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Review Article

Does Lactic Acid Cause Muscular Fatigue?

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Abstract. *Until recently, lactic acid accumulation in muscles and the acidosis resulting from it were believed to cause muscular fatigue in most athletic events. However, early pieces of research that supported this contention suffered from a flaw in design. They were conducted with excised muscle fibers that had been cooled well below normal body temperature before measurements were taken. More recent studies have demonstrated little or no effect of lactic acid accumulation and acidosis on muscle contraction force and velocity when muscle fibers were warmed near normal body temperature (29, 42, 43). This finding has caused many in the scientific community to doubt that acidosis is the principal cause of muscular fatigue. Some are now suggesting that muscular fatigue is caused by creatine phosphate (CP) depletion and, specifically, the increases of inorganic phosphate (Pi) and ADP that occur when the muscle supply of CP is reduced. Despite these findings, a very strong case for acidosis has been made by Knuth and associates (20). They showed that acidosis resulted in a loss of muscle power even when fibers were warmed to 30^{0}C (86^0 F) before testing. Evidence both for and against acidosis as a cause of muscular fatigue has been presented in this paper so the reader can form his or her own opinion on this controversial issue.*

Key Words: *Acidosis, muscle temperature, Muscle pH.*

Introduction.

When people are asked to define fatigue, they frequently use words like “exhaustion” and “pain”. These terms describe only two of several forms that muscular fatigue can take. Exhaustion refers to the kind of fatigue that occurs in events lasting several hours. That fatigue is caused by dehydration and depletion of carbohydrate sources in muscles. Pain and an inability to maintain a desired level of performance are terms associated with fatigue in events lasting one to several minutes. That type of muscular fatigue is the subject of this paper.

In 1924, Nobel Laureate A.V. Hill (16) and his associates suggested that the cause of fatigue in events lasting one to several minutes was lactic acid accumulation in the muscles. This notion has been

widely accepted in the years since and continues today, with only minor modifications, as the classic explanation for muscular fatigue in most athletic activities. The role of acidosis in muscular fatigue is now being questioned, however, with the report by Pate and co-workers in 1995, that high levels of lactic acid did not interfere with muscular contraction at near-normal body temperatures (29). This finding has led to a re-examination of the role of lactic acid in muscular fatigue.

The purpose of this paper will be to describe the proposed role of lactic acid in muscular fatigue as well as other possible causes of fatigue that have been suggested by recent research so that readers may form their own opinion on this issue.

The Mechanisms of Muscular Fatigue.

Before discussing the suggested role lactic acid plays in muscle fatigue, I should list the different mechanisms that give rise to that fatigue. Muscle fatigue can manifest itself in the following ways during exercise; (1) by a reduction in the rate of ATP regeneration, which, in turn, slows the release of the energy that powers muscle contractions; (2) by a reduction in muscle contraction force; (3) by a reduction in muscle contraction velocity; and (4) by a reduction in muscle contraction rate.

The Acidosis Model of Muscular Fatigue

As mentioned earlier, the Nobel Laureate, A. V. Hill (16) and others proposed that lactic acid accumulated in muscles during exercise producing a condition called **acidosis,** which caused most of the energy producing and contractile mechanisms of muscle to weaken, slow, and, ultimately, fail. The process by which Hill and colleagues believed lactic acid accumulation caused acidosis can be explained as follows.

Very little lactic acid is produced in the muscles at rest and during mild exercise and that which is produced can be removed quite readily so no excess accumulation occurs. However, when the production rate of lactic acid is high, as it is during most athletic contests, the mechanisms for reducing its production and removal will be stressed to the limit causing excess lactic acid to accumulate in the muscles. As lactic acid accumulates in working muscles, their (the muscle's) pH, which is normally slightly alkaline (7.04), will be reduced to some acidic value between 6.9 and 6.4 .

When this happens, acidosis occurs. As acidosis progresses in muscles the rate of ATP regeneration is believed to slowed. In addition, the muscle fibers are thought tolose contraction force, and velocity so that their power output is reduced and athletes are no longer able to maintain their initial level of performance.

Many experts believed the effects of acidosis were progressive because reductions of muscle force and power became more prominent with greater declines in muscle pH until at a certain level, thought to be in the neighborhood of 6.4 in humans, muscles became unable to regenerate ATP and therefore, gain the energy they needed for contraction (37).

Acidosis was thought to reduce the rate of energy release because it inhibited the activity of two rate-limiting enzymes of metabolism, phosphorylase, and phosphofructokinase (PFK) (32). The inhibitory effect of these two enzymes, reduced the rate of energy release from ATP and the rate of ATP regeneration so that athletes were not able to supply energy rapidly enough to maintain their desired level of effort. In addition, acidosis was also thought to interfere with the release of calcium from the sarcoplasmic reticulum of muscle fibers, which, in turn, reduced the amount that could bind with troponin on the actin molecule and initiate contraction. (This process will be described in greater detail later in this paper.) To summarize, acidosis was believed to reduce the contraction rate of muscle fibers by (1) slowing the rate of ATP regeneration, while at the same time, (2) reducing their contraction force by reducing calcium release and the formation of actomyosin.

When acidosis occurred, both of these effects were shown to reduce muscular power in several studies where groups of muscle fibers and single muscle fibers were tested. In addition, through the use of muscle biopsies, increased amounts of lactic acid and subsequent acidosis were reported in the muscles and blood of athletes when they became fatigued, lending support to the belief that acidosis caused muscular fatigue.

Lactic acid formation and removal.

Let me briefly describe how lactic acid is produced and removed in muscles before discussing its role in fatigue. The rate of lactic acid accumulation depends upon the demand placed on ATP to release energy for muscular contraction, and the ability of creatine phosphate and aerobic metabolism to meet that demand. After the first several seconds of exercise the muscle supply of creatine phosphate will be reduced considerably and that source will no longer be viable. If at that time, the demand for ATP continues to exceed the ability of aerobic glycolysis to replace it, anaerobic glycolysis will "take up the slack", increasing its rate and producing pyruvate and NADH in excess of the rate at which they can be absorbed into the mitochondria. Consequently, these byproducts will accumulate in the cytoplasm of the muscle fiber where they will be transformed to lactic acid and NAD^+. The processes of anaerobic glycolysis and aerobic metabolism are illustrated by the drawings in figure 1.

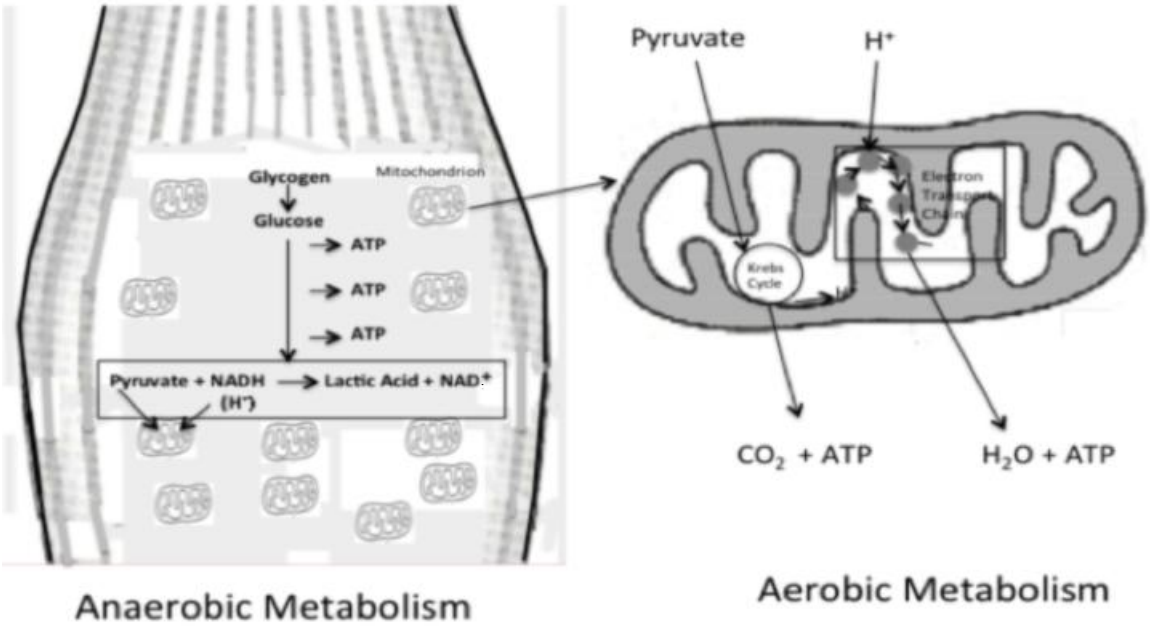

Figure 1. *The drawing on the left depicts the process of anaerobic glycolysis that takes place in the cytoplasm of muscle fibers. During this process, glycogen is metabolized to pyruvate and NADH during which time three (3) molecules of ATP will be regenerated. The pyruvate and NADH can then enter the mitochondria and be oxidized. The drawing on the right shows a mitochondrion where pyruvate, and the hydrogen ions from NADH are metabolized aerobically. Pyruvate enters the mitochondrion and is metabolized to carbon dioxide in Krebs cycle. The hydrogen ions from NADH (and pyruvate) are metabolized to water in the electron transport chain. Thirty-nine (39) ATP molecules can be regenerated by aerobic metabolism. Any pyruvate and NADH that are not absorbed into the mitochondria will be converted to lactic acid and NAD^+ as shown in the drawing of a muscle fiber on the left.*

As mentioned earlier, it happens, that during moderate to high intensity exercise, pyruvate and NADH will be produced faster than they can be transported into the mitochondria and oxidized. As a result, the remaining portions of these substances will combine to form lactic acid and NAD^+ in the cytoplasm of muscle fibers. This occurs when pyruvate takes up a hydrogen ion (H^+) from NADH, and becomes lactic acid while NADH is oxidized to NAD^+. The formation of lactic acid and NAD^+ from pyruvate and NADH is diagrammed in figure 2.

Pyruvate + NADH = Lactic Acid (pyruvate + H^+) + NAD^+

Figure 2. *Lactic acid is formed when NADH donates its H^+ ion to pyruvate. The NADH then becomes NAD^+.*

Once produced, lactic acid can be removed from the muscles by a variety of means. Nevertheless, during intense exercise the rate of lactic acid production will be so high that some will inevitably remain to accumulate in the muscle fibers, and this will lower muscle pH and produce acidosis in the muscles.

In the interest of accuracy, it should be noted that lactic acid is not the direct cause of acidosis. The actual culprit is an increase of the hydrogen ions (H^+) released when lactic acid dissociates to lactate in muscles.

Lactate and lactic acid – how are they related? Lactate is lactic acid that has lost a hydrogen ion (H^+) and gained a sodium (Na^+) or potassium (K^+) ion. This process is illustrated in figure 3.

Lactic acid (pyruvate + H^+) + Na^+ = Lactate (pyruvate + Na^+) + H^+
NADH + H^+ = NAD^+

Figure 3. *Lactic acid must first be converted to lactate before it can leave the working muscles. This occurs when lactic acid gives up its H^+ and the pyruvate remaining combines with Na^+ or K^+. Some of the H^+ ions that are left behind by this process combine with NADH and are oxidized to NAD^+ so they can also enter the mitochondria and be removed from the body as water (H_2O) while others are shuttled into the mitochondria. At the same time, some H^+ ions can also be transported from the muscle fibers in combination with lactate.*

The membranes of muscle fibers will not allow very much lactic acid to pass through or be pumped from them. Consequently, lactic acid must first be converted to lactate and hydrogen ions (H^+) before it can leave the muscles in any significant quantity. As stated, lactate is produced when lactic acid gives up its hydrogen ion and combines with sodium (Na^+) or potassium (K^+). Both the lactate and

some of the hydrogen ions can then diffuse or be transported out of the muscles to the bloodstream. Transportation out of muscle fibers is achieved through the activity of lactate/H^+ transporters. Some of the remaining hydrogen ions can combine with NADH, producing NAD^+, which can enter the mitochondria where the H^+ ions will be oxidized to water (H_2O) by means of the electron transport chain. Hydrogen ions can also be shuttled into the mitochondria of muscle fibers where they will likewise be metabolized to water (H_2O) in the electron transport chain.

Lactate and hydrogen ions accumulate at nearly the same rate during exercise, and, because of this an increase of the former substance in the blood has been used to estimate the magnitude of the latter, and, consequently, the extent of acidosis in the muscles. This is the rationale for the popular practice of blood lactate testing. However, an increase of blood lactate, while it signals an increase of lactic acid production in muscles, can provide only an estimate of the effect of that substance on muscle pH. This is because muscles have several ways of removing lactic acid or weakening its effect on pH during exercise, both before and after it enters the bloodstream. Consequently, the actual extent of H^+ accumulation (pH reduction) in muscles depends on the interplay between four factors; (1) the rate of lactic acid production in the working muscles, (2) the rate at which H^+ ions can be taken into the mitochondria of working muscle fibers, (3) the rate of removal of lactic acid from those same muscles as lactate and H^+, and (4) the buffering ability of those muscles. Obviously, muscle pH will become acidic when the rate of production exceeds the removal and buffering rates, a situation that occurs in all athletic events except for those that are the very short and long.

Does acidosis cause muscular fatigue?

Despite all of the evidence linking lactic acid and subsequent acidosis to muscular fatigue, some recent studies indicate lactic acid may only be associated with muscular fatigue rather than a major cause of it. As mentioned earlier in this paper, it has recently been discovered that earlier studies supporting acidosis as the precipitating cause of muscular fatigue may have suffered from a serous design flaw. They were conducted with excised muscle tissue that had been frozen and then thawed to a temperature of only 15^0 C (59^0 F) before being tested. Now, however, scientists have developed a technique for warming excised muscle tissue near body temperature before testing it. It is well-

known that muscle tissue tends to respond differently to stimuli when it is warm as compared to when it is colder. As a result, several researchers have now reported that the detrimental effects of acidosis on muscle performance are significantly reduced or non-existent in muscle fibers that have been warmed near normal body temperature before being tested.

In several studies acidosis did not markedly reduce muscular force or contraction velocity when the fibers were warmed to (30^0 C (86^0 F) and 32^0C (90^0F) before being tested (5, 29, 43, 46, 50). In one of these studies (43), acidosis caused a 28% decline in peak force for a single fast twitch fiber of mouse muscle at a lower temperature of 12^0C (54^0F) but only a 10% decline at 32^0C. In the same study, contraction velocity was reduced 20% at 12^0C by acidosis while at 32^0C, acidosis did not slow muscle contraction speed at all. These results are summarized in Table 1. As a result of these and similar findings, the belief that lactic acid accumulation and subsequent acidosis cause muscular fatigue is now being questioned. Nevertheless, there are still some researchers who support the acidosis theory.

Table 1. The effect of acidosis on single muscle fiber contraction force and velocity at temperatures of 12^0 and 32^0.

Muscle temperature	Contraction force	Contraction velocity
12^0 C (54^0 F)	**-28%**	**-20%**
32^0 C (90^0 F)	**-10%**	**0%**

Data from: Westerblad, H., J.D. Burton, and H. Lannergren. (1997). The effect of intracellular pH of contractile function in intact, single fibers of mouse muscle declines with increasing temperature. *Journal of Physiology,* 500: 193-204.

One such group is Knuth and associates (20). They set out to determine if acidosis and high muscle temperatures might affect slow and fast twitch muscle fibers differently. Previous studies on warmed muscle had been conducted with only isolated fast twitch muscle fibers. Pate and colleagues (29) used single fibers from the psoas muscle of rabbits, a muscle consisting predominantly of fast twitch fibers, in their study, and Westerblad and colleagues (43) studied the effect of high temperatures on single fast twitch muscle fibers of mice. In other studies, mixed samples of fast and slow twitch muscle fibers were used where the effects on individual fibers types were not reported (49).

It is easy to see that the effect of exercise muscle temperatures and acidosis on mixed samples of muscle tissue could be misleading if the two fiber types differed in their response to either or both of these conditions. This is because a combination of high muscle temperature and acidosis could reduce the contraction force and/or velocity for one type of fiber and this result could be hidden by a compensatory effect on the other fiber type within a mixed group of fibers so that there appeared to be no loss of function. In a like manner, the detrimental effect of acidosis at high muscle temperatures on one particular fiber type, either fast or slow twitch, might not be clearly demonstrated in a mixed group of fibers that contained a preponderance of the other fiber type. Therefore, Knuth and colleagues tested the effect of low pH (6.2) on both the force and contraction velocity of single fast- and slow-twitch muscle fibers at temperatures of both 15^{0}C (59^{0}F) and 30^{0}C. Further, they looked at the effects of acidosis on the contracting power of each fiber type, something that had not been measured in previous studies. As in the other studies, rats were used as subjects. A summary of their results is presented in Table 2. Following is a description of those results

Table 2. The effect of low and high muscle temperatures on the peak force, contraction velocity, peak power, and loaded force and contraction velocity for single slow twitch (ST) and fast twitch (FT) muscle fibers of rats.

	Peak Force		**Contraction Velocity**		**Peak Power**		**Loaded force/velocity relationships**			
							Force		**Velocity**	
Muscle temperature	**ST**	**FT**	**ST**	**FT**	**ST**	**FT**	**ST**	**FT**	**ST**	**FT**
15^0 C (59^{0}F)	**-30%**	**-30%**	**-9%**	**-27%**	**-17%**	**-37%**	**-25%**	**-28%**	**-0%**	**-14%**
30^0 C (86^{0}F)	**-12%**	**-4 to -11%**	**-25%**	**-32%**	**-34%**	**-18%**	**-16%**	**-8%**	**-22%**	**-11%**

Data from: Knuth, S.T., H. Dave, J.R. Peters and R.H. Fitts. (2006). Low cell pH depresses peak power in rat skeletal muscle fibers at both 30^0 C and 15^{0}C: implications for muscle fatigue. *Journal of Physiology,* 575(3): 887-899.

