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### Wolff-Parkinson-White Syndrome: Review of Literature

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#### Review Article

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#### ABSTRACT

Wolff-Parkinson-White (WPW) disorder is a condition in which there is an additional electrical pathway in the heart. The condition can prompt times of fast heart rate (tachycardia). WPW disorder is a standout amongst the most widely recognized reasons for quick heart rate issues in babies and youngsters. The patient with the Wolff-Parkinson-White (WPW) ECG design has much in the same way as patients with different elements, for example, long-QT disorder and Brugada disorder, which are connected with a recognizing variation from the norm on ECG. All are generally amiable in the lion's share of beset people however in any case convey a danger of unforeseen sudden passing, which asks for some endeavour at danger administration. The WPW pattern differs from the others in that a highly effective curative procedure is available, albeit with some risk, leaving us with a decision to manage expectantly or intervene.

#### INTRODUCTION

##### Causes

Normally, electrical signs complete a specific pathway the heart. This helps the heart beat frequently. This keeps the heart from having additional thumps or pulsates happening too early.

In individuals with WPW disorder, a portion of the heart's electrical signs go down an additional pathway. This may bring about an extremely fast heart rate called supraventricular tachycardia [1,2].

A great many people with WPW disorder don't have whatever other heart issues. Be that as it may, this condition has been connected with other heart conditions, for example, Ebstein irregularity [3-5]. A type of the condition likewise keeps running in families (Figure 1).

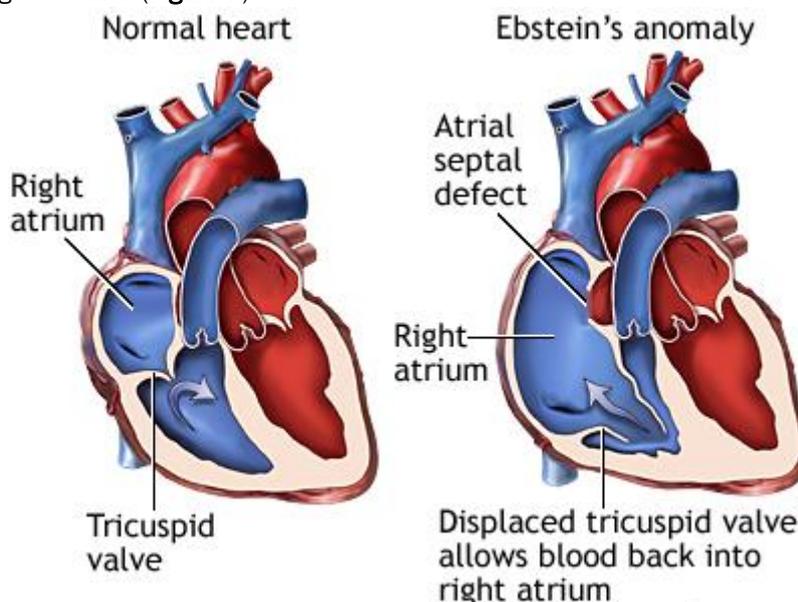


Figure 1: Comparison of normal heart and WPW disordered heart [6].

## Symptoms

The clinical manifestations of WPW syndrome reflect the associated tachyarrhythmia episodes—rather than the anomalous ventricular excitation per se. They may have their onset at any time from childhood to middle age, and they can vary in severity from mild chest discomfort or palpitations with or without syncope to severe cardiopulmonary compromise and cardiac arrest [7-20]. Thus, presentation varies by patient age.

Infants may present with the following:

- Tachypnea
- Irritability
- Pallor
- Intolerance of feedings
- Evidence of congestive heart failure if the episode has been untreated for several hours
- A history of not behaving as usual for 1-2 days
- An intercurrent febrile illness may be present
- A verbal child with WPW syndrome usually reports the following:
  - Chest pain
  - Palpitations
  - Breathing difficulty

Older patients can usually describe the following:

- Sudden onset of a pounding heartbeat
- Pulse that is regular and “too rapid to count”
- Typically, a concomitant reduction in their tolerance for activity

Physical findings include the following:

