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Cerebral palsy and its treatments

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Mini Review

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Introduction

Cerebral palsy may be a neurological disease that has a gaggle of disabilities that influence a child's ability to maneuver and uphold equilibrium and position [1-5]. Encephalopathy is additionally illustrated as a loss or inability to regulate over the body movements; or motor issues in regulation the hand and arms throughout attainment of manual tasks. These difficulties with the timings of motor movements arise because of abnormal or incomplete brain development. Encephalopathy is one amongst the foremost usually found disorders within the developing countries and its prevalence is cited as one or 2/1000 in nascence. Failure in action of milestones of motor development from birth to 5 years like, rolling, sitting, standing, holding or walking etc., area unit doubtless to be the numerous indicators of encephalopathy. Signs and symptoms of encephalopathy vary from person to person [6-9]. This tends to worsen in relevance advancement in age [10-16]. Not solely has the severity rather typically, had the symptoms modified in their nature and trends. As one grows older as an example before six months old-time, could feel stiff or floppy however will pass off because the same child grows older than six months, he/she might not even be ready to appear any direction the least bit. Spastic paralysis will be diagnosed with thorough clinical assessment by Associate in Nursing authentic medico and paeditrician in conjoining reports and observations of biological process psychologists and oldsters [17-19]. Spastic and dyskinetic area unit 2 major subtypes of spastic paralysis. Spastic refers to result tone during which physical forces area unit inflexible (abrupt), and movements area unit uncomfortable, tense and jerky. This class of spastic paralysis affects variegated of the cadaver confidential by these components of the body like handicapped person (one aspect of the body), palsy (both legs), palsy (the entire body), 70-80% cases of spastic paralysis falls during this kind. Jerkiness will be assessed by increase in muscles tone that\'s pertaining to resistance to physical property, by evaluating joint angle [20-25]. Second sort of spastic paralysis is dyskinetic that affects association and coordination of arrangements. Dystonia refers to tonus and reduce motion; choreoathetosis, to asymmetrical, irregular, involuntary movements of the appendages or facial muscles. The caregiving may be a traditional performs of being the parent of a young kid. But this role takes up new kind once the kid is experiencing some impairment that is useful in nature. In such cases, the fogeys need to deal with the challenges of health problems that area unit confronted by their child's physical or intellectual incapacity standing. The caregiving needs of everyday living increase the toll of burden for the fogeys and that they consequently face numerous changes in their physical and psychological well-being, that indirectly affects the family functioning patterns and family dynamics during which all members directly or indirectly get influenced [26-29]. This can be Associate in nursing involved development to be investigated however family dynamics get subtly modified attributable to the presence of special wants member within the family. The approach of estimating the "independent" or "direct" effects of the care-recipient's incapacity on the family functioning might not seem adequate. An additional elaborated analytical approach is required to

know each direct and indirect effect at the same time. Drawback with anybody of the kid produces marked effects on whole family functioning, that describes because the styles of relations associating members of a family system to every alternative. Families develop patterns for managing basic, biological process, and crisis-related tasks. Healthy families uphold the emotional, physical and communal welfare of each member of family. Among the various components that participate during this method area unit a family's internal strengths and therefore the stability of the nuclear family. In contrast to the other grouping, family's area unit competent to deliver the shut emotional resource required to yield self-contained and well-balanced youngsters and adults. Likewise, families that perform in an exceedingly healthy manner area unit well-armed to touch upon the various routine variations and unexpected catastrophes that challenges them throughout their life. So primary perform of the family is to supply opportunities to grow and develop to every member of the family [30-35].

Treatments of Cerebral palsy treatments:

Human Embryonic vegetative cell medical care in encephalopathy Shroff G, Das L (2014)

Treatment of vision defect in kids with CP is difficult, recent analysis during this space has shown right smart improvement with the utilization of stem cells together with neurotrophic agents. the utilization of human embryonic stem cells (hESCs) has been explored recently for the treatment of CVI in kids with CP. though only a few studies evaluating the role of hESCs square measure out there as a result of moot supply and isolation technique of hESCs, a study on visually impaired rates showed 100 percent improvement with the utilization of hESCs. The analysis work of Shroff G, Das L (2014) with helpful results had shown the effectiveness of hESC medical care. CVI is that the visual loss that happens as a result of disturbance within the posterior visual pathway or the cortical region. Lack of element to the brain is that the commonest reason for the prevalence of CVI. aside from this, CVI might result as a result of intracranial pressure/hydrocephaly, brain malformations/head injury, central system infections, poisoning/drug exposure, prematurity/birth trauma, encephalopathy, or seizures/epilepsy. Huo et al. reportable that twenty five.9% of the youngsters with CVI have CP. alternative studies have conjointly reportable that almost all kids with CVI have concomitant CP.

