

Exploration

Other Plausible Mechanisms of Anesthetic Action within the Framework of Spin-mediated Consciousness Theory

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ABSTRACT

We have recently discussed the mechanism of xenon anesthetic action in spin-mediated consciousness theory in light of the recent experimental findings. In this paper, we discuss some other plausible mechanisms of anesthetic action within the framework of spin-mediated consciousness theory. In one scenario, general anesthetics such as xenon may produce anesthesia by direct perturbations and/or distortions of neural spin networks alone or in addition to perturbing and/or distorting O₂ and/or NO pathways in neural membranes and proteins. In another scenario, the mind-pixels are comprised of unpaired electrons carried by transition metal ions, O₂ and/or NO caged-in/bound/absorbed to large molecules in neural membranes and proteins. In the latter, general anesthetics such as xenon may produce anesthesia by perturbing free/unbound O₂ and/or NO pathways in neural membranes and proteins and/or unpaired electron spin networks thus blocking and/or distorting their functions in consciousness. In either of the above scenarios, the nuclear spins of xenon 131 and xenon 129 may partially play the roles of displaced nuclear spins or unpaired electron spins, thus attenuate the anesthetic potency of nuclear-spin-carrying xenon isotopes observed by Li, *et. al.*.

Keywords: Spin-mediated, consciousness, mind pixel, nuclear spin, electron spin, xenon isotopes, xenon 131, xenon 129, mechanism of anesthetic action.

1. Introduction¹

In the spin-mediated consciousness theory put forward in 2002 [8], molecules containing unpaired electron spins, such as oxygen (O₂) and nitric oxide (NO), interact with the mind pixels comprised of various nuclear spins in the brain and activate the latter as one of the steps generating conscious experience. Therefore, general anesthetics such as xenon produce anesthesia by perturbing O₂ and/or NO pathways thus distorting and/or blocking their activation functions in consciousness, as recently discussed in [1], in light of the recent experimental findings [11]. Naturally, as shown in [1], nuclear spins of xenon 131 and xenon 129 may partially play the activating roles of displaced O₂ and/or NO among other possibilities and, thus attenuate the anesthetic potency of nuclear-spin-carrying xenon isotopes found in [11].

It is believed that the anesthetic functions of xenon is associated with its binding to NMDA

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¹See Note 1 for more background discussions; see Note 2 for a brief comparisons of oxygen (O₂), nitric oxide (NO), nitrous oxide (N₂O) & xenon (Xe), please see Note 2.

receptor (See, e.g., [2]). However, there is no commonly accepted theory on how general anesthetics work even after more than 160 years since their discovery. There are two schools of thoughts on the subject. The first and oldest is the “lipid theory” which proposes that anesthetics dissolve into cell membranes and produce common structural perturbation resulting in depressed function of ion channels and receptors that are involved in brain functions (See, e.g., [3, 7]). The second, more popular and recent theory is the “protein theory” which suggests that anesthetics directly interact with membrane proteins such as ion channels and receptors that are involved in brain functions. However, the protein theory doesn't seem to square well with the low affinity and diversity of the general anesthetics. There is no direct experimental evidence to support either theory (See, e.g., [4, 7]). Both theoretical and experimental studies have shown that many general anesthetics cause changes in membrane structures and properties at or just above the clinical concentrations required for anesthesia (See, e.g., [5-6]).

In this paper, we discuss two other plausible mechanisms of anesthetic action within the framework of spin-mediated consciousness theory [8, 19-20, 22]. One scenario concerns general anesthetics such as xenon producing anesthesia by direct perturbations and/or distortions of neural spin networks alone or in addition to perturbing and/or distorting O₂ and/or NO pathways in neural membranes and proteins. Another scenario concerns the mind-pixels being comprised of unpaired electrons carried by transition metal ions, O₂ and/or NO caged-in/bound/absorbed to large molecules in neural membranes and proteins.

2. The Framework of Spin-mediated Consciousness Theory

Within the framework of spin-mediated consciousness theory, quantum spin is the seat of consciousness and the linchpin between mind and the brain, that is, spin is the “mind-pixel”, and the unity of mind is achieved by quantum entanglement of these mind-pixels [8]. The theory is based on the fact that quantum spin is the most basic quantum bit ("qubit") for encoding information and a fundamental quantum process associated with the structure of neural membranes and proteins that are saturated with quantum spin carrying nuclei and these nuclear spins have relatively long quantum coherence time (8, also see [22]). There is also a version of spin-mediated consciousness theory based on unpaired electron spin (See additional refs in [8]).

(1) Spin-mediated consciousness theory of 2002

Spin-mediated consciousness theory of 2002 [8] postulates that: (a) Consciousness is intrinsically connected to quantum spin; (b) The mind-pixels of the brain are comprised of the nuclear spins distributed in the neural membranes and proteins, the pixel-activating agents are comprised of biologically available paramagnetic species such as O₂ and NO, and the neural memories are comprised of all possible entangled quantum states of the mind-pixels; (c) Action potential modulations of nuclear spin interactions input information to the mind pixels and spin chemistry is the output circuit to classical neural activities; and (d) Consciousness emerges from the collapses of those entangled quantum states which are able to survive decoherence, said

collapses are contextual, irreversible and non-computable and the unity of consciousness is achieved through quantum entanglement of the mind-pixels.

As mind-pixel activating agents, the unpaired electrons of the paramagnetic species such as O₂ and NO can interact with nuclear spins through their large magnetic dipoles and collision-induced Fermi-contact mechanism thus activating the neural nuclear spin ensembles [8]. Indeed, because the magnetic dipole moment of an unpaired electron is 658 times larger than that of the ¹H nucleus, O₂ and NO can respectively produce magnetic fields 1,316 and 658 times larger than ¹H (See, e.g., [17]). In addition, O₂ and NO are hydrophobic small molecules so their concentrations in neural membranes are much higher than in aqueous solutions such as cytoplasm (See, e.g., [15]). Thus, as they rapidly tumble and diffuse, they produce microscopically strong and fluctuating magnetic fields. O₂ are the predominant sources of internal magnetic fields in neural membranes and proteins, as evidenced by the strong effect of O₂ on spin-spin and spin-lattice relaxation rates (See, e.g., [18]).

In additions, nuclear spin networks in neural membranes and proteins are modulated by action potentials through *J*-coupling, dipolar coupling and chemical shielding tensors and perturbed by microscopically strong and fluctuating internal magnetic fields produced largely by paramagnetic oxygen [19-20]. Further, both nuclear spin in neural membranes and proteins as mind-pixels and unpaired electron spins of O₂ and NO as mind-pixel activating agents are also modulated by action potentials through relativistic effect of moving spin in strong electric field [20, 22] and, in turn, said modulation of the activating agents influence the nuclear spins in the brain as shown below. Thus, action-potential modulations of both nuclear and unpaired electron spins synchronize their collective dynamics to the neural firings [19-20].