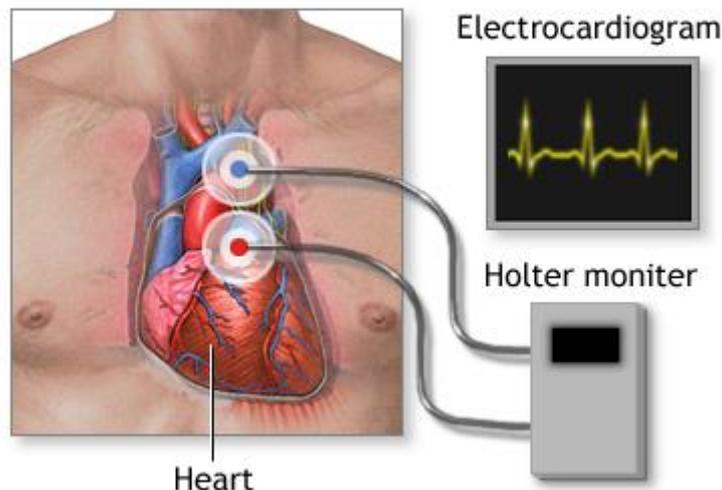
- Normal cardiac examination findings in the vast majority of cases
- During tachycardia episodes, the patient may be cool, diaphoretic, and hypotensive
- Crackles in the lungs from pulmonary vascular congestion (during or following an SVT episode)
- Many young patients may present with resting tachycardia on examination, with only minimal symptoms (eg, palpitations, weakness, mild dizziness) despite exceedingly fast heart rates.

Clinical features of associated cardiac defects may be present, such as the following [21-46]:

- Cardiomyopathy
- Ebstein anomaly
- Hypertrophic cardiomyopathy

## Exams and Tests

A physical exam done during a tachycardia scene will demonstrate a heart rate quicker than 100 pulsates every moment. An ordinary heart rate is 60 to 100 pulsates every moment in grown-ups, and under 150 beats for each moment in babies, newborn children, and little youngsters. Circulatory strain will be typical or low as a rule [47-60]. On the off chance that the individual is not having tachycardia at the season of the exam, the outcomes might be typical. The condition might be determined to have an ECG or with constant or individual activated walking ECG monitoring, for example, a Holter monitor (**Figure 2**).



**Figure 2:** A test called an electro physiologic study (EPS) is done using catheters that are placed in the heart. This test may help identify the location of the extra electrical pathway.

### DIAGNOSIS

WPW is normally analyzed on the premise of the electrocardiogram in an asymptomatic person. For this situation it is showed as a delta wave, which is a slurred upstroke in the QRS complex that is connected with a short PR interim. The short PR interim and slurring of the QRS complex is really the motivation enduring to the ventricles rashly (over the frill pathway) immediately experienced in the AV hub [61-85].

On the off chance that a man with WPW encounters scenes of atrial fibrillation, the ECG demonstrates a fast polymorphic wide-complex tachycardia (without torsade's de pointes). This blend of atrial fibrillation and WPW is viewed as hazardous, and most antiarrhythmic medications are contraindicated.

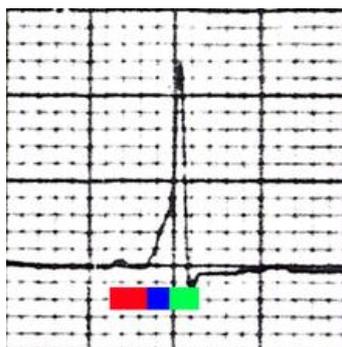
At the point when an individual is in typical sinus cadence, the ECG attributes of WPW are a short PR interim (under 120 ms in length), broadened QRS complex (more prominent than 120 ms in term) with slurred upstroke of the QRS complex, and optional repolarization changes (reflected in ST portion T wave changes).

In people with WPW, electrical movement that is started in the SA hub goes through the adornment pathway and through the AV hub to initiate the ventricles by means of both pathways. Since the adornment pathway does not have the motivation moderating properties of the AV hub, the electrical drive first initiates the ventricles through the extra pathway, and instantly a while later by means of the AV hub. This gives the short PR interim and slurred upstroke of the QRS complex known as the delta wave [86-95].

In the event of sort A pre-excitation (left atrioventricular associations), a positive R wave is seen in V1 ("positive delta") on the precordial leads of the electrocardiogram, while in sort B pre-excitation (right atrioventricular associations), a prevalently negative delta wave is found in lead V1 ("negative delta") (**Figure 3**).

