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The Mechanical Energy-Generation Basis and Evident Neural Restriction of Muscles' Core Power-Rates and Ensuing Force Levels

James J. Perrine

Tucson, Arizona, USA

ABSTRACT

Perrine JJ. The Mechanical Energy-Generation Basis and Evident Neural Restriction of Muscles' Core Power-Rates and Ensuing Force Levels. **JEPonline** 2015;18(5):23-36. The biochemical parts of the complex process that enables skeletal muscles to develop useful force levels have been well studied and described. Numerous experiments have also been done on biophysical properties of muscles. Yet, still to be answered is how the miniscule forces developed by the muscles' tiny sarcomeres enable the substantial force levels that whole muscles develop under some types of loading. An axiom in physics is that forces develop only when mechanical energy is generated, and builds up at a rate that is higher than any rate the energy is transferred to a movable load. Clearly, then, muscles cannot develop forces or do dynamic work against loads unless enabling mechanical energy is first generated at adequate rates. In physics, the term "power" can denote either the rate work is done or the rate that mechanical energy is generated and/or transferred. Past experiments on the physical properties of muscles provided some useful data, but did not elucidate how mechanical energy is generated or how the rates it is generated determine muscles' dynamic force capabilities and force-velocity relationships under various loading conditions, or whether restrictions on those rates are imposed at times by neuromuscular systems. Consequently, modern texts on the structure, function, and mechanics of muscles provide little information on these basic mechanisms. This review offers a new, physics-based perspective on the actual function of the sarcomeres. It is not to generate any useful forces directly but rather to generate a rapid series of tiny impulses of mechanical energy that quickly accumulate under some but not all loading conditions, and thereby enable the buildup of useful force levels. Secondly, this review re-examines and compares data from past

studies of the force-velocity relationships of isolated and in-vivo muscles under various loading conditions and identifies indications therein that intrinsic limits and/or protective neural restrictions on the muscles' core, contractile power-rates likely had occurred. Lastly, it suggests protocols for some future studies that could provide useful, new information about the workings of these basic muscle and/or neuromuscular mechanisms that should be relevant to those engaged in basic research, and ultimately to those seeking the best methods of training muscles for specific activities.

Key Words: Sarcomere dynamics, Contractile power rates, Protective neural inhibition, Force and power-velocity relationships

INTRODUCTION

Prior to 50 yrs ago, almost no one used the term "power" in connection with muscles as anything but essentially a synonym for great muscle strength. A very strong muscle was a "powerful" muscle. Of course before then there was no easy way to directly measure the immediate level or maximum, instantaneous power output of an in-vivo muscle. The introduction of the Cybex isokinetic dynamometer in 1964 enabled muscle forces with respect to time and velocity, and thereby instantaneous power-rates, to be more readily tested and studied.

Although the interest in power capabilities of muscles has grown, there is still a tendency to regard power as a type of force, rather than a rate. For example, in muscle science literature, terms like "speed strength" are sometimes used to describe manifestations of muscles' power-rates. In physics, "power" denotes the rate at which work is done. But since work cannot be done without a supply of enabling mechanical energy, power also represents and often quantifies the rate that mechanical energy is generated and/or transferred. Accordingly, the term "power-rate(s)" will generally be used in this paper instead of simply "power", to make clear that (notwithstanding that the formula for instantaneous power-rate is force times velocity) the term does not denote an amount of force, but rather the rate(s) that enabling mechanical energy is generated and/or transferred.

Force develops when mechanical energy is confined and builds up during loading events. While the interpretation and significance of the power-rate capabilities of muscles may not have been fully appreciated before, it is recognized that simple strength measures are often not adequate to predict the functional capabilities of muscles (especially since many functional activities and most athletic actions require relatively fast movements and/or quick applications of force). In fact, many athletic trainers and physical therapists are now employing various exercise techniques that call for faster movements of sub-maximal weights and/or quicker applications of loads to improve the "power" capabilities of particular muscle groups.

Several studies have been conducted to see whether particular "power training" techniques are more effective than strength training for improving scores in "power" tests, and/or performance in various functional activities (3,6,14,23). The results have ranged from no to yes or both types of training were needed. However a number of governing factors should be considered when seeking to measure or improve the actual power-rate capabilities of muscles. These factors include the type and size of the loads imposed, the relative velocities attained (and perhaps how quickly they are attained), the contractile time durations involved, and the skill of the subjects.

Muscle power-rates are not as simple to measure properly as strength. Yet, not only are the power-rates of muscles important functionally, it appears that timely restrictions of the muscles' core power-rates may be how the neuromuscular system guards against potentially injurious outcomes. The

formulas used to quantify the power output rates of mechanical systems can also be used to quantify the power output rates of muscles. But, to appreciate the relevance of muscles' power-rates, one must have an understanding of their mechanical-energy-generation basis. To elucidate the basis of muscles' power-rate capabilities, and how they enable both rapid force developments and dynamic force levels, a new perspective on the mechanical-energy-generation and buildup process, which physics laws dictate must take place first within whole muscles, will now be presented. This basic process was described in part previously (17-19).

The Mechanical-Energy-Generation Basis of Muscles' Power-Rates

The ability of skeletal muscles to convert chemical energy into mechanical energy is grounded in the dynamics of their sarcomere units. The basic structure of sarcomeres has been known for some time, but the exact way they operate continues to be analyzed by researchers. Discussions of the specific structural details of sarcomeres, and their theoretical operating modes can be found in recent reviews and texts (12,15,24). It is apparent that most models try to envision how sarcomeres can function as "force generators". That is, how do the sarcomeres directly generate sizeable constituent forces, and together with sarcomeres located in other muscle fibers, create and transmit to tendons the high, dynamic force levels achieved by whole muscles? Interestingly, there is no mention of the need for mechanical energy to be generated first, even though the "laws" of physics require that for such dynamic force levels to develop, enabling mechanical energy must not only be generated first but also at adequate rates.

