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VOLITION

Barking up the wrong free: readiness potentials reflect processes independent of conscious will

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Abstract In the early 1980s, Libet found that a readiness potential (RP) over central scalp locations begins on average several hundred milliseconds before the reported time of awareness of willing to move (W). Haggard and Eimer Exp Brain Res 126(1):128–133, (1999) later found no correlation between the timing of the RP and W, suggesting that the RP does not reflect processes causal of W. However, they did find a positive correlation between the onset of the lateralized readiness potential (LRP) and W, suggesting that the LRP might reflect processes causal of W. Here, we report a failure to replicate Haggard and Eimer's LRP finding with a larger group of participants and several variations of their analytical method. Although we did find a between-subject correlation in just one of 12 related analyses of the LRP, we crucially found no within-subject covariation between LRP onset and W. These results suggest that the RP and LRP reflect processes independent of will and consciousness. This conclusion has significant implications for our understanding of the neural basis of motor action and potentially for arguments about free will and the causal role of consciousness.

Keywords Readiness potential · Libet · Volition · Consciousness · Free will

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Introduction

Although our conscious will appears to cause our actions, Benjamin Libet's experiments raised doubts about this apparent causal connection (Libet et al. 1983). Libet and colleagues had subjects make simple motor movements, such as lifting a finger, while they measured a particular EEG marker of brain activity time-locked to the motor act. This marker—a negative potential over central scalp locations that develops in the seconds before participants make self-initiated movements—has been dubbed the "readiness potential" or RP (also known as a 'Bereitschaftspotential' or BP; cf. Kornhuber and Deecke 1965). Libet and colleagues estimated the earliest time of conscious awareness of a will to move by asking subjects to recall where a rotating spot on a clock face had been at the moment when they had first become aware of an urge to move. They found that the RP occurred on average 350 ms before the reported time of awareness (W). This finding has been replicated many times (e.g., Fried et al. 2011; Haggard and Eimer 1999; Lau et al. 2007; Sirigu et al. 2004). Neural activity reflected by the RP reliably preceded a voluntary action and also preceded the time at which participants reported becoming aware that they were about to act. Because a cause cannot come after its effect, Libet concluded that conscious will could not be what initiates the causal process that leads to action.

Libet's results raise a crucial question: Is the RP part of a causal chain that is sufficient to produce seemingly voluntary action without any essential intervening causal role for will or for consciousness? If so, the efficacy of will and consciousness along with moral responsibility are all in doubt. In the end, Libet did not deny free will but instead postulated a veto power that could stop the execution of unconsciously initiated actions. However, many scientists and

philosophers still use Libet-like results to argue against the existence of free will and responsibility (cf. some authors in Sinnot-Armstrong and Nadel 2010).

Despite three decades of theorizing about Libet's findings, the precise role of the RP has not been empirically established. It is still unclear whether the RP is a neural correlate of planning a motor act, anticipating a motor act, preparing to perform a motor act, consciously willing a motor act, a combination of these, or even none of the above (cf. Schurger et al. 2012).

To our knowledge, Haggard and Eimer (1999) conducted the only study that has claimed to have found evidence supporting a causal relationship between readiness potentials and conscious will. Their argument was based on Mill's (1843) observation that a necessary condition of causal relations is covariation of causes and effects. Haggard and Eimer found that RP latency did not covary with W, which undermines a causal relationship between the two. However, they did report a correlation between W and the onset of the lateralized readiness potential (LRP). The LRP is a difference in scalp potentials between the two hemispheres that develops over the motor cortex shortly before participants make a lateralized movement, such as a left or right hand button press. This correlation leaves open the possibility that the LRP reflects processes causally associated with W.

Unfortunately, Haggard and Eimer's (1999) results used data from only 8 subjects. Their table of early versus late awareness LRP onsets indicates that the effect was driven largely by the data from participants 3 and 6. If either of these two participants had been removed from their analysis, the effect would no longer have reached significance. We therefore attempted to replicate their original finding with a larger sample in order to clarify the nature of the RP and LRP and their relation to awareness, will, and action.

