How Humans Move: The Neural Origins of Rhythmic and Discrete Movements

By Nick Korsantia

B.A. in Biology, Psychology, and Cognitive Science, Case Western Reserve University

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Thesis directed by

Donald O’Malley
Professor of Biology
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Abstract of Thesis

Humans have a wide behavioral repertoire ranging from locomotion, object-manipulation, communication, and much more. From the mundane to amazing bodily feats, the nervous system is responsible for controlling movement at all levels. In this literature thesis, the main purpose is to bring together and discuss scientific literature on the control mechanisms of the human movement system, specifically how rhythmic and discrete movements are controlled. Rhythmic movements are defined by being cyclical and periodic, while discrete movements are one-time movements with a well-defined beginning and end (Sternad and Hogan 2004). This type of definition focuses on the external factors of the movement, but misses the internal mechanisms responsible for achieving it. In order to address this and approach the movement system from a more comprehensive viewpoint, this review paper will examine rhythmic and discrete movements using an integrative approach from both a behavioral and physiological perspective. The behavioral approach will cover topics on degrees of freedom, synergies in the human motor system, as well as the context with which rhythmic and discrete movements are performed. The physiological approach will start with an overview of the anatomy of the motor cortex, supplemental areas of the motor system, and how these areas function together. Next, we discuss the descending and ascending corticospinal tracts and the involvement of the brainstem during rhythmic and discrete movements.
We will also discuss two original studies with pilot data on the stimulation of the motor cortex and the posterior parietal cortex. Finally, this review concludes with an overall perspective on how rhythmic and discrete control mechanisms are different and what role they play in human movement as a whole.

We hypothesize that rhythmic and discrete movements have different control mechanisms reflected in the different involvement of the nervous system in each of these movement types. Through our examination of the motor control literature, we find that rhythmic movements are highly associated with the activity of the brainstem and spinal cord, whereas discrete movements are more associated with cortical activity in motor areas of the frontal and parietal lobe. Although discrete movement commands pass down through the brainstem and spinal cord, there is evidence that these movements initiate rhythmic neuronal activity, likely caused by local central pattern generators. There is no evidence that discrete movements cause such activity. Additionally, discrete movements are associated with activity in the primary motor cortex, posterior parietal cortex, supplementary motor cortex, premotor cortex, as well as other bilateral cortical activations. However, rhythmic movements are primarily associated with activity in the contralateral primary motor cortex.
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Chapter 1: Introduction

The majority of organisms on the planet come equipped with some ability to move and manipulate the world around them. We might take it for granted, but arguably one of the most significant functions of the nervous system is the generation of movement. For humans, movement is achieved by the musculoskeletal system when activated skeletal muscles contract and generate force to pull on bones. Voluntary muscle activation is achieved by sending motor commands down from the brain to the spinal cord and finally to its connected muscle. The brain and spinal cord also serve a major control role to make sure the movement is smooth and achieves its goal, especially if adjustments are needed (Rosenbaum, 1980). Even the most basic movement, such as grabbing an apple, has many parameters that need to be controlled: the arm path to reach goal, the muscle sequence needed to achieve this path, the feedback of reaching the destination, and the sequence of hand movements to clasp the apple without crushing it. The brain and spinal cord are responsible for coordinating and controlling all these parameters and ultimately converting them into a sequences of muscle activations that complete the set forth goal. So how does the nervous system do this?
To begin, let’s consider two movement types that are ubiquitous in human movement: discrete, reaching-like movements and continuous, rhythmic movements. Every movement that you are able to make with your body is either discrete, rhythmic, or some combination of both and that alone begs the question: are these two movement types controlled differently? Externally, discrete and rhythmic movements look very different, even when the same muscle groups are involved (Degallier 2010). Regarding discrete movements, a football player raising his arm and preparing to catch a football is a much different feat than raising your arms to wave at a friend. Additionally, these two movements have very different contexts in which they are done and for what purpose they are done under. In the football example, the player must be aware of his arm’s position and trajectory as well as the target’s (the incoming football) position and trajectory. The player must make quick adjustments if he is to successfully catch the ball in this dynamic situation. It becomes clear that the visual system and proprioception, our subconscious perception of movement and spatial orientation, must be involved together.
with the motor system for the football player to achieve a successful catch (Smyth, 1982).
The person who is waving his hand is also activating similar muscles, but under very
different conditions. There is no implicit target when one is waving their hand in the air,
therefore there is less need for visually-guided coordination or spatial orientation.

The important aspect in simple rhythmic movements is the process: the rhythmicity itself
is paramount (Kugler, 1987). As many cyclical movements, such as locomotion, require
less attention to a targeted goal, the movement simply needs to be reliably consistent
and smooth. Some common rhythmic movements, such as reflexively scratching an itch
or restlessly moving your leg while sitting, can often go unnoticed. This is not typically the
case in discrete movements as there is high goal orientation towards target and necessary
sensory feedback needed to adjust trajectory. As there is nothing continuous in a discrete
movement and there is a high demand on reaching a particular target, discrete
movements have many kinematic characteristics that seem strikingly different to
rhythmic movements.

There are many apparent differences between discrete and rhythmic behavior, but it is
important to understand the underlying structures that give rise to these differences.
Physiological research over the last two decades on these two movements types found
that brainstem and spinal cord structures are important in ongoing rhythmic activity
(Schmidt, 2000). Much of this research has cited central pattern generators, which are a
collection of neurons that produce a rhythmic firing pattern, as essential in performing rhythmic movements. At the spinal level, the efferent neurons are the most proximal to their innervated muscles. Within cross-sections of the gray matter of the spinal cord, select groups of interneurons are arranged to produce oscillatory activity (Grillner et al 2005). These CPG groups form synapses with efferent motor neurons and allow the production of rhythmic depolarization of connected muscle, which results in rhythmic contractions and movement in those parts of the body.

Previous research has also shown that greater cortical activity was found during discrete movements compared to rhythmic ones (Schaal 2004). The showed more isolated activation in the primary motor cortex, or M1 cortex, in rhythmic behavior as opposed to the more diffuse activation during discrete movements in the M1 and other motor areas of the cortex, such as the supplementary motor cortex, premotor cortex, and posterior parietal cortex. Additionally, clinical research on stroke patients that were undergoing motor rehabilitation found that discrete movements were more affected than rhythmic ones (Leconte et al 2016). Since stroke generally does not affect deep brain tissue, this gives supporting evidence to suggest that discrete movement rely more heavily on cortical involvement, while rhythmic movements rely on lower brain structures, such as the brainstem. Due to the behavioral and neural differences in activation, we hypothesize that discrete and rhythmic movements have distinctly control mechanisms.
Some early theorists on complex movements found that many behaviors with a repetitive sequence likely are formed by grouping together several simpler elements of the behavior. This grouping of muscle contractions into functional rhythmic movements is one example of a muscle synergy. Muscle synergies can develop through innate physiological development, such as the generation of walking, or can be a new learned skill, such as juggling. Interestingly, many learned rhythmic movements begin as discrete movements that have been sequenced together (Tresch, 2009). As evidence shows that discrete movements often require high cortical activation and the communication between different areas of the cortex, how is it possible for rhythmic movements to be produced if they have a decreased amount of cortical activation? We suggest that muscle synergies are arranged and encoded in areas that are in deep brain regions, such as nuclei of the brainstem. We also hypothesize that as humans learn to perform new rhythmic movements, they begin by combining discrete movements and with practice a new behavior is learned. This model relies initially on increased cortical activation to produce willful discrete movements (Flash 2016). While practicing, the cortical activation is expected to decrease as the new muscle synergy is being learned. The muscle synergy is likely encoded in the brainstem allowing quick access to the sequence of muscle activations. The newly created muscle synergy is now a fully rhythmic behavior rather than a combination of discrete movements. As we review literature that supports the difference in control mechanism for these two movements, we will look back at this model and discuss its validity from the behavioral and physiological perspective.
Chapter 2: Rhythmic and Discrete Behavior

The external characterization of movement is a necessary place to begin our understanding of rhythmic and discrete motor control. Since the products of movement happen in physical space, where the performer and environment interact, it makes sense that early motor scientists primarily used behavioral paradigms and practices to infer principles that organized movement (Latash 2008). Many of these scientists worked not only to deepen an understanding of human motor principles, but also to use these principles in athletics and sports sciences to increase performance in order to gain a competitive edge. Early motor research also had far-reaching implications in the work force, especially in areas where complex tool manipulation was required, new heavy machinery was used, or when a high degree of precision was demanded, such as the case with master artisans.

2. 1 Degrees of Freedom and Redundancy

Early motor scientists used behavioral observations of movement to analyze the motor system. Nikolai Bernstein, a prominent Russian scientist working in the department of labor, made great strides in behavioral analysis and is considered by many today as the father of motor control. Bernstein often used cyclographic techniques (Fig 2) to track movement of athletes. By placing reflective markers on the athlete’s joints and taking
pictures in rapid succession, up to 200 frames per minute, Bernstein and his colleagues were able to examine the movement of the body at a high resolution (Bernstein, 1967). His research showed that most movements, like hitting a chisel with a hammer, are composed of smaller movements, dubbed submovements. Any deviation to these individual submovements would inevitably alter the movement as a whole. Bernstein concluded that the brain must be updating itself during the movement and sequencing its motor commands to ensure smooth continuous control of movement.

Bernstein and his colleagues also found that for every movement there are multiple ways that the body can conform its joints and muscles to achieve the same motion or same outcome. It was known that the number of muscles acting across a joint generally exceeds the number of kinematic degrees of freedom of that joint (Bernstein, 1970).

Fig 2 An example of the cyclographic data. High Speed cameras capture the light sensitive markers on the wrist and hammer and allows us to see the different trajectories that the blacksmith uses, while still achieving a consistent accuracy. (source: Bernstein, Nikolaj A. "The co-ordination and regulation of movements." (1967) pg105-110 open source database)
Bernstein took note of this redundancy: a given movement can be realized with an infinite number of muscle activation patterns and the CNS must be responsible for adequately controlling the many degrees of freedom of the musculoskeletal system to achieve this movement. Solving the redundancy problem can also be considered in the context of learning a new movement or skill: the performer must discover ways to organize the different independent degrees of freedom to achieve the task goal (Bernstein 1967). Looking again at the expert hammering work of the blacksmith in Fig 1, there are several swing paths that he takes to land a hit on the target. These different swing paths are controlled by several muscle groups in the shoulder and forearm and give rise to many degrees of freedom of the trajectory. However, the expert performer still manages to reproducibly hit the same target towards the end of the swing path. Bernstein proposed that humans acquire new behavioral skills by exploring these different degrees of freedom, fixing them, reorganizing them, and finally exploiting their passive mechanical properties (Bernstein, 1970).

2.2 Motor Synergies

Another interesting contribution of Bernstein’s was his observation of compensation in nearby muscles to make task-dependent motions less variable. This was apparent when an expert blacksmith raised and swung his hammer to hit a point target on an anvil. The muscles of the arm are coordinated in such a way that errors and variability in one of the
activated muscles is compensated for immediately by nearby muscles. This type of phenomenon is said to be dependent on learned motor synergies and is present only in expert performers and not novices (Bernstein, 1967). Synergies, literally meaning working together, ease the computational power of the neuromuscular system by arranging many effector muscles and sequences needed in a task under a smaller number of higher up elements. Rather than having the CNS directly communicate with all the effector muscles needed in a motion, Cortical commands can stimulate the activity of a select group of synergies that arrange muscle sequences themselves. Although over the next 50 years the physiological credibility of muscle synergies is still being debated, a large amount of evidence supports the hypothesis that multiple muscle units can be combined and activated together using organizing elements call synergies (Tresch, 2009). As illustrated in Fig 3, this arrangement at the synergy level creates a degree of flexibility, allowing the rearrangement and sequencing of many muscles.
effector muscles into individual units that can be called up at any time. Many motor
synergies, such an innate motor programs are present at birth (Grillner and Wallen 2004).
However, other muscle synergies are not intrinsic within the human body and practice is
necessary to create and reinforce them, meaning that synergies are necessary aspect in
motor acquisition and learning of new skilled movements (Latash, 2010). In each
example, muscle synergies also allow the covariation of the involved muscles to play a
role in error correction during a movement, allowing slight deviation in muscle activity to
be easily corrected for by other muscles involved.