Peak force. At 15^{0}C, acidosis caused a 30% decrease in peak contraction force for both slow- and fast-twitch muscle fibers. These results were in near agreement with those reported previously by Westerblad and associates (-28%), for single fast twitch fibers of mouse muscle at a temperature of 12^{0}C. At a temperature of 30^{0}C, the decline in peak force of fast twitch fibers was also very similar. It was -4% to -11%, in this study versus -10% in the study by Westerblad and colleagues. Knuth and fellow researchers reported a decline in peak force of 12% for slow twitch muscle fibers at a high temperature which was also similar to the decline of 10% reported for fast twitch fibers in the study

by Westerblad and associates. So, where contraction force was concerned the results from this study and previous studies with cold and warm muscle fibers were remarkably similar.

Contraction velocity. Where this measure was concerned, the effect of a low temperature (15^0C), on fast twitch fibers was also similar to that reported by Westerblad and associates for single fast twitch fibers of mouse muscle at 12^0C . Contraction velocity declined 20% in the study by Westerblad, et al., and was -27% in the study by Knuth and colleagues. The effect of low pH on muscle contraction velocity was very different from that reported by Westerblad and associates at higher muscle temperatures, however. Whereas, Westerblad and associates reported no change in contraction velocity for fast twitch muscle fibers at a muscle temperature of 32^0C, Knuth and colleagues found that contraction velocity was 32% slower for this fiber type at a muscle temperature of 30^0C.

In addition, Knuth and colleagues reported that slow twitch muscle fibers contracted 25% more slowly at 30^0C and only 9% slower at a lower temperature of 15^0 C. These results were statistically significant. Obviously, the inhibiting effect of acidosis on the contraction velocity of both fast and slow twitch muscle fibers was increased considerably at a temperature closer to that of the human body during exercise in the study by Knuth and associates than had been reported by researchers in previous studies.

Peak power. In the next part of the study, Knuth and associates combined their results on contraction force and velocity at different muscle temperatures into a measure of peak power, (force x velocity = power). The effect of acidosis on peak power was found to be different for each fiber type although peak power was reduced significantly for both fiber types at a higher vs. lower muscle temperature. The detrimental effect was greater on slow twitch muscle fibers at the higher temperature. Contractile power declined 34% in slow twitch fibers at a pH of 6.2 and a temperature of 30^0 C but only 17% at the same pH but a lower temperature of 15^0C.

The opposite effect was observed with fast twitch fibers. Their peak power declined only 18% at the higher temperature compared to 37% at the lower. It should be noted that the decline in peak power for fast twitch fibers at the higher temperature was more marked than their decline in peak force (-18% for power vs -4% to -11% for force). This indicates that, for fast twitch muscle fibers, a reduction of contraction velocity (-25%) induced by acidosis at a high muscle temperature has a greater detrimental effect on muscle power than does a reduction in force.

The study by Knuth and colleagues, showed that acidosis caused a considerable loss of contractile power in both fiber types at temperatures near those of the human body (-34% loss of power for slow twitch and -18% for fast twitch muscle fibers). As you can see, the loss of power was greater for slow twitch fibers at the high muscle temperature because both their contractile force and velocity were reduced. By contrast, peak power declined less at the higher temperature for fast twitch muscle fibers, although the drop of 18% was still quite significant. Nevertheless, the loss of power in fast twitch fibers was principally caused by a decline in contractile velocity which contradicts the findings of Pate and colleagues (29) and Westerblad and co-workers (43) who reported that contractile velocity did not decline at a muscle temperature of 32^0 C.

Force/velocity relationships. The usual procedure in studies of this type is to measure maximal muscle contraction velocity with no load on the muscle. Although valuable, measuring the velocity of unloaded muscle fibers is not as practical as measuring the effect on force and velocity when muscles are contracting against resistance. For this reason, Knuth and co-workers also measured the effects of three different loads on muscular force and velocity at both high and low temperatures during acidosis.

Slow twitch fibers lost similar amounts of force at 15^0C (-25%) and 30^0C (-16%) when contracting against resistance during acidosis. At the same time, the contraction velocity of slow twitch fibers was not reduced at 15^0C during acidosis while it declined 22% at a temperature of 30^0C. Consequently, slow twitch fibers lost both contraction force (-16%) and velocity (-22%) at high temperatures when working against resistance during acidosis while losing only contraction force

(-25%), at the lower temperature.

Both the force and velocity of fast twitch fibers declined when they worked against resistance during acidosis, regardless of whether the muscle temperature was low or high. The loss of force was considerably less at the higher temperature, however, (-28% at 15^0C vs. -8% at 30^0C). The contraction velocity of fast twitch fibers declined similarly at both low and high temperatures (-14% at 15^0C and -11% at 30^0C). Therefore, fast twitch fibers exhibited declines in force and velocity at both high and low muscle temperatures, although, the effect on force was much less at the higher temperature while the effect on velocity was similar at both low and high temperatures. The smaller loss of force probably accounts for the fact that the power of fast twitch fibers decreased less at the higher temperature, (-18% at 30^0C vs. -37% at 15^0C).

The finding by Knuth and colleagues that acidosis *does* reduce the contractile performance of both fast and slow twitch muscle fibers at near-normal body temperatures stands in marked contrast to the results of those studies where only single fast twitch fibers were used. The effect appears to be more pronounced in slow twitch fibers because they contract slower and with less force. Fast twitch fibers appear to maintain more of their contraction force at high temperatures during acidosis but, like their slow twitch counterparts, contraction velocity declines, causing a smaller, but still significant, loss of power. Obviously, the argument that acidosis *does not* cause muscular fatigue is far from resolved. Nevertheless, as a result of the research of Westerblad and others, many researchers now believe that muscular fatigue is caused by different metabolites and not lactic acid. Let's examine some of the other metabolic products that are now being associated with fatigue to determine their feasibility.

Other possible causes of muscular fatigue.

The recent use of non-invasive NMR techniques has enabled scientists to look at reductions and increases of substances such as ATP, CP, Pi, and H^+, in muscles, immediately following exercise. This research strongly suggests that CP depletion, and muscle calcium changes brought on by increases of inorganic phosphate (Pi) and ADP may be major causes of muscular fatigue. In addition, there is

some support for the notion that high muscle temperatures also cause fatigue. The concentrations at rest and after severe exercise of these and other metabolic products that may be associated with muscular fatigue are listed in Table 3. You can see that ATP, CP, muscle pH, and glycogen decrease during severe exercise while, ADP, AMP, IMP, Pi, and Lactate increase markedly. Since many scientists believe that creatine phosphate (CP) depletion is the leading candidate, it will be discussed first.

Table 3. Metabolite concentrations in human skeletal at rest and following severe exercise in mmols/kg wet tissue.

Metabolites	Rest	Severe fatigue
ATP	**8.2**	**6.2**
ADP	**0.016**	**0.13**
AMP	**0.00008**	**0.002**
IMP	**<0.02**	**1.8**
CP	**32**	**5**
Pi	**3.0**	**28**
Lactate	**2.2**	**26.3**
pH	**7.1**	**6.4**
Glycogen	**175**	**80**

Modified from, M. Houston. (2006). *Biochemistry Primer Exercise Science.* Champaign, IL: Human Kinetics. p. 53 with permission.

Creatine phosphate depletion as a cause of muscular fatigue. You will recall that creatine phosphate is the chemical compound that restores ATP most rapidly during exercise. It does this by donating its phosphate molecule and the energy that binds it to creatine so that ATP can be regenerated from ADP. This reaction is diagrammed in figure 4.

Figure 4. *CP (creatine phosphate) is a chemical compound containing creatine and a phosphate molecule that is bound to the creatine by chemical energy. When CP splits, it releases energy and phosphate that can combine with ADP to regenerate ATP. As indicated earlier, ADP is so-named because it contains two phosphate molecules and needs only one more to become ATP.*

Despite its high rate of use, the muscle's supply of ATP only declines 20% to 40% during exercise so long as there is plenty of creatine phosphate available for regeneration, consequently, depletion of the latter substance, rather than the former, is believed to be associated with muscular fatigue.

Why ATP levels decline only moderately at exhaustion while creatine phosphate levels are greatly depleted is a puzzle. As you can see in Table 3, a muscle fiber's ATP supply was reduced only a small amount (-24%) at severe fatigue while creatine phosphate declines considerably more (-88%).

Although a muscle fiber's CP supply could theoretically be depleted within 6 to 10 seconds of intense exercise, you will notice, in Table 3, that there is still some CP remaining even when athletes are severely fatigued. The muscles apparently defend themselves against complete depletion by slowing their rate of creatine phosphate use after the first few seconds of exercise.

Luckily, the splitting of creatine and phosphate is not the only way the muscles have of releasing energy and regenerating ATP. Other metabolic processes, i.e. anaerobic glycolysis and aerobic glycolysis are also able to perform this function, and, in so doing, prevent the complete depletion of creatine phosphate.

It is a fallacy that all the creatine phosphate in a muscle must be used up before anaerobic glycolysis and aerobic glycolysis begin regenerating ATP. Actually the latter two metabolic processes begin contributing ATP to the effort immediately at the start of exercise. They are slower reacting, however, and require more time to reach their peak rate of ATP regeneration (5 to 6 seconds for anaerobic glycolysis and 1 to 3 minutes for aerobic glycolysis). Consequently, the splitting of creatine phosphate is the major source of ATP regeneration from the first few seconds of exercise until the other two metabolic processes get "up to speed". When that happens, the working muscles' dependence on creatine phosphate will be reduced, allowing some to remain stored in them for later use. However, when the rate of creatine phosphate splitting is reduced and anaerobic and aerobic glycolysis become the major sources for regenerating ATP, exercise will need to proceed at a reduced pace where these slower metabolic processes can keep up with the energy demand. Nevertheless, this is advantageous to the athlete because rapid ATP regeneration would stop if creatine phosphate was completely depleted.

Thus, creatine phosphate declines in two stages during exercise, a fast stage at the start of exercise, followed by a slower phase until the end of exercise. The two phases of CP decline are illustrated by the graph in figure 5. The rapidity of the initial drop and the severity of the final drop are both increased when the intensity of work is increased. Athletes can reduce the rate of creatine phosphate use, by slowing their effort (pacing) in the early stages of a competition so aerobic and anaerobic

metabolism can more nearly match the demand for ATP regeneration. In this way, the muscle's supply of CP will not be so greatly depleted early in the race and more may be available for use later on. On the other hand, if they start a competition too rapidly, the body may protect itself against the shutdown of other metabolic functions, that are necessary to life, by involuntarily, (or, perhaps, voluntarily) reducing muscle contractile force and velocity so that creatine phosphate will not be depleted too rapidly. This is a manifestation of the theory, proposed by Noakes (27), that fatigue is caused by feedback mechanisms in the brain and central nervous system. That theory will be described later in this paper.

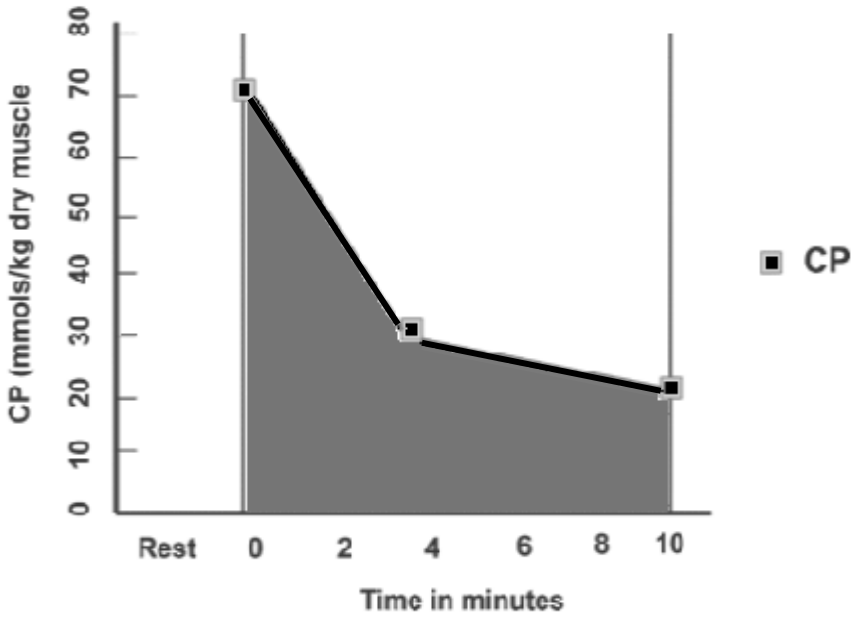

Figure 5. *Creatine phosphate is depleted in two stages during exercise. A rapid stage, from the beginning of exercise until the rates of anaerobic and aerobic metabolism are increased (from 0 to 4 minutes in the figure), and a slower phase until the end of exercise (from 4 to 10 minutes in this figure). Adapted from G.A. Brooks, T.D. Fahey, and K.M. Baldwin. (2005). Exercise Physiology: Human Bioenergetics and Its Applications. Boston, MA: McGraw Hill Book Co., p. 854 with permission.*

One of the reasons **creatine loading** has become so popular is a belief that the rate of creatine phosphate depletion can also be delayed during exercise by increasing the muscles' supply of creatine prior to exercise. A number of studies have suggested this practice will increase muscular strength and performance. The most commonly used procedure for creatine loading is to consume 20

grams daily for 4 to 6 days. The 20 grams are ingested in four doses of 5 grams daily, separated by 4 to 5 hours. This has been shown to increase the CP content of muscles by an average of 11% (19). Athletes with the lowest initial levels of CP tend to store more while trained athletes and those with high initial levels will store less.

Creatine supplementation appears to be most effective for improving performance in events lasting up to 3 minutes"(19). It also seems to improve performance when subsequent efforts are repeated with short recovery periods. Numerous reports from athletes indicate that creatine supplementation also enables them to train harder because they recover faster. In addition, there have been many reports of increased muscle tissue and strength with creatine loading. This effect has not been universally supported, however. Louis and associates (24) found no such effect in their research, while, at the same time, Safdar and colleagues (32) showed that creatine supplementation increased the expression of genes involved in muscle growth. On a negative note, it has been suggested that sustained creatine supplementation will reduce its uptake by muscle cells over time so that the performance enhancing effect of creatine loading will become minimal at best (39).

Increases of Pi and ADP as causes of muscular fatigue.

Other suggested causes of muscular fatigue are increases of inorganic phosphate (Pi) and adenosine diphosphate (ADP) during muscular contraction. ADP is produced, and increases in muscles, when ATP is split for energy during exercise. Both ATP and creatine phosphate (CP) metabolism result in increases of inorganic phosphate (Pi) during exercise. As shown in figure 6, the splitting of ATP and release of its energy results in the formation of ADP (adenosine diphosphate) as well as inorganic phosphate, which probably accounts for the fact that both are associated with muscular fatigue (34). Also diagrammed in figure 6 is the inorganic phosphate that is freed during creatine phosphate metabolism.

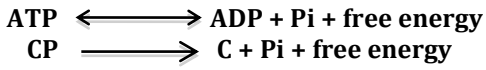

Figure 6. *The splitting of ATP, the release of free energy and the subsequent formation of ADP and Pi. Also diagrammed is the splitting of Creatine Phosphate (CP), and the release of inorganic phosphate and free energy that can be used for regenerating ATP from ADP. The amount of free inorganic phosphate in muscles will also increase when glycogen is used to regenerate ATP. ADP also increases because the rate of ATP regeneration slows as the amount of creatine phosphate in muscles is reduced.*

Both inorganic phosphate and ADP may contribute to fatigue through their effect on calcium release in muscle fibers. Calcium is stored in the sarcoplasmic reticulum of muscle fibers and is essential to muscular contraction because it allows the myosin cross-bridges in myofibrils to attach to the actin filaments and pull them inward so that they shorten, or, to use the proper term, contract. This was a partial description of the very popular sliding filament theory of muscular contraction. This process is pictured and described further in figure 7.

The effects of increased inorganic phosphate (Pi) on muscular fatigue. Contraction force may be reduced when an increase of inorganic phosphate reduces the release of calcium in muscles. This is because a reduction of calcium interferes with the coupling of actin and myosin so that fewer and weaker bonds are formed. When this happens contraction force will be characterized as "weak" rather than "strong". Obviously, when "weak" contractions occur, more fibers will have to contract to maintain a particular level of performance and fatigue will soon follow.

Increases of inorganic phosphate, like those of hydrogen ions, are temperature and fiber type dependent. In one study, an increase of inorganic phosphate reduced muscle contractile force by 54% and 50% in slow and fast twitch muscle fibers respectively at a temperature of 15^0C. This effect was reduced, but still significant in slow twitch muscle fibers (-19%) at a temperature of 30^0 C. The contractile force of fast twitch muscle fibers was much less affected at a high muscle temperature, declining only 5% at 30^0C. Muscle contraction velocity was not reduced by a high level of inorganic phosphate at either low or high temperatures. On the other hand, peak power was reduced 49% for slow twitch and 40% for fast twitch fibers at 15^0 probably because of the considerable loss of contractile force at this muscle temperature. Muscle contractile power was reduced only 16% for slow twitch and 18% for fast twitch fibers at 30^0, however (11). These results were obviously due to smaller reductions of force and no reduction of contractile velocity at the higher muscle temperature. Despite the reduced loss of power at 30^0, it is to be expected that reductions in fast and slow twitch muscle power of 16% and 18% would have a detrimental effect on performance.

