Roles for perceived voluntary motor commands in motor control

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Voluntary contractions of skeletal muscles are accompanied by centrally-generated motor 'commands' that ultimately result in descending inputs to motoneurone pools. Perceived motor commands influence the sensations of muscle force and timing, and can be accurately directed to individual distal muscles in the absence of feedback. In addition, they may also influence respiratory sensations and cardiorespiratory responses to muscular contraction. Data on the neural mechanisms underlying these motor commands suggest that they may be generated differently according to the particular function subserved within the motor system.

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All voluntary contractions are preceded by notional motor commands generated within the CNS. These commands ultimately cause recruitment of motoneurones and production of muscle force. For over a century, physiologists (and philosophers) have wondered whether signals related to such commands directly evoke sensations. In a review of kinaesthetic mechanisms, McCloskey[1] emphasized the role of muscle, joint and skin receptors in sensation of the position and movement of the limb, and presented evidence for a kinaesthetic role for centrally-generated motor commands. The present review evaluates the roles for these commands in different aspects of motor control and discusses the ways in which such commands are generated within the CNS. A comparison is made with motor commands involved in the cardiovascular and respiratory systems.

Roles for motor perceived commands

The perception of signals related to centrally-generated motor commands is involved in the sensation of muscle force and in the timing of muscle contraction. The term 'sensation of muscle force' is used to encompass the sensation of isometric force and the sensation of heaviness associated with shortening and lengthening contractions. The major evidence that favours a role for perceived signals related to motor commands in the estimation of muscle force is derived from the simple observation that a weight lifted by a weakened muscle feels heavy. The generality of this clinical dictum, first stated explicitly by Sir Gordon Holmes in 1922[2], has been established by a number of investigations (see, for example, Refs 3–8). This increase is easily quantified by asking the subject to match the forces generated on both sides when the muscles on one side have been 'weakened'. This has been documented for 'weakness' produced by muscle fatigue[3,6,7,9], local infusion of paralysant drugs[4,5,10], inhibition of the agonist motoneurones by the excitation of muscle spindles in antagonists (Ref. 2 and cf. Ref. 11) or by painful cutaneous stimuli (Gandevia, S. C. and Milne, R. J., unpublished observations). An increase in perceived force has also been documented following removal of the long-latency stretch reflex[12], a reduction in muscle length[13] and in cases of unilateral cerebellar 'hypotonia'[2,14] or 'stroke' without sensory loss[4]. If peripheral signals related to absolute intramuscular tension were involved directly in these judgements then no overestimation should occur during weakness (see below). Because fusimotor neurones are usually activated along with skeleto-motoneurones in voluntary contractions, the discharge of muscle spindle afferents may increase when the drive to the motoneurone pool increases during weakness. While central motor commands are thus translated into peripheral kinaesthetic signals via muscle spindles, their discharge does not directly evoke the sensation of muscle force. Excitation of muscle spindle afferents with vibration is associated with a lessening of perceived force (due to facilitation of the agonist motoneurone pool) rather than an increase which would be required if their discharge were to signal force[3,15]. Muscle spindle afferents already have an established kinaesthetic role in sensing limb position and movement. This role has been confirmed recently in studies using joint anaesthesia[16], longitudinal vibration of muscle tendons to excite spindle endings[17],

