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Original Article

Changes in the isoprenoid pathway with transcendental meditation and Reiki healing practices in seizure disorder

R. Kumar A, P. A. Kurup*

*Department of Neurology, Medical College Hospital, Trivandrum, Kerala, India; and *Department of Biochemistry, University of Kerala, Trivandrum, Kerala, India.*

A quantal perceptive model of brain function has been postulated by several groups. Reiki-like healing practices in seizure disorder (ILAE classification – II E – generalized seizures – tonic clonic), involving transfer of life force or low level of electromagnetic force (EMF) from the healer to the recipient patient, may act via quantal perceptive mechanisms. Increased synthesis of an endogenous membrane Na+-K+ ATPase inhibitor digoxin and a related tyrosine / tryptophan transport defect has been demonstrated in refractory seizure disorder (ILAE classification – II E – generalized seizures – tonic clonic). Reiki-like healing practices in refractory epilepsy results in a reduction in seizure frequency. Reiki-like healing practices produce membrane stabilization and stimulation of membrane Na+-K+ ATPase activity by quantal perception of low levels of EMF. The consequent intracellular hypermagnesemia inhibits HMG CoA reductase activity and digoxin synthesis resulting in the alteration of the neutral amino acid transport (tryptophan / tyrosine) defect. A hypothalamic digoxin-mediated quantal perception model of brain function is proposed. The phenomena of biological transmutation and consequent hypermagnesemia occurring in the resultant neuronal quantal state is also discussed.

Key Words: Reiki healing practices, Transcendental mediation, Seizure disorder, Isoprenoid, Na^+-K^+ATPase, Digoxin, Quantal perception.

Introduction

A quantal perception model for brain function and consciousness has been proposed by several groups of workers. The brain is hypothesized to function as a quantum computer.[1] Reiki-like healing practices involving transfer of life force or low level of EMF from the healer to the patient have been in use in patients with seizure disorders. The Reiki-like treatment practices, if effective, are hypothesized to act via quantal perception since the EMF is too weak to be transferred by normal sensory perceptive mechanisms. The present study was conducted to assess the efficacy of such treatment protocols in epileptic patients. The seizure frequency was used as the end point to assess the efficacy of the treatment. Previous reports have demonstrated an endogenous membrane Na^+-K^+ ATPase inhibition-related biochemical cascade in primary generalized epilepsy.[2-4] Elevated levels of an hypothalamic endogenous membrane Na^+-K^+ ATPase inhibitor digoxin have been reported in epilepsy. It was considered pertinent to study the changes in the membrane Na^+-K^+ ATPase inhibition cascade in seizure patients undergoing Reiki-like treatment practices. The results are discussed in this paper and a hypothalamic digoxin-mediated quantal perceptive model of brain function is proposed.

Material and Methods

Fifteen patients with refractory seizure disorder (ILAE classification – II E – generalized seizures – tonic clonic) (patients with persistent seizures, on 3 or more antiepileptic drugs in full dosage, and total compliance over a period of 3 years) were chosen for the study. They were chosen randomly from those attending the epilepsy clinic of the Department of Neurology, Medical College Hospital, Trivandrum. They were in the age group of 20-30 years. Eight of the patients were males and 7 of them were females. Patients with systemic diseases like hypertension, diabetes mellitus, cardiac, renal and hepatic diseases were excluded from the study. Thrice a week they underwent Reiki-like healing hand therapy, where the healer meditates, reaches a trance-like state and transfers his life force or low level of EMF by the touch of his hand to the patient. They also underwent daily one hour of transcendental meditation. They were clinically assessed with seizure frequency counts at the end of 3 months of therapy. The pre and post-therapy biochemical and clinical parameters were com-

