Strength training has a transformative effect on every physical system. Muscles hypertrophy and change the angle at which they exert their pulling force on the bones. Enzyme concentrations change, and hormones are released, triggering cascades of chemical reactions. The heart and blood vessels adapt to greater pressures, bone grows and becomes denser, and connective tissues become stronger and more able to handle the increased stress applied to them. Even the skin stretches, shrinks, and forms calluses where friction is applied. We have the barbell technology – we can rebuild you.

Eventually, though, structural improvements reach a point of diminishing returns. When sporting movements are over in an eyeblink and when a lifter, wrestler, or gymnast has to squeeze greater and greater performance out of the same body weight, can we accelerate performance by tapping the neuromuscular system? To see if that’s feasible, we need to know how the neuromuscular (NM) system works, how it’s measured, and if there is a way to upgrade this “mind-muscle connection” to get more power out of the same musculoskeletal hardware.

We all know technique is critically important, and it is unquestionably an element of the NM system. Reams of books and way too many pages of the internet have been devoted to finding the “perfect” way to squat/deadlift/curl/thruster/you-name-it. To get to the other half of the NM system though, the unconscious half, we first have to understand how it works.

**Back to school, back to school...**

Here’s the basic script: Whenever you need to contract a muscle voluntarily, the process starts in the cerebral cortex at the upper motor neuron (UMN). The upper motor neuron’s job is to tell a lower motor neuron (LMN, located in the brain stem or the spine) to activate or to stop. Every LMN has a threshold voltage that, when reached, triggers it to send out its own signal, called an “action potential,” down the length of its axon (which acts something like a power cord or information cable) to every muscle fiber it’s connected to. A single LMN and all the muscle fibers it innervates is called a *motor unit* (MU). The motor unit continues to generate action potentials until the UMN tells it to stop. The action potential’s final destination is the NM junction, which sits just up against the
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muscle without touching. When the NM junction receives the action potential, it triggers the release of acetylcholine into the space around the surface of the muscle, which can trigger a chemical cascade which generates a single contraction, called a twitch. If more force is recruited, the MU can generate multiple action potentials in quick enough succession that the muscle will not completely relax before it contracts again (the twitches “fuse”). When the muscle is at maximum sustained contraction, we are at a tetanic contraction – the absolute maximal force a motor unit can generate. The brain has 2 options as resistance increases, then: call more MUs to the fight (recruitment) or excite the MUs to generate more frequent action potentials (firing rate).

For voluntary contractions (ie: squatting), the UMN partially relies on the higher brain to know when to stop. However, excitatory/inhibitory neurotransmitters can affect the quality of muscle contraction outside of your conscious/central control. Caffeine, for instance, inhibits the clearance of acetylcholine after it leaves the NM junction, “priming” the muscle for action, and may have other neuronal effects [47][48]. Intramuscular sensors can also directly affect nearby motor units. The two sensory fibers most commonly referenced in regard to sports are the muscle spindle and the golgi tendon organ (GTO). When the muscle fiber is stretched, the muscle spindle sends a signal to excite the motor unit to help resist whatever is stretching the muscle. This stretch reflex is part of what gives you the “bounce” out of the bottom of a correctly executed squat. The GTO senses increased stretch in a tendon (caused by the strain created by an increased load). As tension rises up to about 50% of max contraction, the GTO sends a weak signal that inhibits that MU, but not strong enough to entirely shut down the contraction.

Knowing that basic script, these factoids may have an impact on our strength training:

1. All the fibers in a MU will be of the same fiber type.

2. #1 is important because all the fibers in a MU are plugged into the same LMN and receive the same signal. Because #1 is true, and the NM junction tends to be very similar within fiber types, the entire MU fires at the same time (the all-or-nothing principle).

