

## The Timing of Mental Events: Libet's Experimental Findings and Their Implications

Benjamin Libet

*University of California at Davis, Davis, California 95616*

The major findings by Libet et al. are briefly summarized. The criticisms and alternative proposals by Trevena and Miller, Pockett, and Gomes (this issue) are analyzed and found to be largely unwarranted. © 2002 Elsevier Science (USA)

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Libet and his colleagues made several fundamental and important experimental discoveries relating to timing factors in achieving a conscious sensory experience and in the cerebral production of a freely voluntary act. They are summarized briefly below.

1. Cerebral cortical activities, in response to a somatosensory stimulus, must proceed for about 500 ms in order to elicit the conscious sensation (Libet et al., 1964, 1967, 1991, 1992a).

2. Activations of shorter durations at the same intensities can produce *unconscious detection* of that input. Increasing the duration of repetitive ascending inputs to the sensory cortex by an *additional* 400 ms converts an unconscious correct detection to a conscious sensory experience (Libet et al., 1991). This is the basis of Libet's "time-on" theory for the transition between unconscious and conscious mental functions.

3. Despite the delay for cortical achievement of *awareness*, for a sensory input, Libet proposed the hypothesis that the *subjective* timing of the stimulus is referred backward in time to coincide with the initial primary evoked response of the sensory cortex to the stimulus. This response appears with a latency of up to about 30 ms depending on the bodily location of the stimulus. This subjective "antedating" results in our *experiencing* a stimulus with no delay after its delivery. A direct experimental test of such referral in time confirmed the hypothesis (Libet et al., 1979).

4. A freely voluntary act was found to be preceded, by about 550 ms, by the readiness potential (a slow surface negative electrical charge that is maximal at the vertex). But subjects reported becoming first aware of the wish or intention to act only about 200 ms (SE  $\pm$  20 ms) before the act (Libet et al., 1983; Libet, 1985). This meant that the brain was *initiating* the volitional process unconsciously, at least 350 ms before the person was aware of wanting to act.

Address correspondence and reprint requests to B. Libet, Center for Neuroscience, University of California at Davis, Davis, CA 95616.

However, Libet noted that the conscious function still had enough time to affect the outcome of the process; that is, it could allow the volitional initiative to go to completion, it could provide a necessary trigger for the completion, or it could block or veto the process and prevent the act's appearance. There is no doubt that a veto function can occur. The argument has been made that the conscious veto process would itself require preceding developmental processes, just like a conscious sensory awareness. But Libet (1999) argued that the conscious veto in a control function, different from awareness per se, need not be a direct product of the preceding processes, as is the case for simple awareness.

The implications of these findings for the concept of free will were considered fully by Libet (1999).

### *Impact and Acceptance of These Findings*

It should be of interest to the reader that many of the world's leading neuroscientists have not only accepted our findings and interpretations, but have even enthusiastically praised these achievements and their experimental ingenuity. These included the editors and referees of the journals that published our articles (including the *Journal of Neurophysiology*, *Science*, *Electroencephalography and Clinical Neurophysiology*, *Brain*, etc.). Individual expressions came from E. D. Adrian, J. C. Eccles, Ragnar Granit, R. W. Gerard, Frederic Bremer, Charles Phillips, Laurence Weiskrantz, Wilder Penfield, David Ingvar, Herbert Jasper, Robert Doty, Robert Schmidt, Anders Lundberg, Per Andersen, Howard Shevrin, Hans Kornhuber, Ainsley Iggo, Petra Stoerig, Pierre Buser, Stuart Hameroff, and many others.

It is interesting that most of the negative criticism of our findings and their implications have come from philosophers and others with no significant experience in experimental neuroscience of the brain. Some notable positive exceptions have been Sir Karl Popper (the leading philosopher of science in the 20th century), Stephen Pepper (late Professor of Philosophy at UC Berkeley), Martin Edman (Professor of Philosophy at the University of Umeå, Sweden), and others.