Individuals with WPW may have more than one extra pathway—now and again, upwards of eight irregular pathways have been found. This has been found in people with Ebstein's abnormality.

Wolff–Parkinson–White disorder is once in a while connected with Leber's inherited optic neuropathy (LHON), a type of mitochondrial infection.



**Figure 3.** One beat from a rhythm strip in V2 demonstrating characteristic findings in Wolff–Parkinson–White syndrome. Note the characteristic delta wave (above the blue bar), the short PR interval (red bar) of 80 ms, and the long QRS complex (blue bar plus green bar) at 120 ms.

#### Differential Diagnosis

- Atrial fibrillation.
- Atrial flutter.
- Atrioventricular nodal re-entry tachycardia (AVNRT).
- Sinus node dysfunction.
- Ventricular fibrillation.
- Ventricular tachycardia.
- Ebstein's anomaly.
- Lown-Ganong-Levine syndrome.
- Other causes of syncope.

#### Treatment

Pharmaceuticals, especially antiarrhythmic medications, for example, procainamide or amiodarone, might be utilized to control or keep a quick pulse. In the event that the heart rate does not come back to ordinary with restorative treatment, specialists may utilize a kind of treatment called electrical cardioversion (shock).

The long haul treatment for WPW disorder is all the time catheter removal. This methodology includes embeddings a tube (catheter) into a vein through a little slice close to the crotch up to the heart zone. At the point when the tip achieves the heart, the little zone that is bringing on the quick heart rate is demolished utilizing an uncommon kind of vitality called radiofrequency or by solidifying it (cryoablation) [96-101].

Open heart surgery to smolder or stop the additional pathway may likewise give a perpetual cure to WPW disorder. By and large, this methodology is done just on the off chance that you require heart surgery for different reasons.

Complications may include:

- Complications of surgery
- Heart failure
- Reduced blood pressure (caused by rapid heart rate)
- Side effects of medicines

The most extreme type of a quick pulse is ventricular fibrillation (VF), which may quickly prompt stun or demise. It can once in a while happen in individuals with WPW, especially on the off chance that they additionally have atrial fibrillation (AF), which is another sort of anomalous heart mood [102-108]. This sort of quick pulse requires crisis treatment and a technique called cardioversion.

#### REFERENCES

1. Beyrouti R, et al. Parkinson Disease Associated with Myasthenia Gravis: A Case Report and Literature Review. *J Neurol Disord*. 2016;4:283.
2. Hong Duck Kim, et al. OMICS as Therapeutic Platform: Environmental Factors to Parkinsons Disease. *J Microb Biochem Technol*. 2016;8:222-225.
3. Antonino Cannas, et al. Suicide in Parkinsons Disease: An Open Question and a Complex and Poorly Explored Phenomenon. *J Neurol Disord* 2016;4:273.
4. Keiko Ikemoto. Imaging of D-cell: Aromatic L-amino acid decarboxylase (AADC)-immunoreactive glial cell found in Parkinsonian striatum. *J Mol Imaging Dynam*.
5. Shuei Sugama, et al. Effect of Chronic Stress in the Onset of Parkinsons Disease: Possible Role of Microglial Cells in Neuroinflammation. *J Neurol Disord*. 2016:S2-001.
6. Hiller ALP, et al. Are Cholinesterase Inhibitors Effective in Improving Balance in Parkinsons Disease?. *J Neurol Disord*. 2016:S2-002.
7. Kataoka H and Ueno S. Frontal Assessment Battery and Falling Related with Freezing of Gait in Parkinsons Disease. *Int J Phys Med Rehabil*. 2016;4:335.
8. Zeilig G and Shiller AD. Advanced Technology to Enhance Rehabilitation Outcomes: Parkinson Disease. *Int J Neurorehabilitation Eng*. 2016;3:e124.
9. Woolhouse MH and Zaraneek A. Intuitive Navigation in Computer Applications for People with Parkinsons. *J Biomusic Eng*. 2016;4:115.