Hence, it might help to illuminate how sarcomeres physically function if consideration is given to how they collectively enable whole muscles' power-rate capabilities. Looking at the structure of sarcomeres from this standpoint, it appears they are suited to converting chemical energy rapidly, but just incrementally into tiny impulses of mechanical energy. Upon excitation by a volley of discrete electrical potentials, local chemical energy substrates are split so that a rapid series of tiny, tractive mechanical energy impulses are created. The forces produced by the energy impulses are likely extremely small. In fact, the force exerted by the myosin motors of individual sarcomeres has been calculated to be ~6 pN (16). However, each energy impulse is obviously sufficient to cause the sarcomeres' actin and myosin filaments to overcome local visco-elastic resistance and overlap slightly by a still not fully understood mechanism, thereby doing a tiny amount of work, and drawing-in their "Z line" borders by a slight amount. Each energy impulse is also very brief, yet the slight overlappings and internal shortenings are progressive. Some models envision that filament cross-bridges keep the overlappings from slipping back between the tiny, cyclical impulses of tractive energy so the overlapping is "ratcheted up". But if local viscous resistance also slows slippage of each incremental overlapping gain, and the excitation frequency activates successive tractive impulses quickly enough, an actual ratcheting action may not be required. In any case, the overlappings rapidly progress, and the tiny, cyclical impulses of tractive mechanical energy quickly accumulate as they are absorbed by, and stored in elements of a series elastic component within the sarcomeres, and/or the myofibrils or fibers in which the sarcomeres are located.

In concentric contractions, the myriad tiny impulses of mechanical energy generated by the active sarcomeres (in series, parallel or pinnate fibers) in a whole muscle, and stored in the various elastic elements, become combined and transmitted to attached tendons. While this discussion is concerned with the mechanical energy that muscles can generate and buildup on their own, it should be noted that mechanical energy can also be added from external sources during impulsive eccentric loadings. Apparently, it is stored (briefly) primarily in the elastic portions of muscle tendons (5). During any loading event, the total amount of "potential energy" stored in a whole muscle-tendon unit at any moment, and not transferred to some external load, creates the useful tension (force) that is

manifested and exerted externally. It could be that the absolute maximum force level a skeletal muscle can achieve is not limited by the strength of the cross-bridges between its sarcomere actin and myosin filaments, but rather by myosin motor kinetics (20) and/or the frequency at which motor neurons deliver the requisite firing stimuli.

Regardless of exactly how it occurs, the aspect of a muscle's own mechanical-energy-generation process that is functionally important is the rate at which it occurs. It can be assumed that when a muscle is contracting and working just concentrically, the rate that all of the muscle's sarcomeres collectively can or are allowed to generate mechanical energy, and apply or transfer it via attached tendons and skeletal segments to an external load ultimately determines how quickly the muscle develops force and how well it maintains a useful level of force against a moving load if loading velocities reach relatively high rates and the energy is being transferred to the load at relatively high rates. This internal work-rate/power-rate may seem at first to not be applicable to a "static", isometric contraction, because there is no external work/power output. But, dynamic work always must be done at some rate within sarcomeres to first generate and then continuously replace internal losses due to viscosity and friction. Therefore, the mechanical energy needed to develop any useful tensions even in isometric contractions, that is, the aggregate, energy-generating work-rate achieved by sarcomeres, determines the time-rates and amplitudes of force developments by whole muscles in both isometric and dynamic contractions.

In summary, muscle power can occur and act in two stages. First, a primary stage where mechanical energy is generated at some aggregate rate and only transferred internally to storage elements during isometric contractions, and second, except during isometric contractions, a secondary stage where the energy is also transferred to a movable, external load. A whole muscle's aggregate, internal, mechanical-energy-generation rate can be called its contractile power rate, or when useful, simply its contractile intensity. Whatever it is called, that core rate, subject to any neural restrictions imposed on it, determines the muscles' functional, time-based and dynamic force capabilities under given load conditions, and thereby their force-velocity relationships.

In-vivo muscles' core, contractile power-rates cannot be measured directly. Although muscles' time-rates of force development (either in isometric or dynamic, concentric contractions) are determined by their core mechanical-energy-generation rates, these force buildup rates only reflect the relative contractile power-rates a given muscle has attained internally in specific contractions. However, thanks to the physics law of conservation of energy, that core mechanical-energy-generation rate can be assumed to be almost fully manifested by the external power output rates attained in dynamic contractions. With suitable instrumentation, those rates can be measured and quantified in either of two ways.

Instantaneous power-rate is given by the product of force and velocity at any moment. Average work/power-output-rate during a single or multiple contractions is given by the respective amount of work done, divided by a unit of time. When testing muscles' instantaneous power-rate capabilities (e.g., when assessing a muscle's ability to support quick joint-stabilizing actions or high-speed athletic movements, or determining the effectiveness of power training regimens, or studying the effects of neural inhibitory mechanisms), measurements of its instantaneous or average work-rate/power-output-rate in just one or two contractions would be least prone to high-power-rate endurance limits, which appear to occur very quickly when muscles work at high contractile power intensities, based on observations by the author during isokinetic dynamometer testing (19). This specific endurance capability deserves further investigation.