### TREVENA AND MILLER'S ARTICLE

Trevena and Miller (2002) have made a valiant experimental attempt to reopen the issue of the timing of the cerebral volitional process relative to the timing of subjective report of intention to act.

Trevena and Miller did not replicate the experiment of Libet et al. (1983), contrary to their claim of doing so. Their subjects were asked to pay attention to which hand to move. That added a complication to the timings at issue here. In the Libet et al. study subjects had the uncomplicated task of moving the same hand in all trials.

Trevena and Miller's subjects were asked to report the time of the *decision* to move now. In the Libet study subjects were asked to report the time of their first *awareness* of the wish (*W*) or urge to act. There could be a significant difference between these two kinds of reports. Indeed, their reported mean decision times were considerably different from those in the Libet study. In the study by Keller and Heckhausen (1990), which did replicate the Libet conditions, the reported timings were similar to those of Libet et al. (1983).

Trevena and Miller raise the possibility that our recorded readiness potential (RP), averaged over 40 trials, may include some individual RPs that started well before most of the others. That could make our starting time for the averaged RP a good deal earlier than is true for most of its components. In that case our finding that the RP began at least 350 ms before the reported *W*'s may not be valid.

First, Trevena and Miller have no actual evidence that this "smearing" of RP occurred. It is purely a speculative possibility. Their Fig. 1 is a "schematic demonstration" of this. It is not based on recorded RPs of individual trials. Second, they fail to note that Libet et al. did not merely eyeball the earliest time of the RP. We also measured the time at which 90% of the area under the averaged RP began, discarding the time of start for the earliest 10%. The "90% times" would be very unlikely to be representative of an initial leading "smear," but these 90% times produced similar values for the RPs to start roughly 350 ms before the *W* time. Third, the onsets of our RPIIs (for the fully spontaneous acts in all 40 trials) were usually fairly abrupt, with a fast rise. That would not be expected if there were a "smearing" of the leading edge of the averaged RP.

Finally, the conclusion by Trevena and Miller, that the "cortical preparation necessary for the movement to happen immediately may not start until *after* the conscious decision to move," is unwarranted on their own evidence (*italics mine*).

The conclusion was based on their finding that "many reported decision-times were before the onset of the Lateralized Readiness Potential" (LRP). They actually found that only 20% of the decision times were earlier than their LRP onsets. Their argument is that if one finds any decision times earlier than the LRP that would be sufficient evidence for their conclusion. They choose to dismiss the significance of the other 80% of their decision times that are *later* than the onset of the LRPs. What is even more appalling, they found that 40% of their decision times "were reported as occurring *after* the movement"! Trevena and Miller then go on to say that participants with such decision times (coming *after* the act) were probably "simply wrong about the time of their decisions."

A more obvious explanation for these peculiar results is simply that their subjects produced a wide spectrum of decision times, ranging from 20% that were earlier than the onset of the LRP to 40% that followed the motor act. There would be nothing magical about the early 20% of reported decision times. There is certainly no basis for drawing their conclusion that the conscious decision to act really comes before the brain's LRP process that prepares for the act. One cannot just ignore or argue away the 80% of reported decision times that follow the onset of the LRP. Ironically, their emphasis on the earliest 20% of decision times is opposite to their argument that the leading edge of onset of RP may be a nonsignificant "smearing" of individual events.

A more ingenious way of getting at this issue was reported by Haggard and Eimer (1999), a study ignored by Trevena and Miller. Haggard and Eimer divided their reported decision times into an early group and a later group. They compared the early decision-time values to the LRPs that were recorded in those trials. They found that the onset of the "early" LRPs appeared well *before* the "early" decision times. That provided a relatively straightforward result that contradicts the conclusion by Trevena and Miller.

## SUSAN POCKETT'S ARTICLE

Pockett (2002) has attempted an exhaustive reinterpretation of Libet's findings. She believes she can offer well-supported alternative interpretations that would contradict Libet's conclusions about substantial delays for producing a conscious experience. I appreciate Pockett's kind words about my pioneering role in the field of experimental research on the relation between cerebral neural processes and the production of subjective conscious experience or awareness. However, her analyses and reinterpretations do not take sufficient account of the full conditions of our experimental findings. Her revised conclusions are shown below to be unwarranted.