The decoherence effect which causes a quantum system to lose quantum coherence through interactions with its environment has been a major concern for any quantum theory of the brain (See, e.g., [23]). However, nuclear spins only have weak interactions with their environments thus long relaxation time after excitation (See, e.g., [14]). Indeed, there are both theoretical and experimental studies indicating the possibility of large-scale quantum coherence with entanglement in the nuclear spin ensembles distributed in the neural membranes and proteins (See, e.g., [24-32]). Further, studies show that decoherence-free subspaces can exist within the Hilbert space of a complex quantum system (See, e.g., [33-34]).

(2) Plausible modification to spin-mediated consciousness theory

If molecules with unpaired electrons such as O₂ and NO do not serve activating agent, the postulates of the spin-mediated consciousness theory of 2002 [8] may be modified as follows: (a) Consciousness is intrinsically connected to quantum spin; (b) The mind-pixels of the brain are comprised of the nuclear spins distributed in the neural membranes and proteins, and the neural memories are comprised of all possible entangled quantum states of the mind-pixels; (c) Action potential modulations of nuclear spin interactions activate and input information to the mind pixels and spin chemistry is the output circuit to classical neural activities; and (d) Consciousness emerges from the collapses of those entangled quantum states which are able to

survive decoherence, and the unity of consciousness is achieved through quantum entanglement of the mind-pixels.

In this modified theory, nuclear spin networks in neural membranes and proteins are modulated by action potentials through J -coupling, dipolar coupling and chemical shielding tensors and perturbed by microscopically strong and fluctuating internal magnetic fields produced largely by paramagnetic O_2 [19-20]. Further, nuclear spin in neural membranes and proteins as mind-pixels are also modulated by action potentials through relativistic effect of moving spin in strong electric field [20, 22]. Thus, action-potential modulations of nuclear spins synchronize their collective dynamics to the neural firings [19-20].

Again, decoherence effect which causes a quantum system to lose quantum coherence through interactions with its environment has been a major concern for any quantum theory of the brain (See, *e.g.*, [23]). However, nuclear spins only have weak interactions with their environments thus long relaxation time after excitation (See, *e.g.*, [14]). Indeed, there are both theoretical and experimental studies indicating the possibility of large-scale quantum coherence with entanglement in the nuclear spin ensembles distributed in the neural membranes and proteins (See, *e.g.*, [24-32]). Further, studies show that decoherence-free subspaces can exist within the Hilbert space of a complex quantum system (See, *e.g.*, [33-34]).

(3) Plausible electron-spin-mediated consciousness theory

As a plausible alternative to nuclear spin based consciousness, the electron spin mediated consciousness theory put forward in 2004 (See add'l refs in [8]) postulates that: (a) Consciousness is intrinsically connected to quantum spin; (b) The mind-pixels of the brain are comprised of the unpaired electron spins distributed in the neural membranes and proteins, the pixel-activating agents are comprised of the unbound/free O_2 and NO , and the neural memories are comprised of all possible entangled quantum states of the mind-pixels; (c) Action potential modulations of electron spin-spin interactions input information to the mind pixels and spin chemistry is the output circuit to classical neural activities; and (d) Consciousness emerges from the collapses of those entangled quantum states which are able to survive decoherence, said collapses are contextual, irreversible and non-computable and the unity of consciousness is achieved through quantum entanglement of the mind-pixels.

Through action potential modulated electron spin-spin interactions and fluctuating internal magnetic field driven activations, the neural electron spin networks inside neural membranes and proteins form various entangled quantum states some of which survive decoherence through quantum Zeno effects (See, *e.g.*, [54]) or in decoherence-free subspaces (See, *e.g.*, [33-34]) and then collapse contextually via irreversible and non-computable means producing consciousness and, in turn, the collective spin dynamics associated with said collapses have effects through spin chemistry on classical neural activities thus influencing the neural networks of the brain (See add'l refs in [8]). It is also argued that the unpaired electron spins inside a network of large molecules may be able to form long-lived macroscopic quantum coherence through tunneling since they are

insulated to certain extent from the noisy brain environment (See add'l refs in [8]). Thus, according to this alternative approach, the unpaired electron spin networks are the “mind-screen,” the neural membranes and proteins are the mind-screen and memory matrices, and unbound paramagnetic small molecules such as O₂ and NO are pixel-activating agents (See add'l refs in [8]). Together, they form the neural substrates of consciousness.

The network of unpaired electrons carried by transition metal ions, O₂ and/or NO caged-in/bound/absorbed to large molecules in neural membranes and proteins are modulated by action potentials through intra and inter-molecular exchange coupling and dipolar coupling and perturbed by microscopically strong and fluctuating internal magnetic fields produced largely by paramagnetic O₂ [19-20]. Further, all unpaired electron spins in neural membranes and proteins are also modulated by action potentials through relativistic effect of moving spin in strong electric field [20, 22] and, in turn, said modulations influence the electron spins in the brain, thus synchronizing their collective dynamics to the neural firings [19-20].

Because the high mobility of the electrons and strong interactions of electron spins with their environments, electron spins have very short relaxation time after excitations [See, e.g., [14]]. This property of electron spins seems to be a major problem for them to serve as the mind-pixels. Paradoxically, the interactions of the neural electron spin networks with their noisy brain environments may enhance quantum coherence through quantum Zeno effect which prevents a quantum system to evolve/decohere through repeated collisions with their environments (See, e.g., [54]). Further, studies show that decoherence-free subspaces can exist within the Hilbert space of a complex quantum system (See, e.g., [33-34]).

3. Action Potential Modulations of Nuclear Spins & Unpaired Electron Spins²

We have illustrated in [1] action potential modulations of two intra- or inter-molecular nuclear-spin system **I**₁ and **I**₂ inside neural membranes or proteins by the following heuristic Hamiltonian:

$$\hat{H} = -\hbar\gamma_1\hat{\mathbf{I}}_1 \cdot (\mathbf{1} - \boldsymbol{\sigma}_{1R} - \boldsymbol{\sigma}_{1A}) \cdot (\mathbf{B}_{1i} + \mathbf{B}_{1e} + \mathbf{B}'_1) - \hbar\gamma_2\hat{\mathbf{I}}_2 \cdot (\mathbf{1} - \boldsymbol{\sigma}_{2R} - \boldsymbol{\sigma}_{2A}) \cdot (\mathbf{B}_{2i} + \mathbf{B}_{2e} + \mathbf{B}'_2) + h\hat{\mathbf{I}}_1 \cdot (\mathbf{J}_R + \mathbf{J}_A) \cdot \hat{\mathbf{I}}_2 + h\hat{\mathbf{I}}_1 \cdot (\mathbf{D}_R + \mathbf{D}_A) \cdot \hat{\mathbf{I}}_2 \quad (1)$$

where **B**_{1i}, **B**_{1e}, **B**'₁, **B**_{2i}, **B**_{2e} and **B**'₂ are respectively the internal, external and relativistic-effect magnetic fields at the locations of first and second spins, γ_1 and γ_2 are respectively the gyromagnetic ratios of the said first and second spins, and other terms are explained below.

² See Note 3 for discussions on relativistic effect in electric field, fluctuating magnetic field & action potential modulation.

