated a HR/workload relationship that was sigmoidal. This HR/physical work curve was characterised by 3 distinct sequential phases: an anticipatory phase, a linear phase where the slope of HR was consistent, and a curvilinear phase where the slope of HR/workload decreased and deviated from the linear trend.[59]

2.2 The Conconi Test

In 1982, Conconi et al.[9] suggested that this sigmoidal HRPC alone could be used as an alternative method to assess critical performance or work intensities. These investigators recruited well trained distance runners (n = 210) who participated in a running test on an outdoor track. The test began at an initial running speed (RS) of 12 to 14 km/h with an attendant speed increase every 200m (0.5 km/h

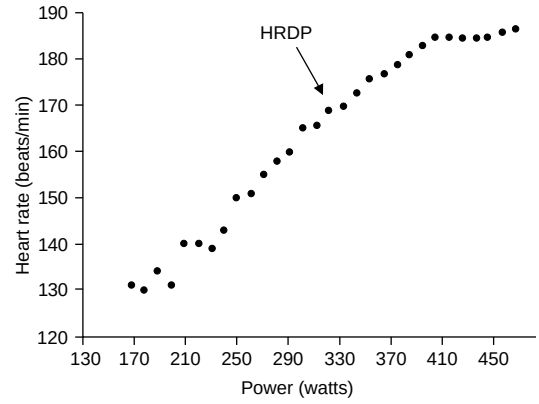

Fig. 1. Heart rate power curve (cycle ergometry) of a 24-year-old volleyball player showing a 'regular' heart rate deflection point (HRDP) and demonstrating a decrease in HR-work slope. The HRDP (arrow) was 169 beats per minute and was assessed using a mathematical model.[18,19,27]

average increase) until further increases were no longer possible. HR was recorded during the last 50m of every segment. A distinct shift in the nature of HR response from a linear to curvilinear trend occurred at near maximal speeds. Conconi et al.[9] originally labelled this loss of linearity between HR and RS the 'deflection velocity'.

The anaerobic threshold was determined using a somewhat unconventional method. Venous blood lactate (B_{LAC}) values from 10 runners were assessed at 3 RS above and below the individualised deflection velocity. Each RS was maintained for 1200m, interspersed with a 15-minute recovery period. Blood samples were extracted 5 minutes after the completion of each run.

These B_{LAC} values were superimposed onto the corresponding HR-RS graph. Straight lines connected the lactate points above and below the deflection velocity, respectively. The RS at the intersection of these lines resolved the anaerobic threshold. Results indicated that the deflection velocity and anaerobic threshold were coincidentally related ($r = 0.99$; $n = 10$).

From these observations, Conconi et al.[9] concluded that the HR-RS relationship could be used to noninvasively measure the anaerobic threshold in runners. They also suggested that this test could be expanded to include other sports, and may be useful for following the training programmes of individual athletes.[9] This field test became known as the 'Conconi test', the popular name for HRDP testing.

2.3 The Conconi Hypothesis

To explain this HR phenomenon, Conconi et al.[9] cited the observations of Pendergast et al.,[60] who demonstrated that above the anaerobic threshold the attendant increase in $\dot{V}O_2$ was smaller than the increase in work intensity. Conconi et al.[9] hypothesised that, in runners, an increase in RS above the anaerobic threshold is partly independent of $\dot{V}O_2$ and HR. They reasoned that if work intensity above the anaerobic threshold increases more than HR, then the deflection in HR could be used to noninvasively and indirectly assess the anaerobic threshold.[9]

3. Methodological Aspects of HRDP Assessment

Subsequent to the work of Conconi et al.,[9] HRDP investigations incorporated various study populations in a variety of sporting activities in both field and laboratory settings. Some of the methods used to calculate HRDP are more objective than others. The protocol of HRDP testing requires a progressive incremental work protocol; however, some of the procedural disparities between independent studies may have added to the controversy surrounding the HRDP concept.[36]

3.1 Methods of HRDP Calculation

Visual inspection is the conventional method for HRDP assessment.[5,10,11,17,28,31,34,41,45] Although it is the most feasible, it is possibly not the most accurate method. Deflections in the HRPC may not always be evident[11,36] and difficulties associated with discerning the deflection point visually may cause an inaccurate HRDP assessment. Ballarin et al.[61] reported significant correlations ($r > 0.94$) between computer-determined methods and visual inspection by experienced observers, and recommended that visual HRDP analysis could be executed by experienced observers.

Mathematical modelling of regular HRDP behaviour has also been administered to assist with HR breakpoint assessment. A third order curvilinear regression equation[3] and logistical function[18,19,27] have been incorporated to describe the regular deflection in the HRPC. The logistical function method utilises the first derivative of the natural log of the HR data points to delineate the HRDP. This method is reported to be both objective and practical, since the HRDP is calculated mathematically without visual cues and may be easily applied using a computer spreadsheet programme.

Regression techniques applied to the HRPC have attempted to make the deflection point more discernible and more objective. These techniques have included simple linear regression,[1,4,33] 2-

compartment linear regression models[3,13,28,35] and monosegmental exponential and bisegmental logarithmic analyses.[34]

The nature of the HRPC must be taken into account if linear regression is used to assess the HRDP.[22] The slope of the regression line will be affected if it includes the initial HR data points previous to the linear segment in the HR-work relationship, which may possibly lead to overestimation of the HRDP.[22]

The most sensitive assessment of the HRPC for the HRDP involves computer-aided regression analysis[14-16,20,22,24-26,62] that takes into consideration the breakpoints [i.e. the aerobic threshold or first lactate turnpoint (LTP_1) and the anaerobic threshold or second lactate turnpoint (LTP_2)] associated with the 3 phases of energy supply.[56] The regression lines used to calculate HRDP are applied between the first lactate turnpoint (LTP_1) and the power output at maximal HR.[14-16,20,22,24-26,62] This method is appropriate for investigational purposes, but not does not have a high degree of practicality.