Damage to the anterior vision pathway, lateral crooked bodies, or the os cortices within the brain among patients with CP might cause vision defect [35-40]. Presently, there aren\'t any medications or surgical therapies out there for the treatment of CVI except rehabilitation medical care. within the gift study, we have a tendency to tried to guage the effectiveness of hESCs within the treatment of CVI in kids with CP. most the youngsters (39 of 40) showed improvement in vision by atleast one grade. Of the eight blind patients enclosed within the gift study, all showed improvement in vision (greater than 2) with just one patient having perception of sunshine at the top of the study. Over half the patients regained traditional vision at the top of the study once receiving hESC medical care.

After being transplanted within the body, the hESCs begin growing within the affected space to exchange the degenerated cell kind. Within the gift study, the new differentiated cells replaced the broken cells and improved insertion was mirrored within the SPECT scan. The patients received hESC medical care with gap amount between 2 treatment phases as a result of hESCs need time to grow and regenerate the affected half to a most extent. Additionally, the gap part provides the medical man AN understanding of the patient's condition and demand for any treatment [41-56]. Stem cells have the potential to speak with the opposite cells of the body from the realm of harm and "home" at the positioning of injury. Chemokine cytokines and growth factors free from the positioning of injury communicate with the stem cells administered within the body. This method is initiated by the upregulation of selectins and integrins on the surface of the stem cells that modify their migration to the broken website resulting in regeneration. Previous studies have shown that the route through that mesenchymal stem cells were administered influenced their potential to migrate and residential at the positioning of injury [57- 61]. Additionally, studies investigation the potential of stem cells for chronic disorders have shown that a niche amount between 2 doses of stem cells is crucial as a result of the stem cells need time to start out multiplying into the specified cell kind. Penha et al. showed that a continuing improvement was discovered over an amount of three months once transplantation of MSCs in dogs with SCI and a motivating improvement was seen eighteen months once the transplantation MSCs. author et al. showed improved eye sight of a five twelvemonth previous CP patient with twine

blood vegetative cell medical care at a pair of months [62-71]. Neural stem cells, hematogenic stem cells and embryonic stem cells have shown potential to revive traditional vision by repopulating the broken areas and by preventing any degeneration of the cells.

Lack of element offer to the brain is one in every of the leading causes of CVI in kids with CP. SPECT scan is an efficient tool within the detection of cerebral impairment, cerebral blood flow, and hypo perfused areas of the brain [72-75]. SPECT scan detects the hypo perfused areas of the brain as a result of reduced tracer uptake in these regions. The extent of tracer uptake shows the extent of insertion in those areas. The regions of the brain with weakened tracer uptake represent hypo perfused areas, those with absence of tracer uptake represent areas with no insertion, and an exaggerated tracer uptake represents hypoperfusion. We have a tendency to used SPECT scan as a prognostic tool for sleuthing the extent of improvement in insertion among the patients during this study. Brain SPECT scan is an efficient watching tool for response to hESC medical care in patients with CP. It detects changes occurring at molecular level.

Sasmal et al. showed that twenty.7% of the patients with CP has concomitant CVI and early ophthalmologic examination is important for optimum management. Alternative prevalence studies have shown high proportion of patients (61.9%) with CP to be utterly blind. Of the various varieties of visual impairments discovered in patients with CP, 47.7% of them expertise CVI. Impairment of vision conjointly ends up in the general incapacity of the youngsters with CP.