Apparently, slow twitch muscle fibers lose a significant amount of their force at near-body temperatures when inorganic phosphate increases, while the loss of force is much less pronounced, but still significant, in fast twitch muscle fibers. This finding has suggested to some, that an increase of inorganic phosphate, and not acidosis, may be the actual cause of muscle fatigue. In the opinions of some, acidosis, may contribute somewhat to muscular fatigue or, it may simply accompany the increase of inorganic phosphate without, itself, contributing to a loss of contraction force or velocity. In support of an enhancing effect for acidosis, the results of one study showed that an increase of hydrogen ions and inorganic phosphate, together, caused muscles to fatigue more rapidly than an increase of Pi alone (28).

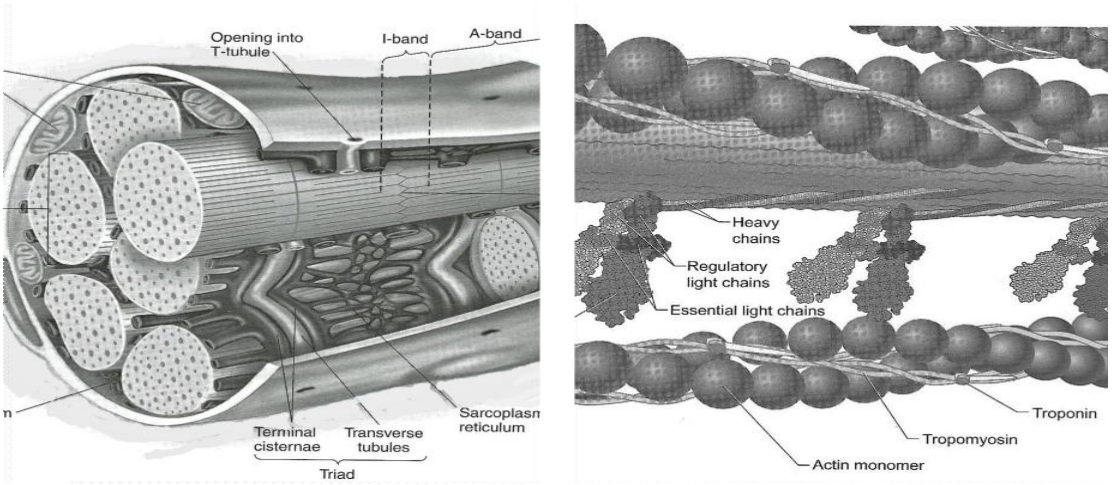

Figure 7. *The drawing on the left depicts a muscle fiber, showing the myofibrils, the sarcoplasmic reticulum where calcium is stored, and the transverse tubules. The drawing on the right shows the arrangement of myosin and actin filaments within a single myofibril. It also shows the cross-bridges from the thicker myosin filaments that allow them to attach to the thinner actin filaments and exert an inward pull that causes the fiber to contract. When a nerve impulse, called an action potential, stimulates a muscle fiber, its charge spreads by way of the transverse tubules to the sarcoplasmic reticulum causing the release of calcium. Calcium spreads to the myofibrils allowing the cross-bridges from myosin to attach to actin and exert an inward pull. Adapted from Wilmore, Costill and Kenney (2008),* ***Physiology of Sport and Exercise.*** *Champaign, IL: Human Kinetics, p. 32, with permission.*

It should be noted that the loss of peak power caused by high levels of inorganic phosphate was less for slow twitch fibers in a study by Debold and co-workers (11) than was the loss resulting from acidosis in the study by Knuth and colleagues that was described earlier (-16% for Pi vs. -34% for

acidosis). It seems, therefore that acidosis may have a greater detrimental effect on slow twitch muscle fatigue at high temperatures than does an increase of inorganic phosphate although both may play a role. On the other hand, acidosis and an increase of inorganic phosphate appeared to contribute equally to reduced power for fast twitch fibers in the studies of Debold and co-workers and Knuth and colleagues. Power declined by 18% in both cases.

Another way an increase of inorganic phosphate can compromise muscle contraction force is by increasing the amount of calcium needed to produce a strong attachment of myosin with actin. Apparently, considerably more calcium is required to initiate contraction when inorganic phosphate accumulates in muscles. With less calcium released, and more needed to maintain a particular level of force when inorganic phosphate increases in muscle fibers, it is easy to see why muscular force could be reduced.

In 2006, Debold and associates (12) measured the calcium needed to produce strong contractions of both the slow twitch (soleus) and fast twitch (gastrocnemius) muscle fibers of rats at both high and low temperatures when inorganic phosphate was increased to an unusually high value of 30 mmols. They reported that high levels of inorganic phosphate did, indeed, reduce contraction force. Slow twitch fibers required more calcium to produce strong contractions regardless of whether the muscle temperature was 15^0 or 30^0. However, they needed twice as much calcium at the higher temperature. By contrast, fast twitch muscle fibers required more calcium only when muscle fiber temperature was increased to 30^0. These results suggest that an increase of Pi will reduce the contractile force of both fibers types at high muscle temperatures.

As an aside to this topic, there is evidence that the stimulant caffeine can reduce the weakening effect of Pi on muscle contraction force. This is because caffeine stimulates calcium release allowing stronger contractions, even when fibers are fatigued (22). Caffeine is almost 100% absorbed into the bloodstream within 5 minutes, and ingestion of 2 to 3 mg of caffeine per kilogram of body weight has been shown to increase muscle power by 7% (21).

The effect of increased ADP on muscular fatigue. An increase of ADP has also been suggested to cause muscle fatigue because it reduces the contraction velocity of muscle fibers. In one study, an increase of ADP decreased the contraction velocity of the toe muscles of rats (44). In another, an increase of ADP reduced the contraction velocity of rabbit psoas muscle by more than 50% at a muscle temperature of 30^0C (8). A reduction of contraction velocity may be particularly detrimental in activities requiring fast movements. This is because a decrease of contraction velocity becomes more important to power output when exercise requires fast movements (41). Therefore, a reduction of contraction velocity will reduce power considerably in activities where speed of movement is a major factor. Consequently, it is easy to see why increases of ADP in muscles might have a detrimental effect on the power they can produce. By the same token, a reduction of contractile force will limit power more than a reduction of contractile velocity when exercise requires the exertion of a great amount of force at slower speeds. As indicated earlier, it is an increase of inorganic phosphate that seems to reduce muscle contraction force. On the other hand, an increased amount of inorganic phosphate seems to have no effect on muscle contraction velocity (7, 44).

One way the body has of reducing the rate of ADP accumulation while, at the same time, increasing the energy supply is by using two ADP molecules to regenerate one molecule of ATP. Two ADP molecules can interact so that the phosphate molecule of one ADP and the energy that binds it to its adenosine molecule will be transferred to the other ADP molecule, leaving one ATP and one AMP (adenosine monophosphate) molecule. This reaction is diagrammed in figure 8. Westerblad and co-workers (46) have suggested that the detrimental effects of acidosis on muscle contraction can be overridden when the rate of ADP accumulation is reduced through this reaction.

ADP + ADP ⟶ **ATP + AMP**
(2 Adenosine + 4 phosphates) **(1 Adenosine + 3 phosphates) + (1 adenosine + 1 phosphate)**

Figure 8. *ATP, can be regenerated when two ADP molecules combine and produce one ATP and one AMP (adenosine monophosphate) molecule.*

ADP accumulation has also been shown to reduce the rapidity of successive muscle contractions by increasing the time it takes for a fiber to relax between those contractions (42). Apparently an increase of ADP slows the time required for calcium to leave the contraction site and return to its place of storage in the sarcoplasmic reticulum. Consequently, it takes longer for the myosin cross-bridges to detach from actin. In one study, the half-time for muscle relaxation following contraction increased from 39 milliseconds to 128 milliseconds when the fibers were fatigued (38). As would be expected, an increase in relaxation time will slow the contraction rate of muscle fibers, which will, in turn, reduce muscle power. It should be mentioned that acidosis has also been shown to increase the time required for muscle fibers to relax between contractions.

Increases of both inorganic phosphate and ADP can also inhibit the activity of enzymes involved in the regeneration of ATP, by slowing their activity, and, thus, reducing the rate of energy release for muscular contraction (36). This has been shown to cause muscle fatigue at low muscle temperatures. Whether this effect is also present at normal or high body temperatures during exercise remains to be seen.

High muscle temperature as a cause of fatigue. Finally, rate at which muscles fatigue may be increased as body temperature increases during exercise. Contractile force will be reduced because more calcium appears to be needed to produce strong myosin/actin bonds as muscle temperature increases beyond normal. As mentioned earlier, the amount of calcium required to effect contraction was increased markedly at 30^0C for both ST and FT muscle fibers compared to the amount required to produce the same contraction force at 15^0C (12). This was particularly true for slow twitch fibers, where the increase was nearly double that required by fast twitch fibers.

Conclusions and implications for training and research.

Lactic acid has been touted as the primary cause of muscular fatigue in most athletic activities for several decades now. This was based on the fact that the hydrogen ions released from it during exercise caused a movement of muscle pH from neutral (higher pH) to acidic (lower pH). Low muscle pH, known as acidosis, was believed to reduce performance by slowing the rate of energy

release and by reducing the force, velocity and rate of muscle contraction. However, early research into this matter may have been flawed because it was conducted with groups of muscle fibers and single fibers that had been frozen and thawed to a temperature that was far colder that of the human body, before being tested.

With improved techniques it has become possible to study the effect of acidosis on fibers that have been warmed to near-normal body temperature. The results of studies where these higher temperatures were used showed that the detrimental effects of pH on muscle performance were reduced considerably or disappeared entirely. This has prompted a re-evaluation of the role of lactic acid in muscular fatigue. At the present time many prominent experts in exercise science no longer believe that acidosis causes muscular fatigue while others continue to assert its importance as a causative factor. Let me present a summary of the cases for and against acidosis using information that was discussed in this paper. The case against acidosis will be summarized first since it has generated the most controversy during the past several years.

The Case Against Acidosis. We have known for decades that lactic acid accumulates rapidly in muscles during hard exercise and that this was accompanied by a reduction of their performance. Consequently, it was quite natural to assume a causative relationship between lactic acid accumulation and muscular fatigue. However, the finding that muscle contraction force and velocity were not greatly reduced by acidosis at higher temperatures has caused many researchers to doubt that lactic acid causes muscular fatigue. They believe the relationship between the two may have been coincidental rather than causative. That is, the accumulation of lactic acid may simply accompany or, perhaps, enhance the effect of other metabolic products that are the primary culprits. Evidence for this belief was presented by Westerblad and co-workers (44) when they were able to fatigue the toe muscles of rats without acidosis occurring.

Even those who think lactic acid plays a minor role in muscular fatigue, believe its effect on muscle pH can be overridden by the action of other metabolic substances. For example, although acidosis is believed to reduce the rate of energy release via anaerobic glycolysis, there is evidence that this

effect can be overridden when two ADP molecules combine to regenerate ATP (33, 46). (See figure 8). Thus, muscles might still be capable of regenerating ATP despite a low pH. Other research that was cited earlier in this paper also indicates the possibility that the effects of low pH can be eliminated, reduced or "overridden" by factors such as increases of muscle temperature and a decrease of creatine phosphate in muscles. This may be particularly true of fast twitch muscle fibers, because a much greater nervous stimulation is needed to activate them and, because their rates of anaerobic metabolism and, thus, their muscle power output are much higher during exercise.

Many who doubt acidosis is a major cause of muscular fatigue, believe the rate of creatine phosphate depletion should get top billing as the actual cause. This may happen because fail-safe mechanisms in the central nervous system and/or the muscles of the body slow muscle contraction speed when they sense the muscle CP supply is being depleted too rapidly. This, in turn, will reduce the rate of ATP splitting so the demand for regeneration can only be met through the slower processes of anaerobic and aerobic metabolism.

Additionally, free inorganic phosphate (Pi) and ADP may act as "governors" reducing muscle contractile force and velocity when they increase in muscles. The accumulation of Pi results from rapid ATP and CP splitting, and may slow the rate of calcium release from the sarcoplasmic reticulum so that muscle force declines. At the same time, an increase of ADP that occurs because of rapid ATP splitting during exercise, may reduce muscle contraction velocity. The final possible cause of muscular fatigue may be muscle temperature. When it increases during exercise, more calcium is needed to produce strong actin/myosin bonds that contract with greater force. On the other hand, when the calcium is not available in adequate quantities, muscle contraction force will be reduced.

Support for creatine phosphate depletion, high body temperature, increases of inorganic phosphate, ADP, and high muscle temperature as causes of muscular fatigue comes from several findings. The fact that muscle contraction velocity is unaffected by acidosis at high temperatures is one of these (5, 29, 43). These results have been refuted by Knuth and associates (20 when they reported that the contraction force, contraction velocity and power output of both slow and fast twitch muscle fibers

was reduced significantly by acidosis. Despite their findings, the possibility exists that some unforeseen factor or factors actually caused the discrepancy between their results and the findings of others, and, suggesting that acidosis is only associated with but does not cause muscle fatigue.

Other arguments against lactic acid come from the results of several studies where the rate of fatigue development in single muscle fibers was not increased when acidosis was exaggerated (5, 50). Also, Lamb and co-workers (23) reported that acidosis did not reduce calcium release in muscle fibers. Sahlin and Ren (35) reported that muscle force was restored within a few minutes of recovery following exercise despite the fact that muscle pH was still below 7.0, thus, suggesting that acidosis was not the primary cause of fatigue.

Further evidence against lactic acid accumulation and subsequent acidosis as the preeminent cause of muscular fatigue comes from the results of several studies where an increase of inorganic phosphate produced reductions in force and power in both slow and fast twitch muscle fibers at high muscle temperatures, that were greater than those produced by acidosis. (11,12). In one of these studies, (12), an increase of inorganic phosphate reduced muscle force by 26% in slow twitch fibers and 18% in fast twitch fibers. By contrast, acidosis reduced muscular force by only 12% in slow twitch and by 4% to 11% in fast twitch fibers in the study by Knuth and colleagues.

Additionally, if the results of different studies can be compared, muscle contraction power, as reflected by a loss of force at different contraction velocities was reduced more by an increase of inorganic phosphate than by acidosis. Power was reduced 38% in slow and 30% in fast twitch fibers when the inorganic phosphate content of those fibers was increased (11). The loss of power was similar to that during acidosis (-34%) in slow twitch muscle fibers. Fast twitch fibers exhibited a much smaller loss of power (-18%) during acidosis than when inorganic phosphate levels were increased, however (20).

Debold and colleagues (12) also reported that an increase of inorganic phosphate reduced muscle power, only, in this case, the cause was a slowing of muscle fiber relaxation time such that fibers were not able to contract as rapidly in succession. They also reported, in 2006, that an increase of

inorganic phosphate increased the amount of calcium needed for muscle contraction to a greater extent at high compared to low muscle temperatures and that this effect was two times greater for fast twitch than it was for slow twitch muscle fibers (12).

Where muscle contraction velocity is concerned, increases of muscle inorganic phosphate have not been found to affect it at either high or low temperatures (14). However, an increase of ADP has been reported to slow contraction velocity considerably more than acidosis at high muscle temperatures, (-50%, (8) vs. -25%, [29]) however.

Whether the differences in these percentage performance decrements indicate greater causation by inorganic phosphate and ADP compared to acidosis remains to be seen. Nevertheless, the possibility must be considered that an increase of one or both may have a more powerful influence on muscular fatigue than does acidosis. We will have to await further research for an answer.

The Case For Acidosis. Perhaps the strongest evidence that acidosis plays a major role in muscular fatigue has been provided by the research of Knuth and colleagues (20). Contradicting the results of other studies that showed little or no effect of acidosis on muscle performance at temperatures near those of the human body, they demonstrated that muscle contractile force, velocity and power *were* reduced by acidosis when muscle temperatures were 30^0C. Where muscle contraction force was concerned their results demonstrated, in agreement with other studies, that it was reduced less at higher, compared to lower temperatures. The magnitude of that reduction differed by fiber type, however. The contraction force of slow twitch fibers was significantly reduced by 12% at a higher temperature compared to a 30% reduction at a lower temperature while, in fast twitch muscle fibers, contractile force was not reduced significantly by acidosis at the higher temperature. The decrease was only 4% to 11% at a muscle temperature of 30^0C versus 30% at a lower temperature (15^0C).

The fact that the deleterious effect of acidosis on the contraction force of muscle fibers did not disappear entirely at higher temperatures in this and earlier research should not be dismissed lightly. Reductions ranging from 4% to 11% (20) and 18% (29) for fast twitch fibers that were reported in two studies might certainly have a detrimental effect on performance when the margin of

victory is often measured in portions of an inch and hundredths of a second. Consequently, acidosis may have a significant detrimental effect on performance even if that effect is not as debilitating as once believed.

While other studies *did not* show that muscle contraction velocity was slowed at temperatures near those of the human body (29, 43), Knuth and associates reported that acidosis definitely had a detrimental effect on this measure. In fact, performance was reduced more for slow twitch muscle fibers at a high compared to a low muscle temperature. The slow twitch muscle fibers that were tested lost 25% of their contraction speed at a muscle temperature of 30^0C and only 9% at a lower temperature (15^0C). Reductions of contractile velocity were considerable for fast twitch fibers at both high and low temperatures although they were slightly, but non-significantly, greater at the higher temperature (-27% at 15^0C versus -32% at 30^0C). Reductions of these magnitudes should be sufficient to reduce muscle performance at exercise body temperatures, therefore, they support the contention that acidosis can cause fatigue. This finding was in stark contrast to the results of Westerblad and associates (43), who found no slowing of fast twitch contraction velocity at higher temperatures during acidosis. They also differed with the result stated by Pate and colleagues (29) who reported a slight, but non-significant, increase of contractile velocity at higher muscle temperatures during acidosis.

Knuth and associates also looked at important aspects of muscle performance that were not included in previous studies. They measured the effect of acidosis on the amount of muscular force at various contraction velocities and they measured the effect of acidosis on muscle power (the product of force and velocity).