3. The current evidence indicates that the proportion of the type of muscle fibers you have (fast twitch or slow twitch – Type 2 and Type 1, respectively) are almost entirely genetically predetermined [16]. A muscle will shift subtypes within types due to training, but if you’re born with a low percentage of fast-twitch fibers in a muscle, you simply don’t have the potential to contract that muscles as quickly as someone with a faster muscular genetic makeup. This also applies between the muscles in a single individual – the hamstrings, for instance, have a lower percentage of Type 2 fibers than the quads.

4. The conventional wisdom says that women have a lower proportion of Type 2 vs. Type 1 muscle fibers than men. The conventional wisdom isn't always right, though -- the literature isn’t very compelling either way [39][40][41]. Even if it has nothing to do with fiber type, though, women generally have a lower NME than men. Women have a lower 1RM at the same lean mass, especially at explosive sports (compare the current world record 56kg male weightlifting total to the 75kg female record total for an enlightening example). However, for various reasons, if a woman has the same 1RM as a man, her isometric endurance and lower-weight strength-endurance are probably significantly higher [42].

5. When an entire muscle group is stimulated and multiple motor units are required to complete a task, the slow-twitch muscle fibers (which have a lower threshold) will contract before the fast-twitch muscle fibers sequentially until the demand for force is met (with a few exceptions
NME for the Strength-Lifter beyond the scope of this article). For this reason, aerobic activities like cycling (or 100 burpees for time) only minimally develop Type 2 fibers and therefore power.

6. The signal from the LMN is binary in that sense that it is either on or off, firing or not. However, the signal’s firing rate, voltage, nerve conduction velocity (how fast the signal travels through the body), and the response of the muscle spindle and GTO all respond to training to different effect (we’ll discuss some of those later).

So... what?

It can be difficult to see the impact of these facts in real life because it feels like your muscles respond almost instantly, but if you measure the force an action generates directly (using a force plate or a dynamometer), you can see exactly how quickly it takes a muscle or movement to build up to its maximum. Plotted on a graph, you end up with a force-time (F-T) curve like the one below (Figure 1).

Imagine you’re sitting in a leg extension machine or sitting in a box squat over a force plate and try to drive the pad (or the bar) as hard and fast as you can. The solid line in this F-T curve might look like your force output. Times will vary between muscles and muscle groups, but a max effort contraction in the knee extensors (our example here) can reach around 90% maximal voluntary force in around 400ms, then it slowly crawls up towards maximum until fatigue or boredom begins to bring it down again [26]. NME seems to have a great deal to do with how much of our “true maximum force” we can generate in a voluntary contraction, but one of the main goals of improving neural drive is to improve one’s rate of force development (RFD) – the slope of this curve. Ideally, we would take an athlete’s F-T curve from looking like the solid line to the dotted line in the graph above. This may not look like a great change, but many sporting movements take less than 400ms to complete. The shot put, high jump, pole vault, baseball throw, and sprint support phase are ready examples [19].

Since the area under the curve before we lose contact with the ground/implement determines how far we throw or how much we can snatch, the thought is that we can drive performance by pushing the F-T curve to the left. For speed athletes, the benefits are obvious. For slow-strength athletes, the transfer is not as significant, but it is still real. Think of an arm-wrestler: between two equally strong wrestlers, the strong-faster wrestler will start off an inch or two ahead with the psychological and mechanical advantages that entails.
For an example we’re more familiar with, consider the squat: an initial RFD that provides just a 10% increase in angular velocity would open the hip and knee 2-3 more degrees in 200 milliseconds [17]. Since the knee/hip moments decrease as they open, a more “explosive” lifter will be able to use less overall muscular effort per rep to achieve the same work because they spend less time at a leverage-disadvantaged position. In other words, the “hole” in the squat is a cold, dark, God-forsaken place. The faster you get out, the better.

At this point, though, we’re still talking in the theoretical. What does coaching experience and valid research tell us about achieving that measly little 10% increase? If you’re considering NME alone… it’s not easy.

“Deedee, to the laboratory!”