Indications of Intrinsic Limits or Neural Restrictions on Muscles' Core, Contractile Power-Rates in the Data from Studies of the Force-Velocity Relationships of Muscles under Different Loading Conditions

Physical evidence of the way core, contractile power-rates were developed and managed was contained in the data of four earlier studies of the force-velocity relationships of muscles. But, the evidence in each study appears to have been missed or misinterpreted. First, there were the evident but little noted indications of intrinsic muscle power-rate limits attained in the classic and still pertinent experiments on isolated, maximally stimulated muscle preparations by A. V. Hill (7, 9). Then, there were the findings of the first and so far only in-depth study of the force-velocity relationship of in-vivo muscles under (now common) accelerated-weight loading by D. R. Wilkie (22). Later there were the overtly different findings of a study conducted by this author and V. R. Edgerton (19) on in-vivo muscles but under a different type of loading and dissimilar loading conditions.

In 1938, A. V. Hill conducted his classic experiment to determine "the heat of shortening and dynamic constants of muscle" (7), which provided data on the force-velocity relationship of in-vitro muscle preparations stimulated to "maximal tetanus", and working against different amounts of inertial mass. Thirty years later (1968) he conducted a similar experiment, but this time used a constant-velocity, after-loading method (9). As before, he found that the force-velocity data were very consistent with his previously determined "characteristic equation" (F + a) (V + b) = a constant, which displayed in graphic form does not form just a "hyperbolic curve" as it is commonly described; it is a rectangular hyperbola wherein the product of the x and y values at (most) every point is constant. The two a and b factors were included so that the characteristic equation would fit his force-velocity data, and represented the energy liberated as both the measured "heat of shortening" and mechanical work done during dynamic contractions. Note that irrespective of the specific a and b values, the relative product of force and velocity at most points (i.e., instantaneous power output rate) was constant (internal friction and viscous resistance may explain the drops near curve ends). So, it seems very likely that Hill's maximally stimulated muscle preparations had attained, and were generally manifesting their maximum contractile power-rates. Hill obviously was familiar with power measures; he commented briefly on "the greatest rate of doing work" near the end of his write-up on the 1938 experiment (7), and referred to "power" specifically in a 1964 paper (8). Yet, curiously, near the end of his write-up on his final (1968-1970) experiment, when he wondered and discussed, "Is the force-velocity relation an instantaneous property of muscle?", he did not note the uniform/constant power output result or the possibility that it represented an intrinsic, maximum power-rate capability. Certainly this maximum mechanical-energy-generation-rate evidence should be considered when contemplating the actual function of sarcomeres and how they carry it out.

In 1950, D. R. Wilkie reported on an in-depth study he did on the force-velocity relationship of in-vivo skeletal muscles (22). He recorded the final velocities attained by in-vivo muscles when they forcibly accelerate progressively smaller weights. However, it appears that Wilkie (22) had no way to directly measure the instantaneous amounts of force his subjects' muscles actually developed. When any mass is accelerated, the force developed at any instant is given by the formula: force equals mass times acceleration. When a weight is not just lifted but is purposely accelerated, the applied force must equal the weight, plus whatever additional amount of force is required to achieve some immediate rate of acceleration. So clearly the tensions created during movements of Wilkie's weight-loaded "isotonic lever" (as he called it) were not in fact isotonic (meaning constant tension); and the force values in Wilkie's experiment represent the average amounts of force that were developed to achieve the final velocities reached with each weight load (22). However, there is little doubt that Wilkie's study (22) still usefully determined the general force-velocity relationship of in-vivo muscles when accelerating weights. Notably, Wilkie (22) stated beforehand that he expected his force-velocity

data from in-vivo muscles would indicate that “the degree of excitation is constant”, and not “a property of the central nervous system” and, then, concluded (when comparing his results with Hill’s data from isolated muscle preparations) “It is clear that the characteristic equation gives a good description of the corrected experimental results”. However, it is valid to now question Wilkie’s conclusion that his weight-loaded in-vivo muscles were not affected by neural inhibitory mechanisms. As will be explained, it is possible their contractile intensities, while constant, were being limited to a sub-maximal, constant level, and thus were not as high as those attained by Hill’s isolated muscle preparations, which obviously lacked any neural controls.

Thirty-seven years ago (in 1978), the author conducted a force and power-velocity study with V. R. Edgerton. Our paper was entitled “Muscle force-velocity and power-velocity relationships under isokinetic loading” (19). The findings, which differed from Wilkie's (22), were reviewed 6 yrs later at the 1984 McMaster International Symposium on Human Muscle Power (18). In the discussion session following that presentation, the questions by the symposium attendees mainly reflected two concerns about its validity: (1) Can the force-velocity relationships exhibited by skeleton-attached, in-vivo muscles be meaningfully compared with those measured directly with or from isolated muscle preparations? (2) Why did our findings with in-vivo skeletal muscles differ from those determined previously by D. R. Wilkie (22)? Both concerns still need to be addressed.

Regarding the first concern, apart from any neural influences, the tension (force) developed by an in-vivo muscle that is transmitted via attached tendons over or around a joint to a skeletal segment could be expected to be affected by intervening mechanical factors that vary over a range of movement. These primarily result from changing tension vectors caused by particular muscle-limb architectures and joint friction forces. But, the effect of varying tension vectors (as well as a muscle’s length-tension variation) can be largely excluded in studies of muscles’ force-velocity relationships by measuring the forces that are developed at various velocities at a specific joint angle, or at least by determining the average forces developed over the exact, same range of movement at various velocities as Wilkie (22) did. Joint friction forces are believed to be very small in relation to muscle forces (at least in young, healthy subjects). Thus, it seems unlikely that joint friction would significantly affect the forces developed and externally manifested by in-vivo muscles at different velocities, and thereby significantly obscure their general force-velocity relationship.

Regarding the second concern, the findings of our study on in-vivo muscles have been cited often, and summarized in published reviews of the various force-velocity studies. But virtually all of them have characterized our findings as showing only a small difference (just in the forces developed on the low end of the velocity scale) in what Wilkie (22) had found for the force-velocity relationship of his subjects’ in-vivo muscles.

