

Neural Correlates of Motor Vigour and Motor Urgency During Exercise

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Abstract This article reviews the brain structures and neural circuitry underlying the motor system as it pertains to endurance exercise. Some obvious phenomena that occur during endurance racing events that need to be explained neurophysiologically are variable pacing strategies, the end spurt, motivation and the rating of perceived exertion. Understanding the above phenomena physiologically is problematic due to the sheer complexity of obtaining real-time brain measurements during exercise. In those rare instances where brain measurements have been made during exercise, the measurements have usually been limited to the sensory and motor cortices; or the exercise itself was limited to small muscle groups. Without discounting the crucial importance of the primary motor cortex in the execution of voluntary movement, it is surprising that very few exercise studies pay any attention to the complex and dynamic organization of motor action in relation to the subcortical nuclei, given that they are essential for the execution of normal movement patterns. In addition, the findings from laboratory-based exercise performance trials are hampered by the absence of objective measures of the motivational state of subjects. In this review we propose

that some of the above phenomena may be explained by distinguishing between voluntary, vigorous and urgent motor behaviours during exercise, given that different CNS structures and neurotransmitters are involved in the execution of these different motor behaviours.

1 Introduction

Scientists have been familiar with the macro-anatomy of the brain as we currently understand it for the best part of 150 years [1]. In contrast, information about the molecular features and circuitry connecting these various anatomical structures is continually increasing in complexity; with this increase becoming exponential over the last 45 years [2].

From text book physiology we know that the execution of voluntary movement, such as that exhibited during exercise, requires muscle recruitment via descending nerve impulses from the motor cortex that synapse with alpha motor neurones situated in the ventral horn of the spinal cord [3]. A more crucial concept that needs to be described neurophysiologically is the fact that the motor cortex does not work in isolation. Rather, there is active regulation of the motor cortex itself [4–6]. Given that sporting performance is dependent on a functional motor system and is also influenced by psychological factors [7–9], it is perhaps time to explore more of the motor and psychobiological circuitry lying ‘beyond’ the motor cortex.

2 Historical Development of Motor Control During Exercise

Santiago Ramon Y. Cajal was the first to provide a detailed analysis of the neuronal circuitries underpinning both

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reflexive and voluntary movement with his famous 1894 Golgi stained illustrations [2]. Two decades later, Krogh and Lindhard [10] tested subjects during heavy cycling exercise to examine the cardiorespiratory changes that occurred during the initial stages of exercise and were led to conclude that it was not only the skeletal muscles, but also the respiratory centres, that were up-regulated by “an irradiation of impulses from the motor cortex”.

Subsequent researchers rarely looked beyond the motor cortex—although ‘central irradiation of impulses’ was modified to, amongst others, ‘central command’ [11, 12], ‘voluntary activation’ [13] and ‘central motor drive’ [14]—as the causal agent driving motor behaviour during exercise [10, 15–18]. Williamson and co-workers linked central command to activation of the insular cortex [19], and showed that this brain area was involved with the regulation of the autonomic nervous system during exercise [20].

While exercise scientists were focusing their research efforts on the circulatory and muscular demands of exercise, neuroscientists set out to measure the neuronal connections between the brain and the circulation and muscles. By the early 1970s, landmark neurophysiological studies, led by researchers like Thach, Evarts and DeLong, had already established that activity in the cerebellum [21, 22], basal ganglia (BG) [23, 24] and thalamus [25] preceded movement, which led Evarts to surmise on p 243-4 [26]:

“... the basal ganglia and cerebellum receive information from all regions of the cerebral cortex, transform this information, and then send a new pattern of signals to the motor cortex. Whereas the traditional view held that the cerebral motor cortex was at the highest level of motor integration and that the subcortical structures were at a lower level, that is, closer to the muscle, it now appears that the situation is quite the reverse ... the motor cortex is more directly connected to the spinal cord motoneurons than either the cerebellum or the basal ganglia. All this is consistent with Phillips’ view that the motor cortex should be thought of in relation to the middle level of Jackson’s hierarchical organization”.

Evart’s so-called ‘new pattern of transformed information’ from the BG and cerebellum is sent back to the motor cortex via the thalamus, an important integrator of motor, cognitive and emotional inputs [27] and also a modulator and driver of afferent sensory inputs [28]. Both the BG and cerebellum have extensive neural input into the motor cortex via the thalamus [29, 30] and can be functionally subdivided into drivers of (1) internally generated (IG) movement speed—via BG and prefrontal cortex (PFC) [31]; and (2) externally or visually triggered (VT) urgent movement—via the cerebellum [32, 33].