Motor synergies are an interesting example of neuromuscular flexibility and adaptability,
allowing people to rearrange a host of muscular elements within a smaller number of
controlling elements to create a desired skilled movement. It is apparent that motor
synergies are not completely rigid as can be seen by Bernstein’s earlier work with
Blacksmith hammer swings (Bernstein, 1967). Each swing of the hammer resulted in a
slightly different trajectory, while still keeping a high end-point accuracy towards the
desired target. Swinging a hammer and reliably hitting a target is not a trivial task and
requires much training to learn this skill and form the necessary muscle synergies.
However, these muscle synergies must account for environmental perturbations (i.e.
weight of the hammer) and errors in processing from the neuromuscular system, which is
innately noisy (Faisal 2008). For this reason, established synergies must account for these
internal and external errors by correcting movement trajectories, especially towards the
of a complete motor pattern. A study, looking at the flexible nature of established synergies, was performed while using EMG recordings and saw that within each cycle of a movement, slightly different EMG patterns arose (d'Avella, Andrea, and Emilio Bizzi 2005). After reducing the EMG signal to its components, it was clear that each cycle of movement recruited slightly different motor units especially towards the beginning and end of the movement. Given that the neuromuscular system, like the rest of the nervous system, is filled with noise and errors in processing, synergies allow the user to have a learned pattern of muscle activations, while still allowing for corrections during the movement without compromising accuracy.

2.3 Rhythmic and Discrete Movements

Apart from submovements and redundancy, another external characterization of all human motions looks at two kinematically distinct movement types: rhythmic and discrete movements. Kinematically, we can identify movements as being rhythmic, discrete, or some combination of both. Rhythmic movements are continuous, cyclical, and often do not have a specific end point, while discrete movements are singular events with a distinct start and finish. Scratching an itch, chopping carrots, and walking are all examples of rhythmic movements, while some examples of discrete movements are: reaching for a glass, picking up a box, and kicking a punching bag. Given that these two classes of movements have very different kinematic features, it follows to reason that
there are different neural control mechanisms producing each type of movement. Three hypotheses have been considered: (1) rhythmic movements originate from combined discrete movement commands, (2) discrete movements originate from shortened rhythmic movement commands, or (3) discrete and rhythmic movements originate from separate control systems (Hogan 2007).

In addition to kinematic constraints set forth by the definition of rhythmic and discrete movements, there are also persistent contexts that each of these movements is done under (Phillips-Silver et al 2010). Take the example of chopping a carrot. We do not trouble ourselves with discretely making each chop and taking a break, instead we opt to make continuous cuts until the carrot is chopped, ensuring speed and increasing the consistency of each cut. In the same way, when we walk, we do not stop every time each foot hits the pavement and think about our next step, rather we continue through our gait until we reach our destination or hit an obstacle. The situation is drastically different when we want to grab an item within reach. There are no continuous movements, only one goal and the necessary muscle movements to achieve this goal. By and large, discrete movements are goal-oriented, require some degree of attention towards the destination, and require feedback to confirm that the goal is truly met (Marvin et al 1999). On the other hand, rhythmic movements are more process-oriented and require less feedback as the movement is regular and consistent (Kugler 1987).
2.4 Dynamic Motor Primitives

The human neuromuscular system involves the integration of many different anatomical parts, including cortical and subcortical brain regions, the spinal cord, peripheral nerves, muscles, and the joint and bones they act on. However, this system is anything but linear in producing movement and is updated and controlled at different levels. Given the inherent high dimensionality of the neuromuscular system’s capabilities, many scientists in the motor control community seek lower-dimensional building blocks or primitives as a basis for complex actions and to ease computational demands for the brain. Previous research posited three dynamic primitives as candidates: fixed-point attractors, limit-cycle oscillators, and mechanical impedance (Hogan & Sternad, 2012). For discrete point-to-point movements, there may be fixed-point attractor dynamics that allow goal-directed movement to approach their target. For rhythmic movements, limit-cycle oscillations may organize complexity into stable dynamic systems that repeat until an inhibitory stimulus is given (Hogan & Sternad, 2013; Sternad, 2008). The third primitive is mechanical impedance, which is a type of mechanical sense created by interacting with objects and the environment allowing for feedback and updating of online movements. These three primitives taken together could model a motor hierarchical system that is able to create rhythmic, discrete, and complex movements.
Submovements, as first recognized by Nikolai Bernstein, are smaller movements that together make up a larger movement. Oftentimes, submovements are used as a way to correct a trajectory that will miss the target. Most often, goal-directed reaching tasks require the performer to propel their arm towards the object and make any necessary changes in trajectory to successfully reach it. The first part of this discrete movement involves an initial submovement that generates the force necessary to reach the goal, but is not precise (Novak et al 2002). For this reason, there are larger variances in the initial part of a discrete movement as opposed to the latter half. To correct for the variance in the first submovement, there is secondary submovement that alters the course closer to the destined target. To make the second submovement, proprioceptive and kinesthetic feedback is necessary to make a corrective motor command and alter the trajectory to reach the target. This is different from rhythmic movements, which do not have any submovements within each cycle, marking a significant control difference between the two types of movements (Wisleder 2007).

In principle, all movements can be comprised of individual submovements in sequence, but biological evidence suggests that movements that are repeated originate from oscillations in the nervous system (Repp 2005). Rhythmic movements are generated from continuous signals that activate the muscles in a cyclical fashion. During steady-state rhythmic activity, there is less emphasis on the neuromuscular system in reaching a target and more focus on consistency and repetition of the action. This can be seen in simple
rhythmic movements such as waving your hands to say hello, there is no target to reach so the extra accuracy of corrective submovements is unneeded. The demand on the neuromuscular system is much greater when there is more emphasis on precise visual, proprioceptive, kinesthetic and feedback, such as when you catch a fly ball or kick a moving target in karate class.

Mechanical Impedance is the third primitive which has its basis in the physical interaction between performer and object (Hogan, 2013). At its very core, mechanical impedance relies on Newton’s 3rd law: every action has an opposite and equal reaction. This is clear when you catch a ball and the ball causes your arm to fling backward in the direction the ball was thrown. For this reason, our neuromuscular system must account for this force before it even occurs, otherwise we might not have enough grip and fail to catch the ball. Furthermore, when interacting with an object, like drinking from a cup of coffee, the performer must actively adapt their muscle activation in response to dynamic object interaction. In the coffee example, the performer must give adequate strength to their grip to prevent the cup from falling and alter this strength as they take a sip in order to account for the moving liquid inside the cup without spilling it. Even though this might sound like a trivial example, the neuromuscular system is burdened with many control parameters that must command, predict, and adapt to a changing environment in which we are interacting and also changing. If this is done too suddenly, too fast, or without coordination the performer will have a stain to clean up. To avoid this situation and other
situations where object manipulation is required, the mechanical impedance informs the
neuromuscular system on how to adapt to the changes happening at the cusp of the
performer and object and looks for an optimal solution (Burdet, 2001). Factors such as
object weight, shape, contents, consistency, texture all play a role in how we interact with
objects, both in predictive commands and online adaptations guided by sensory
feedback.

When combined together, these three primitives of movement, submovements,
oscillations, and mechanical impedance, can explain and characterize any movement and
its interactions. However, there is a lack of evidence to say where these primitives might
be housed and whether these functions might be present in more than one area in the
neuromuscular hierarchy. To look at oscillatory functions, we must look at areas known
for their rhythmic activity within the neuromuscular system, such as the brain stem and
spinal cord. To look at submovements and discrete movements, we turn our sights to
areas of the brain that integrate sensory feedback with motor commands. Additionally,
since discrete movements are highly goal-oriented and require object recognition to
reach the target, we will look at areas that function in motor planning, visual-guidance
and proprioception.
Chapter 3: Physiology of Discrete and Rhythmic Movements

As we have seen, the behavioral aspect of motor control and movement generation is a useful perspective to begin understanding the nature of rhythmic and discrete movements. Observing behavior allowed early scientists to discover the vast range of movements that humans are capable of as well as key underlying concepts of motor control, such as redundancy, degrees of freedom, synergies and error correction.

Behaviorists, like many other schools of scientific inquiry and practice, have largely been isolated and only sparingly integrated works from other scientific communities. Likewise, physiologists and clinical researchers, held their own set of ideas and practices within their community and rarely intermingled with the likes of behaviorists. This type of ‘island-thinking’ within scientific communities began to subside in the late 20th century, as the obvious benefits of sharing ideas and collaborating between neighboring disciplines became clear. For this reason, it becomes increasingly clear that a complete picture of the human motor control system is impossible without considering the biological mechanisms that underlie it. Physiology is the branch of biology that studies the function of the organism and how their individual parts work alone and together. Just like the modelling approach of motor primitives uses biological data to motivate their design, so too will a greater understanding of the physiological aspects of movement compliment and enrich the behavioral aspect of movement. In our physiological review, we will consider the spinal cord, central pattern generator, brainstem, primary motor cortex and posterior
parietal cortex. Although the human movement system relies on the functioning of many other areas, including the cerebellum, frontal lobe, supplementary motor areas and the basal ganglia, we only mention them briefly and in the context of rhythmic vs discrete motor control.

3.1 Spinal Cord and Reflexes

Although the spinal cord is the final common pathway before motor commands reach their respective muscles, it is still highly involved in regulating movement, especially with rapid reflex arcs that happen outside of our awareness (Zehr and Duysens 2004). The most basic example is the monosynaptic reflex shown in Fig 4 containing only two neurons. The simple reflex arc consists of a sensory neuron synapsing and exciting a motor neuron in the spinal cord, which activates a muscle. Most simple reflexes, like the knee jerk reflex, activate the
same muscle where the sensory neuron originated. To take it one step further, complex or polysynaptic reflexes contain interneurons between their sensory and motor neurons, increasing the types and number of ways that the spinal cord can involuntarily control movement. In Fig 5 an inhibitory interneuron (-) is present between the sensory neuron and a different motor neuron that projects to an antagonist muscle, preventing contraction. Polysynaptic reflexes are not limited to just inhibitory neurons synapsing on MNs to the antagonist muscle. The sensory neuron may stimulate an excitatory or inhibitory interneuron that stimulates a motor neuron projecting to an agonist or antagonist muscle. The MN may even project to other muscle groups in different limbs. Although only using a handful of active neurons, reflexes as a whole serve a very important control function at lower levels of the spinal cord, due to their fast and involuntary nature. Such reflexes offer a powerful, rapid, and automatic way to coordinate movements on the spinal level and are often useful in correcting errors in
movement that are otherwise too fast to control using cortical commands or subcortical commands (Folkow et al 1969).

Charles Scott Sherrington was one of the pioneers in observing and describing the mechanisms of muscle reflexes in the context of motor control. His studies on reflex arcs and neuron function in quadrupeds, such as dogs and cats, eventually lead to his receiving the Nobel prize for physiology in 1932. Sherrington used intracellular electrode recordings on the leg muscles of four-legged mammals and used the timing of the signals to model the pathways of the spinal neuron tracts. Sherrington found that when simple reflex arcs in the agonist leg muscles were active, it inhibited the antagonist leg muscles activity in the same limb, which is referred to as the law of reciprocal innervation (Sherrington 1908). This can be shown by summing and coordinating together the two reflexes shown in Figure 5.

