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Aaron Schurger

Pengbo 'Ben' Hu

Joanna Pak

Adina L. Roskies

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Opinion

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Aaron Schurger,[1,2,3,4,7,*] Pengbo 'Ben' Hu,[5] Joanna Pak,[2] and Adina L. Roskies[6,7,*]

The readiness potential (RP), a slow buildup of electrical potential recorded at the scalp using electroencephalography, has been associated with neural activity involved in movement preparation. It became famous thanks to Benjamin Libet (*Brain* 1983;106:623–642), who used the time difference between the RP and self-reported time of conscious intention to move to argue that we lack free will. The RP's informativeness about self-generated action and derivatively about free will has prompted continued research on this neural phenomenon. Here, we argue that recent advances in our understanding of the RP, including computational modeling of the phenomenon, call for a reassessment of its relevance for understanding volition and the philosophical problem of free will.

Highlights

The readiness potential (RP) has been widely interpreted to indicate preparation for movement and is used to argue that our brains decide before we do. It thus has been a fulcrum for discussion about the neuroscience of free will.

Recent computational models provide an alternative framework for thinking about the significance of the RP, suggesting instead that the RP is a natural result of the operation of a stochastic accumulator process of decision-making, analyzed by time-locking to threshold-crossing events.

These models call for a reevaluation of: (i) the ontological standing of the RP as reflecting a real, causally efficacious signal in the brain; (ii) the meaningfulness of temporal comparisons between the 'onset' of the RP and the timing of other phenomena; (iii) the moment at which we, as experimenters, identify that a decision to act has been made; and (iv) the relevance of the RP for discussions of free will.

The Readiness Potential

The **readiness potential (RP)** (see Glossary) or *Bereitschaftspotential* (BP) is a brain signal linked to voluntary movement. Its existence has been used to argue against the possibility of free will. Originally identified by Kornhuber and Deeke [1], the RP emerges from the analysis of electroencephalogram (EEG) data recorded during experimental tasks involving spontaneous or self-paced movements. When EEG traces, recorded during such a task, are **time-locked** to movement onset and averaged together, a slow negative-going electrical potential is evident leading up to movement onset (Box 1). The RP is prominent at central electrode sites located above mesial motor cortical areas and peaks contralateral to the moving limb. In experiments that average data from multiple subjects making self-paced movements, the RP is highly replicable. The RP has traditionally been interpreted as a sign of planning and preparation for movement and it is well-established as a reliable signal that precedes self-initiated movement in the group average. However, recent literature raises questions about the RP's ontological status as a real signal in the brain, its relation to action, its significance for arguments about volition, and its implications for free will. We review this recent literature and offer a reinterpretation of the nature of the signal that undermines its relevance for classic arguments against free will.

Historical Background

The RP gained notoriety largely due to the work of Benjamin Libet in the 1980s [2]. Libet asked subjects to spontaneously and repeatedly perform a simple movement, flexion of the fingers and/or wrist, while he measured EEG activity and electromyography (EMG) from the relevant muscles. Subjects also monitored a rapidly rotating clock dial and were told to note, for each movement, the time on the clock at which they first felt the urge, or will, to move (**W-time**). Their retrospective reports enabled Libet to establish a temporal relationship between a subject's self-reported awareness of willing to move, the time of movement, and the onset of the RP. The results are familiar to many even outside of neuroscience: average W-time is only approximately 200 ms before movement onset, hundreds of ms after the apparent onset of the RP. Libet correctly reasoned that if subjects consciously willed themselves to move after the brain began preparing to move, conscious will could not cause the RP's initiation. Given the RP's perceived status as a preparatory signal to move, he further reasoned that the subjects' brains had already unconsciously initiated movement before their conscious willing. As he put it: '…the brain evidently "decides" to initiate or, at the least, prepares to initiate the act at a time before there is

[1]Department of Psychology, Crean College of Health and Behavioral Sciences, Chapman University, One University Drive, Orange, CA 92867, USA
[2]Institute for Interdisciplinary Brain and Behavioral Sciences, Chapman University, 14725 Alton Parkway, Irvine, CA 92618, USA
[3]INSERM, Cognitive Neuroimaging Unit, NeuroSpin Center, Gif sur Yvette 91191, France
[4]Commissariat à l'Energie Atomique, Direction des Sciences du Vivant, I2BM, NeuroSpin Center, Gif sur Yvette 91191, France
[5]Department of Linguistics and Cognitive Science, Pomona College, Claremont, CA 91711, USA
[6]Department of Philosophy and Program in Cognitive Science, Dartmouth College, Hanover, NH 03755, USA
[7]These authors contributed equally to this work

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*Correspondence:
schurger@chapman.edu (A. Schurger)
and adina.roskies@dartmouth.edu
(A.L. Roskies).

