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Spooky Metaphysics, Weird Neuroscience, Kane and the Varieties of Free Will Worth Wanting

Introduction

This paper will explore Kane's (1996) suggestion that "ultimate responsibility" (hereafter, UR) is a feature of a variety of free will worth wanting, and examine his proposed mechanisms by which it is to be realized. The present paper will thus be divided into four sections. First, it will explain what Kane takes UR to be. Second, it will discuss his proposed mechanism for UR. Third, it will critique these proposed mechanisms in the light of neuroscience, physics and other branches of science. Fourth, it will sketch the beginnings of an alternative account which is remarkably similar to Kane's but without some of the perceived flaws of the third section. This section shall also make a brief remark concerning the role of probabilistic models of nervous systems and future directions for research.

Section One - Kane's definition of UR

Kane states his UR condition as follows (1998, pg. 35, italics in original):

> An agent is ultimately responsible for some (event or state) E's occurring only if (R) the agent is personally responsible for E's occurring in a sense which entails that something the agent voluntarily (or willingly) did or omitted, and for which the agent could have voluntarily done otherwise, either was, or causally contributed to E's occurrence and made a difference to whether or not E occurred, and (U) for every X and Y (where X and Y represent occurrences of events and/or states) if the agent is personally responsible for X, and if Y is an *arche* (or sufficient ground or cause or explanation) for X, then the agent must be personally responsible for Y.

Let us explore the parts of each clause in turn to better understand this thesis so that in section two we can better understand Kane's motivations for his proposed mechanism by UR is to be realized.

(R) is the less controversial part of the definition of UR. As Kane points out, a traditional compatibilist can be happy with

this part of the definition. This does assume that Kane and the compatibilist are both able to give adequate accounts of what "voluntary" means. Note that Kane is not requiring that E is done voluntarily, just a prior occurrence results in E is. This clause is presumably to allow for cases of being responsible for conditions after an individual loses their voluntary control. For instance, according to Kane, one is (generally) responsible for getting drunk and then driving even if the driving was done involuntarily. On this scheme, people in essence are responsible for losing voluntary control in the first place, and that results in them being responsible for the consequences thereof.

The next clause in the (R) conditions concerns "could have done otherwise". Again, the compatibilist can interpret this clause the way she would wish to. For instance, she can adopt the "if the past were different, the choice would have been different" interpretation of the "could have done otherwise". Kane does not wish to disagree with the compatibilist at this stage and thus, welcomes interpretation by compatibilists of this clause in their terms. He remarks in a footnote (1996, pg. 221n10), however, that these past two requirements are actually redundant because they are entailed by the rest of U+R.

The final part of (R), "causally contributed to E's occurrence and made a difference to whether or not E occurred" is the most difficult of the (R) clauses to elucidate. It means that if an event (or state, to allow Kane his metaphysics) occurs, this event would have not occurred in the same way or at all if it weren't for the agent. This seems a bit puzzling. While it is true that one wants the agent to have a role in whether something occurs or not to hold them responsible (*ceritus paribus*), this condition does not specify how much of a role is necessary. (After all, to take an absurdly extreme case: someone standing horrified at a shooting deflects the bullet a minute amount resulting from their gravitational attraction on it.) This is important for the proposed mechanism concerns, which we shall see after our discussion of the ultimacy part of the condition, to which we now turn.

The “ultimacy” portion of UR is the part of Kane’s condition for free will that is contentious. It revolves around the notion of an *arche* or sufficient ground. He characterizes this feature (1996, pg. 73) as follows (italics in original):

> “What U thus requires is that if an agent is ultimately responsible for an action, the action cannot have a sufficient reason of any of these kinds [sufficient conditions, sufficient causes, sufficient motives - K.D.] *for which the agent is not also responsible.*”

This condition requires the agent be responsible for the sources of their actions and of their will to perform the actions. This is important for Kane, for without the second clause of the previous sentence obtaining, agents would possess free action (*ceritus paribus*) but would not have free <u>will</u>.

This entails that there either must be actions which ground responsibility through an indeterminate mechanism, or there are further actions which ground its associated responsibility. This potentially creates an infinite regress.

