

## Problems in the Timing of Conscious Experience

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Libet's (2000) arguments in defense of his interpretation of his experimental results are insufficient. The claims of my critical review (Gomes, 1998) do not suffer with his new statements. © 2002 Elsevier Science (USA)

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Studies involving the stimulation of the cortex with electrical pulses at liminal intensity have shown that a rather long pulse train is needed to produce a conscious sensation (Libet et al., 1964). This led Libet to the hypothesis that a similar period of cortical stimulation (of about 500 ms) might be needed in the case of a single pulse applied to the skin. By coupling peripheral (P) and cortical (C) stimuli, Libet et al. (1979) devised a method intended to time the occurrence of conscious sensation. In order to preserve his original hypothesis, Libet was led by his results into a second hypothesis, that conscious sensation is referred backward in time to the moment of the primary cortical evoked potential. Coupling peripheral stimuli to stimuli applied on the medial lemniscus (LM), Libet and his colleagues obtained results that seemed to confirm this hypothesis (see Gomes, 1998, for a detailed discussion).

Coupling of peripheral and cortical stimuli (P-C coupling) led to simultaneous sensations when P roughly coincided with the end of the Minimum Train Duration of C. P-LM coupling, by contrast, led to simultaneous sensations when P roughly coincided with the onset of LM. This difference was explained by Libet as due to the absence of a primary evoked potential in the case of C.

### *Alternative Hypotheses*

The implausibility of Libet's two hypotheses (Gomes, 1998) led me to search for alternative explanations of the data. The first alternative interpretation I examined follows earlier proposals by Patricia Churchland (1981) and Ian Glynn (1990). It assumes that there is a real latency between the end of the cortical Minimum Train Duration (MTD) and consciousness of the sensation. Different results obtained in P-C and P-LM couplings would then lead us to assume a different duration of this post-MTD latency in each case (Gomes, 1998). Libet (2000, p. 2) dismisses this interpretation, stating: "that added, unknown latency should be the *same* following *any* input [to cortex, to medial lemniscus (LM) or to skin] that is just adequate for

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a conscious experience.” However, this is an unwarranted assumption. Stimulation to LM elicits repetitive evoked potentials in the cortex, which are absent in direct stimulation of the cortex. This might account for a shorter LM latency.

Libet states that none of my proposals “takes into account the experimental observations that the presence or absence of the primary evoked potential appears to be crucial to the subjective timing of the sensory experience” (Libet, 2000, p. 6). This is in striking contrast to the following quote from my article: “[LM-latency] may well have a much shorter duration. And the presence of the Evoked Potentials in the case of LM stimulation would possibly be the cause of the difference (or be related to it)” (Gomes, 1998, p. 578).

There is a second alternative explanation for the difference in results obtained with P-C and P-LM couplings (Gomes, 1998, pp. 582–583 and Fig. 7). This is based on the different intensity of the peripheral stimuli used by Libet in the two cases. This difference alone, leading to a longer latency for the peripheral stimulus in P-LM coupling, could account for the different results obtained by Libet in each case. There is simply no mention of this alternative—and more parsimonious—hypothesis in Libet’s reply (Libet, 2000).

The first alternative hypothesis assumes that LM latency is shorter than C latency. Discussing the figures in my article, Libet says I had to change “real” latency values (from Fig. 5 to Fig. 6). “The LM latency is portrayed as much shorter than was the C latency” (Libet, 2000, p. 5). But this is precisely the first alternative hypothesis (justification for which is given in my article), not an inconsistency in my interpretation. There was no “change” from Fig. 5 to Fig. 6, since they deal with different latencies.

Libet goes on: “The C latency is now reduced so as to match the LM latency.” Perhaps he is referring to my Fig. 7, where the second alternative hypothesis (which he does not discuss) is represented. “And, surprisingly, the latency for the peripheral skin stimulus is now *three times* as long as it was in Fig 5!” (Libet, 2000, p. 5). What he makes seem as an absurd inconsistency is in fact my second alternative hypothesis, based on the fact that the intensity of the peripheral stimulus used by Libet in P-LM coupling was much weaker than in P-C coupling. Weaker stimuli lead to longer latencies, as shown in perceptual masking experiments.