Box 1. The Readiness Potential, Past and Present

The empirical literature on the RP is vast and varied and difficult to fit under a single theoretical account. Libet's [2] classic study distinguished between two types of RPs: type I RPs, involving 'preplanning', had significantly earlier onsets (approximately 1000 ms prior to movement) than type II RPs, associated with spontaneous movement (approximately 500 ms prior), but *cf.* [66] who found the opposite to be true with a larger cohort of subjects. The RP is commonly characterized as having an early and a late component [29]. The early component (~1500–400 ms prior to movement onset) is a slow but gradual increase in negativity, symmetrical between the two hemispheres, that has been attributed to activity in the supplementary motor area and premotor cortex, whereas the late component (~400–0 ms) is generated by activity in the primary motor cortex. The spatial focus and amplitude of the RP are dependent on particulars of the experimental task, including movement effector [67–69], limb dominance [70], attention to timing of movement [71] or the intention to move [72], and other task factors [73,74], including possibly beliefs in free will [75]. Recent work suggests that the RP may be more prominent in arbitrary versus reason-based decisions [65].

Numerous studies linking the RP to a variety of phenomena and experimental manipulations complicate its interpretation, especially as a phenomenon indicative of volition. For example, the RP may be related to and modulated by anticipation of the sensory and/or somatosensory consequences of the action [76,77] and by awareness of movement intention [78]. Some have suggested that the RP is linked to decision uncertainty [41,79], but at least in one way of operationalizing uncertainty, this is not borne out [80]. Consistent with other work [65], it appears to be associated more with endogenous than external cues [80]. The RP is diminished in amplitude or absent with cerebellar lesions [81] and diminished by damage to the prefrontal cortex [82] and in neuropsychiatric conditions such as Parkinson's disease [83] and obsessive compulsive disorder (OCD) [84]. It is unclear whether or not the (seemingly involuntary) tics of Tourette's patients are preceded by an RP [85]. The breathing cycle, a largely involuntary phenomenon, has been linked to a modulation in the amplitude of the RP [86]. However, because breathing strongly influences the onset time of spontaneous voluntary movement [86], it is difficult to determine whether these observations reflect a causal relationship or an incidental one.

any reportable subjective awareness that such a decision has taken place. It is concluded that cerebral initiation even of a spontaneous voluntary act, of the kind studied here, can and usually does begin unconsciously…These considerations would appear to introduce certain constraints on the potential of the individual for exerting conscious initiation and control over his voluntary acts.' [2]. Since many believe that conscious initiation and control over one's voluntary acts are required for free will, Libet's work was widely interpreted as undermining the possibility of free will [3–8].

Libet himself recognized at least two ways his stark conclusions negating free will were limited. First, he postulated that we could veto our brain's unconscious decisions in the period between when we became aware of our intention to move and the movement itself; this **veto power** is commonly referred to as 'free won't' [9]. Subsequent studies have shown that such conscious veto decisions are also preceded by an RP and thus subject to the same problematic delay before W-time, making such a position seem untenable [10–12]. Also, a recent study estimated the point of no return in vetoing self-initiated movements to be about 200 ms before movement onset [13], roughly the same time that Libet thought the veto window opened. This also makes 'free won't' seem untenable, because the window of time in which Libet suggested subjects could veto a movement begins precisely when subjects can no longer veto a movement. More helpfully, Libet suggested that many of our actions result from conscious deliberation that unfolds over much longer time-scales [2], which would not be subject to these timing issues. Indeed, philosophers have argued that few of the decisions for which responsibility (and thus free will) is of concern fit the profile of Libet-style tasks [14–16].

It is difficult to overstate the degree to which the conclusions of Libet's papers on the RP have permeated the intellectual *zeitgeist*. Despite numerous critiques [3,14,17–20], many neuroscientists concur that our brains decide before we do that our actions are unconsciously initiated and that we therefore lack free will [5,21–24]. Furthermore, Libet's work has sparked ongoing debates in philosophy with many thinkers accepting his conclusions regarding the relative timing of awareness and action initiation, even if they reject its ultimate relevance to the free will debate. His work

has also been showcased in the popular press [6,8,25–27] with little critical commentary. Recent fMRI evidence showing that free decisions can be predicted several seconds before the choice is made [22,28] has helped to cement this view. Note, however, that although prediction accuracy was statistically significantly above chance, it was still only marginally better than a random guess.

Because of the dramatic conclusions drawn from Libet's work, the RP continues to be a topic of interest in the brain sciences, including among neurologists. Research on the RP was thoroughly reviewed in 2006 [29], but our understanding of the RP, and the underlying phenomenon of a **premovement buildup (PMB)** of neural activity (Box 2) more generally, has been challenged in recent years. In what follows, we review recent work on the RP and discuss how new models inform our interpretation of the RP and its relevance to questions of volition and free will. As the classical view takes the RP to mediate between volition (or conscious intention) and action, we begin by focusing on its relevance to both phenomena.