Since there cannot be an infinite regress of responsibility (humans being finite creatures), Kane postulates that at some moments in a (presumably typical) person’s life there are what he calls self forming actions (SFAs) by which one’s character is developed. These SFAs thus ground some degree of future responsibility. SFAs cannot therefore be determined as these are supposed to be the sense in which our actions are “up to us” in a way that is incompatible with determinism. These do involve a sense of “could have done otherwise” that the compatibilists do not accept. The world was genuinely open to two distinct possibilities in the future, and <u>we</u> made it go one way rather than the other. This brings us into the question of how Kane thinks this occurs - i.e., his proposed mechanism.

<u>Section Two - Kane’s proposed mechanism</u>

As Kane states it, the proposed mechanism for a freely willed action appears to be rather simple: it starts with a quantum level undetermined event, one which gets chaotically amplified by a neural network in the brain, eventually leading to a choice that

“could have gone the other way.”

Kane does not give us any direct discussion over what type of quantum events he has in mind.

This mechanism can also operate in SFAs, grounding responsibility for actions in the future that the agent had less or no choice over. Here, Kane is acutely aware of cases where an agent did a supremely moral act and had apparently no choice whether or not to so act at the time. Kane refers to Dennett’s Luther example; I prefer my own case of such an occurrence. I shall set it out so we can analyze Kane’s mechanism, in part using it as a test case.

Last year, around March or so, I was with an extremely close friend in Montréal, leaving campus to go to a yoga class. We came to the entrance to the Métro, and it turned out that she had somehow misplaced her wallet and thus, we (a) couldn’t go to yoga together, and (b) she had to return to campus to hunt down her wallet. At that moment, I knew even as I turned and left with her that I had only one possible choice - to stand by her. So back to campus I walked with her, and so on, knowing I could not have done otherwise. Yet, I regard this as one of the most powerfully moral experiences I have ever had, and one of the reasons I know I care for her to this day.

If Kane were right, a neurologist looking into my brain at some point in my history in principle could find a quantum level event that lead to an assembly of neurons somewhere in my nervous system that would be guaranteed to fire in concert when exposed to “dear friend in serious difficulty”[1].

So although my behaviour was (perhaps) compelled completely at the time I acted, I acted in way in which I could be morally responsible. This is in virtue of a free willed action (or inaction) in the past whose “world line” with a quantum event in

[1] Fuller (2000) suggests that settling the free will debate presupposes solving the mind-body problem. This may very well be correct, and this comes out especially in this type of situation. This is because it is not clear at all whether one should talk about stimuli to the nervous system in this fashion, but this concern is too much of a digression to handle in the present paper.

my nervous system.

Let us turn to the second large feature of Kane's proposed mechanism concerns chaos. Kane does not suggest much in the way of what parts of the nervous system would be chaotic. The references he provides are not much help; see below for details.

Section Three - Critique of mechanism

In this section I shall provide three reasons to suppose that Kane's proposed mechanism is implausible. The first of these has to do with proposed chaotic mechanisms. The second two concern the neuroscientific details of what he proposed and comes in two parts. One concerns the putative quantum level effects in the brain, and the second concerns the mechanism behind the phenomenology of willing he proposes.

It helps to have a good elucidation of "chaos" on the table in order to determined whether any subsystems of the brain are chaotic in the technical sense. First, a misconception: note that mere sensitivity to initial conditions is not sufficient for chaos: a system modeled by the simple differential equation (say, if x is a displacement and t, time):

$$\frac{dx}{dt} = x$$

is very sensitive to initial conditions, but it is not chaotic in the mathematical sense. This is important for Kane's purposes as little conditions are supposed to on his account have radically different effects, not simply wildly less or more of a given effect.

If a merely amplificative process of the type modelable by the above differential equation, rather than a chaotic one, were involved in deliberation, it would seem that this would result in small perturbations being more influential. Suppose someone was 51% likely to do action A and 49% to do B. This sort of amplification would result in the 1% difference being quickly expanded to a larger amount (it would double every "time period"). This consideration of chaos vs. merely large sensitivity to initial conditions is important, because it further constrains the

parts of the nervous system by which his mechanism could work. Sekuler and Blake (1994) imply that the visual system works in the latter fashion, for instance. But, Kane needs some amplification, as must have, to speak loosely, some mechanism for turning small effects into big ones. (Note that Kane's quotations on page 129 of his 1996 deal only with the amplicative effects, and none of them propose a mechanism by which this is to occur.)

