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Cortical transients preceding voluntary movement

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ABSTRACT

The process of initiating a voluntary muscular movement evidently involves a focusing of diffuse brain activity onto a highly specific location in the primary motor cortex. Even the very simple stereotypic movements used to study the 'contingent negative variation' and the 'readiness potential' begin with EEG indicative of widely distributed brain activity. In natural settings the involvement of diffuse cortical networks is undoubtedly even more important. Eventually, however, activity must coalesce onto specific neurons for the intended movement to ensue. Here we examine that focusing process from a mathematical point of view. Using a digital simulation, we solve the global equations for cortical dynamics and model the flow from diffuse onset to localized spike. From this perspective the interplay between global and local effects is seen as a necessary consequence of a basic cortical architecture which supports wave propagation. Watching the process evolve over time allows us to estimate some characteristic amplitudes and delays.

1. Introduction

The process of initiating a voluntary muscular movement evidently involves a focusing of diffuse brain activity onto a highly specific location in the primary motor cortex. Even the very simple stereotypic movements used to study the 'contingent negative variation' (CNV) [1,2] and the 'readiness potential' [3,4], begin with EEG apparently indicative of widely distributed brain dynamics. In natural settings the involvement of diffuse cortical networks is undoubtedly even more important [5]. Eventually, however, activity must coalesce onto specific neurons for the intended bodily movement to ensue. Here we examine that focusing process from a mathematical point of view. Using a digital simulation, we solve the global equations for cortical dynamics [6] and model the flow from diffuse onset to localized spike. From this perspective the interplay between global and local effects is seen as a necessary consequence of the basic cortical architecture which supports wave propagation.

An understanding of brain activity preceding normal movements could have important practical implications. As prosthetic devices become more fully functional imitations of the limbs and organs they replace, the interfaces for their control take on added significance. To achieve smooth activation of these complex devices, engineers seek 'intuitive' control mechanisms, mimicking the natural flow insofar as they are able and interfacing to the body's neural system as early as understanding and technology permit. 'Mind reading' might summarize the goal. If a user's intentions for fine motor movements could be reliably detected, then mechanical devices might become much more useful to him. And if that detection took place at the cortical level, then response times could be shortened and prosthetic devices used even by those with severe injuries to the peripheral nervous system. Advances in this technology might prove especially beneficial to so-called 'locked-in' patients suffering (mainly) from ALS and brainstem strokes. Already some success with brain-computer interfaces has been achieved, enabling patients to move a cursor on a computer screen [7] and to control an artificial hand [8].

Regrettably little is known about the specific cortical events that precede voluntary muscle movements. The 'motor strip' itself is well mapped, and it is clear that highly specific neurons in it must fire to initiate the contraction of particular skeletal muscles. The necessary cortical activity must have a distinctly localized character, focusing on certain cells and excluding others located nearby. But these same neurons are richly interconnected with others in a structure that supports complex wave activity and precludes their firing as isolated entities. Indeed, EEG patterns recorded preceding voluntary movements are characterized by very diffuse waves at the onset. The mathematical simulation reported here represents an attempt to understand and model how this diffuse beginning leads to the eventual sharply localized result. Watching the process evolve over time allows us to estimate some characteristic amplitudes and delays.

Both the 'contingent negative variation' (CNV) [1,2] and the 'readiness potential' [3,4] are EEG waves marked by a negative voltage spread over a wide region at the top of the head. This voltage grows in magnitude over a period of about half a second prior to a muscular response. In the case of the CNV, larger voltages are

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associated with more intense involvement in the task and with shorter reaction times [2]. Occasionally the CNV is sufficiently large as to be observed in the raw EEG, but its study usually requires time-locked averaging. The readiness potential is similarly small, but somewhat differently distributed. In particular, it is more pronounced over the hemisphere contra-lateral to the responding hand. Alan Givens writes, 'About 200 milliseconds after the second stimulus, the CNV ends in a sharp positive-going ($\sim$10 Hz) wave termed the CNV resolution' [2]. The readiness potential appears to end less suddenly [4]. In any event, this marks the onset of muscle movement, and concomitant inputs to the adjoining somatosensory cortex contribute to the EEG from this point forward.

To see how a sharply localized response can evolve in this richly interconnected system, we have examined the global equations for 'neocortical dynamics', used successfully by Nunez [6,10,11], Katznelson [9], Nunez and Srinivasan [11] and others to elucidate widespread cortical rhythms as observed in EEG. These equations are based on a columnar model of the cortex with inhibitory interconnections that are predominantly short-range and with excitatory connections that at their longest span a cortical hemisphere. The density of these long fibers decreases with distance, with a characteristic length of about 10 cm. Their finite conduction velocity gives rise to the propagation of waves in synaptic activity. Both Nunez [10] and Jirsa and Haken [12] have joined the global equations to the contributions of local networks in the generation of EEG. The current work stops short of this goal, considering only global relations.