The RPs Connection to Action

Understanding the functional significance of the RP is complicated by difficulties in experimentally distinguishing it from other slow wave potentials. For example, the contingent negative variation (CNV) and stimulus-preceding negativity (SPN) look very similar to the RP in shape and spatial distribution (Box 3). The most notable difference between them is the experimental paradigms in which they are generated. It remains unresolved whether these signals are meaningfully distinct. In addition, although the RP has traditionally been regarded as an indicator of motor preparation or movement initiation, some studies have reported RPs in decision tasks that do not involve motor activity at all [30]. Thus, these studies call into question the tight relationship postulated between the RP and action.

The surest way to verify that the RP reflects the proximal cause of movement would be to detect it on single trials and show that it predicts movement**.** If one reliably found RP-like events preceding individual self-initiated movements (ideally by a fixed interval), but not at any other time, this would indicate that RPs reflect decisions to move (note that, unless stated otherwise, the phrase

Box 2. The Premovement Buildup (PMB)

The RP was first discovered using EEG, hence the name readiness 'potential' (referring to the electrical potentials measured with scalp electrodes), but it ultimately reflects a gradual buildup of neural activity in the neural tissue below the scalp EEG electrode. This buildup can be measured in other ways: when measured using magnetoencephalography, we refer to it as a 'readiness field', and when measured using implanted electrodes at the single-neuron level we refer to it as a 'readiness discharge'. The underlying neural phenomenon is the same, a slow buildup in the firing rate or recruitment of individual neurons in premotor and motor areas, which we might refer to as a PMB.

The PMB is not a uniquely human phenomenon [37] as it has been observed in monkeys [87–90], rodents [91–94], fish [95], and even crayfish [96,97]. The fact that the PMB is also observed in invertebrates tells us that it is not unique to the cerebral cortex, even though the PMB is often referred to in the EEG literature as the 'cortical readiness potential'. The signal measured using EEG most certainly originates in the cortex, but the buildup itself may emerge from an interaction between cortex and subcortical structures, notably the basal ganglia [98,99]. We suggest using the label 'RP' to refer to the movement-related signal measured with EEG and 'PMB' to refer to the underlying neural modulation measured in multiple species and with various techniques.

The particular temporal profile of the PMB in general, and the RP in particular, is an oft-neglected feature and has not been systematically studied in the way that, for example, the onset or amplitude has. Often it appears to exhibit a nonlinear exponential-looking buildup [1,13,41,86,100,101], but in other studies it appears as more of a linear ramp [2,66,102,103]. It makes sense to speak of an 'onset' time if the buildup is linear, but if it is nonlinear then it seems to decay asymptotically into the past and there is no principled reason to declare it to 'begin' at one point in time rather than another. Whether the RP/PMB is fundamentally a linear or nonlinear ramp and what the factors are that determine its shape remains unclear.

Glossary

Agent-causation: theory that free actions must be caused by agents that are themselves uncaused to act.
Compatibilism: philosophical view that free will is compatible with determinism.
Early-decision account: holds that decision occurs well prior to action, at the point when the RP deviates from baseline.
Incompatibilism: philosophical view that free will cannot exist if determinism is true.
Late-decision account: holds that decision occurs very close in time to action, when threshold is crossed.
Lateralized readiness potential (LRP): a signal computed as the difference between contralateral and ipsilateral electrodes positioned over primary motor cortex. The difference becomes lateralized roughly 200–400 ms before movement onset and is more directly related to motor execution than is the RP.
Point-of-no-return: point in time after decision to act after which an agent can no longer veto or withhold a muscle contraction.
Premovement buildup (PMB): buildup of neural activity prior to action that is seen across many modes of measurement.
Readiness potential (RP): the signal identified in averaged EEGs time-locked to movement onset that shows a premovement buildup of activity prior to action. The signal is measured over premotor cortex and precedes movement by anywhere from 0.5 to 1.5 seconds.
Stochastic accumulator model: a model of decision making and/or action initiation that involves taking a temporal integral over a constant (evidence or imperative) plus noise and (optionally) leakage. The decision is made and/or action initiated when the integral reaches a given threshold.
Stochastic decision model (SDM): a model that incorporates ongoing stochastic fluctuations that influence the precise time at which a decision is made and/or an action is initiated.
Threshold crossing: in stochastic decision models, the point at which a decision threshold is crossed.
Time-locking: EEGs measure electrical signals that vary with time. When individual trials contain identifiable events that can be indexed to the EEG traces, the traces can be aligned with

Box 3. Different Event-Preceding Potentials

Several different slow cortical potentials have been found, distinguished primarily by the experimental paradigms that elicit them. The RP is recorded in self-paced motor tasks. Paradigms in which a warning stimulus (S1) is presented, followed by an imperative stimulus requiring a motor response (S2) after a fixed interval, are used to elicit the contingent negative variation (CNV). Like the RP, the CNV has early and late components. The early component seems to be related to S1 and the late component involves the preparation process in anticipation of S2. Another slow cortical potential, the stimulus-preceding negativity (SPN), is seen in paradigms that involve no movement. Instead, subjects anticipate a stimulus containing important information, such as feedback about earlier performance. The similarity between the three signals suggests that similar neural processes may be at work during anticipation in the absence of movement.