P. A. Kurup
Gouri Sadan, T. C. 4/1525, North of Cliff House, Kattu Road, Kowdiar P.O., Trivandrum-695003, Kerala, India. E-mail: kvgnair@satyam.net.in

pared in the refractory epilepsy group. An equal number of age and sex matched healthy subjects served as controls for the pre-therapy refractory epilepsy group. The controls were chosen randomly from the general population of Trivandrum city. They were free from systemic diseases like hypertension, diabetes mellitus, cardiac, hepatic and renal diseases. They were not on any drug therapy for any disease. All patients and controls were on the same dietary regimen which gave adequate amounts of trace elements, especially magnesium throughout the course of the study. The following biochemical parameters were assessed at the start of the therapy and at the end of 3 months – plasma HMG CoA reductase, serum digoxin, serum magnesium and RBC membrane Na^+-K^+ ATPase activity. The serum levels of tyrosine, dopamine, noradrenaline, tryptophan, serotonin and quinolinic acid were also assessed. Fasting blood was taken from each of the patients for various estimations. RBCs were separated within 1 hour of the collection of the blood for the estimation of membrane Na^+-K^+ ATPase. Serum was used for the estimation of HMG CoA reductase activity. Plasma /serum was used for the estimation of the other parameters. All biochemicals used in this study were obtained from M/s Sigma Chemicals, USA. The activity of HMG CoA reductase of the plasma was determined using the method of Rao and Ramakrishnan, by determining the ratio of HMG CoA to mevalonate.[5] For the determination of the Na^+-K^+ ATPase activity of the erythrocyte membrane, the procedure described by Wallach and Kamat was used.[6] Digoxin in the plasma was determined by the HPLC procedure described by Arun et al.[7] Magnesium in the plasma was estimated by atomic absorption spectrophotometry.[8] Tryptophan was estimated by the method of Bloxam and Warren[9] and tyrosine by the method of Wong et al.[10] Serotonin was estimated by the method of Curzon et al[11] and catecholamines by the method of Well-Malherbe et al.[12] Quinolinic acid content of plasma was estimated by HPLC (C18 column micro BondaparkTM 4.6 x 150 mm), solvent system 0.01 M acetate buffer (pH 3.0) and methanol (6:4), flow rate 1.0 ml/minute and detection-UV 250 nm. Statistical analysis was done by the Students 't' test with modified degree of freedom.

Results

Pre-therapy the activity of HMG CoA reductase and the concentration of serum digoxin were increased and RBC membrane Na^+-K^+ ATPase activity and serum magnesium were reduced. Post-therapy the activity of HMG CoA reductase and the concentration of digoxin were reduced and RBC membrane Na^+-K^+ ATPase activity and serum magnesium were increased (Table 1).

The concentration of serum tryptophan, quinolinic acid and serotonin was increased in the plasma while that of tyrosine, dopamine and noradrenaline was decreased in the pre-therapy group. Post-therapy the concentration of serum tryptophan, quinolinic acid and serotonin was reduced in the plasma while that of tyrosine, dopamine and noradrenaline was increased (Table 2).

The post-therapy seizure frequency showed a significant decrease (Table 3).

Discussion

The results showed that plasma HMG CoA reductase activity and serum digoxin were increased in seizure disorder (ILAE classification – II E – generalized seizures – tonic clonic). Previous studies in this laboratory have demonstrated incorporation of 14C-acetate into digoxin in a rat brain indicating that acetyl CoA is the precursor for digoxin biosynthesis.[13] The elevated HMG CoA reductase activity correlates well with elevated digoxin levels and reduced RBC membrane Na^+-K^+ ATPase activity. The increase in endogenous digoxin, a potent inhibitor of membrane Na^+-K^+ ATPase, can decrease this enzyme activity.[14] The inhibition of Na^+-K^+ ATPase by dig-

Table 1: Concentration of serum digoxin, magnesium and RBC membrane Na^+-K^+ ATPase activity in refractory primary generalized epilepsy

Groups	HMG CoA reductase Reductase ratio of HMG CoA/ mevalonate	Digoxin ng/dl	Na^+-K^+ ATPase (µg/p_i/mg protein)	Magnesium mg/dl
1 Control	1.15±0.12	12.80±1.09	5.04±0.221	2.40±0.24
2 Epilepsy pre-therapy	0.884+0.075*	23.05+1.76*	1.48+0.139*	2.08+0.11*
3 Epilepsy post-therapy	1.12+0.12*	14.0±1.07*	4.02+0.132*	2.56+0.22*

Mean of the values from 15 samples + SD., * p less than 0.01; Group 2 has been compared with group 1, Group 3 has been compared with group 2.