In the above equation (1), the two nuclear spins are coupled to each other through J-coupling tensor (including “through space” J-coupling, see, e.g., [39]) $\mathbf{J} = \mathbf{J}_R + \mathbf{J}_A$, where \mathbf{J}_R is for resting potential and \mathbf{J}_A accounts for contribution from action potential modulation, and dipolar coupling tensor $\mathbf{D} = \mathbf{D}_R + \mathbf{D}_A$, where \mathbf{D}_R is for resting potential and \mathbf{D}_A accounts for contribution from action potential modulation [19]. Second, chemical shielding tensor of each nuclear spin which also contains contribution from action potential modulation of its surrounding covalent bonds are taken into account - For the first spin $\boldsymbol{\sigma}_1 = \boldsymbol{\sigma}_{1R} + \boldsymbol{\sigma}_{1A}$ and for the second spin $\boldsymbol{\sigma}_2 = \boldsymbol{\sigma}_{2R} + \boldsymbol{\sigma}_{2A}$ where $\boldsymbol{\sigma}_{1R}$ and $\boldsymbol{\sigma}_{2R}$ are the chemical shielding tensors at resting potential and $\boldsymbol{\sigma}_{1A}$ and $\boldsymbol{\sigma}_{2A}$ are the first-order contribution to $\boldsymbol{\sigma}_1$ and $\boldsymbol{\sigma}_2$ respectively from action potential modulations. Third, the effects of internal magnetic field \mathbf{B}_i from other spins, external magnetic field \mathbf{B}_e and the magnetic field \mathbf{B}' due to relativistic effect on moving nuclear spin in the electric field of action potentials are also taken into accounts [20].

In the above equation (6), \mathbf{J}_A , \mathbf{D}_A , $\boldsymbol{\sigma}_{1A}$, $\boldsymbol{\sigma}_{2A}$, \mathbf{B}_{1i} , \mathbf{B}'_1 , \mathbf{B}_{2i} and \mathbf{B}'_2 are all functions of membrane voltage V_m which is driven by action potentials. Thus, the above Hamiltonian of interaction allows the action potentials to modulate nuclear spin dynamics through J-coupling, dipolar coupling, chemical shift, internal magnetic field and relativistic effect of moving spin in electric field of the action potential. Importantly, the spin-spin interaction in the above Hamiltonian causes the two nuclear spins in the two-spin system to form entangled quantum states known as Bell States.

We have further illustrated in [1] action potential modulations of one nuclear-spin system \mathbf{I} and one unpaired electron spin \mathbf{S} inside neural membranes or proteins by the following heuristic Hamiltonian:

$$\hat{H} = -\hbar\gamma_I \hat{\mathbf{I}} \cdot (\mathbf{1} - \boldsymbol{\sigma}_R - \boldsymbol{\sigma}_A) \cdot (\mathbf{B}_{1i} + \mathbf{B}_{1e} + \mathbf{B}'_1) + \hbar\gamma_S \hat{\mathbf{S}} \cdot (\mathbf{B}_{2i} + \mathbf{B}_{2e} + \mathbf{B}'_2) + \hbar \hat{\mathbf{I}} \cdot (\mathbf{A}_R + \mathbf{A}_A) \cdot \hat{\mathbf{S}} \quad (2)$$

where \mathbf{B}_{1i} , \mathbf{B}_{1e} , \mathbf{B}'_1 , \mathbf{B}_{2i} , \mathbf{B}_{2e} and \mathbf{B}'_2 are respectively the internal, external and relativistic-effect magnetic fields at the locations of nuclear-spin system \mathbf{I} and unpaired electron spin \mathbf{S} , γ_I and γ_S are respectively the gyromagnetic ratios of the said nuclear spin and unpaired electron spin, and other terms are explained below.

In the above equation (2), the nuclear spin and unpaired electron spin are coupled to each other through dipolar coupling tensor $\mathbf{A} = \mathbf{A}_R + \mathbf{A}_A$, where \mathbf{A}_r is for resting potential and \mathbf{A}_a accounts for

contribution from action potential modulation. Second, chemical shielding tensor σ of the nuclear spin which also contains contribution from action potential modulation of its surrounding covalent bonds are taken into account, That is, $\sigma = \sigma_R + \sigma_A$ where σ_A is the first-order contribution to σ from action potential modulations. Third, the effects of internal magnetic field magnetic field \mathbf{B}_i from other spins, external magnetic field \mathbf{B}_e and the magnetic field \mathbf{B}' due to relativistic effect of moving spin in the electric field of action potential are taken into accounts.

In equation (2), \mathbf{A}_a , σ_A , \mathbf{B}_{li} , \mathbf{B}'_I , \mathbf{B}_{Si} and \mathbf{B}'_S are all functions of membrane voltage V_m which is driven by action potential. The above Hamiltonian of interactions allows unpaired electron spin \mathbf{S} of O_2 and/or NO to activate/polarize/interact with nuclear spin \mathbf{I} and the action potentials to modulate nuclear-electronic two-spin system dynamics through dipolar coupling, chemical shift, internal magnetic field and relativistic effect of moving spin in electric field of the action potential. Importantly, the nuclear spin and electron spin interaction in the above Hamiltonian cause the two spins to form entangled quantum states known as Bell States.

We now illustrate action potential modulations of two intra- or inter-molecular unpaired electron spin system \mathbf{S}_1 and \mathbf{S}_2 inside neural membranes or proteins by the following heuristic Hamiltonian:

$$\hat{H} = \hbar\gamma_1\hat{\mathbf{S}}_1 \cdot (\mathbf{B}_{li} + \mathbf{B}_{1e} + \mathbf{B}'_1) + \hbar\gamma_2\hat{\mathbf{S}}_2 \cdot (\mathbf{B}_{2i} + \mathbf{B}_{2e} + \mathbf{B}'_2) + h\hat{\mathbf{S}}_1 \cdot (\mathbf{J}_R + \mathbf{J}_A) \cdot \hat{\mathbf{S}}_2 + h\hat{\mathbf{S}}_1 \cdot (\mathbf{D}_R + \mathbf{D}_A) \cdot \hat{\mathbf{S}}_2 \quad (3)$$

where \mathbf{B}_{li} , \mathbf{B}_{1e} , \mathbf{B}'_1 , \mathbf{B}_{2i} , \mathbf{B}_{2e} and \mathbf{B}'_2 are respectively the internal, external and relativistic-effect magnetic fields at the locations of first and second spins, γ_1 and γ_2 are respectively the gyromagnetic ratios of the said first and second spins, and other terms are explained below.

In the above equation (3), the two unpaired electron spins are coupled to each other through exchange tensor $\mathbf{J} = \mathbf{J}_R + \mathbf{J}_A$, where \mathbf{J}_R is for resting potential and \mathbf{J}_A accounts for contribution from action potential modulation, dipolar coupling tensor $\mathbf{D} = \mathbf{D}_R + \mathbf{D}_A$, where \mathbf{D}_R is for resting potential and \mathbf{D}_A accounts for contribution from action potential modulation, and g-factor anisotropies of both unpaired electron spins are neglected. Second, the effects of internal magnetic field \mathbf{B}_i from other spins, external magnetic field \mathbf{B}_e and the magnetic field \mathbf{B}' due to relativistic effect on moving electron spin in the electric field of action potentials are also taken into accounts (See , e.g. [14]).