The literature demonstrates that the relationship between these potentials is contested. Some have posited that the RP is identical to the late component of the CNV [104,105], while prior work maintained that the RP is more lateralized than the late CNV [106]. It has been suggested that the RP is identical to the CNV and related to anticipation [107]. It also remains possible that the RP has a different neural mechanism from the CNV [108,109] or that they share the same cortical generator, yet differ in subcortical sources [109].

Meanwhile, others have hypothesized that the CNV is a combination of both motor and non-motor preparatory processes (i.e., the RP and SPN, respectively) [110]. This was challenged by the finding that the summation of SPN and RP resulted in a different scalp distribution than the CNV [111]. More recently, it was observed that the amplitudes of the waveform obtained by superimposing the SPN on the RP was similar to that of the CNV at central and parietal sites but was smaller at frontal sites [112]. They suggest that the CNV and SPN may both be related to attention, sharing underlying neural mechanisms in the parietal area but differing in frontal regions.

In sum, the relationship between the RP and other slow cortical waves remains murky. The commonalities among them make it difficult to determine which aspects of the RP, other than the **lateralized readiness potential (LRP)**, are specifically linked to motor preparation.

respect to those occurrences before averaging. This time-locking will tend to emphasize what is common to that type of event by reducing uncorrelated signal (noise).

Veto power: ability to change one's mind after a decision to act and withhold action.

W-time: the time, measured in Libet-style studies from self-report, at which the agent decides or wills.

'decision to move' refers to a hypothetical neural event that leads to movement and that may or may not be accompanied by a conscious mental event). However, as the RP signal is an order of magnitude weaker than the noise, averaging many (>30) trials has been necessary to reveal the RP and examine its properties. We therefore do not know if the (averaged) RP is just a de-noised version of what is present on each trial, or if it is an artifact of trial averaging. Other waveforms, suitably distributed, can in theory result in a trace identical to the RP when averaged together [31]. Ultimately, the question remains whether or not the RP exists as such in single trials. If it does, then we should, at least in theory, be able to measure it at the single-trial level (e.g., using an appropriate filter) [32]. However, doing so would require a correct hypothesis about what an individual RP looked like and a good signal-to-noise ratio, which is unlikely to be obtained using EEG.

Moreover, false positives severely limit our ability to determine the causal relationship between individual RPs and movement. Suppose one finds a signal that looks like an RP but is not followed by movement. In that case, it is impossible to determine whether or not that particular signal is just a random deflection in the time series that resembles one's hypothesized template (a false positive), or a real RP that fails to initiate movement (and thus evidence against the interpretation that RPs are causal precursors of movement).

Nevertheless, RP detection in single trials has been attempted and there are a handful of published accounts. One study [33] measured RPs in single trials with multielectrode fusion methods. However, signals detected on individual trials had a variable temporal relationship to movement. In addition, purported RPs in individual trials were statistically indistinguishable from normal fluctuations in resting-state brain activity. Prior efforts to detect individual RPs in real time based on EEG managed to predict movement onset only modestly better than chance [34–36]. One might suspect that single-unit recordings in humans would settle the score [37], but, although the reported averages are compelling, the pre-average raster plots show that the

buildup observed in the average is not readily apparent at the single-trial level. The upshot is that we are certain neither about the causal link between occurrences of the RP and motor initiation, nor about the profile of neural activity during single trials, which, in the aggregate, produces the reliable signal we call the RP.

The RP's Connection to Volition and Awareness

Although most experiments that measure the RP involve subjects intentionally willing their movements, few have explicitly tested the relationship between the RP and awareness of the intention to act. One study [38] explored whether the presence or absence of an RP-like signal correlates with an individual's feeling of intending when engaging in self-paced movements. Subjects' awareness of their intentions to act were probed at different times during a self-paced movement task using an EEG-based brain–computer interface (BCI). Subjects were more likely to report a conscious intention when the probe (a sensory cue) was triggered by an RP-like signal, compared with when the probe was triggered by the absence of an RP-like signal. In a similar vein, a different group [39] interrupted subjects at random times while they performed a self-paced motor task. Subjects were instructed to react to the interruption (by pressing a button) only if they happened to be preparing to move at the time. An RP-like signal was more prominent preceding interruptions when there was awareness of movement intention. However, other work [40] argues against there being a relationship: researchers found that when subjects were hypnotized to move their wrist without conscious intention, canonical (average) RPs were still evident, suggesting that RPs can occur before movement even in the absence of conscious willing.