*"Facilitation"*

Pockett's reinterpretation of Libet's data rests heavily on her proposal that progressive facilitation occurs during a train of stimulus pulses. That could result in a delay to reach the threshold level of neuronal responses needed to produce a conscious sensory experience. She argues, then, that the delay for appearance of a conscious sensation would be a kind of artifact of stimulating the sensory cortex directly.

But there are at least two kinds of facilitation that can appear in the cerebral cortex (and indeed in other neural structures). There can be facilitation that builds up between one stimulus pulse and the next. This kind of facilitation is generally short-lasting and would disappear quickly after a train of pulses. Then there are longer lasting facilitations that can persist for minutes, hours, or even days. Sherrington had observed, in the early 1900s, that a longer lasting facilitation could be seen in the motor cortex of nonhuman primates. There is, of course, the more recently discovered long-term potentiation (LTP); in the brain this has been prominent in synaptic transmission involving glutamate. These longer lasting facilitations appear to involve additional metabolic steps, and so their onset and development are relatively slow. A long-lasting enhancement, discovered in sympathetic ganglia by Libet and Tosaka (1970), involves an action by dopamine on the muscarinic receptors for acetylcholine. Another excitatory response lasting 30 min or more was discovered by Nishi and Koketsu (1968). This effect is mediated by a peptide, LHRH (Jan, Jan, & Kuffler, 1979). It begins to appear only after about 1-s delay, and rises slowly over the next 5 or 10 min after a brief, <1-sec, train of preganglionic impulses (Ashe & Libet, 1981).

Clearly, then, the slow facilitatory effect, described by Libet et al. (1964) for brief stimulus trains of stimuli to somatosensory cortex, cannot be automatically invoked by Pockett as one appearing *during* such a train of pulses. We did indeed observe evidence *against* any facilitation developing between successive stimulus pulses. We recorded the direct cortical responses (DCRs) of the cortex to each stimulus pulse in a 0.5-s train at 20 pps; the intensity (peak current) of the pulses was at the liminal level for producing a conscious sensory experience (see Libet, 1973, p. 766; or Libet, 1982, p. 237). There was essentially no change in the magnitude or form of the successive DCRs in the whole 0.5-s train of 10 such responses.

The DCRs appear to represent postsynaptic responses of the dendrites of pyramidal cells that receive afferent inputs. Had there been any significant facilitation developing during the 0.5-s train, that should have been reflected in a buildup of the DCRs.

These DCRs were well below the maximal amplitudes possible, and so there was considerable available potentiality for any buildup to occur.

Additionally, with stimuli applied to the medial lemniscus (LM) pathway (to the somatosensory cortex), the proposal of facilitation has no merit at all. The facilitation factor proposed by Pockett cannot occur in the nerve fiber bundle of LM. And the reports of a slow cortical facilitation have been found only with direct stimulation of the cortex. There is no evidence for facilitation of the responses evoked in the sensory cortex by trains of ascending impulses initiated in LM. Pockett correctly noted that no value was given for the time intervals between successive trains of stimulus pulses in LM. I neglected to indicate that an interval of 15 s was used, just to be on the safe side against any late facilitation here. No changes in effectiveness were seen with successive trains. Also, we did routinely check the threshold level at the end of a series of trials to make sure no significant change had occurred.

An additional datum against facilitatory buildup to threshold level: A single stimulus pulse to LM at 40 times the intensity needed for a train at threshold level failed to produce *any* sensations. That strong pulse should have been more than enough to excite the neurons that Pockett proposed needed the facilitatory buildup.

#### *Other "Reinterpretations" by Pockett*

I select only her potentially significant points for this discussion.