IG movement speed has been defined as ‘motor vigour’ to distinguish the energy cost of movement from the accuracy of that movement in individuals suffering with Parkinson’s Disease (PD) [34]. These PD sufferers chose to move slower (i.e. paced themselves) because of the relatively steeper energy costs associated with normal movement speed than that found in healthy individuals. Given that PD is a BG-related disorder suggests that the BG are important for the regulation of IG movement speed, or, more specifically, with the energy costs associated with movement [2]. This agrees with Robbins and Everitt’s [35] conclusion in laboratory animals that the vigour and frequency of ‘behavioural activation’ (i.e. movement speed changes caused by changes in internal arousal) is regulated by the dorsal striatum, forming the input nuclei of the BG [27].

On the other hand, upregulation of externally or VT urgent movement as mediated by the cerebellum [36] has been termed paradoxical kinesis or ‘motor urgency’ [37]. Motor urgency is best elicited when an urgent external situation is cued by a visual trigger [38]. This effect was tested in patients with Parkinson’s disease (PD) who attended a referral area in Northern Israel exposed to enemy Katyusha and mortar rocket attacks for 1 month during an ongoing war. It was discovered that a 65-year-old male patient, who had been unable to run for many years and could only walk about 12 steps before losing his balance, was nevertheless able to run after his wife when a rocket warning siren sounded. Significantly, this patient was unable to elicit more than a shuffle during dozens of other rocket warning sirens before and after this incident. According to the patient, the difference during this particular warning siren was that his wife grabbed his arm while he was slowly getting out of his chair and he then ran following in her exact footsteps [38]. The interpretation is that he was able to observe/visualize (1) how to run when he (2) saw his wife performing the activity in a (3) urgent situation, with motor drive from the cerebellum rather than the dysfunctional BG. It is probable that motor urgency plays a key role when sprinting for the finish line during an endurance race, where visualizing the end is probably as powerful a VT as physically seeing the finish line [36].

3 Primitive Versus Fine Movement Control

Given that the focus of this review is on endurance running and cycling races longer than ~30 min, an important concept that needs qualification is the distinction between fine and primitive motor control [39]. To illustrate by example, a primate with a primary motor cortex (M1)

lesion and unable to grasp a nail to open a box containing a peanut (fine motor control) was nevertheless able to grasp a wire of the same thickness for the purpose of climbing (primitive movement) [40]. This finding may be explained by the fact that affectively triggered (i.e. subcortically generated movement in the fulfilment of a biological need such as hunger or reproduction) gross motor behaviour is primarily regulated by the BG [41].

The BG regulates subcortical locomotor regions (LR), identified in the mesencephalon (MLR), diencephalon [42] and in the cerebellum [43] that are able to independently generate locomotor patterns [44–47]. There is also evidence that brainstem locomotor regions and central pattern generators (CPGs) in the bipedal human [43, 48–50, 167], are functionally similar to that described in quadruped vertebrates [51–54], and in electric CPG models [55]. Further support for the functionality of locomotor pattern generators in humans come from the fact that direct stimulation of the lumbar spine elicits locomotor patterns in paraplegics [56], and from EMG recordings, which show that precise motor programmes (not under cognitive control) are activated to initiate walking at different speeds [57]. Indeed, a recent review supports not only patterned locomotion in humans, but provides evidence for patterned control during many other motor behaviours in humans, e.g. swimming, jumping and arm reaching [58].

The important lesson from the primate with the M1 lesion is that, while direct cortico-motoneuronal input may be imperative for motor activities requiring fine motor [59] and fine force control of the upper limbs [60], and during motor learning [61, 62], it is not as essential for the more primitive locomotor control of the lower limbs [60] necessary to run and cycle [39, 57, 63, 64]. This concept extends the transient hypofrontality hypothesis proposed by Dietrich and Audiffren, whereby, allocation of metabolic resources are shunted away from brain areas not critically needed for execution of exercise, areas like the PFC and limbic system [65], to include substantial parts of the motor cortex, which is deoxygenated during high-intensity exercise [66] to the list of brain areas not absolutely essential for goal acquisition.