However, there are some important distinctions between our study and Wilkie’s that appear to have not been appreciated before. Our study (19) did not represent just a more direct way of measuring how much force in-vivo muscles can develop at various velocities, nor did it find only a small difference in what had been found earlier by Wilkie (22). In our study, in-vivo skeletal muscles revealed how much force they can develop at various velocities under isokinetic loading, where any further acceleration once under load was prevented by an isokinetic dynamometer. Wilkie’s earlier study (22) had determined how much force in-vivo skeletal muscles can develop at various loading velocities, when the loading means (in his case hanging weights) do not prevent accelerations, and indeed were necessary and encouraged for his experiment.

When looking at graphed displays of Wilkie’s findings (22) and ours (19), a marked difference can be seen. His force-velocity data showed that when trying to accelerate progressively smaller weights to

as high a speed as possible, calculated (average) force capability immediately and rapidly falls off from its maximum at zero velocity. From the start, it does appear to follow a generally hyperbolic curve, seemingly consistent with what A. V. Hill had earlier found for isolated muscle preparations. However, our study (19) found that under isokinetic loading, maximum force capability can be attained not only at zero velocity, but even when an isokinetic dynamometer's controlled loading velocity has been increased until (reasonably fit) muscles are contracting at velocities perhaps as much as 25% or more of their usual, loading/shortening-velocity range. Not until muscles are contracting at velocities above about 50% of their likely, usual loading-velocity range does the force-velocity relationship appear to closely follow the rectangular hyperbolic curve found for maximally stimulated muscle preparations. In other words, under isokinetic loading, the point on the velocity scale where in-vivo muscles appear to finally reach their intrinsic, maximum, contractile power-rates has shifted considerably to the right. See Figure 1 to view the general force-velocity relationships found by each of the three studies displayed side-by-side. While the force and velocity axes of this composite graph are necessarily relative, absolute values are not needed to see (among other things) the marked difference between the low-velocity portions of the general, in-vivo muscle force-velocity relationship found under controlled/constant speed loading, and that found under accelerated-weight loading.

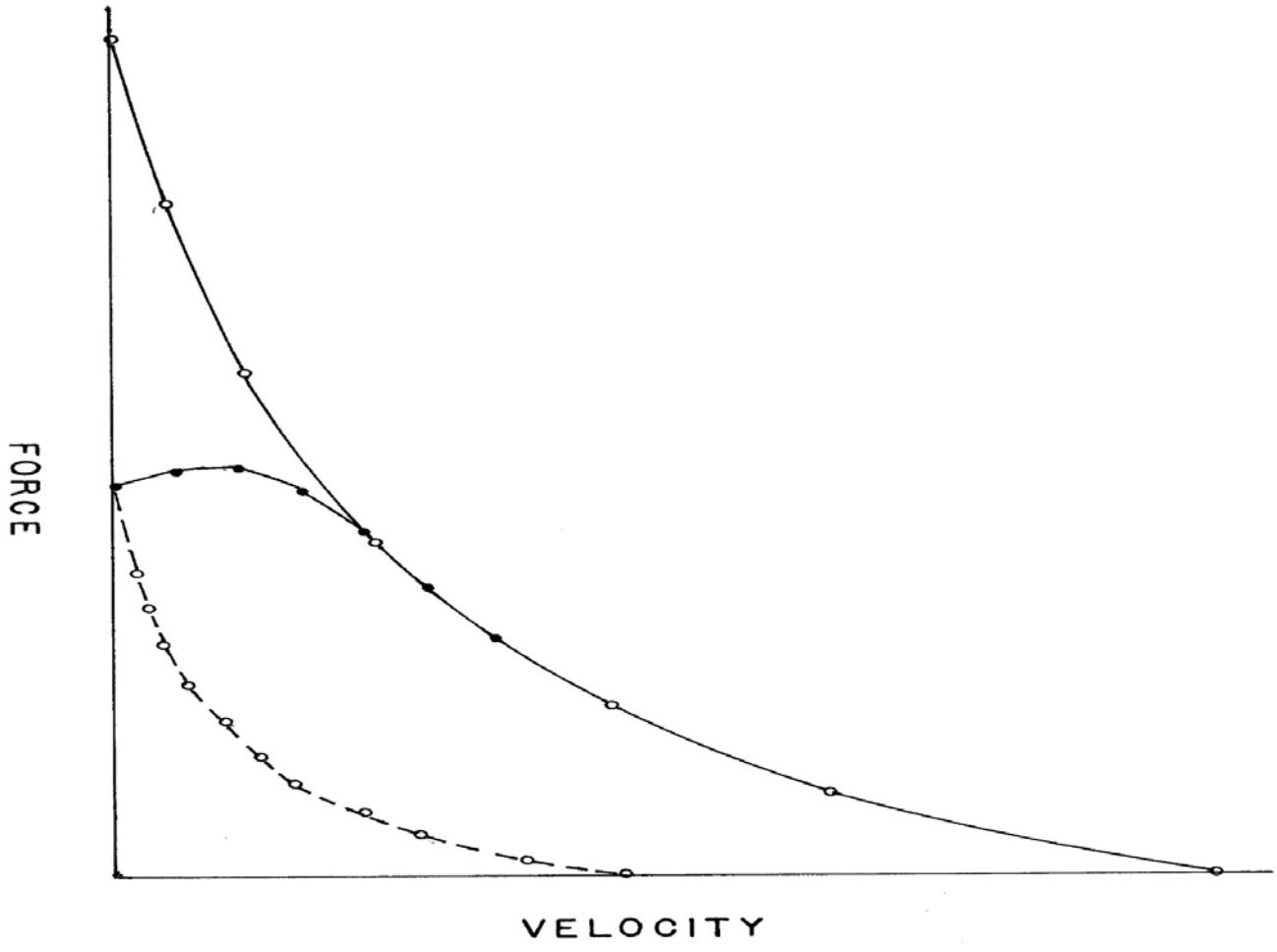

Figure 1. General Force-Velocity Relationships of Isolated Animal and In-Vivo Human Muscles as Determined in Three Separate Experiments under Different Loading Conditions.