10. Biswas A and Das SK. Alzheimer and Parkinsons Disease -Two Faces of the Same Disease?. J Alzheimers Dis Parkinsonism.
11. Prasad RKA, et al. A Review on Techniques for Diagnosing and Monitoring Patients with Parkinsons Disease. J Biosens Bioelectron. 2015;7:203.
12. Werner FM and Coveñas R. Efficacy of the Deep-Brain Stimulation in Parkinsons Disease According to a Neural Network. J Cytol Histol.
13. Oliver Kaut, et al. DNA Methylation of Imprinted Loci on Autosomal Chromosomes and IGF2 are not Affected in Parkinson's Disease Patients Peripheral Blood Monocytes. Brain Disord Ther. 2016;5:211.
14. Pieroni MA, et al. Novel Therapeutic Approach for Parkinson Disease during REM Sleep. J Health Edu Res Dev. 2016;4:164.
15. Birman N, et al. Decreased Dopamine Transporter Binding Ipsilateral to the Clinically More Affected Side in Parkinson's disease: Which Side to Take?. J Neurol Neurophysiol.2016;7:361.
16. Bayle N, et al. Movement Smoothness Differentiates Voluntary from Parkinsonian Bradykinesia. J Addict Res Ther. 2015;7:264.
17. Iijima M, et al. Odor Identification Function Differs between Vascular Parkinsonism and Akinetic-Type Parkinson's Disease. J Alzheimers Dis Parkinsonism.
18. Park BJ. How Should We Deal with Parkinson's Disease?. Single Cell Biol. 2016;5:129.
19. Gera G, et al. Identification of Balance Deficits in People with Parkinson Disease; is the Sensory Organization Test Enough? Int J Phys Med Rehabil 2016; 4.
20. Li M Fu. Genomics Reveals Similar and Dissimilar Pathogenesis between Alzheimer's and Parkinson's Diseases. Biol syst Open Access. 2015;5:148.
21. Andrew Tran et al. The Role of Coffee in the Therapy of Parkinsons Disease. J Alzheimers Dis Parkinsonism.
22. Olivola Enrica et al. Low Serum 25(OH)D Levels in Parkinson Disease; a Non Specific Marker of Neurodegeneration?. J Alzheimers Dis Parkinsonism.
23. Piemonte MEP, et al. Extensive Training Promotes Performance Improvement but not Automaticity in Patients with Parkinson's disease. J Neurol Neurophysiol. 2015;6:324.
24. Farheen SA, et al. Translational Science of Psychosis in Parkinsons Disease. Clin Depress.
25. Cocco T and Papa S. Molecular Targets for Improvement of Parkinson's Disease Therapy. Brain Disord Ther. 2015;4:173.
26. Gambhir PK. How Well can a Person with Parkinsons Swallow?. Int J Neurorehabilitation Eng 2015;2:e114.
27. Marcela Díaz et al. Assessment of the Protective Capacity of Nanosomes of Quercetin in an Experimental Model of Parkinsons Disease in the Rat. Gen Med (Los Angel) 2015;3:207.
28. X Deng, et al. Adverse Effects were not the Main Causes for Rotigotine Patch Withdrawal in Parkinsons Disease. J Alzheimers Dis Parkinsonism. 2015;5:195.
29. Shroff G. Use of Human Embryonic Stem Cells in the Treatment of Parkinsons Disease: A Case Report. Int J Emerg Ment Health. 2015,17:260.
30. Carpi S, et al. Non-motor symptoms in Parkinson disease. CNB.
31. Bokhari FA. Non-Wolf Parkinson White (WPW) Preexcitation Syndrome. Angiol. 2015;3:152.
32. Dow CT. Parkinsons -Just another Infectious Disease. J Neuroinfect Dis. 2015;6:183.
33. O'Connor M, et al. The Relationship between Strength and Balance in Individuals with Parkinsons Disease. J Neurol Disord. 2015;3:239.
34. Ryan JJ, et al. Temporal Disorientation Base Rates in Alzheimers Disease and Parkinsons Disease. J Gerontol Geriatr Res. 2015;4:221.
35. Pallanti S and Marras A. Transcranial Magnetic Stimulation Treatment for Motor Symptoms in Parkinsons Disease: A Review of Two Decades of Studies. J Alzheimers Dis Parkinsonism. 2015;5:191.
36. Salama M. mTOR Silencing in Parkinson's Disease both in vitro and in vivo. Brain Disord Ther. 2015;4:167.