Pockett proposes that a surface negative evoked potential (EP), with a peak at about 80 ms after a threshold skin pulse in one subject (Fig. 1A in Libet et al., 1967), should be taken to indicate the time delay for eliciting a conscious sensory response. She neglects to point out that this EP component appeared even with completely *subthreshold* skin pulses (no conscious sensation at all). In Fig. 1B there, for another subject, *two* early negative peaks appeared, one at about 65 ms and the second at about 113 ms. In Fig. 1 of Libet et al. (1975), the first significant negative EP, in the response of somatosensory cortex to pulses at the skin or the median nerve, had peak times of about 175 ms. (a) These early negative EPs have different delays in different subjects. (b) These EPs can also appear with subthreshold and no conscious sensations. (c) The later EP components that go on for at least 500 ms are more consistently correlated with the presence of a conscious sensory experience of the skin pulse. (d) When the late components are *selectively* suppressed by a general anesthetic or by atropine, the conscious responses to skin stimuli disappear or are depressed. There is no such evidence for the so-called 80-ms EP.

Pockett also dismisses the evidence of retroactive *enhancement* of the conscious experience of a skin pulse (Libet et al., 1992). This retroactive effect was produced by a train of pulses to somatosensory cortex that *began* up to about 400 ms or more after the skin pulse. This effect provided additional strong evidence for the view that there is a delay of up to about 500 ms for the appearance of awareness of the stimulus. During such a delay another input could modify the nature or the content of the sensation that is to appear. Pockett argues that the retroenhancement is due to "memory retouching." This is purely an ad hoc construction with no experimental evidence. The only evidence for retroactive effects on memory is for a disruption or abolition of a memory trace. And this kind of effect has been found only with strong

and spatially broader electrical stimulation, as in electroshock convulsive therapy. The near-threshold stimuli delivered via an electrode with a very small contact area has never been shown specifically to disrupt memory.

The “usefulness” of a delay in the appearance of sensory awareness is evident, in part, in the phenomenon of psychic modification of the content of a sensory experience. Freud and others in psychiatry and psychology have recognized that the conscious experience reported by a person may be quite altered from the actual image or even completely repressed. The delay of up to about 500 ms provides a physiological opportunity during which the individual can *unconsciously* detect the nature of the image and generate neural processes to alter the content of the conscious experience. (Pockett sarcastically remarks that the article on retroenhancement did not appear until 20 years after the experiments were done. But the reason for this delay was noted in our article. We wanted to wait for additional trials, which did not materialize.)

Pockett makes a number of errors in her detailed consideration of our experiments and many ad hoc assumptions to fill out some arguments. I refer to only a few examples of them.

1. Pockett suggests that the neurological abnormalities of our subjects may have affected our experiments. Our subjects, in fact, appeared to have a normal somatosensory system, even when compared to normal people.

2. Pockett believes that in later articles we “transmuted slightly” our findings with a single pulse applied in the central somatosensory system. Pockett seems unaware of the experimental basis for this “transmutation.” In the early experiments, with stimuli applied directly to somatosensory cortex, a strong single pulse could elicit a sensation. But that was commonly associated with the production of muscle twitch and could not be regarded as a sensation generated in the cortex. (Incidentally, Pockett’s suggestion that the muscle twitch was due to current spread to the motor cortex is not supportable. The motor cortex responds very differently to even a subthreshold train of pulses.) Our later conclusion that a single pulse stimulus is completely unable to elicit a conscious sensation was based on later tests with a coaxial electrode in the medial lemniscus (LM). A single pulse in this sensory pathway produced no muscle responses, even at 20 or 40 times the liminal intensity needed for an effective train of pulses (Libet et al., 1967, 1975, see p. 297). These very strong single pulses were also completely unable to elicit any sensation.

3. Pockett states, “The fact that Libet’s interpretation fails to predict the actual data” (that a skin pulse was reported to appear without the 0.5-s delay) “is not taken by him as evidence that his prediction was wrong.” She did not read our article as carefully and objectively as she claims to have done. In fact, we immediately stated that one option was that our delay hypothesis did not apply with a peripheral stimulus (Libet et al., 1979, p. 200). We then stated that an experimental test of the alternative option was required to indicate which was valid. The tests, matching of LM stimulus trains with a skin pulse, provided the crucial evidence.