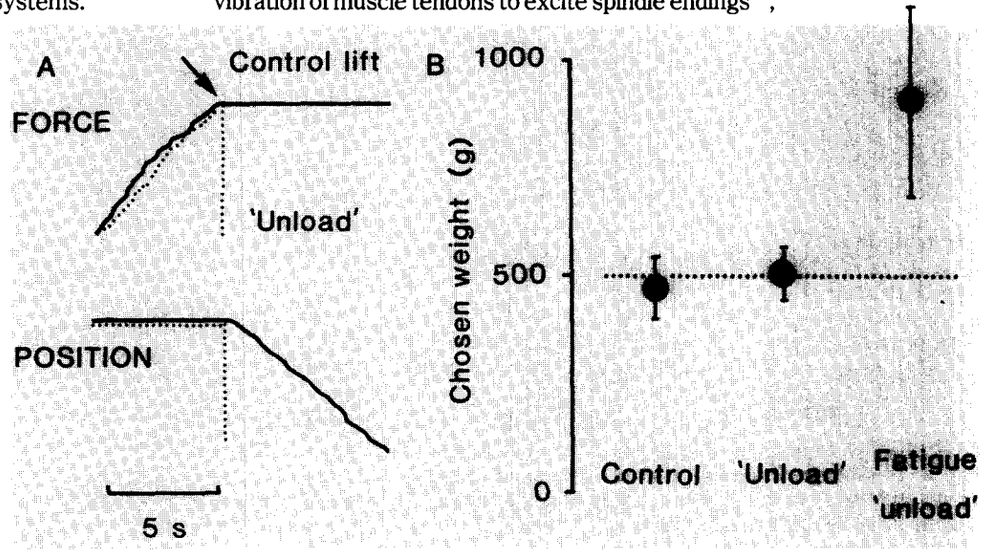

Fig. 1. (A) *Diagrammatic representation of a study in which the ability to match a force generated isometrically by the long flexor of the thumb was compared with the ability to match a weight lifted isotonically by the same muscle[6]. During a 'control lift' (solid lines) the reference force (500 g) was lifted on the left and matched to a variable weight lifted on the right. During the 'unload' condition (dotted lines) the contraction was unloaded by a motor when the reference force was reached so that the muscle did not shorten while carrying the load. The digital nerves of the thumb were anaesthetized on both sides.* **(B)** *Data for a group of subjects (mean ± SEM). When the reference force was unloaded by the motor, the perceived force was the same as that during the control lift. This suggests that afferent inputs generated after the critical force was achieved are not required for accurate judgement. Perceived force was significantly overestimated when the reference 'unloaded' force was generated by a fatigued muscle. This suggests that subjects were biased in their force judgements by a signal of the centrally-generated motor command.*

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and electrical stimulation of their afferent fibres[18].

Four problems with the postulated role for motor commands in the sensation of muscle force should be mentioned.

(1) The relationship between the size of the motor-command signal used in force sensation and the degree of weakness is complex. A common finding during weakness is that the overestimation of force does not increase in proportion to the degree of weakness. When muscle strength is halved, perceived force does not usually double; it may increase by only 30–50% (Refs 4, 5). While this suggests a non-linear relationship between the perceived signal of motor command and the force output from a muscle (a finding supported from classical psychophysics), a study by Roland and Ladegaard-Pedersen[10] implies that there may also be a signal generated within the CNS that does increase in proportion to the degree of experimental weakness. Although this question requires further investigation, there is a technical problem with these studies, particularly those in which neuromuscular blockers are used. Ideally a constant degree of weakness of the same muscle fibres should be maintained while perceived forces are measured. If the fibres of some motor units recruited in the contractions are unaffected by the experimentally induced weakness, then the small increase in perceived force with a halving of muscle strength could be explained. Furthermore, the increased peripheral drive from muscle spindle afferents during weakness and other changes in drive to the motoneurone pool may also explain the implied non-linearity.

