# Motor Control Programs and Walking

YURI P. IVANENKO, RICHARD E. POPPELE, and FRANCESCO LACQUANITI

The question of how the central nervous system coordinates muscle activity is central to an understanding of motor control. The authors argue that motor programs may be considered as a characteristic timing of muscle activations linked to specific kinematic events. In particular, muscle activity occurring during human locomotion can be accounted for by five basic temporal components in a variety of locomotion conditions. Spatiotemporal maps of spinal cord motoneuron activation also show discrete periods of activity. Furthermore, the coordination of locomotion with voluntary tasks is accomplished through a superposition of motor programs or activation timings that are separately associated with each task. As a consequence, the selection of muscle synergies appears to be downstream from the processes that generate activation timings. NEUROSCIENTIST 12(4):339–348, 2006. DOI: 10.1177/1073858406287987

Although the origin of the motor programs or formulas of movement (Bernstein 1967) embraces numerous brain structures and probably cannot be separated from sensory feedback, the final common pathway (Sherrington 1906) is considered the activation of the α-motoneurons (MNs). Thus, researchers have often relied on electromyographic (EMG) signals to conjecture what is being programmed in the central nervous system (CNS). A number of recent studies have used this approach to explore the bases of central motor programming by decomposing muscle activation patterns as a means to look backward from the periphery to the CNS (Davis and Vaughan 1993; d'Avella and others 2003; Hart and Giszter 2004; Ivanenko and others 2004).

In this review, we will focus on the general design of the motor output during human locomotion and its interaction with voluntary movements. The key issue is the multidimensional (multimuscle) analysis of motor patterns. Although the review is largely centered on recent work in our laboratory, the purpose is to bring together a number of recent findings on the organization of the motor output rooted in the temporal structure of the phasic action of muscles. We will initially consider the spatiotemporal organization of α-MN activation in the human spinal cord and then a complementary statistical analysis. Finally, we will consider a conceptual framework for a superposition of locomotion and voluntary motor programs. We will not review any detailed kinematic control of walking (see Lacquaniti and others 1999, 2002).

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## Spatiotemporal Maps of MN Activation during Locomotion Show Discrete Periods of Activity

Spinal pattern generators for locomotion have now been studied in several mammals (Orlovsky and others 1999). However, the details of such circuitry in the human spinal cord are still largely unknown (Winter 1989; Duysens and Van de Crommert 1998; Lacquaniti and others 1999; Edgerton and others 2001; Capaday 2002; Ivanenko and others 2003; Dietz and Colombo 2004; Grasso and others 2004). The current understanding about these spinal circuits is based largely on studies in the cat. Pattern-generating oscillators are believed to be primarily localized in the cervical and lumbosacral segments of the cord where they control the locomotion movements of the corresponding limbs. Early studies by Brown (1911) suggested there are one or more half-center central pattern generators (CPGs) for each limb (see also Jankowska and others 1967; Grillner and Zangger 1979). More recent data suggest a major site for CPG activity in the upper lumbar segments may serve a pacemaker role, together with other, perhaps separate, generator sites in more caudal segments (Kremer and Lev-Tov 1997; Lev-Tov and others 2000; Cazalets and Bertrand 2000). Although the CPGs organize the sequencing of motor activity in locomotion, they are also controlled in turn by both central commands and proprioceptive feedback (Drew and others 2004; Pearson 1995). For example, electrical stimulation applied to the mesencephalic locomotor region in the cat (Shik and others 1966) can elicit locomotion at various speeds, depending on the intensity of the stimulation. Moreover, the transition period from rest to locomotion, which may be characterized by a specific tuning of the spinal cord circuitry, is also strongly influenced by descending activity (Shik 1983; Mori and others 1989). In addition to the segmental feedback from proprioceptors, it has also been suggested that MNs themselves may be integral elements of CPGs (Marder 1991; O'Donovan and others 1998).
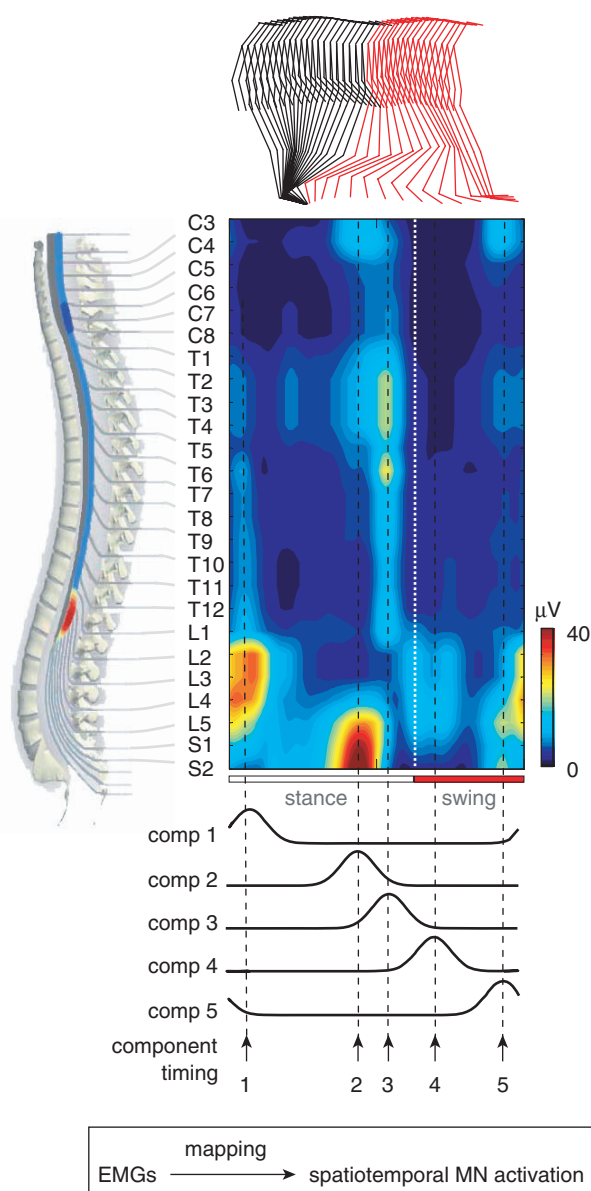
**Fig. 1.** Spatiotemporal patterns of ipsilateral α–motoneuron (MN) activity along the rostrocaudal axis of the spinal cord during walking on a treadmill at 5 km/h. Pattern is plotted versus normalized gait cycle. Output pattern was constructed by mapping the recorded electromyographic (EMG) waveform of 32 ipsilateral limb and trunk and shoulder muscles (nonnormalized method, adapted from Ivanenko, Poppele, and others [2006] with permission) onto the known charts of segmental localization. Five Gaussian activation components that correspond to five major discrete periods of activity and account for ~90% of total variance are shown on the bottom.