Fig 6. A diagram of local spinal-muscle connections that connect different reflex arcs in muscles across distributed body parts. Note that all reflexes arcs share a common path, which allow for the production of compound reflex arcs (Source: Sherrington Charles (1908) The Integrative Action of the Nervous System, open source document)
In addition to reflexes acting on the same limb, Sherrington observed that when one side (i.e. Left) was active it would inhibit its contralateral side (i.e. Right). Similar findings of separate reflexes activating or inhibiting muscles onto different limbs and parts of the body allowed Sherrington to map out this system of complex reflexes illustrated on the previous page in Figure 6. Some of these reflexes crossed vast distances across the body and used interneurons that crossed multiple spinal segments to create long motor reflexes. Later studies identified these specific interneurons as propriospinal neurons and were important in distal motor control as well as sensory processing (Skinner et al 1989). Predecessors to Sherrington mainly observed reflexes in isolation from other muscle reflexes. However, it was clear to Sherrington that every reflex could potentially stimulate the receptor and sensory neuron of a different muscle and reflex arc with the possibility of coordinating many reflexes together shown in Figure 7. Using this logic, Sherrington reasoned that it was possible to control complex, whole-body movements, such as locomotion, at the spinal level. He was convinced that this happened by way of chaining many reflexes together in time, sequentially activating different muscles in different areas.

Fig 7 Schematic of 3 different chained reflexes proposed by Sherrington, R-receptor, E- effector (1) A receptor in a muscle is stimulated and activates a (Sherrington, Charles. Reflex activity of the spinal cord. CUP Archive, 1910, adapted from Herrick, Schwalbe's Neurology, open source document)
of the body to produce a coordinated movement. Sherrington named this multi-reflex activation a compound reflex (Sherrington, 1910). Sherrington postulated that chaining together compound reflexes sequentially at the spinal cord level enabled quadrupeds to activate the needed muscles to transition across the different phases of a walking or running gait. In principle compound reflexes at the spinal cord level had the capacity to fire rhythmically and self regulate some movements independently.

Later studies in the 1980’s found much of Sherrington’s claim of compound reflexes as insufficient to achieve coordinated locomotion and other complex rhythmic behavior (Baev 1992). Rarely are spinal reflexes the only control factor in achieving such complex behavior. Rhythmic generators at the level of spinal cord and brainstem serve as an important means of generating and sending reliable rhythmic signals to the desired muscles (MacKay-Lyons 2002). Additionally, motor commands from the cortex and brainstem were seen as the main organizing signal that controlled spinal and muscle activation (Trevor et al 2004).

Although there is dependency of top-down commands on initiating movement, the function of the spinal cord in organizing and sequencing muscle activity is critical. This is highlighted when looking at humans and other animals with spinal cord injuries. Many people with segmental spinal cord injuries have a difficult time regaining motor function, even using modern neurorehabilitation methods and physical therapy (McDonald and
Sadowsky 2002). Studies on quadruped animals with thoracic spinal cord injury saw that locomotion is impaired, especially of the hind-limbs, but it was partially possible to regain function. Using sensory input associated with stepping, dogs were able to regain some function of the hind limbs while on a treadmill (Behrman, 2000). Such improvements were not as pronounced when clinical trials were applied in humans, and only a few subjects were able to achieve any independent locomotion. It is possible that the spinal cord interneuron arrangement and spinal control is quite different in humans compared to non-human mammals.

3.2 Central Pattern Generators

Moving away from compound reflexes to explain spinal control, the motor community reengaged the possibility that central pattern generators rhythmically motor movement at the level of the spinal cord and brainstem (MacKay-Lyons, 2002). Central pattern generators (CPGs) are a series of neurons that are able to fire rhythmically and independently and have been studied since the 1960’s in many different model organisms. Although invertebrates were known to have many active CPGs to control rhythmic behavior, further evidence suggests vertebrates such as lampreys, macaque monkeys, and even humans actively use CPG arrangements (Grillner and Wallen 1985). Neurons of a CPG are arranged in a way that continued sensory input is not necessary to generate subsequent action potentials at a consistent rhythm, unlike chain reflexes. As seen in Figure 8, chain reflexes can be contrasted with CPG arrangements by their neuron
arrangement. Sensory neurons and interneurons are necessary in linking two reflexes together, however a central oscillator that rhythmically generates its own signal does not need these integrating components. As long as the CPG fires regularly, it will stimulate MNs and their associated muscles to fire rhythmically. Studies in deafferented animals with their sensory neurons knocked out proved to still retain rhythmicity, supporting the role of CPGs, rather than chain reflexes, in producing rhythmic behavior (Pearson 1987).

The common arrangement of such CPGs are half-center oscillators, which are two neurons that are reciprocally coupled through inhibition (Marder and Bucher 2001). As seen in Figure 9, the left neuron (green) stimulates an interneuron (purple) that inhibits the right neuron at the same time stimulating its motor neuron with its muscle and inhibiting the antagonist motor neuron and muscle. After a short time, the right neuron is released from inhibition and fires own pulse, mirroring the effect of the left neuron and functionally creating oscillatory pulses in antagonist muscles and generating rhythmic
movements. These CPGs have been identified in the spinal cord and likely play a critical role in motor control. It is important to note that although sensory input is not needed to achieve rhythmicity, sensory inputs do serve a modulatory role in modifying the rhythmic activity and potentially terminating it in the right circumstances. In this way only interneuron arrangements are sufficient in the spinal cord to produce oscillatory behavior in their connected muscles, without needing to wait for input from sensory neurons (Marder, 2001).

It is likely that rhythmic locomotion and other rhythmic movements begin as a motor command in the brain and activate the relevant spinal CPGs to promote ongoing rhythmic muscle activations. Diverting the rhythmic producing elements to the spinal cord interneurons allows us to save valuable cortical resources and simplify the organization of motor commands (Grillner, 2009). A striking example is the mollusk, Tritonia Diomedea, that has a single excitable neuron in its motor cortex that controls a complex motor

Fig 9 A common CPG neuron arrangement. The green neurons serve as the half center oscillators: as the left neuron fires, it inhibits the right neuron through an inhibitory interneuron (purple), stimulates its motor neuron directly and inhibits the antagonist motor neuron via an interneuron (brown). As the right motor neuron is released from inhibition, it fires a signal and subsequently inhibits the left neuron and the agonist motor neuron. (Source: Brown and Lundberg 1911, open source document)
pattern: escape swimming (Frost, 1996). This behavior lasts up to a minute, as the CPG in its dorsal ganglia continually activates its tentacles, flapping away from the initial danger stimulus. Research on zebrafish, which only contain approximately 300 descending neurons, shows that more complex motor patterns, such as prey capture, may be organized from several separate motor patterns arranged in sequence (Budick and (Budick and O’Malley, 2000).

In isolation, the activity of the central pattern generators in the spinal cord can produce multiple forms of movement, from locomotion to defensive withdrawal. As the number of elements that can be organized within the spinal cord increases so too does the computational power of neuromuscular system, which allows for greater flexibility in creating different motor patterns. More recent research on turtles and tadpole CPG activity, saw that there exist specialized spinal interneurons that have a multifunctional role in achieving different

Fig 10 a schematic of a typical extensor-flexor reflex arc (B) with an added central pattern generator regulating overall rhythmic bursts (C). Notice that the CPG is indirectly inducing rhythmicity by alternating releasing inhibition via the GABA-ergic path followed by stimulating the la inhibitory sensory neuron in succession (Hochman Lab- Emory University School of Medicine; Department of Physiology, permission pending)
rhythmic generation and increasing the possibilities in movement modification during rhythmic activity (Berkowitz, 2010). It is important to note that although spinal CPGs are able to continuously generate signals, they are unable to do this without some type of descending control or sensory reflex to initiate the rhythmic activity. Additionally, the termination of such CPGs can be achieved by an intrinsic timer within the CPGs or a distinct termination signal originating from the brain or periphery (Grillner, 2005).

CPGs play a vital role in rhythmic movement control and potentially simplify the computational power of the brain in producing movement by localizing rhythmic activity in the spinal level. However, it is also possible that mammals use similar CPG arrangements in movement control to ease the demands of discrete movements with continuous patterns (Bizzi 2008). Some discrete movements contain patterned responses even when the movement itself is not rhythmic. Mathematical decomposition of EMG signals in rhythmic and discrete arm movements have found that there is very similar timing of muscle activity within a bursting muscle (Ronsse et al 2009). Even though bursts may have occurred at varied times between discrete and rhythmic conditions, the identification within the bursts suggest similar neural activity at the spinal cord level. One possible explanation for this is the common use of similar CPGs and interneurons within the spinal cord to perform discrete and rhythmic movements. Although explicit evidence for CPG activity has been found in discrete movements, coordination and co-activation of muscle groups involved in performing discrete movements is clear. Since many discrete
movements contain at least two submovements, it is very possible that the coordination of these submovements is accomplished with a rhythmic oscillator within the spinal cord (Flash and Hochner 2005).

Through genetic identification techniques in rats, many spinal cord interneurons have been identified with unique functional properties in the spinal cord CPGs: V0, V1, V2, V3, and Hb9 neurons. Fig 11 illustrates the hypothesized arrangement of these interneurons in organizing motor patterns, coordinating left-right activity, and rhythmic generation of a mouse (Kiehn, 2011). Of these individual neuron types, it has been discovered that a subtype of interneurons, V1 cells, located in the mouse spinal cord CPGs are involved in locomotor networks in adjusting the speed of motor neuron outputs (Gosgnach, 2006). These
interneurons serve an inhibitory role in the local spinal CPGs and promote fast motor bursting behavior when activated. Using similar genetic methods to identify spinal interneurons, it is clear that humans also contain V1 neurons as well as other CPG interneurons found in the rat model. Using only neurons in the spinal cord, recruitment of the necessary muscles in walking can be achieved, modulated for frequency, and even alter the type of gait pattern.

Spinal cord CPGs not only are able to organize motor elements in different sequences to achieve unique rhythmic behavior, but they are able to adjust frequency of movement, the timing within movements, and burst strength at the local spinal level (Kiehn, 2006). It is particularly noteworthy that the physiological substrate that organizes intricate sequences of muscle activation, frequency adjustment, and phase changes can be located in the spinal cord, so close to the effector muscles. But are CPGs only involved in rhythmic activity or can these interneuron arrangements aid in discrete movement generation, where there is a clear target and goal? Can CPGs explain submovements and corrections in trajectory while aiming at a desired location? While the computational power of the spinal cord is great, it is likely that such behaviors and adjustments require a higher level of processing to create and organize their motor commands, which is likely a function of cortical networks.
3.3 Brainstem

The brainstem is the bottom-most level of the brain where neural tracts exit to form the spinal cord. It sits directly bellow the forebrain, which is further subdivided into the cortex and deeper brain structures, such as the limbic system. The brainstem controls the flow of signals between the brain and the body and is also involved in crucial life sustaining functions. Breathing, swallowing, heart rate regulation, blood pressure control, and wakefulness are all functions of the brainstem. Many of these functions contain a rhythmic element and it is said that the brainstem contains CPGs that regulate the activity of these physiological behaviors (Drew et al 2004). Moreover, the brainstem is also directly involved in motor regulation within muscles of the head, such as in mastication or swallowing. There is also compelling evidence that brainstem function is involved in motor control in the rest of the body.

Scott Sherrington also pioneered work on locomotion in decerebrate cats, with their brainstem in tact (Sherrington 1910). Observing the electrical recordings from the four
limbs, the decerebrate cat was placed on a moving treadmill. These cats achieved steady rhythmic locomotion when the treadmill was active, but could not initiate any voluntary movement, when the treadmill stopped. No cortical top-down commands are possible in decerebrate animals, so the cat must only rely on brainstem-spinal level control to achieve locomotion. Although the initiation of locomotion was impossible voluntarily, the movement of the treadmill likely induced some sensory-feedback pathways to allow for the cat to reciprocally activate its leg muscles and follow a ongoing reflexive loop using its intrinsic CPGs (Hiebert 1999).

Although in humans the brainstem is small compared to the rest of the cortex, it contains many individual areas that functionally have independent as well as complimentary roles, such as the midbrain, pons and medulla oblongata. These areas are further subdivided into populations of neuron cell bodies (nuclei) and tracts that deliver descending motor commands and receive ascending sensory inputs. Additionally, the brainstem is connected to the cerebellum, part of the hindbrain, via the pons, which is highly involved in the processing of movement, especially in posture, eye movement and breathing (Saladin 2007). The cerebellum receives cortical and peripheral sensory inputs and functions to regulate voluntary motor coordination, balance, posture, and speech.

