Haemoglobin, Blood Volume, Cardiac Function, and Aerobic Power

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Abstract/Résumé

Alterations in [Hb], which are mediated through changes in arterial oxygen content, and alterations in BV, which are mediated through changes in cardiac output ($\dot{Q}$), have a significant effect on both $\dot{V}O_2$max and aerobic performance. If BV is held constant, a decrease in [Hb] (anaemia) causes a decrease in $\dot{V}O_2$max and aerobic performance, while an increase in [Hb] (blood doping) causes an increase in $\dot{V}O_2$max and aerobic performance. If [Hb] is held constant, an increase in BV can cause an increase in both $\dot{V}O_2$max and aerobic performance, while a decrease in BV can cause a decrease in both $\dot{V}O_2$max and aerobic performance. In addition, an increase in BV can compensate for moderate reductions in [Hb] through increases in $\dot{Q}$, allowing $\dot{V}O_2$max to remain unchanged or even increase. Also, a large portion of the difference in the enhanced cardiovascular function of endurance athletes is due to their high BV and the resultant enhancement of diastolic function. Hence, optimizing both [Hb] and BV is a very important consideration for endurance performance.

Les variations de la concentration d'hémoglobine [Hb], médiées par les variations du contenu artériel d'oxygène, et les variations du volume sanguine (BV), médiées par les variations du débit cardiaque ($\dot{Q}$), influencent grandement le $\dot{V}O_2$max et la performance aérobie. Si le BV est maintenu, une diminution de [Hb] (anémie) se traduit par une baisse du $\dot{V}O_2$max et de la performance aérobie alors qu'une hausse de [Hb] (dopage sanguin) se traduit par une augmentation du $\dot{V}O_2$max et de la performance aérobie. Si la [Hb] est

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maintenue, un accroissement de BV peut se traduire par une amélioration du $\dot{V}O_2$max et de la performance aérobie alors qu'une réduction de BV peut se traduire par diminution du $\dot{V}O_2$max et de la performance aérobie. De plus, un accroissement de BV peut compenser une faible réduction de la [Hb] grâce à une augmentation de $\dot{Q}$, permettant ainsi au $\dot{V}O_2$max de se maintenir et même de s'améliorer. En outre, une forte proportion de l'amélioration de la fonction cardio-vasculaire chez l'athlète d'endurance est imputable à leur BV élevé qui améliore la fonction diastolique. Ainsi, optimiser [Hb] et BV constitue un atout considérable chez l'athlète d'endurance.

Introduction

Although the interrelationships among haemoglobin concentration ([Hb]), blood volume (BV), cardiac function, and aerobic performance have received considerable attention, their relative influence on $\dot{V}O_2$max and aerobic performance is not entirely clear. $\dot{V}O_2$max is determined by oxygen transport—the product of arterial oxygen content (CaO_2) and cardiac output ($\dot{Q}$). Since $\dot{Q}$ is influenced by BV, and CaO_2 is largely determined by the blood [Hb], manipulation of both BV and [Hb] can markedly affect systemic oxygen transport, thereby having a significant influence on $\dot{V}O_2$max and aerobic performance. Under normal conditions, the oxidative capacity of muscles exceeds that of oxygen transport to the muscles. Therefore, during maximal exercise, oxygen transport is the limiting factor for $\dot{V}O_2$max and aerobic performance. If systemic oxygen transport is increased, additional oxygen is available to the working muscles and the result would likely be an increase in $\dot{V}O_2$max and aerobic performance.

Haemoglobin Concentration and Oxygen Transport

Oxygen is carried both in physical solution and in combination with Hb. The amount of dissolved oxygen is proportional to the partial pressure of oxygen (PO_2) in blood as follows: $PO_2 \times 0.003$ ml oxygen per 100 ml blood. Hence, under normal atmospheric conditions, dissolved oxygen contributes relatively little to the overall transport of oxygen in blood. Therefore, oxygen's combination with Hb accounts for most of the blood's capacity to transport oxygen.