Validation of HRDP has incorporated the mathematical calculation of the direction and degree of HRDP. This method incorporates a second degree polynomial representation of the HRPC, satisfying the condition of least error squares.[20-24,26,62] Tangents of various points between LTP_1 and maximal power were calculated to give a value called 'factor k_{HR}', which describes the nature of the deflection. Using this configuration, a regular deflection would have a positive k value and an inverse deflection would have a negative k value.

3.2 Anaerobic Threshold

The expression of the anaerobic threshold in the HRDP literature is diverse and has contributed to the challenges of validation. Although some investigators[10,32] utilised the unconventional multistage protocol created by Conconi et al.,[9] most inquiries applied simultaneous protocols. Several HRDP papers incorporate the Skinner and McLellan[56] theory of energy supply during progressive work and refer to the first breakpoint as the aerobic threshold[14,16,62] or the first lactate turnpoint (LTP_1).[20,22,26] The second breakpoint is referred to as the lactate turnpoint[14,16,62] or the second lactate turnpoint (LTP_2).[20,22,26] A fixed blood lactate of 4 mmol/L has also been used to express the anaerobic threshold,[6,12,33,38,41,63] as well as the first lactate breakaway as the lactate threshold.[28] The VT has also been cited in several HRDP investigations.[4,5,11,13,18,19,27,35,36]

3.3 Protocols

3.3.1 Field Testing

HRDP field studies have incorporated running,[9,27,28,34,39] swimming,[10] rowing,[31] cycling,[31,64] canoeing, cross-country skiing, roller-skating, ice-skating, walking[31] and kayaking.[12] With the exception of kayaking (which used increases in HR to monitor work intensity),[15] all field testing simulated the original Conconi et al.[9] methodology wherein the volunteer gradually increased speed after set distances to increase work intensity. The Conconi method of field testing could be considered a 'fixed distance' stage protocol, since each stage is a set distance. Field protocols are summarised in table I.

3.3.2 Laboratory Testing

Although field testing more closely resembles the athlete's natural environment, laboratory testing allows for a more controlled environment to analyse the HRDP. Research has primarily incorporated cycle ergometry,[11,13,14,17-19,22-24,29,33,35,38,41,42,62] wherein the cadences have been either fixed[11,13,16,17] or self-selected.[3,14,18,19,22-25,27,35,37,38,41,62] Rowing ergometry,[33,64] treadmill running[2,4,5,12,38,41] and arm-cranking ergometry[6] have also been included. Unlike field protocols, laboratory protocols are generally characterised by 'fixed time' stages, which increase work in the form of watts, speed or elevation at regular time intervals. An exception to this is the cycle ergometry protocol of Francis et al.,[36] who increased cadence by 5 revolutions every 30 seconds instead of watts. Furthermore, Jones and Doust[45] increased treadmill speed by 0.14 m/sec every 200m instead of at regular time intervals. Laboratory protocols are summarised in table II.

Table I. Field protocols for heart rate deflection point assessment

Study	Activity	Protocol
Vachon et al.[7]	Running	Warm-up for 10 minutes, 10-14 km/h start with 0.5 km/h speed increase every 200m
Stathus & Sucec[8]	Running	7 miles/h start with 0.5 miles/h increase every 200m
Conconi et al.[9]	Running	12-14 km/h start with 0.5 km/min increase
Cellini et al.[10]	Swimming	Speed increase every 50m
Hofmann et al.[15]	Kayaking	Heart rate 130 beats/min start with 5 beats/min increase
Petit et al.[27]	Running	10.3 km/h start with speed increase every 200m
Tokmakidis & Leger[28]	Running	9 km/h start with 1 km/h increase to 14 km/h; 0.5 km/h increase thereafter
Droghetti et al.[31]	Multiple sports	Canoeing and rowing: 2400m distance in rectilinear channel. Water velocity through this distance 36 m/h. Speed increase every 200m Cross-country skiing (asphalt): 1680m distance (5.5% grade) with speed increase every 140m Cross-country skiing (snow): speed increases on frozen lake Cycling: speed increase every 335m in velodrome Ice-skating: 400m track with speed increase every lap Roller-skating: 325m asphalt track with speed increase every lap Walking: 400m track with speed increase every 200m
Tokmakidis & Leger[34]	Running	9 km/h start; pre-recorded audio cassette signaled speed increases
Ballarin et al.[39]	Running	Outdoor: 5-7 km/h with speed increase every 100m Indoor: figure-8 course with slow speed increase
Conconi et al.[64]	Cycling	Velodrome: Warm-up for 15-30 minutes, 28-30 km/h start speed with slight speed increase every lap (average 1.0 km/h) Uphill ascent of 1.8km at 10% constant grade: 12 km/h start speed with slight increase in speed every 150m

3.4 Revisions to the Conconi Protocol

Conconi et al.[65] revised the original HRDP field protocol[9] based on 12 years of practical application. These investigators believed that methodological problems may have contributed to some of the unsuccessful tests reported in the literature. The updated procedures necessitate speed increases in a ramp-like manner, based on time rather than distance.[61,65]

Specifically, this requires an augmentation in workload such that HR does not increase by more than 8 beats/min. In cases where speed is the modality, the point of transition between submaximal and maximal exercise intensity should be characterised by an accelerated rate of increase until volitional exhaustion. This point may be determined intrinsically by the athlete or by the investigator monitoring external signs of impending fatigue. Furthermore, a correlation coefficient of $r \geq 0.98$ for the linear portion of the HR-work relationship is considered a critical component for successful HRDP assessment.[61,65]

3.5 The Protocol as an Explanation for the HRDP

The literature provides evidence that a completely linear HRPC may occur.[1,2,11,16,21,22,33,36,45] Therefore, some researchers are doubtful that the HRDP is a normal physiological occurrence.[11] Ribeiro et al.[11] reported difficulties assessing the HRDP in 50% of their study participants and suggested that the biological origin of the deflection point is somewhat dubious.