Although this paper will not delve into the complex role that the cerebellum has in rhythmic and discrete movement control, it is worth noting that its influence on the brainstem activity modulates and alters descending motor commands and movement as a
whole. In humans, the medulla oblongata serves a minor role in motor coordination below the neck, but is important in regulating heart and blood vessel function, breathing, sneezing and swallowing (Afifi 1998).

These functions deal with the autonomic rather than the somatic aspects of movement control and have little impact on voluntary movements. On the other hand, the pons, due primarily to its position at the crossroads of cerebellum, midbrain and the spinal cord, serves an important function in movement regulation. Some functions, just to name a few, include wakefulness, respiratory rhythm, swallowing, equilibrium, eye movement, and postures. These 3 anatomical areas, the cerebellum, pons, and medulla oblongata all compose the hindbrain, the evolutionary oldest part of the brain.

The midbrain, although in the brainstem, lies at the intersection of the forebrain and hindbrain. It primarily acts as the major channel that integrates visual and auditory sensory signals with motor commands from the cortex (Sherman 2007). However, the midbrain does not just passively relay information to and from the body and cortex, it also continually integrates and modifies signals received from the cerebellum and motor
areas of the cortex (Garcia-Rill and Skinner 1988). Additionally, it is known that the red nucleus, a collection of cell bodies in the midbrain receive proprioceptive input from the vestibular system of the inner ear and the Golgi tendon organs from peripheral muscles and integrates this information to update subsequent motor commands (Cunnington 2002).

3.4 Corticospinal and Other Motor Tracts

The major link between the primary motor cortex, the brainstem and the spinal cord is the Corticospinal tract (CS). The M1 contains specialized large pyramidal Betz cells, which constitute the majority of the descending corticospinal tract and a large portion of the corticobulbar (CB) tract (Meyer 1987). The CS controls voluntary muscles below the neck, whereas the CB controls voluntary muscles of the head. The M1 area, like the rest of the cortex, contains 6 main layers of cells with efferent, afferent, or associative functions. Layer 5 serves an efferent function and contains the excitatory Betz cells, which are upper motor neurons (UMNs), of the CS and CB tracts that project onto lower motor neurons (LMNs) that directly innervate muscles. Together, the CS and CB axons leave the cortex and begin forming what is known as the pyramidal tract, due to its triangular shape. CB axons project onto the neurons of the brainstem, whereas CS axons project onto neurons of the spinal cord. As we follow the CS bundle of axons, from different part of the M1, they come together to form the internal capsule and descend past the medulla at which point the vast majority of fibers cross over to the contralateral side at the pyramidal
decussation forming the lateral corticospinal tract, which controls fine motor movements of the appendicular skeleton (Davidoff 1990). The rest of the fibers continue descending on the ipsilateral side and form the anterior corticospinal tract, which controls the majority of the trunk muscles of the axial skeleton. Both of these tracts of fibers descend
into the spinal cord where they synapse onto lower motor neurons in the anterior grey column.

Some axons of the CS branch off before reaching the spinal cord at the brainstem, specifically at the red nucleus of the midbrain (Fig 14, Left). As we will discuss later, the midbrain is involved in rhythmic motor control, but primarily for the mouth, head, and lungs. This tangential projection synapses onto cell bodies of the red nucleus and continues as the rubrospinal tract. In vertebrates without a significant corticospinal tract, such as amphibians and reptiles, the rubrospinal tract acts as the major motor control path for locomotion and other rhythmic movements (Grillner 2011). In higher mammals, such as humans and macaque monkeys, the rubrospinal tract acts to maintain tone in muscles and adds stability during movement, allowing actions to be smooth and fluid. It is noteworthy to mention that babies rely on this tract to generate muscle patterns needed for crawling, but it is unknown how this tract functions when learning bipedal locomotion and in later developing rhythmic activities in humans.

The corticospinal tract also contains side projections in the lower areas of the brainstem, specifically in the pons and medulla. This wide area, known as the reticular formation, is the beginning of the reticulospinal tract, known to control posture as well as locomotion. Research on lampreys has shown that there is rhythmic firing of reticular clusters of neurons when performing rhythmic undulations during swimming and burrowing.
(Deliagina 2000). The reticulospinal system carries other important motor control commands involved in locomotion initiation, steering and equilibrium control. It is known that in humans the reticulospinal system does not have such a dominant role in performing rhythmic movements as Lampreys, however evidence has shown that movement modulation due to sensory inputs is associated with reticulospinal firing in humans (Roberts et al 2000).

It is important to note that some UMs have neuromodulatory influences from the brainstem, meaning the rate or pattern of firing is altered using brainstem neurons projecting onto. Neuromodulatory influences work in a different way than standard inhibitory and excitatory signals and often change the electrical conduction properties of the affected neurons to alter their normal firing pattern (Kobayashi and Tadashi 2002). It is likely that ascending sensory feedback from the body is integrated in brainstem and modify later movements, make adjustments in trajectory, or alter the firing pattern of rhythmic activity (Nguyen and Kleinfeld 2005). Some of these UMs directly synapse on to their spinal LMs, which directly innervate muscles, while other UMs are separated by an interneuron before reaching their destined UMs and muscle fibers. These interneurons are the same interneurons that are responsible for complex reflex production, central pattern generation, and organization of synergies and are pronouncedly active during rhythmic motor commands (Berkowitz et al 2010). Interestingly enough, the direct connections from UMs to LMs are evolutionary more
recent than the intermediary step in the spinal cord and seem to be the most pronounced in humans (Lemon and Griffiths 2005). Since humans rely heavily on fine hand movements and object manipulation, this lends credence to the fact that direct connections to LMNs must allow for greater motor control in individual muscle fibers in completing delicate, single-muscle dependent behaviors. It is likely that such direct connections to muscles may allow for discrete control of a small set of muscles, rather than producing continuous rhythmic patterns in the underlying limbs.

Direct control of individual muscles produces more possibilities in muscle activations and thus increases the amount of behaviors one can perform. However, such a task is computationally demanding due to the lack of automaticity in the neural system. When CS neurons are indirectly connected muscles using interneurons that command an array of muscles, the computational demand decreases. The power of the interneuron separating the UMN and LMN allows muscle activation to be grouped, sequenced and coordinated in different ways. This may be very useful in overlearned movements, common behaviors that do not need any skill training, such as walking and running or even skillful covariation of muscles during a blacksmith’s hammer swing, but might be very difficult when learning new movements or constructing new behaviors, such is the case when a novice performer attempting to swing a hammer for the first time (Trevor et al 2008). It is useless to use previously learnt synergies for new tasks without somehow modifying them, it could be the case that direct UMN to LMN connections allow for beginners to practice the muscles and later rearrange using interneurons at intermediate
learning stages. Additionally, non-rhythmic behaviors might use the direct UMN to LMN connections to bypass any interneuron control. Although autonomous control is favorable in some cases with continuous or overlearned movements, direct muscle control is also necessary when learning new skills or performing discrete movements that are more precisely goal-oriented.

3.5 Motor Cortex and Supplemental Areas

The importance of the brainstem and spinal cord are paramount in achieving movement, especially since they are the final checkpoints before muscle activation. However, the movement system is far more expansive in its construction and it is necessary to look at cortical functions and connections to investigate the control of voluntary actions. For this reason, a brief look at the anatomy of the M1 and its descending tracts is necessary. The primary motor cortex, or M1 cortex (Brodmann area 4), was largely investigated by a preeminent Canadian neurosurgeon, Wilder Penfield. In addition to expanding methods and techniques in brain surgery, Dr. Penfield used stimulating electrodes on the brain to observe the connectivity on other parts of the body, while recording the activity of many muscles (Penfield 1961). Penfield found there was a direct somatotopic mapping from the M1 cortex that enervated all the muscles of the body or simply referred to as the motor homunculus. In other words, a stimulus to one part of the M1 would consistently excite and cause a twitch in one muscle group. Additionally, if you move the stimulating probe
over only a millimeter and you would be exciting the adjacent muscles of the body. These characteristics of the motor system have proven to be conserved across primates and other vertebrates. This finding into the organization of the primary motor cortex is the starting point in understanding motor control hierarchy and opened up a host of possible stimulation studies to probe for the function and connectivity of the motor cortex.

In much the same way Penfield was able to stimulate the muscles of dogs and cats through electrical stimulation in the 1940s, in today’s times it is possible to safely stimulate human brains using transcranial magnetic stimulation or TMS (Robert 2000). Although TMS has been used as scientific tool before the 1980’s, it was not until the advent of Dr. Anthony Barker’s Magnetic
Nerve Stimulation (MNS) technique that TMS saw a real boom in popularity (Barker et al 1985). The MNS technique was able to create transient lesions in the brain, which would disrupt the activity of the targeted tissue and causes excitability changes in connected neurons. The TMS machine worked by using a large battery source that discharged a heavy current into a wire that coiled around itself many times. By applying Faraday’s law of electromagnetic induction, such a coiled wire construction, would produce a large magnetic field to be produced at the center of the coil. Through this mechanism, an experimenter that wanted to probe the brain for function or connectivity can place the coil on the desired cranial area, stimulate, and record the result. If the coil is engaged and stimulates the motor cortex above a specific intensity threshold, fibers of the corticospinal tract will fire and activate the muscles that they enervate.

Fig 16 TMS activation and Faraday’s Induction in the brain. The large electric current flowing through the magnetic coil (black), creates a substantial magnetic flux (purple) that induces an electric potential (green) in neurons deep to the stimulator. If the current is strong enough, these neurons will fire action potentials. (Source: The Neurocognition and Psychophysics Laboratory, Department of Psychology, University of Saskatchewan, open source material)
The primary motor cortex not only serves as a relay for motor commands, it also serves as an integrating role for projections coming from cortical areas such as the Supplementary Motor Area (SMA), the dorsal and ventral Premotor Cortex (PMC), the somatosensory area (SSA), and posterior parietal cortex (PPC) as well as subcortical areas such as the thalamus, basal ganglia, and cerebellum (Purves 2001). Although an in-depth analysis of each area is beyond the scope of this paper, we will review key aspects that will become relevant to the control of discrete vs rhythmic behavior. Of these areas that connect to the M1, the SMA, PMC, and PPC are highly active during discrete hand movements, but absent in similar rhythmic movements.

The supplementary motor area has a similar somatotopic map as the M1 cortex and has been implicated in self-generated movements that do not require a sensory stimulus to initiate. Although the true function of the SMA is still unknown, SMA activation has been
readily seen while performers sequence a group of movements and perform bimanual
tasks. The function of the premotor cortex (PMC) is implicated in movement planning,
spatially guided movements, and recognizing and understanding other peoples’ actions.
Both these areas not only send projections to the M1, but also connect directly to spinal
lower motor neurons. Additionally, the SMA and PMC are involved in postural control and
compensatory muscle activation for balance while performing a task.

The posterior parietal cortex is a very large area of the brain at the crossroads of sensory-
visual processing and motor control. It is likely that that tasks that depend on visually
based sensory stimuli rely on the PPC to coordinate muscle activation (Corbetta 2000).
The function of the PPC has been elucidated by lesion studies in primates and humans.
Damage to the PPC has resulted in many sensorimotor impairments, including decrease in
memory and perception of spatial relations, poor accuracy in reaching and grasping
movements, and control of eye movement (Azanon et al 2010; Bologini et al 2007).
Additionally, research in children with infantile autism has found considerably decreased
in overall volume in the parietal lobe, especially in the PPC (Courchesne et al 1993). As
many children with autism have poor hand-eye coordination, reduced accuracy in
reaching and holding on to objects, as well as difficulty in visually mimicking other people,
it lends some support to the importance of the PPC in performing discrete movements.
Interestingly, many children with autism also suffer from frequent bouts of rhythmic head
and arm movements brought about by irregular oscillations in the nervous system.
As scientists come to understand the different functions of the nervous system in creating and controlling movement, further experiments are needed to bridge the gap between physiological data and behavioral data. To bolster the current understanding of motor control as whole, we have conducted two pilot studies and collected preliminary data on rhythmic and discrete movements performed in humans. Using TMS techniques that were previously discussed, we combined a behavioral task with added magnetic stimulation on two specific areas that are known to be involved in motor control: The M1 and the PPC. The conclusions of these studies in combination with the literature review on behavioral and physiological aspects of rhythmic and discrete movements will allow us to have a more comprehensive understanding in motor control differences between these different movements.
Chapter 4: Experiments

Within this chapter we will discuss two preliminary experiments I have conducted using transcranial magnetic stimulation (TMS) on human subjects. The following 2 studies focus on two areas of the brain that are known to be involved in motor control, the primary motor cortex and the posterior parietal cortex. We use TMS pulses on these areas of the brain to see if they affect rhythmic and discrete movement generation. In both experiments each subject will be performing forearm flexion and extension, either rhythmically or discretely.