Peak power is considered the most discriminating measure of muscle performance because it reflects the ability to do work. Knuth and associates reported that power declined for both fiber types at high temperatures during acidosis, although the effect on fast twitch fibers was less pronounced. The power of fast twitch fibers diminished by 18% at 30^0C versus 37% at 15^0C. At the same time, slow

twitch muscle fibers demonstrated a significantly greater loss of power at the high temperature. They lost 34% of their contraction power at 30^0C versus only 17% at 15^0C.

When the relationship of force exerted at different contraction velocities was compared, the force exerted by slow twitch muscle fibers was reduced by acidosis at each different contraction velocity when the muscle temperature was 30^0C (average of -16%). This was less than the 25% reduction exhibited at a lower temperature but still represents a significant loss of force under loaded conditions. Fast twitch muscle fibers demonstrated less reduction in force at a higher temperature (-8% at 30^0C vs. -28% at 15^0C). Their contraction velocity was reduced similarly at both the higher and lower temperatures, however (-14% at 15^0C vs. -11% at 30^0C). Thus, when loaded, slow twitch muscle fibers lost a considerable amount of both force and velocity at a temperature of 30^0C (-16% for force and -22% for velocity). On the other hand, reductions of both contractile force and velocity were considerably less, but still significant, for fast twitch fibers at the higher temperature (-8% for force and -11% for velocity).

Contrary to the results of others, Knuth and co-workers demonstrated that acidosis had a profoundly detrimental effect on muscle performance when that effect was differentiated according to fiber type. While the effect was more pronounced in slow twitch muscle fibers it was, nevertheless, of consequence in fast twitch fibers. An18% reduction in their muscular power of the latter fiber type at a higher temperature, although less than the 37% reduction at a lower temperature, should have been of sufficient magnitude to cause a reduction in performance.

Why did Knuth and co-workers find that acidosis caused muscular fatigue at high temperatures when the results of other researchers indicated otherwise? Perhaps it was because the studies that demonstrated little if any effect of acidosis on muscle contraction force and velocity were done with groups of mixed muscle fibers where the proportions of fast and slow twitch fibers were not identified, or with single fast twitch fibers whose contraction force and velocity were not hampered so greatly at higher temperatures. As mentioned earlier, in mixed samples of muscle, this may have allowed fast twitch fibers, whose performance did not decline very much, to compensate for the

reduced performance of slow twitch fibers so that muscle performance did not appear to be affected in an adverse manner. At the same time, the profound detrimental effect of acidosis on single slow twitch muscle fibers was missed in studies where only single fast twitch fibers were used.

It was mentioned earlier that, a reduced rate of calcium release, and not acidosis, is thought, by some, to be the cause of muscle fatigue during exercise. However, a compelling bit of support for acidosis as the actual cause is that peak force decreases during acidosis even when the rate of calcium release is high in muscle fibers (26). If an increase of inorganic phosphate were the culprit, we would expect to see an inhibition of calcium release accompanying the loss of muscular force. This suggests that an increase of hydrogen ions will cause fatigue even when the release of inorganic phosphate is high in muscles. Along this same line, the finding that the reduction in calcium release that accompanies an increase of inorganic phosphate, is intensified when it is accompanied by an increase of hydrogen ions, is another point in support of acidosis as a factor in muscular fatigue.

Further support for acidosis as a major cause of muscular fatigue is found in the fact that increased amounts of calcium are required to produce a particular level of force when muscle pH is low (45). Additionally, acidosis has also been demonstrated to cause a slowing of muscle relaxation time at exercise body temperatures (5). In this respect, Westerblad and Lannergren (42) reported muscle fibers did *not* take longer to relax between fatiguing contractions when acidosis was prevented.

In another attempt to substantiate the role of acidosis in muscular fatigue, Bangsbo and colleagues (1) showed that the endurance of leg muscles was reduced if the lactate concentration of the legs was first increased by arm exercise immediately prior to testing the legs. (lactate produced by the arms had been transported to the legs during arm exercise and prior to leg exercise.) Of course, it could be argued that the increase of lactate was coincidental, in that, exercise may have also increased other metabolites that were actually responsible for reducing leg endurance. Nevertheless, this is one more indication that acidosis may cause muscular fatigue.

Finally, there is a theory by Noakes (27) that muscle fatigue is the result of feedback mechanisms residing in the central nervous system. According to this theory, the brain is predisposed to a certain level of distress during a particular exercise through prior exposure and will send out inhibiting messages to the heart, lungs and muscles when that level is exceeded even when it does not pose an immediate danger to life and limb. The brain senses distress from several different sources with a low muscle pH as well as unusually high respiratory and heart rates and high body temperature being four of the more prominent signals. According to this theory, when the central nervous system (the brain and autonomic nervous system) sense unusual bodily distress during exercise, they try to lessen it by reducing (inhibiting) the rate of muscular contraction and, perhaps, by also changing the pattern of muscle activation toward greater recruitment of enduring slow twitch muscle fibers in preference to more rapidly fatiguing fast twitch fibers. The rate of work would necessarily slow down when slower-contracting fibers are substituted for fibers that contract more rapidly and have a greater potential for anaerobic energy release.

Noakes believes that improved performances occur when those inhibiting effects are removed or their severity lessened by "re-programming" the brain, through training, to accept a previously inhibiting level of distress as manageable. In this respect, acidosis may be the most prominent distressing event because of the pain and nausea it produces. Thus, if the onset of distress and/or the severity of inhibiting feedback can be delayed and/or lessened through training that reduces the rate and severity of a decline in muscle pH, athletes should be able to maintain a particular level of effort for a longer period of time.

It is also possible that the effect of acidosis on performance may be mitigated by psychological factors as much or moreso than by those that are physiological in nature. That is, athletes may consciously slow their efforts and their paces as acidosis and other forms of distress become severe, because they lose the motivation to push on, or because they become frightened that they will not be able to complete the event at a respectable level of effort. Thus, although it may be the brain and central nervous system that are sending out distress signals, conditions like acidosis may be generating those signals. Consequently, a reduction in the rate of decline of muscle pH may lessen the severity

of distress signals such that athletes' performances are not diminished to the same extent they might otherwise be if the acidosis were more severe. In the next section we will consider an important question, *"What are the implications of this information for training athletes?".*

Implications for training.

It appears now that muscular fatigue may be more complicated than we once thought. Lactic acid accumulation and the acidosis it produces may not be the only cause of muscular fatigue, nor even the most crippling cause. The rate of creatine phosphate use and the rate of inorganic phosphate and ADP increases during exercise may fatigue muscles even more, whereas, lactic acid accumulation and acidosis may only accompany or, perhaps, enhance their effect. Based on this information, "Have we been training the wrong physiological processes all these years?

For decades, our approach to training has been concerned with improving the physiological mechanisms that reduce lactic acid production and accumulation in muscles. Training methods that were proven to increase cardiac output, VO_{2max}, and buffering capacity were front and center in this effort. Recent additions to this list have been training methods that increase mitochondrial density and lactate transporters. Do our methods require a radical change or only some modifications?

I don't believe a radical change is required. Our training methods have worked even though the reasons for doing so may be different than we once thought. This is probably because fatigue results from the need for high rates of energy release and, therefore, an increase in the rate of anaerobic metabolism that is required to attain those rates. Therefore, training methods, such as those mentioned previously, that have been shown to reduce the demand for anaerobic metabolism, or mitigate its deleterious effects, should continue to be justified even if increases and decreases of certain byproducts of the anaerobic process other than lactic acid prove to be the actual causes of muscular fatigue. That being said, there are some modifications to our traditional training methods that may be in order while some suggestions for future research certainly are.

Suggested training modifications and avenues for future research. It seems reasonable that more emphasis should be placed on training that that has been proven to increase mitochondria, rather than VO_{2max} (although the latter will also increase as a result of that training). It has been well documented that muscles never use all of the oxygen available to them (27). There is more oxygen in the blood stream than can be taken up by the muscles as it (oxygen) passes by them. Thus, some of the oxygen does not diffuse into the muscles because the rate of blood flow is fast and because there is not enough myoglobin to transport it through the cytoplasm and enough mitochondria to accept it. So, the primary factor limiting the rate of aerobic metabolism in muscles may not be the amount of oxygen available to muscle fibers, but, instead, the amount that can be consumed, or *taken up* by them as it passes by in the bloodstream.

Many experts believe that the amount of oxygen that gets taken up by the muscles depends more on the size and number of mitochondria at hand to consume it than any other single factor. Evidence for this belief can be taken from two related studies where VO_{2max} had only a moderate relationship (0.74) with endurance in groups of endurance-trained and untrained rats while the number and size of their mitochondria had a much higher relationship of 0.92 (9,10). As such, a measure of VO_{2max} may not be important in and of itself, but only as an indicator that more chemical factories (mitochondria) are available to take up the oxygen.

Mitochondrial density is the term used to designate the percent volume of a muscle cell occupied by mitochondria. While VO_{2max} is related to a rapid rate of aerobic metabolism, it is probably mitochondrial density that determines that rate. In other words, VO_{2max} may simply be a byproduct of the size and number of mitochondria available to take up oxygen from the circulation. Therefore, when mitochondrial density increases, the oxygen supply to muscles (VO_{2max}) should also increase.

In addition, with more and larger mitochondria, muscles should also be able to metabolize greater amounts of the pyruvate, hydrogen ions, lactic acid, and NAD+ that were formed during exercise. Consequently, an increase in mitochondrial size and number should certainly reduce an athlete's reliance on anaerobic metabolism even if the actual reason for doing so is to reduce the rate of

creatine phosphate use and the rates of increase of inorganic phosphate and ADP rather than reducing the rate of lactic acid accumulation.

Overdistance training has been reported to double mitochondrial density (18), allowing athletes to generate more ATP aerobically during exercise. In one study, endurance training improved the size and number of mitochondria in the muscle fibers of rats by 100%, which resulted in a 400% increase in their running time to exhaustion. At the same time, their VO_{2max} improved only 15%. A control group of rats also increased their VO_{2max} by 15% with sprint training, but they did not increase mitochondria nor did they improve their running endurance. This finding may have occurred because the sprint group improved their VO_{2max} by increasing mitochondria and capillaries in and around fast twitch fibers, more so than they were increased in and around the slow twitch fibers that were doing most of the work during the rodents' runs to exhaustion.

Regarding other physiological adaptations, those methods we now use to increase cardiac output should continue to occupy a "front and center" role in training. A substantial increase of cardiac output will continue to carry more nutrients to the muscles and increase the rate of metabolite removal from them even if reducing the muscle content of certain of those metabolites is more important to maintaining performance than the removal of lactate.

Buffering capacity and the training of monocarboxylate transporters (MCT's) are the questionable processes. Buffering refers to the body's ability to weaken lactic acid so that more hydrogen ions are required to reduce pH by a given amount. MCT's transport lactate and hydrogen ions out of fibers where they are being produced and into resting fibers and organs where they can be metabolized. If acidosis is not a cause of muscular fatigue, neither of these processes may be important to performance. Yet, there is a reasonable amount of scientific evidence that indicates improved performance and increased buffering capacity go hand in hand. (This is an important point in support of acidosis as a major cause of muscular fatigue). Research on the effect of MCT's is relatively new. Training has been shown to increase their activity but little is known concerning the

effect of that increase on performance. Consequently, until we know for sure that acidosis is not involved, it would be wise to continue training to improve both of these physiological mechanisms.

In the meantime, research on reducing the rate of creatine phosphate use during exercise, increasing its rate of restoration after exercise, and the effects of supplementation of this substance on performance, should be accelerated. Research on ways that the rates of accumulation of inorganic phosphate and ADP can be reduced, or mediated, within working muscles during exercise should also become a priority. The possibility that training may also increase their rates of removal from working muscle fibers through either active or passive metabolic procedures is also a topic worthy of study. Likewise, new training techniques that may achieve these effects should also be explored.

We should also consider the possibility that muscular fatigue results from feedback mechanisms that elicit inhibiting responses by the brain and central nervous system. Therefore, coaches and athletes should pay more attention to training at race pace while gently and progressively diminishing possible inhibiting effects, i.e. high heart and breathing rates, loss of CP, increases of ADP and Pi and/or acidosis. This should allow them to increase their pace for the duration of a race. Accomplishing this requires applying the principle of progressive overload with very controlled and specific training sets. There are several methods for applying progressive overload, the most prominent of which involve, (1) swimming progressively more repeats at race pace, (2) swimming at race pace with progressively less rest between repeats and sets of repeats, and, (3) swimming standard sets of repeats at progressively faster average speeds.

Finally, we should not dismiss the role of lactic acid in muscular fatigue as inconsequential. After all, at the present time, acidosis has not been absolutely discredited as a cause of muscular fatigue.

References:

1. Bangsbo, J., K. Madsen, B. Kiens, and E.A. Richter. (1996). Effect of muscle acidity on muscle metabolism and fatigue during intense exercise in man. *Journal of Physiology (London),* 495: 587-596.
2. Bogdanis, G.C. (2009). Fatigue and training status. In C. Williams and S. Ratel (eds). *Human Muscle Fatigue.* New York, NY: Routledge, pp. 164-204.
3. Brooks, G.A., T.D. Fahey, and K.M. Baldwin. (2005). *Exercise Physiology: HumanBioenergetic and Its Application.* New York, NY: McGraw-Hill.
4. Brooks, G.A., H. Dubouchaud, M. Brown, J.P. Sicurello, and C.E. Butz. (1999). Role of mitochondrial lactic dehydrogenase and lactate oxidation in the "intra-cellular lactate shuttle." *Proceedings of the National Academy of Science.* 96: 1129-1134.
5. Bruton, J.D., J. Linnergren, and H. Westerblad. (1998). Effects of CO_2-induced acidification on the fatigue resistance of single mouse muscle fibers at 28^0C. *Journal of Applied Physiology.* 85: 478-483.
6. Coggan, A.R., S.C. Swanson, L.A. Mendenhall, D.L. Habash, and C.I. Kien. (1995). Effect of endurance training on hepatic glycogenolysis and gluconeogenesis during prolonged exercise in men. *American Journal of Physiology.* 268: E375- E383.
7. Cooke, R. and E. Pate. (1985). The effects of ADP and phosphate on the contraction of muscle fibers. *Biophysical Journal,* 48: 789-798.
8. Coupland, M.E., G.J. Pinniger and K.W. Ranatunga. (2005). Endothermic force generation, temperature-jump experiments and effect of increased (MgADP) in rabbit psoas muscle. *Journal of Physiology,* 567(Pt 2): 471-492.
9. Davies, K.J.A., L. Packer and G.A. Brooks. (1981). Biochemical adaptation of mitochondria, muscle, and whole-animal respiration to endurance training. *Archives of Biochemistry and Biophysiology.* 209: 539-554.
10. Davies, K.J.A., L. Packer and G.A. Brooks. (1982). Exercise bioenergetics following sprint training. *Archives of Biochemistry and Biophysiology.* 215: 260-265.

11, Debold, E.P., H. Dave, and R.H. Fitts. (2004). Fiber type and temperature dependence of inorganic phosphate: implications for fatigue. *American Journal Physiology and Cell Physiology,* 287: C673-C681.

12. Debold, E.P., J. Romatowski, and R.H. Fitts. (2006). The depressive effect of Pi on the force calcium relationship in skinned single muscle fibers is temperature dependent. *American Journal of Physiology and Cell Physiology,* 290: C1041- C1050.

13, Donovan, C.M. & M.J. Pagliassotti. 1990. Enhanced efficiency of lactate removal after endurance training. J. Appl. Physiol. 68(3):1053-1058.

14, Fitts, R.H. (2008). The cross-bridge cycle and skeletal muscle fatigue. *Journal of Applied Physiology,* 104: 551-558.

15. Hermansen, L. and J. Osnes. 1972). Blood and muscle pH after maximal exercise in man. *Journal of Applied Physiology,* 32: 304-308.

16. Hill, A.V, C.N.H. Long and H. Lupton. (1924). Muscular exercise, lactic acid, and the supply and utilization of oxygen (Pt. I to VIII). *Procedures Royal Society* (London) Ser. B, 96: 438-475; 97: 84-138; 97: 155-176.

17. Holloszy, J.O. (1975). Adaptation of skeletal muscle to endurance exercise. *Medicine and Science in Sports.* 7(3): 155-164.

18. Hood, D.A. (2001). Contractile activity-induced mitochondrial biogenesis in skeletal muscle. *Journal of Applied Physiology,* 90: 1137-115.

19. Houston, M. (2006). *Biochemistry Primer for Exercise Science.* Champaign, IL: Human Kinetics.

20, Knuth, S.T., H. Dave, J.R. Peters and R.H. Fitts. (2006). Low cell pH depresses peak power in rat skeletal muscle fibers at both 30^0 C and 15^0C: implications for muscle fatigue. *Journal of Physiology,* 575(3): 887-899.

21. Kovaks, E., J. Stegan, and F. Brouns. (1998). Effect of caffeinated drinks on substrate metabolism, caffeine excretion, and performance. *Journal of Applied Physiology,* 85: 709-715.

22. Lannergren J. and H. Westerblad. (1989). Maximum tension and force-velocity properties of fatigued, single Xenopus muscle fibers studied by caffeine and high K^+. *Journal of Physiology (London),* 409: 473- 490.

23. Lamb, G.D., E. Recupero, and D.G. Stephenson. (1992). Effect of myoplasmic pH on excitation-contraction coupling in skeletal muscle fibers of the toad. *Journal of Physiology (London),* 409: 211 - 224.

24. Louis, M., J.R. Poortmans, M. Franceaux, E. Hultman, J. Berre, N. Boisseau, V.R. Young, K. Smith, J.A. Babraj, T. Waddell, and M.J. Rennie. (2003). No effect of creatine supplementation on human myofibrillar and sarcoplasmic protein synthesis after resistance exercise. *American Journal of Physiology,* 285: E1089 - E1094.