Approximately 45% of the volume of normal blood is red blood cells (RBCs) and this percentage is called the haematocrit (Hct). The RBCs contain Hb, and each gram of Hb, if fully saturated with oxygen, can combine with up to 1.39 ml of oxygen. The CaO_2 depends on the arterial PO_2, the oxygen-carrying capacity of [Hb], and the percent oxyhaemoglobin saturation. It is calculated as: (1.39 ml oxygen/gram Hb) × ([Hb] (grams Hb/100 ml blood)) × (oxyhaemoglobin saturation) + $0.003\ PO_2$. Therefore, the higher the [Hb], the greater the oxygen-carrying capacity of blood. At a constant $\dot{Q}$, this will result in an improved oxygen transport to the working muscles, enabling an increased $\dot{V}O_2$max and aerobic performance.

Sports Anaemia

The mean [Hb] for men is 15.5 g · 100 ml^{-1} blood (range 14.0 to 18.0), and for women it is 13.8 g · 100 ml^{-1} blood (range 12.0 to 16.0). When [Hb] falls below this normal range, it is termed anaemia. The incidence of clinical anaemia is very low in well-nourished athletes; however, a subnormal [Hb] is frequently reported

in this population (Clement and Sawchuk, 1984; Pate, 1983). This condition is termed "sports anaemia" and refers to the [Hb] of an athlete that is below average in the absence of any clinical rationale.

ANAEMIA AND AEROBIC PERFORMANCE

Given the importance of [Hb] in oxygen transport, sports anaemia is of considerable interest to athletes, as indicated in previous reviews (Carlson and Mawdsley, 1986; Clement and Sawchuk, 1984). Patients with pronounced anaemia display a significantly reduced $\dot{V}O_2$max compared to normal healthy individuals (Davies et al., 1973; Sproule et al., 1960), while $\dot{Q}$ is little affected (Celsing et al., 1987). Small reductions in [Hb] are also associated with reductions in both $\dot{V}O_2$max and endurance performance in healthy individuals. Early investigations of blood withdrawal (Freedson, 1981; Horstman et al., 1974; Woodson et al., 1978) revealed that, in healthy individuals, a reduced [Hb] is generally associated with a concomitant decrease in $\dot{V}O_2$max and/or endurance performance. Whereas others have observed that oxygen transport can be maintained despite a decreased [Hb] via concomitant increases in SV and $\dot{Q}$, allowing $\dot{V}O_2$max to remain unchanged (Horstman et al., 1974).

In a series of investigations, Kanstrup and Ekblom (1982, 1984) examined the effects of alterations in [Hb] and BV on aerobic performance. The consensus of these investigations was that total body Hb plays a key role in the determination of $\dot{V}O_2$max and physical performance. A reduction in [Hb] with a maintained or reduced BV will result in concomitant reductions in $\dot{V}O_2$max and endurance performance. Whereas an increase in $\dot{V}O_2$max and endurance performance can be achieved when an elevated [Hb] is associated with an unchanged or increased BV. However, these authors also reported that the reduced CaO_2 as a result of acute PV expansion can be compensated by an increased $\dot{Q}$max, allowing $\dot{V}O_2$max to remain unchanged.

Schneiderman-Walker (1987) examined the effects of a suboptimal [Hb] on physiological and performance capacities in 5 men and 5 women. The participants had blood removed on four occasions, each separated by 48 hours, to bring about a progressive decline (4% each withdrawal) in [Hb] without causing clinical anaemia. The mean total decrease in [Hb] was 1.8 g · 100 ml^{-1}, and this was accompanied by an average drop of 6% in $\dot{V}O_2$max. Endurance performance time was also significantly reduced (~14%). Anaerobic performance was not affected by the reductions in [Hb]. These results highlighted the importance of [Hb] in $\dot{V}O_2$max.

In summary, the majority of investigations on acute and chronic isovolemic anaemia in humans indicate that a reduced [Hb] results in a decreased $\dot{V}O_2$max. As an approximation of the relationship between anaemia and $\dot{V}O_2$max, a reduction in [Hb] of 0.3 g · 100 ml^{-1} corresponds to a fall in $\dot{V}O_2$max of 1% (Gledhill, 1982) (Figure 1), and this would be accompanied by a fall in endurance performance of ~2%. Thus, if the [Hb] of an endurance athlete fell from 15.5 to 14 g · 100 ml^{-1}, concomitant decreases of 5% in $\dot{V}O_2$max and 10% in endurance performance would be expected.