Currently, however, opinions diverge as to whether the mammalian CPGs are localized or distributed (Kiehn and others 1998; Orlovsky and others 1999).

There is relatively little data relevant to understanding the organization of the CPG circuitry in humans. However,

it is possible to visualize α-MN activation during locomotion by mapping the activity patterns from a large number of simultaneously recorded muscles onto the approximate rostrocaudal location of the MN pools in the human spinal cord (Fig. 1; Ivanenko, Poppele, and others 2006). This technique, similar to one recently reported for the cat (Yakovenko and others 2002), does not show the organization of the CPG directly, but it does show how the CPG output is directed to the MN pools within each spinal segment. We applied this technique by recording EMG activity from 32 ipsilateral leg and trunk muscles during locomotion and adding up the contribution of each muscle to the total activity in each spinal segment according to the published myotomal charts of segmental innervation in humans (Sharrard 1964; Kendall and others 1993). The implicit assumption is that the rectified EMG provides an indirect measure of the net firing of spinal MNs innervating that muscle.

Maps constructed from data recorded during treadmill locomotion at several different speeds show a number of common features (Ivanenko, Poppele, and others 2006). One is that MN activity tends to occur in bursts that are temporally aligned across several spinal segments. For each spinal segment, there are generally two activity bursts occurring in each locomotion cycle corresponding to one burst during the stance phase and the other during swing (Fig. 1). These maps are relatively invariant across subjects, especially at high walking speeds (Ivanenko, Poppele, and others 2006), despite the fact that the root innervation of many muscles does show interindividual variations (see Wilbourn and Aminoff 1998).

A simple inspection of the maps suggests that almost all of the EMG activity during locomotion can be represented by five separate periods of MN activation. The timing of these activity peaks tends to be associated with the major kinematic and kinetic events in the gait cycle, namely (see Fig. 1, *bottom*), weight acceptance (1), loading/propulsion (2), trunk-stabilization activity during the double support phase (3), liftoff (4), and heel strike (5). Thus, it appears that the activation patterns may represent the drive provided by spinal pattern generators and/or sensory feedback.

## Locomotion Motor Program as a Characteristic Timing of Muscle Activations

Another line of evidence relating to the nature of the locomotion motor program has come from analyses of EMG activity using factor analysis. A couple of studies in the 1980s and 1990s showed that the activity patterns of leg muscles during locomotion could be accounted for by four to five specific bursts of activation (Patla 1985; Davis and Vaughan 1993). That is, the activity patterns resulting from the modulation of leg muscle activity over a step cycle could be reconstructed for each muscle from a weighted sum of a few (5) basic temporal components (Fig. 2, see also below). Most recently, we showed that this analysis gives the same basic result when trunk and arm muscles are included in the analysis and independently of walking speed or body weight support (Ivanenko and others 2004, 2005). Furthermore, the
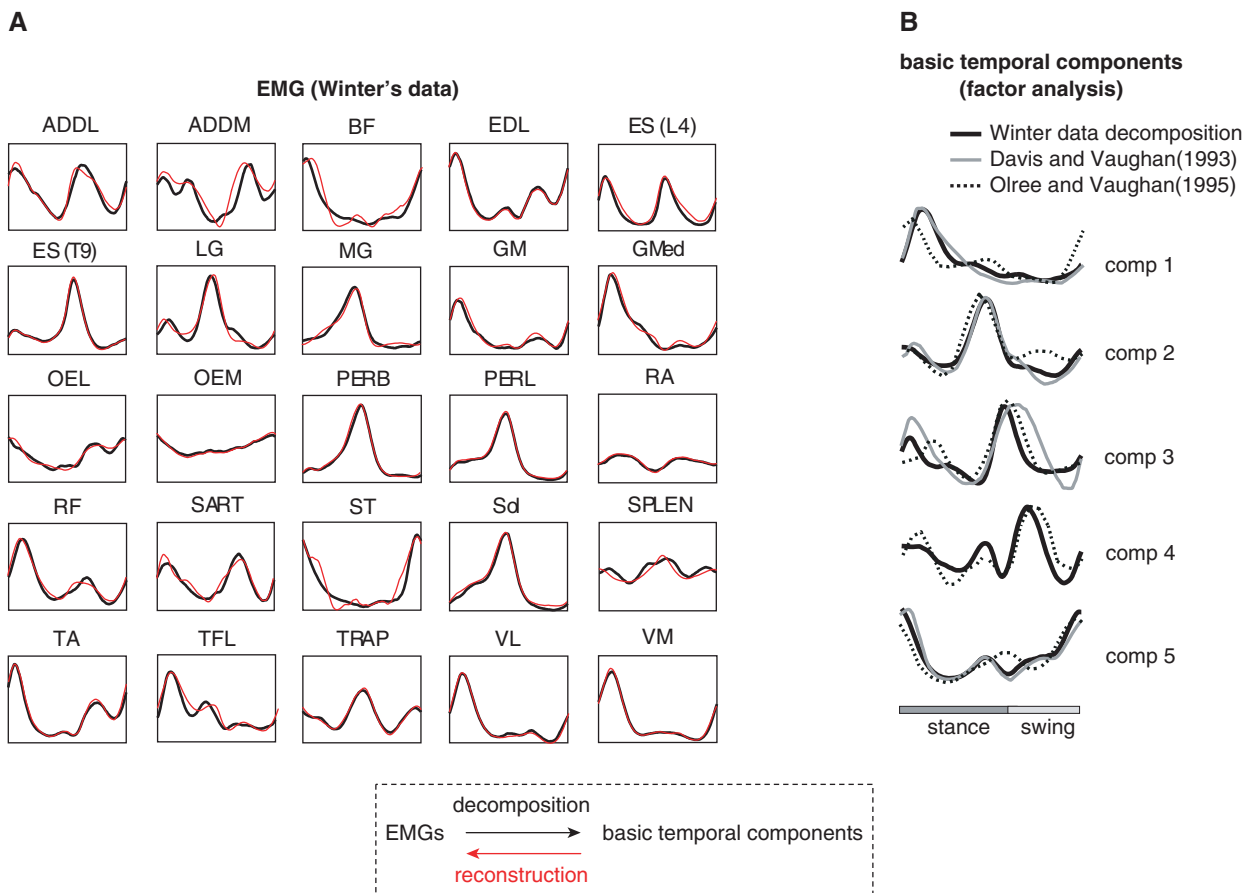
## A