Solid curve drawn through open circles from data on isolated, maximally stimulated animal muscles determined in 1968 and reported by Hill in 1970 (9). Solid curve drawn through dots from data on in-vivo human muscles under isokinetic loading reported in 1978 by Perrine and Edgerton (19), scaled and positioned to yield the best fit with the isolated muscle curve. Curve drawn with dashed line from data on in-vivo human muscles under accelerated-weight loading as reported in 1950 by Wilkie (22), with velocity and force axes reversed, and scaled and positioned so that the isometric force value coincides with the corresponding value observed in the Perrine and Edgerton study (19).

It should be noted here (as we did in the 1978 paper) that although two other researchers, Komi in 1973 (11) and Thorstensson in 1976 (21) also obtained in-vivo muscle data under controlled, constant speed loading, neither of them found anything inconsistent with Wilkie's (22) conclusion that in-vivo muscles have similar force-velocity relationships as isolated muscle preparations. The reason here is likely because of significant differences in our respective dynamometer testing methodologies. In our study, one important reason muscle forces did not immediately drop when tested at low to moderate controlled velocities is probably because, once we noticed that subjects had difficulty maintaining a truly maximal effort for more than about 500 msec, we instructed the subjects to delay the start of their maximal efforts until they were nearing our test position in the range of movement at the lower loading speeds (i.e., so that they would not have to maintain an all-out effort for more than 500 msec). Also, we used one specific position, 30° before full knee extension, for our force-capability determinations rather than simply peak torque, which is subject to substantial force-affecting time and position differences. Thirty degrees was both a functional knee position, and was sufficiently distal in the available range of movement to provide time for a muscle to develop and stably manifest its full force capability at each of the controlled loading speeds. The force measurements in Hill's final 1968 study (9) with isolated muscle preparations, where he also employed a constant speed after-loading method were always made at a specific muscle length and after a muscle had reached its full, stabilized tension capability at each velocity.

The Figure 1 comparison of our 1978 data with Hill's 1968 data on isolated maximally stimulated muscle preparations also offers evidence that an in-vivo muscle's force levels under constant-speed loading can be markedly restricted. Indeed it indicates that it can be to as little as 50% of its actual, intrinsic potential (at zero velocity). It seems a reasonable assumption that series Golgi tendon organs, which neurophysiologists have found can sense levels of tension and have an inhibitory affect on the motor neurons innervating the tensioning muscles (10) may prompt impositions of this overt force limitation. Hopefully, given the functional relevance of this evident inhibitory effect, future studies will determine whether it can occur with all kinds of loading and, perhaps, confirm that such substantial force restrictions are in fact associated with GTO activity.

It appears that the typical neuromuscular system has various ways to adjust or limit the immediate, aggregate rate of internal energy generation by whole muscles (and thereby the force levels that can quickly result if the energy is not transferred, and accumulates) by modulating firing frequencies, and/or the number, and synchrony of the motor units activated. It could be that the herein-delineated, maximum, core, contractile power-rate capabilities of muscles do not limit the force levels attained in typical maximal, voluntary/in-vivo, isometric contractions except, perhaps, on rare occasions when highly incentivized individuals may be able to "overrule" the evident neural limit on force levels and cause some of their muscles to reach their actual, intrinsic, contractile power-rate limits. Of course when energy is added from external sources in eccentric loadings, force levels could be expected to exceed any limits implemented by neural restrictions on the rate a muscle can generate mechanical energy itself.

Other than the evident neural restriction of contractile intensity to prevent forces from rising above "safe" levels in concentric contractions, is there another need to restrict contractile intensity when muscles are employed to accelerate only sub-maximal weight loads? Let's look again at the earlier comparison of our data and Wilkie's near-hyperbolic force-velocity data. In Wilkie's data, indicated power output (force times velocity) appears to have remained nearly constant over the tested velocity range; whereas, in our study, power output rates rose considerably as controlled velocities increased (and, with our subjects' knee extensors, first peaked at around 240°·sec^{-1}, refer to Figure 2). This indicates that under isokinetic loading that prevents accelerations once under load an in-vivo muscle's underlying contractile intensity can be higher at higher controlled loading velocities and, perhaps, be considerably higher than when muscles are forcibly accelerating sub-maximal weight loads. What causes this evident power-rate difference? There would seem to be two basic possibilities. When trying to accelerate sub-maximal weights, manifested power-rates remain constant with increasing speeds because: (1) they are partly counteracted and balanced by forces developed by contractions of antagonist muscles; and/or (2) antagonist muscles do not immediately contract (and keep observed power levels constant), but the contractile power-rates of agonist muscles is held in check when they are employed to forcibly accelerate sub-maximal weight loads.