The differences between 'fixed time' protocols in laboratory testing and 'fixed distance' stage protocols characteristic of field testing might account for the low incidence of HRDP and scope of HRDP variability in the literature.[2] Fixed time protocols incorporate increases in work intensity at constant time intervals. However, fixed distance protocols incorporate increases in work intensity (usually speed) over set distances.

Fixed distance stage protocols have been targeted as an explanation for the occurrence of a deflection.[66] Since stage distances are constant, any

accretion in exercise intensity must arise from an increase in speed. This effectively decreases the duration of the stage, especially near the end of the test. The time interval of each stage will decrease progressively to the extent that the circulatory system cannot effectively adapt to the increasing workload. This will be physically manifest as a lagging in HR response and visually observed as a deflection in linearity. This decreased continuance of the stage coincident with ineffective cardiovascular adaptation implies that the HRDP may be an artifact of the protocol.[66]

However, the cardiocirculatory adaptation to increases in work intensity are reported to occur within 10 to 20 seconds if the adjustment in speed increments are 0.5 km/h or less.[9,67] Recently, Vachon et al.[7] reported a steady state HR (± 2 beats/min) within 15 to 30 seconds using the revised Conconi protocol. The time lag between the onset of HRDP and the beginning of the final acceleration to complete the test is sufficient to allow for cardiocirculatory adaptation. However, Conconi et al.[67] reported that, in runners, the final acceleration during incremental staging occurs after the HRDP.

Perhaps more convincingly, the HRDP has been observed with fixed stage protocols utilising the cycle ergometer and treadmill in which there is no decrease in time as the stages progress.[2,3,11-16,18,22,25,29,35,41] This contradicts the notion that the HRDP is an artifact result of protocol.

3.5.1 Influences of Protocols on HRDP

Vachon et al.[7] reported that although 100% of study participants (n = 8) demonstrated an HRDP using the Conconi protocol in the field, only 50% (n = 4) showed an HRDP utilising a fixed time treadmill protocol.

The degree of HR deflection is highly dependent on the type of protocol used.[21] Treadmill protocols marked by time-based increases in work intensity ($test_1$ and $test_2$) were compared with treadmill protocols that initiated an acceleration phase in the middle of phase III of energy supply ($test_3$).[56] An acceleration phase that began in between phase I and phase II[56] was designated $test_4$.

In young males (n = 11) with a pronounced HRDP in the time-based tests, the degree of deflection in $test_3$ and $test_4$ expressed by factor k_{HR} was significantly changed, but the HRDP was not (179 ± 10 *vs* 176 ± 8 *vs* 178 ± 12 *vs* 177 ± 10 beats/min for $tests_{1-4}$, respectively).[21] On the other hand, volunteers (n = 7) who expressed a linear HRPC in the time-based tests demonstrated a HRDP in protocols that included acceleration phases in tests 3 and 4 (HRDP 190 ± 11 *vs* 180 ± 10 beats/min, respectively).[21] Pokan et al.[21] concluded that test protocols which incorporated nonlinear time or speed increments allowed for arbitrary designations of the HRDP and were therefore not valid.

4. Validity of the HRDP

As the HRDP is reputed to be a noninvasive method to assess the anaerobic threshold, this implies that physiological variables such as power, speed, HR, B_{LAC} or $\dot{V}O_2$ at the HRDP and the anaerobic threshold should be in a high degree of relationship. The ability of the HRDP to assess the anaerobic threshold appears to be equivocal because of sundry results in the literature.

Conconi et al.[9] originally reported that the RS at the HRDP and speed at the anaerobic threshold were significantly related ($r = 0.99$; $n = 10$). Similar correlations between the power at HRDP and the anaerobic threshold have been reported for rowing ergometry: 253.27 ± 32.74 *vs* 256.82 ± 31.80W, respectively ($r = 0.991$; $n = 11$).[32] Droghetti et al.[31] found coincidence between speed at the HRDP and speed at the anaerobic threshold in cycling, cross-country skiing, roller-skating, walking and rowing.

These findings have been criticised by Tokmakidis and Leger,[28,34] who suggested that the discontinuous multistage lactate threshold protocol implemented by Conconi et al.[9] may have introduced an experimental bias that accounted for the strong relationships between RS at the HRDP and RS at the anaerobic threshold. According to Tokmakidis and Leger,[28] this point will always arrive close to RS at the HRDP.