Although this data has not been published, it is used as a first look at how TMS might be used to see the difference in rhythmic and discrete movements. Magnetic pulses are used, while subjects perform a rhythmic or discrete forearm flexion and extension. Muscle EMG and kinematic differences during these movements are recorded and analyzed. Differences in these results will inform us of possible motor control mechanisms that are involved and how they might be different in these two types of movements. Possible follow-up studies are mentioned and applications of the results are discussed within the larger scope of motor control.
4.1 M1 Stimulation Experiment

First support for the distinct nature of rhythmic vs. discrete movements in humans was seen in a fMRI neuroimaging study (Schaal et al, 2004). Their results showed that distinct brain regions are activated when wrist flexion and extension are alternated rhythmically, compared to a sequencing of discrete flexions and extensions (Fig 18). While rhythmic movements were associated with primary motor areas, discrete movements required additional activation in parietal and prefrontal regions of the brain. Furthermore, M1 activity during rhythmic movements was significantly decreased compared to discrete movements in the fMRI experiment.

The goal of our study seeks to further distinguish the control mechanisms present in performing human rhythmic and discrete movements, while undergoing transcranial magnetic stimulation (TMS) to the M1 cortex. Based on the previous fMRI findings of Fig 18. fMRI activation of cortical regions of the brain in response to discrete wrist rotation and rhythmic wrist rotation. (Left) The red area represent cortical response during discrete movements subtracted by background activity and the yellow areas represent cortical response during rhythmic movements subtracted by background activity; (Right) The blue areas represent the discrete response subtracted by the rhythmic response and the green areas represent the rhythmic response subtracted by the discrete response. (Source: Schaal S., Stemad D., Osu R., Kawato, M. “Rhythmic movements are not discrete” Nature Neuroscience, Vol 7 Num 10 (2004) p1136-1143, with permission)
increased primary motor cortex activation in discrete movements, we expect that TMS will have a greater effect while performing discrete movements. TMS (Magstim, D70^2: 1.9T) pulses were perturbed the contralateral M1 area to corroborate the differential cortical involvement between these two types of movements. Using single-pulse TMS, subjects were stimulated during discrete and rhythmic forearm movements, while having their muscle activity recorded. Since rhythmic movements had considerably reduced activation in these areas, less perturbation is expected in the moving arm. Conversely, discrete movements were expected to have large perturbations. Subjects were seated with their trunk and head stabilized, the right forearm resting on a manipulandum allowing flexions and extensions in the horizontal plane. Continuous displacements were recorded. Condition-1 (rhythmic): Subjects oscillated their dominant forearm at their preferred frequency for a 2-min trial. At a random time, a single TMS pulse (100μs rise time, 1ms duration) was applied to the contralateral parietal area at the Pz electrode location. Only supra-threshold stimulation magnitudes were used, eliciting overt muscular disruptions. Condition-2 (discrete): Subjects performed self-paced flexions and extensions separated by random intervals (minimum of 3s) over the course of a 5-min trial. During discrete movements, the TMS pulse was timed to occur at peak speed. The velocity profile of the discrete movement showed significant perturbations, however rhythmic movements saw similar average perturbations, but not for all phases of motion. Additionally, discrete movements saw a much higher variability when responding to TMS compared to rhythmic movements, in each phase of motion.
4.1.1 Methods

Participants were seated in a custom-fitted chair and rested their right forearm on moveable pivot arm equipped with an optical encoder that recorded forearm kinematics (Fig 19). An alcohol prep-pad was applied on the upper arm to improve electrical conductance and EMG electrodes were attached to the right biceps, triceps and grounding wire. The activity of the muscles was observed on an oscilloscope (Virtual Oscilloscope) to confirm the placement of electrodes. Participants were then given a 5-minute training period where they practiced the discrete and rhythmic movements until satisfactory consistency in performance was reached.

Figure 19. Setup of Experimental Design with Essential Components:
A monitor in front of the volunteers displayed the position of the right forearm angle (psychophysics toolbox, 32-bit Matlab ver. 2012a). Two circular targets were displayed on the virtual environment and the volunteer was instructed to move from one target to the next in each dynamic condition shown on the next page in Figure 20. The targets were separated by 100 degrees. For the rhythmic condition, the subject was instructed to move continuously between each target in 1.5 seconds, making sure not to stop. For the discrete condition, the subject was instructed to move to the next target when a ‘GO’ cue appeared (the current target turns red) and stop at the next target until the next ‘GO’
Care was taken to ensure that the time of movement in the discrete and rhythmic half-cycle was 1.5 seconds. Each volunteer went through 2 rhythmic and 2 discrete blocks in randomized order, with each block containing 24 pulses and running approximately 2 minutes.

All volunteers were verbally told of the procedures and each individual signed a consent form, signifying their understanding of the experimental techniques involved and agreement to take part in the study. Volunteers were given a questionnaire to identify any exclusion criteria, such as medication and health factors as well as cataloging any potential effects on cortical excitability. Approval for the study was given by the Northeastern Institutional Review Board (Approved: 1/8/14 ID# 12-11-07).
TMS Hot spot and Motor Threshold

In order to identify the best location for the magnetic stimulator prior to data collection, we used neuro-navigation software (Brainsight ver. 2013) with attached tracking camera to probe the motor cortex. By applying magnetic pulses and monitoring muscle activity, we were able to locate areas of the brain that evoked an excitatory potential in the biceps. These muscle activations are called motor evoked potentials (MEPs) and are reliably reproduced using sufficient TMS stimulation in the corresponding location in the primary motor cortex. MEPs appear at an invariant time (~24msec) after pulse onset in the biceps and any response that had a peak-to-peak voltage value over 50uV in this time window was considered a noticeable MEP (Fig 22). To hone in on the area of interest after finding an initial MEP, we fired pulses on a systematic array of cortical targets, with the aid of the neuro-navigation interface. The targeted area that resulted in maximal biceps activation was termed the “hot spot”, which was used as the stimulator location for all subsequent stimulations.

Figure 22. Sample EMG Trace with MEP. This graph depicts the onset of pulse, characterized by a sudden electromagnetic interference from the TMS pulse, called the TMS artefact (red). The motor evoked potential (green) that is followed approximate 20msec after. The highest and lowest points of the MEP were used to determine the peak-to-peak amplitude.
For each volunteer, we selected the desired amplitude of stimulation by first finding the amplitude that resulted in a noticeable MEP (above 50uV) in non-moving biceps 50% of the time, termed the motor threshold (MT). We used an adaptive motor threshold assessment program (AdaptivePEST, MUSC dept. of Psychiatry) to estimate this threshold. As a standard practice in other TMS protocols, we used a suprathreshold stimulation intensity of MT + 20% to excite the motor cortex.

Data Acquisition

All data acquisition was done with the aid of the Matlab data acquisition toolbox. A custom-created program read in the 12-bit kinematic data of the pivot-arm encoder and converted it to angles. A 3000 Hz sampling frequency was for kinematic data acquisition. Additionally, a digital signal is sent from the stimulator every time a pulse was fired, which synchronized the kinematic data with the time of stimulation. EMG data is recorded in parallel and was synchronized with the kinematic data. The same sample frequency was used to prevent any time stretch, when synchronizing data. The final raw data file for each block of

![Sample subject data of pulse, kinematics, and EMG](image)

Figure 23. Sample Subject Data in One Block. The pulse timing (red lines), Kinematic data (green trace), and EMG data (blue trace) are depicted for a 15 second segment of rhythmic movement.
movement included (1) timing (2) pulse firing (3) kinematic data (4) EMG signal. We can visualize these parameters on one plot and observe trends over time (Fig 23). Each subject contained 4 data files for each block of movement. Since we performed 2 rhythmic and 2 discrete blocks, each containing 24 pulses of TMS, each condition contained 48 pulses, with a grand total of 96 pulses for all 4 blocks.

**4.1.2 Data Analysis**

The first step in analysis is to calculate the kinematic phase of movement during the onset of each pulse using custom Matlab scripts (64-bit Matlab 2014b). Since the movement contained flexion and extension components, the phases were divided in equal sections with 0-50% being flexion and 50-100% being extension, by definition. Each TMS pulse was time-stamped and matched with the kinematic phase of movement of the forearm.

Since MEPs were experimentally identified as being present 15-30msec after pulse, amplitude of MEP was estimated by taking the peak-to-peak value in this time window. In summary each pulse was characterized by identifying the kinematic phase of the forearm as well as the presence and amplitude of the resultant MEP. For each of the dynamic conditions 48 data points were taken with phase being the independent variable, and MEP amplitude being the dependent variable.
The kinematic phases were binned in 8 groups, 12.5, 25, 37.5, 50, 62.5, 75, 87.5, and 100. This matched the protocol of Carson et al 2004. For each subject, the MEP amplitudes were normalized to the maximum MEP within a block making the dependent value ‘percentage of maximum MEP’. This manipulation allowed us to compare MEP phase-dependencies across subjects. The data was averaged across each subject and a standard deviation was calculated for each phase bin.

The bursting pattern of muscles was characterized in terms of total net activity of the active muscle to control for the variable EMG activity and intensity of the muscles across different dynamic conditions. Bursting activity in the muscle was found by rectifying the EMG signal, using a detrending.

Figure 24. Sample EMG with Overlaid Envelope. The EMG signal (blue) is depicted with its filtered envelope (red) with two bursts. The 25% of max envelope mark (green) is the threshold at which a burst can be identified.

Figure 25. Exemplary Kinematic Data from one Subject During Rhythmic Movement. Bursting activity in biceps occurred from 80-100% (red line) during the beginning of flexion and end of extension. Green line depicts bursts occurring 30-80% of the time.
function and a low-pass 2.5Hz Butterworth filter to create an EMG envelope (Fig 24). This process gives us information of the overall intensity of EMG firing across time. As standard practice, if EMG activity was above 25% of the maximum envelope, the muscle is considered to be bursting. Bursting durations were averaged across each cycle (Fig 25) of movement and compared between dynamic conditions.

4.1.3 Results

Final binned results for all subjects comparing motor evoked potentials in rhythmic and discrete forearm rotation are depicted on the next page in Fig 26. A one-way ANOVA with two nominal variables, rhythmic and discrete, was done for each binned phase. Each binned phase failed the ANOVA by giving a large F value that did not satisfy $F<1.000$ needed to reject the null-hypothesis. All $p$ values for MEP effects were $p>0.05$ (TABLE 1A). It is possible there is a minor effect in the phase corresponding to 12.5% of the movement that gave the smallest ratio of effect over variance, but this was not a significant result. A similar one-way ANOVA using the standard deviations (variability) as the inputs gave much lower F-values and were significant in the beginning of flexion and ending of the extension phase (TABLE 1B).
Figure 26. Plots of MEP Response Across Phase. Both plots are constructed using data from Table 1. Panel A depicts rhythmic phase dependency on MEP and Panel B depicts discrete phase dependency on MEP. Note the consistent increase in error bars between dynamics conditions. Phases 0 (100), 12.5, 25 show significant increases in variability in the discrete movements.
A custom pattern-fitting program was used in Matlab to find average burst durations for each subject. One possible problem with the comparison between rhythmic and discrete movement MEPs is the muscle burst duration for each movement. If the burst duration for a similar movement is naturally larger without TMS stimulation, the MEP responses would also be increased due to increased corticospinal activation. A one-way ANOVA test compared the bursting durations in the two dynamic conditions and no significant differences were found between the two conditions. For this reason, differences in bursting duration were not considered a variable that could confound analysis of the kinematic phase dependency of MEP generation.