Kane makes use of several reasonably recent articles concerning chaotic behaviour in the brain. It bears investigating the pieces he references to see whether they can be made to bear the type of burden Kane wants them to. First, both the article Kane refers to (Bablotantz & Destexhe 1985) and a similar one (an der Heiden & Mackey 1985) does not support chaos in the brain in a way that Kane would want. The article concerns recordings from the human electroencephalogram (hereafter, EEG).

In order to demonstrate the implausibility of this measurement being an indication of what Kane wants, let us rehearse what a EEG measures. As Churchland and Sejnowski (1996 [1992], pg. 438) point out:

> "[...] the EEG recording is a composite signal from volume conduction in many different parts of the brain, and it is far from clear what a signal means in terms of how individual neurons in the relevant networks are behaving."

In gist, the EEG measures a property of <u>global</u> brain functioning. Not all of the brain's systems are involved in willing, decision making, action, etc. For Kane's hypothesis to be vindicated, there would have to be chaotic amplification in the applicable system - for his hypothesis exactly, in the decision making centers. Demonstration of chaos in the brain as a whole does not indicate this. Decision making centers in the brain would have to be chaotic along one dimension only for his account to work; otherwise, it seems one would be flighty. This dimension is the issue of quantum influence on decision making in "just the right way". For instance, the influence cannot be such that outside influences are irrelevant to the "quantum coin toss" - somehow the probability distributions must change sensibly. But, Kane provides

no hypotheses concerning how this is to work. He does gesture at some work in non equilibrium thermodynamics, but these are insufficient - let us see why.

Kane's references along the aforementioned lines, to papers in *Dissipative Structures and Spatiotemporal Organization Studies in Biomedical Research* (Scott & McMillen 1980) also concerns global brain functioning. Kane is right that the nervous system is a far from equilibrium system (in the thermodynamic sense discussed in the aforementioned collection). But, this is again a global property of the nervous system as a whole (and, indeed, apparently of organisms as a whole). Scott has also edited a further book, (Scott 1991) which Kane also references concerning such matters. The present author has not reviewed this book, however, Kane does not mention enough of its content to adequately make a case using it regardless. In summary, then: Kane and his supporting texts do not clearly make the case for relevant chaos, both in respects of what parts of the nervous system are chaotic and how this chaos is to be "tuned".

This brings us to the second feature of Kane's account which is dubious. No mechanism of action is proposed by Kane by which a quantum level event would have neurological significance. He refers to a book by Stapp (1993), one very similar in spirit to the speculations of Penrose (1989) whom he also refers to several times concerning issues of consciousness as they pertain to the free will issue. Many criticisms of such speculations about quantum level events in the brain have been raised in the literature. First is the issue of size - most quantum systems[2] are very small (known exceptions include, but are not limited to, neutron stars and Böse-Einstein condensates (BEC)). Now, the nervous system is not a neutron star or a BEC, so there are apparently two alternatives. One possibility is that something

[2] Here "quantum system" is used to mean any system in which quantum properties are not "drowned out". One way that this "drowning out" can be looked at (metaphorically) is to imagine a token on a grid. Roll one eight sided die and move the token according to the direction indication chart: [1: up-left 2: up 3: up-right 4: right 5: down-right 6: down 7: down-left 8: left] This token represents a tiny quantum system. Note that its position is very significantly changeable. Now compare that to the case of (say) one billion billion tokens on the grid. The fluctuations tend to be averaged out by the large numbers involved.

very small is somehow isolated in its quantum behaviour. This is necessary, for its effect must be (according to Kane) chaotically amplified before it is drowned out by quantum "noise"[3]. Penrose suggested microtubles in neurons are suitable for this. Stapp claims (according to Kane) that the global character of consciousness and quantum mechanics is the reason to hypothesize a connection between the two. Stapp's proposal makes much use of the famous EPR experiment because (supposedly) brains and EPR are holistic in character (Stapp 1998).