37. Hatzifilippou E, et al. High Levels of Anti-Ganglioside Antibodies in Patients with Parkinsons Disease Associated with Cognitive Decline. *Int J Neurorehabilitation Eng.* 2015;2:159.
38. Vadivelan M. Parkinson's Disease. *J Sleep Disord Ther.* 2015;4:200.
39. Petrosyan TR. Bacterial Melanin as a Potential Targeted Therapy for the Parkinsons Disease. *Pigmentary Disorders.* 2015;2:173.
40. Bagchi A, et al. Organic Farming Practice for Quality Improvement of Tea and Its Anti Parkinsonism Effect on Health Defense. *J Phys Chem Biophys.* 2015;5:178.
41. Daniela AP, et al. Markers of ParkinsonsDisease Progression Using Cerebrovascular, Autonomic and Small Fiber Polyneuropathy Features. *J Neurol Disord* 2015;3:217.
42. Bhande S. Parkinson Diagnosis using Neural Network: a Survey. *IJRSET.*
43. Kovacs A, et al. Induced Psychosis or Psychotic Relapse? An Unexpected Effect of Anti-Parkinson Treatment. *Int J Emerg Ment Health.* 2015;17:191.
44. Fritsch T, et al. L-DOPA-Induced Disinhibition in Parkinson Disease: More than Agonistic Behavior. *Biol Syst Open Access.* 2012;2:115.
45. Felix-Martin W and Coveñas R. Treatment of Psychotic Symptoms in Parkinsons Disease. *J Cytol Histol.* 2015;6:e115.
46. Utkin YN, et al. What Animal Models of Parkinsonism Tell us About the Distinct Nicotinic Acetylcholine Receptors Involved in Pathogenesis?. *J Alzheimers Dis Parkinsonism.* 2015;5:181.
47. Devasena T and Francis AP. Nanotoxicity-Induced Alzheimer Disease and Parkinsonism: Not Further than Diagnosis. *J Alzheimers Dis Parkinsonism.* 2015;5:178.
48. Swathi G, et al. Evaluation Of Rotenone Induced Parkinson's Disease On Glutamate Metabolism And Protective Strategies Of Bacopa Monnieri. *IJPAES.*
49. Yamamoto T, et al. Assessment of A New Magnetic Device to Monitor Swallowing in Parkinsons Disease. *J Neurol Neurophysiol.* 2015;6:267.
50. Santiago JA, et al. Understanding the Role Diet Plays in Parkinson's Disease Could Lead to Better Disease Management. *Clin Exp Pharmacol.* 2014;5:e135.
51. Felix-Martin W and Coveñas R. Might Combined GABAA Agonists and NMDA Antagonists have a Therapeutic and maybe a Prophylactic Effect in Alzheimer's and Parkinson's Disease?. *J Cytol Histol.* 2015;6:298.
52. Olalekan OM and Olurotimi JS. NMDA R/VDR in Fish Melanocytes; Receptor Targeted Therapeutic Model and Mechanism in Parkinsons disease. *J Biomol Res Ther.* 2014;3:114.
53. Arkun K, et al. Effect of Lewy Bodies on Mitochondrial DNA Copy Numbers and Deletion Burden in ParkinsonsDisease Substantia nigra Neurons. *J Alzheimers Dis Parkinsonism.* 2015;4:175.
54. Tortolero GS, et al. EEG Findings in Diffuse Lewy Body Disease and Parkinson's Disease with Dementia. *Brain Disord Ther.* 2015;4:156.
55. Stephens SJ, et al. Fluoxetine-Induced Atypical Serotonin Syndrome with Hallucinations Masquerading as a Parkinsonian Syndrome. *Fam Med Med Sci Res.* 2014;3:147.
56. Mohammed NB, et al. Single Dose Does Matter! An Interesting Case of Parkinsons Hyperpyrexia Syndrome. *J Neurol Disord.* 2014;2:191.
57. Oguro H, et al. Randomized Trial of Repetitive Transcranial Magnetic Stimulation for Apathy and Depression in Parkinsons Disease. *J Neurol Neurophysiol.* 5:242.
58. Sayaka Ono et al. FMT-PET for the Early Diagnosis of Parkinsons Disease. *J Neurol Disord.* 2014;2:104.
59. Kathryn R, et al. Personality Style in Behavioural Disturbances in ParkinsonsDisease. *J Neurol Neurophysiol.*5:251.
60. Hinson VK, et al. Forced Exercise for Freezing of Gait in Post STN DBS ParkinsonsDisease Patients. *J Alzheimers Dis Parkinsonism.* 2014;4:171.