Table II. Laboratory protocols for heart rate deflection point assessment

Study	Activity	Protocol
de Wit et al.[1]	Cycle ergometry	Constant duration test: power value = 120-130 beats/min start with mathematically calculated stage increase every minute Constant distance test: start = 0.50-1.50 W/kg bodyweight; stage 1 = 2 minutes; stage 2 = 1 minute; stages 3-5 = 50 seconds; stage 6-end = 40 seconds
Jones & Doust[2]	Treadmill	3.33 m/sec start with 0.14 m/sec increase every 200m
Kara et al.[3]	Cycle ergometry	40W start; electronically braked resistance
Mahon & Vaccaro[4]	Treadmill	3 miles/h at 0% inclination with 0.5 miles/h increase until 5-7 miles/h; 2.5% increase in elevation thereafter
Zacharogiannis & Farrally[5]	Treadmill	Predetermined speed at start with 1.0 km/h increase every minute
Schmid et al.[6]	Arm-crank ergometry	Warm-up for 3 minutes at 20W, 20W start with 10 W/min increase. Crank frequency constant between 50-60 revolutions/min
Vachon et al.[7]	Treadmill	10-minute warm-up at 50% heart rate reserve, initial speed 11-12 km/h with 0.5 km/h increase every minute
Ribeiro et al.[11]	Cycle ergometry	30W start with 30 W/min increase; cadence fixed at 70 revolutions/min 25W start with 25 W/min increase
Maffulli et al.[12]	Treadmill	Borg scale 'very light' or 'fairly light' start with 0.083-0.16 m/sec for 1-, 2- and 4-minute stages
Bunc et al.[13]	Cycle ergometry	40W start with 10 W/min increase at 70 revolutions/min
Hofmann et al.[14]	Cycle ergometry	40W start with 10 W/min increase
Hofmann et al.[16]	Cycle ergometry	40W start with 20W increase every 90 seconds; cadence fixed at 70 revolutions/min
Thorlund et al.[17]	Cycle ergometry	60W start with 30W/2 min; cadence at 60 revolutions/min
Bodner et al.[18]	Cycle ergometry	50W start with 30 W/min ramped increase
Bodner et al.[19]	Cycle ergometry	50W start with 30 W/min ramped increase
Pokan et al.[20]	Cycle ergometry	20W start with 10W increase every 90 seconds
Pokan et al.[21]	Treadmill	Test 1: 6 km/h start with 0.6 km/h increase every 60 seconds Test 2: 5.6 km/h start with 0.2 km/h increase every 20 seconds Test 3: 5.6 km/h start with 0.2 km/h increase up until middle of phase III energy supply. Thereafter, 0.3 km/h increase every 20 seconds for first minute, 0.4 km/h increase every 20 seconds for second minute, etc. Test 4: same as test 3 except acceleration starts at beginning of phase II energy supply
Hofmann et al.[22]	Cycle ergometry	40W start with 20 W/min increase
Pokan et al.[23]	Cycle ergometry	40W start with 20W increase every 90 seconds
Hofmann et al.[24]	Cycle ergometry	40W start with 20 W/min increase
Pokan et al.[26]	Cycle ergometry	40W start with 20 W/min increase
Bunc & Heller[29]	Cycle ergometry	PWC minus 40W start with 20 W/min increase
Foster et al.[30]	Cycle ergometry	15W start with 15 W/min increase
Droghetti[32]	Rowing ergometry	Men: 170-200W start with 10-15 W/min increase Women: 150W start with 8-12.5 W/min increase
Kuipers et al.[33]	Cycle ergometry; treadmill	60% predetermined maximal workload with 10 W/min increase; 20-minute warmup; 10 km/h start with 0.5 km/h increase every 30 seconds
Bunc et al.[35]	Treadmill; cycle ergometry	Treadmill: 13 km/h (5% inclination) with 1 km/h increase Cycle ergometer: physical work capacity of 170 beats/min plus 20W start with 20 W/min increase
Francis et al.[36]	Cycle ergometry	50 revolutions/min at 100W start with 5 revolutions/30 seconds increase
Nikolaizik et al.[37]	Cycle ergometry	Males: 50W start with 10 W/increment increase Females: 30W start with 10 W/increment increase Proportional time reduction per stage
Gaisl & Hofmann[38]	Cycle ergometry; treadmill	Cycle ergometer: 10W start with 10 W/min increase; 40W start with 10 W/min increase (females); 60W start with 10 W/min increase (males) Treadmill (5% grade): 7-8 km/h start with speed increase every 200m; 6 km/h start with 0.5 km/h increase every minute

Table II. Contd

Study	Activity	Protocol
Baraldi et al.[40]	Treadmill	6.5 km/h with 2% inclination/min
Gaisl & Weisspeiner[41]	Cycle ergometry	0W start with 10 W/min increase
Rogers et al.[43]	Treadmill	5.6 km/h start with 2% inclination/min
Jones & Doust[45]	Treadmill	3.33 m/sec start at 1% inclination with 0.14 m/sec increase
Pokan et al.[62]	Cycle ergometry	40W start with 20 W/min increase
Bourgois & Vrijens[63]	Rowing ergometry	15-minute standardised warm-up, initial power 120-140W, increase in number of flywheel revolutions/min

PWC = physical work capacity.

Studies involving running, utilising more conventional LT protocols (i.e. 3 or 4 minute continuous stages) conducted separately from HRDP protocols, have produced findings that are generally in opposition to those of Conconi et al.[9] HR values were significantly related (r = 0.85) and not significantly different (HRDP 176.2 ± 10.8 beats/min; HR at LT 175.8 ± 9.3 beats/min); however, Tokmakidis and Leger[28] observed low correlation coefficients (r = 0.50) between RS at the HRDP and RS at LT. Running velocity at HRDP was 13.4% higher than that at LT. These values are almost identical to those of Jones and Doust,[45] who also reported a 13% higher average running velocity at HRDP (5.08 ± 0.25 m/sec) and HR values (186 ± 9 beats/min) than at the lactate turnpoint (4.39 ± 0.2 m/sec and 172 ± 10 beats/min, respectively). RS at the HRDP and LT were moderately related (r = 0.688; n = 7) in the work of Vachon et al.[7] However, the RS at the HRDP was significantly higher than that at LT (17.99 ± 1.51 *vs* 14.92 ± 1.43 km/h, respectively).