Treatment with hESC derived retinal pigment epithelial tissue has shown improvement in vision on the graph in rats with retinal unwellness. The utilization of hESCs has not been clinically viable within the overdue to problem in harvest home these cells in an exceedingly xeno free surroundings. However; we have a tendency to use a proprietary (United States Granted Patent No-WO 2007/141657A PCT/1B 2007 revealed thirteen Dec 2007)in-house methodology for the isolation of hESCs to explore their potential within the treatment of visually impaired patients with CP. Treatment of AN 11-year previous patient United Nations agency had CP and vision defect with bone marrow mesenchymal stem cells showed improvement in electrophysiologically examined vision once treatment for an amount of vi months . The utilization of hESC medical care within the treatment of vision defect in patients with CP has not been experimented extensively however; the utilization of hESCs within the treatment of ocular diseases of variable etiologies has shown improvement in vision. Neural stem cells (NSCs) derived from hESCs preserve photoreceptors and visual operate while not formation of tumour cells. presymptomatic animal studies have incontestable effective transplantation of NSCs in hypoxic-ischemic brain with plant tissue injury.

Neural stem cells square measure noted to revive traditional vision by repopulating the broken areas and by preventing any degeneration of the cells. The hESCs initiate regeneration of broken cells by multiplying into similar cell kind. Otani et al. showed that hematogenic stem cells fixed retinal operates and vascularisation in mice with retinal degeneration unwellness. Ameele et al. showed that transplantation of embryonic stem cells within the brain ends up in corticogenesis and improvement in vision within a amount of 6-8 days in an exceedingly placental mammal model [76-80]. Once the new differentiated cells replaced the broken cells, improved insertion was discovered within the gift study that mirrored within the SPECT scan. Author et al. showed improved eye sight of a five twelvemonth previous CP patient with twine blood vegetative cell medical care at a pair of months [80-82].

Vision plays an important role in development of gross and fine motor skills, visual-motor coordination, and walking capabilities in kids. The event of control is lacking in visually impaired kids as a result of lack of eye and hand or foot interlock. As compared with encephalopathy kids having traditional vision, the encephalopathy kids with vision impairment would like lots of help in their daily activities. we have a tendency to analyzed the GMFCS E & R immeasurable our twenty three patients in whom SPECT scan was out there and discovered that there was a motivating improvement in their scores before and once improvement in vision. Though, improvement in GMFCS E & R scores could be multi-factorial however this facet cannot be utterly dominated out [83-85].

In conclusion, the utilization of hESC medical care in patients with CVI has shown helpful results. Considering fewer therapeutic choices for the treatment of CVI in kids with CP, hESC medical care has provided hope to many patients with this condition. Though the results of the current study have shown the effectiveness of hESC medical care; any studies with larger sample size square measure a necessity for creating this therapy out there clinically.

Combination of Hippotherapy with Technical Bobath methodology in Body striated muscle management of a Patient with Tetraplegia as a result of Cerebral Palsy: Provin D, Briel AF, Guerino adult male (2012)

The hippotherapy may be a therapeutic methodology supported the follow of accomplishment activities that uses the horse as main kinesiotherapic agent. Already the Neuroevolutive thought of Bobath consecrated there square measure years, it's used of specific techniques of inhibition, facilitation and fascicle stimulation, objectifying to change commonplace bodily property tenseness and abnormal movements, facilitating commonplace motors of additional acceptable movements. Provin D, Briel AF, Guerino adult male (2012) analysis work had results with vital improvement in body part hunchback by quantifying trunk alignment, improvement of static balance at sitting position adopted attributable to the taken position while not support on the horse, improvement in time recovery and aspect recovery, improvement of MMSS muscle strength through exaggerated time within the cat position on the horse and on the platform, and tonic changes enabled by the advantages favored by the Bobath methodology techniques combination and hippotherapy.

Effectiveness of Myofascial unharnesses on jerkiness and Lower Extremity operates in Diplegic Cerebral Palsy: randomised Controlled Trial: Kumar C, Vaidya Sn (2015).

The effectiveness of Myofascial unharnesses together with typical therapy on jerkiness of calf, hamstring and adductors of hip and on lower extremity operate in spastic diplegic subjects [86-90].

Spasticity is outlined as a velocity-dependent resistance to stretch. Spastic CP is caused by injury to the pointed components of the brain. Bone and joint changes in encephalopathy result from muscle jerkiness and contraction. The spine and also the joints of the lower extremity square measure most typically affected. Spinal curvature might progress speedily and will continue once skeletal maturity. Progressive hip flexion and motion result in inhospitable deformity, exaggerated limb anteversion, apparent cotyloid joint valga and luxation, deformity of the limb head, hip dislocation, and formation of pseudoacetabulum. Within the knee, flexion contraction, patella alta, and os sesamoideum fragmentation square measure the foremost usually seen abnormalities. Progressive equinovalgus and equinovarus of the foot and articulatio talocruralis square measure related to rocker-bottom deformity and luxation of the talonavicular joint. Early recognition of progressive deformity in subjects with encephalopathy permits timely treatment and hindrance of irreversible modification.