MONITORING AND TREATMENT OF SPORTS ANAEMIA

In athletes, the term sports anaemia is often a misnomer for dilutional anaemia and therefore does not pose a serious problem to aerobic performance. However, true anaemia does exist in some athletes, particularly women, usually as a result of iron

deficiency, which may have a significant effect on aerobic performance. Therefore the monitoring of haematological status in athletes is very important.

Haemoglobin is an iron-containing protein, and its synthesis depends on adequate stores of iron in the bone marrow. Thus, in addition to monitoring [Hb] and Hct, measures of iron related variables are useful for the detection of potential hematologic problems. If only one iron index is to be monitored, serum ferritin is recommended, since there is a good correlation between serum ferritin levels and iron stores in bone marrow. It is recommended that the [Hb] and serum ferritin levels of athletes be monitored every 6 to 12 months (Clement and Sawchuk, 1984). Care must be taken when comparing values for the same athlete measured using different assay kits or different commercial laboratories. It also has been suggested that the use of a single value for the determination of "normalcy" or "iron deficiency" should be avoided in favour of kit-specific and laboratory-specific norms (Ondracka and Gledhill, 1988).

Sports anaemia (if associated with iron deficiency) should be offset by dietary intake of iron and other nutrients involved in the synthesis of [Hb]. When excessive iron intake is needed, such as for high intensity training, dietary iron supplementation may be warranted (Pate, 1983). However, iron depletion does not

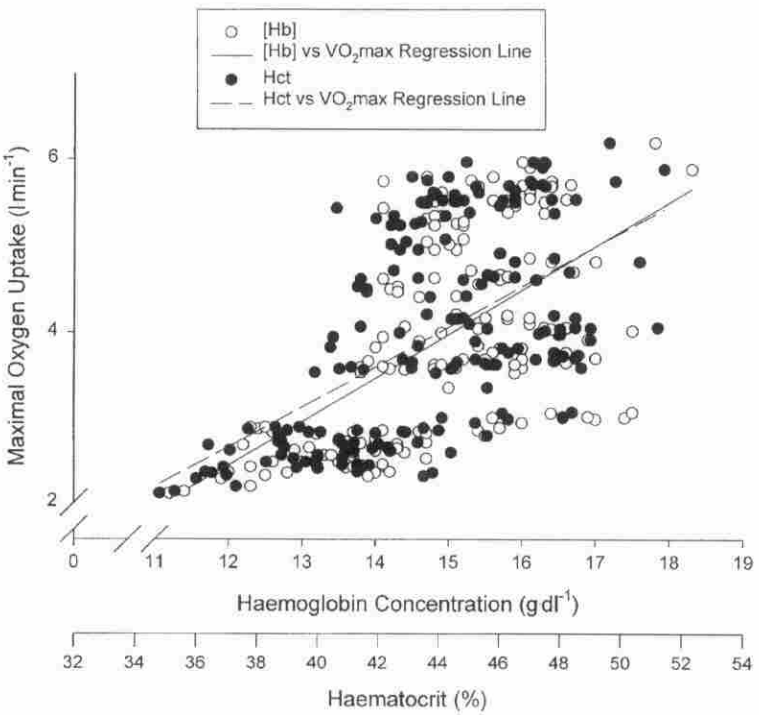

Figure 1. Maximal oxygen uptake as a function of haemoglobin concentration and haematocrit. Values reported are those observed from blood doping and blood removal studies conducted in our laboratory.

necessarily limit erythropoiesis and/or endurance performance unless iron deficiency anaemia exists. Therefore, iron supplementation will only improve performance in athletes with iron deficiency anaemia. In those athletes, it may also be necessary to advocate a modification or reduction in the volume of training (Pate, 1983).

Blood Doping: Induced Erythrocythemia

Blood doping is a term used to describe the procedure of inducing an above normal [Hb] (erythrocythemia). It is generally achieved by removing a volume of blood from an individual, separating and storing the RBCs for some time, then re-infusing the cells into the donor. The underlying theory of blood doping is that while the RBCs are being stored, erythropoiesis is stimulated in the blood donor such that RBCs are produced at an accelerated rate, thereby returning [Hb] to normal levels. Therefore, when the stored RBCs are re-infused, the [Hb] is above normal, resulting in a dramatically increased oxygen-carrying capacity of blood.