**EMG (Winter's data)**

ADDL  ADDM  BF  EDL  ES (L4)

ES (T9)  LG  MG  GM  GMed

OEL  OEM  PERB  PERL  RA

RF  SART  ST  Sd  SPLEN

TA  TFL  TRAP  VL  VM

## B

**basic temporal components
(factor analysis)**

— Winter data decomposition
— Davis and Vaughan(1993)
···· Olree and Vaughan(1995)

comp 1

comp 2

comp 3

comp 4

comp 5

stance  swing

decomposition
EMGs ⟶ basic temporal components
⟵ reconstruction

**Fig. 2.** Linear decomposition of electromyographic (EMG) waveforms. *A*, averaged EMG activity recorded from 18 subjects for 25 muscles during a single cycle of overground locomotion at a natural speed (~5 km/h). EMG records were filtered with a low-pass cutoff of 3 Hz. Data taken from Winter (1991). *B*, basic temporal components derived from these recordings by factor analysis (FA; black traces). Results are compared with the results, of an FA of EMGs recorded from 16 leg muscles (Davis and Vaughan 1993; gray traces) and from eight muscles in each leg (Olree and Vaughan 1995; dotted traces). Components are designated in a "chronological" order of their main peak in the cycle beginning with touchdown. Red curves in each of the plots in *A* show the reconstructed EMG waveforms (using five basic temporal components depictured in *B*).

five temporal components correspond to the bands of activation seen in the spinal maps (Fig. 1, *bottom*).

The basic rationale for the factor analysis is to represent the original EMG data set $E$ ($m \times t$ matrix, where $m$ is the number of muscles and $t$ is the time base) as a linear combination of $n$ basic temporal components ($n < m$):

$$E = W\,C + \text{residual},$$

where $W$ equals weighting coefficients ($m \times n$ matrix) and $C$ represents basic temporal components ($n \times t$ matrix). Over the past decade, a number of related factor analysis methods have been developed and applied to decomposing multiple EMG records, each based on a different assumption (Davis and Vaughan 1993; Bell and Sejnowski 1995; Lee and Seung 1999; Tresch and others 2006). We recently compared three of these methods with the EMGs recorded during locomotion: factor analysis (FA), independent component analysis (ICA), and nonnegative matrix factorization (NMF; Ivanenko and others 2005). Although each of these statistical approaches places different restrictions on the outcomes (e.g., nonnegative values in NMF), they all converge on a similar solution about the temporal structure of the EMG activity pattern during human locomotion (Fig. 3*A*). The same five basic components accounted for about 90% to 95% of the variance in the original data set regardless of the factor analysis technique used (see also Davis and Vaughan 1993; Olree and Vaughan 1995).