4. Pockett attempts to explain our finding that subjects reported an awareness of actually moving *before* the movement on the basis of her “reinterpretations.” She is not aware of the work by McCloskey et al. (1983), who reported a similar result with a completely different technique.

Finally, Pockett finds it “difficult to understand (the) ease with which (Libet’s)

interpretations have been accepted by many in the consciousness research community.” This expression can, of course, be turned around: How is it that Pockett’s reinterpretations didn’t agree with the versions accepted by so many eminent experts, including the editorial reviewers for the journal *Brain*, who went over our articles at length and in fine detail!

### REBUTTAL BY GILBERTO GOMES

Gomes (1998) had written a “critical review and reinterpretation of Libet’s research.” After I carefully reviewed his article I wrote a reply to Gilberto Gomes (Libet, 2000). Both of these articles appeared in this same journal (*Consciousness and Cognition*). I recommend that the readers, if interested in this interchange, should go back and read Gomes’s 1998 paper and my lengthy reply to it (Libet, 2000).

Gomes (2002) now still does not accept my arguments against his criticisms. He simply repeats much of his original proposals. These were rife with ad hoc assumptions and hypotheses that lacked evidence and were mostly untestable experimentally. As an experimental neuroscientist I have only published hypotheses for which I carried out experimental tests or proposed an experimental test design, and I presented conclusions directly based on the experimental evidence. As a philosopher Gomes exhibits characteristics often found in philosophers. He seems to think one can offer reinterpretations by making unsupported assumptions, offering speculative data that do not exist and constructing hypotheses that are not even testable. If someone seriously believes he has a good hypothesis let him or her devise and carry out an experimental test of it. Experimental scientists do not generally engage in trying to design and carry out tests of hypotheses which they feel have no merit and/or appear to be untestable. I give only a few examples from Gomes’s present rebuttal to illustrate his style of thinking.

1. Gomes proposed that the latency for sensory awareness may be shorter with an LM stimulus than with one to sensory cortex. This purely ad hoc construction is not based on evidence. Gomes speculates that the primary cortical evoked potentials in response to LM stimulus pulses “might account for a shorter LM latency.” But primary evoked responses, even with very strong pulses in LM, were shown experimentally to be completely ineffective for eliciting any conscious sensory response. What basis could there then be for their shortening the latency of sensation?

2. Gomes tries to justify his assumption that the sensory latency for the skin stimuli, in our experimental pairing of skin and LM stimuli, was much longer for these skin stimuli than for LM. For technical reasons we used skin pulses that were slightly weaker than a threshold strength single skin. But we showed that repetition of these slightly subthreshold pulses quickly reached threshold for sensation within 30 ms or less. So one may be justified to increase sensory latency with this train of skin pulses by 30 ms. Gomes, however, proposes an increase of hundreds of milliseconds over that for a single skin pulse at threshold intensity (see Figs. 6 and 7 in Gomes, 1998). In order to justify his much longer latency for these skin stimulus trains, Gomes proposes an additional speculation: He says “that the *latency* itself may change with the stimulus intensity.” This nonsensical proposal is thrown in simply to make his overall hypothesis workable.

Gomes's comments on our experiments on brain processes in freely voluntary acts have already been addressed in my reply to the commentaries in Libet (1985) and in my discussion of Trevena and Miller (2002) in this issue of *Consciousness and Cognition*. Gomes's arguments that are based on the work of Keller and Heckhausen (1990) ignore my discussion of these issues in Libet (1992b).

Well, enough of this. I do not think any of this discussion will change Gomes's mind. I offer it only to acquaint the reader with some of the issues involved. Again, I would ask readers who are interested in a fuller treatment of the issues raised by Gomes to read my full reply to him in Libet (2000).

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