Methods

We followed Haggard and Eimer's methods precisely, which used the traditional Libet paradigm as follows. Participants sat 50 cm from a computer monitor on which we displayed an analog clock with a 1.3-cm-long hand that rotated clockwise once every 2,560 ms. Clock positions were labeled with the numbers 1 through 12. Participants performed the same number and type of trials as in Haggard and Eimer: 40 trials each of free (i.e., response hand chosen by the participant) or fixed (response hand chosen by the experimenter) movements, judgments of W or M (the time at which they actually moved), and left or right button presses, totaling 320 trials for each approximately hour-long session. Participants were told to move spontaneously, that is, without preplanning when to move in any given trial or following a pattern of responses across trials. Exactly as in Haggard and Eimer, participants were told to report "when you first began to prepare your movement" for W trials and "when you pressed the key" for M trials. Participants reported times in clock units which were converted to milliseconds.

Twenty-one participants (9 females, 20 right-handed) with mean age 28.9 years (SD 11.6 years) gave consent to participate. One participant only completed half of the experiment due to a computer program error, and one participant's data were discarded before further analysis because of excessive noise. EEG data were recorded at 2,048 Hz using BioSemi Ag/AgCl high impedance active electrodes located at 32 scalp locations according to the 10–20 system (Jasper 1958), on the left and right mastoids, and on the back of each index finger. Only eight of these electrodes—at Fpz, Fz, Cz, Pz, C3, C4, and the two mastoids—were used in the analyses. Scalp data were referenced offline to the average mastoid signal, bandpass filtered from 0.016 to 70 Hz, and epoched from 2,600 ms before to 400 ms after each key press. Each epoch was baseline shifted using the average signal from −2,600 ms to −2,500 ms. Epochs deviating more than 80 μV from baseline at Fpz or Pz were rejected to eliminate eye artifacts. On average, 18.8 % (SD 22.2 %) of trials were rejected for each participant due to either participant error or eye artifact. The RP was measured at Cz and the LRP was measured at C3 and C4 using the double subtraction method (Eimer 1998).

Results

Our results replicated those of Haggard and Eimer for fixed versus free and M trials: manipulating whether movements were fixed or free did not affect the RP, LRP, M, or W. Although Haggard and Eimer's paradigm did not allow for an exact measurement of RP onset, we also clearly replicated Libet's original finding: mean W was −196 ms (SE 31.9 ms) relative to the key press, mean RP onset occurred at or before the start of our epoch at −2,600 ms, and mean LRP onset as measured by Haggard and Eimer (see below) was −779 ms. As our primary concern was to investigate whether LRP onset is correlated with W, we will only report results relating to early versus late W trials in order to address Haggard and Eimer's main finding.

We followed the method of Haggard and Eimer in using pre-movement RP amplitude as an indication of RP latency. Replicating their findings, we found no significant differences in RP amplitude from −1,000 to −500 ms between early and late W trials (see Fig. 1 for grand mean time courses). The mean RP amplitude during this time period was −6.649 μV (SE 1.197 μV) for early W trials and −6.677 (SE 1.426 μV) for late W trials [$t(19) = 0.03304$, $p = 0.9740$]. This result supports their conclusion that the RP does not covary with W.