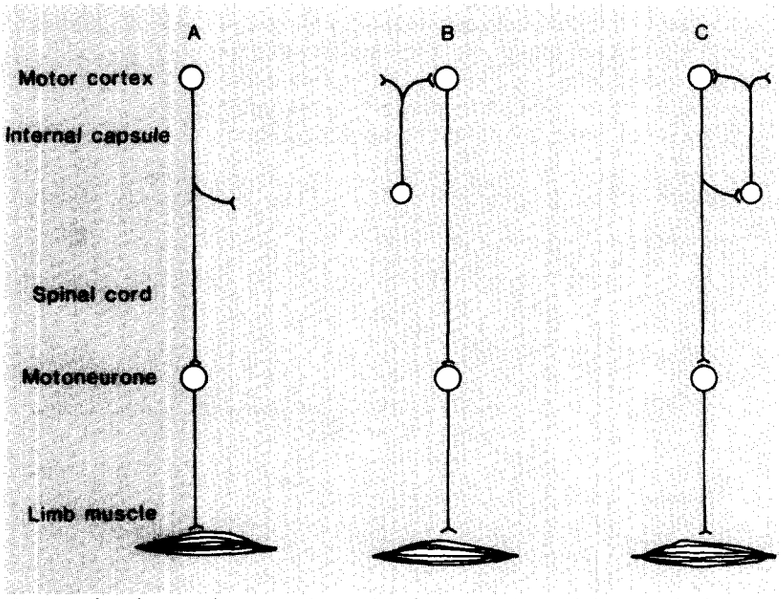

Fig. 2. *This depicts three simple models for generation of the signal of motor command that is used in the estimation of force. A pathway descending from the motor cortex via the internal capsule, motoneurone and muscle are shown diagrammatically. In* **(A)** *the relevant signal of command is generated from a collateral of the descending pathway. Although arguably the most economical model, the concept that the activity in corticofugal pathways generates a sense of force is not supported by observations during stimulation of the motor cortex or internal capsule. In* **(B)** *the relevant signal ascends to the motor cortex from a subcortical site via the internal capsule. Observations on patients with pure motor hemiplegia due to cortical or capsular lesions have led to the idea that a projection to, or from, the motor cortex is essential for generation of the signal of motor command used in force estimation. Note that the model in (B) requires an ascending signal. Further discussion of relevant mechanisms is contained in the text. In* **(C)** *a combination of the circuitry depicted in (A) and (B) is shown.*

(2) The relationship between the actual motor signal that impinges on the motoneurones (and interposed interneurones) and the perceived signal of motor commands used in kinaesthetic judgements is not clear. The most economical explanation is that the actual motor command and the signal that evokes sensation are closely related. Most simply the latter would arise merely as a collateral from the former. The isolated observations on patients with discrete neurological lesions are consistent with such a close relationship, or at least do not provide evidence against it. Different models for generation of the relevant command signals are considered below (see Fig. 2).

(3) A signal of motor command cannot be interpreted as a quantitative signal of external events, such as the force needed to move a particular object, unless a peripheral input signals the success or failure of the contraction in moving it. However, the role of afferent inputs in calibration of this signal of perceived motor command has received little attention. In a study designed to investigate this[6], subjects judged the magnitude of an isometric force produced by flexion of the distal joint of the thumb when the force was immediately 'unloaded' by a motor once it had been achieved (Fig. 1). Under these constrained conditions, subjects were as accurate in their judgements as when an equivalent weight was moved in an isotonic contraction. As perceived force increased during unloaded contractions when the muscle was fatigued (Fig. 1B), subjects were presumably relying on centrally-generated commands. A signal generated in intramuscular receptors by the unloading must have been used to calibrate the perceived signal of motor command since the joint and cutaneous afferents from the thumb were anaesthetized. The relative unsophistication of this afferent signal can also be inferred from the residual ability to control movements after deafferentation[19]. It is as if the crude afferent input signals to the CNS the critical point in a ramp of increasing motor command at which the command succeeds in supporting or lifting the weight. This critical level of command may then provide the estimate of force. Further central processing is required to 'translate' this estimate so that it specifies uniquely the size of any external load on the limb. It is necessary to know the orientation both of the limb and of the applied load with respect to gravity[20].

(4) In addition to signals of motor command, peripheral signals of intramuscular tension reach consciousness[3,10]. On the available evidence from psychophysical studies, peripheral inputs related to absolute force do not provide a dominant perceived signal used to control voluntary movement, although recent evidence shows that Golgi tendon organs (which respond to active muscle force) do project to the sensorimotor cortex[21]. Signals of absolute force can be perceived independently of changes in perceived motor commands when the excitability of the motoneurone pool is altered artificially by muscle vibration[3]. However, there are conflicting reports as to whether subjects 'extract' this signal of force when the muscles are fatigued by sustained contractions. Signals of command and absolute force were distinguished during fatigue of the inspiratory muscles[22] but during fatigue of the elbow flexors subjects relied on the signals of motor command and could not distinguish a peripheral signal of force[23]. It is relevant that patients

with reduced muscle power usually complain not that their muscles are weak but that a greater motor command or effort is required to use them.

