Perception of effort during exercise is independent of afferent feedback from skeletal muscles, heart, and lungs

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Perception of effort, also known as perceived exertion or sense of effort, is a major feature of fatigue (7), and it is widely used to monitor and prescribe exercise intensity (18). However, despite its importance, the neurophysiological bases of this atypical sensation are poorly understood. A model popular among physiologists investigating central regulation of exercise performance is that perceived exertion results from the complex integration of different inputs to the central nervous system (CNS). These inputs include afferent feedback from the peripheral organs most active during aerobic exercise (i.e., skeletal muscles, heart, and lungs) and other interoceptors, with or without additional inputs from the CNS itself, such as knowledge of the exercise task endpoint (3, 6, 28).

The purpose of this article is to challenge this view by reviewing evidence that perception of effort during dynamic whole body exercise is independent of afferent feedback from small-diameter muscle afferents, heart, and lungs. For isolated and/or isometric exercise, the reader is referred to other reviews demonstrating that the sense of effort is independent of sensory inputs from muscle spindles and Golgi tendon organs (15, 24).

Definitions. The common assumption that afferent feedback contributes significantly to perception of effort may derive from a too broad operational definition. Indeed, if subjects are instructed that rating of perceived exertion (RPE) refers to the discomfort experienced during exercise, then muscle pain, thermal discomfort, and thirst can be appropriately included in the perceptual rating because hedonicity is one of the four dimensions of any sensation (quality, intensity, duration, and hedonicity) (3). However, these and other unpleasant sensations experienced during exercise have their own specific neurophysiological mechanisms and verbal descriptors (i.e., quality) and can be differentiated from perception of effort (e.g., Ref. 19). Therefore, we believe that a narrower definition of exertion as “the effort expended in performing a physical activity” (Oxford Dictionary of Sports Science and Medicine) is physiologically more appropriate. Furthermore, this definition is in line with the verbal descriptors chosen by Borg for his RPE scale (“heavy/hard” and “light/easy”) (18) rather than ratings of hedonicity (“pleasant/unpleasant,” “feeding good/feeling bad,” “comfortable/uncomfortable”) (3).

Perceived exertion and afferent feedback from skeletal muscles. Skeletal muscles are richly innervated by group III and IV fibers that provide afferent feedback related to metabolic, ionic, thermal, and mechanical stress (12). These small-diameter afferent fibers project, through lamina I neurons, to various spinal and supraspinal sites including the sensory cortex (5). Given the existence of such sensory system and the well-known increases in muscle metabolic by-products, potassium, temperature, and mechanical stress during intense exercise (12), it seems logical to assume that perception of effort is based on these afferent stimuli (Fig. 1A). Furthermore, there are high correlations between markers of metabolic stress (e.g., blood lactate concentration and %VO2max) with RPE during incremental exercise (18). However, the presence of the sensory machinery and a high correlation between the proposed metabolic stimuli and perceived exertion does not necessarily mean that the two are causally related. Indeed, several experimental studies in which perception of effort and metabolic stress have been dissociated argue against such common assumption.

For example, when skeletal muscle weakness is induced by a small dose of curare (a drug that blocks transmission at the neuromuscular junction without affecting afferent pathways) there is a significant increase in RPE during cycling exercise (9). In theory, recruitment of glycolytic fibers due to the selective effect of curare on oxidative fibers may increase metabolic stimuli and perception of effort. However, the small increase in blood lactate concentration (0.36 mM) compared with the disproportionate increase in perceived exertion (4.8 points on Borg’s 15-point RPE scale) argue against this hypothesis. Similarly, changes in afferent feedback from prefatigued locomotor muscles cannot explain the significant increase in perceived exertion we measured during a high-intensity cycling test (14). Indeed, we induced a significant reduction in locomotor muscle force using an eccentric exercise protocol known to affect primarily type II fibers without any metabolic stress or changes in muscle receptors sensitivity. Increased afferent feedback related to inflammation (which manifests itself at perceptual level as delayed-onset muscle soreness) was also controlled by performing the high-intensity cycling test 30 min after the intermittent 100 drop jumps.

So what is the neural signal underlying perception of effort in these experimental conditions? The findings reviewed above support the hypothesis suggested more than 150 years ago that perception of effort is a “sensation of innervation” (24). Basically, it is proposed that the sense of effort is centrally generated by forwarding neural signals, termed corollary discharges or efference copies, from motor to sensory areas of the cerebral cortex (Fig. 1B). Therefore, it is not surprising that the increase in central motor command necessary to exercise at the same workload with muscles weakened by partial neuromuscular blockade or locomotor muscle fatigue is perceived as increased...
A  **Afferent feedback model of perceived exertion**

![Diagram of afferent feedback model]

- Central command (pre-motor and/or motor areas)
- Sense of effort (somatosensory areas)
- Skeletal muscles

B  **Corollary discharge model of perceived exertion**

![Diagram of corollary discharge model]

- Central command (pre-motor and/or motor areas)
- Sense of effort (somatosensory areas)
- Skeletal muscles

Fig. 1. Simplified afferent feedback (A) and corollary discharge (B) models of perceived exertion.