The equations are normally written in integral form, with the activity at a particular cortical column being determined by the subcortical and commissural inputs to that location plus an integral over connections from the surrounding cortex, taking activity at earlier times as required by the intervening distance and the conduction velocity. About 92% of the long axon nerve fibers arriving at a cortical column are from other locations in the same hemisphere [9]. Although the inhibitory connections are often associated with a substantial delay, we follow treatments by Nunez in which their effects, along with those of the short-range excitatory fibers, are considered as 'instantaneous' (i.e. as comparable to post-synaptic delays within a cortical column, which are also neglected). The dynamics of the system depend in part on a ratio of overall positive to negative feedback as captured in a single parameter, B, which controls the gain for inputs of relatively long wavelength. We have taken $B = 0.95$ which preserves stability of the linearized equations. Larger values of B lead to more labile behavior and are considered more realistic by some workers, but they require constant subcortical inhibitory inputs to keep the system operating in a quasi-linear region [13].

In the work reported here, the equations have been transformed from their usual integral representation to an equivalent partial differential form better suited to digital simulation (see Appendix A). They are solved on a closed spherical surface with a circumference of 80 cm, corresponding approximately to a cortical hemisphere 'inflated' so as to smooth its fissures and undulations. The conduction velocity of the long fibers is taken to be 800 cm/s. The digital simulation used discrete cortical elements with centers separated by no more than 0.8 mm and time steps of 1.0 ms.

2. Initial priming

Our simulation begins with a diffuse, low-level input held constant for half a second. The input has a broad maximum over the eventual focus point in one hemisphere and tapers off to zero on its opposite side. The resulting cortical activity takes on a similar pattern and reaches somewhat larger amplitude. The response rises slowly with a time constant of about 100 ms and levels off with a relative gain of about 1.8. (The absolute gain is considerably greater, but we wish to demonstrate a benefit of priming with long wavelengths. As mentioned in Appendix A, we have reduced to unity a multiplicative constant that affects all wavelengths equally.) Fig. 1 shows the steady input (green) and increasing response (red), now nearing its final amplitude at the peak location. This response represents increased post-synaptic activity in pyramidal neurons across a large region. These cells are aligned perpendicular to the cortical surface, and their collective activity is reflected in a similarly widespread negative voltage on the scalp, corresponding to the initial phases of the CNV and readiness potential.

3. Trigger onset

At any time after initial priming, motion can be triggered by changing the input over a disc surrounding the focus point. Here a small negative input (*i.e.* a reduction from prevailing ambient) is applied over a disc of radius 35 mm as shown in Fig. 2. Responses corresponding to inputs of short wavelength are oscillatory and under-damped. A small wave front begins to propagate in both directions from the discontinuity. The out-bound wave decreases in amplitude as it propagates both because of the overall preponderance of negative feedback and because the wave front spreads over an increasingly larger perimeter. The in-bound wave, however, grows in amplitude as it concentrates over a decreasing perimeter, this effect out-weighing that of the negative feedback. A disc with radius 35 mm is near the optimum for the ratio of positive to negative feedback used in the simulation ($B = 0.95$). For larger discs the inbound wave initially loses amplitude, while smaller ones would benefit from a greater circumference of initial activity.

4. A big bounce

The wave of reduced activity grows in amplitude as it propagates toward the disc center as shown in Figs. 3 and 4 (red[1] traces) where it converges to a trough after 62 ms. The locally reduced activity is reflected in a momentarily positive scalp voltage. The system will rebound in a few milliseconds. All input was removed 5 ms after the trigger pulse was applied. (Green traces in Figs. 3 and 4 show zero.) The consequent second wave of opposite polarity is also converging and will reinforce the oscillatory maximum.

5. Focusing pulse

As the effects of the trigger pulse and its removal converge, a small focusing pulse is applied to select the particular motor neurons necessary for the desired muscle movement. Being of very short spatial extent, the effect of this pulse is almost immediate. It combines with the waves initiated earlier to drive the activity to a large value at a highly specific location. This is shown in Fig. 5 where the overall pulse amplification is more than 13:1. The narrow width of the resulting activity places it beyond the validity of the global wave equations. A more detailed accounting would require considering interactions with the local motor network [10].

Although the trigger and focusing pulses are of low amplitude, their relative timing is crucial. For the 35 mm trigger disc used in our simulation, the focusing pulse must follow the trigger by 69 ms to achieve its greatest effect. This constitutes an appreciable fraction of the very fast reaction times often recorded in experiments in which the CNV is prominently seen.

[1] For interpretation of the references to color in Fig. 4, the reader is referred to the web version of this paper.