Computer simulations have shown that bicycle pedalling requires as few as three brainstem-generated signals that drive six different muscle groups (three for each leg) to control variables like cadence, power output, efficiency and smoothness of the pedalling action [63]. Thus, the major contribution from the cerebral hemispheres (motor cortex and BG) in cycling is presumably the recruitment of additional muscle fibres and the coordination and modulation of the overall excitatory drive to the brainstem locomotor regions that control spinal CPGs to ensure fulfilment of biological needs [42, 67].

4 Goal Directed Movement

It is challenging to describe voluntary movement, or feed-forward control ‘irradiating from higher brain centres’, physiologically; however, Bernstein had set the stage by mapping out the putative brain structures needed to coordinate the ‘actions of the organism upon their environment’ in fulfilling biological needs in a 1947 paper [68]. It is in meeting these biological needs in the outside world via goal-directed actions [69] that humans engage in voluntary movement and, by extension, in voluntary exercise. The formulation of the goal to be executed represents the top level of Dietrich’s [70] hierarchical organization of consciousness and is localized to the dorsolateral part of the PFC [71], while the medial frontal cortex (MFC) is essential for incentivized action selection [72].

In this regard, neuroscientists and clinical neurologists alike accept the Sherringtonian notion that the only part of voluntary movement that is volitional is its goal or purpose, which also serves to distinguish it from involuntary movement (e.g. Tourette’s syndrome and choreiform movement) that is goalless [26]. Once the goal is clearly formulated the motor responses necessary for goal acquisition are made up of a variety of reflex processes [26, 73]. For the purposes of this paper, the ‘goal of the intended movement’ will be seen as the signal from the PFC, not the motor cortex, to initiate movement [74]. In this regard, direct measurement of pyramidal tract neurons in the motor cortex clearly established that it is the goal of the movement rather than the afferent sensory input into the motor cortex that regulates the motor cortex [26]. Given that motor cortex activation effects movement, the purpose or goal of the intended movement would necessarily be the primary regulator of the motor cortex [75].

5 Motor Cortex Versus Basal Ganglia in Motor Control

There are five cortico-basoganglionic-thalamo-cortical circuits [76–78] consisting of motor, oculomotor, motor association, prefrontal and limbic circuits in the human brain [79]. These circuits are anatomically segregated, largely closed and re-entrant [25, 78] that is, they start and end in similar cortical areas and thereby tightly link the frontal cortex with the BG and the thalamus, but also allow for the continual updating of information [27] (Fig. 1). An examination of the disturbances resulting from brain lesions in human patients gives us some insight into the functional importance of the caudate nuclei, forming part of the dorsal striatum of the BG. In a case study, Martin described a Parkinsonian patient who displayed ‘irresistible propulsion’ for long periods of his illness, observing as

information. Apart from novel stimuli, motor cortex inhibition is also brought about by afferent feedback from fatiguing quadriceps muscle during exercise [87] associated with insular activation [19], and Hilty and co-workers found increased thalamo-insular activation just prior to handgrip exercise task failure [88]. These authors also found a fatigue-induced increase in communication between the insular and motor cortices during cycling exercise [6]. Presumably, afferent feedback during endurance exercise will enter the cortico-BG-thalamo-cortical loops [76] via the thalamus [3] to continually update goal-directed behaviour and bring about motor cortex inhibition as needed to prevent premature fatigue [87]. However, if the exercise is at a set work load rather than self-paced, task failure is seemingly preceded by activation of a thalamo-insulo-motor cortical circuit [6, 88].

This is presumably the level at which the physiological control of pacing is situated. During shorter endurance events the immediate goal of the athlete presumably leads to greater striatal inhibition of the pallidum. The pallidum has extensive inhibitory neural inputs into the motor cortex via the thalamus [30], thus inhibition of the pallidum would lead to greater motor cortical [89] and presumably brainstem locomotor centre activation [67], resulting in greater motor vigour during shorter than more prolonged races (Fig. 2).

In this regard, subjects who randomly completed 5 km, 10 km, 40 km and 100 km cycling time trials, were able to produce greater average power outputs throughout their 5 km time trials than they were able to during their 40 km and 100 km time trials [90]. One way of explaining these findings is that the relatively lower motor vigour of subjects during their 40 km and 100 km than during their 5 km time trials is due to subjects' ongoing cost-benefit calculations [7, 91] and the so-called 'net reward rate' [92]. The net reward rate is equal to the 'influx of net benefit per unit time' whereby subjects continuously weigh up, most likely by way of self-talk [93] whether the lower 'energy cost' of performing any action more slowly is worth the lost benefit due to the extra execution time (Fig. 2) [92]. And the perceived benefit of any particular action is directly proportional to the tonic level of striatal dopamine [94] such that motor vigour is either up or downregulated to precisely match the perceived benefit as set by the tonic striatal dopamine levels [95].

