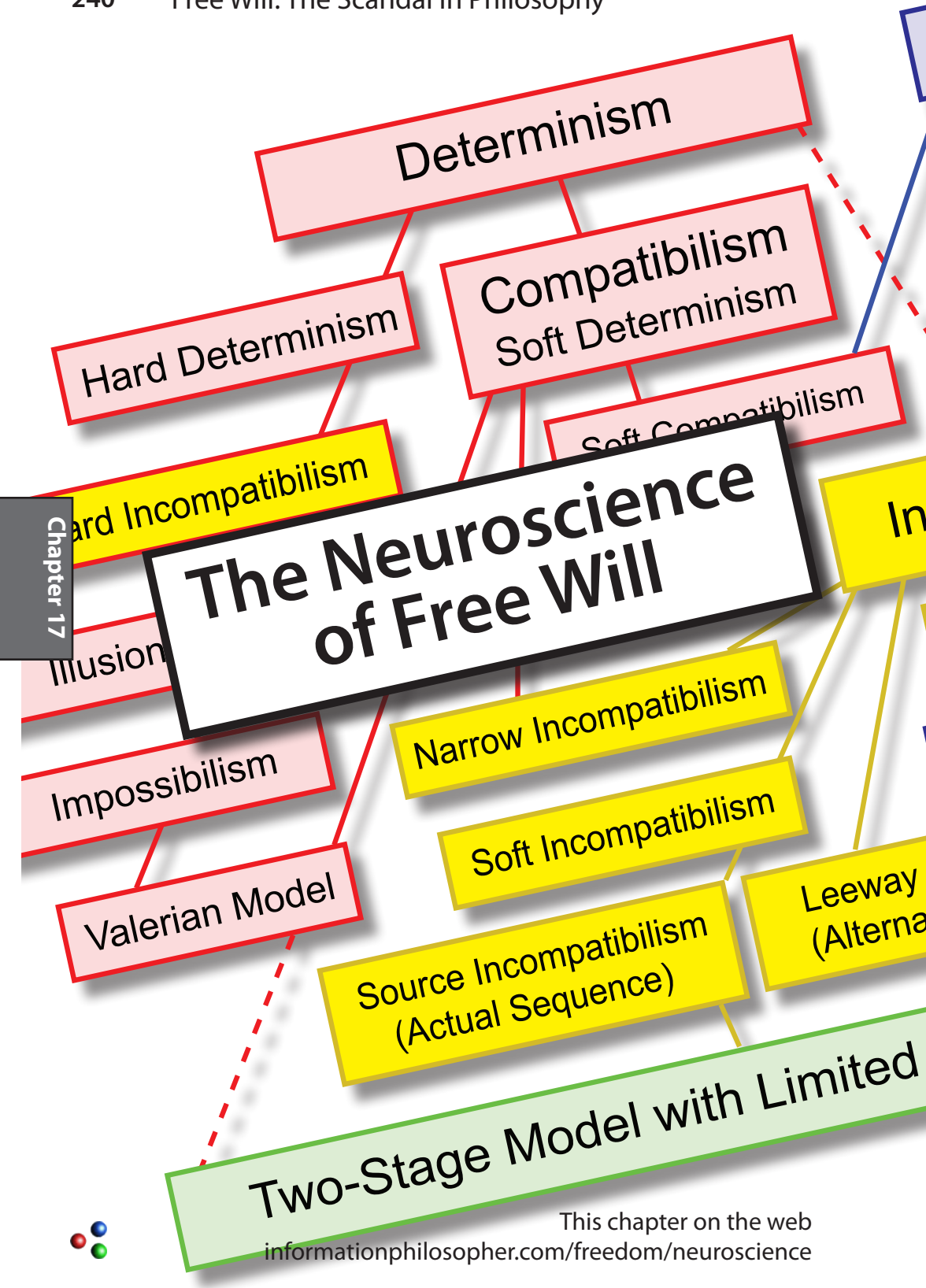

This chapter on the web
informationphilosopher.com/freedom/neuroscience

The Neuroscience of Free Will

Molecular biologists have assured neuroscientists for years that the molecular structures involved in neurons are too large to be affected significantly by quantum randomness.

Nevertheless, some neurobiologists looked for structures small enough to be affected. John Eccles identified what he called "critically poised neurons," whose synapses might discharge their vesicles with thousands of neurotransmitters as a result of "downward causation" from the mind. Roger Penrose and Stuart Hameroff see the microtubules in the cellular cytoskeleton as small enough to produce quantum coherence, perhaps some nonlocal entanglement.