The above are implausible for several reasons. First, Penrose's speculations are shown to be unlikely (a) because not just neurons have microtubles (they are part of the mechanism of cell division). Do our livers (for instance) have free will? And do all animals with neurons have free will to the same degree? The microtubles in the cells of a frog are much the same as those in humans. Admittedly, the chaotic amplification system Kane proposes may be different in each case, but then one has show how to connect the two, which leads to (b) are they suitably "quantum" in nature. The evidence appears to be "no". Penrose has at best shown it is possible, not that it is the case. See Churchland and Grush 1995 for more on these issues and other reasons to suppose this sort of speculation is misguided. Kane has to deal with similar objections even if Penrose's specifics are not adopted. How is it that only nervous systems are sensitive in the way he wants?

Stapp's speculations (1993), of which Kane gives more attention, are even more implausible. Stapp's speculations and interpretation of the EPR "paradox" (see above) make intense use of the Copenhagen interpretation of quantum mechanics, which while originally orthodoxy, is slowly being regarded as not only inconsistent with the physics as actually practiced, but philosophically stultifying (see, e.g., Stenger 1995, Bunge 1967). Stapp's use of this (mis)interpretation of quantum mechanics is thus really no help to Kane. As pointed out by several authors in Bunge 1967, the Copenhagen interpretation is a 20th century version of age old subjective idealism, and if that's not invoking

[3] The effect may also get drowned out by other fluctuations (e.g. thermal, chemical). I am ignoring these as I am trying to be as charitable to Kane as possible.

“panicky metaphysics” (Russell 2000) to solve one’s problems of free will, what is?

Stapp also considers quantum level effects determining whether a given neuron is to be inhibited or excited in a given situation. Kane quotes these favourably. It is unclear how this would work, as a neuron is still largely a classical system (Churchland and Grush 1995). One would need another chaotic amplifier at the neuron level for this to work; Kane and Stapp have no proposed any such mechanism.

The other alternative is that the nervous system is some sort of (as it is said) coherent system analogous to the neutron star or BEC cases above. Kane (1996) does not give us any reasons to suppose that this is the case, only that his account may require such a situation. Unless special conditions occur the decoherence time (roughly a measure of the degree to which a system is “quantum” in nature) of even the smallest of brain subsystems would be microseconds.

It should be noted that the chaotic amplification problem also is doubled by Kane as it requires the amplification twice, one for each of two distinct systems of the brain. On page 130, we find his summary of the viewpoints he is canvassing in the form of his thesis T25, which reads as follows:

> Imagine that the indeterminate efforts of will of T24 are complex chaotic processes in the brain, involving neural networks that are globally sensitive to quantum indeterminacies at the neuronal level. Persons experience these complex processes phenomenologically as “efforts of will” they are making to resist temptation in moral and prudential situations. The efforts are provoked by the competing motives and conflicts within the wills of the persons described in T22 and T23. These conflicts create tensions that are reflected in appropriate regions of the brain by movement further from thermodynamic equilibrium, which increases the sensitivity to micro indeterminancies at the neuronal level and magnifies the indeterminacies throughout the complex macro processes which, taken as a whole, is the agent’s effort of will.

The two amplifications are thus (1) the mechanism by which the

action takes place and (2) the monitoring process that "observes" this mechanism of action. Both of these would have to be such in order that this "observation" process be correct[4]. They must be distinct, as it is reasonably well established in neuropsychological circles that these two systems are distinct. (Petrides 1989). This leads us to our final objection with Kane's account, namely, that the neuroscience proper necessary for Kane's account is confused.

This concerns the apparent assumption of Kane that will is a global property of the nervous system or brain. This seems unlikely for three reasons. One concerns mechanisms of will that are known. It has been suggested on neurological grounds (Jahanshahi & Frith 1998) that the willed action "route" is goals/plans ⇒ willed intention ⇒ action ⇒ response. This is not compatible with Kane's account as (1) the will on the neuroscientific scheme is a stage in the process, not something that accompanies the whole process and (2) there is no feedback to speak of in many neuronal systems of action until the action is completed, unlike on Kane's account which suggests more monitoring along the process than is there. For a discussion of this fact and its relevance to philosophy of action, see Dennett 1991, pgs. 139-170, especially 168-169.)

It may be rejoined that the above objection equivocates on the meaning of will - that neuropsychology and philosophy use the term differently. Well, perhaps this is so, but Kane has given us his own idiosyncratic definition which does not match on to what is known about what does match on to the conventional definition of will. He is thus the one making a persuasive definition. (After all, Jahanshahi and Frith make a point of dealing with that objection to their thesis in their paper.)