4. Mechanism of Anesthetic Action in the Framework of Spin-mediated Consciousness Theory

We describe here mechanism of anesthetic action in the framework of the spin-mediated theory [8, also see, 19-20, 22].

Figure 1(a) schematically shows the normal diffusion of O₂ and NO without anesthetics dissolved into the neural membranes and proteins. As these molecules rapidly diffuse through the membranes, they interact with the neural membrane components and generate strong and fluctuating internal magnetic fields which are modulated by action potentials. Figure 1(b) schematically shows anesthetic perturbations of O₂ and NO pathways and *neural membranes themselves* by anesthetic molecules and xenon atoms and the resulting distortion and/or obstruction of these pathways and *neural membranes themselves*. Such perturbations render O₂ and NO and/or *neural membranes themselves* not able to perform their normal functions thus resulting in unconsciousness.

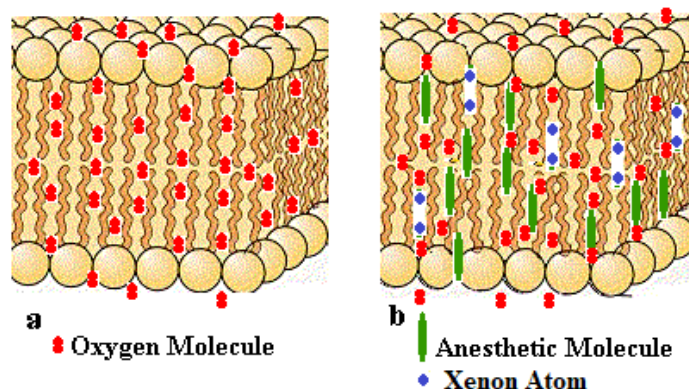


Figure 3. Illustration of anesthetic action. **a** shows the normal diffusion of O₂ without anesthetics dissolved into neural membranes. **b** shows xenon and anesthetic molecule perturbations of O₂ pathways and *neural membranes themselves*.

Further, as illustrated in Figure 2 below, anesthetic molecules and xenon atoms perturb not only O₂ and NO pathways and *neural membranes themselves* but also its lateral movement within the membrane and the movement within a hydrophobic pocket of the protein.

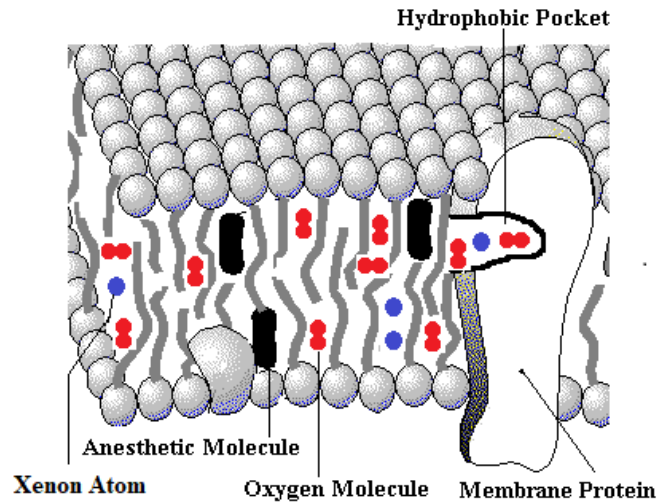


Figure 2. Perturbation of O₂ and NO pathways and *neural membranes themselves* in membranes and proteins by anesthetic molecules and xenon atoms. In the framework of spin-mediated consciousness theory, anesthetic molecules and xenon atoms block, dislocate, distort or otherwise interfere with O₂ and/or NO pathways and neural membranes and proteins.

(1) Spin-mediated consciousness theory of 2002

In the spin-mediated consciousness theory put forward in 2002, general anesthetics such as xenon produce anesthesia by perturbing O₂ and/or NO pathways in neural membranes and proteins thus blocking and/or distorting their activation functions in consciousness. Naturally, the nuclear spins of xenon 131 and xenon 129 may partially play the activating roles of displaced O₂ and/or NO [1], thus attenuate the anesthetic potency of nuclear-spin-carrying xenon isotopes as observed by Li *et. al.* [11].

Using a system of one nuclear-spin **I** and one (or two) unpaired electron spin **S** inside neural membranes or proteins under the action potential modulation as an illustration, one may write a heuristic Hamiltonian for the two-spin system as [1]:

$$\hat{H} = -\hbar\gamma_I \hat{\mathbf{I}} \cdot (\mathbf{1} - \boldsymbol{\sigma}_R - \boldsymbol{\sigma}_A) \cdot (\mathbf{B}_{li} + \mathbf{B}_{le} + \mathbf{B}'_l) + \hbar\gamma_S \hat{\mathbf{S}} \cdot (\mathbf{B}_{si} + \mathbf{B}_{se} + \mathbf{B}'_s) + \hbar \hat{\mathbf{I}} \cdot (\mathbf{A}_R + \mathbf{A}_A) \cdot \hat{\mathbf{S}} \quad (4)$$

After the unpaired-electron-carrying molecule such as O₂ or NO is displaced by an anesthetic such as xenon 132 or xenon 134 which has no unpaired electron spin and nuclear spin, the Hamiltonian becomes:

$$\hat{H} = -\hbar\gamma_I \hat{\mathbf{I}} \cdot (\mathbf{1} - \boldsymbol{\sigma}_R - \boldsymbol{\sigma}_A) \cdot (\mathbf{B}_{li} + \mathbf{B}_{le} + \mathbf{B}'_I) \quad (5)$$

which contains no interaction between nuclear spin **I** and the activating agent O₂ or NO.

On the other hand, if the unpaired-electron-carrying molecule such as O₂ or NO is displaced by an anesthetic such as xenon 129 or xenon 131 which has no unpaired electron spin but has nuclear spin **I**₂, the Hamiltonian (9) becomes:

$$\begin{aligned} \hat{H} = & -\hbar\gamma_I \hat{\mathbf{I}} \cdot (\mathbf{1} - \boldsymbol{\sigma}_R - \boldsymbol{\sigma}_A) \cdot (\mathbf{B}_{li} + \mathbf{B}_{le} + \mathbf{B}'_I) - \hbar\gamma_2 \hat{\mathbf{I}}_2 \cdot (\mathbf{1} - \boldsymbol{\sigma}_{2R} - \boldsymbol{\sigma}_{2A}) \cdot (\mathbf{B}_{2i} + \mathbf{B}_{2e} + \mathbf{B}'_2) + \\ & \hbar \hat{\mathbf{I}} \cdot (\mathbf{J}_R + \mathbf{J}_A) \cdot \hat{\mathbf{I}}_2 + \hbar \hat{\mathbf{I}} \cdot (\mathbf{D}_R + \mathbf{D}_A) \cdot \hat{\mathbf{I}}_2 \end{aligned} \quad (6)$$

which contains both intermolecular (“through space”) J-coupling (See, e.g., [39]) and dipolar coupling between nuclear spin **I** and xenon 129 or xenon 131’s nuclear spin **I**₂ modulated by action potentials – These modulated couplings may then attenuate the anesthetic potency of nuclear-spin-carrying xenon 129 and xenon 131 as observed by Li *et. al.* [11].