The temporal components are each characterized by the same relatively narrow peak of activation (Gaussian-like) occurring at a specific point in the normalized gait cycle (Fig. 3*A*). The implication of this is that the activity drive represented by each component is scaled by the cycle duration so that muscle activation has a short duration at higher speeds (about 90 ms for walking at 9 km/h) and a much longer duration at low speeds (about 250 ms at 1 km/h). If these periods of muscle activation
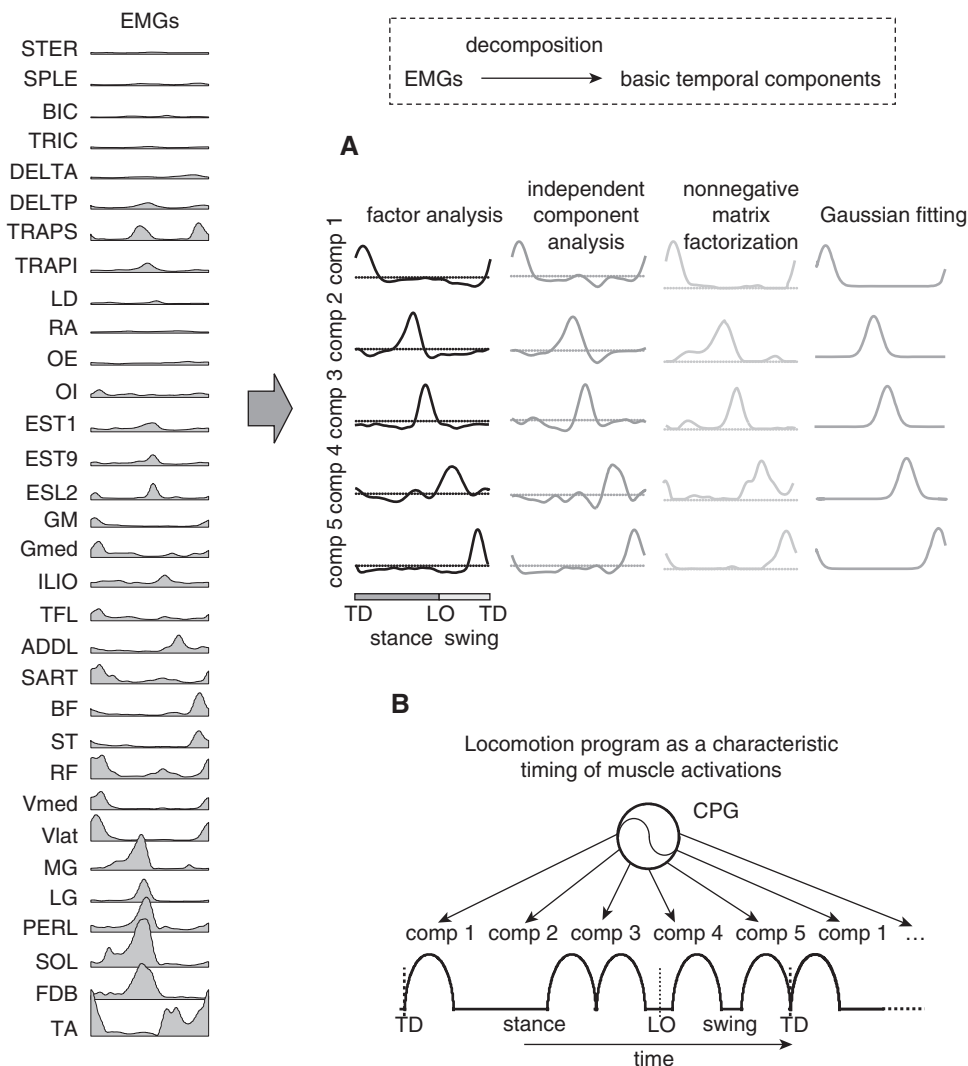
**Fig. 3.** Decomposition of 32 ipsilateral electromyographic (EMG) waveforms into a smaller set of five basic temporal components during normal walking at 5 km/h. *A*, Comparison of activation components obtained using different statistical methods (factor analysis, independent component analysis, and nonnegative matrix factorization). The five basic components are each characterized by a relatively narrow peak of activation (Gaussian-like) at a particular phase of the cycle. Components are designated in chronological order. *B*, Proposed locomotion motor program as a sequence of activation pulses. TD = touchdown; LO = liftoff; CPG = central pattern generator.

represent the activity of a CPG, then they imply that the duty cycle or fraction of the cycle over which the CPG is active is considerably less than 50%. This is unlike the classic half-center oscillator that is postulated to be active for one-half the cycle and inhibited for the other half. Once again, however, this behavior might have its origin in the sensory input, which would naturally be scaled to the biomechanical events.

Another possibility is that the result might be a kind of statistical artifact. Each of the factor analysis techniques attempts to represent the total EMG activity with a set of independent components. It could be that any set of five or so components with appropriate duration and temporal spacing within the step cycle could accomplish this. Thus, it is noteworthy that the EMG activity can also be fitted (using multiple linear regression) with five

Gaussian activation components having a standard deviation of ~6% of the cycle duration (Fig. 1*B*). Unlike FA, ICA, and NMF components, which have minor activity peaks in addition to a main peak (Fig. 3*A*), the Gaussian components are more elemental and perhaps even more likely to account for the total EMG activity. However, arbitrary uniform temporal distributions of five such components can account for only 60% to 80% of the EMG variance, whereas the most variance (~90%) is explained only when the Gaussian peaks are aligned with the main peaks of the FA components (Ivanenko, Poppele, and others 2006).

Another reason to suppose that the result is not a statistical artifact is that the timing of muscle activation revealed by the factor analysis aligns with the activation loci observed in the spinal maps (Fig. 1). The maps also

show that each activation period is associated with MN in particular spinal segments and not in others.

## Bilateral Coordination

Although each limb controller has a relatively autonomous ability to generate rhythmic pattern (as, for instance, can be observed when walking on a treadmill with split belts; Forssberg and others 1980; Yang and others 2005), a remarkable feature of the natural bilateral coordination of MN activity during locomotion is that four of the five activation components are temporally synchronized on both sides of the body (Fig. 3). This was first discovered by Olree and Vaughan (1995), who recorded from leg muscles on both sides of the body and found basically the same five components, two of which (1 and 2) were copies of the other two (3 and 5) but phase shifted by exactly one-half the cycle. This is illustrated schematically in Figure 4. The implication was that the two sets of components were primarily driving muscles on opposite sides of the body (Fig. 3). In fact, Olree and Vaughan (1995) found that components 1 and 2 were predominantly weighted on the ipsilateral leg muscles, whereas components 3 and 5 were more significantly weighted on contralateral leg muscles. It is also evident in the maps (Fig. 1) that although components 1 and 2 are quite prominent in the lumbar and sacral segments, respectively, components 3 and 5 are more evident in the trunk and cervical segments. The latter would be associated with trunk and arm movements that are in opposite phase to the ipsilateral leg and in phase with the contralateral leg. The bilateral synchronization of activation timings occurs around the heel strike events of both legs (Fig. 4A). In contrast, component 4, which explains the least variance and is associated with the ipsilateral foot lift or swing, has no obvious contralateral analog (Fig. 4).

## Invariance of Activation Timings

The pattern of EMG activity for individual muscles in the gait cycle exhibits a great deal of intersubject and context-dependent variability (Winter and Yack 1987; Ivanenko and others 2004). The question arises as to whether the above rhythmic patterning elements are invariant in conditions in which muscle activation patterns can be different from those observed in normal walking. That is, does the variance in muscle activation across subjects or locomotion context imply a variance also in the motor program?