25. Martyn, D.A. and A.M. Gordon. (1992). Force and stiffness in glycerinated rabbit psoas fibers. Effects of calcium and elevated phosphate. *Journal of General Physiology,* 99: 795-816.

26. Metzger, J.M., and R.L. Moss. (1987). Greater hydrogen ion-induced depression of tension and velocity in skinned single fibres of rat fast and slow muscles. *Journal of Physiology.* 393: 727-742.

27. Noakes, T.D. (2000). Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. *Scandanavian Journal of Medicine and Science in Sports,* 10(3): 123-145.

28. Nosek, T.M., K.Y. Fender, and R.E. Godt. (1987). It is diprotenated inorganic phosphate that depresses force in skinned skeletal muscle fibers. *Science,* 236: 191-193.

29. Pate, E., M. Bhimani, K. Franks-Skiba, and R. Cooke. (1995). Reduced effect of pH on skinned rabbit psoas muscle mechanics at high temperatures: implications for fatigue. *Journal of Applied Physiology,* 486: 689-694.

30. Pilegaard, H., K. Domino, T. Noland, C. Jeul, Y. Hellsten, A.P. Halestrap, and J. Bangsbo. (1999). Effect of high-intensity exercise training on lactate/H^+ transport capacity in human skeletal muscle. *American Journal of Physiology,* 276: E255-E261.

31. Rasmussen, H.N., G. van Hall, and U.F. Rasmussen. (2002). Lactate dehydrogenae is not a mitochondrial enzyme in human and mouse vastus lateralis muscle. *Journal of Applied Physiology,* 541: 575-580.

32. Safdar, A., N.J Yardley, R. Snow, S. Melov, and M.A. Tarnopolsky. (2008). Global and targeted gene expression and protein content in skeletal muscle of young men following short-term creatine monohydrate supplementation. *Physiological Genomics,* 32: 219 – 228.

33. Sahlin, K. (1986). Muscle fatigue and lactic acid accumulation. *Acta Physiologica Scandanavica* (Supplement), 556: 83–91.

34. Sahlin, K. (2009). Metabolic factors in fatigue. In M. Hargreaves and L. Spriet (eds.). *Exercise Metabolism.* Champaign, IL.: Human Kinetics, pp. 163-186.

35. Sahlin, K. and J.M. Ren. 1989). Relationship of contraction capacity to metabolic changes during recovery from a fatiguing contraction. *Journal of Applied Physiology,* 67: 648-654

36. Sahlin, K., M. Tonkonogi, and K. Soderlund. (1998). Energy supply and muscle fatigue in humans. *Acta Physiologica Scandanavica,* 162: 261-266.

37. Sahlin, K., R.C. Harris, B. Nylind, and E. Hultman. (1976). Lactate content and pH in muscle samples obtained after dynamic exercise. *Pflugers Archives,* 367: 143 - 149.

38. Sjoholm, H. K. Sahlin, . Edstrom, and E. Hultman. (1983). Quantatative estimation of anaerobic and oxidative energy metabolism and contraction characteristics in intact human skeletal muscle in response to electrical stimulation. *Clinical Physiology,* 3: 227-239

39. Snow, R.J. and R.M. Murphy. (2003). Factors influencing creatine loading into human skeletal muscle. *Exercise and Sport Sciences Reviews.* 31: 154-158.

40. Spriet, L.L. and M. Hargreaves. (2006). Overview of exercise metabolism. In M. Hargreaves and L. Spriet (eds.). *Exercise Metabolism.* Champaign, IL.: Human Kinetics, pp. 1-6

41. Westerblad, H., and D.G. Allen. (2009). Cellular mechanisms of muscular fatigue. In C. Williams and S. Ratel (eds). *Human Muscle Fatigue.* New York, NY: Routledge, pp. 48-75.

42. Westerblad, H. and J. Lannergren. (1991). Slowing of relaxation during fatigue in single mouse muscle fibers. *Journal of Physiology,* 434: 323-336.

43. Westerblad, H., J.D. Burton, and H. Lannergren. (1997). The effect of intracellular pH of contractile function in intact, single fibers of mouse muscle declines with increasing temperature. *Journal of Physiology,* 500: 193-204.

44. Westerblad, H., A.J. Dahlstadt, and J. Lannergren. (1998). Mechanisms underlying reduced maximum shortening velocity during fatigue of intact, single fibers of mouse muscle. *Journal of Physiology,* 510:269-277

45. Westerblad, H., J.D. Bruton, D.G. Allen, and J. Lannergren. (2000). Functional of Ca^{2+} in long-lasting fatigue of skeletal muscle. *European Journal of Applied Physiology,* 83: 166-174.

46. Westerblad, H., D.G. Allen, and J. Lannergren. (2002). Muscle fatigue: lactic acid or inorganic phosphate the major cause? *News in Physiological Sciences,* 17: 17- 21.

47. Wilmore, J.H., D.L. Costill, and W. L. Kenney. (2008). *Physiology of Sport and Exercise.* Champaign, IL: Human Kinetics.

48. Wilson, J.R., K.K. McCully, D.M. Mancini, B. Boden, and B. Chance, (1988). Relationship of muscular fatigue to pH and deprotonated Pi in humans: A 31P- NMR study. *Journal of Applied Physiology,* 64: 2333-2339.

49. Wiseman, R.W., T.W. Beck, and P.B. Chase. (1996). Effect of intracellular pH on force development depends on temperature in intact skeletal muscles from mouse. *American Journal of Physiology,* 271: C878-C886.

50. Zhang, S.J., J.D. Bruton, A. Katz, and H. Westerblad. (2006). Limited oxygen diffusion accelerates fatigue development in mouse skeletal muscle. *Journal of Physiology,* 572: 551-559.

Original Research

The Effect of Real-Time Feedback on Swimming Technique

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Research was conducted at Manta Mechanics Swim Flume, Lahaina, Hawaii
Running Head: Real-time feedback

ABSTRACT

We examine a new approach for accelerating the learning of efficient stroke mechanics: using a flume equipped to deliver multi-perspective live video footage and force analysis data simultaneously to the swimmer and the coach. A preliminary study of the effectiveness of this approach with a small group of age group swimmers shows gains in ability to generate force of around 20% and to improve swim velocity with only two hours of application.

***KEY WORDS**: Hand force, video, real-time, feedback*

INTRODUCTION

When learning the execution of a complex physical motion, the rate at which the skill is mastered typically depends on the evaluation of how well the motion was performed (the "feedback"), the time between the execution of the motion and the evaluation (the "feedback time"), and the number of repetitions performed. If the motion is highly repetitive (such as in swimming) and the evaluation is poor or the feedback infrequent, then the acquisition of the desired skill can be at best slow, and at worst never achieved. In extreme cases the use of poor technique over thousands of repetitions, with infrequent correction, can lead to debilitating injuries. The goal for successful coaching of stroke technique in swimming is therefore clear; provide high-quality feedback in the shortest possible feedback time on a frequent basis.

Swimming strokes are three-dimensional in nature and involve a complex interaction between the arms, legs, body and head. Indeed, there is still much debate about how we actually propel ourselves through water and therefore what constitutes optimal technique. Consequently, an objective evaluation of stroke technique is far from trivial. Technique evaluations are typically based on visual inspection of the stroke and extend from simple "on deck" viewing of the stroke (which provides very limited information), to the use of underwater mirrors, to multiple-angle, under and above water video footage. The quality of the visual evaluation depends on the expertise of the evaluator (the coach or athlete) and is always subjective. Recently the evaluation process has been expanded to include a force analysis of the stroke that is synchronized with video footage [1]. This represents an important step toward providing an objective evaluation. Moving forward with the assumption that the expertise of the evaluator is sufficient, then what is important for high-quality feedback is to provide the evaluator with as much objective information as possible in a timely manner.

Ideally, feedback is provided continuously to the athlete as they swim, in this way they can correct their technique in real time. Currently this can be done by locating mirrors at the bottom of, or above a swimming flume (visual feedback) and/or by electronic audio communication (from a coach who may be watching the athlete in the flume or via live video footage). All other feedback methods require the swimmer to stop swimming and are therefore slower (in many cases, much slower!).
In this paper we examine the advantages of providing multi-perspective live video footage and force analysis data to both the athlete while they are swimming and also to the coach. That is, we examine the benefits of providing high-quality feedback in real time.

METHODS

Approach to the Problem

The general approach is to deliver multi-perspective live video footage and force analysis data simultaneously to the athlete while they are swimming and the coach. To this end we use a flume (the Elite version of the Endless Pool) equipped with video cameras located below, above, to each side and forward of the swimmer, and a force sensor system attached to each of the athlete's hands (the

Aquanex system [1]). The force data and video streams are piped in real time to a coach's monitor by the side of the pool. The coach selects which data are simultaneously piped to the swimmer's monitors: one near the bottom of the pool, the other above the pool. Each monitor is located such that the swimmer can view the screen while swimming without compromising his or her head position. An additional monitor is located by the side of the pool for immediate playback of recorded video and force data.

Subjects

For this study we used four swimmers from a year-round age group team: two boys (ages 13 and 14) and two girls (ages 15 and 16). The study was focused on backstroke and lasted four days. The athletes represented a wide range in ability in backstroke, from USA-Swimming's "All-Times" to "AA" standard.

Procedures

The first step on day 1 was to record video and force data for each athlete before using the real-time feedback system. This allowed us to determine the swimmer's initial force profiles for each stroke (both shape and strength) and establish their current stroke mechanics. The second step was to educate each swimmer about the target profile for the stroke force curve – a linear increase in force with time. This target profile has both an empirical and theoretical basis [1,2]. The next step was for the coach and athlete to evaluate the force and video data just acquired. The final step was a 30-minute "in pool" session where the athlete focused on improving their stroke profile by monitoring their force profile in real time while swimming. During the first 20 minutes of this period the speed of the water in the flume was set so that the athlete could focus on their stroke without fatiguing too quickly. The athletes would then swim while watching their force profiles in real time and try to adjust their stroke to provide the target profile. Meanwhile the coach monitored both the multi-angle video streams and the force data. When a technique limitation was identified the coach would stream the video feed that best highlighted the limitation, with (or without) the force data, to the athlete and make suggestions on how to correct the limitation. The athlete would then strive to improve their force profile through the elimination of the identified technique limitation.

During the last 10 minutes the athletes worked on maintaining their stroke profiles for one-minute intervals with a one-minute rest period between efforts. That is, throughout the entire 30-minute sessions the athlete was able to work on improving their technique on a stroke-by-stroke basis. The four steps were repeated each day of the study period. During this period the athletes also continued to train with their team as normal but with the request that they practice their new stroke technique as much as possible.

Statistical Analysis

We assume that the variations we see in the force data over each trial are normally distributed around some mean value.

RESULTS

To evaluate the effectiveness of our proposed real-time feedback coaching approach we compare the average peak force per stroke cycle and the average force per stroke cycle over a 60 second swim as measured at the beginning and end of the study. We used these measures as it has been shown that there is a direct correspondence between hand-force and swimming velocity [3]. We also compare the “before” and “after” stroke profiles. We emphasize that during the evaluation measurements, the swimmers did not have access to any feedback. This was done to ensure that any measured changes reflected meaningful changes in stroke. That is, the swimmer had assimilated the necessary change and was not being guided in any way.

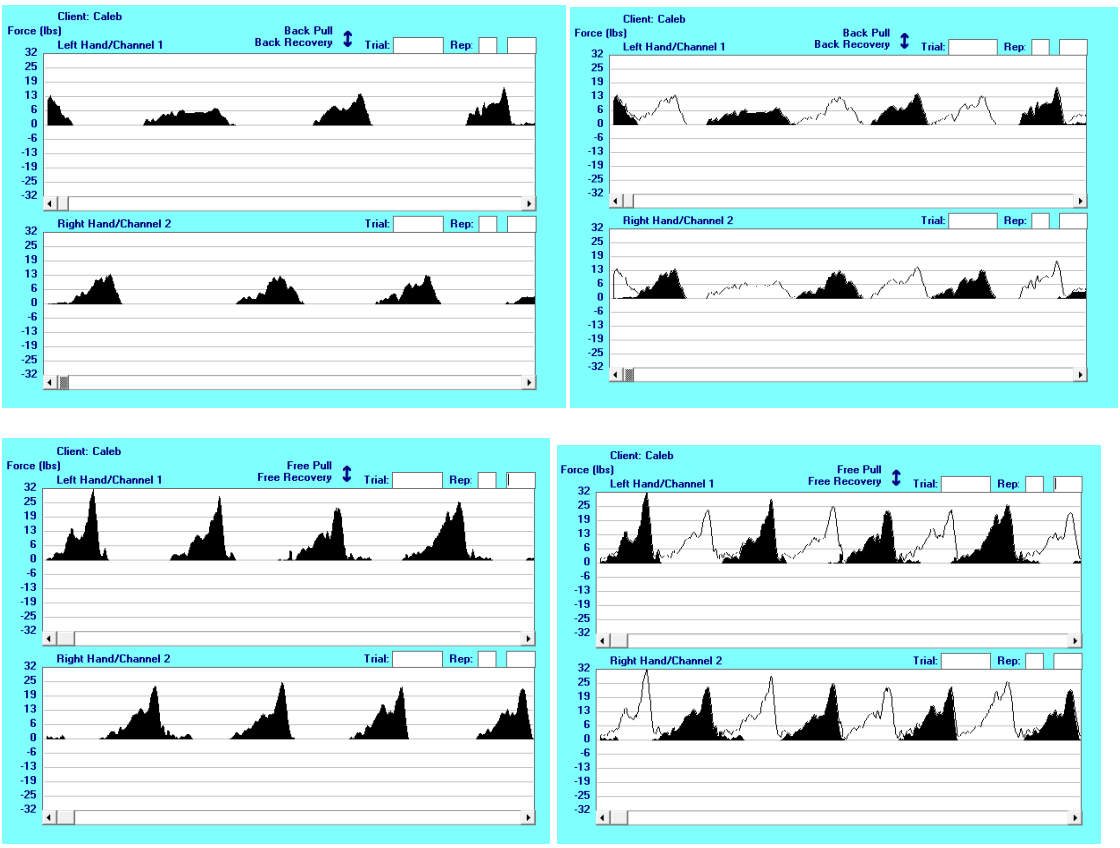

Figure 1. *This shows the hand force profiles for one of the male athletes swimming backstroke at the beginning (top row) and end (bottom row) of the study. The left column shows the individual hand force profiles, the right column shows the overlap of the force profiles.*

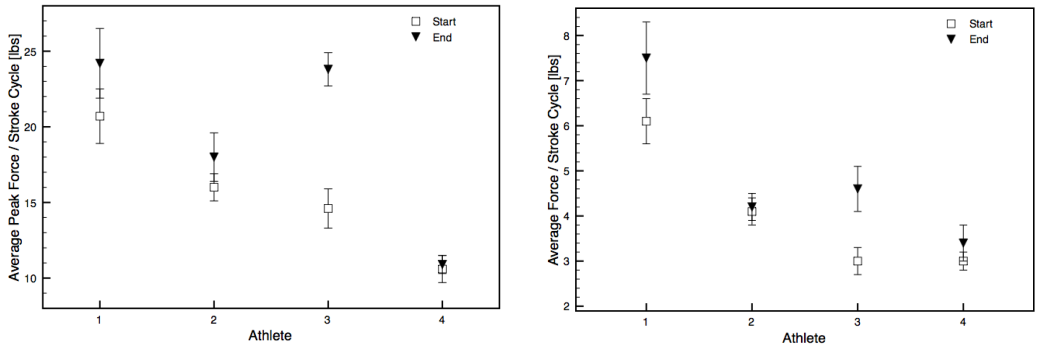

Figure 2. *This shows the average peak force/stroke cycle (left) and average force/stroke cycle (right) for each athlete at the start of the study (open squares) and the end (solid triangles). The ±1-sigma error bars are calculated assuming that the spread in the observed data is normally distributed. The improvements in average peak force/stroke cycle and average force/stroke cycle for the group as a whole were 24% and 22%, respectively.*

We found that after just two cummulative hours of real-time feedback, all four swimmers showed marked improvements both in the shape of their force profiles (e.g., Figure 1) and their ability to generate force during their stroke (Figure 2). Moreover, interestingly, the rate of improvement was

seen to increase throughout the study suggesting that there is some acclimation time that the athlete goes through before the feedback process becomes efficient. This raises an interesting question, "How quickly does this rate of improvement level off?" In addition, we found that the flume water velocity had to be increased for each athlete over the course of the study. This is an indicator that their stroke efficiency had increased.

DISCUSSION

First and foremost, we note that the number of subjects in the study group was small and thus caution has to be exercised in the interpretation of the results. Having said that, it is extremely encouraging that with as little as two hours of real-time feedback we see a significant improvement in both the efficiency of the athletes to generate force in their stroke (of order 20%) and in the application of this force throughout the stroke. We also have some evidence that these improvements translated directly into increased swimming velocity. First, the flume water speed for each athlete had to be increased over the period of the study. Second, one of the athletes had posted a personal best time (by 0.68 seconds) in the 100m backstroke of 1:16.88 two days before the study. They raced the same event again at a meet the day after the study and posted a time of 1:12.82. The above results, along with the recognition that the force produced by a swimmer throughout the stroke provides an insight into the effectiveness of the technique [5], support our hypothesis that real-time feedback is beneficial for accelerating the learning of efficient stroke mechanics. Lastly, the real-time feedback goes to both the swimmer and the coach. For the swimmer, the power of the real-time feedback is two-fold. First it allows them, on a stroke-by-stroke basis, to detect exactly when in the stroke they are losing power and to then experiment with their stroke at this point and to see the results immediately. Second, the feedback then helps them to maintain the new stroke while the body learns what to do. For coaches, the combination of real-time force and multi-perspective video data not only enable them to see stroke limitations, such as subtle hand pitch changes, that would be otherwise be extremely difficult to detect, it also helps them guide the athlete toward a more efficient stroke in a shorter time interval. For example, the effects of suggested changes will be apparent in a very short time, consequently changes that are not beneficial can be quickly abandoned. We note that

during the study we found that the main causes of loss of force were a shallow hand entry, late engagement of the water in the pull phase of the stroke, lack of capitalizing on the push phase and unnecessary (often subtle) hand pitch changes throughout the stroke.