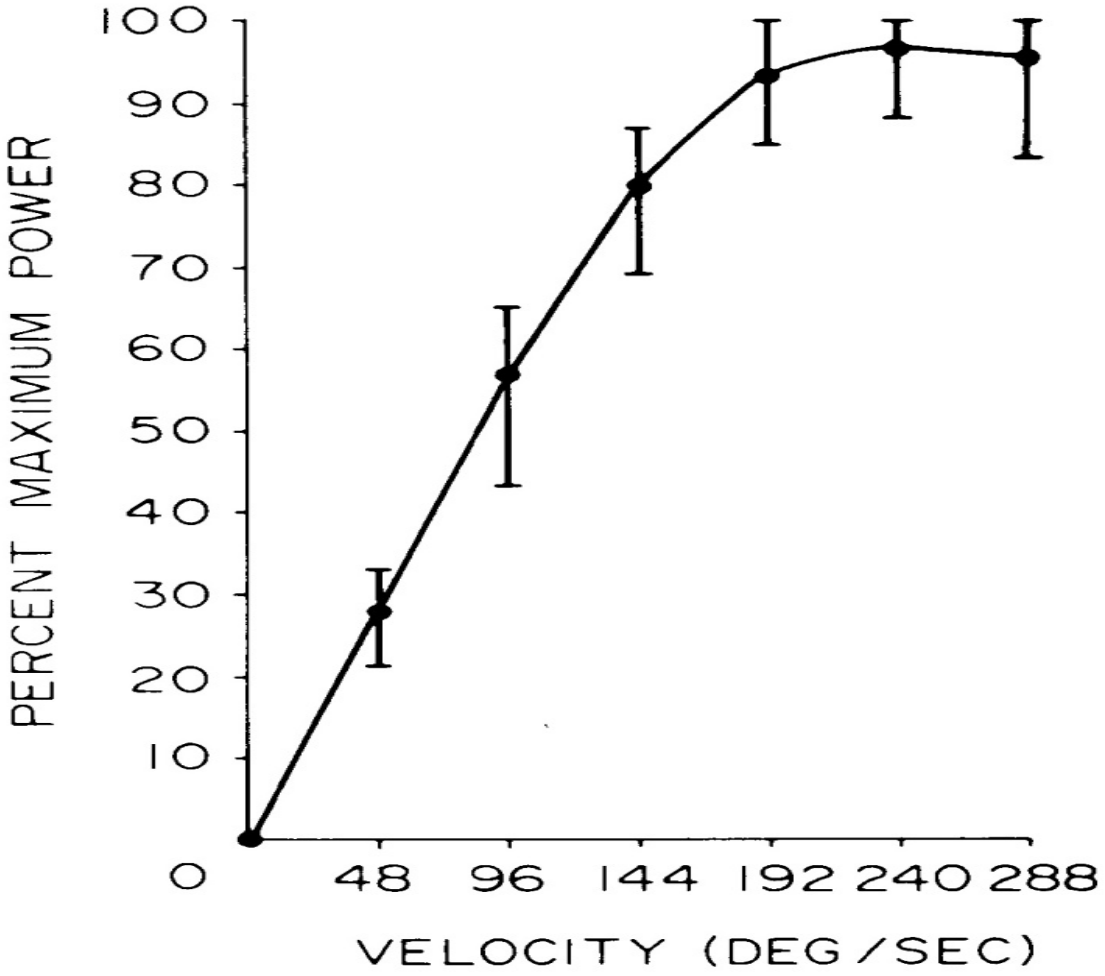

Figure 2. Power-Velocity Relationship of In-Vivo Knee-Extensor Muscles under Isokinetic Loading Determined in 1978 by Perrine and Edgerton (18). Dots represent means and vertical bars the range of the subject's muscles' maximum power-rate values.

As for the first possibility, neurophysiologists have known for some time that the structure of muscle spindles enables them to sense the rate that coupled muscles are lengthening, and deliver an excitatory signal to the motor neurons innervating those muscles (10). It has long been observed that quick stretches of muscles (e.g., by a tendon tap) can elicit a stretch reflex contraction. It has also been observed that antagonist muscles sometimes reflexively contract during some high-speed limb movements (e.g., those driven by powerful extensor muscles) conceivably to prevent a joint's ligaments from being damaged at the end of its range of movement. However, Wilkie's force-velocity data were obtained from elbow flexor muscles. If antagonists still contracted to slow movements before the end of the range, one would expect that the effect on the force-velocity relationship would not be uniform over the entire velocity range (i.e., an antagonist muscle presumably would not need to be immediately contracted at the lower velocities when ample time remained before the end of a range). Wilkie (22) reported that except in two records he saw no electromyographic evidence of reciprocal innervation of the triceps brachii muscles under the loading conditions or range of movement provided for in his experiment.

Thus, the second possibility that the contractile intensity of agonist muscles is held in check for some reason when they are employed to accelerate sub-maximal weights must be considered. The fact that the indicated power output rate was nearly constant in Wilkie's (22) experiment supports that possibility. That is, if the actual, instantaneous forces quickly attained against the heavier weights were (as appears to happen under isokinetic loading) limited to a safe upper level, and thus always reached that same level initially as the muscles began to accelerate the heavier weights, then, the indicated power-rate should have also increased. The best explanation for why it did not would seem to be that the contractile intensities (core power-rates) of Wilkie's subject's muscles could not increase with higher test velocities (so forces could continue at first to reach the force limit imposed by GTOs) because loads were in fact accelerating. This evident, acceleration-associated restriction will have to be investigated further, but neurophysiologists have found that spindle Ia afferents bifurcate in the spinal cord. In addition to exciting their enclosing (and lengthening) muscles to contract, they can achieve, through an inhibitory interneuron, reductions of the contractions of opposing muscles (10).

Also, a recent study by Dimitriou and Benoni (4) found that spindle Ia afferent discharge patterns are different with increasing (accelerating) rates of stretching. So, perhaps, when an agonist muscle is forcibly shortening and stretching opposite spindles just at a relatively high, controlled/constant rate, that condition alone will not elicit a spindle response that limits that muscle. But, an increasing rate of forcible agonist muscle shortening/antagonist lengthening might elicit a spindle Ia afferent response that does promptly limit an agonist muscle's contractile intensity. The purpose of this other restriction may not be to also ensure that forces do not eventually exceed a safe upper limit or to prevent joints from imminent injury, but perhaps to gain a preemptive "head start" on averting the possibility that a weaker antagonist muscle could be injured if and when it has to contract. For this new risk, the contractile intensity/power-rate of agonist muscles is also restricted by spindles located in antagonist muscles, and held to a constant level. As a result, the force levels, during Wilkie's (22) experiment fell right away in inverse proportion to velocity.