This further suggests that the decision of subjects to invest a lower 'energy cost per unit time' during their 40 km and 100 km versus their 5 km time trials presumably resulted from relatively lower tonic striatal dopamine levels [96] during the longer versus shorter time trials; and the relatively lower tonic striatal dopamine levels during the longer time trials can be explained by global motor inhibition; a BG-mediated affective response that

optimizes the probability of goal achievement. Given that the longer time trials may well have resulted in exhaustion before completion at the average 5 km power output, striatal inhibition of the pallidum would have released [84, 85] only that part of the motor system needed to optimize the probability of finishing the longer time trials even if this meant a longer execution time. Presumably, goal optimization in the laboratory would be slanted more towards finishing, while goal optimization during an important sporting event would be slanted more towards speed [97].

7 The Behavioural Effects of Dopamine in the Striatum

Pessiglione and co-workers' functional magnetic resonance imaging (fMRI) research directly linked the monetary reward prediction errors in the behavioural choices of humans to dopamine-dependant modulations in the striatum [98]. fMRI research also identified distinct neuronal activations subsuming the delay costs (ventral striatum and the ventromedial PFC) and the expected energetic costs (anterior cingulate cortex and the anterior insula) associated with pursuing an erotic reward [99]. More recent research showed that the striatum is also important for feedback evaluation related to goal achievement in a motor task [100]. These researchers examined fMRI brain activations in their subjects while subjects evaluated their performance on a force matching handgrip task. Activations in the subjects' dorsal striatum were greater after those trials where a monetary reward was on offer, irrespective of whether their handgrip performance was good or not, while activations in the ventral striatum were greater following good versus bad handgrip performances [100].

As a corollary, one would expect that handgrip matching performance would be disassociated from monetary reward in persons with BG lesions. One such patient group presenting with BG lesions was identified by Laplane and Dubois in 2001, which they aptly named auto-activation deficit (AAD) [101]. fMRI work with an AAD patient group presenting with bilateral striato-pallidal lesions showed that handgrip-matching performance was indeed disassociated from monetary incentive [91]. Crucially, these AAD subjects' maximal handgrip force and instructed handgrip-matching force were similar to that of healthy control subjects, but they nevertheless failed to increase their handgrip force output when greater monetary rewards were offered, contrary to the healthy control subjects who increased their handgrip force output. In summary, the above research suggests that dopamine modulation in the striatum is crucial for the up- and downregulation of IG movement vigour according to cost-benefit analyses.

Palmiter in his extensive research to unearth the ‘process by which animals become energized to initiate goal directed behaviour, concluded that the midbrain release of dopamine into the dorsal striatum is the critical pathway to effect energized behaviour [107].

8 Dopamine Release into the Striatum

Dopamine enters the striatum via synaptic release from midbrain dopamine neurons situated in the substantia nigra pars compacta (SNc) and the ventral tegmental area (VTA) [108]. Synaptic release of dopamine into the striatum can be modulated in a number of ways as follows:

- (1) By the ascending reticular activating system (ARAS) acting through the pedunculopontine tegmental nucleus (PPT) in the brainstem [109], which preferentially stimulates the SNc, but also the VTA dopamine neurons in the midbrain [110];
- (2) by rewarding/aversive stimuli that directly activate/depress the midbrain dopamine neurons [111–113];
- (3) by inputs from the hippocampus that up- or down-regulate the percentage of spontaneously firing midbrain dopamine neurons [114].
- (4) Significant correlations have also been found between personality traits, like novelty seeking and reward dependence, and activation of both SNc and VTA dopamine neurones [115].
- (5) Linking it to exercise, 6 weeks of wheel running training in rats resulted in a downregulation of basal dopamine (D₂) autoreceptor messenger RNA in the SNc, which would enable greater synaptic dopamine release into the striatum and increase exercise drive [116] (Fig. 1).