Signals related to motor commands also influence the perceived timing of muscular contractions[1,24]. By varying the timing between a cutaneous stimulus and a voluntary movement it was shown that subjects distinguish between the time at which the muscle contraction was commanded and the time at which the resulting contraction generated kinaesthetic information. The perceived signal for timing motor commands to the arm muscles was perceived to arise about 100 ms before electromyographic (EMG) activity, even when the limb was anaesthetized. When instructed to make simultaneous contractions with muscles of the jaw and of the foot, some subjects aligned the motor commands for the two muscle groups, and some aligned sensory information set up by the resulting contractions. Thus some subjects preferred a central rather than a peripheral signal as their 'timing marker'. Presumably the perceived signals of timing of motor commands can be used both to sequence rapid voluntary movements that are too fast to be adjusted by afferent feedback and to time the adjustments to ongoing movements.

A recent study has emphasized the precision with which centrally generated commands can be controlled[25]. Normal subjects learned to direct motor commands to particular sets of neurones in the CNS without afferent feedback. Needle electrodes were positioned to record from the first-recruited motor unit in each of two intrinsic muscles of the hand. When requested by the experimenter, the subject was required to 'focus' a motor command upon one or other of the pair of muscles *without* recruitment of motoneurones in either muscle or movement of the hand. The ability to produce 'subthreshold' motor commands directed to one of the motoneurone pools was determined by delivery, at random times, of liminal stimuli to the contralateral motor cortex using percutaneous stimulation[26]. In less than an hour, subjects learned to focus subthreshold commands such that only motor units in the requested muscle were activated by the ('test') cortical stimuli. This ability can be expressed without recourse to afferent feedback because there was no voluntary movement of the hand. It cannot be explained by selective activation of muscle spindle afferents via the fusimotor system because subjects do not learn to activate this system without also activating motor units[27]. Presumably this ability indicates that commands can be precisely monitored and fractionated for the individual intrinsic muscles of the hand. It is likely that this ability is useful for organizing fine manipulative behaviour and learning complex movements.

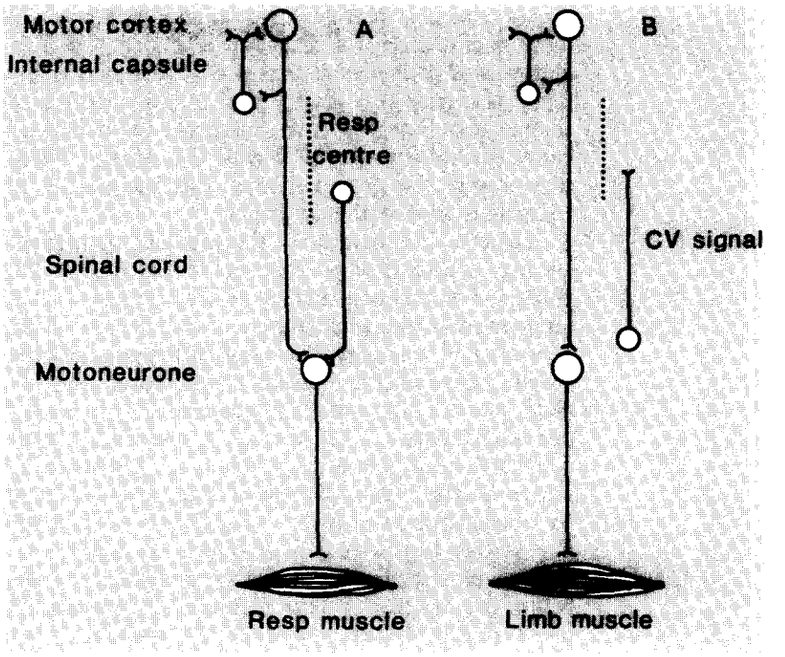

Fig. 3. (A) *One mechanism by which signals of voluntary motor command can influence the sensation of inspiratory muscle force. Respiratory motoneurones receive descending commands from several sources: some commands for voluntary action arise in the motor cortex and other non-voluntary commands from hindbrain respiratory centres. There is evidence that sensations of inspiratory muscle force are influenced by signals of motor command generated voluntarily (the descending input shown at the left of the respiratory motoneurone) in a similar way to sensations of force generated by limb muscle. Motor commands from respiratory centres in isolation may not give rise to the sensation of inspiratory muscle force or the related sensation of breathlessness.* **(B)** *Cardiovascular signals that elevate blood pressure and heart rate during muscle contraction can arise from centrally-generated signals of motor command and from peripheral inputs. When the spinal cord is transected at low thoracic levels, attempted contractions of the lower limbs produce no increase in cardiovascular variables, but they do if the subject contracts muscles in the upper limb or if normal subjects attempt to contract muscles paralysed by ischemia[32]. After spinal transection, motor commands can still give a perceived signal of force but these commands cannot generate the usual cardiovascular changes to attempted contractions.*