Not all research on NME is valid for human athletes. For instance, some of the early ideas about NM function were derived from studies done on cats with their spines removed, and the resulting conclusions don’t always play out quite the same in vivo. With human subjects, many important studies have been done using single-joint movements on force plates and isokinetic machines. They’re not perfect, but they’ve taught us a few things:

**Signal Voltage:** Electromyography (EMG) reads an increase in local nervous activity by measuring voltage either directly via needle or through the skin (surface EMG/sEMG). Increases in the amplitude read by EMG in response to strength training are evident even before hypertrophy can be detected [29] and appear to have some impact on maximum force, but not nearly at a 1:1 return. For instance, a study on the effects of six weeks’ resistance training showed a 134% increase in EMG voltage, but max force on a knee extension only increased by 16% [18]. This makes sense. Increased recruitment = more MUs called into action = greater local electrical activity.

In general, though, we have to be cautious about information derived from EMG studies, especially sEMG. Dr. Sullivan went into detail on many of the ways the readings can be taken incorrectly in his 2012 *Sports Science Review*, but even when done correctly, EMG measures can be unreliable. One study found a 35% and 45% variability in mean and peak EMG readings when athletes attempted to generate MVC in isolated muscle groups over multiple attempts [24].

Consider also that hypertrophy tends to lower EMG activity for the same load. The bigger the muscle and more force it’s capable of generating, the fewer MUs are needed to meet the same demand. Also consider the “meaning of the measure” – Bret Contreras measured himself completing various exercises and found (using sEMG) that his BW chin-up generated ~50% higher mean and peak readings in the rectus abdominis than the same chin-up with a 90# weight added. He also found a weighted front squat generated greater spinal erector activity than an equally weighted good morning (where the moment arm on the lower back should be significantly greater) [25]. That’s not to say these studies are “bad” – the information may have been recorded perfectly, but consider what the data might mean (or not mean) before making a definitive conclusion.

**Incomplete Recruitment:** Trained lifters can work their nervous systems harder than novices on forced reps and at high intensities [30] in large part because a novice is unable to call every relevant MU into action. This is not to say, however, that a muscle is “only capable of using 60% of its power in the gym” (an oft-misused Zatsiorsky saying). In various muscles, researchers have found than an untrained lifter can be coached after multiple attempts to reach near-max contraction in isolation movements in
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a single session [26]. Using MRI, researchers recorded untrained lifters activating ~70% of the muscle fibers relevant to the movement on their first session, and various studies have shown that this gap closes with training, sometimes quickly [28] [30] [31].

Although maximum contraction may not be possible, one review in *Sports Medicine* found MVC often left only 2-5% on the table [36]. These numbers don’t imply that maximizing recruitment would provide a 28% increase in final squatting/jumping power, though. These studies were applied to isolated movements that don’t involve all the confounding factors of volume day squats. Still, they are a significant part of gains during the first few months of training.

**Firing Frequency:** As discussed earlier, to generate force without calling more MUs, the NM system has to trigger the twitches at a higher frequency to bring them to a tetanic contraction. This appears to be one of the NM system’s quickest adaptations, with increases of 15-49% being measured between 2 sessions of knee extensions done only 48 hours from each other [37]. With further training, the MU will fire in “doublets” at the beginning of rapid movements, potentially to jump-start the action. The effects were found to be significant each session for up to 6 weeks [34], providing a scientific basis for the claim that a 1RM test done within 6 weeks of a novice starting training is meaningless.

**Muscle Spindle and GTO:** We mentioned the power of the muscle spindle to excite connected motor neurons in response to stretch. The muscle spindles are what makes your knee extend when your doctor taps it with a hammer, and it has implications for strength squats, jumps, and other stretch-shortening actions. Luckily, it appears that it grows stronger after chronic high intensities, and its time to respond grows shorter [35].