While the tonic striatal dopamine levels are pre-eminently dependent on the release of dopamine from the midbrain into the striatum, factors other than the amount of synaptic dopamine release also play a role. Factors like (1) the interplay between the striatal acetylcholine and dopamine neurotransmitter activities [117] (see Sect. 9); (2) whether the striatal medium spiny neurons are in an up- or down-state [108]; (3) the nature of the cortical input into the striatum [118, 119] and, additionally; (4) behaviourally salient burst firing is only possible in those midbrain dopamine neurons that are spontaneously active [120].

In the rat, midbrain dopamine neurons make ~2,700 million synaptic contacts with the striatum [121]. Considering that about 70 % of the afferent input into the midbrain dopamine neurons are inhibitory GABAergic inputs [122] that serve to depress movement, the upregulation of synaptic dopamine release into the striatum (via

disinhibition and/or excitation of the midbrain dopamine neurons) have the potential to hugely impact motor vigour. Furthermore, each midbrain dopamine neuron that releases synaptic dopamine into the striatum has extensive axonal and dendritic arborizations that make between 10,000 and 100,000 synaptic contacts with other neurons [123]. Thus, midbrain dopamine neurons are ideally suited to have a tonic influence on brain function. BG function is remarkably similar across all vertebrate species, from fish and amphibians through reptiles, birds to mammals [124], suggesting that these findings in rats are broadly transposable to humans.

9 Striatal Dopamine Versus Acetylcholine Activity

Apart from dopamine, the striatum also contains tonically active cholinergic interneurons that release acetylcholine into the striatum. The effects of acetylcholine and dopamine in the striatum have traditionally been interpreted as antagonistic only [125, 126], but recent findings suggest a more sophisticated interaction [117]. Striatal acetylcholine acts presynaptically to strongly polarize how opposing dopaminergic neuron activities are transduced into dopamine release. A pause in the tonically active cholinergic neurons seems to presynaptically filter the effect of the dopamine neuron activity at the dopamine release site [117].

Tonically active cholinergic neurons don’t fire in relation to body movements, but respond to sensory stimuli associated with reward [127]. Sensory stimuli reporting a reward elicit a pause response in the tonically active cholinergic neurons in the striatum and, simultaneously, burst discharges in dopamine neurons in the SNc. The pause in the acetylcholine release amplifies the dopamine signal. The striatum is functionally divided according to the behavioral relevance of a stimulus [127].

Furthermore, salient sensory signals also increase acetylcholine release in sensory areas to enhance cortical processing of thalamic inputs to enhance cognitive flexibility [128]. Cognitive flexibility is important as it enables humans to override those salient stimuli, with concomitant burst discharge of midbrain dopamine neurons, which may be harmful in the long run, e.g. drugs of abuse [96, 103, 104, 129]. However, cognitive flexibility, as mediated by forebrain acetylcholine, may well interfere with the more primitive motor control, as modulated by striatal dopamine, necessary for running or cycling by slowing or stopping exercise to attend to an unexpected/interfering stimulus [130]. The brain areas associated with the upregulation of primitive motor control during motivated behaviour has been extensively modelled in animals [131] and will be covered next.

10 Behavioral Control Column and Behavioural State Controller

Disconnection of the cerebral hemispheres from the hypothalamus and brainstem in the cat does not prevent spontaneous ingestive, reproductive and defensive behaviour in response to a direct sensory input [2]. However, these animals are unable to anticipate, pointing to the necessity of cortical input for the formulation of anticipatory goal-directed movement and pacing during exercise. Animals with a disconnection at the hypothalamic/midbrain junction display no spontaneous movement, attesting to the importance of these brain areas for movement initiation and regulation. In an impressive review paper, Swanson laid out the neuronal circuitry in the hypothalamus and midbrain involved in the regulation of motivated behaviour in animals [131]. In this paper he introduced his so-called ‘Behavioral Control Column’ (BCC), tasked to initiate and coordinate all motivated behaviours [131].

Swanson further proposed a ‘Behavioural State Controller’ (BSC) working in conjunction with the BCC, similarly located rostrocaudally from the medial hypothalamus to the midbrain [132]. The BSC facilitates intrinsic motivated behaviour by upregulation of not only the skeletomotor and visceromotor systems; but also the secretomotor system via a host of neurotransmitters and hormones released by neuron populations located within the proposed BSC (Fig. 2) [132]. Note that Swanson specifically referred to ‘motivated behaviour’ in defining his BSC and distinguished it from voluntary (i.e. under cognitive control [133]) and reflexive behaviour [131]. In Swanson’s model, the BCC and BSC subsumes the circuitry necessary to mediate the three basic types of motivated behaviours, defensive, ingestive and reproductive, essential for survival. It follows that the greater the threat to life and limb, the greater the upregulation of the three motor systems to ensure biological goal attainment [7].