Table 2: Tyrosine and Tryptophan catabolic patterns in refractory primary generalized epilepsy

Groups	Tyrptophan (mg/dl)	Tyrosine (mg/dl)	5HT (µg/dl)	Dop (ng/dl)	Norepi (ng/dl)	QA (ng/dl)
1. Control	1.11±0.08	1.14±0.09	20.9±1.9	12.89±0.67	45.15±2.35	370.60±21.07
2. Epilepsy	1.96±0.09*	0.883±0.05*	59.5±4.6*	8.53±0.53*	34.18±1.11*	549.34±41.21*
3. Epilepsy post-therapy	1.12±0.07*	1.11±0.06*	25.8±1.7*	11.88±0.66*	44.15±1.12*	390.62±20.07*

Mean of the values from 15 samples + SD, * p less than 0.01; Group 2 has been compared with group 1, Group 3 has been compared with group 2, 5 HT – Serotonin; DOP – Dopamine; Norepi – Nor-epinephrine; QA – Quinolinic acid

Table 3: Seizure frequency in refractory primary generalized epilepsy pre and post-therapy

Seizure	Frequency 2	
	Pre-therapy	Post-therapy
1. Male 21	12 per month	2 per month
2. Male 32	10 / month	3 / month
3. Male 35	8 / month	3 / month
4. Male 26	12 / month	3 / month
5. Male 28	9 / month	1 / month
6. Male 19	9 / month	1 / month
7. Male 34	8 month	4 / month
8. Male 33	12 / month	2 / month
9. Female 21	15 / month	3 / month
10. Female 28	12 / month	3 / month
11. Female 26	11 / month	3 / month
12. Female 32	11 / month	4 / month
13. Female 26	8 / month	2 / month
14. Female 30	9 / month	0 / month
15. Female 22	9 / month	2 / month
*Average seizure frequency	9 / month	2 / month

*P less than 0.01, Group 2 was compared with group 1

oxin is known to cause an increase in intracellular calcium and a reduction in intracellular magnesium. This has been reported previously in the literature.[15] Serum magnesium was found to be reduced in seizure disorder (ILAE classification – II E – generalized seizures – tonic clonic). Membrane Na^+-K^+ ATPase inhibition can produce defective neuronal membrane repolarization and a paroxysmal depolarization shift resulting in epileptogenesis.[16]

There is an increase in tryptophan and its catabolites and a reduction in tyrosine and its catabolites in the patient's serum. This could be due to the fact that digoxin can regulate neutral amino acid transport system with preferential promotion of tryptophan transport over tyrosine.[17] The decrease in membrane Na^+-K^+ ATPase activity in seizure disorder (ILAE classification – II E – generalized seizures – tonic clonic) could also be due to the fact that the hyperpolarizing neurotransmitters (dopamine and noradrenaline) are reduced and the depolarizing neuroactive compounds (serotonin and quinolinic acid) are increased.[18] Quinolinic acid and serotonin being NMDA (N-Methyl D-Aspartate) agonist can contribute to NMDA excitotoxicity reported in epilepsy.[19] In the presence of hypomagnesemia, the magnesium block on the NMDA receptor is removed leading to NMDA excitotoxicity.[20] The plasma membrane glutamate transporter (on the surface of the glial cell and presynaptic neuron) is coupled to a Na^+ gradient which is disrupted by the inhibition of membrane Na^+-K^+ ATPase, resulting in decreased clearance of glutamate by presynaptic and glial uptake at the end of synaptic transmission.[20] By these mechanisms, inhibition of neuronal membrane Na^+-K^+ ATPase can promote glutamatergic transmission and excitotoxicity contributing to epileptogenesis.