Centrifugal motor commands and neural mechanisms

The evidence above suggests several roles for centrally-generated signals of motor command but unfortunately there are few data on the specific neural mechanisms involved. Before specific mechanisms are proposed, it should be noted that the frequently cited 'outflow' hypotheses of corollary discharge and efference copy describe neural circuits for a stereotyped motor programme; neither hypothesis implicity requires direct perception of the motor command (for further discussion see Ref. 8). Nonetheless, an efferent signal of motor command (or corollary discharge/efference copy) may influence perception of afferent signals indirectly. For example, during voluntary contractions only the muscle spindle discharges that are greater than 'commanded' or 'expected' are perceived as an imposed movement[28]. Signals of motor command can calibrate but not generate sensations of limb movement[29].

There is a theoretical difficulty in designating specific centres as involved in the generation of the perceived signals related to motor commands. If a particular unilateral lesion consistently produces an over- or under-estimation of forces exerted on one side, then it cannot necessarily be concluded that the ablated centre is involved in generation of the relevant central signal. The centre may simply have provided a background level of excitation or inhibition (respectively) at some level in the pathway, even at a spinal motoneuronal level, rather than have generated the perceived signal of motor command. The temptation to ascribe the generation of these signals to a particular CNS centre has proved difficult to resist[14,30]. Thus, the increased effort required to initiate movement in Parkinson's

disease implies merely that some 'assistance to movement' which is usually dependent on the nigrostriatal pathway has been lost. It does not imply that this pathway gives rise to the perceived commands. Similarly, the increase in perceived heaviness associated with unilateral cerebellar hypotonia cannot be used to show that the perceived commands arise in the cerebellum[14].

Subject to the caveats of the previous paragraph, specific central lesions that spare or abolish the perceived motor command involved in the sensation of force may provide insight into which structures directly contribute to the sensation. Surgical transection of the corpus callosum and anterior commissure does not affect the accuracy of force matching, nor does it prevent transfer of the increased perceived motor command from one hemisphere to the other during muscle fatigue[9]. Also, clinically complete transection of the spinal cord (at or below mid-cervical levels) does not abolish the sensation of effort (or force) that accompanies attempts to contract paralysed muscles below the lesion[31,32]. The simplest explanation of these results is that a perceived signal of motor command can arise without input from either the neocortical commissures or the spinal cord.

Complete hemiplegia without conventional sensory disturbance (clinically referred to as pure motor hemiplegia[33]) is the one disturbance reported to abolish specifically the sensation of force that usually accompanies attempts to contract paralysed muscles[31]. This observation was first noted by Ernst Mach in 1898[34] when he suffered a transient hemiplegia, and has subsequently been confirmed by additional subjects experiencing damage to the motor cortex and the posterior limb of the internal capsule[31]. These subjects have no deficit of cutaneous sensibility or sensation of limb movement but, as long as a muscle remains completely paralysed, the sensation of force (or effort) that usually accompanies attempts to contract it is lost. As voluntary strength returns, the sensation of force reappears and it declines in intensity as muscle strength recovers. These observations suggest that neural traffic reaching or leaving the motor cortex via the internal capsule provides a critical component of the signal required for the perceived motor command involved in force sensation. By contrast, following lesions below the internal capsule that produce pure paralysis, the sense of motor command on attempted movement is not lost. Subjects with complete hemiplegia due to midbrain/pontine infarction (with little or no sensory disturbance) note an intense sensation of force on first attempts to move the paralysed limbs (Gandevia, S. C., unpublished observations).