When analysis was finished demographic info of participants, no statistically vital distinction was found showing that subjects square measure matched for baseline characteristics. There was no vital distinction between pre GMFM, pre MAS and pre MTS score in 2 teams for calf, hamstring and skeletal muscle, that shows that 2 teams square measure statistically matched at baseline level.

When comparison was done between pre and post intervention levels for each the teams, the values for MAS, MTS and GMFM score were statistically vital for all calf, hamstring and skeletal muscle.

When post intervention comparison was done between blood type and blood type it had been found that there was vital distinction between post MAS, post R1 price of MTS for calf, hamstring and skeletal muscle muscle; whereas no vital distinction was found in post R2 price of MTS and post GMFM score in between the teams.

it is in distinction with the previous study done by Alexis B. Hansen et al. on Myofascial Structural Integration terminated that myofascial structural integration may be a specific, complementary technique to loosen and line up muscles and will facilitate improved motor operate in young kids with spastic encephalopathy. Improvement in GMFM score during this study could also be as a result of longer period of intervention, relatively lower sample size and completely different treatment protocol used for core and extremity.

Another previous study done by Burris Duncan in 2008 on “effectiveness of treatment within the os field and MFR (OMT) versus stylostixis as complementary treatment for kids with CP” terminated that, a series of treatments victimization treatment within the os field, MFR, or each improved motor operate

in kids with moderate to severe spastic CP. however they didn't get improvement in jerkiness, that they themselves have proven subjective to be of import [91-93].

In MTS 2 parameters square measure thought-about that square measure quality of muscle reaction (X) and angle of muscle reaction (Y), each square measure calculated at completely different velocities. In gift study solely Y parameter is evaluated as it's believed that the X parameter within the MTS isn't acceptable in assessing the severity of jerkiness as a result of the V1 speed will solely score zero or one to not elicit the innate reflex. We have a tendency to might score three and four scores with V2 or V3 velocities however they weren't acceptable for proximal muscle teams. Throughout testing we have a tendency to discovered convulsion solely in skeletal muscle and striated muscle muscles.

The probable mechanism for results might wear down neuroreflexive modification that happens with the applying of manual force on the system whereas giving MFR. The hands on approach provide receptive stimulation through receptors, which supplies response by central process at the medulla spinalis and plant tissue levels. Receptive stimulation oftentimes ends up in motor inhibition. This principal is employed in MFR technique once the receptive stimulation of a stretch is applied and also the operator waits for motor inhibition to occur in order that relaxation results.

The accomplishment of relaxation of muscle and exaggerated flexibility of muscle through MFR; stretching, strengthening and weight bearing activities got, that helped in reducing jerkiness and in increasing lower extremity operate expeditiously. The myofascial unharness technique is resultive and subjects with spastic paralysis would expertise bigger enhancements in jerkiness as compared to traditional treatment alone in management of adverse effect as a result of jerkiness.

Conclusion:

Considering fewer therapeutic choices for the treatment of encephalopathy, hESC therapy with further studies with larger sample size are effective for the treatment of cerebral palsy.

REFERENCES

1. Masood A, Arshad R, Mazahir S (2015) Families of Children with Cerebral Palsy: Family Functioning Domains. Int J Sch Cog Psychol 2:119.
2. Nafi OA (2011) Clinical Spectrum of Cerebral Palsy in South Jordan; Analysis of 122 Cases. Pediatr Therapeut 1:101.
3. Hurley DS, Sukal-Moulton T, Gaebler-Spira D, Krosschell KJ, Pavone L, et al. (2015) Systematic Review of Cerebral Palsy Registries/ Surveillance Groups: Relationships between Registry Characteristics and Knowledge Dissemination. Int J Phys Med Rehabil 3:266.
4. Merino ST, Bonilla MDRT, Chavez BAL, Fong DM, Barrios JAG (2015) Functional Polymorphism of the Interleukin-1beta Gene Promoter is Associated with Increased Risk for Cerebral Palsy in Mexican Children with Perinatal Hypoxia-Ischemia Antecedents. J Neonatal Biol 4:167.
5. Kumar C, Vaidya SN (2015) Effectiveness of Myofascial Release on Spasticity and Lower Extremity Function in Diplegic Cerebral Palsy: Randomized Controlled Trial. Int J Phys Med Rehabil 3:253.
6. Mohamed A Jaber, Taha Allouch (2015) Dentofacial Abnormalities and Oral Health Status in Children with Cerebral Palsy. J Interdiscipl Med Dent Sci 3:164
7. Shroff G, Das L (2014) Human Embryonic Stem Cell Therapy in Cerebral Palsy Children with Cortical Visual Impairment: A Case Series of 40 Patients. J Cell Sci Ther 5:189.