Early investigations on this topic were equivocal and reviewers could not support the beneficial effect of blood doping on aerobic performance (Pate, 1976; Williams, 1980). However, early investigators used blood storage techniques that caused substantial RBC loss and therefore were unable to achieve a significant increase in [Hb]. Later investigators avoided this methodological problem and revealed that blood doping resulted in a significant improvement in $\dot{V}O_2$max and endurance performance (Brien and Simon, 1987; Buick et al., 1980; Celsing et al., 1987; Robertson et al., 1982; Spriet et al., 1986; Williams et al., 1981).

METHODOLOGY OF INDUCING ERYTHROCYTHEMIA

It is possible to induce erythrocythemia by transfusing fresh blood from a matched donor (homologous transfusion), as was undertaken by some members of the U.S. cycling team during the 1984 Los Angeles Olympics (Pavelka, 1985). Homologous transfusions are routinely used during the treatment of life-threatening medical conditions. However, such transfusions, even when strict clinical precautions are taken, are associated with significant risks including the development of fever, chills, malaise, and the potential of acquiring fatal infections such as hepatitis B and acquired immune deficiency syndrome.

The removal, storage, and subsequent re-infusion of a subject's own blood (autologous transfusion) avoids the dangers of homologous transfusions. Most stored blood is preserved by refrigeration at 4 °C. However, after the maximal allowable 3-week storage period, only 60% of the RBCs are still viable. When blood is freeze-preserved, it is possible to maximize the recovery of the stored RBC (approx. 85%) and also to delay re-infusion of the cells as long as necessary to ensure that the donor's normal RBC count has been re-established. The [Hb] increases to approximately 10% above control after re-infusion of 900 ml freeze-preserved blood, then progressively declines toward the control level over the next 120 days. This condition of erythrocythemia exists for an extended period of time while the RBC count gradually decreases to the control level (Gledhill, 1982). Therefore, the benefits of the increased oxygen-carrying capacity are not only short-term, but are also sustained for several weeks thereafter.

STUDIES OF BLOOD DOPING

The first investigation of blood doping and aerobic performance in which RBCs were freeze-preserved was reported in a double-blind, sham-controlled, crossover experiment in 1980 (Buick et al., 1980). An important aspect of this investigation was that the researchers examined the effects of blood doping after the subjects' hematologic levels returned to control values. They reported that the re-infusion of 900 ml of autologous, freeze-preserved RBCs in 11 elite endurance runners resulted in significant increases in [Hb] (15.1 to 16.5 g · 100 ml^{-1}, or 9.3%), $\dot{V}O_2$max (5.1%), and treadmill running time to exhaustion (34%). These results conclusively established the ergogenic benefit of blood doping.

A subsequent investigation from the same laboratory (Spriet et al., 1986) examined the effects of re-infusing both 900 ml and 1,350 ml of freeze-preserved autologous blood on 4 elite endurance runners. A primary aim of this investigation was to examine whether there was any impairment in cardiovascular function as a result of induced erythrocythemia. No evidence of cardiovascular compromise as a result of the 900 and 1,350 ml re-infusions was observed throughout light to maximal exercise, despite a significantly increased Hct (7.9 and 10.8%, respectively) and $\dot{V}O_2$max (3.9 and 6.6%, respectively). These results confirmed the findings of the previous investigation and revealed that the effect of blood doping is even greater when the volume of blood is increased. However, during exercise with the highest re-infusion volume, the customary hemoconcentration resulted in a Hct level that approached the clinical diagnostic value of erythrocythemia. Therefore the researchers suggested that the use of re-infusion volumes greater than 1,350 ml should be avoided.

Further support for the ergogenic properties of blood doping has been confirmed by several other investigators (Goforth et al., 1982; Robertson et al., 1982; Thomson et al., 1982; Williams et al., 1981). These authors reported significant improvements in aerobic performance and/or $\dot{V}O_2$max as a result of the re-infusion of 900 ml or more of freeze-preserved autologous blood.