PRACTICAL APPLICATIONS

This study suggests that the use of real-time feedback has significant potential as a teaching/training tool. Unfortunately, not every team is going to have access to a suitably equipped swimming flume. The question then is, how to achieve the benefits of real-time feedback in a typical swimming pool? One way would be to have the athletes swim on thethers with a mirror beneath (or above) them and a coach observing nearby underwater. The potential downside to this is that there is evidence that the mechanics of the swimmer's stroke may change when they are on a tether [4].

REFERENCES

1. Havriluk, R., Analysis of swimming performance using advanced technology, CoachesInfo.com, 2009
2. Havriluk, R., Personal Communication, 2012
3. Havriluk, R., Hand force and swimming velocity, XVth Federation Internationale de Natation World Congress, Indianapolis, IN, 2004
4. Maglischo, E. W., Maglischo, C. W., Zier, D. J., & Santos, T. R., The effects of sprint- assisted and sprint-resisted swimming on stroke mechanics. *Journal of Swimming Research, 1,* 27- 33, 1985
5. Sanders, R., Thow, J., Alcock, A., Fairweather, M., Riac, I. and Mather, F., How can asymmetries in swimming be identified and measured?, *J. Swimming Research*, Vol. 19:1, 2012.

Commentary

Two Butterfly Styles

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Abstract: Butterfly is a complicated stroke, but similarities among elite butterfly specialists are immense. Fly requires a synchronized movement between the arms, body, and legs to help raise and lower the body at the surface. This timing is crucial in both main styles of fly: shoulder driven and body driven fly. The shoulder driven stroke is geared towards sprint with a fast tempo and low amplitude, while the body driven strokes utilizes a larger amplitude undulation for longer races. Fly coordinates rhythm and strength to propel the swimmer forward. This article is a detailed biomechanical view on the stroke of butterfly addressing the biomechanics and hypothesizing the future of butterfly.

Keywords: Fly, biomechanics, shoulder drive fly, body driven fly, technique

Introduction

Biomechanics

Minimal research studies are present on the exact biomechanics of butterfly. This limitation is due to the high variability seen between elite swimmers. Despite the variability, this sequence of events is commonly agreed upon around coaches:

Arms

The arms and shoulder blade muscles are the main contributors of the arms for forward force production. Shoulder stability uses the large trapezius, latissimus dorsi, and many more muscles for optimal force production.

- Hand entry: The hands enter approximately 6 - 8 inches apart, in front of the shoulders in full extension. The finger tips should enter the water first, with the elbows pointed laterally. Keep the elbows higher than the hand or wrist during the entry.

- Initial Catch: The catch begins with the hands pressing down from outside to inside to move the hands medially. In a body driven stroke, the in-sweep is more pronounced, then the shoulder driven stroke. Some would argue, there is no in-sweep in a shoulder driven stroke.
- Catch: The hands continue to move toward midline. The elbows remain high and the palms stay facing towards the feet. The hands move in the vertical plane, and then gradually move horizontally at the end of the catch.
- Push: The hands continue to move backwards, finishing the end of the propulsive phase.
- Recovery: The arms exit the water via the littlest finger, and then slide slightly over the water.

Kick

The majority of force production during the kick is from the quadriceps with the hamstrings and gluteal muscles providing secondary force production. The knee moves from approximately 0 - 90 degrees knee flexion (Richardson1986). Large amounts of ankle plantar flexion are necessary for maximal propulsion.

- First Down-kick: Occurs as the hands enter the water
- First Up-kick: Occurs as the hands start the initial catch.
- Second Down-kick: Occurs during the push.
- Second Up-kick: Occurs during the recovery.

Undulation

Despite the high amount of force production during the kick, the undulation and timing of kick is more conducive with fly elite butterfly.

- Down and Forward Undulation: Chest moves down and hips up as the hands enter the water.
- Up and Forward Undulation: Chest rises and hips move forward, underneath the body during the catch.

A confused aspect of fly is the role of the spine. In fly, keeping the head tucked during the undulation helps facilitate a flat coordinated lumbar spine. Butterfly posture uses a flat back, not an arching low back as many believe. Unfortunately, many flyers raise their hips with their lumbar extensor muscles, not via undulation. These flyers arch their back to elevate the hips, increasing injury risk and impeding velocity, as

a rounded spine creates a less hydrodynamic position.

Like all the strokes, drag is more important than propulsion, therefore finding streamline is essential:

"Time gap differences between skill levels were related to the capacity of elite swimmers to assume a more streamlined position of trunk, head and upper limbs during leg actions, adopt a shorter glide and higher stroke rate to overcome great forward resistance, and generate higher forces and use better technique during the arm pull. Thus, coaches are advised to begin monitoring arm-leg coordination earlier in swimmers' careers to ensure that they attain their highest possible skill levels" (Seifert 2008).

This coordination is essential for undulation.

Timing

A few studies have looked at coordination and timing during butterfly and determined

this coordination separates elite from novice flyers.

"Raising the center of mass was transmitted caudally and contributed to a propulsive 'whip-like' action (Sanders 1995)".

This whip-like action is essential for fluidity and elite fly. Another study said:

"It is concluded that high intra-cycle variation of the velocity of the centre of mass was related to less efficient swimming and vice versa for the butterfly stroke (Barbosa 2005)."

This confirms the essence of coordinated movement during the undulation, to ensure the whip-like action.

Once again, coordination is essential.

Coordinating the body undulation while maintaining a streamline is essential for elite fly.

Breathing

The breath must initiate during the initial catch and return to a tucked head position before the hands finish the recovery.

Breathing requires dissociation from the cervical and thoracic spine. Body driven flyers (see below) are able to breathe more frequently with less stroke disruption. However, breathing is less propulsive than non-breathing in most swimmers

"Breathing: The total time gap was greater with breathing (23.3% VS. 19%), showing less propulsive

continuity between arm and leg actions ($p<0.05$). This was due to the shorter downward leg kick and longer arm catch and upward leg kick that led to longer glide time. Conversely, breathing leads to greater coupling between the hand exit and the end of leg propulsion, which was due to a shorter arm push phase to facilitate the head exit to breathe (Seifert 2010)."

Each breath shortens the down-kick, the most propulsive component of the kick.

Analysis

To generalize, there are two successful styles of butterfly: shoulder and body driven. These are theories on these strokes from video observation; no studies have assessed these differences.

Shoulder Driven Fly

A shoulder driven fly is typically seen in sprint fly. Storied coach Mike Bottom, Head Coach of the University of Michigan, and Milorad Cavic popularized this style of butterfly. These swimmers's use a higher tempo stroke, as the thoracic uses less undulation, specifically in the thoracic spine. This is partially due to the high activity of the scapular stabilizing muscles, potentially inhibiting full thoracic range of motion. No studies are currently provided on a shoulder driven butterfly, but it seems the shoulder driven stroke uses an earlier and more aggressive initial catch through high activity of the shoulder stabilizing muscles. This stroke increases shoulder muscular activation to increase force production. This increase in force production and higher tempo likely increase lactic acid production, and is less rhythmic than the body driven fly.

Body Driven Fly

Michael Phelps and many past Olympians utilize the body driven fly. The high amplitude stroke uses large body undulation as the hands enter the water, decreasing the tempo. Swimmers using this stroke typically spend more time during the entry phase of the stroke, allowing the body to perform a larger undulation. This form of fly is more rhythmic, and allows a larger core connection, but doesn't allow as much catch with the arms and typically has a slower tempo. This stroke does not allow optimal shoulder muscle activation and requires a pause at the entry of the stroke, decreasing tempo and shoulder force production.

Conclusion

The future of fly depends greatly on swim suits. With the current rules, the flotation provided via suits is minimal, as essential contributor to the shoulder driven stroke. In the long course venue, the shoulder driven stroke will increase lactic acid product due to use of more peripheral muscles, without the aid of walls. This combined with less buoyant suits make it hard for this swimmer to be as successful in the 100 and especially the 200 butterfly with a shoulder driven stroke in the long course venue.

However, in the 50 fly, an emerging event, the shoulder driven stroke will prosper due to its higher work capacity and force generation. Therefore, the future of the stroke and biomechanics depend on the event, but provide clear and logical reasons for their purpose.

Practical Applications

The style of butterfly depends on the distance of the race and individual anthropometrics. Typically, shorten distances, poor mobility, and fast tempo are indications for a shoulder driven butterfly. Longer distances, high mobility, and slower tempos are associated with a body driven butterfly.

These skills must be assessed early in a career, to ensure proper motor learning. However, these skills may change over time, therefore frequent assessment is required.

References

- Richardson AR. The biomechanics of swimming: the shoulder and knee.*Clin Sports Med*. 1986 Jan;5(1):103-13.
- Sanders RH, Cappaert JM, Devlin RK. Wave characteristics of butterfly *Swimming.J Biomech*. 1995 Jan;28(1):9-16.
- Seifert L, Chollet D, Sanders R. Does breathing disturb coordination in butterfly? *Int J Sports Med*. 2010 Mar;31(3):167-73. Epub 2010 Feb 17.

- Barbosa TM, Keskinen KL, Fernandes R, Colaço P,LimaAB, Vilas-Boas JP. Energy cost and intracyclic variation of the velocity of the centre of mass in butterfly stroke. *Eur J Appl Physiol*. 2005 Mar;93(5-6):519-23. Epub 2004 Dec 17.
- Seifert L. Differences in spatial-temporal parameters and arm-leg coordination in butterfly stroke as a function of race pace, skill and gender. *Hum Mov Sci*. 2008 Feb;27(1):96-111. Epub 2007 Nov 1.

Book Review

COR SWIMMER'S SHOULDER BOOK

Editors: DR. G. JOHN MULLEN.
Bibliographic Data: 94 pages
Subjects: Sports Medicine, Rehabilitation, Strength and Conditioning

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Description

The aim of swimming training is to achieve optimum performance on the day of competition via three processes; working hard in training to create the required training stimulus, training according to a well planned structured program to maximize adaptations to the training stimulus, and training the behaviors and physiology specifically for competition. Pre-habilitation and injury prevention is sometimes overlooked. Long term planning must target the factors that could hinder training and performance. Training smart should promote an enhancement of performance by reducing the risk of injury via pre habilitation.

The evolution of new knowledge from sports medicine research, such as presented in this book, usually starts with a stark concept that must be further refined; to move from rehabilitation to pre-habilitation, from 'one size fits all' to the individual needs and practices of different swimmers, and from single issues to an integrated picture of injury prevention. The translation from science to practice usually requires a large body of follow-up studies as well as experimentation in the field.

This new book includes fresh information regarding injury prevention and rehabilitation in swimming and resistance training for the shoulder. The objectives of the book are; to explain the need for injury prevention in swimming and provide a plethora of methods to assist in shoulder injury prevention of swimmers.

FEATURES

A well structured discussion about; the anatomy and physiology of the human body, with an emphasis on the shoulder. The book also includes a long explanation with relevant examples about the importance of shoulder injury prevention for swimmers, which finishes off with practical techniques that could be used by health professionals. This textbook is composed of sixteen parts with subsections in most of them. The topics of the parts are: Effectively Hard, Genes, 10,000-Hour Theory, Potential Outliers, Build the Ship,The Simplistically Complex Shoulder, Pathologies,"Having" and "Feeling" Aren't the Same, Spot the Location, Unique Aquatic Creatures, Return to the Pool ,Swim Coaches Guide to Shoulder Taping, Taping Procedures, Three Day a Week Training Program, Two Times a Week Training Program. Each specific chapter has been systematically developed. The tables and figures are numerous, helpful and very useful.

AUDIENCE

The book provides a very useful resource for trainers, strength and conditioners, coaches and healthcare professionals in the fields of sports medicine. However individuals should be certified in the methods recommended for preventing shoulder injuries before using them. The readers are going to discover that this is a reference book for the reasoning behind injury prevention and also techniques that could be used for injury prevention. A great book for undergraduates in sports science with an interest in swimming.

ASSESSMENT

This book is compulsory reading for anyone interested in; an in-depth explanation of the importance of shoulder injury prevention for swimmers and techniques that may be used for injury prevention, sport medicine and swimming, and for those wishing to develop a better understanding of shoulder anatomy. The fact that the author is also a contributor to swimming blogs and swimming magazines makes this book an enjoyable read.

Reviewed by: *Andrew Sortwell Ph.D.c, MsExSci, Grd Dip Sprt Nut, BHMS(Ed).*

Kinetic Density: A Different Approach to Recruit and Retain Swimmers

Miguel Corsi
(Member of the International Society of Swimming Coaching)

Gaston Bachelard was a french philosopher that demonstrated how the progress of science could be blocked by certain types of mental patterns, creating the concept of "epistemological obstacle". This kind of psychological hindrance makes difficult the learning of new concepts in the field of science.

In a similar way, Thomas Kuhn, author of "The Structure of Scientific Revolutions" wrote in his book:

Because the student largely learns from and is mentored by researchers "who learned the bases of their field from the same concrete models" there is seldom disagreement over fundamentals. Men whose research is based on shared paradigms are committed to the same rules and standards for scientific practice. That commitment and the apparent consensus it produces are prerequisites for normal science, i.e., for the genesis and continuation of a particular research tradition.

Swimming training, an activity where the so called hard and soft science are interrelated, is also chained to the epistemological burden of practice through the years. Entire generations of coaches and swimmers had been forged and trained based mainly in an adaptation of what elite swimmers practiced to be the best in a certain moment.

The continuous improvement of the performances reflects the progression in the methods of training, which suggests that, as long as the swimmers keep breaking their own records, there is no need to rethink the way we coach...or there is?

Since James Counsilman published his first book *The Science of Swimming* in 1968, the meters swam have been growing in a substantial way. At the beginning, to swim 8.000 meters daily was ok, but then, a sort of inflationary process took place and 12.000 and 16.000 were numbers not so weird.

Nowadays there is a shy tendency to questioning the validity of this philosophy of training and some authoritative researchers are suggesting that there is another way to reach high levels of sportive excellence.

David Costill, one of those scientific, said:

"Most competitive swimming events last less than two minutes. How can training for 3-4 hours per day at speeds that are markedly slower than competitive pace prepare the swimmer for the maximal efforts of competition?"

http://coachsci.sdsu.edu/swim/bullets/taper6.htm

There is also an interesting french investigation:

A group of researchers studied the training and performance of competitive 100m and 200m swimmers over a 44-week period. Their findings were as follows:

Most swimmers completed two training sessions per day; Swimmers trained at five specific intensities. These were swim speeds equivalent to 2, 4, 6 and a high 10 mmol/l blood lactate concentration pace and, finally, maximal sprint swimming.

Over the whole season the swimmers who made the biggest improvements were those who performed more of their training at higher paces. The volume of training had no influence on swim performance.

http://www.pponline.co.uk/encyc/swimming-training-why-high-intensity-training-is-more-productive-for-swimmers-than-high-volume-training-213

Certainly we could recall several anecdotes about swimmers that trained relatively few meters in a season but improved his/her personal best.

To elucidate the advantages or not of swimming many meters to reach high levels in competitive swimming is not a minor issue. The pursuit of a method of training that requires much less time in the swimming pool and in the gym is beyond the effectiveness of it. It could avoid quitting the activity in a significant numbers of potential competitive swimmers, those that are expected to expand the basis of the pyramid.

The planning of a workout should not only take in account the technical and physiological aspects of a swimmer. The most important and most overlooked feature in our human material is their emotional and psychological maturity. That is why I mentioned the interrelationship between the hard and soft sciences: they are taller than us, they perform awesome in the water but, deep inside, they are suffering the process of growth, both physical and psychical. That means that not all are willing to spend or waste endless weeks of hard training, sacrificing other interests and activities as important as swimming.

It´s a pity, but this is a fact: a huge number of potential world class swimmers have abandon or even could not have been detected simply because they were not willing to put his head into the water in a, for them, monotonous activity that last 3-4 hours daily, six days a week.
Are they wrong?

Ok, let´s assume that those individuals would not change their mind regarding to the traditional way of training, BUT, they could be interested in train 120 minutes a day, 5 or perhaps 6 days a week, and still have interesting performances. Is it possible to swim a fraction than the others and still succeed at world levels?

Yes, it is.

Remember Michel Gross?

From Wikipedia:

He was probably the finest swimmer in the world in the 200-meter butterfly race from 1981 to 1988. In this period he set four world records, won two world titles, four European titles and one Olympic gold medal. He is perhaps the finest European swimmer ever.

On July 29th, 1984 (Olympic Games, Los Angeles) he set a world record in the 200 freestyle: 1:47.44. That record lasted until September 1988.

The remarkable thing here is not the record itself, but the method of training that he (and his team) applied: quality, not quantity.

Let me share a short passage from an article published a few days before the 1984 Olympics, *"The Americans have so much time—ja?—that they sometimes waste it," says Gross. "You can train for four hours a day and do nothing or you can train two hours a day very strong. That is more important, instead of only swimming meters and meters."*

http://sportsillustrated.cnn.com/vault/article/magazine/MAG1122295/2/index.htm

As a result of all the preceding and with the goal of overcome the constraints of time, I selected a number of ideas from different fields and combined them in a method that I called ***"Kinetic Density"***. I developed and tested that method, first with myself as master swimmer and later with the teenagers of my club. In both cases the results were positive.