Libet also states that the skin stimulus (P) used in P-LM coupling reached its threshold after the first two or three pulses in a 60-pps train. “Consequently, only 33 ms or less might be added to any skin latency. But Gomes is adding about 400 ms to this putative delay!” (Libet, 2000, p. 5). Here, he is clearly confusing two different things. If there is a Minimum Train Duration for the peripheral stimulus, this should in fact be added to the “real” latency to make up the time interval from the onset of the stimulus to the onset of the conscious sensation. However, this was not my argument in hypothesizing a longer latency for the skin stimulus in the LM experiments. What we should also take into account is that *the latency* itself may change with the stimulus intensity. Backward masking experiments suggest that the latency for conscious sensation is longer when the stimulus is weaker. Thus, the point is not that there is a Minimum Train Duration to be added to the latency, but that the latency itself may be much longer as a consequence of stimulus intensity.

*The Latency for Conscious Sensation of Peripheral Stimuli*

Libet (2000, pp. 3–4) cites four lines of evidence as support of his inference that there is a delay of about 500 ms for conscious sensation of a peripheral stimulus:

(1) He notes that the presence of the late components of the evoked potential is correlated with conscious sensation of the stimulus. However, this tells us nothing about the precise moment at which conscious sensation starts.

(2) *Backward masking and backward enhancement.* Libet states that backward masking was obtained with intervals of 225–300 ms between skin pulse and end of an effective cortical train in most cases. However, as the cortical train lasted 100 ms, its disruptive effect on the production of a conscious sensation may have started at its beginning, even if conscious sensation is not finally prevented when the cortical train lasts less than 100 ms. Therefore, these data only suggest that conscious sensation had not yet been produced 125–200 ms after the peripheral stimulus. As for the rare cases of longer interstimulus intervals anecdotally reported by Libet, one must consider the possible effect of the brain pathology of the subjects and the very low (threshold) intensity of the peripheral stimulus that was used. That the latency for conscious sensation may be rather long in rare cases of slightly perceptible stimuli in subjects suffering from brain pathology does not support Libet's advocating of the existence, in general, of "a lag in conscious awareness of the real world by up to about 500 ms" (Libet, 2000, p. 4).

Experiments in which backward enhancement was observed (Libet et al., 1992) involved comparing two peripheral stimuli separated by a 5-s interval. The presence of a third sensation (referred to the same skin area but produced by a cortical stimulus) tended to favor judgment of the second as being stronger than the first (in fact, they had the same intensity). This happened with intervals between the second (peripheral) and the third (cortical) stimuli of up to 500 ms. However, subjects' responses depended not only on *consciousness* of the second stimulus but also on the *judgment* of the relative intensities of the first two stimuli. Therefore, all we can conclude is that this *judgment* was not completed 500 ms after the second stimulus, since it was influenced by the presence of the third. No inference on the latency for conscious sensation itself is warranted by these experiments. *Consciousness* of the second stimulus, as it first occurs, may have been unaffected by the presence of the third. It is when *judging* the intensity of the second stimulus, relative to the first, that its close temporal association with the third stimulus may have prejudiced its evaluation as being stronger than the much more distant first stimulus. Even if "subjects did not confuse the  $S_2$  sensation with the one produced by the cortical stimulus that followed  $S_2$ " (Libet, 2000, p. 4), the latter may have influenced judgment of the former relative to the first one. And if one doubts whether this influence on judgment is possible, without conscious sensation itself having been affected, one should be reminded that even in the absence of the third stimulus, subjects failed this apparently simple judgment task in 35% of trials; that is, they said that one of the two stimuli was stronger when they were in fact equal (Libet et al., 1992, Table 1).

(3) Libet mentions the finding by Jensen that reaction times (RT) jumped from 250 to 650 ms when subjects were asked to lengthen their RT by the smallest amount

possible. This only shows, however, that this difference must correspond to the time needed to refrain from reacting immediately and next to release the reaction.