However, Petit et al.[27] demonstrated that RS at the HRDP (16.3 ± 2.1 km/h) and VT (16.4 ± 2.3 km/h) were highly related (r = 0.95; n = 11) and not significantly different. Similar results were obtained for HR (HRDP 178 ± 7.7 beats/min; VT 180 ± 9.9 beats/min; r = 0.79; n = 11).

In terms of other activities using conventional LT methodology, power at HRDP (251 ± 21W) was shown to be greater than power at the individual anaerobic threshold (195 ± 31W; $p < 0.01$) and at a fixed blood lactate level of 4 mmol/L (234 ± 31W) in 10 young rowers.[63] Kuipers et al.[33] also showed that power at the HRDP (286 ± 32W) was significantly greater than that at the 4 mmol/L level (250 ± 51W) using cycle ergometry.

There is evidence to support the validity of the HRDP when physiological variables at the HRDP and anaerobic threshold are derived concurrently from the same graded testing procedures. A high degree of relationship has been observed between HRDP and LTP_2, HRDP and lactate turnpoint, and HRDP and VT with such assessments.[11,13,14,16,20,22,25,29,35,38]

Correlation coefficients greater than 0.90 have been observed between power variables at the HRDP and the LTP_2,[11,13,16,22] and the VT[35] utilising cycle ergometry. Hofmann et al.[22] reported significant correlations (r = 0.905; $p < 0.001$) between power at the HRDP and LTP_2, and between HR at the HRDP and LTP_2 (r = 0.889; $p < 0.001$) in 213 healthy young male volunteers utilising cycle ergometry. This finding was inclusive of both regular and inverse deflections. Power values at the HRDP (234.5 ± 69.5W) and the LTP_2 (240 ± 67.1W) were also significantly related (r = 0.92; n = 11) but not significantly different in a group of trained and untrained males.[11] Similar results were reported by Bunc et al.,[35] who showed that power values at the HRDP (224.16 ± 21.68W) and VT (220.00 ± 23.47W) were significantly related (r = 0.938; $p < 0.01$) but not significantly different in 17 untrained young males.

HR values in trained runners (n = 28) at the HRDP (177.0 ± 6.0 beats/min) and at VT (176.0 ± 6.0 beats/min) have been significantly related (r = 0.93; $p < 0.01$). $\dot{V}O_2$ in these runners at the HRDP (4.07 ± 0.32 L/min) and VT (4.04 ± 0.37 L/min) was also highly related (r = 0.916; $p < 0.01$). Speed

variables were also related ($r = 0.902$; $p < 0.01$) and not significantly different.[35] Similar results in 22 untrained females utilising cycle ergometry were reported by Bunc et al.[13] HR at the HRDP (170.8 ± 5.5 beats/min) and HR at VT (168.3 ± 4.8 beats/min) were significantly related ($r = 0.812$; $p < 0.001$) along with $\dot{V}O_2$ values ($r = 0.867$; $p < 0.001$).

However, Zacharogiannis and Farrally[5] reported that velocity, $\dot{V}O_2$ and % of maximum HR values at the HRDP were significantly higher than VT (8.26, 7.2 and 9.5%, respectively) in trained runners ($n = 12$), despite significant correlations between the HRDP and VT for these values.

The dissociation between metabolism and cardiovascular implication is evident in the assessment of the HRDP in patients with cystic fibrosis (CF).[37] Individuals with CF present a unique situation with respect to HRDP. Although their cardiovascular function is normal, oxygen diffusion across the alveolar-capillary interface is compromised. As a result, premature metabolic acidosis may occur because of hypoxaemia associated with poor oxygen diffusion. Results of HRDP assessment showed relatively significant relationships for power between HRDP and LTP_2 ($r = 0.76$; $p < 0.0005$) but the mean power at the HRDP (115.9 ± 29.6W) was significantly higher than at LTP_2 (97.1 ± 33.9W).[37] This study suggests that HRDP-based exercise prescription would result in workloads that are too strenuous for patients with CF.

The disparity of power outputs at the HRDP across varied nutritional states suggests that HRDP validity may be questionable even if threshold assessment is concurrent. Thorlund et al.[17] reported that the HRDP does not provide a stable assessment of LT across normal and glycogen-depleted conditions.[17] These investigators concluded that there is no causal relationship between HRDP and LT. This finding limits the usefulness of HRDP applied to prolonged training or competition because glycogen reserve reduction is inevitable and potentially chronic with such activities.[17] However, Conconi et al.[67] state that the disparity in RS or power across HRDP testing following prolonged training does not disqualify the validity of the HRDP nor its usefulness. Rather, these investigators suggest that HRDP may be used to signal modifications in the nutritional status of the athlete in such instances.