To study this issue, we examined motor patterns at different locomotion speeds and under weight-supported conditions. For a speed range of 1 km/h to 9 km/h, we found that the same five activation components accounted for the EMG activation of up to 32 ipsilateral muscles. There was, however, a consistent speed-dependent phase shift that corresponded to the shift in relative stance duration with walking speed. This again might imply some modulation by sensory input.

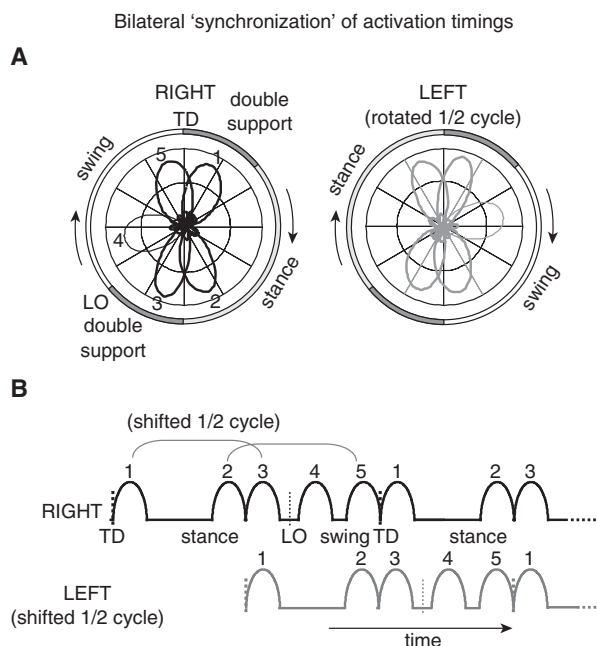By supporting various levels of body weight, we also placed different biomechanical demands on the limbs



Bilateral 'synchronization' of activation timings

**Fig. 4.** Bilateral synchronization of activation timings during normal walking. *A*, Polar plots of factor analysis components. Polar direction denotes relative time over gait cycle (time progresses clockwise), and radius denotes amplitude of component. Double support phase is marked in dark gray. *B*, Superimposed sequence of activation timings on the left and right sides of the body. Contralateral components are obtained from the one-half cycle shift of ipsilateral components. Two of the components (3 and 5) are phase-shifted copies of components 1 and 2. Component 4 has no phase-shifted analog on the contralateral side. TD = touchdown; LO = liftoff.

during locomotion. Although this might be expected to alter the activation patterns of specific muscles (and it did), we again found that the overall muscle activation could be accounted for by the same five activation components. Once again, each activation component described a short period of synchronous activation or relaxation of a particular set of muscles. It should be noted, however, that at the lowest speeds (1 km/h) and higher levels of body unloading (>75%), there tended to be some distortion in the statistically defined components, possibly due to a very low activity in some muscles in these conditions. Nonetheless, the results supported the idea that the same sequence of activation components (Fig. 3) provides a stable motor program that is basically unaltered by changes in locomotion speed or by body weight support during locomotion (Fig. 5).

We also studied patients with spinal cord injury trained to step on a treadmill with body weight support (Ivanenko and others 2003). Patients learned to produce foot kinematics similar to that of healthy subjects but with activity patterns in individual muscles that were generally different from the control group. The corresponding spatiotemporal maps of MN activity are also different (Grasso and others 2004). Spinal lesions

**Same 5 activation components at different speeds and loads**

stance    swing          stance    swing

speed                    body weight support

| — 1 km/h | — 2 km/h |
| — 3 km/h | — 5 km/h |

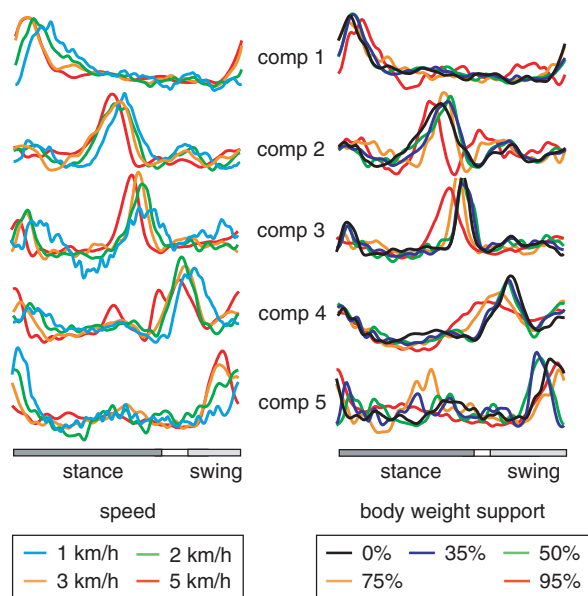| — 0% | — 35% | — 50% |
| — 75% | — 95% | |

**Fig. 5.** Effect of walking speed (*left*) and body weight support (*right*) on component factors for treadmill locomotion. Five common components across conditions are located in chronological order. Adapted from Ivanenko and others 2004.

probably trigger multiple forms of plasticity including modified synaptic strengths (synaptic plasticity), sprouting, and anatomical development of new circuits (anatomical plasticity), as well as plasticity of unlesioned descending pathways, including both subcortical and cortical motor areas. In particular, stepping may depend more heavily on cortical (and voluntary) control after severe spinal lesions than it does in healthy subjects, in whom locomotion may be more automatic. Yet we found a similar set of five temporal components in the EMG activity of the patients, implying that a flexible combination of those components could account for the wide range of muscle patterns we observed. Moreover, muscles both rostral and caudal to the lesions could be strongly weighted on a given component. It seems that the invariance of activation timings in these patients reflects in some way the global kinematic goal of an appropriate foot trajectory (a motor equivalent solution; Ivanenko and others 2003).