As small as these structures are, they still contain many thousands of quantum level objects (atoms and molecules). How can any single quantum event in the brain get amplified to become macroscopically important? This is the question that has faced everyone who wants quantum randomness to be the basis for human freedom.

Will neuroscientists ever be able to look at a neuron and see exactly what it is thinking? Maybe not, but some have thought they can use brain activity measurements to prove that free will does not exist.

Libet's Experiment

Benjamin Libet's famous neuroscience experiments are widely regarded as having established that conscious will is an illusion, starting with Libet's own claim (mistaken, we shall argue below) that the readiness potential (RP) that he observed a few hundred milliseconds before the awareness of conscious will and the consequent muscle motion, "initiates" and is the **cause** of both the will and the action. [1]

As Alfred Mele has shown, the experimental data do not support a causal relationship. We can see this by interpreting the rise

1 Libet (2004) p. 136.

in the RP as the early stage in the two-stage model. The brain may only be considering its alternative possibilities!

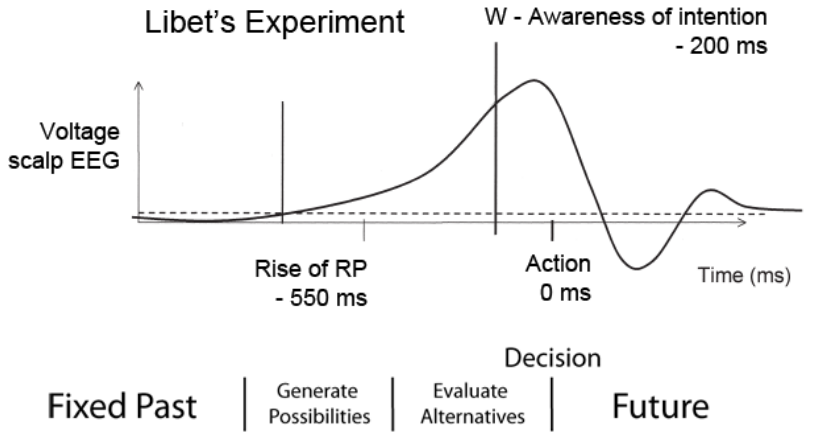

Figure 17-1. Early brain activity may only be considering possibilities.

Note that Libet proposed that the will could nevertheless be free, if there was time for it to "veto" its own prior decision, which had been caused by the early rise of the RP. But his main mistake was to conclude that the first sign of activity was causative, rather than merely enabling the later decision.

Although the abrupt and rapid decisions to flex a finger measured by Libet bear little resemblance to the kinds of two-stage deliberate decisions needed for responsibility, it seems reasonable to assume that neuronal activity might arise as the mind considers whether to flex or not to flex, when it forms the intention to flex. Roderick Chisholm argued that at least one alternative possibility always exists, we can always say no. So Libet's "veto" is already in the running as a possibility, and Libet need not have worried that there is too little time for it be effective, as his critics have maintained.

Libet, Patrick Haggard, Daniel Wegner, and the others who say the conscious will is not the cause of the action, because your neurons have already made the decision, cannot prove a causal relation between RP and action.

They are in fact begging the question of free will by assuming that a deterministic relation already exists between the early stage RP and the, action simply because it shows up earlier than the action (*post hoc, propter hoc*).

What if the early RP is just the first stage of developing options, followed by evaluating them, then deciding? In such an arbitrary choice - to flex or not flex, we should expect to see the readiness potential occasionally rise up, but then *not be followed by the W* point, and of course no muscle motion. The fact that Libet reports none of these may appear to lend weight to the idea that RP and muscle motion are indeed causally related. But this is a mistake, as pointed out by Alfred Mele.[2]

All the Libet experiments work by permanently storing the last few seconds of data that have been collected, when triggered by detecting the wrist flex itself. If there is no wrist flex, there is no data collected. The equally likely (in my view) cases of a rise in RP followed by no wrist flex would have been systematically ignored by Libet's method of data collection.

Should new versions of the Libet experiments find this missing data, it would establish that there is no causal connection between RP and action, only between RP and considering alternative possibilities, to flex or not to flex, in the two-stage model of free will.

Libet and the Two-Stage Model

In his late work *Mind Time*, Libet surprisingly describes more than one "initiative," disconnecting the RP from the action.

> "We may view the unconscious initiatives for voluntary actions as "burbling up" unconsciously in the brain. The conscious will then selects which of these initiatives may go forward to an action, or which ones to veto and abort, so no action occurs." [3]

These initiatives are **alternative possibilities**, "burbling up" suggests they "present themselves" randomly, as William James says, and selection is clearly the **adequately determined** second stage of our two-stage model.

2 Mele (2010) p. 53.
3 Libet (2004) p. 148.