(2) Modified spin-mediated consciousness theory

In this scenario, general anesthetics such as xenon may produce anesthesia by direct perturbations and/or distortions of neural spin networks alone or in addition to perturbing and/or distorting O₂ and/or NO pathways in neural membranes and proteins in (1) above.

Using a system of two intra- or inter-molecular nuclear-spin system **I**₁ and **I**₂ inside neural membranes or proteins under the action potential modulation as an illustration, one may write a heuristic Hamiltonian for the two-spin system as:

$$\begin{aligned} \hat{H} = & -\hbar\gamma_1 \hat{\mathbf{I}}_1 \cdot (\mathbf{1} - \boldsymbol{\sigma}_{1R} - \boldsymbol{\sigma}_{1A}) \cdot (\mathbf{B}_{1i} + \mathbf{B}_{1e} + \mathbf{B}'_1) - \hbar\gamma_2 \hat{\mathbf{I}}_2 \cdot (\mathbf{1} - \boldsymbol{\sigma}_{2R} - \boldsymbol{\sigma}_{2A}) \cdot (\mathbf{B}_{2i} + \mathbf{B}_{2e} + \mathbf{B}'_2) + \\ & \hbar \hat{\mathbf{I}}_1 \cdot (\mathbf{J}_R + \mathbf{J}_A) \cdot \hat{\mathbf{I}}_2 + \hbar \hat{\mathbf{I}}_1 \cdot (\mathbf{D}_R + \mathbf{D}_A) \cdot \hat{\mathbf{I}}_2 \end{aligned} \quad (7)$$

After one of the nuclear spin, e.g., \mathbf{I}_2 is displaced by an anesthetic such as xenon 132 or xenon 134 which has no unpaired electron spin and nuclear spin, the Hamiltonian becomes:

$$\hat{H} = -\hbar\gamma_1\hat{\mathbf{I}}_1 \cdot (\mathbf{1} - \boldsymbol{\sigma}_{1R} - \boldsymbol{\sigma}_{1A}) \cdot (\mathbf{B}_{1i} + \mathbf{B}_{1e} + \mathbf{B}'_1) \quad (8)$$

which contains no interaction between nuclear spin \mathbf{I}_1 and \mathbf{I}_2 .

On the other hand, if \mathbf{I}_2 is displaced by an anesthetic such as xenon 129 or xenon 131 which has no unpaired electron spin but has nuclear spin \mathbf{I}_3 , the Hamiltonian (x) becomes:

$$\begin{aligned} \hat{H} = & -\hbar\gamma_1\hat{\mathbf{I}}_1 \cdot (\mathbf{1} - \boldsymbol{\sigma}_{1R} - \boldsymbol{\sigma}_{1A}) \cdot (\mathbf{B}_{1i} + \mathbf{B}_{1e} + \mathbf{B}'_1) - \hbar\gamma_3\hat{\mathbf{I}}_3 \cdot (\mathbf{1} - \boldsymbol{\sigma}_{3R} - \boldsymbol{\sigma}_{3A}) \cdot (\mathbf{B}_{3i} + \mathbf{B}_{3e} + \mathbf{B}'_3) + \\ & h\hat{\mathbf{I}}_1 \cdot (\mathbf{J}_R + \mathbf{J}_A) \cdot \hat{\mathbf{I}}_3 + h\hat{\mathbf{I}}_1 \cdot (\mathbf{D}_R + \mathbf{D}_A) \cdot \hat{\mathbf{I}}_3 \end{aligned} \quad (9)$$

which contains both intermolecular (“through space”) J-coupling (See, e.g., [39]) and dipolar coupling between nuclear spin \mathbf{I} and xenon 129 or xenon 131’s nuclear spin \mathbf{I}_3 modulated by action potentials – These modulated couplings may then attenuate the anesthetic potency of nuclear-spin-carrying xenon 129 and xenon 131 as observed by Li *et. al.* [11].

(3) Electron-spin-mediated consciousness theory

In this scenario, the mind-pixels are comprised of unpaired electrons carried by transition metal ions, O_2 and/or NO caged-in/bound/absorbed to large molecules in neural membranes and proteins. General anesthetics such as xenon may produce anesthesia by perturbing free/unbound O_2 and/or NO pathways in neural membranes and proteins and/or unpaired electron spin networks thus blocking and/or distorting their functions in consciousness.

Using a system of two intra- or inter-molecular unpaired electron spin system \mathbf{S}_1 and \mathbf{S}_2 inside neural membranes or proteins under the action potential modulation as an illustration, one may write a heuristic Hamiltonian for the two-spin system as:

$$\begin{aligned} \hat{H} = & \hbar\gamma_1\hat{\mathbf{S}}_1 \cdot (\mathbf{B}_{1i} + \mathbf{B}_{1e} + \mathbf{B}'_1) + \hbar\gamma_2\hat{\mathbf{S}}_2 \cdot (\mathbf{B}_{2i} + \mathbf{B}_{2e} + \mathbf{B}'_2) + \\ & h\hat{\mathbf{S}}_1 \cdot (\mathbf{J}_R + \mathbf{J}_A) \cdot \hat{\mathbf{S}}_2 + h\hat{\mathbf{S}}_1 \cdot (\mathbf{D}_R + \mathbf{D}_A) \cdot \hat{\mathbf{S}}_2 \end{aligned} \quad (10)$$

After one of the electron spin, e.g., \mathbf{S}_2 is displaced by an anesthetic such as xenon 132 or xenon 134 which has no unpaired electron spin and nuclear spin, the Hamiltonian becomes:

$$\hat{H} = \hbar\gamma_1\hat{\mathbf{S}}_1 \cdot (\mathbf{B}_{1i} + \mathbf{B}_{1e} + \mathbf{B}'_1) \quad (11)$$

which contains no interaction between unpaired electron spin \mathbf{S}_1 and \mathbf{S}_2 .

On the other hand, if \mathbf{S}_2 is displaced by an anesthetic such as xenon 129 or xenon 131 which has no unpaired electron spin but has nuclear spin \mathbf{I} , the Hamiltonian (\hat{H}) becomes:

$$\hat{H} = \hbar\gamma_1 \hat{\mathbf{S}}_1 \cdot (\mathbf{B}_{li} + \mathbf{B}_{le} + \mathbf{B}'_1) - \hbar\gamma_I \hat{\mathbf{I}} \cdot (\mathbf{1} - \boldsymbol{\sigma}_R - \boldsymbol{\sigma}_A) \cdot (\mathbf{B}_{li} + \mathbf{B}_{le} + \mathbf{B}'_1) + \hbar \hat{\mathbf{S}}_1 \cdot (\mathbf{A}_R + \mathbf{A}_A) \cdot \hat{\mathbf{I}} \quad (12)$$

which contains dipolar coupling between electron spin \mathbf{S}_1 and xenon 129 or xenon 131's nuclear spin \mathbf{I} modulated by action potentials – These modulated couplings may then attenuate the anesthetic potency of nuclear-spin-carrying xenon 129 and xenon 131 as observed by Li *et. al.* [11].