(4) He stresses the fact that about 500 ms of stimulation of ventrobasal thalamus at liminal intensity are needed to produce a conscious sensation. Yet this tells us nothing about the latency for conscious sensation of a peripheral stimulus.

We see that all four lines of evidence mentioned by Libet are completely inconclusive in relation to his hypothesis that conscious sensation of skin stimulus is delayed by up to 400–500 ms.

### *Quality of the Evidence*

We must acknowledge the many difficulties involved in the experimental study of the temporal relation of stimuli in neurological patients. However, one should also recognize the technical limitations of the experiments that Libet and his group were able to conduct (Gomes, 1998, section 4.7). Libet counters this kind of criticism appealing to the authority of his coauthor in statistical matters and arguing that a proper study of a small number of cases may be conclusive (Libet, 2000, pp. 1–2).

The statistical treatment of his results may well be adequate to the available data. All the same, the quality of the evidence is poor. Only three subjects were tested (during surgical procedure) in the case of peripheral–cortical (P-C) coupling. One of these was not tested in the most crucial condition, with the peripheral stimulus (P) at the end of the Minimum Train Duration (when simultaneity of sensations should occur). Another subject was tested with a visual stimulus (which may have a different latency for conscious sensation) instead of a somatosensory one. In the case of coupling P with a stimulus to medial lemniscus (P-LM coupling), statistically treated results involved only two patients, who were tested in a different experimental condition (using chronically implanted electrodes) (Libet et al., 1979). (Note that the contrast between the results of P-C and P-LM couplings is crucial to Libet's argument.)

### *The Backward Referral Hypothesis*

As support for his backward referral hypothesis, Libet (2000, p. 6) insists on the difference in timing in P-C and P-LM couplings. However, in addition to the technical problems just mentioned, he offers no solid argument against the two previously mentioned alternative hypotheses. The fact is that these alternative explanations were not controlled for in his experiments.

Libet (2000, p. 6) also mentions the case of a patient with unilateral damage to the specific somatosensory projection, for whom there was “a delay of 200 to 400 ms in the subjective timing for a peripherally-induced sensation on the abnormal side, relative to one on the normal side” (Libet et al., 1979, p. 218). He says my proposals cannot explain these results. However, it is clear that they can easily do so. Damage to the somatosensory projection can be seen as prolonging the latency for the conscious sensation of peripheral stimuli on the affected side.

Libet also thinks that my proposals “do not explain the intuitive feeling of people that there is no delay in their subjective timing of sensory events” (p. 7). However,

if there is—as I think—a latency for *all* conscious experience, how could people possibly be aware of this delay?

### *Timing of Conscious Intention to Act*

A different set of experiments by Libet was intended to time the conscious intention to act (Libet et al., 1983). His subjects were instructed to perform a previously defined movement at a freely chosen moment. They were looking at a fast-rotating clock and were instructed to report either the moment of the decision (W) or the moment of the movement (M). At the same time, readiness potentials (RPs) were recorded. In control experiments, subjects were asked to time the sensation of a peripheral stimulus (S).

In my critical review (Gomes, 1998, pp. 589–591), I have considered three kinds of problems regarding this method. These are not adequately discussed in Libet's new article. One is that the validity of his method is dependent on his own hypothesis of backward referral of sensory experience. This was shown to be highly implausible and not adequately corroborated by his results. If the subject reports that the rotating spot was at a certain position at the moment he had a certain conscious experience, this would only give us the real time of occurrence of the conscious experience if there was no latency for the sensory perception of the spot or if this latency was corrected for by backward referral. "If there is no backward referral, 'clock-times' reported for S or M do not correspond to the real times of S or M conscious experience" (Gomes, 1998, p. 591). Libet's insistence that these "were the *actual times* reported by the subject" (2000, p. 9, his emphasis) is not an adequate answer to this criticism. Actual *reported* times are not necessarily actual times of *occurrence*, since the subject may have been conscious of a spot position that had occurred *some time before*. He refers to his previous reply to this argument (which had already been raised by other commentators) but does not address my observation that this reply is biased by his adherence to the backward referral hypothesis (Gomes, 1998, p. 591).<sup>2</sup>