ERYTHROPOIETIN: A NEW FORM OF BLOOD DOPING

Recombinant DNA technology has allowed for the cloning of the gene for erythropoietin, and it has been approved for the treatment of anaemia (Cowart, 1989). Recombinant human erythropoietin (rHuEPO) has been proven effective in the treatment of anaemia in patients with chronic renal failure (Winearls et al., 1986). Unfortunately, rHuEPO could also be employed to improve endurance performance in the same manner as blood doping—by enhancing [Hb] and thereby oxygen transport. Hence, the use of rHuEPO has the potential to replace blood doping by a series of injections.

Recent investigations have revealed that rHuEPO administration improves aerobic capacity (Ekblom, 1996; Rosenlof et al., 1989) and endurance performance (Ekblom, 1996). This results from the stimulation of erythropoiesis (i.e., increased RBC and Hb), which leads to an increased oxygen content. However, the use of rHuEPO is also associated with potentially serious complications including hypertension and thrombolytic or convulsive events. In athletes, the increased Hct may pose more serious problems, since during exercise Hct may be elevated to dangerous levels such that viscosity increases to a point that can cause heart failure. A

series of mysterious deaths in elite European cyclists may have been associated with the misuse of rHuEPO. However, at present it is very difficult to detect rHuEPO use. Thus, the use of rHuEPO not only represents a potential ergogenic aid for endurance events but also may lead to serious cardiovascular complications that may result in death. Therefore the use of rHuEPO represents a major problem for endurance sports.

Role of Blood Volume in the Enhanced Cardiovascular Function of Endurance Athletes

It has long been accepted that BV is important in cardiac function and aerobic performance. Changes in BV affect ventricular preload via the Frank-Starling mechanism and thus influence oxygen transport by altering SV and thereby $\dot{Q}$. Changes in $\dot{Q}$max in turn affect oxygen transport capacity and consequently $\dot{V}O_2$max. Hence, alterations in BV can influence both cardiac function and $\dot{V}O_2$max.

Early investigators of the effects of BV manipulations generally concluded that [Hb], through alterations in total body Hb, plays the dominant role in the influence of BV and [Hb] on $\dot{V}O_2$max and endurance performance (Kanstrup and Ekblom, 1982, 1984). However, recent investigators have shown that $\dot{V}O_2$max can be increased as a result of acute plasma volume expansion in untrained individuals (Coyle et al., 1990; Krip et al., 1997). These findings provided support for the contention that BV plays a greater role in the determination of $\dot{V}O_2$max than was previously thought (Warburton, 1998).

It is commonly accepted that endurance athletes have a significantly larger BV than their non-endurance-trained counterparts. However, the importance of this augmented BV in the athletes' enhanced cardiovascular performance has not received due consideration. An expansion of BV has been shown to increase preload (Coyle et al., 1990; Krip et al., 1997; Warburton et al., 1998) and augment the velocity of the rapid filling phase during diastole (Courtois et al., 1987). Coincident with an expanded BV, endurance athletes possess an increased left ventricular dimension (George et al., 1991), an enhanced myocardial compliance (Levine, 1993), an increased force of atrial contraction, an augmented elastic recoil, and increased negative left ventricular pressure (Courtois et al., 1987; Matsuda et al., 1983). These adaptations all serve to enhance diastolic filling, and therefore may allow athletes to make greater use of the Frank-Starling mechanism during exercise (Gledhill et al., 1994).

MYOCARDIAL CONTRACTILITY VS. THE FRANK-STARLING MECHANISM

There is considerable debate as to which factor, myocardial contractility (enhanced sympathetic stimulation) or the Frank-Starling effect, has the greatest influence on cardiovascular function during incremental exercise. For instance, several researchers have postulated that at low and moderate exercise intensities, the Frank-Starling mechanism is mainly responsible for increasing SV and $\dot{Q}$ (Ginzton et al., 1989; Plotnick et al., 1986). This occurs due to an increased end-diastolic volume (EDV) and therefore an increased SV, due to the Frank-Starling effect. However, at higher levels of exercise (approx. 40% of $\dot{V}O_2$max), the time available for diastolic filling is reduced, thus SV is thought to reach a plateau. It was therefore hypothesized that tachycardia and myocardial contractility have more effect on

increasing $\dot{Q}$ than the Frank-Starling mechanism during the later stages of vigorous exercise (Ginzton et al., 1989; Plotnick et al., 1986).