Nonclassical Interpretations of the RP

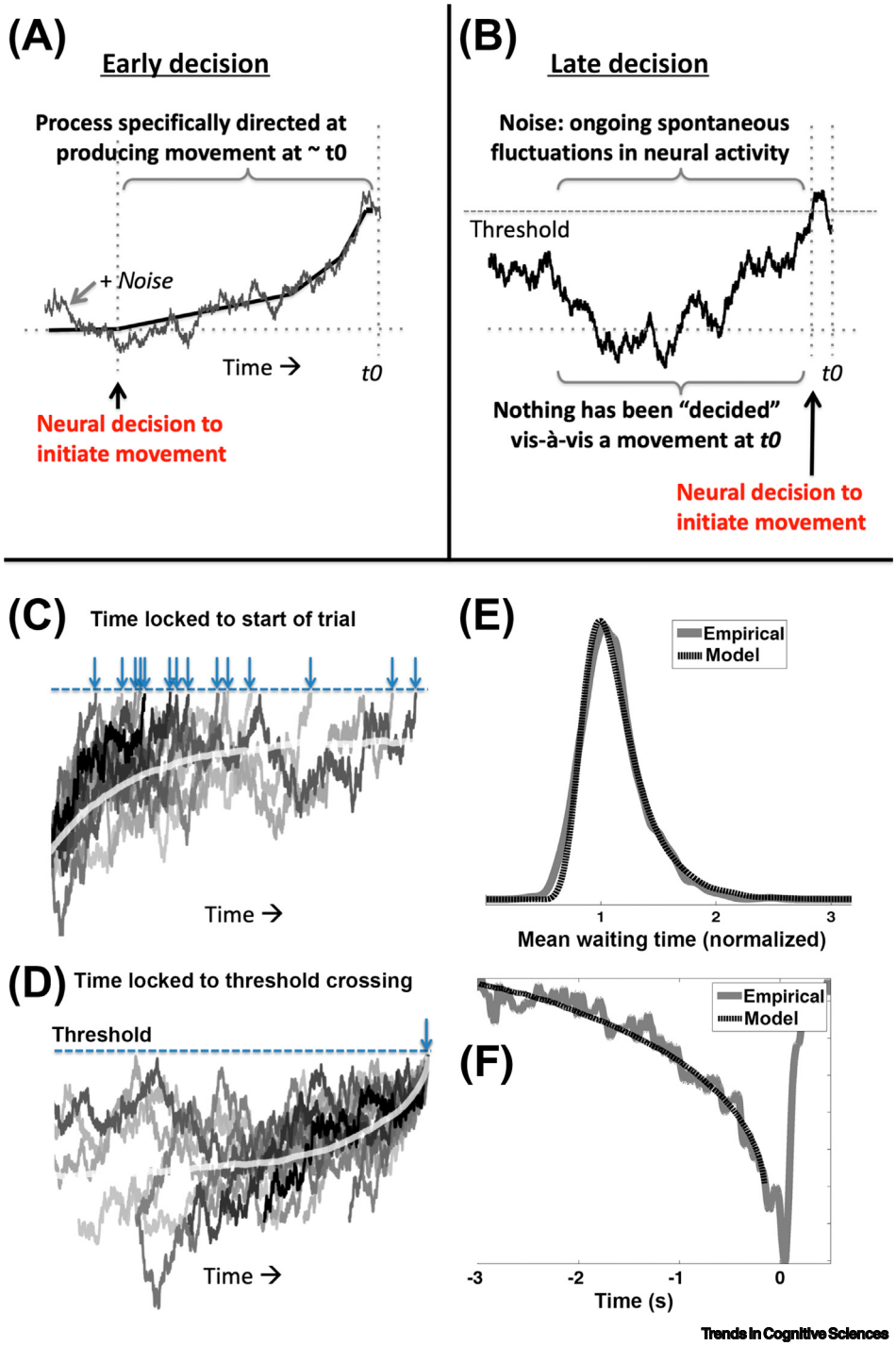
The RP is generated by sampling only epochs that culminate in movement. In Libet-like tasks we never observe what happens when movement is not triggered. This raises the possibility that the RP is due to biased sampling, an artifact of the analysis process. This insight underlies a family of models that offer an entirely different interpretation of the RP. This class of models, known as '**stochastic decision models**' **(SDMs)**, envision the RP as a necessary consequence of trial averaging coupled with at least a tendency for movement onset to coincide with negative deflections in electrical potential. The first of these, the accumulation-to-bound model (AtBM) [41–43] holds that the precise moment of movement onset is partly determined by ongoing stochastic fluctuations in neural activity, especially when the imperative to move is weak or absent (Figure 1, Key Figure and Box 4). These fluctuations appear in the movement-locked average as a slow buildup. According to SDMs, the RP reflects neural activity antecedent to the decision to move, or perhaps the process of arriving at a decision to move, rather than the outcome of a decision to move. Thus, unlike the classical interpretation, which is an **early-decision account**, the SDMs offer a **late-decision account** (Figure 1).