It may be possible to sense this preemptive contractile intensity restriction. For example, imagine this (subjective) experiment. You are pushing/pulling some simple (e.g., a free-swinging or rolling) mass load whose speed is perceptively increasing as a result. You can keep applying some muscle force against it, but can you sense you would have difficulty making an all-out maximum effort? If possible, compare that to the sense of pushing or pulling a load that is not accelerating as a result, like a slow-moving vehicle or an isokinetic-loading device. Doesn't the constant loading speed make it feel more stable, and thereby "safer" to make a maximum effort?

Apart from what this evident, acceleration-associated, contractile power-rate restriction may demonstrate about the protective functions of muscle spindles, the restriction may affect functional muscle contractions more often than the evident, maximum force restriction. Thus, it would be useful if a study could be done with in-vivo muscles using both weights and an isokinetic dynamometer to confirm whether instantaneous muscle power output-rates are in fact relatively lower when muscles work against accelerating loads, versus when they work against loading modes that prevent accelerations. For this new study, modern means of directly measuring the immediate forces developed against various weight loads (e.g., using strain gauge transducers) could be used to answer the following questions: What instantaneous force levels are attained when one tries to maximally accelerate progressively smaller weights? Are antagonist muscles in fact not immediately contracted? What are the actual, comparative magnitudes of the respective power output rates, and therefore the muscles' core, contractile power-rates?

The Pursuit and Promise of Muscle Power Improvements

As noted earlier, various studies have explored the relative effectiveness of different strength and “power” training techniques, and have gotten mixed results. The power training techniques involved working muscles concentrically against different, sub-maximal amounts of weight or eccentrically-concentrically against free-weights and/or masses. In 2008, the question: “Does an optimal load exist for power training?” was debated by Cormie and Flanagan (3). To address the question, two participants cited the results of various studies in which muscles were ostensibly loaded to different amounts of their maximal, isometric force capability. Essentially, both participants were considering how different training methods did or could alter an in-vivo muscle’s current force-velocity relationship(s) under certain types of free-weight or mass loading. But, it appears that no consideration was given then to what neural-inhibitory effects load-acceleration rates may have had on muscle power-rates in the studies cited.

It could be that muscle power-rate capabilities, like strength capabilities, can be improved by employing the “overload” principle (e.g., repeatedly working muscles so that they can develop relatively high if not their absolute maximum contractile intensities). Many studies have suggested that strength (maximal force) training methods work, first because they induce adaptations in a neural mechanism, and thereafter produce improvements in the size and makeup of muscle fibers. Training techniques that enable muscles to develop and maintain their maximal “allowed” contractile power-rate intensities against accelerating weight or mass loads may also induce adaptations in the postulated, spindle-mediated, power-restricting mechanism. "Plyometric" methods that seek to both reflexively and eccentrically boost contractile forces during quick and rapid stretch-shortening cycles seem to work, but it is not clear exactly how the two different aspects of those methods (the reflexive boost or the eccentric loading aspect) contribute to the observed improvements. While the energy added during impulsive, eccentric loading can drive force levels higher than during voluntary concentric loading, it remains to be seen whether those methods evoke any higher, underlying contractile intensities in the resisting muscles and thereby improve their power-rate capabilities better than ordinary concentric methods.

Isokinetic loading, by preventing accelerations, might permit muscles to safely attain their actual maximum contractile intensities and provide, when desired, for optimal muscle power-rate training. As noted earlier, our 1978 force-velocity relationship study (19) indicated that muscles can attain relatively high (perhaps, maximum) power outputs working just concentrically against isokinetic loading at sufficiently high controlled speeds. In addition, the 1981 study by Caiozzo et al. (1) on the effects of training muscles at two different isokinetic loading speeds provided evidence for force-

velocity training specificity. Training knee extensors at the velocity where we had earlier found peak torque (rotary force) to occur ($96°\cdot sec^{-1}$) resulted in the highest percentage gains at or below that velocity. Training knee extensors at the velocity where we had found peak power output to first occur ($240°\cdot sec^{-1}$) resulted in the highest percentage torque increases at and above that velocity (where maximum contractile power-rate development presumably is required).

The preceding discussions of muscle power-rates and training thereof (by any method) should not be seen as questioning the rationale for strength training. Adequate strength may be all that is needed to perform a particular activity. Also, as noted, regular strength training is believed to both temper a neural inhibitory mechanism and in time bring about an increase in the size and makeup of muscle fibers. Those adaptations would also tend to increase whole muscle power-rate capabilities so that performance of dynamic activities like running, jumping, and throwing could improve. In addition, as was previously noted, different neuromuscular mechanisms appear to affect force and power-rate capabilities depending on loading conditions. Thus, it is likely that no single training regimen would be able to improve both strength and power-rate capabilities optimally. Some combination of both types of training might be needed to optimally improve the performance of some activities. When a specific improvement of a muscle's power-rate capability is deemed to be an appropriate goal of the training, a practical question may be: "Would it be helpful to also improve the muscle's power-endurance capability to optimize the desired functional and/or sports gains?"