8. Oliveira T, Carollo J, Robertson D, Pan Z, Heyn P (2014) Incidence of Epilepsy in Adults with Cerebral Palsy and Secondary Health Outcomes: A Review and Proposed Feasibility Study. J Neurol Disord 2:188.
9. Eseigbe EE, Anyiam JO, Wammanda RD, Obajuluw SO, Rotibi BB, et al. (2014) A Review of Gross Motor Function in Children with Cerebral Palsy in Zaria, North-Western Nigeria. Int J Phys Med Rehabil 2:236.
10. Azzam AM (2014) Efficacy of Induced Spherical Based Modified Balance Board on Improvement of Sitting Level Stage in Spastic Cerebral Palsy Children. J Nov Physiother 4:210.
11. Pate R, Chu SK, Gerstman B (2014) Prevalence of Musculoskeletal Pain in Adults with Developmental Disabilities. Int J Phys Med Rehabil S5:008.
12. Zwick D (2014) How Posture Goes Wrong: Body Shape Distortion in Cerebral Palsy. J Yoga Phys Ther 4:e115
13. Maeda K (2013) A Proposal to Reduce Congenital Cerebral Palsy. J Health Med Informat 4:135.
14. Loudon JA (2013) Repurposing Amlexanox as a 'Run the Red Light Cure- All' with Read-through – a 'No-Nonsense' Approach to Personalised Medicine. J Bioanal Biomed 5:079-096.
15. Venkata Krishnan R (2013) Restoring Motor functions in Spinal cord injury, Hemiplegic Cerebral Palsy, and Stroke by Botulinum toxin-induced Synaptic Competitive-Learning Therapy. J Neurol Disord 1:134.
16. Bugajski S, Christian A, Oâ€™Shea RK, Vendrely AM (2013) Exploring Yogaâ€™s Effects on Impairments and Functional Limitations for a Nine-Year-Old Female with Cerebral Palsy: A Case Report. J Yoga Phys Ther 3:140.
17. Jr HL, Joshi A, Lorenz Z, Miller F, Dabney K (2013) Pediatric Cerebral Palsy Life Expectancy: Has Survival Improved Over Time? Pediat Therapeut 3:146.
18. Kirton A (2013) Modulation of Developmental Plasticity with Non-Invasive Brain Stimulation in Cerebral Palsy. Int J Phys Med Rehabil 1:135.
19. Sharma A, Kulkarni P, Sane H, Gokulchandran N, Badhe P, et al.(2012) Positron Emission Tomography-Computed Tomography Scan Captures the Effects of Cellular Therapy in a Case of Cerebral Palsy. J Clin Case Rep 2:195.
20. Azzam AM (2012) Effect of Hand Function Training on Improvement of Hand Grip Strength in Hemiplegic Cerebral Palsy in Children. J Nov Physiother 2:116.
21. Provin D, Briel AF, Guerino MR (2012) Combination of Hippotherapy with Technical Bobath Method in Body Extensor Control of a Patient with Tetraplegia due to Cerebral Palsy. J Nov Physiother 2:111.
22. Zadnikar M, Rugelj D (2011) Postural Stability after Hippotherapy in an Adolescent with Cerebral Palsy. J Nov Physiother 1:106.