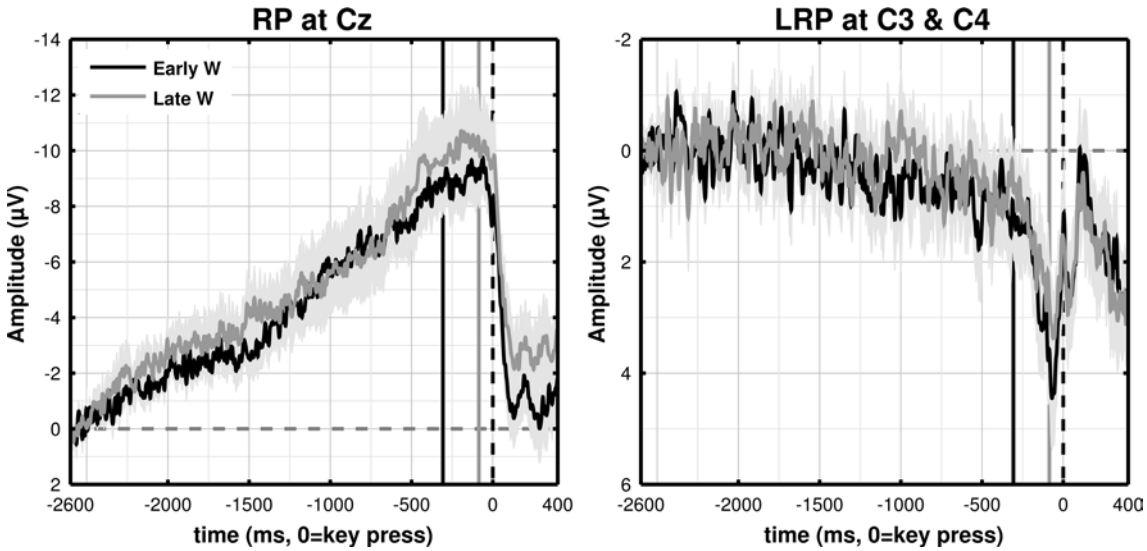

Fig. 1 No significant differences exist between early and late awareness RPs at Cz or LRPs at C3 and C4. *Solid black vertical line* is the mean early W time (−307 ms). *Solid gray vertical line* is the mean late W time (−85.5 ms). *Dotted black vertical line* is the time of button press

However, we did not reproduce their LRP finding. Haggard and Eimer calculated LRP onset by fitting each pre-movement time course to a function defined piecewise as a linear portion followed by a linear + quadratic portion. The "join point," which is the time point at which the linear transitioned to the linear + quadratic portion, was defined by them as the time of LRP onset. We used their method and found no significant differences between early and late W trials. LRP onset was −719 ms (SE 119 ms) for early W trials and −851 ms (SE 124 ms) for late W trials [$t(19) = 0.8574$ (one-tailed), $p = 0.7990$]. Note that these values trend in the opposite direction one would expect if the LRP reflects processes that cause W, with LRP onset occurring earlier for late W trials. We analyzed the data twice more using different definitions of LRP onset: once using 50 % peak latency and once with 25 % peak latency. In both cases, we found no significant difference between early and late W trials. Mean 50 % peak latency was −383 ms (SE 64 ms) for early W trials and −426 ms (SE 86 ms) for late W trials [$t(19) = 0.4086$ (one-tailed), $p = 0.6563$]. Mean 25 % peak latency was −695 ms (SE 108 ms) for early W trials and −647 ms (SE 130 ms) for late W trials [$t(19) = -0.3864$ (one-tailed), $p = 0.3517$].

Because the LRP is a relatively small signal relative to the level of noise in EEG signals, we considered the possibility that our single-subject means were too noisy to adequately resolve the LRP onset. We examined the signal-to-noise ratio (SNR) of the single-subject mean LRPs, with SNR defined as the LRP peak amplitude divided by the maximum amplitude of the signal over the baseline period. The mean SNR for early awareness, late awareness, and all trials pooled was 6.1 (SE 1.4), 4.0 (SE 0.72), and 9.7 (SE 2.5), respectively, suggesting that our data have adequate power. However, Miller, Patterson, and Ulrich (1998) suggest using a jackknife procedure for assessing LRP onset latency differences between conditions. In a jackknifed analysis with N subjects, N grand means of the data are calculated, each with one subject left out. The analysis is then performed on these grand means with corrections applied for the jackknife-induced decrease in variance. In the case of noisy estimates that occurs when calculating onsets from single-subject LRP time courses, this procedure can provide cleaner results while not biasing estimates of significance. We tried jackknifed versions of each of the three analyses above but still did not find significant differences in LRP timing between early and late W trials (see Fig. 2 for results of these within-subject analyses). While the results of the 25 and 50 % peak latency methods correlated significantly with each other ($r = 0.655$, $p = 0.002$ for non-jackknifed analyses; $r = 0.761$, $p < 0.001$ for jackknifed analyses), neither peak latency method correlated significantly with the LRP onset method of Haggard and Eimer (r ranging from 0.057 to 0.345, $p > 0.05$ in each case).