The defender of Kane may remark at this stage that appealing to Jahanshahi and Frith's paper is anachronistic, as it was released two years after Kane's book. I grant this objection in so far as

[4] Consider Figure 1 below (see page 15 of this text). The grey line represents the true chaotic behaviour of the system; the black line represents an imperfect monitoring mechanism. Notice how right it is sometimes and yet how wrong it is at others.

it is correct to say that Kane (of course) need not have considered the article in question but the issue of the current paper is whether Kane's account can still hold up generally.

Second (of the will problems), Kane gives no indication that there is such a global monitoring device in the nervous system that is necessary to support his claim. Recall that Kane thinks we experience efforts of will; in order for this to be the case, there must likely be a monitoring system of some sort in the brain. This is part of what distinguishes occurrences we are aware of from those we are not, though not necessarily those involved in "consciousness" in the contentious sense. (Once again, we have to skirt the mind-body problem.) The self monitoring mechanism (1996, page 140) he borrows from Koch, etc. is not chaotic; in fact, the whole issue concerning the "binding mechanism" hypothesis is that it involves a steady neuronal firing frequency (Koch & Crick 1994).

Third and finally (and perhaps most interestingly to those concerned with free will proper), Kane gives no mechanism by which willing would be the sort of process to move the brain further from thermodynamic equilibrium. Assuming that the relevant systems of the brain become more chaotic during moments of great willing, this means they become more and more sensitive to initial conditions. If these are quantum level conditions and in the brain, they could be brought about by an environmental radioisotope[5] (i.e. one outside the agent) or the like. And so deliberation could thus be more sensitive to external influence (and, moreover, irrelevant external influence) according to Kane's account. We could thus be made less free. It may be argued that the exact source of the undetermined event is unimportant; merely that the system it applies to be the agent's own. This is plausible, except for the fact that what guarantees the choice at the right time? This collapses into the self monitoring mechanism worries - how does the system by which the undetermined events work "sometimes". In the light of the present concern, it is merely that the decision making systems would be repeatedly

[5] Decay of a radioisotope is one possibility for Kane's undetermined events. As I shall remark shortly, though, this is not discussed in adequate detail in Kane's work.

affected from outside by radioactive decay without regard to the rest of the nervous system.

(The aforementioned effect not significant in normal accounts of brain functioning because quantum level effects (of any kind) are usually “washed out”, as discussed above.)

There are two ways Kane can get out of this difficulty, neither of which are discussed in his work. The first could be that the appropriate system is well insulated from outside events of the appropriate sort. (Kane has not specified even what sort of quantum event his mechanism uses, so it is rather difficult to speculate on what sort of “shielding” would be appropriate.) Kane could also presumably bite the bullet and assert that areas with slightly more ambient radioactivity (for instance) do result in people having slightly different phenomenology of willing. This would be because the events that “spark off” the willing on his account would occur slightly more often. It is unclear what this would do to the agent, however.

Section Four - Hints of future direction

I have shown that Kane’s account of the mechanism of UR is implausible in the light of what is actually known about the brain and nervous system. In this section I will propose the starting point of a remarkably similar account, but one without (I hope!) the dubious neuroscience of the previous question. The insight I will draw upon concerns the difference between partially self-caused and externally caused raised in the previous section. The account is necessarily very incomplete, as I feel we do not have enough knowledge of the nervous system’s functioning to give a more substantial account. For instance, this account will largely ignore cortico-limbic interactions. This is a weakness of Kane’s account as well; here I simply acknowledge that my account has this weakness as and hope that it is interesting enough for someone to pick up from where I left off. My final note in this paper shall be a remark about the use of probability in modelling the nervous system.

The first important facts to realize about the nervous system are first the two levels of spontaneity that occur, and second, what

exactly is meant by spontaneity in this context. Neurons both spontaneously fire (Sekuler & Blake 1994) and spontaneously self-assemble into systems (Hebb 1949). What is meant by spontaneity is important for the understanding of free will here. As we have seen, Kane wants UR. Spontaneity gives him the exact toe in the door he wants, for it means the appropriate events (firing and self-assembly) occur in the absence of external stimuli[6]. (One can regard this as an experimental disproof of behaviourism.) Of course, as Kane himself notes, this is not sufficient for free will, as after all, assembly and firing must occur with some sensitivity to input. Here we can make use of SFAs, discussed above.