4.1 HRDP and Steady State

Incremental testing contributes to a metabolic state that is continuously in transition.[68] However, the identification of physiological variables at the anaerobic threshold or VT derived from these tests reflects a similar metabolic state that occurs during maximal steady state exercise.[58,69] The validity of the HRDP to assess the anaerobic threshold can be strengthened if it can be expanded to include a high degree of relationship to the MLSS.[57]

Conconi et al.[9] compared RS at the HRDP to average RS during competition and reported significant correlations for the 5000m race [20.15 ± 1.15 *vs* 19.13 ± 1.08 km/h ($r = 0.93$; $n = 19$)], for the marathon [17.4 ± 1.14 *vs* 18.85 ± 1.15 km/h ($r = 0.95$; $n = 55$)] and for the 1-hour race (18.65 ± 0.92 *vs* 18.7 ± 0.98 km/h ($r = 0.99$; $n = 31$)]. Droghetti et al.[31] also reported significant correlations ($r = 0.93$; $n = 11$) between cycling speed at the HRDP and average cycling speed in a 16km simulated race.

Only one study has used HRDP to predict competitive performance. Petit et al.[27] applied an objective mathematical model to Conconi field test results to assess the HRDP in a group of runners. HRDP predicted times and actual times for 10km running performance for 17 runners were highly correlated ($r = 0.98$; $p < 0.01$), although the times were significantly different (mean difference 0.8 minutes; $p < 0.01$). The unseeding of some runners ($n = 5$) was given as a source for this discrepancy due to the large number (20 000) of participants in the race. Seeded runners ($n = 11$), analysed separately, demonstrated a nonsignificant time difference (0.1 ± 0.5 minutes).

According to Hofmann et al.,[14] it is possible that steady state intensities may be derived from traditional, nonsteady HRDP assessments. Time courses of blood lactate levels, $\dot{V}_E$, $\dot{V}O_2$ and surface electromyogram (EMG) of working muscle in untrained female students ($n = 16$) were stable during

successful 20-minute cycle ergometry at workloads equivalent to 10% lower than power at the HRDP.[14] This was not observed at 10% above power at the HRDP where all parameters measured increased continually (with the exception of EMG). Additionally, none of the students were able to complete the 20-minute task. In a reverse manner, $\dot{V}O_2$ and HR values at the HRDP (3.90 ± 0.56 L/min and 168.7 ± 6.9 beats/min, respectively) were shown to be significantly related to, but not significantly different from, those obtained at 10 (3.60 ± 0.58 L/min and 164.3 ± 10.4 beats/min) and 15 minutes (3.71 ± 0.54 L/min and 166.4 ± 11.9 beats/min) during steady state cycling at VT in trained cyclists.[19]

Twenty white water kayakers took part in 70 HRDP investigations that assessed HR and blood lactate parameters during steady state kayaking.[15] HR at the HRDP and steady state HR in 12 cases were strongly correlated ($r = 0.882$; $p < 0.001$) and not significantly different. Fifty-eight tests revealed that HR at the HRDP (170.5 ± 8.3 beats/min) was not significantly different from steady state HR (169.0 ± 8.3 beats/min) and lead to a steady state B_{LAC} response of 4.2 ± 0.9 mmol/L. The steady state loads at a predetermined HRDP lead to steady state lactate values around 4 mmol/L in kayakers under field conditions.[15]

Only 1 runner out of 7 was able to complete a 30-minute run at 0.14 m/s below RS at the HRDP in the study of Jones and Doust.[45] B_{LAC} levels in all study participants increased continually until volitional fatigue (8.1 ± 1.8 mmol/L) and final mean HR was equivalent to 99% of maximal HR. Conversely, all runners were able to complete 30 minutes of running at 0.14 m/s below RS at the lactate turnpoint (final mean blood lactate values 2.4 ± 0.5 mmol/L).

Similar results have been observed with rowing ergometry.[63] Only 4 of 10 young rowers reached a target time of 30 minutes at a power output corresponding to the HRDP. Six rowers were unable to complete the test. Times ranged from 4.92 to 30 minutes with a mean of 17.5 ± 11.1 minutes. B_{LAC} values increased continually for 9 rowers, with only 1 rower meeting the criteria for steady state B_{LAC}.

In a study involving paraplegic athletes (n = 8) and nonparaplegic individuals (n = 8), 6 paraplegic and 5 nonparaplegic individuals were unable to complete a 24-minute prolonged exercise test using arm-cranking ergometry.[6] The test incorporated 3 consecutive 8-minute time periods corresponding to 10W below, power equivalent to, and 10W above power output at the HRDP, respectively.

It is speculated that the cause of the disparities for the validation of HRDP is the accuracy of the method used to calculate HRDP[14] or the differences in the protocols used to assess the anaerobic threshold, in particular the LT.[7]

5. Reproducibility and Reliability

A lack of repeatable deflections has been interpreted to indicate that the HRDP is not reliable.[2] Some studies have documented 100% success discerning the HRDP,[9,33,62] but others report results of 94,[22] 93,[29] 89,[41] 75,[43] 72,[11] 68,[3] 57[37] or 46%,[33] or no demonstrable deflection point.[36] This suggests that the HRDP may not be reproducible across dissimilar populations. Indeed, the evidence for HRDP reproducibility appears to be equivocal. Ribeiro et al.[11] observed that only 50% of study participants demonstrated a HRDP when assessed for reproducibility. However, VT was confirmed in all participants. This finding is comparable with the results of Jones and Doust,[2] who reported that only 40% of their study participants exhibited a clear curvilinear shift in HR during incremental treadmill testing. The 60% remaining showed either no HRDP (26.7%) or displayed a HRDP in only 1 of the tests (33.3%). Similar results (45%, 31% and 24%, respectively) were reported by de Wit et al.[1]