To test whether we could find any covariation at all between LRP onset and W, we performed between-subject correlation analyses comparing W with the above measures calculated on each participant's average time course (no longer split between early and late awareness trials). Of the six analyses we performed, only one was significant, namely the correlation between jackknifed W and LRP onset as defined by Haggard and Eimer ($r^2 = 0.412$, $p = 0.002$). Figure 3 shows scatter plots for these six between-subject correlation analyses.

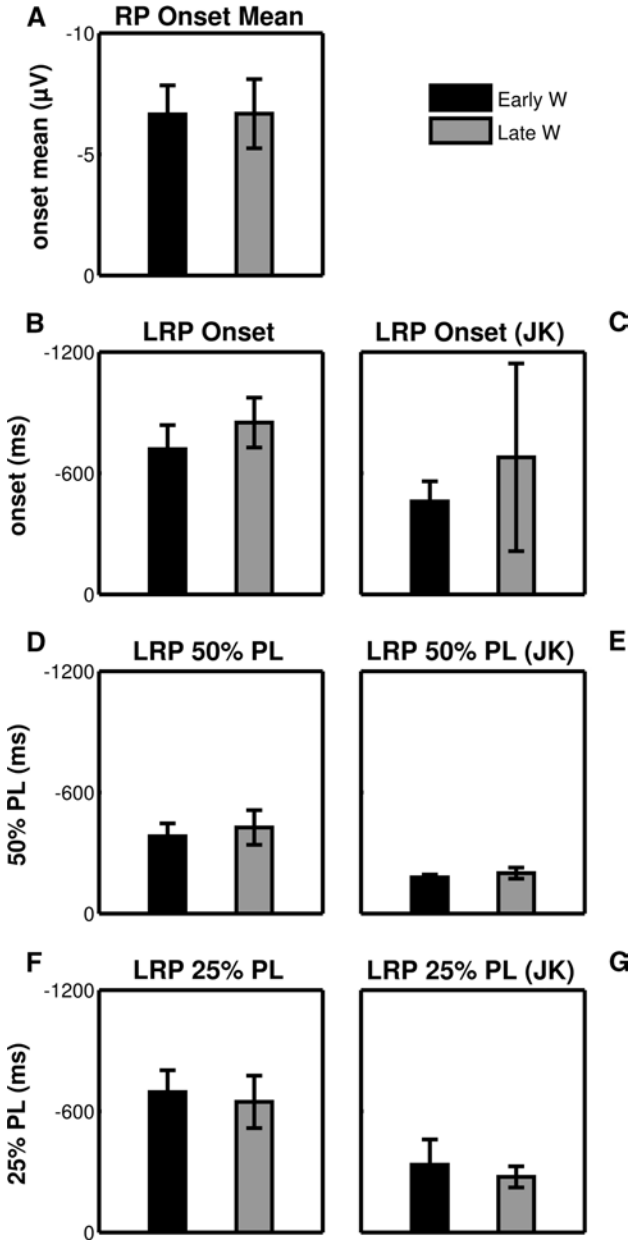

Fig. 2 Summary of within-subject *t* tests comparing early and late awareness trials. *Error bars* indicate standard errors of the mean. **c**, **e**, **g** are jackknifed versions of **b**, **d**, **f**. **a** Mean RP amplitude from −1,000 to −500 ms before key press. **b** LRP onset as measured in Haggard and Eimer (1999). **d** 50 % peak latency of LRP. **f** 25 % peak latency of LRP. No differences reach significance

In sum, we found no within-subject covariation between LRP onset and W and no significant between-subject covariation between LRP onset and W except in one test (to be discussed below). Table 1 summarizes the results from the 12 LRP analyses. Overall, our experiment suggests that the RP and LRP do not correlate with—and, hence, do not reflect the cause of—the conscious experience of will.