61. Whitesman P. Preliminary Set Theory-Type Analysis of Proteins Associated With Parkinsons Disease. *J Alzheimers Dis Parkinsonism*. 2014;4:170.
62. Bitner A, et al. The Role of Multidrug Interactions in the Safety of Pharmacotherapy for Concomitant ParkinsonsDisease and Arterial Hypertension in Poland. *J Pharmacovigil*. 2014;2:151.
63. Bryan Lieber BA, et al. Motion Sensors to Assess and Monitor Medical and Surgical Management of ParkinsonsDisease. *Int J Phys Med Rehabil*. 2014;2:221.
64. Leroi I, et al. Apathy and Emotional Blunting in Parkinson's Disease. *Brain Disord Ther*. 2014;3:141.
65. Jianfeng Lu. Modeling Parkinson Disease with Human Induced Pluripotent Stem Cells. *Clon Transgen*. 2014;3:3.
66. Felix-Martin W and Covenas R. Classical Neurotransmitters and Neuropeptides involved in Parkinson's Disease: A Multi-Neurotransmitter System. *J Cytol Histol*. 2014;5:266.
67. Lieberman A. Falls in Parkinson Disease: The Relevance of Short Steps. *J Nov Physiother*. 2014;4:209.
68. Sadek HL, et al. The Inflammatory Cytokines in the Pathogenesis of ParkinsonsDisease. *J Alzheimers Dis Parkinsonism*. 2014;4:148.
69. Bhowmick SS, et al. Postencephalitic Parkinsonism in a Patient with Mumps Infection: A Case Report. *JNID*. 2014;5:162.
70. Joanna C. Rehabilitation Procedures Aimed at Decreasing Motor Symptoms in Parkinson's Disease. *Int J Phys Med Rehabil*. 2014;S5:009.
71. Wheeler CJ, et al. T-Lymphocyte Deficiency Exacerbates Behavioral Deficits in the 6-OHDA Unilateral Lesion Rat Model for Parkinson's Disease. *J Neurol Neurophysiol*. 2014;5.
72. Lieberman A, et al. Comparison of Parkinson Disease Patients Who Fell Once with Patients Who Fell More than Once (Recurrent Fallers). *J Alzheimers Dis Parkinsonism*. 2014;4:140.
73. Evaluation of Metabolic State in Striatal Rat Model of ParkinsonsDisease: before and after Deep Brain Stimulation. *J Obes Weight Loss Ther*. 2014;4:205.
74. Scovassi AI. Parkinsons Disease: New Insights. *Biochem & Pharmacol* 2012;4:e146.
75. Wesnes KA and Burn DJ. Compromised Object Pattern Separation Performance in Parkinsons Disease Suggests Dentate Gyrus Neurogenesis may be Compromised in the Condition. *J Alzheimers Dis Parkinsonism*. 2014;4:131.
76. Polgar S, et al. Stem Cell Therapy for Parkinsonsdisease: Are Double-Blind Randomized Control Trials the Best Design for Quantifying Therapy Outcomes?. *J Neurol Neurophysiol* 2013;4:170.
77. Kabel AM and El Kholi SS. Effect of Ubiquinone and Resveratrol on Experimentally Induced Parkinsonism. *J Res Development*. 2013;3:112.
78. Rodriguez S. Are Animal Models of ParkinsonsDisease as Bad as they Seem to Be?. *J Neurol Disord*. 2014;2:e107.
79. Radaei F and Gharibzadeh S. The Increase of AMP-activated Protein Kinase during Exercise and its Effect on Reducing Parkinson's Disease Symptoms. *Brain Disord Ther*. 2013;2:110.
80. Thanasan S, et al. Clozapine Withdrawal Catatonia or Lethal Catatonia in a Schizo affective patient with a family history of Parkinsons Disease. *Afr J Psychiatry*.
81. Radaei F and XGharibzadeh S. The Effect of Carbidopa in Carbidopa-Levodopa Combination on Reducing Osteoporotic Symptoms in Parkinsons Disease Patients. *Brain Disord Ther*. 2013;2:107.
82. Nocera JR, et al. Exercise to Improve Non-Motor Symptoms of Parkinson's Disease. *J Yoga Phys Ther*. 2013;3.
83. James LE and Asuni AA. Parkinsons Disease and the ?Sunshine? Vitamin. *J Alzheimers Dis Parkinsonism*. 2013;3:120.
84. Khanh vinh quoc, et al. Environmental Factors in Alzheimers and Parkinsons Diseases. *J Alzheimers Dis Parkinsonism*. 2013;3:119.