**Effort.** Despite this kind of experimental evidence in favor of the corollary discharge model of perceived exertion (15), lack of a neurobiological mechanism and the discovery of muscle receptor function by Sherrington made most people believe that these receptors were responsible for all sensations related to muscle contraction (24). However, in recent years, corollary discharge pathways have been described in animals, including a single interneuron forwarding inhibitory signals from motor to sensory areas of the male singing cricket nervous system (23). This important discovery provides a "proof-of-principle" for the existence of neurons and pathways serving other corollary discharge functions including perception of effort.

Lastly, convincing evidence against the widespread assumption that perceived exertion arises from afferent muscle feedback is provided by experiments using epidural anesthesia. Indeed, despite a marked reduction in sensory inputs from the lower limbs, RPE during cycling exercise is either unchanged or increased by spinal blockade (13, 27). The latter effect is due to the muscle weakness induced by the epidural anesthesia used in these studies and the compensatory increase in central neural drive to the motoneurons innervating the locomotor muscles. Even when this reduction in muscle strength is taken into account by exercising at the same relative intensity, RPE is not reduced by blockade of sensory inputs from the lower limbs (16). This is strong experimental evidence against the notion that somatosensory feedback from active muscles is important for perception of effort during exercise (6).

**Perceived exertion and afferent feedback from the heart.** The correlation between perception of effort and heart rate (HR) during incremental exercise is so strong that the 6–20 RPE scale was originally developed by Borg to reflect the range of HR found in young and fit subjects (60 beats/min at rest, 200 beats/min during maximal exercise) (18). There is also evidence consistent with an afferent cortical representation of cardiac action (10), which is not surprising given the existence of both sympathetic and vagal afferent pathways from the heart (8), and chest wall mechanoreceptors (4). Indeed, some subjects can accurately report cardiac activity (4), and the crushing, burning, and squeezing sensations of angina pectoris are far too familiar to patients with ischemic heart disease (8). However, various studies in which cardiac function has been manipulated experimentally do not support a causal relationship between HR and perception of effort (18). For example, RPE during cycling exercise is unchanged or even increased when HR is reduced by calcium channel and β-adrenergic blockade (17). Therefore, it seems that normal subjects do not attend to afferent cardiac signals when rating perceived exertion. Furthermore, cardiac transplant patients (who have a denervated heart) report normal perception of effort during an incremental exercise test (1). Indeed, RPE is recommended by the American Heart Association for prescription of exercise intensity after transplantation (22).

**Perceived exertion and afferent feedback from the lungs.** Dyspnea, from the Greek "difficult breathing," is a major component of overall perceived exertion (18) and it has been extensively investigated because of its clinical relevance (20). As for the discomfort associated with intense exercise, the word "dyspnea" can refer to several sensations with different verbal descriptors and neurophysiological mechanisms. Inhibitory and excitatory afferent inputs from central and peripheral chemoreceptors and mechanoreceptors in the chest wall, lungs, and airways are thought to play an important role in generating air hunger, chest tightness, and unsatisfied inspiration (20). However, the dyspnea experienced during aerobic exercise by healthy individuals without restricted breathing and normal blood gases largely reflects the sense of respiratory effort (11, 20, 21). This respiratory sensation is generated by corollary discharges of the central motor commands to the respiratory muscles (11, 20), and it is clearly independent of feedback from pulmonary vagal afferents. Indeed, patients with double lung transplantation can estimate the magnitude of inspiratory resistive loads based on their normal sense of effort (31). Similar results have been obtained in healthy subjects with anesthetized airways (2). Importantly, the common neurophysiological mechanism shared by perceptions of effort referred to breathing and locomotor muscle contraction justifies their integration in the overall rating of perceived exertion.

**Conclusions.** Although essential for a variety of other physiological and perceptual processes such as cardiorespiratory regulation (12) and sense of position and movement (24), afferent feedback from skeletal muscles, heart, and lungs does not contribute significantly to perception of effort during exercise, which is the conscious awareness of the central motor commands to the locomotor and respiratory muscles. Preliminary imaging studies in humans (30), lesion studies in animals (29), and psychopharmacological studies in both humans (26) and animals (25) suggest that the anterior cingulate cortex, insular cortex, thalamus, dopamine, and endogenous opioids are important for perception of effort and related decision making, e.g., disengaging from a time to exhaustion test (14). Additional studies on the origin [primary motor cortex or upstream? (24)], neural pathways, and sensory processing of corollary discharges related to perceived exertion are warranted. Similarly, the role played by other neurocognitive processes (e.g., response inhibition and conflict monitoring)
and activation-dependent plasticity in setting perception of effort during prolonged exercise needs to be established.

REFERENCES