Research has established that it is particularly the midbrain (caudal part of the BSC) release of dopamine into the BG input nuclei or striatum that regulates motivational drive [92, 103–107]. Thus, while motivated behaviour is more primitive, given that it is driven by the midbrain and cerebellum [134], this drive is nevertheless coordinated by the BG situated inside the cerebral hemispheres. Additional neuronal circuitries from the cortex to the BG enable higher organisms to exert voluntary cortical control over the brainstem locomotor centres [84].

Swanson’s BCC and BSC are based on animal behaviour studies [131]; nevertheless, Swanson’s distinction between motivated, voluntary and reflexive behaviour fits easily into the exercise continuum in humans as well. Reflex behaviour includes maintenance of homeostasis and brainstem reflexes like breathing [42] and postural muscle

tone [46, 134]. Voluntary behaviour occurs during participation in exercise in general, for example, to maintain fitness or to lose weight; while motivated behaviour describes the upregulated exercise intensity needed to achieve a more specific goal like winning a race or setting a personal best time in the face of stiff competition. Note that any form of exercise participation requires some form of motivation, so it is better to think of a continuum from purely voluntary exercise to purely motivated exercise, with most exercise bouts falling somewhere in between. Escaping a life or death situation would be predominantly motivated, while walking to get from the car to the office is a predominantly voluntary action. The nature of the goal serves to distinguish more motivated from more voluntary exercise in that the performance of motivated exercise necessarily requires more intensive training and peaking and also greater arousal levels before and during exercise compared with the performance of voluntary exercise. Note, the periaqueductal grey (PAG) and hypothalamus are under modulatory control of the frontal cortex via, amongst others, the amygdala [135–137]. Thus, motor drive during motivated goal achievement coordinated by the BCC and the BSC is upregulated both via frontal cortical disinhibition and amygdalar excitation.

11 Cortical Override of Pain via Stress-Induced Analgesia

While non-opioid analgesics increase pain tolerance and exercise performance [138], stress-induced analgesia (SIA) operating via the opiate system, may well achieve the same effect without drugs. Even without stressful conditions endogenous opioids results in behavioural activation in mice [139]. Within a stressful and/or painful situation, the release of endogenous opioids is a well-known phenomenon investigated as placebo effect, which has been shown to be dependent on the PFC [140]. Recently the impact of the placebo effect, classically limited to clinical or experimental settings, has been broadened to physical performance [141]. Compared to a control group without conditioning the conditioned participants were able to tolerate pain, resulting from squeezing a hand spring exerciser under ischaemic conditions, significantly better [141]. Endorphin release in endurance sports (runner’s high), independent of conditioning, has been shown in a positron emission tomography study [142]. This is indicative of cortical overriding of fatigue via a rewarding subjective experience, which would be reflected in the RPE (Fig. 2) [138].

An alternative pathway of SIA is the endocannabinoid system. In the PAG stress produces two endogenous cannabinoids, the lipids 2-arachidonoylglycerol and

anandamid, which might mediate SIA [143]. The highest concentration of the cannabinoid-1 (CB₁) receptor is found in the BG, especially the output nuclei, and the cerebellum [144] suggesting a strong influence of the endocannabinoid system in both motor vigour and motor urgency. Cannabinoids seem to modulate primarily the GABAergic and glutamatergic synapses, but recent findings identified a direct influence on the dopamine system via the transient receptor potential cation channel, vanilloid-1 receptor [145].

This then leads us to another form of motivated behaviour, a neural driver of the motor cortex via external prompts that needs to be described neurophysiologically, the end spurt.

12 Motor Urgency—The End Spurt Phenomenon in Exercise

An fMRI study showed that motor urgency engages the cerebellum and the sensorimotor cortices bilaterally [36] that agree with fMRI work that linked sensory movement processes to the cerebellum [146]. Given that neural inputs from the cerebellum into M1 and premotor (PM) cortices are profuse [30], it suggests that the various sensory stimuli surrounding the approach to the finish would provide additional drive to M1.