<table>
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<tr>
<th>Phase</th>
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Table 1: One-way ANOVA analysis. Gives the F-values for a one-way ANOVA for the 8 different phases. Panel A depicts ANOVA analysis using %MEP across dynamics conditions and Panel B depicts ANOVA analysis using standard deviation across dynamic conditions. Significant values are highlighted in green.
4.1.4 Conclusions

We hypothesized that there would a difference in the pattern of biceps activation across the two different movement conditions: rhythmic and discrete. Using one-way ANOVA, the data do not support this hypothesis. However, there was an increase in within-subject variability during the beginning of flexion and ending of extension in discrete conditions compared to rhythmic conditions. From the data, it is clear that the cortex increases its excitability primarily when the biceps is active, both when it functions as a primary mover (beginning of flexion) or as the breaking force (ending of extension). On average this excitability is not controlled differently with rhythmic and discrete movements, but there is a significant increase in variability during these phases in discrete movements compared to rhythmic movements. From this, one can infer that there is more variable control during these phases for discrete movement, which could reflect the increased amount of inputs that the M1 receives during these movements. Ultimately, these data support the assertion that there are different control mechanisms exerted during rhythmic and discrete behaviors in the M1, even when similar muscles are activated.

In 10 subjects, it was observed that on average there is no significant changes in biceps EMG amplitude during stimulation between the two movement conditions. This was true for each of the eight phases of movement between the two conditions with the exception of phase (2), which corresponded to the 25-50% flexion. Stimulation during the discrete
movement condition during this phase resulted in a significantly higher amplitude (p value 1). This trend was true for each of the 10 participants.

Additionally, we have found that variability of the EMG amplitude was significantly greater (p value 2) in all phases of movement during the discrete condition. This trend was true for all 10 participants. Although we did not see any significant changes in MEP excitability in discrete movements over rhythmic movements in this study, there was a significantly greater variability in MEP responses in discrete movements compared to rhythmic movements. The increase in variability might indicate a different type of processing occurring in the M1 cortex during discrete movements compared to rhythmic movements, even though the average responses were different. Since discrete movements have been shown to have many more cortical areas active compared to rhythmic movements, it would make sense that the M1 stimulation during discrete movements would lead to a greater variability in MEP responses compared to rhythmic movements.

Although the fMRI study showed differences in the primary motor cortex activation in the two dynamic conditions, other parts of the brain might be better candidates in finding control differences. This is an important consideration since the M1 area is likely responsible for integrating and sending out the final motor command towards the muscles, rather than taking a dominant control role. Some important considerations are
the different perceptual and planning demands required by discrete movements, which likely reflect the increase in activity in parietal and supplementary motor areas. Since the posterior parietal cortex (PPC) was found to be active during discrete wrist flexion, it might be expected that stimulation in this area will only affect the activity during discrete movements and not rhythmic ones. To further understand the differences in control of discrete and rhythmic movements, single-pulse TMS was performed on the PPC to observe the elicited MEPs in the biceps in our subsequent study.

4.2 PPC Stimulation Experiment

The Posterior Parietal Cortex (PPC) is another important area involved in the motor control system and lies posterior to the primary motor cortex. The PPC is highly involved in sensorimotor integration and is important in perception of movement, memory of spatial relations, and behavioral attention (Anderson, 1989). Previous views of the PPC label it as purely sensory cortex where different sensory modalities were associated, including visual and proprioceptive modalities. However, more recent studies found that lesions in the primate and human PPC area resulted in serious deficits in movement and motor planning (Corbetta, 2000). Further revisions of the role of the PPC find that activity
in this area was related to object fixation, smooth eye pursuit during movement, and even shape representation for action planning (Anderson, 2009). Another study found that visual and somatosensory association in the PPC enabled humans to have representations of touch in external space (Azanon, 2010). Although much of the PPC is indeed sensory association cortex, the ultimate goal of this sensory coupling is driven towards smooth movement generation during goal-oriented tasks.

Based on the findings by Schaal, the PPC has negligible activation during rhythmic movements, but is highly active during discrete movements. Previously believed to be sensory cortex, it is now known that the PPC serves a sensory-motor integration role in motor planning, intentions, and visually guided movements (Andersen 2002). Primate studies that looked at reaching and gaze found that a subsection of the PPC is specifically active when primates made reaching movements with their arm towards a desired object within arm’s reach (Citation). This area would be known as the parietal reaching region.

Fig 27. A Topographical map of motor association areas of the brain. Of particular importance are the location of the primary motor cortex and posterior parietal cortex (Source: thebrain.mcgill.ca, open source material)
Similarly, there are other populations of neurons in the PPC that fire when the primate grasps for the item it was reaching for, after it has reach its destination: the parietal grasp region (Vingerboets, 2014). These findings taken together, emphasize the PPC’s role in producing and controlling common discrete movements, such as reaching. Interestingly enough, these parietal regions have analogues in the human brain (Citation). Since all reaching movements are discrete, the PPC is likely involved in the control of this specific type of movement, whereas rhythmic movements are unlikely to activate the PPC. For this reason, we hypothesize that TMS pulses during discrete movements will cause noticeable kinematic perturbations, whereas rhythmic movements will be unaffected.

Taking a very similar approach from Experiment 1, this study’s aim was to further elucidate mechanisms of control rhythmic vs discrete movements. Instead of targeting the primary motor cortex, the TMS pulse was aimed at the PPC. Although the PPC does have corticospinal upper motor neurons that descend to your skeletal muscles, there are too few to achieve any noticeable MEP using EMG electrodes (Shadmehr and Krakauer...
2008). For this reason, the motor perturbations caused by PPC stimulation were identified by the error in trajectory and end-point variance from intended target.

4.2.1 Methods

Prior to the experiment, all six volunteers were given a brief introduction and description of the PPC experiment as well as verbal instructions on the types of the forearm movements to be performed. A questionnaire was given for each volunteer to evaluate their eligibility in performing in the experiment. Exclusion criteria included any physical or environmental factors that could affect cortical excitability, such as caffeine intake, amount of sleep, medications, or pre-existing medical conditions. Following, participants were seated in a custom-fitted chair and rested their right forearm on moveable pivot arm equipped with an optical encoder that recorded forearm kinematics. This set up was identical to the previous experiment (Fig 18). Both movement types required the subject to rotate the pivot arm along the horizontal axis limited to 100 degrees of movement. One cycle of each movement would oscillate between flexion and extension: moving from the midline of the body and extending outward. For rhythmic movements, subjects will oscillate between flexion and extension with no stop, while discrete movements will see at least a 1 second stop in between cycles.

Participants were then given a 5-minute training period where they practiced the discrete and rhythmic movements until satisfactory consistency in performance was reached. No
electrodes were needed since no muscle activation or motor evoked potential was expected from PPC stimulation and so EMG data could not be collected. Additionally, we used the same virtual display (Fig 19) to give subjects their online forearm position as they perform the movement.

One complication in probing the posterior parietal cortex was finding the right stimulator intensity to adequately disrupt the activity in the PPC. This was not a problem in the M1 experiment since reliable MEPs produced and the motor threshold could be adjusted. However, since TMS pulses would not directly evoke contractions in muscles while stimulating the PPC, another method for calculating stimulator intensity was needed. For this reason, we used the next best approximation, by using the primary motor cortex excitability as in the previous experiment. We used the same protocol in the previous experiment to determine the M1 excitability for each of the six subjects. Using an adaptive motor threshold assessment program (AdaptivePEST, MUSC dept. of Psychiatry) we adjusted subsequent TMS stimulus amplitudes until they converged at the threshold amplitude. As standard practice we use the stimulus intensity of threshold +20% for all subsequent PPC stimulations.

To locate the posterior parietal cortex, a grid was set up on a swimming cap with approximate known locations of relevant cortical areas. The grid was based on a 64 channel electrode cap setup and the midpoint between area P3 and P1 shown in Fig 25 is
known to be the approximate placement of the Posterior Parietal Cortex (Ball et al 1999).

A pen mark is placed in this area, which will be used by all 6 subjects. Given that the swim cap conformed to the head shape of each subject, specific care was taken to confirm that the position of PPC was correct. A 64-channel electrode cap was fastened on top of the swimming cap and marked the exact mid-point area between P1 and P3 with a pen mark, shown in figure 29 with a green box. Then the positions of the two pen marks are averaged and used as the final target for the TMS stimulator. The coil is placed over the marked swimming cap and a TMS pulse is fired in the desired marked area.

Following training and target acquisition of the PPC, we would perform the movement trials with TMS stimulation. There were 4 type trials: rhythmic with TMS, rhythmic with sham TMS, discrete with TMS, and discrete with sham.

Fig 29 A schematic diagram of a 64-electrode cap on a subject’s head. The PPC focal point is highlighted by the area between P1 and P3 in the green box. The sham trial target is located in area TP7 highlighted by the red box. (Source: Kielar and Joanisse. "Graded effects of regularity in language revealed by N400 indices of morphological priming.” Journal of Cognitive Neuroscience 22.7 (2010): 1373-1398. Permission pending)
TMS. The sham trials used TMS stimulation, but on an unrelated part of the cortex that should not effect movement: the superior temporal gyrus. This area corresponds to the TP7 electrode (red box in figure 29) on the 64-channel cap and was marked as the sham target on the swimming cap. This area is highly involved in memory and hearing and its stimulation should not directly affect motor performance. The sham condition was introduced as a control to determine if there were any generalized effects of TMS on subject behavior.

The trials were arranged in a random blocked pattern so that each would contain 1 rhythmic with TMS, 1 rhythmic with sham, 2 discrete with TMS, and 2 discrete with sham. Since discrete trials have a significant wait period where no movements are done, twice as many discrete trials were completed to match the amount of successful pulses in rhythmic trials. Since the PPC is involved in motor planning in goal-oriented tasks, it would make sense that TMS pulses would not only affect the trajectory during movement, but would also affect subsequent motor activation before movement begins. A successful pulse in the discrete condition would be considered only if the pulse was between .5 seconds before movement onset and up until the end of the discrete forearm movement. Any pulse that was outside of this window was not considered in the final data analysis. Conversely, since rhythmic trials had consistent movements, no exclusion criteria were used and all TMS pulses were considered successful.


4.2.2 Data Analysis

Motor evoked potentials are rarely seen when stimulating the posterior parietal cortex, due to the limited direct fibers that project onto the corticospinal tract (Shadmehr and Krakauer 2008). For this reason, we could not use the EMG signal from the biceps and triceps to determine an effect on stimulating the PPC during rhythmic and discrete movements. Instead, we would specifically look at the trajectory data and the end-point variance to see if there were any deviations in movements with and without TMS stimulation.

The raw position data was compiled together for each of the six subjects with the time stamp of each TMS pulse as shown in Fig 30. Additionally, the velocity at each position was calculated using the first derivative in a custom Matlab program and superimposed
on to the original graph. For discrete movement trials, it was essential to know the beginning and end of each discrete movement. For the beginning of the movement, we used a minimal velocity threshold that was calculated using the same custom Matlab program for each subject. It was insufficient to use a zero acceleration condition to determine the beginning of the discrete movement due to slight movements in the arm while waiting. A velocity threshold was much more accurate in predicting true starts and was confirmed by manual inspection. To calculate position and time when discrete movements ended, determining the first zero acceleration after beginning the movement was sufficient and no threshold condition was needed.

To calculate end-point error for discrete movements, the true start and stop positions were used as described in the previous paragraph. These positions would be compared to the target positions on the visual display and the difference would yield the end-point error for each movement. Only movements that had TMS pulses between .5 seconds of movement onset and up to the end of the discrete movement were considered to be under the pulsed category of movements. All other discrete movements were assumed to have no TMS influence. Similarly, to calculate end-point error for rhythmic movements, each peak of the sinusoid-like wave movement was taken and compared with the target value. Each end-point was grouped under whether it had a TMS pulse preceding it within one cycle. If there was no TMS within one cycle, the movement was assumed to have no
TMS influence. The sham and non-sham trials for end-point error were calculated identically.