5. Conclusions³

In this paper, we have discussed two other plausible mechanisms of anesthetic action within the framework of spin-mediated consciousness theory [8, 19-20, 22] in light of the recent experimental findings of Li, *et. al.* [11].

In one scenario, general anesthetics such as xenon may produce anesthesia by direct perturbations and/or distortions of neural spin networks alone or in addition to perturbing and/or distorting O₂ and/or NO pathways in neural membranes and proteins.

In another scenario, the mind-pixels are comprised of unpaired electrons carried by transition metal ions, O₂ and/or NO caged-in/bound/absorbed to large molecules in neural membranes and proteins (See add'l refs. in [8]). In the latter, general anesthetics such as xenon may produce anesthesia by perturbing free/unbound O₂ and/or NO pathways in neural membranes and proteins and/or unpaired electron spin networks thus blocking and/or distorting their functions in consciousness.

In either of the above scenarios, the nuclear spins of xenon 131 and xenon 129 may partially play the roles of displaced nuclear spins or unpaired electron spins, thus attenuate the anesthetic potency of nuclear-spin-carrying xenon isotopes as observed by Li *et. al.* [11].

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³ See Note 4 for discussions on the Experimental Results by Li *et. al.* [11].

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Note 1. Some background information

Since both O₂ and general anesthetics are hydrophobic, we proposed in 2001 within the framework of conventional neuroscience that general anesthetic may cause unconsciousness by perturbing O₂ pathway in neural membranes and O₂-utilizing proteins, such that the availability of O₂ to its sites of utilization is reduced, which in turn triggers cascading cellular responses through O₂-sensing mechanisms, resulting in general anesthesia [7]. In the spin-mediated consciousness theory of 2002 [8], we hypothesized that anesthetic perturbations of oxygen pathways in neural membranes and proteins themselves are the direct cause of unconsciousness within the framework of quantum biology/brain. Indeed, the low affinity, diversity and pervasiveness of general anesthetics point to this direction [8].

Since spin-mediated consciousness theory was put forth in 2002 [8], the idea that nuclear spins and/or unpaired electron spins in the brain may play some roles in consciousness has gains traction recently (See, e.g., [9-13]). Importantly, Li, *et.al.* have found experimentally that nuclear spins of xenon isotopes, xenon 131 and xenon 129, attenuate their anesthetic potency in mice [11]. The authors therein suggest that "the quantum property of nuclear spin in the monoatomic anesthetic xenon promotes conscious processes at the xenon site of action, consistent with theories proposing quantum mechanisms in consciousness" and speculate that quantum entanglement of nuclear spins may be involved in consciousness [11]. However, for whatever reason (hopefully benign such as oversight), the authors therein did not cite or mention [8], which was the first, to the best knowledge of the herein first author, to propose nuclear/electron spin mediated/based consciousness theory back in 2002.

Note 2. Brief comparisons of oxygen, nitric oxide, nitrous oxide & xenon

Each O₂ contains two unpaired valence electrons thus is strongly paramagnetic and at the same time chemically reactive as a bi-radical [8]. It is capable of producing a large fluctuating magnetic field along its diffusing pathway thus serves as a natural contrast agent in MRI (See, e.g., [14]). The existence of unpaired electrons in stable molecules is very rare indeed. O₂ are the only paramagnetic specie to be found in large quantities in the brain besides enzyme-produced nitric oxide (NO) [8]. In addition, O₂ is an essential component for energy production in the central nervous system.

O₂ and NO are hydrophobic small molecules so their concentrations in neural membranes are much higher than in aqueous solutions such as cytoplasm (See, e.g., [15]). Both O₂ and NO, the latter being an unstable free radical with one unpaired electron and a small neural transmitter (See, e.g., [21]), are well known in spin chemistry - a field focused on the study of free-radical mediated chemical reactions where very small magnetic energies can change non-equilibrium spin conversion process (See, e.g., [16]).

In contrast, nitrous oxide (N₂O) and xenon (Xe) contain no unpaired electrons and are general anesthetics. They are also hydrophobic small molecules so their concentrations in neural membranes are much higher than in aqueous solutions such as cytoplasm (See, e.g., [8]). Xenon has 9 stable isotopes among which xenon 129 has a nuclear spin of 1/2, xenon 131 has nuclear spin of 3/2, and the other seven isotopes have no nuclear spin (See, e.g., [11]).

Note 3. Relativistic Effect in Electric Field, Fluctuating Magnetic Field & Action Potential Modulation

The range of electric field strength E_m inside the neural membranes during a typical action potential as calculated from $E_m = V_m/d$ where V_m and d are respectively the membrane voltage and thickness is shown in Figure 1. It oscillates between -9 to +6 million volts per meter during the course of each action potential [19-20]. These strengths are comparable to those causing electroporation of cell membranes and dielectric breakdown of many materials at which the covalent bonds of the constituent molecules are torn apart (See, *e.g.*, [35]). So it significantly affects the conformations and collective dynamics of the neural membrane components such as phospholipids, cholesterol and proteins. Indeed, voltage-dependent ion channels perform their functions through electric field induced conformation changes of the constituent protein (See, *e.g.*, [36]) and studies on the effects of electric fields on lipids support the above conclusion (See, *e.g.*, [37-38]).

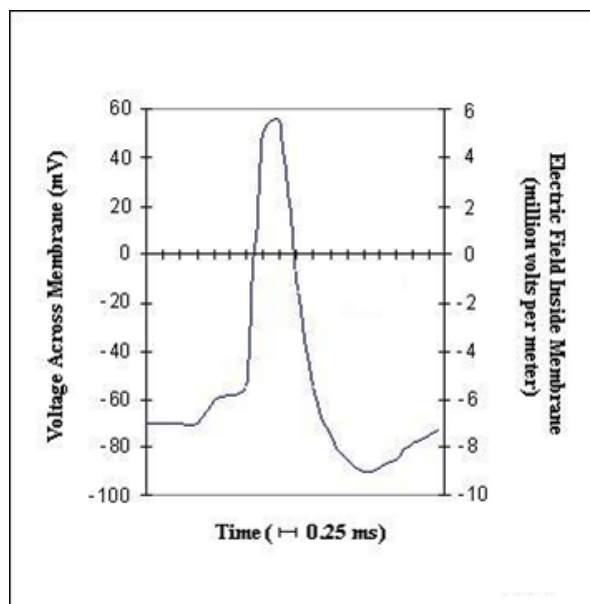


Figure 1. Electric field strength inside neural membrane during the course of an action potential. The calculation is done by assuming a typical membrane thickness of about 10 nm and the results are shown in the unit of one million volts per meter with “-” and “+” indicating that the direction of electric field is respectively pointing outward or inward inside the neural membrane.