For a better understanding of the *Kinetic Density* method I divide it in three main parts:

- Hydrodynamics
- Neural efficiency
- High Intensity Interval Training

Hydrodynamics - Square of the velocity

Let´s go back to the aforementioned swimming classic *The Science of Swimming* by James Counsilman. At the beginning of the book he briefly explains that the drag a body creates as it moves through a fluid increases approximately with the square of its velocity. i.e. swimming 2 % faster creates 4 % more drag.

Many books and articles that discuss the hydrodynamic aspect of competitive swimming mention this principle, but they put the emphasis in other analysis, like the angle of attack, lift forces etc. Of course that kind of researches are important, but, as there is less coverage of the Square of Velocity principle, perhaps the swimming community underestimates its crucial importance.

The basis of the *Kinetic Density* method is this, sometimes forgotten, hydrodynamic law. A deep comprehension of it would facilitate its practical implications.

There are several indicators to prescribe different loads or intensities: Borg Rating of Perceived Exertion (RPE), Heart Rate, a percentage of the time for a specific event, etc.

To maximize the effectiveness of the training, we prefer to use this last criteria, but, we are conscious that it could be a tricky one.

Suppose that we have a swimmer, with a PB of 58 sec., that usually swims 15 x 100 freestyle every 1:30, at an average of 1:12 (80.55 % -1.38 m/s). To elicit a physiological adaptation we ask him to improve his average time to 1:10 (82.85 % -1,42 m/s)

If we feel comfortable with a spreadsheet, we could quickly develop a number of combinations to plan a whole season:

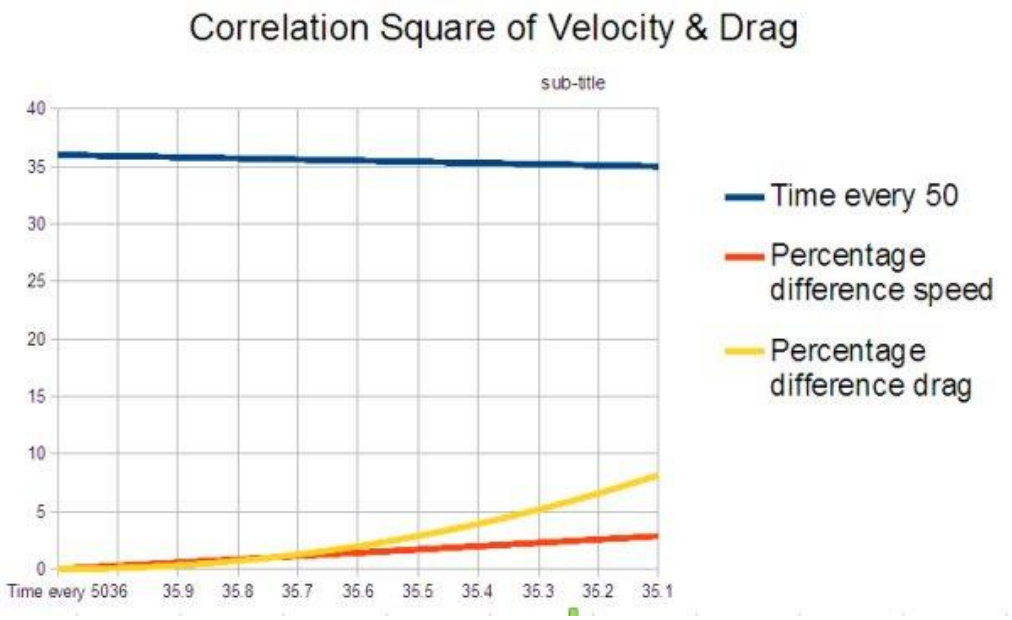
- 10 x 200 at 80 %
- 16 x 50 at this or that percentage
- 12 x 100 , first at 80%, second at 85 % and third at 90%

This could give us a false sense of confidence, as we think that the percentages of velocity are percentages of effort.

Unfortunately, this is not the case. Like us or not, the square of the speed is there to remember its existence, in a painful, lactacid way.

In our example of 15 x 100 (1:12 to 1:10) , the increase of speed in 2.85 % equals, according to the Square of velocity law, to an increase of the drag of 8.16 %

Time every 50	Speed m/s	Percentage difference speed	Percentage difference drag
36	1.3888888889	0	0
35.9	1.3927576602	0.278551532	0.077590956
35.8	1.3966480447	0.5586592179	0.312100122
35.7	1.4005602241	0.8403361345	0.706164819
35.6	1.404494382	1.1235955056	1.26246686
35.5	1.4084507042	1.4084507042	1.983733386
35.4	1.4124293785	1.6949152542	2.872737719
35.3	1.4164305949	1.9830028329	3.932300235
35.2	1.4204545455	2.2727272727	5.165289256
35.1	1.4245014245	2.5641025641	6.574621959
35	1.4285714286	2.8571428571	8.163265306

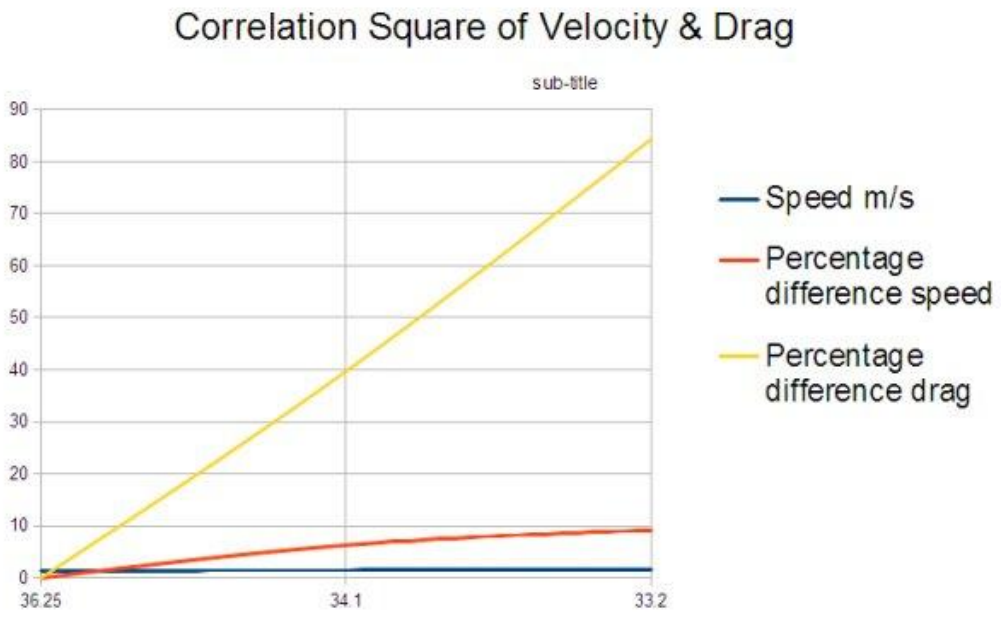


But lets see what happens with the 12 x 100 descending 1-3 workout for our 58 secs swimmer.

Swim at 80 % of his personal best means an easy 1:12.5, then 85 % is 1:08.2 and finally 90 % equals to 1:06.4 (well done, boy!)

If we express that in velocity (meters/seconds) and correlate the difference in percentage respect to the lower level of effort, according to the Square of velocity law, the eloquence of the graph leave us speechless.

Time every 50	Speed m/s	Percentage difference speed	Percentage difference drag
36.25	1.3793103448	0	0
34.1	1.4662756598	6.3049853372	39.7528401
33.2	1.5060240964	9.186746988	84.39632022



In just three seconds faster every fifty meters, the drag/resistance jumps an astonishing 84 %.

I suppose that next time we prescribe a descending workout like this, we would be more conscious of the impact that this kind of effort provokes in the swimmer. Said that, lets move to the other issue.

Neural efficiency

In this section we will use a free interpretation and application of Computational neuroscience. As you will see later, this is an important component of *Kinetic Density.* *(*http://en.wikipedia.org/wiki/Computational_neuroscience*)*

Swimming is an activity included in the group of cyclical sports, which could provide the false idea of an easy task. After all, you just need to teach the proper timing to coordinate stroke and breathing and that´s it.

Of course not, you are yelling, and I could not agree more.

The level of kinetic sensibility needed to swim effortless at a decent speed requires a lot of practice, under the sound guidance of a swim teacher or coach. This is not something that you could master in a short period of time. Practice-feedback-practice-feedback again and again and again (hey!, it is a cyclical sport, remember?)

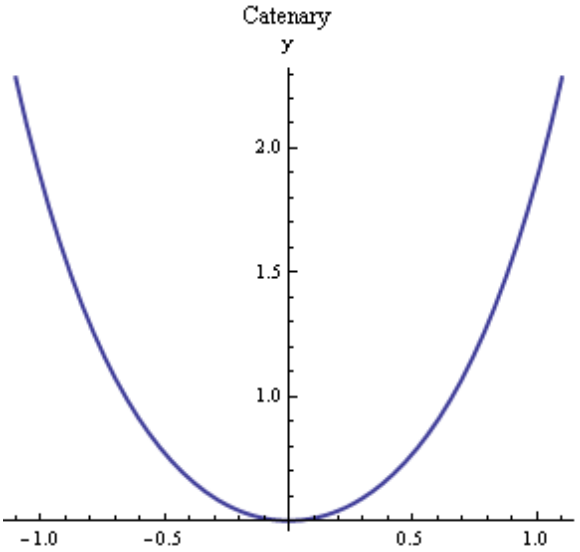
Walking up and down by the side of the swimming pool we are permanently monitoring the efficiency of the stroke in our swimmers. A number of items are checked to verify if they are performing according to our criteria. But how many times, if ever, do we ask where such amount of information is stored? What are the process involved to sort, classify and prepare for a later utilization?

Let´s assume that the information of the sportive gesture could be graphically represented and that representation is stored, somehow, in a database allocated in the cerebral cortex for its posterior retrieve and application.

(Cortical rewiring and information storage.

http://www.biomedsearch.com/nih/Cortical-rewiring-information-storage/15483599.html)

For example, if we draw in a paper a mathematical function, the brain should store the route of the hand in only two axis: (*x,y)*



f (x,y)

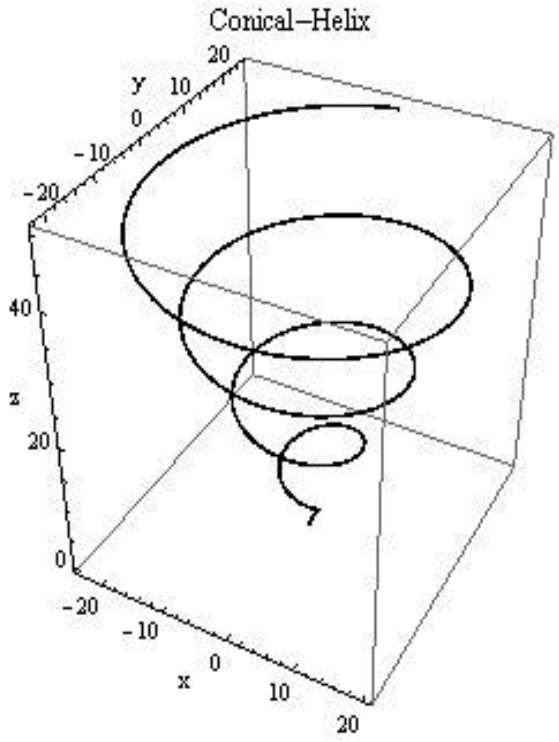
Plane Curves: Catenary from Differential Geometry Library. http://digi-area.com/DifferentialGeometryLibrary/PlaneCurves/Catenary.php

Now let's situate ourselves at the side of the swimming pool, when we are trying to show the gesture of any stroke. Leaned forward, we move our hands repeating as a mantra "reach farther, keep your elbows up, roll your body"

In this case, our hand would be moving in a three dimensional pattern (*x,y,z*)

A 3D mathematical function could give us an approximation to what I want to express . Of course, the example given *does not represent the stroke*, is... just maths.

$\int (x,y,z)$



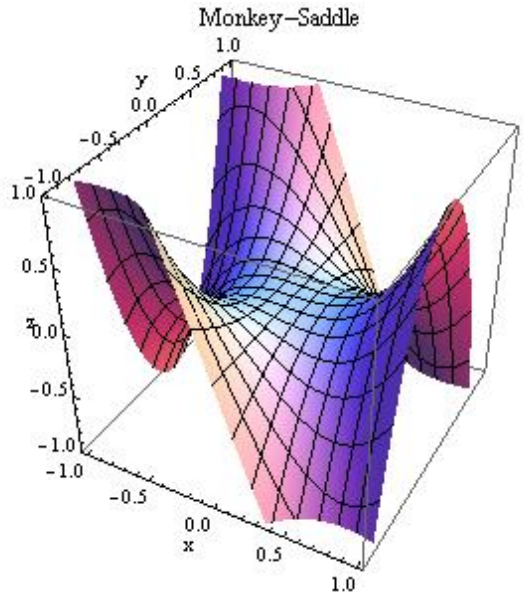
Space Curves: Conical helix from Differential Geometry Library. http://digi-area.com/DifferentialGeometryLibrary/SpaceCurves/Conical-Helix.php

The aforementioned mathematical representations only describe the sweep of the hand, without any reference to speed or the conditions of the environment.

In our computational model, the representation, storing and retrieving of the swim is remarkably complex.

The cerebral cortex of the swimmer process a huge amount of data but to simplify this theorization we put the focus in the most relevant. To the last 3D graph now we add four more variables: speed, pressure, level of lactic acid and perceived effort

ƒ (x,y,z,s,p,Lact, pe): x,y,z, speed, pressure, level of lactic acid, perceived effort



Surfaces: Monkey saddle from Differential Geometry Library. http://digi-area.com/DifferentialGeometryLibrary/Surfaces/Monkey-Saddle.php

Now, the stored model is a 3D surface (not a line), with a variation of colors that means different data. The stored information to swim crawl at 90 % is different to swim at 85 % or at 100 %. Yes, perhaps the path that describes the sweep of the hand, the (x,y,z) function, *is similar* in the different percentages, but the rest of information - speed, pressure, level of lactic acid and perceived effort - , as a consequence of the Square of the Speed Law, deeply alter the data stored in the cerebral complex. A little change in the speed, faster or slower, elicits a re-

arrange of the data that the cerebral cortex is forced to update. That little change in speed alters the perception of the resistance generated by the water, varies the stroke (http://w4.ub.uni-konstanz.de/cpa/article/viewFile/620/545), modifies the concentration of lactate and impacts on the perceived effort. And all this information goes to that hypothetical database, where is processed and stored to be later retrieved. If we could represent that information in a mathematical fashion, probably the formula would read as follow

Freestyle for 100 maximal effort:

$$\int \text{100 Free } (x_{100F}, y_{100F}, z_{100F}, v_{100F}, p_{100F}, Lact_{100F}, pe_{100F})$$

Freestyle for a workout of 10 x 200 :

$$\int \text{10x200 Free } (x_{10x200}, y_{10x200}, z_{10x200}, v_{10x200}, p_{10x200}, Lact_{10x200}, pe_{10x200})$$

	∫ 100 Free	∫ 10x200 Free
x,y,z	similar	similar
Speed	15 % faster	-
pressure/drag	200 % greater	-
lactate	16-20	4
Percived effort (Borg scale)	Extremely hard	Somewhat hard

It could be an interesting speculation on how is retrieved that stored information in different circumstances. During a race, for example: is there any kind of contradictory information between the conscious movement that the swimmer wants to performs and the learned (an slower) skills? Which of those learned skills would take control during a swim at maximal speed?

I invite the reader to “change the hat”, in De Bono (http://en.wikipedia.org/wiki/Edward_de_Bono) words, and start thinking the training of swimming as the training of a complex skill and not as the development of several physiological functions.

If you take this way of thinking, at least for a while, you could have a new perspective in the debate about the advantages or disadvantages of swimming many meters at slow pace.

If we adopt the criteria of improve a skill instead of focus on levels of lactate or heart rate, we need to modify radically the quantity of the meters swam and, most important, the type of exercises.

Here is where comes into play the third and last component of Kinetic Density

High-intensity interval training

Let me put it clear from the very beginning: aerobic training is very important when we plan according to Kinetic Density Method

BUT

the percentages and volumes utilized are much smaller than those used by traditional methods. In our criteria, after swimming a certain number of meters in AT , the gains are smaller in comparison with the time devoted at such effort. We try to optimize the ROI (return of investment) and our capital (time) is scarce. So, we do use subaerobic workouts, but in a smaller proportion than other clubs do.

Also, we refer to the sets and workouts that compound Kinetic Density with a different nomenclature, as they reflect better the goal of each workout.

The structure of the season is undulatory, following a pattern of 2 weeks load x 1 recovery or 3 x 1. Within the weekly planning also we alternate days of load with recovery ones, but here we are more flexible in function of the performance of the team.

However, as soon we start the season, we include high percentages of exercises specific to the competition and VO2max. Why? As those elements need a considerable amount of time to be mastered by the swimmer, we don´t see the point to wait till the peak of the season to remember that we need to focus on them.

Our approach is to improve a complex skill and we work with that goal in mind. According to our standards the measure of any improvement is given by, among other factors, swim efficiency, the amount of meters swimming at or close to race pace and level of effort to perform that workout. "*To swim at race pace*" should not be confounded with lactacte tolerance. We adjust the ratio effort/rest to permit the swimmer to learn the feeling of the race without produce high level of lactate. Short distances (25, 50 and 75) with 15 to 30 seconds rest are the usual and up to 1200 -1500 meters for the total volume of this workout.

This kind of workout is to improve the neural efficiency.