Possible neural circuits

Three possible schemes by which the relevant signal could be generated are shown in Fig. 2. In simplistic terms, it may (1) arise directly from corticofugal paths via a collateral distal to the internal capsule, (2) involve a subcortical structure that projects to the motor cortex via the capsule, or (3) arise from a loop with one or both limbs traversing the internal capsule. Corticofugal activity alone (presumed to include corticospinal activity) which is 'read off' via a collateral below the internal capsule may not be sufficient to provide the required signal (Fig. 2A). Subjects fail to report more than one sensation of force during a rapid movement when there is sequential activation of agonist, antagonist and then agonist muscle. Stimulation of the motor cortex at sufficient levels to activate powerfully an intrinsic hand muscle in normal subjects does not produce a sensation of force (Rothwell, J. C. and Gandevia, S. C., unpublished observations and Refs 26, 35, 36), nor does stimulation within the internal capsule (Tasker, R., pers. commun.). Subjects note merely that the muscle has twitched. Further studies are required to evaluate the hypothetical circuits described above. It would be particularly helpful to find a subcortical lesion that abolished the perceived signals of motor command. While the available clues do not yet allow crucial distinction between the possibilities in Fig. 2, they tentatively implicate both the motor cortex and a subcortical input. They further suggest that the latter does not arise in the pons, midbrain or cerebellum.

If subjects with pure motor hemiplegia retain the ability to time accurately their attempts or commands to move but lose the usual sensation of muscle force, then the neural mechanisms underlying the two signals of motor command (one for timing, one for force sensation) may be generated independently within the CNS. Available data indicate only that such subjects know that an attempt to move has been made[31,34]. Further support for the suggestion that the two neural mechanisms are distinct comes from the observation that virtually all subjects are influenced by signals related to central motor commands when judging force[3,6,23] but some use peripheral signals to estimate the timing of muscle contraction[24]. In addition, signals related to perceived timing arise before muscular contraction, whereas those of muscle force arise as force is generated.

Other signals of motor commands

This review concludes with a brief description of the operation of some other putative signals of perceived motor commands that involve the respiratory and cardiovascular systems. Other examples of motor commands could equally well be given. The neural mechanisms underlying the role of motor commands in the genesis of sensations of respiratory muscle force and in cardiorespiratory control during exercise are not the same, nor are they identical to those described for control of limb muscles (Fig. 3).

Voluntary motor commands to inspiratory muscles, but not those from autonomic pontomedullary centres for respiration, strongly bias the sense of inspiratory muscle force (i.e. inspiratory pressure). It has been argued that the sensation of breathlessness may simply reflect the increased voluntary command to achieve a certain pressure or airflow[22,37,38]. The interaction of the voluntary and non-voluntary command signals and the many afferent inputs in the sensation of breathlessness is complex. However, it seems that signals related to the levels of arterial blood gases (and/or respiratory centre activity) cannot generate by themselves the sensation of breathlessness.

The idea that there is 'cortical irradiation' of cardiovascular centres by motor commands to produce the appropriate elevations in blood pressure and heart rate during muscular contraction was introduced by Krogh and Lindhardt in 1913 and has received experimental support[39]. A common tacit assumption is

that the perceived motor command signals used in force sensation are the same as the central signals used in cardiovascular control. However, there is recent evidence that the two signals can be dissociated during attempted contraction of paralysed leg muscles in subjects with complete transection of the thoracic spinal cord. The ability to perceive motor commands used in force estimation remains, but the ability to elevate cardiovascular variables in the usual way is lost[32]. This loss is unlikely to be due to failure of peripheral inputs to reach the cerebrum because attempts to contract muscles paralysed by anoxia in normal subjects produce increases in heart rate and blood pressure. This suggests that whatever signals of motor command generate the sensation of force during the attempted contractions cannot produce the usual cardiovascular changes in the absence of an ascending signal in the spinal cord. Such a signal may arise through spinal circuits that receive a descending motor input and subsequently project it rostrally (Fig. 3B), possibly via the ventral spinocerebellar tract[40]. This mechanism would offer yet another way by which central motor commands can exert their effects within the CNS.

Concluding remarks

Although this review has largely focused on perceived motor commands associated with limb muscles, there are other neural systems involving sensorimotor and integrative control that are influenced by motor commands. Such systems broaden the concept developed here that motor commands subserve many different functions within the CNS and are generated via different neural mechanisms. Furthermore, while it is usual to emphasize tangible feedback signals in motor control, the perceived signals of centrally-generated motor commands discussed here have important roles in directing, quantifying and timing the outputs to muscles.

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