Self-forming actions on this account would make some use of the notion of spontaneous assembly. The assemblies that result in these cases have properties that are not attributable directly to the stimulus received. One such place in the brain where this is significant from the perspective of free will is the frontal cortex.

This is because decision-making in humans occurs in the frontal cortex (Milner & Petrides 1984). Patients who have severe frontal damage are not able to make decisions of various kinds, and often engage in persistent actions even in the light of evidence that these actions are not optimal in a given case. This might (if suitably investigated) lead us to postulating the kinds of agents which cannot appropriately make choices (moral or otherwise). Some literature suggests that moral doing is even anatomically separable from moral reasoning (e.g. Jennings 1999), so a hypothesis about one feature of a morally responsible agent would be one in which these subsystems are conjoined properly both at the moment of action and at the time of self forming actions. Of course, one could very well be responsible for the dissociation, in which case typical evaluation of responsibility for the actions that result after the dissociation applies. (This is much the case as (e.g.) the usual evaluation of cases of actions under the

[6] Those who have "agent causation" worries about this should recall that self activity is not limited to neuronal systems. As Bunge (1979) points out, inertia falsifies the peripatetic maxim that everything that moves is moved (only) by another.

influence of alcohol.)

We can get a version of UR out of this under certain circumstances, as well. To the extent that neuronal assemblies in the decision making centers of the brain (for instance) are spontaneous assemblies, rather than merely ones created in response to stimuli, they are ultimately ours. Note that this allows us to understand the case with my friend and her wallet, above. “I” had previously spontaneously assembled a “love” or “friendship” assembly and it in this case led to action, although its outcome in that case was (apparently) not open to choice at all.

I was ultimately responsible for (not) choosing to help my friend because of this previous assembly which itself was a free creation “of mine”. (It is, strictly speaking, inappropriate to say that I create my neuronal assemblies. One would want to say that they are me.) I was also, presumably, on this account, morally responsible because a causal chain originated in my frontal cortex and lead to action, as opposed to, say, a being a brute stimulus-response action. On this account one traces a causal path back to an agent decision making centres to see whether an agent had UR. (This account is of course, incomplete - there are many complicating factors - but this is the zero-order approximation, to speak metaphorically.) Furthermore, since the assemblies there are large enough, worries about external sources of random noise do not apply any more or less than usual during intense deliberation. Sensitivity to the environment is mediated by redundancy of neurons up to a point. But see the next point for issues of randomness in nervous systems.

Finally, note that if an important feature of Kane’s account (1996) to be salvaged is the probabilistic nature of his mechanism, then moving the level of the discussion to the single neuron overcomes the worries about quantum mechanics discussed in this paper. See Stevens 1994 for information on probabilistic, stochastic and deterministic accounts of cortical functioning. In gist, a model of free will that was essentially probabilistic would require a neuronal level model. Probabilistic attributes of

neurons tend to get washed out at the level of cortical systems, though this is not always the case. I suggest then that the area to look at for hypothesizing free will is at the level of the cortical system. The cortex is important in this context as it allows us to distinguish between animals. Humans have some degree of free will because they have a sizable cortex; animals with no or little cortex would have no or less free will on this conception. (This is not merely because of the probabilism involved, but also because the cerebral cortex is responsible for so called “higher” brain functions.)

To develop more of an account of free based on the information in the aforementioned article would require developing a general theory of action, as has been remarked several times previously. As that is off the subject for the present paper, I shall leave this remark as an “embryo” to suggest further research.

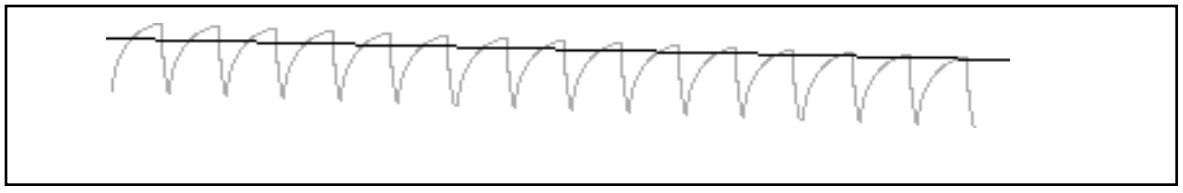
Conclusion

Kane’s UR mechanism fails on several grounds, but can hopefully be rehabilitated (in broad outline) by paying attention to one maxim. Bunge (1983) writes concerning principles of investigation:

> Do not skip levels. E.g. do not try to write the Schrödinger equation for the brain.