While late-decision accounts of the RP may be less intuitive than the goal-directed interpretation offered by the classical early-decision account, they are, arguably, more parsimonious. Why should there be such a long and highly variable lag, of up to one second or more, between the decision to initiate movement and movement onset? Why has the RP not proven to be a very reliable real-time predictor of movement onset? And why are subjective reports of the (conscious) decision time so late relative to the supposed 'onset' of the RP? These puzzling questions are not at all puzzling from a late-decision perspective, so the onus should be on proponents of the early-decision account to explain why the more parsimonious late-decision view is false.

Recent physiological evidence embraces the decision-to-bound framework and corroborates the late-decision interpretation. Neurophysiological work in rats [44] found that ramping activity in the rat secondary motor cortex (M2) could be explained similarly. This ramping activity preceded

Key Figure

Early- versus Late-Decision Accounts of the Readiness Potential (RP)/ the Stochastic Accumulator Model



(See figure legend at the bottom of the next page.)

Box 4. The Accumulation-to-Bound Model

The AtBM posits that the RP reflects ongoing stochastic fluctuations in neural activity that play a role in fixing the precise time of movement onset when timing is relatively unconstrained [41]. When there are no external inputs to that decision, such as in the Libet task, the question 'do I move now, or later?' has to be resolved somehow. The ongoing temporally autocorrelated stochastic ebb and flow of brain activity could be one factor. If ongoing fluctuations can tip the scales in favor of moving now, then time-locking to movement onset becomes equivalent to time-locking to crests in the underlying autocorrelated neural time series. The time-locked average will then, at least in part, reflect the random buildup to those crests (see Figure 1 in main text). While this may not explain the peak of the RP, which probably originates in the primary motor cortex, it can explain the early phase of the RP. The AtBM is a 'late decision' account in that the threshold crossing event that leads to movement is proximal to the movement and late in the evolution of the classic RP. The RP onset (deviation from baseline) on this model is physiologically meaningless: it does not mark a causally efficacious neural event or process.

The underlying process is modeled using a leaky stochastic accumulator, a kind of AtBM (or accumulator to bound model). Such models have been used for decades to model various kinds of cued decision-making tasks [113–116] and have also been used to account for decision-related neural activity, notably in posterior parietal cortex, in perceptual decision-making tasks [117–119]. The accumulator process is given by

$$dx = (I - kx)dt + c\xi\sqrt{dt} \quad [\text{I}]$$

and has three free parameters: an imperative signal I, a leak current k, and a threshold on x (which is called the decision variable). On average, the decision variable will climb to a mean value of I/k and then fluctuate randomly around that mean value, eventually reaching an appropriately chosen threshold. The average trajectory of the decision variable as it approaches the threshold can be fit to electrophysiological data, such as the RP, and the distribution of first threshold-crossing times can be fit to the empirical distribution of waiting times in a self-initiated movement task [41]. More recently it has also been shown that, under certain assumptions, the average input to the accumulator also has a ramping character that can fit the RP [43].

spontaneous decisions to abandon waiting for a large reward and instead opt for a small but certain reward. The authors were able to account for this ramping activity using a leaky **stochastic accumulator model**, the same model used by [41] to capture the RP profile in human EEG. Subsequent work found that the buildup's stochastic component was reflected by activity in area M2, but not medial prefrontal cortex (mPFC), which instead accounted for deterministic biases in action timing [45]. This suggests a two-stage model of the RP in which a signal that can lead to movement only does so when it coincides in time with a stochastic fluctuation that then pushes the system over the threshold for movement.

Consistent with the stochastic account, some neuroscientists [46] propose that the RP reflects a tendency for movement initiation to coincide with negative-going fluctuations in the EEG slow-cortical potential (*cf.* [47]). They call this idea the selective slow-cortical-potential (SCP) sampling hypothesis. Like the AtBM, the SCP sampling hypothesis rests on the premise that there are ongoing (presumably stochastic) slow fluctuations in cortical potential. Furthermore, the motor system is somewhat more excitable during the negative phases of these fluctuations, making spontaneous voluntary movement more likely at those times. Like all SDMs, this account raises the same challenge to the classic interpretation: the RP does not reflect a process of 'planning

Figure 1. (A) Early-decision accounts of the RP propose that the onset of the RP marks an inflection point in neural activity, the start of a process of planning and preparation for movement that culminates in a movement at $t0$. According to early-decision accounts, the neural decision to initiate movement is marked by the onset of the RP. (B) In late-decision accounts, the RP reflects the average time course of ongoing spontaneous fluctuations in neural firing rate, recruitment, or excitability when data are time-locked to crests in those fluctuations. The accumulation-to-bound model (AtBM; C–F) offers a late-decision account of the RP. The AtBM accounts for the RP using a leaky stochastic accumulator. The distribution of first crossing times (blue arrows in C) can be used to account for the distribution of waiting times in Libet's [2] task (E). When the decision variable is time-locked to the threshold crossing (D), its average trajectory (sign reversed) as it approaches the threshold can be fit to the shape of the RP (F).

and preparation for movement' and cannot be used to argue against the existence of conscious volition.