23. Berry T, Howcroft CJ, Klejman S, Fehlings PED, Wright V, et al. (2011) Variations in Movement Patterns during Active Video Game Play in Children with Cerebral Palsy. J Bioeng Biomed Sci S1:001.
24. hao Y (2015) The Images of Primary Glioblastoma Cells Isolated from Human Glioma Specimens. Zhao et al., Brain Disord 4:i102.
25. Alsaadi T, Shahrou TM (2015) Depressive Disorders in Patients with Epilepsy: Underdiagnosed and Appropriately Managed?. Brain Disord Ther 4:163.
26. Allakhverdieva AE, Grant AR, Quon LJ, Ricciardi SA, Diluna ML (2015) Extradural Decompression for the Treatment of Oculomotor and Oropharyngeal Symptoms in Chiari I Malformation: A Case Report . Brain Disord Ther 4:162.
27. Litovchenko T, Iakubenko I (2015) Identification of Disturbances of Autoregulation of Cerebral Hemodynamics and Blood Vessels Reactivity in Patients with Consequences of Mild Traumatic Brain Injuries. Brain Disord Ther 4:161.
28. Almushayti Z, Almuhaish H (2015) Unusual CT and MRI? Appearance** of an Epidermoid Cyst: A Case Report. J Blood Disord Transfus 4:160.
29. Gomez AMG, Vasquez JGF, Alquezar AL, Salvat GQ, Nada MM, et al. (2015) Clinical Functioning in a Cohort of Patients with Severe Mental Disorder, before and after Joining a Workplace Reintegration Program. Brain Disord Ther 4:159.
30. Flavia M, Chiara S, Patrizia P (2015) Left Tactile Agnosia Amelioration by Prism Adaptation Sustains Unilateral Spatial Neglect-Based Hypothesis. Brain Disord Ther 4:158.
31. Naisberg Y (2015) Macro Biophysical Physiological Neuropsychiatry. Brain Disord Ther 4:157.
32. Tortolero GS, Fragoso M, Espanol G, Estevez M, Rey A (2015) EEG Findings in Diffuse Lewy Body Disease and Parkinson´S Disease with Dementia. Brain Disord Ther 4:156.
33. Sivathanu S, Sampath S (2015) Childhood Chronic Inflammatory Demyelinating Polyneuropathy – A Report of Two Cases. Brain Disord Ther 4:155.
34. Xie F, Ma X (2015) Molecular Hydrogen and its Potential Application in Therapy of Brain Disorders. Brain Disord Ther 4:154
35. Klingner CM, Brodoehl S, Hohenstein C, Winning J, Kummer L et al. (2015) A Case with 7 Min Door-To-Needle-Time and an Outline of Ultrarapid Stroke Management. Brain Disord Ther 4:153.
36. Fang C, Garbuzova-Davis S, Tan J, Obregon D (2015) C1q as a Regulator of Brain Development: Implications for Autism Spectrum Disorders. Brain Disord Ther 3:152.
37. Masters A, Pandi-Perumal SR, Seixas A, Girardin Jean-Louis G, McFarlane SI (2015) Melatonin, the Hormone of Darkness: From Sleep Promotion to Ebola Treatment. Brain Disord Ther 3:151.
38. Wang XH, Sperry L, Olichney J, Farias ST, Shahlaie K, et al. (2015) Impact of Deep Brain Stimulation Therapy on Autonomic Disturbances and Related Symptoms of Parkinson's Disease. Brain Disord Ther 4:150.