The nuclear spins carried by the nuclei such as ^1H , ^{13}C and ^{31}P inside the neural membranes and proteins form complex intra- and inter-molecular spin networks through various intra-molecular J- and dipolar couplings and both short- and long-range intermolecular dipolar couplings [19] and even through-space J-couplings (See, *e.g.* [39]). Since J-coupling is the indirect interaction between two nuclear spins through covalent bonds and dipolar coupling is the direct interaction of two nuclear spins through space, their strengths and anisotropies strongly depend on the conformations of the neural membrane components (See,

e.g., [40-41]). Further, the chemical shielding of each nuclear spin also depends on the conformations of surrounding covalent bonds (See, *e.g.*, [42]). Thus, when these spin networks are subjected to the enormous changing electric field produced during each action potential, the J-coupling, dipolar coupling and chemical shielding tensors oscillate with it [19]. Studies on the effects of electric fields on these tensors (See, *e.g.*, [40-42]) also support this conclusion.

Further, nuclear and electron spins interact with electric field through relativistic effect due to their motion in electric field [20, 22] – This is also the cause of spin-orbital couplings and can be vigorously derived from Dirac equation as will be shown in a separate paper. The motion producing the relativistic effect of moving nuclear spin or electron spin seeing/experiencing a magnetic field in its rest frame includes molecule motion such as rotation, electron orbital motion in an atom, rotation of multi-nucleon nucleus and motion a sub-nuclear particle such as quark inside a nucleon (proton or neutron) as illustrated below.

In special relativity, a moving particle in an electric field E sees a magnetic field B' in its own rest frame (See, *e.g.*, [43]):

$$\mathbf{B}' = \frac{\mathbf{E} \times \mathbf{v} / c^2}{\sqrt{1 - v^2/c^2}} \approx \frac{\mathbf{E} \times \mathbf{v}}{c^2} \quad (1)$$

However, due to Thomas precession [44], the magnetic field B' seen by the nuclear spin I is:

$$\mathbf{B}' \approx \frac{\mathbf{E} \times \mathbf{v}}{2c^2} \quad (2)$$

Thus, E exerts a torque (twisting force) on moving proton spin I at speed v as illustrated below in Figure 2:

$$\mathbf{f} = \mathbf{m}_I \times \mathbf{B}' = g_I \mu_I \mathbf{I} \times \frac{\mathbf{E} \times \mathbf{v}}{2c^2} \quad (3)$$

Similarly, E exerts a torque (twisting force) on moving electron spin S at speed v as illustrated in Figure 2:

$$\mathbf{f} = \mathbf{m}_e \times \mathbf{B}' = -g_e \mu_e \mathbf{S} \times \frac{\mathbf{E} \times \mathbf{v}}{2c^2} \quad (4)$$

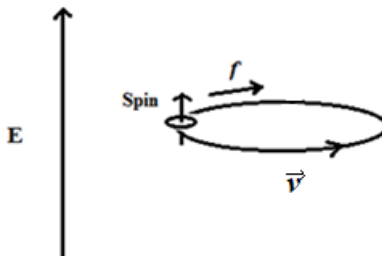


Figure 2. Illustration of spin transverse torque/force f exerted on moving nuclear spin inside the neural membranes and proteins due to molecular rotation, nucleus rotation or

sub-nucleon motion, or moving unpaired electron spin inside the neural membranes and proteins due to molecular rotation or atomic electron orbital motion.

Therefore, the interactions between the moving nuclear/electronic spins in neural membranes and proteins and the varying high-voltage electric fields inside neural membranes and proteins driven by action potentials are capable of directly feeding information into the neurons [20].

We now estimate the strengths of B' seen by nuclear spin and electron spin carried by various molecules in neural membranes and proteins using the formula:

$$B' = Ev/(2c^2) \quad (5)$$

and the estimated $E \sim 9 \times 10^6 \text{ V/m}$ as the maximal electric field strength in neural membranes or proteins of a thickness/length $\sim 10 \times 10^{-9} \text{ m}$.

**(1) Strength of B' seen in the rest frame of rotating lipid molecule
by a nuclear-spin-carrying proton/hydrogen**

For a lipid molecule with a diameter $\sim 0.9 \times 10^{-9} \text{ m}$ and rotation frequency along the chain $\sim 1 \times 10^7/\text{s}$ (See, e.g., [45]), the estimated molecular rotation speed of a spin $\frac{1}{2}$ proton/hydrogen on the lipid molecule is $2 \times 3.14 \times 0.45 \times 10^{-9} \text{ m} \times 1 \times 10^7/\text{s} = 0.0283 \text{ m/s}$.

Thus, B' seen on lipid molecule rest frame by a proton/hydrogen atom due to rotation along the chain is $\sim (9 \times 10^6 \text{ V/m} \times 0.0283 \text{ m/s}) / (2 \times (2.99 \times 10^8 \text{ m/s})^2) = 7.122 \times 10^{-13} \text{ Vs/m}^2$ (Tesla), that is, 0.7122 pico-Tesla.

**(2) Strength of B' seen in the rest frame of rotating nucleus of multiple nucleons
such as deuteron, C^{13} , P^{31} and Xe^{129} by a nuclear-spin-carrying proton/neutron**

For a nucleus of multiple nucleons with diameter $\sim 4.2 \times 10^{-15} \text{ m}$ (e.g., deuteron) and rotation frequency $\sim 4.74 \times 10^{21}/\text{s}$ (See, e.g., [46]), the estimated rotation speed of the nucleus is $\sim 2 \times 3.14 \times 2.1 \times 10^{-15} \text{ m} \times 4.74 \times 10^{21}/\text{s} = 58.95 \times 10^6 \text{ m/s}$.

Thus, B' seen in the nucleus rest frame by a spin $\frac{1}{2}$ nucleon (proton or neutron) due to rotation of nucleus of multiple nucleons is $\sim (9 \times 10^6 \text{ V/m} \times 58.95 \times 10^6 \text{ m/s}) / (2 \times (2.99 \times 10^8 \text{ m/s})^2) = 14.836 \times 10^{-4} \text{ Vs/m}^2$ (Tesla), that is, 14.836 Gauss or 1.484 milli-Tesla.

**(3) Strength of B' seen in the rest frame of rotating sub-nucleon particle
such as a spin-carrying quark inside a nucleon**

For a nucleon with rotating sub-nuclear particle with orbital diameter $\sim 0.8 \times 10^{-15} \text{ m}$ (e.g., the size of a proton) and rotation frequency $\sim 4.74 \times 10^{21}/\text{s}$ (a pure guess based on [46]), the estimated orbital speed of a sub-nuclear particle inside a nucleon is $\sim 2 \times 3.14 \times 0.4 \times 10^{-15} \text{ m} \times 4.74 \times 10^{21}/\text{s} = 11.907 \times 10^6 \text{ m/s}$.

Thus, B' seen in the sub-nucleon rest frame by a spin $\frac{1}{2}$ sub-nucleon particle due to sub-nucleon rotation is $\sim (9 \times 10^6 \text{ V/m} \times 11.907 \times 10^6 \text{ m/s}) / (2 \times (2.99 \times 10^8 \text{ m/s})^2) = 2.997 \times 10^{-4} \text{ Vs/m}^2$ (Tesla), that is, 2.997 Gauss or 0.300 milli-Tesla.