Other variants of the stochastic account are possible. One posits that subjects periodically rehearse the movement they were instructed to make and that these subthreshold rehearsal events interact with ongoing fluctuations in the motor system. When the sum of the ongoing fluctuations and the motor-rehearsal events crosses the threshold, the movement is initiated. This sum will also produce a slow buildup in the average time-locked to movement onset. Another possibility is that motivational signals produce brief subthreshold motor impulses at random times, which are integrated downstream. A leaky integral over this input would reflect the temporal density of the impulses, which would lead to movement onset when it reaches a threshold. While these alternatives may have slightly different philosophical implications, they are notably similar to the late-decision accumulator model.

Since the introduction of the AtBM [41] a few studies have challenged the late-decision view, or bolstered the early-decision view. One [48] looked at across-trial variance in the RP as a function of time (see [31]) and found that variance decreased at a greater rate before self-initiated movements than cued movements. This could be accounted for by incorporating a reduction in noise over time, before self-initiated actions, into the same stochastic accumulator model used by Schurger *et al*. [41]. While this decrease in variance over time leading up to movement onset may seem to be a compelling sign that the motor system is preparing a movement, it is no more compelling than the profile of the RP itself, which had seemed like incontrovertible evidence of preparation until the SDM provided an alternative, arguably more compelling, interpretation. A change in some variable (whether the mean or variance) in advance of an event, however compelling it may seem, does not entail a process of intentional preparation for that event [42]. Future studies may clarify these issues.

According to the AtBM, the RP appears in the movement-locked average because the onset of self-initiated movement tends to coincide in time with crests in ongoing stochastic fluctuations in neuronal activity. If true, then deflections, perhaps resembling the RP, approaching but not crossing the movement threshold, should happen regularly even when the subject is not making any movements. In one study [49], the (averaged) RP was used as a filter to search for RP-like events in EEG data from subjects performing a self-initiated movement task. Because the noise in an EEG recording is an order of magnitude greater in amplitude than the signal and because that noise is temporally autocorrelated, any such filter applied to single-trial EEG data will uncover RP-like events. But these turn out to be unrelated to the weaker, hidden deflections that give rise to the RP in the movement-locked average. Therefore, further research is required, ideally with invasive recordings, to determine whether or not RP-like events happen all the time.

Whether a late- or early-decision account more aptly accounts for the data remains an open question, but, as mentioned previously, the late-decision position has parsimony on its side. Also, other evidence points to a decisive event within the last 200 ms before movement onset, consistent with the late-decision account. There is an abrupt increase in corticospinal excitability in the last 100 ms before muscle contraction (~150 ms before movement), but no changes in corticospinal excitability prior to that [50]. More recently, a study that used a clever paradigm involving a BCI [13] estimated the time of the **point-of-no-return** in self-initiated movement to be about 200 ms before movement onset (cf. [51]). Finally, Libet [2] and many replications since then have found that, when asked when they had (consciously) decided to move, subjects reliably and consistently point to about 150–200 ms before movement onset. According to the classic early-decision account of the RP, this must mean that the brain was already preparing

to move long before awareness of a decision to move. But according to the late-decision account, it is simply a sign that, when asked to report the time at which a decisive neural event took place, subjects are reasonably accurate in doing so.

Relevance of the RP for Discussions of Free Will

The notion that neuroscience could demonstrate the inefficacy of conscious will naturally captured much attention from philosophers and psychologists and it remains an active subject of debate [52]. The arguments for the inefficacy of conscious will rely upon the following three associated implicit commitments. First, that the RP itself is postdecisional: it occurs after the decision to move. Second, that the RP is ontologically real: it reflects an identifiable, causally efficacious process that begins at RP onset and culminates in movement. Third, since the signal reflects a real process, it is natural to assume that the onset of the RP marks a significant moment at which the process leading to movement begins.