However, recent evidence (Gledhill et al., 1994; Krip et al., 1997; Warburton et al., 1998) and previously overlooked findings (Crawford et al., 1985; Rerych et al., 1980; Spriet et al., 1986) indicate that the SV of endurance athletes may continue to increase throughout incremental exercise to maximum. It was also observed that the major difference in exercise cardiac function between endurance athletes and their non-endurance-trained counterparts is mainly in diastolic filling rather than systolic emptying, which was attributed to an increased use of the Frank-Starling mechanism (Gledhill et al., 1994).

BLOOD VOLUME AND DIASTOLIC FUNCTION

The increased ability to use the Frank-Starling mechanism to augment diastolic function is thought to be mainly due to an enhancement in preload due to the athletes' increased BV (Gledhill et al., 1994; Krip et al., 1997; Warburton et al., 1998) (Figure 2). This contention supports previous investigators who have shown that diastolic function is improved after endurance training (Matsuda et al., 1983) with little or no effect on left ventricular systolic function (Fagard et al., 1987). The enhanced BV allows for more complete filling during exercise, resulting in an increased SV and improved cardiovascular performance.

Krip et al. (1997) examined the relative importance of BV in the enhanced cardiovascular function of endurance athletes in comparison to untrained individuals. The athletes had significantly greater BV (16%), maximal diastolic filling rate (47.4%), maximal ventricular emptying rate (24.6%), maximal SV (31.6%), $\dot{Q}$max (29%), and $\dot{V}O_2$max (54.5%). Following a BV reduction of 500 ml of whole blood in the endurance athletes and a 500-ml BV expansion (using 6% dextran) in

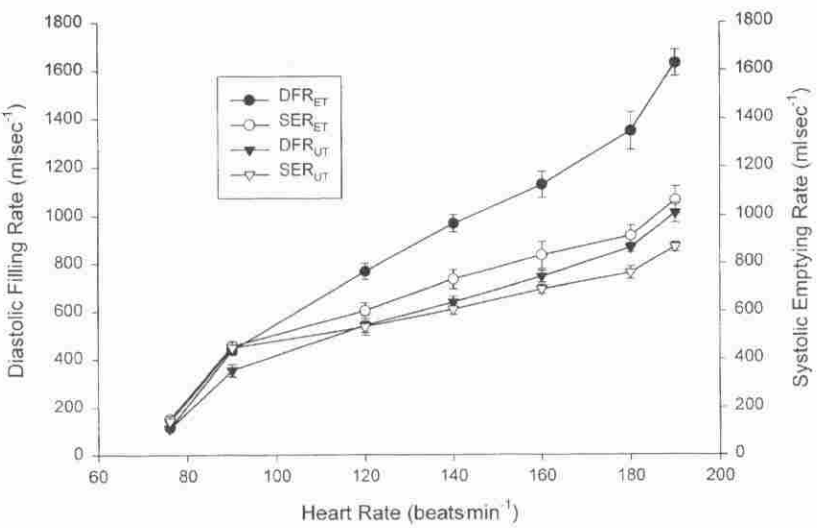

Figure 2. Diastolic filling rate (DFR) and systolic emptying rate (SER) of endurance-trained (ET) and untrained (UT) individuals at preexercise and exercise heart rates.

the untrained participants, there were immediate reversals in the initial cardiovascular differences between groups. For instance, following BV expansion in the untrained participants, maximal diastolic filling rate, maximal SV, $\dot{Q}$max, and $\dot{V}O_2$max all increased significantly (22.5, 9.1, 8.9, and 12.7%, respectively). Whereas BV removal in the athletes resulted in significant decreases in maximal diastolic filling rate (27%), maximal SV (14.3%), $\dot{Q}$max (14.7%), and $\dot{V}O_2$max (7%). Concurrently, however, there were no significant changes in the rate of systolic emptying as a result of BV expansion or reduction. Krip et al. concluded that a large portion of the difference in cardiovascular function between trained and untrained individuals is due to the higher BV of endurance athletes mediated through alterations in diastolic function.