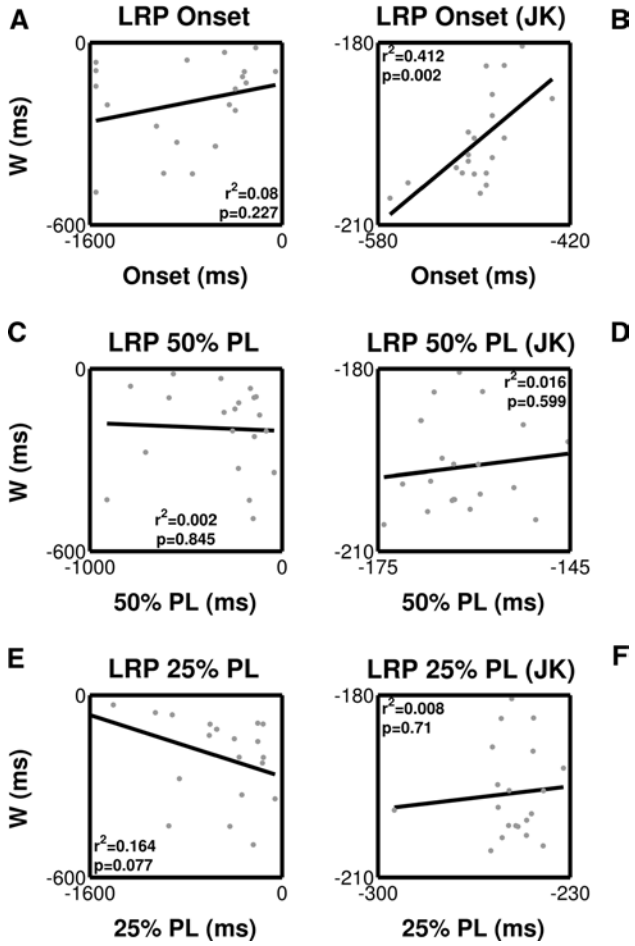

Fig. 3 Between-subject correlations of LRP onset and W. **b**, **d**, **f** are jackknifed versions of **a**, **c**, **e**. **a** LRP onset as measured in Haggard and Eimer (1999). **c** 50 % peak latency of LRP. **e** 25 % peak latency of LRP. Only the jackknifed LRP onset versus W is significant. 25 % peak latency of LRP shows a near-significant inverse correlation with W

Discussion

Several decades ago, Kornhuber and Deecke discovered an electrocortical potential that preceded volitional motor action. They called this response the Bereitschaftspotential and it later came to be known as the *readiness potential*. Subsequently, the seminal experiments of Libet suggested that the onset of this potential precedes awareness of the urge to move, a finding apparently at odds with lay views of free will. In the decades since, researchers have sought to answer exactly how the RP and the associated lateralized RP (LRP) relate to conscious will. In one of the most important contributions to this literature, Haggard and Eimer reported that the LRP is temporally yoked to the feeling of will, suggesting that the LRP might index processes that cause W. We sought to replicate this finding and failed.

Table 1 Summary of analyses

T test	Early	Late	*t*	*p*
RP onset mean	−6.65 μV	−6.68 μV	0.0330	0.974
LRP onset	−719 ms	−851 ms	0.857	0.799
LRP onset (jackknife)	−460	−679	0.448	0.670
LRP 50 % peak latency	−383	−426	0.409	0.656
LRP 50 % peak latency (jackknife)	−179	−200	0.740	0.766
LRP 25 % peak latency	−695	−647	−0.386	0.352
LRP 25 % peak latency (jackknife)	−335	−275	−0.408	0.344
Correlation		*r*	r^2	*p*
LRP onset versus W	–	0.283	0.0801	0.227
LRP onset versus W (jackknife)	–	0.642	0.412	0.00230
LRP 50 % peak latency versus W	–	−0.0468	0.00220	0.845
LRP 50 % peak latency versus W (jackknife)	–	0.125	0.0157	0.599
LRP 25 % peak latency versus W	–	−0.404	0.164	0.0770
LRP 25 % peak latency versus W (jackknife)	–	0.0888	0.00789	0.710