SDMs provide a compelling alternative. Underlying SDMs is the idea that ordinary decision processes give rise to the decision to move. This seems almost tautological, but it is revolutionary for the interpretation of the RP. Standard accumulation-to-bound mechanisms, when time-locked to movement and averaged, and when the 'evidence' is weak relative to the noise, necessarily produce a trace that has the profile of the RP. The decision to move occurs when the threshold is reached prior to movement and the 'onset' of the RP does not correspond to a meaningful event: it is just an artifact of the averaging process. In any individual trial that leads to movement, it may not be possible to identify a point at which the process began since the excursion to threshold may not be monotonic. Furthermore, since the 'onset' of the RP does not correspond to a physiologically real event in individual trials, it is senseless to consider the relationship of the time of that (non)event to any other, such as W-time. In other words, the stochastic model accounts for all the measurable phenomena of the RP in a way that connects naturally with everything else we know about the neural basis of decision-making and strips away the conceptual basis for relating the RP to the timing of conscious awareness. Indeed, if the decision to move is marked by the time of **threshold crossing**, then awareness of conscious intention to move coincides with the decision point, as common sense would suggest.

The implications for this picture of volition for the free will debate are not entirely straightforward, as the details interact with substantive philosophical positions on free will. Stochastic models may yet pose problems for free will, but not because of the relative timing of awareness and volition. According to the AtBM, threshold crossings are partly the result of noise in the system that drives the decision variable over threshold. This suggests that noise is the source of an agent's decision to move when they did, as opposed to at some other moment. **Agent-causation** theorists, who hold that freely willed action requires that the agent be uncaused to act but yet be the cause of its action, will likely look dimly on noise as an agential font of action [53–55]. Other theorists who want to identify the source of action as the agent will have to tell a story that somehow makes a case for the noisy trigger being part of or attributable to the agent [55]. As the AtBM incorporates a shift close to the threshold as a background for the determinative influence of noise, this shift can arguably be agentive or volitional.

Incompatibilists who hold that free will requires that our actions be undetermined may be happy with the AtBM, if the noise in the signal is indeterministic [56,57]. Nothing in the model itself dictates whether or not that is the case and neuroscience is unlikely to definitively answer that question [58,59]. Compatibilists can also be satisfied by this model, since determinism tends not to be an obstacle for them [14,15,60–63]. However, some **compatibilists** may be bothered by the decisions being precipitated by noise and not, one might argue, fully determined by the

agent. These worries may be lessened in that many compatibilists will reject the standard Libet paradigm, in which an agent has no reason to choose to move at any particular moment, as a central case in which freedom of the will is at issue. Instead, they will be content to allow an agent to abdicate causal control to a subpersonal or nonpersonal mechanism in circumstances where no considerations bear on the outcome [14,15,64]. This contentment persists only if, in cases where one decides for reasons [65], it is the weight of the reasons and the agent's weighing of those reasons that are determinative causal factors. A strength of SDMs is that they provide a unifying story that seamlessly allows agents to move between reason-driven and random decisions, as the spontaneous action case is just an SDM driven by noise in the absence of evidence/reasons.

Other versions of the SDM in which excursions to threshold are driven by noise and signal, whether motivational impulses or motor ideation, are even more congenial interpretations in which the agent is the source of threshold crossings. These signals have interpretations that can be linked to motivation or other agentive processes, making it easier to see the agent as the source of individual motor acts.

Concluding Remarks

The RP continues to be both a methodological tool and an object of study, but there are significant areas about which we remain unsure, despite advances (see Outstanding Questions). If recent models of the RP are on the right track, we cannot infer from the existence of the phenomenon that it reflects an actual signal in the brain that, in individual trials, has the characteristics of the RP, or that has causal efficacy. Because of this, one cannot infer that we lack conscious free will based on the temporal profile of the RP. If these models are correct, they may have implications for our understanding of free will, but none that avoid significant and substantive philosophical commitments. But given all the other reasons that have been raised for rejecting the classical interpretation (e.g. [3,14,16,17]), even if SDMs are mistaken and the RP does reflect a real neural signal, albeit one difficult to detect on individual trials, the RP would still fail to support the classic inference for the inefficacy of conscious will.

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Declaration of Interests

No interests are declared.

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Outstanding Questions

Why is there so much variability between subjects in the temporal profile and amplitude of the RP?

Why do some experimental subjects not exhibit a readiness potential at all?

What is the difference/connection between the RP, CNV, and SPN? Do they all share a similar set of neural processes? Is the difference between them a reflection of the differential recruitment of these processes due to slightly different task demands?

Does the readiness potential reflect initiation of action, or something more general such as anticipation or attention?

Which is correct: late- or early-decision models (or neither)?

What is the relationship between the RP and consciousness? Are the neural processes involved in generating the RP the same processes that lead to consciousness of intention?

Is there a discrete neural RP-like signal present on every trial, but just hidden in the noise? Or does it only exist in the average?

If there is a discrete neural signal that reflects the initiation of movement present on every trial or for every movement, what is its shape and its temporal relationship to movement onset?

If movement generated as a response to stimuli or to reasons is the result of an accumulation-to-bound process operating on evidence, then is self-generated movement just the same decision process, but in this case operating without external inputs in a stochastic milieu?

What brain activity is genuinely predictive of the onset of uncued movement?

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