A deficiency of repeatable HRDP in the literature has been attributed to differences among the training or fitness status of volunteers.[11] This hypothesis is supported by Ribeiro et al.[11] and Francis et al.,[36] whose study populations were composed primarily of healthy, active but relatively untrained individuals. Lesser trained individuals may lack the volition to finish and may quit prematurely.[2] As a result, the completed test may not be of an appro-

priate length to assess HRDP. However, an HRDP has been observed in heart disease patients[20] whose capacity for exercise is greatly diminished. Furthermore, homogeneous groups of well trained individuals demonstrated test-retest reproducibility disparities.[2]

Not all HRDP investigations have demonstrated disparate reproducible results. Conconi et al.[9] tested 147 runners between 3 and 80 times each and reported strong reproducibility, but unfortunately did not quantify the data. Other investigators[61] have also demonstrated reproducible HRDP in heterogeneous and homogeneous populations using a modified Conconi protocol. Mean HR values of 165.1 and 164.9 beats/min ($r = 0.95$) were observed in 31 runners across 2 testing periods using Conconi methodology. RS values at the HRDP were also related (9.08 and 9.23 miles/hour; $r = 0.88$).[8] Incorporating cycle ergometry, Bodner et al.[18] noted that the HRDP derived by mathematical modelling was reproducible for HR ($r = 0.84$; $p < 0.001$) and power ($r = 0.95$; $p < 0.001$). This relationship was strengthened by the fact that no significant differences were observed across repeated testing.[18] Maffulli et al.[12] also reported that HR deflection was reproducible in trained runners with a test-retest correlation of $r = 0.97$. The HR deflection point was reproducible if the length of the protocol stage was 1, 2 or 4 minutes.

Although repeated testing may result in some variance in the regression slopes of the HR-workload response, some researchers allege that the convergence of the HR breakpoints across repeated testing is not differentiated.[61] Conversely, the individual metabolic response to graded testing may influence the reliability but not the reproducibility of the HRDP. This infers that the HRDP may be observed across repeated testing (reproducibility) but the values of physiological variables at the HRDP may be significantly different.

Pokan et al.[21] reported that in young male volunteers with a regular HRDP the reliability of the HRDP is not affected by the type of protocol. In cases where protocol remains constant, HRDP reliability appears to be influenced by factors such as glycogen depletion[12,17] and hydration.[12] These observations are supported by Conconi et al.,[65] who suggest that the HRDP-anaerobic threshold relationship may be modified if glycogen levels are disturbed. Variability in the HRDP is demonstrated following a marathon performance.[12,65] However, changes in location of the HRDP are not related to low-test reproducibility, but rather to an alteration in nutritional states.[65] HRDP testing should be conditional upon training status and basal diet to minimise reproducibility difficulties.[12]

The cornerstone of reliability of the HRDP is the objective calculation of HR deflection. Validation becomes problematic if assessments of HRDP are unreliable. The ability to determine HRDP is the weakest aspect of HRDP investigation.[14] Although visual analysis is a conventional method, it is prone to subjective interpretation which may lead to errant or diverse HRDP values. On the other hand, mathematical modelling of HR behaviour provides an objective option for HRDP determination.[27]

6. Physiological Basis of the HRDP

The physiological mechanisms that govern the HRDP phenomenon have not been fully elucidated. However, intrinsic myocardial function, the influences of catecholamines and neural systems, and potassium have been suggested as possibilities.

6.1 Myocardial Function

The HRDP may not represent a physiological substrate in as much as it may represent a coupling of cardiovascular control mechanisms.[70] Pokan et al.[23] produced the first physiological rationale for slope changes in the HRPC during incremental exercise which suggested a relationship between myocardial function and the HRDP.[23] Myocardial function was expressed as left ventricular ejection fraction (LVEF), assessed by radionuclide ventricular scintigraphy. Twelve of 15 study participants demonstrated a regular HRDP or nearly linear HRPC. The other 3 exhibited an increase in slope of the HRPC at higher work intensities. LVEF characteristically reached the highest values before maximal workloads were attained, but indicated an inflection or

breakpoint that coincided with the HRDP ($r = -0.673$; $p < 0.01$). Patterns where the LVEF increased slightly or levelled off after the breakaway were interpreted as augmented myocardial function characteristic of a regular HRDP. Linear HRPCs that demonstrated a marked decrease in LVEF at higher levels of exercise were interpreted as being related to a decreased stress-dependent myocardial function.

Age differences may be related to different LVEF behaviour. The findings of Hofmann et al.[25] showed that older males (aged 50 ± 10 years) expressed greater decreases in LVEF and greater increases in end systolic volume beyond the LT than younger males (aged 23 ± 2 years) utilising similar incremental cycle ergometry.[25] Younger males displayed a regular HRDP, whereas older males demonstrated an inverse response. Cardiac output increased throughout testing in both groups, suggesting that increased HR frequency may be necessary to compensate for possible age-related losses in myocardial function.

Age may be a potential factor in the difference in direction of deflection. However, Hofmann et al.[22] observed a completely linear response in 6.2% and an inverse deflection in 7.9% of healthy young volunteers. Additionally, Bunc and Heller[29] observed a HRDP in the majority of middle-aged men (aged 51.8 ± 5.4 years). Physiologically, this limitation in LVEF and HR responses may be related to differentiated catecholamine sensitivity of the myocardium.[16]

The majority of patients (89%) who experienced posterior wall myocardial infarctions have displayed an inverse HRDP in the HRPC.[20] In these cases, the LVEF significantly decreased between LTP_2 and maximal power. A decrease in LVEF > 5% after the LTP_2 resulted in an inverse HRDP. In this study, the degree and direction of the HRPC appeared to be dependent on left ventricular function during incremental exercise. Foster et al.[30] suggested that the inverse HRDP characterised in patients with stable coronary heart disease may be a compensatory mechanism to maintain cardiac output in cases of left ventricular dysfunction.