39. Hahn MK, Sylvia Gomes S, Remington GJ (2015) Low-Dose, Off-Label Quetiapine Use, Metabolic Syndrome and Impaired Fasting Glucose in an Elderly Man: A Case Report. Brain Disord Ther 4:149.
40. Hori K, Konishi K, Tani M, Akashi N, Kitajima Y, et al. (2015) Anti-Dementia Agents are Partially Symptomatic Treatment and Partially Disease Modifying Treatment. Brain Disord Ther 4:148.
41. Naveed S, Hameed A, Nadeem SM (2015) Knowledge of Amyotrophic Lateral Sclerosis (ALS) in Pharmacy Students. Brain DisordTher 4:147.
42. Bailus BJ (2014) Variations on a Theme: Crispr Models for 15q11-Q13 Disorders and Beyond. Brain Disord Ther 3:146.
43. Quliti KWA (2014) A Case Study of Partial Seizure with Secondary Generalization Induced by Clozapine in Patient with Treatment Resistant Schizophrenia. Brain Disord Ther 3:145.
44. Tanaka H, Hori K, Inamoto A (2014) Relationship with Bipolar Temperament and Behavioral and Psychological Symptoms of Dementia in Alzheimerâ€™s Disease. Brain Disord Ther 3:144.
45. Larson EW, Peterson HE, Lamoreaux WT, MacKay AR, Fairbanks RK, et al. (2014) Case Series: Gamma Knife as Salvage Therapy for Recurrent Glioblastoma Multiforme. Brain Disord Ther 3:143.
46. Saad S and Wang TJC (2014) Neurocognitive Deficits after Radiation Therapy for Brain Malignancies. Brain Disord Ther 3:138.
47. Leroi I, Perera N, Harbishettar V, Robert P (2014) Apathy and Emotional Blunting in Parkinsonâ€™s Disease. Brain Disord Ther 3:141
48. Shivaramegowda NH, Borkar SA, Garg K, Suri V, Sharma MC, et al. (2014) Central Neurocytomas: A Comprehensive Review with Special Emphasis on the Role of Gamma Knife Stereotactic Radiosurgery. J Hematol Thrombo Dis 3:140.
49. Mait P, Manna J (2014) Activation of Heat Shock Proteins by Nanocurcumin to Prevent Neurodegenerative Diseases. Brain Disord Ther 3:139.
50. Nejtek VA (2014) Diffusion Tensor Imaging in Bipolar Disorder with Cocaine Dependence vs. a Healthy Control: Preliminary Findings. Brain Disord Ther 3:142.
51. Loutfi G, Linder J, Hariz GM, Hariz M, Blomstedt P (2014) Pallidal Deep Brain Stimulation in the Treatment of Huntingtonâ€™s Chorea. Brain Disord Ther 3:136.
52. Rakesh B, Bharath S, Bagepally BS, Saini J, Sadanand S, et al. (2014) A Retrospective Study on Relation between Cognitive Performance and Lobar Perfusions of Brain in Alzheimerâ€™s Dementia using Single Photon Emission Computer Tomography . Brain Disord Ther 3:135.
53. Mitoma H, Yoneyama M (2014) A Newly Developed Wearable Device for Continuous Measurement of Gait-Induced Accelerations in Daily Activities . Brain Disord Ther 3:134.
54. jung YG (2013) Capital-Skill Complementarity and Jobless Recovery. J Stock Forex Trad 2:104.
55. Clark JF, Middendorf A, Hasselfeld KA, Ellis JK, Divine J (2014) Aggressive Rehabilitation Pathway Targeting Concussion Symptoms: Illustration with a Case Study. Brain Disord Ther 3:131.

56. Rajamani U (2014) Causes of Neurodegeneration in Diabetes: Possible Culprits and Therapeutic Targets. Brain Disord Ther 3:130.
57. Settle T, Klandorf H (2014) The Role of Uric Acid as an Antioxidant in Neurodegenerative Disease Pathogenesis. Brain Disord Ther 3:129.
58. Lisotto C, Savi L, Pinessi L, Guidotti M, Omboni S, et al. (2014) Efficacy of Frovatriptan vs. Other Triptans in Weekend Migraine: Pooled Analysis of Three Double-Blind, Randomized, Crossover, Multicenter Studies. Brain Disord Ther 3:128.
59. David Andres PM (2014) Trem2 Variants and Risk of Alzheimerâ€™s Disease. Brain Disord Ther 3:127.
60. Paris D, Beaulieu-Abdelahad D, Ait-Ghezala G, Mathura V, Verma M, et al, (2014) Anatabine Attenuates Tau Phosphorylation and Oligomerization in P301S Tau Transgenic Mice. Brain Disord Ther 3:126.
61. Li YY,Zhoua ZH, Wang XF, Xiao N, Chen YM (2014) The Contrast of Efficacy and Safety between Carbamazepine and Other Antiepileptic Drugs in Patients with Postencephalitic Epilepsy: Experience from a Developing Country. Brain Disord Ther 3:125.
62. Umetsu M, Fukumoto K, Sakurai S, Sakai A (2014) Circadian Rhythm Sleep Disorder in Alzheimerâ€™s Disease. Brain Disord Ther 3:124.
63. Burhanoglu S, Sayar GH, Islk U, Arikan Z, Cosar B et al. (2014) Differences in Executive Functions and Problem Solving Styles of Protracted Sober and Relapsed Alcohol-dependent Patients. Brain Disord Ther 3:123.
64. Timofeeva E, Calvez J (2014) Neuronal Substrate of Eating Disorders. Brain Disord Ther 3:121.
65. Spector R (2014) Vitamin Transport Diseases of Brain: Focus on Folates, Thiamine and Riboflavin. Brain Disord Ther 3:120.
66. Polussa J, Schneider A, Hagerman R (2014) Molecular Advances Leading to Treatment Implications for Fragile X Premutation Carriers. Brain Disord Ther 3:119.
67. Gharibzadeh S, Darvishi A, Darvishi M (2014) Reactivation of NMDA Receptors by Synaptic Reentry Reinforcement, a Probable Cause of Auditory Hallucination in Schizophrenia. Brain DisordTher 3:118.
68. Narula S, Waldman AT and Banwell B (2014) Clinical Trials in Pediatric Multiple Sclerosis: Pending Landscape and Challenges . Brain Disord Ther 3:117
69. Mehanna R (2014) Cognitive Changes after Deep Brain Stimulation in Parkinsonâ€™s Disease: A Critical Review. Brain Disord Ther 3:116.
70. Tejada-Simon (2014) Small GTP-binding Proteins: A Future for the Treatment of Cognitive Disorders?. Brain Disord Ther 3:114.
71. Zhao Q, Luo JJ (2014) Epilepsy in Elderly. Brain Disord Ther 3:115.
72. Mathur T, et al. Mathur S (2014) A Case of Glioblastoma Multiforme Masquerading as Rapidly Progressive Dementia. Brain Disord Ther 3:113.