While Kane has not committed such a large howler as the one in the above example, he does appear to (as we have seen) make a large jump between quantum effects and psychological notions of willing and action. Replacing his quantum effects with more properly neuropsychological accounts (as we have hinted at in section four, above) may hopefully yield the results he wants without the dubiousness of the mechanisms he has proposed.

Figure 1



References

an der Heiden, U. & Mackey, M. 1985. Mixed Feedback: A Paradigm for Regular and Irregular Oscillations. In Rensing, an der Heiden and Mackey 1985.

Bablotantz, A. & Destexhe, A. 1985. Strange Attractors in the Human Cortex. In Rensing, an der Heiden and Mackey 1985.

Bunge, M. (ed.) 1967. *Quantum Theory and Reality*. New York: Springer-Verlag.

Bunge, M. 1979. *Causality and modern science*, 3e. New York: Dover Publications.

Bunge, M. 1983. *Treatise on Basic Philosophy*, Vol. 6: *Understanding the World.* Dordrecht: Rediel.

Churchland, P. & Grush, R. 1995. Gaps in Penrose's Toilings. Reprinted in Churchland, P. & Churchland, P. 1998. *On The Contrary: Critical Essays 1987-1997*. MIT Press: Cambridge.

Churchland, P. & Sejnowski, T. 1996 (1992). *The Computational Brain*. Cambridge: MIT Press.

Dennett, D. 1991. *Consciousness Explained.* Boston: Little, Brown & Co.

Fuller, L. 2000. Personal communication.

Hebb, D. 1949. *Organization of Behavior*. New York: Wiley.

Jahanshahi, M. & Frith, C. 1998. Willed Action and its Impairments. Cognitive Neuropsychology, 15 (6/7/8), pg. 483-533.

Jennings, C. 1999. The neurobiology of morals. Nature, Tuesday 19 October 1999. Available on Internet at http://helix.nature.com/nsu/991021/991021-6.html.

Kane, R. 1996. *The significance of free will*. New York: Oxford University Press.

Koch, C. & Crick, F. 1994. Some Further Ideas Regarding the Neuronal Basis of Awareness. In Koch & Davis 1994.

Koch, C. & Davis, J. (eds.). 1994. *Large Scale Neuronal Theories of the Brain.* Cambridge: MIT Press.

Milner, B. & Petrides, M. 1984. Behavioural Effects of frontal-lobe lesions in man. Trends in Neurosciences, 7 (11), pg. 403-407.

Penrose, R. 1989. *The Emperor's New Mind.* London: Vintage.

Petrides, M. 1989. Frontal lobes and memory. In Buller, F. & Grafman, J. (eds.) 1989. *Handbook of Neuropsychology*. Elsevier Science Publishing Co., pg. 75-90.

Rensing, L., an der Heiden, U. and Mackey, M. (eds.) 1985. *Temporal Disorder in Human Oscillatory Systems*. Berlin: Springer-Verlag.

Russell, P. 2000. Unpublished remark, proceedings of University of British Columbia Course PHIL 530, Winter 1999-2000 Session.

Scott, G. (eds.) 1991. *Time, rhythms, and chaos in the new dialogue with nature*. Ames: Iowa State University Press.

Scott, G. & McMillen, M. (ed.) 1980. *Dissipative Structures and Spatiotemporal Organization Studies in Biomedical Research.* Ames: Iowa State University Press.

Sekuler, R. & Blake, R. 1994. *Perception*. (3e). McGraw-Hill: New York.

York.
Stapp, H. 1993. *Mind, matter, and quantum mechanics*. Berlin: Springer-Verlag.
Stapp, H. 1998. Quantum Ontology and Mind-Matter Synthesis. Available on Internet at: http://www-physics.lbl.gov/~stapp/40722.ps
Stenger, V. 1995. *The Unconscious Quantum: Metaphysics in Modern Physics and Cosmology*. Amherst: Prometheus Books.
Stevens, C. 1994. What Form Should a Cortical Theory Take? In Koch & Davis 1994.