## Superposition of Motor Programs

A further test of this concept of a stable, temporally defined motor program for locomotion was the extent to which it might be preserved when the basic locomotion pattern is perturbed by voluntary movements. An important aspect of everyday locomotion is an extremely flexible supraspinal modulation and accommodation of the locomotor pattern, for example, gait acceleration/deceleration, turning, and combination with other motor

activities. Does this require a comparable flexibility in the basic locomotion program, or is the voluntary muscle activity somehow superimposed on the locomotion program? To gain some insight about how this interaction might take place, we examined several voluntary tasks added to overground walking in which subjects kicked a ball, stepped over an obstacle, or reached down and grasped an object on the floor (with weight support on either the right or the left foot; Ivanenko and others 2005). Our basic finding was that five basic activation timing patterns underlying locomotion were invariantly present when voluntary tasks were performed during locomotion (Fig. 6*A*). The voluntary tasks were accompanied by an additional sixth activation component timed to the voluntary task and by different loadings on the five basic temporal components.

The appearance of new task-specific components suggests that the basic locomotion timing pattern was not adequate for a coordinated voluntary task. Indeed, when the same voluntary tasks were performed while standing (step-in-place and stoop-in-place task; i.e., without locomotion), we revealed few significant components (Fig. 6*B*) that accounted for more than 60% of the variance, with higher order components being variable from trial to trial. Moreover, the timing of these components was strictly linked to specific kinematic events (the onset of foot lift and of the vertical object movement). If superimposed relative to this event, a temporal summation of the locomotion (Fig. 7*A*) and voluntary (Fig. 7*B*) programs resulted in a combined sequence of activation timings (Fig. 7*C*). This phenomenon could be expected if the characteristic activation timing for locomotion was generated separately from any voluntary activation timing, although a coupling of corticospinal with propriospinal circuits (Dietz 2002; Drew and others 2004; Zehr and Duysens 2004) might result in partial synchronization of activation components (Figs. 6, 7; see also Nashner and Forssberg, 1986) and/or sharing of neural elements in the locomotor pattern generator with those for a different behavior (Stein and Smith 1997; d'Avella and Bizzi 2005).

## Muscle Synergies and Timing Generation Networks

The idea that motor tasks employ synergies specific to the task is long standing in the motor control literature (Latash 1999). A muscle synergy is usually considered as the set of muscles that are synchronously activated in a task. For instance, a mixed muscle synergy (hip and knee extensors plus ankle flexors) becomes active in locomotion at the moment of heel strike, whereas a synchronous activation of the ankle extensors comes into action by the end of the stance phase during normal locomotion. Another set of examples that has been well documented in the literature is the set of muscle synergies participating in automatic postural responses to stance perturbations (e.g., the so-called hip or ankle strategies; Horak and Nashner 1986; see also Krishnamoorthy and others 2003; Ting and Macpherson 2005). In addition, specific correcting synergies seem to be incorporated into the
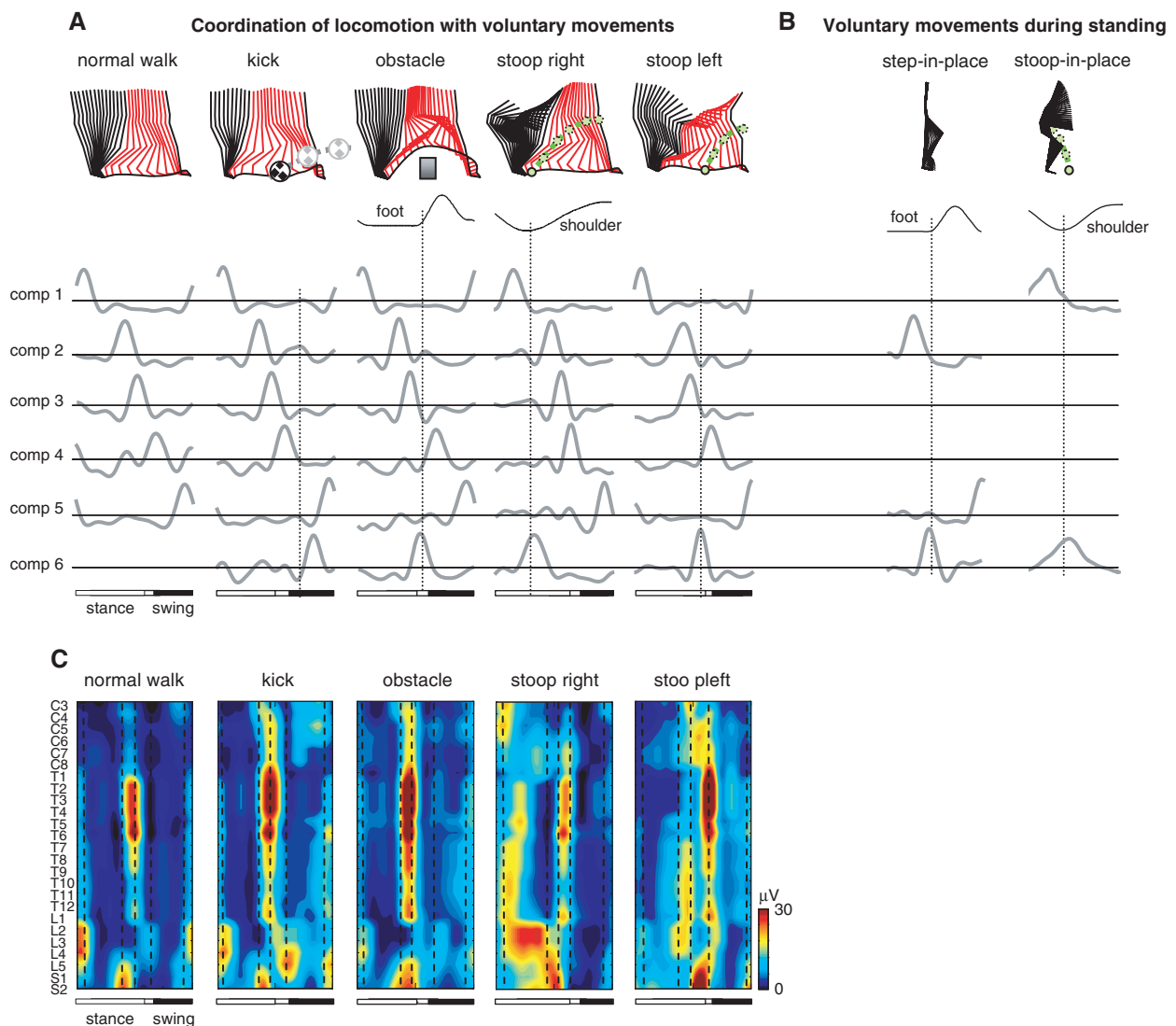
**Fig. 6.** Coordination of locomotion with voluntary movement. Recordings were made during normal walk, kick, obstacle, stoop right, and stoop left tasks. *A*, Averaged activation components derived from muscle activity patterns by factor analysis (adapted from Ivanenko and others 2005, with permission, copyright 2005 by the Society for Neuroscience). The gait cycle and ipsilateral activation components were defined with respect to the right limb in all conditions. Five common components across conditions are located in chronological order, whereas the new sixth component in the voluntary tasks was plotted separately. Stance phase was somewhat different across subjects as indicated by the hatched area of the stance-swing bar. Dotted vertical lines correspond to the timing of the voluntary task estimated as the onset of the horizontal ball movement in the kick condition, the onset of the foot (fifth metatarsum marker) elevation in the obstacle task, and the onset of the vertical object movement in the stoop tasks. *B*, Most significant activation components during single foot lift (step-in-place) and reach-and-grasp (stoop-in-place) movements performed while standing. Dotted vertical lines denote the timing of the voluntary tasks (the onset of the foot lift and of the vertical object movement, respectively). *C*, Corresponding spatiotemporal maps of motoneuron activation in the spinal cord. Same format as in Figure 1. Vertical dashed lines denote the timing of the main peak of five basic components in *A*.