The GTO gets a lot of press in the sports media because of its connection with “proprioceptive neuromuscular facilitation” stretching – PNF. Based on how it’s described, you would expect that the GTO would be attenuated by training. In the NM description, it seemed to act as a NM brake, right? Ride on it long enough and you should wear it down. Not necessarily. Currently, no studies have confirmed this. In fact, in some actions, the GTO excites the nervous system rather than inhibit it, and it appears to be entirely overridden during a max contraction anyway [36]. The GTO likely has more implications for rehab and PNF stretching than strength training.

**Inhibition/Excitation:** The NM system is constantly being excited and inhibited by different signals to allow for efficient movement. When a muscle contracts, inhibitory interneurons partially inhibit their antagonist to allow for more force in the desired direction. The antagonist, on the other hand, will eccentrically co-activate to stabilize a joint or slow down the movement at its end-range (preventing hyperextension) [43]. This co-activation pattern will change in different ways depending on the type of training. Power-specific training seems to decrease or maintain co-activation whereas traditional isolated exercises may actually increase co-activation [44], but I couldn’t find any research on this subject that was definitively convincing. To paraphrase one review, we’re not really sure whether the NM system will optimize integrity or force/speed in a given movement [29]. These interneuronal actions can even act outside of the MU where they started – action in a contralateral limb will excite some activity in its opposite limb, with implications for rehab and unilateral lifting [28].

Are these adaptations useful? Some of them are, certainly. Do they prove that neural drive is “the edge” in sports training or that it’s highly trainable? The evidence isn’t there yet. Some of the gains can be attributed to factors other than neural drive: one study from the *Journal of Applied Physiology* found a statistically significant improvement in neural drive for novices in the first 50ms of a max
effort leg extension after resistance training, but the effect size of the change was marginal compared to the other physiological adaptations that took place [18].

In summary, the research suggests that the performance improvements based on neural drive are positive, small, and some of them are tapped out early in the training progression. Still, none of these studies has tested anything relevant to the athlete on the field or the gym. How do we test whether someone has become more powerful in a sporting task? We jump. Why jumps and not the Olympic lifts? Let’s take a brief detour:

Unweighted jumps are the most powerful movements we can do. Even performed by skilled weightlifters, the clean and snatch generate less power than the vertical jump, which has been recorded at power levels of over 9000 W [19] (bonus points if you get the joke). Compared to the Oly lifts, jumps:

• Require less equipment, training, and space
• Are innate movements that require less technical proficiency
• Are more reliable between athletes (and within multiple attempts by the same athlete)
• Are more relevant to bodyweight sports (increased strength from muscle mass is in part tempered by the increased bodyweight)

The (Not-So-Simple) Jump

Before we discuss the studies involved, we have to look at what constitutes a “jump” in the research. The Journal of Strength Conditioning Research published a study evaluating various tests including these three: the countermovement jump (CMJ), squat jump (SJ), and Sargent’s vertical jump test (SVJ) [20]:

CMJ: athlete maintains hands on the hips and jumps off of a force plate with a voluntary countermovement (dip). Jump height is measured by calculation.

SJ: Same as CMJ, but athlete jumps from the “dip” position with no countermovement.

SVJ: Hands are free to swing during the countermovement, height is measured by the difference between the athlete’s highest jump reach and standing reach with neutral shoulders.

The SVJ is what most people think of when they think “standing vertical jump,” but the CMJ was found to be marginally more reliable and valid than all the others and thus dominates the research – something to keep in mind when some of these studies report unusually low jumps. Even considering their validity, vertical jumps are not an accurate pure test of neural drive. They are affected by many other factors. As a field test, though, they’re the closest we have, and we have a decent sampling of how trainable the vertical jump has been under research conditions (the summary follows, but refer to Table 1 where the data is referenced for the curious/nerdy).

The studies ranged the field in terms of relevance to our question: Some training programs [2] [11][46] were pretty well documented, progressive lifting programs with heavy weights. Others [6] did not record the athlete’s training, allowing them to prepare for their sport ad libitum. Measurement methods were usually well described, but sometimes not [5][16]. Study durations were relatively short, the longest being 12 weeks, so the end-trainability of the SVJ wasn’t directly recorded (though we can infer some things). Few studies included a diagram demonstrating individual subject’s improvements,
Table 1.