Another problem with the timing method concerns the differentiation between W (awareness of intent) and M (awareness of movement onset). Libet (2000) does not address my argument that "[w]e usually have just a unitary awareness of voluntarily moving" (Gomes, 1998, p. 589), not two distinct awarenesses, first, one of wanting to move and then one of moving. He objects to my statement that it seems he "exaggerates when he says that subjects had no difficulty in distinguishing W and M moments" (Gomes, 1998, p. 589). However, Libet and collaborators' own original report support this statement. For example: "Some subjects stated, on their own, that . . . [i]n a W series there was a feeling of active attention to or '*watching for*' the awareness of wanting to move, so as to be able to note the time of its appearance. . . ." (Libet et al., 1983, p. 627, my emphasis).

<sup>2</sup> Incidentally, an additional problem for Libet's control "S" series (in which the subject timed the sensation of a peripheral stimulus by reference to the rotating spot) is highlighted by Nijhawan's finding that moving and stationary stimuli have different latencies for conscious perception (Nijhawan, 1994; 1997).

A third problem, which has to do with the influence of training during his experiments (Gomes, p. 590), was not addressed by Libet (2000).

### *Conscious and Nonconscious Acts*

Keller and Heckhausen (1990) found that RPs are present before both conscious and nonconscious acts. This goes against Libet's assumption that RPs are specific of conscious and voluntary acts. My conclusion was as follows: "conscious and nonconscious acts share more than a certain conception of conscious voluntary acts would be ready to admit. (. . .) Consciousness of the intention and consciousness of the act itself seem to be processes that are added to the process of generating and performing a nonconscious motor act" (Gomes, 1998, pp. 587–588).<sup>3</sup>

However, Libet implies that I disregarded the differences between the RPs for conscious and nonconscious acts, and he stresses "the evidence from Keller and Heckhausen, that the unconscious "RPs" involve a different neural pathway" (Libet, 2000, p. 8). First of all, Libet appears to be skeptical about the real existence of RPs preceding nonconscious movements. Apart from the inverted commas for "RPs" in the previous quotation, he also states that Keller and Heckhausen recorded "a *kind* of RP" (p. 8, my emphasis) with nonconscious movements. In fact, Keller and Heckhausen's published results leave no doubt that *real* RPs were recorded prior to the observed nonconscious movements. Concerning the differences between RPs for conscious and nonconscious acts: I have explicitly discussed them in my article, contrary to what Libet implies. I have even pointed out one difference that is not mentioned by Keller and Heckhausen themselves but which is apparent on the tracings they published (Gomes, 1998, p. 588).

The existence of these differences is not inconsistent with my view. On the contrary, if consciousness of the intention and consciousness of the act are brain processes that are added to the generation of a motor act, then there must be specific neural activations that correspond both to these conscious experiences and to the conditions that lead to their occurrence. However, there is enough similarity between the RPs for conscious and nonconscious movements to justify the conclusion that the preparation and initiation of the two kinds of acts have much in common.

As regards the "different neural pathway": although Keller and Heckhausen speculate on the neural generators that may be responsible for the observed differences, they also state that "it is impossible to determine the underlying neural structures by the method of movement-related potentials (. . .)" (Keller and Heckhausen, 1990, p. 359). Anyhow, their hypothesis is that "each type of RP is *predominated* by different neural generators" (*ibid.*, my emphasis), not that these are altogether different in the two cases. The fact remains that in both conscious and nonconscious acts, RPs were observed and that despite their differences, they have much in common.

In the last part of his reply, Libet (2000) restates his views on free will and conscious control. A fuller discussion of these can be found in Gomes (1999).

<sup>3</sup> I have expanded this view in a later article (Gomes, 1999).

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