To meliorate the VO2max we were inspired in the research of Veronique Billat, the french investigator. One of hers famous investigations is "*Very Short (15 s±15 s) Interval-Training Around the Critical Velocity Allows Middle-Aged Runners to Maintain VO2 max for 14 minutes"*

http://www.lephe.org/attachments/044_37.2001-Billat-very%20short%2015-15-IJSM.pdf

Starting from this and other Billat´s papers we developed several routines to build up VO2 max. As is very difficult to our club to afford biochemical analysis of lactate, we concede that a good rule of thumb to calculate the optimal speed to elicit VO2 max, is to take the time of a 400 meters at full speed. Then, the workouts are based on distances from 25 to 100 meters at that speed. The test is valid for three strokes: freestyle, backstroke and breaststroke.

However, neural efficiency and a good amount of meters at VO2 max are not enough to have a balanced program.

I am not referring here to more aerobic work, I already put that clear at the beginning of this section.

When we started to combine neural efficiency and the Billat´s method the workouts were encouraging. But when the moment of truth came, swimming meets, the results were not correlated with the amazing times that the team had been performing.

After some (numerous really...) analysis we arrived to the hypothesis that those disappointed outcomes were the consequences of the advantages of have been swimming many meters at high speeds with little production of lactic acid...oops!

1-800-HELP-A-COACH

To make the long story short, at the end of another round of investigations, we read about the Tabata´s method. Here is a short explanation from Wikipedia:

Tabata method

A popular regimen based on a 1996 study by Izumi Tabata uses 20 seconds of ultra-intense exercise (at an intensity of about 170% of VO2max) followed by 10 seconds of rest, repeated continuously for 4 minutes (8 cycles). Tabata called this the IE1 protocol. In the original study, athletes using this method trained 4 times per week, plus another day of steady-state training, and obtained gains similar to a group of athletes who did steady state (70% VO2max) training 5 times per week. The steady state group had a higher VO2max at the end (from 52 to 57 ml/kg/min), but the Tabata group had started lower and gained more overall (from 48 to 55 ml/kg/min). Also, only the Tabata group had gained anaerobic capacity benefits.

http://en.wikipedia.org/wiki/High-intensity_interval_training

The majority of the practical applications of Tabata are aimed to terrestrial activities, very few mention our environment, the water. Even though this HIIT is very, very hard, in the water is possible to resist it better for three advantages

- The body is in horizontal position, so there is less workload to lift and pump blood
- There is no impact
- The water dissipates heat more effectively than the air

So, the logical next step was to adapt Sensei Tabata´s method to our planning.

A classical (for us) Tabata´s workout is 8 x 25 more than full speed, starting every 30 secs.

So, to recapitulate, Kinetic Density is compounded by:

- Be conscious of the Square of speed law: small variations in speed are reflected in big changes of the drag/resistance, with a strong impact in the stroke and the fatigue of the swimmer
- Neural Efficiency: to swim many meters at high speed with efficiency and with little production of lactic acid. Short distances, from 25 up to 75 with 15 to 30 seconds of rest are the best option.
- A lot of VO2 max according the method of Billat
- Tolerance to lactic acid, Tabata´s Protocol

After several months of been using this method we noticed the following advantages:

- Swimmers are more focused in the technical aspect of the stroke. As the workouts are composed by shorter distances their attention is more effective, is less probable that they change to “automatic mode” as usual in longer series (8 x 400 perhaps?)
- The sense of rhythm and effort is stimulated in a daily basis. As a positive consequence, they learn faster how to regulate themselves in a competition
- The loads in the workouts are more intense than other “traditional” methods, but as the time devoted every day is less than 120 minutes, there is no major danger of accumulated/hidden fatigue, the kind of tiredness that could drive to overtraining. In this sense the period of recovery is shorter and the same applies to the taper.
- The type of workload allows a more specific stimulation of the fibers, according to the distance for which the swimmer is preparing to.

Perhaps I would put a question mark in the case where the swimming meet would be strenuous: a lot of races with qualifying heats, semifinal and finals. Maybe I would add more meters; this is a homework to be made.

At the present moment, we use Kinetic Density only with swimmers of 13-14 and up, with at least one year of traditional training. I don´t think this method should be used in younger kids.

Now, the practical examples, just in case you were asking.

First (please, don´t laugh) let me put my personal example.

Actually I discover this method almost by accident. Back in 2005, after a short hiatus in master swimming measured in years, I remember finishing a tearful 5 x 400 in an unconfessed time and promising myself to “invent” a more realistic way of training. That was when I started to looking for other strategies. Later on, in the 2007 Argentinian Master Championship I won the 800 meters (http://www.fen.org.ar/resultados_2007/2JORNADA.TXT) and the 400 IM (http://www.fen.org.ar/resultados_2007/3JORNADA.TXT). A few days after the meeting I realized that I had the times to swim in the XII FINA World Masters Championships – Perth AUS- 2008. So, all that summer I trained using Kinetic Density and then flew to Western Australia (http://www.fina.org/project/docs/masters/ma_2008_sw_M.pdf) . In 2009 I swam in the Southamerican Masters Championships https://skydrive.live.com/?cid=6a7578c36a176a54&resid=6A7578C36A176A54!1947&id=6A7578C36A176A54!1947

Ok, for an old man in his 50´s is a nice story, but how about younger swimmers?

I have been using this method with my swimmers since 2011. In this few months our kids improved their times and started to perform well at regional and even at national level. In the summer 2011/2012 we swam only once a day, no more than 4500 meters and complemented the training visiting the gym three times a week. Now in the winter we swim an average of 3000-3500 meters 5 days a week.

Results?

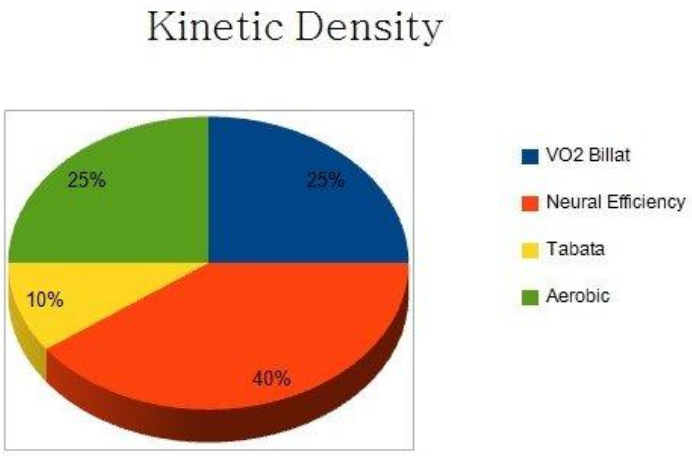
All the team improved their times, from 50 to 800 meters freestyle and also in styles. The three best swimmers (born in 1997 and 1998) integrated the team that won the EPADE games (patagonia region) http://natacionpatagonica.blogspot.com.ar/2012/04/rio-negro-campeon-de-los-epade-2012.html

One of these swimmers won the 200 breastroke and the other one made the time to participate in 200 backstroke Argentinian Championship (2:20.67). This last championship is for the fastest swimmers, not for age groups. Also, two of those swimmers, a girl and a boy, classified to represent Argentina in the Giochi della Gioventù 2012 (Games of the Youth) http://giochidellagioventu.coni.it/ in Italy.

Miscellanea

Perhaps you are asking yourself “Why this funny name, Kinetic Density?”

Well, it surged naturally. If you put the percentage of the different workouts in a pie chart....



it is obvious that there are much more quality workouts than the orthodox way, the speed training has more relative weight than the rest of the exercises, in other words, more **Kinetic Density ...**

Further reading

http://coachsci.sdsu.edu/swim/bullets/energy39.pdf

http://www.swimmingcoach.org/Journal/jsr_home_docs/Maglischo%20part%20II.pdf

The Heat Sheet --- An Autobiography

Allan Kopel (M Ed, MBA)

Hello. I am a heat sheet and I want to tell you a little bit about myself. Depending on the number of years you have been swimming, you may have differing perspectives on what my purpose in life is. It may not seem glamorous but I am actually very popular at certain times. There are, unfortunately, some misconceptions about me.

My basic role in life is to help people stay organized at swim meets. Swimmers and coaches read me to find out what heats and lanes they are assigned to for their races. This same information lets parents know when their child will be swimming. Officials also use me in order to stay organized as they oversee swim meets. At championship meets with on site announcers and media coverage, I am used to help those people follow the events and do their jobs well. So far it is rather simple.

At most swim meets the heat sheets are printed ahead of time. Coaches receive a copy of me with any other pertinent meet information upon arrival at the meet. Coaches can often be seen highlighting the names of their swimmers so they can observe every swim. Parents and other spectators typically receive a heat sheet when they pay their admission to the meet. The first thing most people use me for is to find out where they are seeded in their races. Seeding is based on the entry time submitted. Let's take an example of what you may see when you read me.

Assume that in the 100 meter freestyle, there are 48 swimmers entered and you are swimming in an eight lane pool. This means there will be 6 heats (48 entries / 8 lanes = 6 heats). The entries will appear slowest to fastest. Generally, if the meet is a "timed final" format, the top 8 entries will be in the last heat (heat 6 in this case); the next fastest 8 in the next heat, etc. Timed final, in case you do not know, means that swimmers race one time to determine the order of finish and to record their time for the event. The final order of finish is determined solely on the time swum, so you should not assume that the order on the heat sheet has any bearing on the time you will swim or the order you will finish. Within each heat the swimmers are assigned lanes based on their entry time. The fastest entry is in lane 4, then lane 5, lane 3, lane 6, lane 2, lane 7, lane 1, and finally lane 8. A common misconceptions about me is that the entry time, heat and lane assignment printed in the heat sheet dictates the results. The heat and lane assignments are based solely on the entry times, and have no bearing on the possible order of finish after the race has been swum. Remember to use me only to know when and where you swim. I am not an

indicator of how you will swim or where you will rank among the competitors. Each swimmer has one's own lane in which to have a super race. When you have fun and race great, your time may improve and the order of finish may be very different from the order of the entries.

Let's use the same example of 48 swimmers in an 8 lane pool and look at a swim meet with a preliminary and final format. All swimmers are placed into a heat and lane for the preliminary swim, during which each swimmer races for the opportunity to compete in the championship finals. Times from the preliminary heats are official and can count for records and rankings. During the preliminary heats, the last 3 heats are the "seeded heats" and are entered in a "circle seeded" fashion. These "seeded heats" include the fastest entry times: the fastest 24 in an 8 lane pool; the fastest 18 in a 6 lane pool, etc. Follow the pattern ahead and you should see why they are called "circle seeded" heats. The top entered time will be in lane 4 of heat 6 (the final heat of this race). Lane 4 of an 8 lane pool is right in the middle and is considered the fastest lane. The next fastest time goes in lane 4 of heat 5; the next in lane 4 of heat 4 and the next (the 4th fastest entry) in lane 5 of heat 6; then lane 5 of heat 5; lane 5 of heat 4; and lane 3 of heat 6. This pattern keeps going, moving from the center lanes to the outside lanes, until the fastest 24 entries fill out the lanes of the last three heats in the event. Are you ready for a little quiz? What lane and heat will the 8th, 15th and 22nd fastest seed times swim in? (Seed 8 is in heat 5, lane 3; seed 15 is in heat 4, lane 2; and seed 22 is in heat 6, lane 8). How did you do? Once again, entries are only for the purpose of having a fair

and organized race setting. Racing in a seeded heat can produce a finish with people way ahead and behind others because the times in a circle seeded heat may differ quite a bit. Always race your very best. You always have a chance to achieve a good time, and the actual finish within a heat can be very misleading relative to one's overall finish in the event.

Remember that we have only taken care of 24 of the 48 entered swimmers so far. The remaining swimmers are entered fastest to slowest, the same as in a "timed final" event. So, heat 3 will have the 25th through 32nd times; heat 2 has the 33rd through 40th, and heat 1 has the 41st through 48th entry times. Remember that the fastest entry in the heat is assigned to lane 4, then lane 5, lane 3, lane 6 and so on.

Whereas in the timed final format swimmers race just one time for their team points and for the awards being presented, in a preliminary and final format, swimmers race first in the preliminary with the fastest persons from those heats earning the right to race in the finals. Finals may include one, two or three sections, often called "A", "B" and "C", or Championship, Consolation and Bonus Sections respectively. A new set of me (heat sheet) gets printed for the finals sessions. I typically get referred to as "finals sheets", but for all intent and purpose, I still function as a heat sheet for that session.

In preliminary heats, swimmers may finish in any order, based solely on the time from the preliminary swims. During finals, a swimmer may only rank as high or low as the places within his final heat. For example, if after the preliminaries you qualify for the championship final (top 8 in an 8 lane pool), as long as you finish your race legally in finals, you can not finish any lower than 8th place. Similarly, if you qualify 9th through 16th from the preliminaries and swim in the "B" section of finals, you could possibly set a world record in your "B" section final, and it would count, but the highest you could finish in the meet would be 9th. Notice also that you must complete your race legally in the finals in order to score points for your team and to earn any awards. Getting disqualified in a final session would be like racing fast in the preliminary heats and then choosing not to participate in the finals session. Within the heats of the finals sessions, swimmers are assigned lanes similar to what we've seen already. That is, the top qualifier from the preliminaries is in the championship, or "A" heat in lane 4 (of the 8 lane pool), and the next fastest is in lane 5, then lane 3, lane 6, etc. See, there is a pattern to this. By the way, your preliminary heat swim has no bearing on your finish within the finals. Everyone in finals starts even, with the only difference being the lane you are assigned to. It is not like some sports where one's score from preliminaries may carry over to finals.

Some swim meets such as the Olympic Games and World Championships function with an additional heat of racing; a "semi-final". In those meets, the first round of heats (sometimes called the trials) are seeded the same as any meet with preliminaries and finals. The final three heats of the preliminaries are circle seeded

as already described and the remaining heats (if the number of entries exceeds the capacity for three heats) are seeded as described (I.E. – the 4th from the last heat has the next fastest 8 swimmers – for an 8 lane set up – etc..). These "preliminary heats" determine the lane and heat assignment for the "semi-finals", with new heat sheets (Yippee – more of ME) printed. These semi-final heats are circle seeded but with just 2 heats. The fastest qualifier is in lane 4 of the 2nd semi-final; the next fastest qualifier is in lane 4 of the 1st semi-final; the 3rd qualifier is in lane 5 of the 2nd semi-final etc. As in all other "HEATS" and assignments on the Heat Sheet, the lane and heat is only to insure a fair race. Whoever swims one of the fastest 8 times within the two semi-final heats earns the right to race in the Championship FINALS and thus, earns a right to win a medal at that swim meet.

So are we a little more familiar with who I am and what you can expect to learn from me? The heat sheet is helpful but should NEVER be thought of as determining how you will swim or where you will finish in the rankings. There are a few more things you may notice when you read me. Some meet hosts use me, along with the chance to run a meet, as an opportunity to raise money. One way to do this is to sell advertising space in the pages in the heat sheet. You can sell ad space to businesses as well as to people wishing to offer support and kind words to their favorite team or swimmer. It takes a lot of work to sell this ad space but it is an important job as this is one way to keep your beautiful swim team financially strong. You might also see times listed that pertain to the swimmers at the meet. At a scholastic meet for example, at the top of each event you might find times like the

meet record; the state high school record; the national high school record; and the standard to submit for National Honors. Other swim meets have similar times listed which make people aware of standards to shoot for. In an age group championship meet, you might see the time standards like the meet record; the region record; and the time to qualify for all-star or higher level meets.

One final item you may find in me is a time line. A time line is an approximation of when each event will begin. This is just an estimate. If a time line is posted, the events should not begin earlier than the posted start time. It is important that you confer with your coach to learn if the meet is observing a set time line because you do not want to miss your event.

I hope you have enjoyed and learned something from this. I enjoy being your heat sheet, but I do not want you to ever think that I am anything more than a guide for knowing your heat and lane assignment. Your coach may have some keen observations to share with you, but you should always try your very best and never assume that the order of times in the heat sheet indicates what the order will be after you and the others race. Remember to ask your coach questions about the heat sheet or anything else you may wish to know. Coaches are eager and able to help you. Also remember that heat sheets are put together by people. Even though these people work hard and want to do things well, they sometimes make mistakes. If, for example, your name is left out of an event or your name is misspelled, point this out to your coach. Your coach can usually make the necessary corrections. If

there is a mistake in the heat sheet, please do not let it get you upset. Let your coach take care of the problem, while you go through your warm up and get ready to have a great day of racing. Have fun and race like a champion. I look forward to seeing your name printed on me at your next swim meet.

Journal of the International Society of Swimming Coaching (JISOSC)

JISOSC considers the following types of articles:

- **Book Reviews:** a broad perspective of texts, technical and lay books on swimming, physiology, biomechanics, nutrition, metabolism and biochemistry will be considered for Book Review.
- **Brief Communications:** Original research of high quality with limited goals or restricted focus will be considered in this category.
- **Commentaries:** focused and opinionated articles on subjects within the journal's scope. These articles are usually related to contemporary issues, such as recent research findings.
- **Critical Reviews:** Authoritative summaries of any subject within the journal's scope with dispassionate and rational critique of the field. Critical reviews may be unsolicited or proposed by the editorial staff.
- **Letters:** they can take three forms: a substantial re-analysis of a previously published article, or a substantial response to such a re-analysis from the authors of the original publication, or an article that may not cover 'standard research' but that may be relevant to readers.
- **Methods:** Papers describing new methods that advance research in swimming will be published.
- **Original Research:** should be clinical or basic research with clear conclusions and appropriate terminology. All outstanding clinical or basic research with clear conclusions and appropriate methodology should be submitted to the International Journal of Swimming Kinetics.
- **Perspectives:** normally commissioned and usually based on one or more recently published articles interesting enough to warrant a short commentary.

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