Does the LRP index processes that cause the awareness of an intention to move? Causation requires two facts to be true: (1) the cause precedes the effect and (2) the timing of the cause is correlated with the timing of the effect. If either of these does not hold, then there is no causal relation. Thus, if the LRP does not precede awareness related to an intention to move, or if the timing of LRP onset is not correlated with the timing of awareness, then the LRP does not index processes that cause the awareness of an intention to move. We consider these two issues in order.

(1) *Does LRP onset precede awareness?* We replicated the original Libet finding that the onset of the RP precedes reports of timing of awareness of wanting to move (Libet's "W"). We also replicated Haggard and Eimer's (1999) finding that the onset of the LRP, while later than the RP, also precedes W. However, an intention to move may unfold over time and arise at multiple levels of processing. It is possible that the awareness of wanting to move, reported as having occurred at W, is a final stage of a process that begins much earlier, perhaps even before the beginning of the LRP. In an attempt to address this, Matsuhashi and Hallett (2008) adopted a probe paradigm to assess stages at an earlier potential level of relevant processing. In their study, Matsuhashi and Hallett reported that "the time when subjects have access to movement genesis as intention" could be accessed via an external probe more than one second before W. They dubbed this time "T" and found that it occurred on average 1,420 ms before movement. This is well before the LRP onsets we measured, which occurred around 500–850 ms before movement. Thus, although "T" is not the same as "W", it is possible that subjects could be directed (via a probe) to the genesis of their motor intention before the onset of the LRP.

(2) *Does LRP onset covary with awareness?* Our main question was whether we could replicate Haggard and Eimer's principal finding that tied LRP onset to W. This replication would more firmly establish that the motor system not only causes motor actions but also causes the feeling of will for those actions. Our experiment failed to replicate Haggard and Eimer's principal finding of a within-subject correlation between LRP onset and W. In total, we conducted six within-subject tests of this correlation, and all six failed. We also conducted six between-subject analyses on data from a single experiment and found a significant correlation in one of them. Although the significance of one of the six tests does not provide conclusive evidence for an effect, it is worth considering how between-subject effects could arise in the absence of within-subject effects. A simple and likely possibility is that some participants move more slowly than others. LRP onset and W times are measured relative to the detection of a key press. Therefore, a slower movement, that is, a movement in which a greater amount of time elapses in between the onset of muscle contraction and the completion of the finger press, would lead to an earlier LRP onset and earlier awareness of movement intention relative to the completion of the finger press (the time at which the key press is detected). The within-subject analysis therefore provides a more appropriate test of a temporal correlation between the LRP and W, and no such correlation was found. Thus, the reasoning that Haggard and Eimer used to argue against any causal relation between the RP and W leads us to conclude that we also find no causal relation between the LRP and W.

It is still possible that W is not a good measure of awareness. For instance, if the W judgment were particularly noisy or arbitrary (i.e., if participants do not have conscious access to the time of awareness), then we may also expect to find no relationship between the RP or LRP and awareness as measured by W.

The conclusion that the LRP does not reflect processes causal of W is consistent with research demonstrating that the feeling of will can be influenced by multiple cues beyond action-intrinsic ones such as efference-copy (Synofzik et al. 2009; Wheatley and Looser 2010). Many empirical studies have demonstrated that cues over which one has no control and that cannot influence action can nonetheless influence the feeling of will for that action (Aarts, and van den Bos 2011; Desantis et al. 2011; Dijksterhuis and Aarts 2010; Moore et al. 2009a, b). In one example, participants reported intention for an action that someone else performed when led to believe that they alone had a consistent (action-related) thought moments before the act occurred (Wegner and Wheatley 1999). Banks and Isham (2009) showed that the timing of W could be influenced by introducing a deceptive tone that purportedly occurred along with a participant's button press but that actually occurred anywhere from 5 to 60 ms afterward. Lau et al. (2007) manipulated the experienced onset of intention using transcranial magnetic stimulation. Most recently, Aarts and Van den Bos (2011) demonstrated that the feeling of self-agency for a fixed action can be manipulated by the presence or absence of subliminal primes. In short, several studies have manipulated the feeling of will independently of movement, adding behavioral support for the finding here that the neural antecedents of movement are at least partially independent of the neural antecedents of the feeling of will.