85. Fei C, et al. Experimental Study on Parkinson Disease Model of Rats. *J Neurol Disord*. 2013;1:127.
86. Fei C, et al. Experimental Study on the Neuronal Toxic Effect of Levodopa and the Inhibition of Ginkgo Biloba Extract on Parkinson Disease in Rats. *J Neurol Disord*. 2013;1:126.
87. Young HE, et al. Parkinson Disease with Adult Stem Cells. *J Neurol Disord*. 2013;1:121.
88. Frazzitta G. Rehabilitation and Parkinsons Disease: A Happy Marriage!. *Int J Phys Med Rehabil*. 2013;1:109.
89. Lieberman A. Did Adolf Hitlers Parkinson Disease Affect his Conduct of World War II. *Biol Syst Open Access*. 2013;2:111.
90. Shih-Wei L, et al. No Association between Chronic Osteomyelitis and ParkinsonsDisease in Older People in Taiwan. *J Alzheimers Dis Parkinsonism*. 2013;3:112.
91. Doherty GH. Homocysteine and Parkinsons Disease: A Complex Relationship. *J Neurol Disord* 2013;1:107.
92. Tuite PJ, et al. Magnetic Resonance Imaging (MRI) in Parkinson's Disease. *J Alzheimers Dis Parkinsonism*. 2013;Suppl 1:001.
93. Ridgel AL, et al. Effects of Repeated Bouts of Segmental Vibration Therapy on Balance in Parkinsons Disease. *Int J Phys Med Rehabil*. 2013;1:104.
94. Yasuda Y. Application of Glycerin Poultice to the Semitendinous and Semimembranous above the Popliteal Fossa Improves Motor Disturbance in Parkinson Disease. *J Nov Physiother*. 2012;S1-005.
95. Okada Y, et al. Galvanic Vestibular Stimulation for Camptocormia in ParkinsonsDisease: A Case Report. *J Nov Physiother*. 2012;S1-001.
96. Laine R and Tino Unlap M. IPX-750, A Dopamine Gluconamine That Binds D1/D5 Receptors and Has Anti-Parkinsonian Effects in Three Animal Models, is Transported across the Blood Brain Barrier. *J Biotechnol Biomater*. 2012;2:5.
97. Pasinetti GM. Role of Personalized Medicine in the Identification and Characterization of Parkinson's Disease in Asymptomatic Subjects. *J Alzheimers Dis Parkinsonism*. 2012;2.
98. Hondzinski JM. Specificity of Training, Not the Only Therapy Option for ParkinsonsPatients. *J Nov Physiother* 2012,2:e119.
99. Ghribi O. Do Increased Levels of Oxysterols Underlie Alzheimer?s Disease/ParkinsonsDisease Overlap?. *J Alzheimers Dis*. 2012.2:e111.
100. Perry G, et al. A Pleotrophic Age for Alzheimer and Parkinson disease. *J Alzheimers Dis*. 2012;2:e109.
101. Chong R. Closed-Loop VR-Based Interaction to Improve Walking in Parkinson's Disease. *J Nov Physiother*. 2011;1:1.
102. Melissa LM, et al. Mesenchymal Stem Cell-Based Therapies for ParkinsonsDisease: Progress, Controversies and Lessons for the Future. *J Stem Cell Res Ther*. 2011;S2-005.
103. Takasaki S. Mitochondrial Haplogroups Associated with Japanese Centenarians, Alzheimers Patients, Parkinsons Patients, Type 2 Diabetes Patients, Healthy Non-Obese Young Males, and Obese Young Males.
104. Francisco R, et al. Wolff-Parkinson-White and Prolonged? Q-T? Patterns in the Same Electrocardiographic Record. *J Clinic Experiment Cardiol* 2011;2:118.