Hofmann et al.[16] noted different LVEF breakpoint (LVEFBP) responses in individuals with and without an HRDP. The decrease in LVEF at LVEFBP was more conspicuous in those without HR deflection. In individuals (n = 8) with a regular HRDP, HR and power at HRDP were significantly related to HR and power at LVEFBP ($r = 0.628$ and $r = 0.884$; $p < 0.001$, respectively). Additionally, there was a significant correlation between power ($r = 0.878$; $p < 0.001$) and HR ($r = 0.690$; $p < 0.05$) at LTP_2 (194.2 ± 32.7W and 163.5 ± 5.8 beats/min, respectively) and those values at the LVEFBP (182.6 ± 31.7W and 157.2 ± 11.0 beats/min, respectively). To explain the relationship of the HRDP to the LTP_2, these investigators hypothesised that the augmentation of glycolytic mechanisms culpable for HR deflection are related to the intrinsic function of the heart. A limited cardiac output because of diminution of myocardial function above the anaerobic threshold may contribute to a retarded $\dot{V}O_2$.[16] This reduction in $\dot{V}O_2$ demands that glycolytic pathways must be strongly activated to meet the energy needs for increased power production during the latter stages of heavy incremental work.

6.2 Catecholamine, Neural and Potassium Influences

It is reasonable to hypothesise that catecholamines may be a constituent in the mechanism(s) of HR deflection, since they contribute to the tachycardic response during exercise.[62] However, time courses of plasma adrenaline (epinephrine) and noradrenaline (norepinephrine) levels juxtaposed with both regular and inverse HRDPs during cycle ergometry in 21 individuals displayed no significant relationship. At the same time, a significant relationship was noted between the time course of plasma adrenaline levels and blood lactate levels ($r = 0.723$; $p < 0.005$). Plasma catecholamine levels appear to be independent of HR deflection behaviour.

Hofmann et al.[24] observed slight modulations in the deflection of the HRPC in individuals under the influence of parasympathetic blockade. These investigators concluded that it was unlikely that parasympathetic regulation was a cogent explana-

tion for the HRDP. The results of this study may be supported by recent evidence demonstrating that exercise intensities corresponding to 50 to 60% of maximal $\dot{V}O_2$ are devoid of vagal influences on HR.[71] Cardioacceleration beyond this point is mediated completely by the sympathetic drive.

However, recent findings by Pokan et al.[26] indicated that the parasympathetic drive does influence the HRDP in the HRPC. Twenty individuals under the influence of parasympathetic blockade demonstrated lower values of factor k_{HR} (representative of a more linear HRPC or inverse HRDP) compared with a placebo trial in the same group. The effects of parasympathetic blockade were as follows: individuals who normally had a regular HRDP demonstrated a more linear HRPC; inverse HRDPs were associated with individuals who demonstrated linear HRPCs; and a greater upward swing in the HRPC was seen in individuals who normally responded with inverse HRDPs.

The degree and deflection of the HRPC (k_{HR}) appeared to have no significant relationship to the changes in pH and lactate levels between the LTP_2 and maximal power during incremental cycling in 17 male volunteers.[72] However, a nonsignificant relationship ($r = –0.328$; $p = 0.051$) between k_{HR} and the change in potassium levels between LTP_2 and maximal power suggests that potassium might play a role in the individual HR response during incremental cycle ergometry.[72]

7. Applications

For training regulation purposes, the HRDP possibly provides a noninvasive marker of exercise intensities related to the anaerobic threshold. From a practical perspective, this allows a cheap and easy way to adapt individualised training intensities to athletes.[31,32] HR and work intensities related to the HRDP appear to be modified by training, detraining and illness,[39] as well as glycogen depletion.[12,17] This suggests that HRDP may indicate changes in training status over time and the relative effectiveness of training programmes implemented. Studies have shown that where speed variables can be obtained, the HRDP can be used to predict performance in endurance events.[9,38,42]

The findings of Hofmann et al.[12] in white water kayakers suggested that the HR at the HDRP represented an intensity corresponding to the MLSS. They reasoned that HR at HRDP could be used to regulate endurance training, assuming that the lactate steady state represents the optimal intensity to develop endurance performance. However, Hofmann et al.[22] cautioned that although the HRDP may be useful from a training prescription perspective, universal applicability may not be warranted since the HRDP was not observed in 6.2% of 227 young men.

A significant relationship exists between an inverse HRDP and decreased performance of left ventricular function in patients following myocardial infarction. From a clinical perspective, the HRDP may provide a useful, noninvasive signal to set parameters on myocardial loading for cardiac rehabilitation programmes.[20] Additionally, the HRDP may help to determine exercise intensities associated with left ventricular dysfunction not detected by the ECG, or workloads that are associated with the risk of untoward episodes.[30]

8. Conclusion

The literature indicates that the HRDP is a significant biological occurrence and not an artifact of the protocol used to derive it. Although the exact physiological mechanisms causing the HRDP remain unclear, the intrinsic myocardial function of the heart is implicated. Controversy surrounding the ability of the HRDP to assess the anaerobic threshold may be attributed to differences in methodology (i.e. methods of HRDP calculation, assessment and definition of the anaerobic threshold). The HRDP appears to be related to the LTP_2, but more research is needed to relate the HRDP and the MLSS. Clinically, the HRDP may be relevant as a noninvasive marker by which to set parameters for cardiac rehabilitation training regulation. Further research is required to determine the exact mechanisms involved with the HRDP as well as the relationship to the MLSS.

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