basic locomotor program (Nashner 1980; Nashner and Forssberg 1986). However, even though fixed muscle synergies for locomotion may exist, they should be temporally organized to produce the full locomotor pattern. It appears then that the question of muscle activation during locomotion can be divided into two parts, the timing of activation and the specification of which muscles to activate. In fact, the necessity for such a dual motor

pattern representation has given rise to a nontraditional definition of muscle synergy: a time-varying muscle synergy (d'Avella and others 2003).

The timing of muscle activation during locomotion clearly involves the spinal pattern-generating circuitry. It is tempting to speculate by analogy with the animal literature (Brown 1911; Kiehn and others 1998; Orlovsky and others 1999) that this locomotion circuitry may
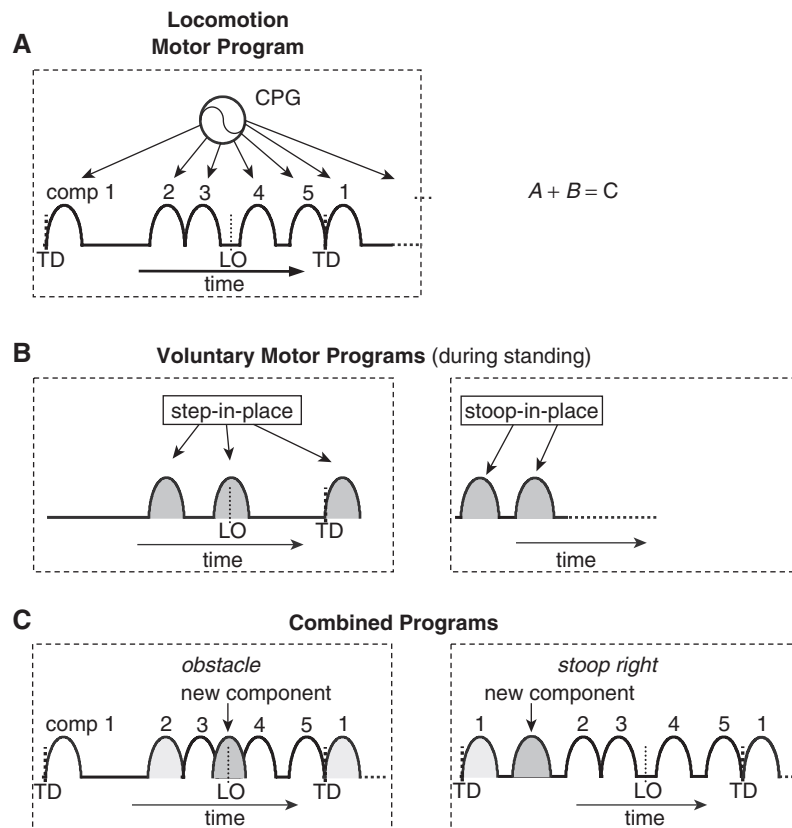
**Fig. 7.** Superposition of motor programs. Locomotion (white, *A*) and voluntary (gray, *B*) programs are considered as temporal sequences of activation pulses. To produce a compound movement (*C*), the basic underlying programs are combined: $A + B = C$. A coupling of corticospinal with propriospinal circuits may result in partial synchronization of activation components (light gray). TD = touchdown; LO = liftoff.

consist of a few simple oscillating circuits that provide the major input to the active MNs during locomotion (Patla and others 1985; Davis and Vaughan 1993). However, this leaves open the question of how the temporal sequence of activation timing comes about and how it is directed to the MNs.

It is clear that muscle activation timing is linked to specific kinematic events, but it is not clear to what extent the kinematic events may drive activation via proprioceptive feedback (e.g., Pearson 1995; Duysens and Van de Crommert 1998) or to what extent intrinsic motor programs drive the kinematics. There is recent evidence from the cat that the spinal circuitry itself encompasses critical periods that may help shape the sequence of muscle activation (Saltiel and Rossignol 2004).