To calculate trajectory deviations, the kinematics of normal discrete and rhythmic forearm movements were analyzed. It was seen that rhythmic movements have a sinusoidal nature, with a rise and dip in a repeated sequence that resembled a sine wave. Discrete movements, however, had a closer resemblance to sigmoid functions. To start calculating trajectory deviations for rhythmic movements, non-TMS movement samples were used to find a sinusoidal function of best-fit by using the Matlab systems identification toolbox (MatLab 2016b). This derived function is subtracted from each cycle of other non-TMS movement samples and integrated.

Fig 31 Graphs representing kinematic trajectory. Top: An ideal sigmoid curve was fitted for each discrete movement. Middle: an ideal sinusoid was fitted for each rhythmic movement. Bottom: The difference between ideal movement and actual movement was to find the deviation in trajectory.
over time to find the area under the curve. This area represents the average trajectory deviation for non-TMS movements. The same procedure was used for each trial to calculate trajectory deviations in TMS movements. The average deviations between non-TMS and TMS movements were later be compared by first using a standard T-test to see any primary effects. This same procedure is done for discrete movements but using a sigmoid function of best fit, also calculated by the Matlab systems identification toolbox. Similarly, trajectory deviations would be calculated by subtracting this function from each cycle of non-TMS movements and then TMS movements to compare differences. The area of the curve was found by using integration, which represents the trajectory deviation.

4.2.3 Results

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Table 2 Average trajectory deviation for 4 subjects. Each cell represents the average deviation, by subtracting fitted trajectory with actual trajectory. For both discrete and rhythmic trials, TMS movements, non-TMS movements, and movements during sham TMS were included.
Both rhythmic and discrete movements saw slight errors in path trajectory when the PPC was stimulated, but among all 4 subjects, discrete movements had significantly higher end-point errors compared to rhythmic movements during stimulation (p<0.01). Subject 2 was the only outlier that did not have a significant effect when comparing discrete and rhythmic movements with TMS. Interestingly enough, discrete movements without TMS were naturally more varied compared to rhythmic movements without TMS. In order to account for this observation, we normalized the TMS trials to the non-TMS trials and also found there was a significant effect between normalized discrete and normalized rhythmic movements with TMS (p<0.05). There were no main effects when comparing non-TMS trials with sham-TMS trials.

Table 3 Average end point error for 4 subjects. Each cell represents the average position error by subtracting the true stop from the actual movement position. For both discrete and rhythmic trials, TMS movements, non-TMS movements, and movements during sham TMS were included.
Both discrete sham and rhythmic sham trials had significantly less end-point errors compared to the normal PPC-TMS movement trials, indicating that there was no immediate motor perturbation when stimulating in a neutral location, specifically the anterior temporal lobe (p<.05). Unlike with trajectory deviations, average end point error without TMS was higher in rhythmic trials compared to discrete trials. When we normalized the TMS trials with the non-TMS trial data, we found that the difference between discrete and rhythmic movements was even more pronounced (p<.01). Within subjects 2 and 3 sham-TMS had a slight effect on discrete movement trials compared to rhythmic trials, but this was not the case when take group data, where there was no significant difference (p>.05).

4.2.4 Conclusions

The literature has shown that the posterior parietal cortex is involved specifically in reaching and grasping movements that require visual attention and are goal-oriented (Reichenbach 2014). Additionally, fMRI data show that higher activity in the PPC is seen when performing discrete wrist rotations as opposed to rhythmic rotations, which saw little to no PPC activation. Since reaching movements fit the profile of discrete behavior, we hypothesized that TMS stimulation in the PPC would perturb discrete movements and have negligible effects on rhythmic movements.
Although in our pilot data there were perturbations in trajectory in both movement types, this could have been due to the visual display that showed the subject’s online hand position and targets. One confounding problem with our experimental setup was the visual dependency in both rhythmic and discrete behaviors. Normally, rhythmic behaviors are not heavily goal-oriented and do not require reaching for a target, this might explain why there was some effect in the deviations in intended trajectory when stimulating the PPC during rhythmic movements. However, the alternative was to use a self-paced rhythmic rotation and this would not be comparable to a discrete movement pattern with given targets. Additionally, self-paced rhythmic and discrete movements are more difficult to calculate intended trajectory, due the random periodic shifts that created non-uniform kinematics.

Although subjects 1 and 3 did have a significant difference between rhythmic trials with and without TMS, the group data suggest that there is no significant effect (p>0.05). Since the end-point variance in discrete movements was significantly higher between no-TMS trials, this supports the claim that discrete movements are reliant on the activity of the PPC. We suspect that errors in the second corrective submovement of a discrete arm rotation were caused due to perturbations in the PPC. End point accuracy for rhythmic arm rotation during TMS was not significantly different from the no-TMS condition. This result supports the claim that rhythmic movement regulation is not highly dependent on PPC activity, but is in discrete movements.
Chapter 5: Discussion

We have looked at movements and the movement system from a behavioral and physiological perspective. The behavioral perspective informed us of the external characteristics of movement and also allowed us to hypothesize on the demand this must create for the underlying physiological structures. As shown by the increased cortical activation during discrete movement, this type of behavior puts more cognitive demand on the neuromuscular system than rhythmic movements, even when the same muscles and joints are used (Schaal, 2004). Discrete movements result in greater cortical activation compared to rhythmic movements. Additionally, due to the goal-oriented nature of many discrete movements, a greater amount of attention is needed in performing discrete movements. Discrete movements rely heavily on attention and sensory feedback to inform the body on how to correct its motion. It is likely that each discrete movement has one initial submovement to move the limb toward its destination and at least one corrective submovement to account for any errors in trajectory and allow for a smooth movement towards the target (Novak 2002).

It is relatively easy to define rhythmic and discrete movements from an external perspective. However, such a clear distinction is more difficult when considering how these movement types are controlled by the neuromuscular system. Although there is clear evidence that rhythmic and discrete movements have vastly different brain activity
signatures, the true physiological underpinnings are still unknown. Moreover, it is unclear whether there truly are independent control mechanisms for these two movement types or a shared control mechanism for all movements types. Do we control the rhythmic and discrete aspects of a movement or do we control more fundamental motor elements that are more essential in the performance of these movement types? In the following paragraphs we will review, discuss and elaborate our previous introduction of the behavioral and physiological aspects of movement. We will also bring in relevant studies from a clinical perspective and propose possible models of rhythmic and discrete motor control that can explain our findings.

5. 1 Rhythmic Movements

As discussed in our physiological exploration of movement control, the spinal cord and brainstem largely control the rhythmic aspects of movement, specifically using central pattern generators. Although primary motor cortex is activated during rhythmic movements, it is unclear whether there are continuous motor commands emanating from the primary motor cortex during the steady-state rhythmic movement from the primary motor cortex. It is possible that continuous signals produced by the central pattern generators in the brainstem and spinal cord ease the computational demand on the primary motor cortex by directly recruiting muscles. In this way, the M1 serves as the primary command agent that delivers the motor command to lower motor
processing regions. The brainstem and spinal cord serve as a secondary and tertiary processing region and turn the singular motor command into a sequence of rhythmic activations that the muscles turn into a movement (Sherman et al 2015).

It is known that the corticospinal tract, which originates mainly from the primary motor cortex, descends through the brainstem and has side projections that modify activity of the brainstem. This is especially true in the midbrain where two important regions have high involvement in rhythmic movements: The mesencephalic (Midbrain) locomotor region (MLR) and the pedunculopontine nucleus (PPN). When electrically stimulated, the MLR has been shown to elicit continuous locomotor flexions and extensions in mice (Esposito 2016). The MLR is part of the reticular formation, which contains the reticulospinal tract, connecting nerve pathways in the brainstem such as the PPN, spinal cord, cerebellum, and cortex. A different study on non-human primates looked at the activity of the PPN during locomotion and at rest (Goetz et al, 2016). Using extracellular recordings, the PPN showed rhythmic bursts of activity during locomotion. These bursts were not seen when the same animal was at rest. Although it is still unknown how directly these results transfer on to non-locomotion rhythmic behavior, it is likely that the brainstem regions, especially in the midbrain, are involved in rhythmic movements and producing continuous sequences of behavior.
5.2 fMRI and TMS

Although the corticospinal tract has axonal projections to the brainstem as mentioned above, it also continues uninterrupted to the spinal cord. This construction allows direct control from the cortex to the spinal cord, while having a parallel control system from the brainstem to the spinal cord (Li et al 2015). Additionally, at the spinal level, compound reflexes offer a local means of coordinating and executing rhythmic activity within a group of agonist/antagonist muscles and across both sides of the body through the action of interneurons. Through fMRI imaging, scientists have found that the spinal cord has a high level of plasticity when learning new sequenced skills and have concluded that interneurons frequently change their connectivity and strength (Cohen 2016). Although higher cortical levels are normally responsible for sending the initial signal in voluntary rhythmic movements, it is these spinal organizing interneurons that organize the muscle sequence at the lower level (Vahdat 2015).

We have seen from fMRI data that M1 activation is seen during rhythmic human wrist movements and this activation is substantially different from discrete wrist movements, even when using similar muscles (Schaal 2004). However, due to the poor temporal resolution of fMRI data, it was unclear whether this M1 activation was prevalent throughout the entire rhythmic sequence or only at the beginning and end
of the entire behavior. In our TMS study on the M1, we did not observe any decrease in excitability during the middle of the rhythmic sequence compared to the ends. However, when we asked the subjects to transition between rhythmic and discrete movements or discrete to rhythmic movements, the MEPs were dramatically higher. This finding suggests that the beginning and end of a rhythmic movement sequence has a much higher involvement of the M1 compared to the steady-state cycles in the middle of the rhythmic movement.

In the same study, 8 phases were observed in rhythmic and discrete movements: 4 extension and 4 flexion phases. On average, the MEPs in rhythmic and discrete conditions were not significantly different within corresponding phases. However, rhythmic movements produced MEPs that were far less variable, suggesting that different control mechanisms were at play. A possibility in this decreased variation could be due to the fact that rhythmic movements had a continuous oscillatory nature and may also have predictability cyclical neural state changes that accompany it. This is unlike the discrete movement trials where a randomized time variable was imposed between each movement to prevent predictability. Periodic and predictable neural state changes would be more difficult if the cue to start moving would be different from one cycle to another. It may be possible that during the middle interval of rhythmic movements, less involvement of the M1 is required, favoring the automatic activity of the midbrain and spinal CPGs.
5.3 Constraints: Rhythmic and Discrete

Discrete movements have largely been studied under the narrower category of reaching and grasping movements using primates as subjects. One notable study looked at reaching of cats while stimulating the superior colliculus of the midbrain. Cats stimulated in this way had a notable deviation in their reaching trajectory and were often very poor at reaching their target (Courjon et al 2015). The same study compared reaching movements performed by monkeys under the same stimulation. Surprisingly, the monkeys did not have the same perturbation in movement and often had negligible effects on trajectory and successful target reaching. The superior colliculus, being part of the midbrain and containing the reticulospinal tract, should be important in achieving rhythmic behavior. However, it is evident that this area is clearly involved in reaching behaviors in cats. So then why is this trait not shared with the monkeys? It is possible that through evolution the apparent roles of the midbrain have been reorganized to favor rhythmic movement control over discrete control.

Rhythmic and Discrete movements consist of different elements, both externally on the behavioral level and internally at the physiological level. However, it would be incorrect to group all rhythmic movements under one general category without considering the possible variations or under which contexts each rhythmic movement is done under. The same is true for generalizing all discrete movements. Our pilot
studies observed the initial and end cycles of rhythmic movements and compared them to the middle interval cycles of rhythmic movements and individual discrete movements. The study probed the M1 cortex using TMS and preliminary data showed that the initial and end cycles of rhythmic movements corresponded to very similar cortical excitability compared to discrete movements. The MEPs for the initial and end cycles were statistically much higher than then MEPs captured in middle interval of rhythmic movements. Additionally, these MEPs were on par with the MEP amplitude of individual discrete movements. Through this example, rhythmic movement initiation and termination may involve discrete physiological elements, compared to the middle interval rhythmic movements, which may be driven by purely rhythmic physiological elements.