CONCLUSIONS

A new view of how muscles build up useful force levels by generating enabling mechanical energy was presented. The instantaneous, aggregate rate that essential mechanical energy is generated within whole muscles, which is termed their core, contractile power-rate, was identified to be a crucial factor underlying their ability to achieve usefully rapid force buildups and dynamic force levels under load and, thereby, their force-velocity relationships. Three earlier studies of the force-velocity relationships of isolated muscle preparations and of in-vivo muscles under two different kinds of loading were reviewed to see what they may have revealed about the way contractile power-rates were limited intrinsically, or by the effect(s) of the various neural inhibitory structures and pathways identified by neurophysiologists. Overt differences between the respective force-velocity relationships were identified. A side-by-side comparison of the power output rates estimable from the data of these earlier studies was presented. It indicates that the underlying contractile intensities/power-rates of the muscles were both limited intrinsically, and protectively restricted at times (apparently by two different inhibitory mechanisms) depending on the type of loading encountered. Both imminent force levels and the occurrence or not of risky load accelerations seem to be factors affecting whether or not neuromuscular systems restrict the contractile intensities/power-rates of muscles under particular loading conditions. Some governing factors to be considered when testing muscle power-rate capabilities or when seeking to improve them were identified. Basic protocols for clarifying research studies were also suggested.

Full disclosure: The Cybex dynamometer was invented by the author. All commercial ties to and royalties received from the manufacturer ended when the last patent on isokinetic loading and the associated devices expired 23 yrs ago.

Address for correspondence: James J. Perrine, 5330 N. Via Celeste, Tucson, AZ 85718, Email: jjperrine@icloud.com

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REFERENCES

1. Caiozzo VJ, Perrine JJ, Edgerton VR. Training-induced alterations of the in-vivo force-velocity relationship of human muscle. ***J Appl Physiol: Respirat Environ Exercise Physiol.*** 1981; 51:750-754.

2. Cormie P, McCauley GO, McBride JM. Power versus strength-power jump squat training: Influence on the load-power relationship. ***Med Sci Sports Exerc.*** 2007;39(6):996-1003.

3. Cormie P, Flanagan SP. Does an optimal load exist for power training? ***J Strength Cond Res.*** 2008;30(2):67-69.

4. Dimitriou M, Benoni BE. Discharges in human muscle spindle afferents during a key pressing task. ***J Physiol.*** 2008;586(22):5455-5470.

5. Finni T, Ikegawa S, Lepola V, Komi PV. Comparison of force-velocity relationships of vastus lateralis muscle in isokinetic and in stretch-shortening cycle exercises. ***Acta Physiol Scand.*** 2003;177:483-491.

6. Henwood TR, Riek S, Taaffe DR. Strength versus muscle power-specific resistance training in community-dwelling older adults. ***J Gerontology*****.** 2008;63A(1):83-91.

7. Hill AV. The heat of shortening and the dynamic constants of muscle. ***Proc R Soc Lond B.*** 1938;126:136-195.

8. Hill AV. The ratio of mechanical power developed to total power expended during muscular shortening. ***Proc Roy Soc Lond B*****.** 1964;159:319-324.

9. Hill AV. ***First and last experiments in muscle mechanics.*** Cambridge University Press, London, 1970, pp. 23-41.

10. Knierim J. Spinal reflexes and descending motor pathways. ***Neuroscience Online.*** 1997, Section 3: Chapter 2.

11. Komi PV. Measurement of the force-velocity relationship in human muscle under concentric and eccentric contractions. ***Medicine and Sport.*** 1973;8:224-229.

12. Komi PV (Editor). ***Strength and power in sport.*** (2nd Edition), Blackwell Science, 2003.

13. Kyrolainen H, Avela J, McBride JM, Koskinen S, Andersen JL, Sipila S, Takala TES, Komi PV. Effects of power training on muscle structure and neuromuscular performance. ***Scand J Med Sci Sports.*** 2005;15:58-64.

14. Lamas L, Ugrinowitsch C, Rodacki A, Pereira G, Mattos EC, Kohn AF, Tricoli V. Effects of strength and power training on neuromuscular adaptations and jumping movement pattern and performance. ***J Strength Cond Res.*** 2012;(12):3335-3344.

15. MacIntosh BR, Gardiner PF, McComas AJ. ***Skeletal muscle form and function****.* (2nd Edition), Champaign, IL, Human Kinetics, 2006, pp.151-175.

16. Piazzesi G, Reconditi M, Linari M, et al. Skeletal muscle performance by modulation of number of myosin motors rather than force or stroke size. ***Cell.*** 2007;131:784-795.

17. Perrine JJ. Isokinetic exercise and the mechanical energy potentials of muscle. ***J Health Phys Ed Rec.*** 1968;39:40-44.

18. Perrine JJ. The biophysics of maximal muscle power outputs: Methods and problems of measurement. ***Human muscle power*** (Proceedings from the 1984 McMaster International Symposium on Human Muscle Power). Human Kinetics, Champaign, IL, 1986, pp.15-25.

19. Perrine JJ, Edgerton VR. Muscle force-velocity and power-velocity relationships under isokinetic loading. ***Med Sci Sports.*** 1978;10:159-66.

20. Seow CY. Hill's equation of muscle performance and its hidden insight on molecular mechanisms. ***J Gen Physiol.*** 2013;142(6):561-573.

21. Thorstensson A, Grimby G, Karlsson J. Force-velocity relations and fiber composition in human knee extensor muscles. ***J Appl Physiol.*** 1976;40:12-16.

22. Wilkie DR. The relation between force and velocity in human muscle. ***J Physiol (Lon).*** 1950; 110:249-280.

23. Zaras N, Konstantinos S, Spyridon M, et al. Effects of strength vs. ballistic-power training on throwing performance. ***J Sports Sci Med.*** 2013;12:130-137.

24. Zatsiorsky VM, Prilutsky BI. ***Biomechanics of skeletal muscles.*** Human Kinetics, Champaign, IL, 2012, pp.131-213.