(4) Strength of B' seen in the rest frame of rotating oxygen molecule by an unpaired electron spin

For an oxygen molecule with size/diameter $\sim 3.46 \times 10^{-11} \text{ m}$ and rotation frequency $\sim 1.07 \times 10^{12} / \text{s}$ (See, e.g., [47]), the estimated rotation speed of a spin-carrying unpaired electron on the oxygen molecule in neural membranes or proteins is $2 \times 3.14 \times 1.73 \times 10^{-11} \text{ m} \times 1.07 \times 10^{12} / \text{s} = 1.162 \times 10^2 \text{ m/s}$.

Thus, B' seen in the rest frame of oxygen molecule by a spin-carrying unpaired electron due to rotation of oxygen molecule is $\sim (9 \times 10^6 \text{ V/m} \times 1.162 \times 10^2 \text{ m/s}) / (2 \times (2.99 \times 10^8 \text{ m/s})^2) = 2.924 \times 10^{-9} \text{ Vs/m}^2$ (Tesla), that is, 2.924 nano-Tesla.

(5) Strength of B' seen in the rest frame of electron orbital in oxygen molecule by an unpaired electron spin

The estimated electron orbital speed in a Bohr atom is $\sim c/137$ (See, e.g., [48]). Using this speed as an estimate of the electron orbital speed in oxygen molecule, B' seen in the rest frame of electron orbital by a spin-carrying unpaired electron due to electron orbital motion is $\sim (9 \times 10^6 \text{ V/m} \times (1/137 \times 2.99 \times 10^8 \text{ m/s})) / (2 \times (2.99 \times 10^8 \text{ m/s})^2) = 54.93 \times 10^{-6} \text{ Vs/m}^2$ (Tesla), that is, 54.93 micro-Tesla or 0.549 Gauss.

The fluctuating internal magnetic fields are produced by the paramagnetic species such as O_2 and NO and spin-carrying nuclei themselves such as ^1H , ^{13}C and ^{31}P . Table 1 shows the maximal magnetic field strengths produced by the magnetic dipoles of the unpaired electrons of O_2 and NO and the nucleus of ^1H along the axes of said dipoles at given distances [19]. Because the magnetic dipole moment of an unpaired electron is 658 times larger than that of the ^1H nucleus, O_2 and NO can respectively produce magnetic fields 1,316 and 658 times larger than ^1H [19].

As distance r increases, the strength of the magnetic dipole field quickly attenuate according to

$$B_i = \frac{\mu_0 m}{4\pi r^3} \quad (8)$$

where μ_0 is the permeability of free space and m is the magnetic dipole moment. As mentioned earlier, O_2 and NO are hydrophobic small molecules so their concentrations in neural membranes are much higher than in aqueous solutions such as cytoplasm (See, e.g., [15]). As they rapidly tumble and diffuse, they produce microscopically strong and fluctuating magnetic fields. Indeed, O_2 are the predominant sources of internal magnetic fields in neural membranes as evidenced by the strong effect of O_2 on spin-spin and spin-lattice relaxation rates (See, e.g., [15, 18]).

Table 1. Magnetic Fields Produced by O₂, NO and ¹H

Distance (Å)	O ₂ (Tesla)	NO (Tesla)	¹ H (Tesla)
1.0	3.713940	1.856970	0.002821
2.0	0.464243	0.232122	0.000353
3.0	0.137553	0.068777	0.000104
4.0	0.058030	0.029015	0.000044
5.0	0.029712	0.014856	0.000023
10.0	0.003714	0.001857	0.000003

Again, these fluctuating internal magnetic fields are modulated by action potentials through relativistic effects on nuclear/electronic spins moving in electric field and continuously perturb the neural spin networks. The intensities of said perturbations depend on the concentrations of O₂ and NO that are highly regulated in the brain. Thus, these modulated perturbations not only activate various neural spin networks but also are likely capable of enhancing the synchronization of these dynamics to the neural spike trains through non-linear processes such as stochastic resonance that is known to occur in the brain (See, e.g., [49-51]).

The collective dynamics of the neural spin networks under modulations by action potentials and perturbations by fluctuating internal magnetic fields which are also modulated by action potentials represent meaningful information to the brain [19-20, 22]. An analogy is the mechanism of liquid crystal display (LCD) where information-carrying electric voltages applied to the pixel cells change the optical properties of the constituent molecules such that when lights pass through these cells their phases get rotated differently which in turn represent different information to the viewer of the LCD screen (See, e.g., [52]). Accordingly, the cause of unconsciousness by general anesthetics can be explained as the direct consequence of their effects the O₂ and/or NO pathways inside neural membranes and proteins and/or the neural membrane structures themselves [7-8, 19-20].

However, how can one explain that cognitive functions seem in general insensitive to environmental and even medical strength external magnetic fields such as those generated by the power lines and the ones used in MRI? It is argued that most of these disturbances do not represent meaningful information to the brain and, further, the brain likely have developed other mechanisms through evolution to counter the effects of external magnetic fields. In the cases where external magnetic disturbances were reported to have observable effects on cognition, the above discussions provide a basis for interpreting these effects as said disturbances contain meaningful information to the brain [7-8, 19-20].

Note 4. Discussions on the Experimental Results by Li *et. al.*

Recently, Li *et. al.* postulated that xenon isotopes might have different anesthetic potencies and experimentally tested the postulates in mice [11]. They found that “[t]he potency of two xenon isotopes with nuclear spin, xenon 129 and xenon 131, is less than the potency of two xenon isotopes, xenon 132 and xenon 134, that do not have nuclear spin; t]his difference in potency cannot be explained, either by differences in outer electron shells (there are none) or the variations in atomic mass “[11].

The conclusion by Li *et. al.* is that “[x]enon isotopes with nuclear spin are less potent than those without, and polarizability cannot account for the difference[; t]he lower anesthetic potency of ^{129}Xe may be the result of it participating in conscious processing and therefore partially antagonizing its own anesthetic potency” [11]. Li *et. al.* suggest that “the quantum property of nuclear spin in the monoatomic anesthetic xenon promotes conscious processes at the xenon site of action, consistent with theories proposing quantum mechanisms in consciousness” and speculate that quantum entanglement of nuclear spins may be involved in consciousness [11].

As shown and discussed in [1], spin-mediated consciousness theory [8, 19-20, 22] naturally explains the attenuation of the anesthetic potency of nuclear-spin-carrying xenon 129 and xenon 131 found by Li *et. al.* [11]. Therefore, the important results of Li *et. al.* [11], if replicable, provide direct and strong support to the spin-mediated consciousness theory put forward in 2002 [8] by the herein authors and its later developments [19-20, 22].

Li *et. al.* [11] did not provide a detailed mechanism of their own to explain their results but cites some work done recently on possible role of nuclear spin in consciousness or cognition in order to offer a plausible explanation. However, it is herein authors’ views that the work cited by Li *et. al.* on the subject may be largely without merits to be discussed in the future if the circumstances call for such discussions.

Among other plausible explanations of Li *et. al.*’s results [11], one is the different quantum behaviors of some xenon isotopes being fermions and some of them being bosons. Xenon 129 and xenon 131 are fermions but xenon 132 and xenon 134 are bosons (see, e.g., [53]). Thus, besides the difference in formation of quantum entanglement, their roles in spin chemistry and/or quantum dynamics of the brain, if not negligible, may be different.