<table>
<thead>
<tr>
<th>试样</th>
<th>CMJ</th>
<th>SVJ</th>
<th>OLY</th>
<th>OLY</th>
<th>TW</th>
<th>PT</th>
</tr>
</thead>
<tbody>
<tr>
<td>10/17</td>
<td>Mean: 25.6 +/− 4.6</td>
<td>Mean: 39.9 +/− 3.8</td>
<td>Mean: 34.6 +/− 4.2</td>
<td>Mean: 40.3 +/− 4.6</td>
<td>Mean: 35.5 +/− 4.2</td>
<td>Mean: 51.0 +/− 5.7</td>
</tr>
</tbody>
</table>

**Note**: The table above lists the results of various strength and power measures for different groups. CMJ, SVJ, OLY, and TW are different measurements used in the assessment of athletic performance, with CMJ referring to cm jumps, SVJ to standing vertical jumps, OLY to olympic lifts, and TW to total weight. The mean values are presented with their respective standard deviations (±).
even in studies with small sample sizes. In several studies [1][44][45], the control group showed significant improvement over pre-test baselines, which suggests possible confounding factors (age/growth effects, retest reliability). Despite the challenges involved, a few trends stand out:

1. Plyometrics, Olympic lifting, and strength training at high volume/intensity consistently improve the vertical jump better than simply training for the sport.

2. Whereas maximum strength may retain or decay slowly, vertical jump improvements are lost fairly quickly without continued jumping work.

3. 10% improvement in 12 weeks is a reasonable gain among trainees who are not very practiced in jumping.

4. The novice effect applies – practiced jumpers don’t tend to improve SVJ quickly or to a great extent. Those with high-end vertical jumps improve less in training.

5. Normal weight loss (<10%, such as would be common when cutting for an athletic event) improved jumps by a small margin, but if the weight loss negatively affects your hydration status or athletic readiness, you may be worse off than you started.

6. The highest single data-point increase I found (except in [16], whose methodological failures lead to question its validity) was 17% in 12 weeks.

In *Practical Programming for Strength Training*, Rip states: “A guy with an SVJ of 10 inches will never improve this ability much more than 25%, no matter how hard he trains.” [22] This is, based on the research, a fair statement. Although most athletes will train for longer than 12 weeks, gains in the studies I read which conducted intermediate measures of growth showed a slow-down of increases by 12 weeks. There will be outliers. If a contestant in “The Biggest Loser” measures their SVJ pre-and-post show (~0″ to age-group normal), gains will be phenomenal. The same applies to less dramatic situations: imagine a novice who hasn’t done max jumps since his recess days decides to test his SVJ (Why? “It’s the WOD”). If he practices the movement, trains jumping strength, and applies event-specific tricks to improve day-of-performance [21] when the WOD comes up 6 months later, I wouldn’t be shocked if his jump improved 25-30%. If his second test methods doesn’t exactly reflect the first test (say… he retracts his shoulder in the standing reach), you might see even more. The statement still holds. If the test is performed correctly, training alone is highly unlikely to generate much more than a 25% increase. Combined with the studies we looked at early, NME will likely account for less than 25% of those gains. What’s in your genetic cards will decide your SVJ max, and them’s the breaks.

**Practical Recommendations:** NM exercise physiology is a relatively new field that is rapidly expanding, and any and all discussion is more than welcome. Nothing I’ve written here is definitive, though I hope it’s been useful at some level. My coaches and bosses always told me to end every brief with a real-life take away, though, so here are the highlights:

1. It is relatively easy to spot athletes who are either very efficient or the opposite (those Rip affectionately refers to as “motor morons”). The hardest ones to tell are those who are technically skilled through practice but naturally inefficient. Personal experimentation/history will tell best, but here are a few rough guidelines to check for low NME:

   a) Test your SVJ. Give yourself a good warmup of light, quick squats and practice jumps.
Jump 3 times and take the highest. You might have low NME if you are in the “below-average” category for your peer group (<16in. for adult males, <12in for adult females).