If not conscious will, what do the RP and LRP index? Existing research suggests first that slow negative potentials such as the RP reflect something more general than the neural activity leading to a movement. Herrmann et al. (2008) found that RP onset occurred before participants could have begun to prepare a specific motor plan and concluded that the RP could not then specifically determine behavior. Trevena and Miller (2010) made a similar argument by showing that RP-like potentials preceding decisions to move were indistinguishable from those preceding decisions not to move (but see Gomes 2010, Castro et al. 2005). Baker et al. (2012) argued that a key component of the RP is the anticipation of an upcoming movement. Similarly, Donchin et al. (1972) found that slow negative potentials such as the contingent negative variation (CNV) and the stimulus-preceding negativity (SPN) can occur in conditions in which no motor task or even no task at all are involved [van Boxtel et al. (1993), van Boxtel and Brunia (1994) argue that the CNV is a combination of the RP and SPN (but see Ikeda et al. 1994, Ikeda et al. 1997)]. Although there may be some components of the RP and CNV that are related solely to stimulus anticipation, others may be related to anticipation of, or preparation for, movement (e.g., Schröter and Leuthold 2008; Van Boxtel and Brunia 1994). Future research should explore separate components of these waveforms.

In contrast to the RP, the LRP appears to be more closely tied to the production of movement. Researchers have shown that the LRP is generated in primary motor cortex, is not detected in the absence of movement, and does not require anticipation or preparation for action (cf. Eimer 1998; Haggard 2008). Our data suggest that the LRP, which may indeed signal the initiation of movement, is not temporally yoked to W.

If the RP and CNV correlate with anticipation rather than motor planning or execution, this would be problematic for Libet's argument that the RP indexes cerebral initiation of a voluntary movement since the RP would reflect processes that are not causal of the movement itself. Note, however, that the LRP is associated with movement and precedes W; this is consistent with a more general view of Libet's claim that movement-related neural activity precedes W.

It is important to note that the present study only tested awareness as defined by the original Libet and Haggard and Eimer studies. How the feeling of conscious will should be reported and defined is a matter of some debate (Sinnott-Armstrong and Nadel 2010). Here, we replicated Haggard and Eimer's (1999) instructions to participants to report "when you first begin to prepare your movement." However, what is meant by "beginning to prepare" is left to the participant to decide; a decision that necessarily introduces subjectivity and inter-participant variability. Within-subjects statistical tests help account for the inter-participant variability but the inherent subjectivity in defining W remains. Subjectivity-related timing concerns are particularly difficult to avoid under the assumption that W (however defined) is not a directly accessible event but an inferential process (Dijksterhuis and Aarts 2010; Wegner 2002).

Conclusion

Our data do not support the existence of a causal relationship between either the RP or LRP and W, even though both the RP and LRP precede W, as shown by Libet et al. (1983). Neither the RP nor LRP appears to be a correlate of consciousness or conscious volition. The RP may instead be a correlate of general anticipation or preparation or merely of ongoing activity that is neither anticipatory nor preparatory, and the LRP may be a correlate of the activation of a motor strip command to move particular muscles. However, more work is needed to further disentangle the processes of preparation, anticipation, awareness, and volition and to determine how each may relate to the RP and LRP.

The classic results of Libet and Haggard and Eimer, as well as many recent discussions of free will and the causal roles of will and of consciousness, need to be rethought in terms of these findings.

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