This fMRI work follows on from direct electrophysiological recordings of single motor cortical neuronal cells by Edward Evarts [147]. Evarts found that primates, who were psychologically prepared for an anticipated stimulus (psychomotor set), had much quicker motor responses compared with when M1 was not primed [147]. Evarts proposed that the ‘set’ signal that primed M1 came from the supplementary motor and PM cortices, as well as from the PFC. He further proposed that the ‘go’ signal that targeted the primed M1 cortex came from the dentate nucleus in the cerebellum. From his primate data, Evarts surmised that the cerebellum go signal would activate M1, if and only if M1 was primed by an anticipatory set signal (Fig. 3).

We thus propose that during an endurance event, the athlete’s M1 would be primed by teleo-anticipatory set signals. Visualization of the end, especially during the last 10 % of the event [90, 148], presumably serves to initiate the go signal from the cerebellum necessary to maximally activate M1 within the constraints of parameters such as muscle glycogen/total carbohydrate reserves [149, 150] and by external conditions such as heat [151]. In this regard, Kay and co-workers showed that muscle recruitment during five of six 1-min all-out maximal sprints completed one every 10 min during a 60-min performance cycle were constrained relative to the last sprint completed

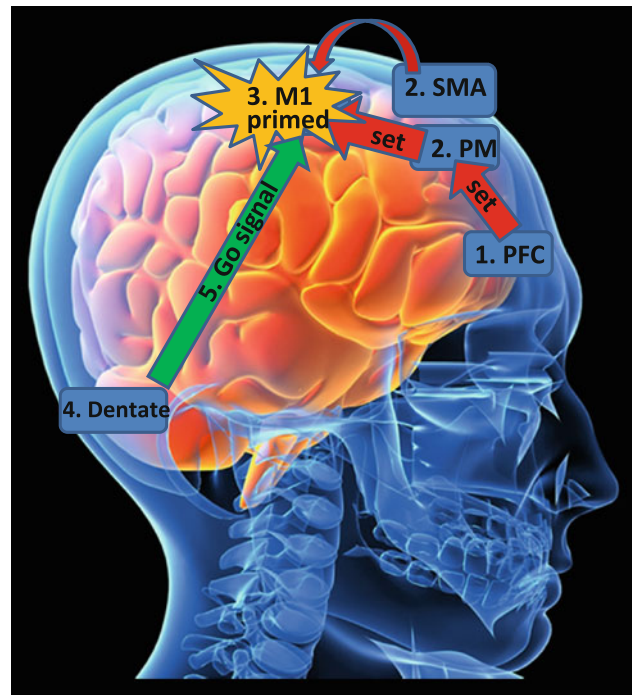


Fig. 3 Motor urgency is also dependent on the PFC such that (1) the PFC and (2) the PM and SMA upregulate and prime (3) the M1 via teleoanticipatory set signals. When the finish line or an anticipated competitor comes into sight (4) the dentate nucleus in the cerebellum gives the (5) ‘go’ signal to activate the primed M1. *PFC* pre-frontal cortex, *PM* pre-motor, *M1* primary motor area, *SMA* supplementary motor areas

at 60 min [152]. The increase in both power output and integrated electromyography during the final sprint relative to the 50th minute sprint, despite the presumably greater levels of peripheral fatigue, suggest a significant role for M1 teleoanticipatory priming and the postulated end-of-trial go signal from the cerebellum in determining this end spurt. Note that the putative role for motor urgency during exercise—based on animal and human reaction time studies and on the extensive neural input from the cerebellum into the motor cortex—is more speculative than the role of motor vigour.

13 Cortical Override of the Basal Ganglia

Clearly the BG and, particularly, the tonic dopamine levels in the striatum play a major role in continually driving the motor system during exercise, whereas the cerebellum may provide additional M1 drive during the end spurt. However, the highest controller of the motor system is situated in the PFC and, as such, it can bypass/override the influence of the BG and cerebellum on movement via direct corticomotoneuronal drive [46]. This override ability increases the cognitive flexibility at the expense of primitive motor

behaviour and motivated exercise. Dampening of primitive motor behaviour seems to be facilitated by increased norepinephrine concentrations that are usually observed with increased exercise duration [153] and intensity. Working with mice, Dziraza and co-workers [166] found that norepinephrine decreased the coherence between the reward expectancy circuitry and the striatum and increased the coherence between the cortex and the striatum. This is consistent with top-down attentional control over subcortical incentive driven structures [131, 133] that would serve to increase cognitive flexibility at the expense of exercise drive [154].