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b) If your technique in the power clean is good and you can only power clean ⅓ of your DL. Regardless of what the test says, the next 2 items are only starting suggestions. Log your training and adjust it as needed – how your body responds to training will always be more important than what any test says.

2. At the same relative effort, a lower-NME athlete will produce less stress on their muscles, bones, and connective tissues than a more efficient athlete. Since adaptation occurs in direct response to how much stress is applied, this is one of the reasons two people on the same percentage-based program can get different results. If sets of 5 (especially in the heavier lifts like squats and deadlifts) aren’t providing enough stress to drive growth, this may be the reason. Consider increasing total volume (say, 2x5 DL rather than 1x5) or increasing intensity and keeping volume stable (5x3 instead of 3x5) while carefully monitoring how your body responds. Some Starting Strength coaches have found this to be a common modification for women, as they naturally have a lower NME than men (maybe not as universally low as we thought [27], again requiring training to be personalized, but still generally lower).

3. Highly efficient athletes can inflict incredible stress on their bodies. If you’re one of these lucky souls, you already know it. Monitor your stress closely and build good technique first. You probably respond to strength training faster than me [16] (I’m jealous of that, by the way), but your margin of error is slimmer at a heavy load because you’re operating at a higher percentage of your body’s “true max.”

4. Just because you were a top-performer at your “box” doesn’t necessarily mean you’re super-efficient. Strength-endurance and endurance performance are more dependent on lactate clearance, VO2max, pacing, and technique than NME. Don’t make a mistake I made early in my novice progression and build in more rest days than you need to because you have a 35 minute “Murph” time. That’s another mistake: doing Murph during your novice progression. Just don’t.

5. The SVJ can be a useful metric when used intelligently, but don’t treat it like a max effort circus trick or try to “top your last score” occasionally. You’re more likely to “game” it then and lose what value it had to begin with, unless you’re trying for The Combine or otherwise testing for placement in your sport.

6. If you are trying for The Combine, build your squat and deadlift strength, incorporate plyometrics into your training if appropriate, practice the jump (duh), and warm up with dynamic squats and jumps prior to the test. Avoid static and PNF stretching before the test – they tend to weaken high-intensity performance, including jumps, for a period of time after their use [32][33]. Don’t wear squishy shoes that will absorb power that should be transferred to the floor. Wear your lucky placebo socks. Every bit helps.

7. **Big point #1**: Take a look at Figure 2. In study after study, text after text, this is how the F-T curve responds to resistance training after a few months. This has been my experience and the experience of many others: when max strength increases, RFD increases. For a million reasons
(structural, neural, psychological), the stronger the athlete becomes, the more explosive he is, and at the novice level, strength = power. After a point, the transfer will slow down or stop, but specifically training for power is inefficient at best if you haven’t put in the time on developing basic strength.

8. Big Point #2: Finally, one last graph (Figure 3). This is what the previous curve looks like “normalized” (where every data point is compared to the max point in the data set). It represents the fact that, after training, we do tend to see an improvement in neural drive (a little of that “pushing to the left” we talked about earlier), but not much. In other words, unless you’re going to be a speed athlete or a top-level strength athlete, don’t sweat the brain stuff. Start by getting stronger and practicing your sport. You’ll get the biggest bang for your buck there. If you reach the point where the dashed line is the difference between you and the podium, then we’ll talk.

CJ Gotcher is an active duty Naval officer of 5 years and an amateur raw powerlifter. When he’s not picking things up and putting them down, you can find him geeking out on research reviews and games of all kinds. CJ is a certified NSCA-CSCS and Precision Nutrition Level 1 coach.
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