A quantal perception model of brain function has been postulated by several groups of workers.[1] A low level of EMF from the healer is probably transferred to the recipient patient by quantal perception. The perceived element in quantal or subliminal perception could be the quanta of matter-dependent electric and magnetic fields. The brain functions as a quantum computer with the quantum computer memory elements consisting of superconducting quantum interference devices–the SQUIDS—which can exist as superposition of macroscopic states.[1] Bose condensation, the basis of superconductivity is achievable at room temperature in the Frohlich model in biological systems. The dielectric protein molecules and polar sphingolipids of the neuronal membrane, nucleosomes which are a combination of basic histones and nucleic acid, and cytoplasmic magnetite molecules are excellent electric dipole oscillators which exist under a steep neuronal membrane voltage gradient. The individual oscillators are energized with a constant source of pumping energy from outside, by digoxin binding to membrane Na^+-K^+ ATPase and produce a paroxysmal depolarization shift in the neuronal membrane. This prevents the dipole oscillators from ever settling into thermal equilibrium with the cytoplasm and interstitial fluid which is always kept at constant temperature.[1] This results in a neuronal quantal state. There are direct connections between the hypothalamus and the cerebral cortex and digoxin may function as a modulator of the hypothalamocortical synapses. Bose condensed states produced by digoxin-mediated dielectric protein molecular pumped phonon system could be used to store information which might be encoded–all within the lowest collective frequency mode–by appropriately adjusting the amplitudes of and phase relations between the dipole oscillators. The external world sensory impressions exist in the cortical dipole oscillators as probabilistic multiple superimposed patterns—the U phase of quantum mechanics. The part of the incoming quantal data maps of the external world built by quantal perception in logical sequence and corollary to the pre-existing cortical external world maps built by conscious perception is chosen. Hypothalamo-cortical connections modulated by digoxin acting on the neuronal membrane help to magnify the chosen map to 1 graviton criteria. This model of quantal perception gives a mechanism for extrasensory or subliminal perception.

Reiki-like healing practices can transmit low level of body EMF from the healer to the recipient by quantal perceptive mechanism. Post-therapy, there was an increase in RBC membrane Na^+-K^+ ATPase activity and serum magnesium and a reduction in HMG CoA reductase activity and digoxin synthesis. Also, the level of tyrosine and its hyperpolarizing catabolites was increased while that of tryptophan and its depolarizing catabolites was decreased. A low level of EMF can stabilize the neuronal membrane and increase neuronal membrane Na^+-K^+ ATPase activity.[22] The stimulation of the neuronal membrane Na^+-K^+ ATPase is known to cause a decrease in intracellular calcium and an increase in intracellular magnesium.[15] Magnesium excess is known to inhibit HMG CoA reductase activity.[16] This leads to reduced digoxin synthesis. Reduced levels of digoxin can stimulate membrane Na^+-

K^+ ATPase activity further and increase intraneuronal magnesium to a greater extent. This starts off a cascade which stimulates membrane Na^+-K^+ ATPase further and stabilizes the neuronal membrane. The stimulation of membrane Na^+-K^+ ATPase can promote neuronal membrane repolarization and inhibit the generation of a paroxysmal depolarization shift and epileptogenesis. Digoxin is known to promote tryptophan transport over tyrosine.[17] Low levels of digoxin can lead to an increase in serum tyrosine levels and a decrease in serum tryptophan. This leads to an increase in the levels of hyperpolarizing tyrosine catabolites and a decrease in the levels of depolarizing tryptophan catabolites, which inhibits epileptogenesis. The increased levels of noradrenaline and dopamine have an antiepileptic action.[18] The increase in serum magnesium also helps to downregulate glutamatergic transmission and inhibits epileptogenesis. The magnesium block on the glutamate NMDA receptor is strengthened.[20]

The increase in serum magnesium in the post-therapy group could also be due to the phenomenon of biological transmutation.[24] Serum magnesium levels are increased, suggesting an increase in the total body magnesium rather than functional replacement of calcium with magnesium. Biological transmutation has been postulated by several groups of workers. Hypothalamic digoxin induced dielectric protein molecular pumped phonon system produces a quantal state within the neuron and in the cell membrane. In this quantal state biological transmutation can happen leading to an increase in serum magnesium levels despite only adequate intake.

The effect of Reiki-like treatment practices and transcendental meditation on seizure count and frequency as well as on biochemical pathways related to membrane Na^+-K^+ ATPase stimulation provides evidence regarding quantal perception and brain function. It also provides evidence on the regulation of metabolic processes by quantally perceived, low levels of EMF induced changes in neuronal transmission. The phenomena of psychoneuromolecular, biological and environmental low level of EMF mediated regulation of metabolic processes needs further study.

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