Hagberg et al. (1998) also recently reported that an expanded BV plays a large role in the enhanced cardiovascular function and $\dot{V}O_2$max of endurance-trained older men in comparison to age-matched controls. They observed that the cardiovascular differences between the trained and untrained men, in peak SV, peak end diastolic volume, and $\dot{V}O_2$max, were directly related to BV (r = 0.67, 0.45, and 0.70, respectively). They postulated that the expanded BV of the endurance-trained men increased diastolic volume and SV via an increased utilization of the Frank-Starling mechanism, leading to an enhanced $\dot{V}O_2$max.

Based on the above findings, we speculate that even before endurance athletes begin training, they are genetically endowed with a large BV. This high BV results in an enhanced diastolic function, SV max, $\dot{Q}$max, and $\dot{V}O_2$max, which allows them to succeed in endurance events, and success in these events provides the impetus for further endurance training. Endurance training in individuals with a genetically endowed high BV further increases the BV, which enables the development of an even higher SV max, $\dot{Q}$max, and $\dot{V}O_2$max.

However, it also appears that highly trained endurance athletes have a BV that places them at the limits of their diastolic reserve capacity. This is based on research in athletes in which minimal changes were observed in cardiac function and aerobic performance as the result of acute plasma volume expansion (Coyle et al., 1986; Hopper et al., 1988; Warburton et al., 1998). That is, endurance athletes may already be at a BV that places their myocardium near or at the limits of its ability to fill. Any further BV expansion would therefore not augment cardiac function.

Conclusion

Alterations in BV affect $\dot{V}O_2$max via changes in $\dot{Q}$, and alterations in [Hb] influence $\dot{V}O_2$max through changes in CaO_2. Therefore, changes in either variable will have a significant effect on aerobic performance (Table 1). Sports anaemia refers to the presence of a subnormal [Hb] and is associated with reductions in $\dot{V}O_2$max and endurance performance. Blood doping results in significant improvements in oxygen transport, $\dot{V}O_2$max, and endurance performance. A new and potentially dangerous form of blood doping is the use of recombinant erythropoietin.

In general, if [Hb] remains unchanged, an accompanying decrease in BV will generally lead to decreases in $\dot{V}O_2$max and endurance performance. However, an increase in BV can compensate for moderate reductions in [Hb] through increases in $\dot{Q}$, allowing $\dot{V}O_2$max to remain unchanged or even increase. If BV remains unchanged, an accompanying significant decrease in [Hb] will lead to decreases in

Table 1 The Interrelationships Among Total Body Hb (TBHb), Haemoglobin Concentration ([Hb]), Blood Volume (BV), $\dot{V}O_2$max, and Aerobic Performance

Condition	TBHb	[Hb]	BV	$\dot{V}O_2$max	Aerobic perform.
Normovolemic normocythemia	0	0	0	0	0
Hypervolemic anaemia (athletes)	0 or –	–	+	0 or +	0 or + or –
Hypervolemic anaemia (normals)	0 or –	–	+	0 or +	0 or + or –
Normovolemic anaemia	–	–	0	–	–
Hypovolemic anaemia	–	–	–	–	–
Hypervolemic normocythemia (athletes)	+	0	+	0 or +	0 or +
Hypervolemic normocythemia (normals)	+	0	+	+	+
Hypovolemic normocythemia	–	0	–	0 or –	0 or –
Hypervolemic erythrocythemia	+	+	+	+	+
Normovolemic erythrocythemia	+	+	0	+	+
Hypovolemic erythrocythemia	0	+	–	0 or –	0 or –

Note. 0 = no change; – = decreased; + = increased.

$\dot{V}O_2$max and endurance performance, while an increase in [Hb] with no change in BV will produce increases in $\dot{V}O_2$max and endurance performance. It can be concluded, therefore, that [Hb] and BV are both very important in determining $\dot{V}O_2$max and endurance performance. Also, a large portion of the difference in the enhanced cardiovascular function of endurance athletes is due to their high BV.

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