Apart from the modulating effects of acetylcholine and norepinephrine over dopamine modulated primitive motor behaviour, serotonin (5HT) substantially reduced the hyperactivity in mice with artificially elevated striatal dopamine levels [155], showing that 5HT has a calming effect related to feeling secure during times of heightened arousal [156]. Further, midbrain serotonergic pathways upregulates the excitability of alpha motoneurons [157, 158], a critical component of rhythmic motor behaviour in the mammalian spine [159]. 5HT has also been linked to facilitation of excitability in the human neuromuscular system [160].

Presumably the correct CNS balance of predominantly dopamine, serotonin, norepinephrine [161] and acetylcholine [117] as well as of endorphins [141, 142] and cannabinoids [144, 145], during an important sporting event will enable an athlete to remain calm and focussed under pressure, to increase motor vigour during, and to add motor urgency at the end of a race. Over-arousal at the start of a race may lead to premature motor urgency at the expense of motor vigour.

14 Location of the Central Governor

From the above discussion in Sect. 13 we thus see that the net reward rate (and therefore exercise performance) as set by the tonic striatal dopamine levels runs deeper than simple substrate depletion or accumulation [154, 161] but is rather a tightly regulated process (Fig. 1). That is to say, the greater the training adaptations and the previous experience, the more novelty seeking the individual, the greater the reward and the more crucial the goal itself, the greater the tonic dopamine levels in the striatum would be. Since motor vigour is set by the tonic striatal dopamine levels, it must thus be of overriding importance in setting the pace during a sporting event via striatal disinhibition of the motor cortex. The corollary of this argument would be that if there is no upregulation of tonic dopamine levels in the striatum, then exercise would be more voluntary than motivated. In a laboratory setting exercise might have a

greater voluntary than motivational component, while the preparation, peaking and pressure of a major sporting event would help to swing the ratio the other way, and approach 100 % motivational behaviour during a life or death situation. Crucially, the prevailing brain chemistry is dependent on the goal subsumed in the PFC and reformulation of the goal (e.g. if the probability of goal achievement becomes more or less achievable during the race) would lead to an altered brain chemistry that would impact performance. For example, if the probability of winning is increased, the brainstem would release more dopamine and ‘fatigued’ muscles would be further upregulated and RPE downregulated (Fig. 2) [162]. According to this interpretation, the Central Governor that is postulated to regulate exercise performance [9, 163] will not be found in a specific location. Rather, it is the prevailing neurochemical balance in the centrally located motor nuclei of the three motor systems that drives the body onwards to goal fulfilment, slows it down or stops it in its tracks if goal achievement becomes less realistic or important during the course of a race.

15 Conclusion

The premise of this review is that voluntary muscle recruitment by M1 during exercise can be upregulated in one of two ways. Firstly by increasing motor vigour, mediated by the basic seeking/wanting brain circuitry that enhances autonomic arousal over and above what is required for homeostatic purposes. In this situation M1 drive is proposed to be enhanced by dopaminergic modulation of the five cortico-basoganglionic-thalamo-cortical circuits. Greater tonic striatal dopamine concentrations would facilitate primitive motor behaviour [92] until the afferent sensory signals impinging on the PFC-BG-thalamo-cortical neuronal circuit becomes excessive relative to the goal [87, 164], with the resultant decrease in striatal dopamine modulation leading to decreased motor vigour and slowing or stopping of exercise [120]. An essential component of motor vigour is that it is driven by the ongoing net reward rate [92, 100, 103–107], rather than by the delayed reward of finishing within a set time [99]. We further propose that the increased norepinephrine concentrations with prolonged exercise would strengthen the coherence between the cortex and the striatum and weaken subcortical primitive motor behaviour, thereby increasing voluntary behaviour and decreasing motivated behaviour.

Secondly, M1 drive can also be enhanced by motor urgency suggested to result from teleo-anticipatory priming of M1 in combination with the ‘go’ signal from the cerebellum. This effect would be limited to those times when urgent anticipatory sensory stimuli are provided such as at

the finish line, when overtaking/being overtaken by a fierce competitor or when following directly in the footsteps of a close competitor. Crucially, given that optimization of goal achievement is set by the BG; excessive motor urgency drive from the cerebellum, especially immediately prior to a race when performance anxiety is heightened, may derail a carefully planned teleoanticipatory strategy and lead to premature fatigue.

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Conflict of interest To the knowledge of the authors, there are no conflicts of interest that are directly or indirectly related to the contents of this article.

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