The sequence of activation components in locomotion bears some resemblance to the sequential activation of premotor drives or muscle synergies activated with pulses in decerebrate and spinal frogs (Hart and Giszter 2004). Those studies showed that a variety of different motor behaviors, both reflex and spontaneous, were associated with activation pulses having a characteristic duration. Although different speeds of movement were not explored in this system, the result seems to be different from the locomotion result in which the duration

of the activation pulses was scaled to the duration of the gait cycle. Another result of the frog study was the finding that each premotor drive was associated with a small core group of muscles or a muscle synergy. The picture that seems to emerge from this is that motor activity may be constructed from a series of discrete activation events (pulses) generated by premotor circuits. A relatively small number of such circuits activates a given set of muscles (a synergy), and appropriate combinations of synergies can produce any given movement.

The temporal patterns we observed in muscle activation during locomotion may correspond in some way to the discrete pulses observed in the frog. The linkage between these pulse events and the muscle activation during locomotion is not so clear, however. For example, a temporal component might be loaded on proximal leg muscles in one condition, whereas the same component is loaded on more distal leg muscles in another condition. Such a redistribution of the component loadings across muscles occurs with speed and under weight-supported conditions (Ivanenko and others 2004). For instance, at 5 km/h, the hamstring muscle loaded heavily in all subjects on component 5 ($r \sim 0.9$), whereas at 1 km/h, it loaded heavily as well but on components 1, 2, 4, and 5 ($r = 0.7–0.95$) depending on the subject.
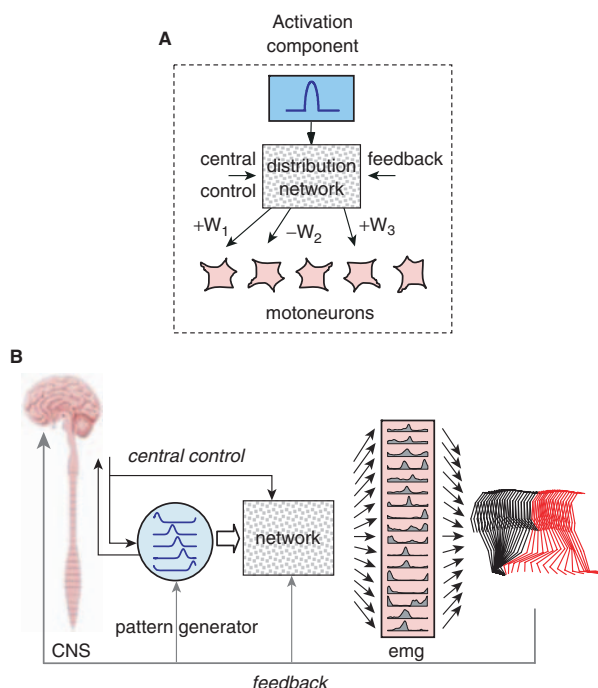
**Fig. 8.** Hypothetical scheme of muscle activation in human locomotion. *A*, Activation components. Activation pulse acts via a distribution network to activate a dynamic synergy controlled by feedback and central commands with $+W_n$ and/or $-W_n$. *B*, Activation components are expressed as a temporal sequence by spinal pattern generators and/or descending commands that are modulated by afferent signals. Ascending pathways and corresponding rhythmic modulation of the central structures provide the basis for a superposition of locomotor motor program with voluntary motor program. The distribution of activation components occurs downstream from the timing in a network that is dynamically configured by sensory feedback and central control.

At the higher levels of body weight support, the component loadings changed for most of the muscles. In addition, the individual loadings are generally different in spinal cord injury patients, although they show a similar set of five temporal components (Ivanenko and others 2003).

A marked redistribution of components across muscles could also be seen in part in corresponding spinal maps of MN activity (Fig. 6*C*): Component 4 is strongly loaded on lumbar segments to produce a kick, whereas component 2 is strongly loaded on the proximal muscles (upper lumbar spinal segments) in the stoop tasks but on the distal extensor muscles (sacral segments) in the normal walk condition. Trunk stabilization activity in the cervico-thoracic regions is typically loaded on component 3 during normal walking, whereas it is significantly delayed in stoop conditions and loads on component 4. It is also worth noting that although some muscles are loaded heavily on single components, many muscles have loadings on more than one component.

A straightforward interpretation of our results is that locomotion timing generation networks activate MNs via a distribution network that sets weightings according to various feedback and feed-forward parameters (Fig. 8; Perret and Cabelguen 1980; Patla and others 1985; Poppele and Bosco 2003; Ivanenko and others 2004). A major part of the proprioceptive feedback, along with the cyclic excitability of spinal interneurons and ascending and descending pathways (Orlovsky and others 1999; Dietz 2002; Drew and others 2004), might interact to shape the motor output to provide an appropriate kinematic pattern (Lacquaniti and others 1999). What is suggested by our findings is that the selection of muscle synergies is downstream from the processes that generate activation timings (Fig. 8).

## Concluding Remarks

Locomotor movements can be readily accommodated to various external conditions, and some of the suggestions in this article based on statistical methods may possibly be revised as empirical data on the neural substrates underlying natural locomotor behavior accumulate. Nevertheless, some strong conclusions can be made from the above considerations. Muscle activity during human locomotion seems to be driven by a similar set of five temporal activation components distributed to various muscles depending on the kinetic and kinematic demands of the limbs (Fig. 8). In addition to a spatial architecture of neuronal networks (Bizzi and others 2000; Giszter and others 2001; Hultborn 2001; Lemay and Grill 2004; Ivanenko, Wright, and others 2006), which selects the appropriate muscle pattern for compound movements, there may also be a temporal architecture that uses discrete timing intervals to coordinate the elements of compound movements.

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