In examining rhythmic and discrete movements, a common problem is oversimplifying the behavior. Specifically, not all rhythmic movements have the same external execution criteria. Hand and arm movements, such as the previously mentioned example of waving your arm, do not have any explicit target to aim for, but this is not true for all rhythmic movements. Some rhythmic movements, have explicit targets, which do not change. An example is playing a trill on a piano, which is playing alternating two (or more) neighbor notes continuously. This example illustrates the initial need to aim and successfully achieve the correct two notes, confirmed by visual or auditory sensation. However, subsequent iterations of the same trill only require
you to repeat the two-note pattern, rather than aim for new notes. Interestingly enough, beginner pianists have difficulty doing this behavior and each successful press resembles a discrete movement, which greatly limits the speed at which the trill can be done. This example not only captures the nature of rhythmic and discrete movements, but also suggests the possible learning mechanisms for acquiring new skills. Since playing a trill or any piano composition is not an innate behavior, it would make sense that amateur performers do not have the necessary muscle activations sequenced and grouped together into higher level organizing elements, as is the case with muscle synergies (Bizzi and Cheung 2013).

Unlike cyclic finger movements on a piano, some rhythmic movements are almost autonomously learned by the body, such as the development of locomotion. As babies develop into toddlers, their musculature becomes more pronounced and they are able to move by crawling on all fours using their arms and legs. Interestingly enough, such movement patterns of four-limbed locomotion highly resemble non-human four-legged locomotion (Patrick et al, 2009). Using surface electrode recordings and high speed motion capture on crawling toddlers, it was clear that muscle activation patterns were similar to those found in the rat, cat, and macaque monkey. This suggests a phylogenetic conservation of locomotor patterns across mammals and likely indication of oscillatory motor primitives that are shared within these distantly related species. These results support the idea that, through evolution, older motor
primitives were retained and built upon rather than opting to overthrow and fundamentally reorganize the neural and motor elements of behavior. Further evidence of this can be seen when looking more closely at a toddler’s first bipedal steps. Each step resembles a discrete reaching movement, but performed with the leg and foot. It is possible that this novice walker is learning a new synergy by organizing already existing motor elements (Righetti et al 2015).

5.4 Rhythmic and Discrete Movement Disorders

Most toddlers naturally develop locomotive patterns through development and experience, but there are some genetic conditions that disrupt this progression. A study looked at behavior of children that had developmental coordination disorder (DCD), previously named clumsy child syndrome. DCD is the lack of coordination with normal movements, such as walking, sitting, and talking, in developing children’s behavior due to a cognitive-motor deficiency (Piek et al 2008). This deficiency does not normally affect intelligence and may be observed early in childhood, even before a child starts walking. Inability of newborns in learning how to suckle and swallow as well as noticeable impairments in sitting, crawling, and walking are common in DCD. In fact, children with DCD often skip the crawling phase of locomotion completely, which is highly rare in normal motor development (Geuze, 2001). A further study discussed the decreased control of eye movement as well as manual control in
children with DCD. The same study saw that children with DCD had a reduced parietal activation, which might explain sensory deficits and difficulties with integrating sensory information with motor output (Kashiwagi, 2009). Researchers believe that one primary cause of this motor deficiency lies in the way the brain develops and likely is linked with genetic factors. This fact that DCD children skip crawling altogether may indicate some problem with the underlying integrity of one’s motor primitives in performing and learning new movements and skills.

While, babies take their first steps discretely, before they their bipedal motion becomes truly rhythmic, many patients with progressed Parkinson’s disease have serious deficiencies in initiating and changing gait while walking. These patients, often have no problem in continuing rhythmic movements when they start, but initiating these movements can be very difficult and often a large amount of physical therapy is necessary to regain some control (Wu and Hallet, 2005). Additionally, when a simple transition is added to the walking gait, such as a turn or a pivot, the PD patients often have a difficult continuing in their rhythmic walk, freeze up their muscles, and have some degree of tremor in their legs and hands. In this case, the rhythmic elements of movement are functioning well, however the discrete elements that begin each movement are malfunctioning. In our model, the rhythmic elements are all located in the brainstem and spinal cord, whereas the discrete elements require some level of cortical activation, especially in the M1.
PD patients have a reduced dopamine production and have a poor connection with the reward pathways in the brainstem. The presence of Lewy Bodies, abnormal protein aggregates, in areas of the brainstem is likely the cause of many motor deficits (Seidel et al 2015). High numbers of Lewy Bodies have been identified in the substantia nigra, ventral tegmental area, pedunculopontine and raphe nuclei, and reticular formation. The reward pathways are highly reliant on the substantia nigra, as it produces most of the brain’s dopamine, and the raphe nuclei, which produces the majority of serotonin involved in motivation and are often used in movement production involved with the basal ganglia (Miguelez et al 2016). The rest of the nuclei are directly involved in motor production by synapsing to brainstem nuclei that project to the descending corticospinal and reticulospinal pathway (Ortiz-Rosario et al 2014).

Apart from activity in the primary motor cortex, fMRI show that discrete-reaching movements activate the posterior parietal cortex (PPC), which is all but silent during rhythmic movements (Schaal 2004). The PPC is often used as a sensorimotor hub that uses incoming sensory feedback to update reaching behaviors and ensures that the target is reached. Without any apparent effort, we are able to adjust our reaching movement using incoming visual and proprioceptive feedback to complete our goal directed task (Reichenbach et al 2014). The continuous feedback and motor updating
supports the dynamic primitive model that discrete movements are updated by submovements. Unlike discrete movements, rhythmic movements are not required to be continuously updated, which presents a stark distinction in control between these types of movements (Prochazka 2010). In fact, fMRI scans show that rhythmic movements have a reduced activation in other cortical areas that are responsible for motor control compared to discrete movements. Evidence shows that discrete movements recruit a larger cortical network than rhythmic movements, especially in the PPC, the supplementary motor cortex, and the premotor area (Leconte et al 2016). The same study showed that patients with stroke have discrete movements more affected than rhythmic ones. Additionally, when these stroke patients had undergone motor standard motor rehabilitation, such as physical therapy, greater recovery rates were seen in rhythmic movement production as opposed to discrete movement production. Since stroke often affects cortical structures more than deep brain structures, this supports the idea that discrete movements rely more heavily on cortical motor control compared to rhythmic movements.

As we have seen, the posterior parietal lobe has a large involvement in production of sensory guided reaching and grasping tasks. In many children with infantile autism, their parietal cortex has a notable decrease in volume compared to same-age children without autism. Many symptoms of autism spectrum disorder include cognitive-behavior deficits, such as decreased sensory-perceptual processing, sudden bouts of
rhythmic movements, and decreased spatial accuracy when reaching for and manipulating objects (Amaral et al. 2008). These deficits give further support to the role of the parietal lobe in performing discrete movements. The rhythmic episodes that children with autism exhibit deserve a closer look. There are three rhythmic movements that are commonly present in children with autism: head-banging, body rocking, and arm flapping. These behaviors are also common in children with a related disorder, Rhythmic movement disorder (Richard 2005). In fMRI studies, both groups of children with either disorder have a strikingly abnormal activation in the thalamus and cerebellum.

Previous studies found that there are markedly different functional relations between cerebral-brainstem activity compared to children without autism spectrum disorder and rhythmic movement disorder (Kinsbourne 1987). These studies are expanded upon by new findings that trace early embryo abnormalities in the developing brainstem (Delafield-Butt and Colwyn 2017). Specifically, functional disruptions in the inferior olive of the brainstem have been linked to irregularities in timing of cerebellar signals. As the timing of these signals have been linked to the proper development of motor and affective properties of the brain, consistent delay in these signals lead to dysfunctional cognitive development. Although many scientists still are unsure of how these uncontrolled rhythmic body movements are produced in children with autism spectrum disorder and rhythmic movement disorder, evidence supports the idea that
dysfunctional brainstem development is associated with poor motor and emotional control seems likely.

By observing the movement system when the brain is not functioning properly, we are able to gain better insight into how certain aspects of movement are controlled and operated. The clinical perspectives of patients with developmental coordination disorder, Parkinson’s disease, some types of stroke, rhythmic movement disorder, and autism spectrum disorder all add to our understanding of how the nervous system controls movements and how specifically rhythmic and discrete movements are differentiated. In the disorders considered here, dysfunctions in brainstem, thalamus, and cerebellar pathways have led to difficulties in rhythmic movement production. Even though other aspects of movement are compromised as well, rhythmic movements are notably compromised compared to discrete movements. Stroke patients, on the other hand, have less deep brain damage and usually preserve and regain rhythmic movement production after recovery. However, discrete movements are more affected, making reaching behaviors as well as initiating rhythmic behaviors more difficult. Interestingly, PD patients have problems initiating and transitioning from one rhythmic movement to another, but often have no problem continuing rhythmic movements when started. PD involves degeneration in many aspects of the brain involved in movement and is not just limited to the brainstem. Although rhythmic movements are compromised, discrete movements also suffer decreased
coordination. Some explanations of this can be seen in the overall decrease in cortical activation in PD patients over the frontal and parietal regions known for being involved in sensory-motor processing and reaching behaviors.

5.5 Concluding Thoughts

A large body of evidence indicates that rhythmic and discrete movements have different control mechanisms and activate radically different areas in the nervous system. Rhythmic movements largely use the brainstem and spinal CPGs to repeat their cyclic contractions, while having little cortical activation. Discrete movements have high cortical activation, especially in the primary motor cortex, posterior parietal cortex, and supplementary areas. Although initiation of rhythmic movements resembles that of discrete movements, through commands from the M1, later middle interval rhythmic activity shows a decreased activity in the primary motor cortex. Even though both rhythmic and discrete movements share the same final common pathway in the corticospinal tract, these movements are processed and controlled differently at many levels of the motor system.

Many of our rhythmic movements develop innately, such as scratching, walking and breathing. However, evidence shows that new rhythmic movements are able to be formed by means of plastic connections in the spinal cord and the formation of new
muscle synergies. Many such movements begin as the combination of discrete movements, such as single note piano playing, to more robust rhythmic ones, such as playing a sequence of notes in a trill. This learning paradigm suggests that discrete movement sequences, which require more attention and goal-orientation, can be automatized into rhythmic movements. This model for skill learning and motor control across cortical areas, brainstem, cerebellum, spinal cord and likely the basal ganglia allows for a diverse repertoire of movements. A combination of discrete movements requires a great deal of conscious effort and cortical activation to perform. However, when the same movements are sequenced and grouped into rhythmic motor synergies, the amount of cortical control is decreased and ability to easily recall and perform such rhythmic movements is increased. As discrete movements are grouped and rhythmic synergies are made, the dynamic nature of the rhythmic and discrete movement production and control is made clear. Although there are clear signs that there are shared motor control mechanisms governing movement as a whole, there is also a hierarchical separation between rhythmic and discrete movement control.

In exploring the behavioral aspects of movement, we have become aware at the different superficial characterizations of rhythmic and discrete movements. Additionally, we also see how these movements are done in context and when each movement type is needed. Although many movements are a combination of discrete and rhythmic elements, pure rhythmic and discrete movements contain many striking
differences on the behavioral level. Physiologically we have seen that discrete
behaviors rely heavily on cortical activation and integrate many sensory-perceptual
inputs with the final motor output. Discrete behaviors are created with several
corrective submovements insuring that the final target is reached with accuracy.
Rhythmic movements, on the other hand, rarely contain submovements during their
periodic movements. Even though we see primary motor cortex activity during the
initiation of rhythmic movements, the repetitive oscillatory muscle contractions are
primary controlled by central pattern generators distributed in the brainstem and
spinal cord. Although more research is needed to elucidate specific control
mechanisms underlying rhythmic and discrete movements, the clear benefits lie in the
distributed structure of the neuromuscular system in dynamically controlling these
movements as needed by the environment.
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