73. Pirker-Kees A, Schmied C, Dal-Bianco P (2013) T-Cells Show Increased Production of Cytokines and Activation Markers in Alzheimerâ€™s Disease. Brain Disord Ther 3:112.
74. Sasani F, Javanbakht J, Nourmohammadzadeh F, Hassan AM, Moghaddam MR, et al. (2013) Clinical, Pathological, and Hematological Study of Post-Injection Neuritis of the Sciatic Nerve by Alfatrim 24% Intramuscular Injection in Small Ruminants. Brain Disord Ther 2:111.
75. Radaei F, Gharibzadeh S (2013) The Increase of AMP-activated Protein Kinase during Exercise and its Effect on Reducing Parkinsonâ€™s Disease Symptoms. Brain Disord Ther 2:110.
76. Fang P, Li X, Luo JJ, Wang H, Yang X (2013) A Double-edged Sword: Uric Acid and Neurological Disorders. Brain Disord Ther 2:109.
77. Yokoyama S,Aoki M, Azuma K, Jimbo K, Konishi K, et al. (2013) Escitalopram for Delusion in an Oldest Old Patient with Alzheimerâ€™s Disease. Brain Disord Ther 2:108.
78. Mitoma H, Nanri K, Mizusawa H (2013) Is Anti-Gliadin Antibody Pathogenic in Gluten Ataxia? Analysis using Rat Cerebellar Slices and Patch-Clamp Recording. Brain Disord Ther 2:105.
79. Radaei F, Gharibzadeh S (2013) The Effect of Carbidopa in Carbidopa- Levodopa Combination on Reducing Osteoporotic Symptoms in Parkinsonâ€™s Disease Patients. Brain Disord Ther 2:107.
80. Hori K, Konishi K, Tanaka H, Yokoyama S, Aoki M, et al. (2013) Mini Review: Pharmacotherapy for Behavioral and Psychological Symptoms in Alzheimerâ€™s Disease. Brain Disord Ther 2:106.
81. Jacob A, Cohen S (2013) A Biomedical Imaging Analysis of the Prevalent Neuropsychiatric Disorders. J Alzheimers Dis Parkinsonism 3: 117.
82. Saleem W, Broderick PA (2013) Biomarkers for Brain Disorders Electrochemically Detected By BRODERICK PROBEÂ® Microelectrodes/Biosensors. J Biosens Bioelectron S12:003.
83. Mizui T, Kojima M (2013) BDNF and Synaptic Plasticity: The Recent Cell Biology for Understanding of Brain Disorders. Clin Pharmacol Biopharm S1:004.
84. Niu Y, Li H, Herrup K, Zhang J (2012) Neuronal Cell Cycle Regulation of Cdk5 in Alzheimer's Disease. Brain Disord Ther S1:004
85. Pareek TK, Zipp L, Letterio JJ (2012) Cdk5: An Emerging Kinase in Pain Signaling. Brain Disorders Ther S1:003.
86. McLinden KA, Trunova S, Giniger E (2012) At the Fulcrum in Health and Disease: Cdk5 and the Balancing Acts of Neuronal Structure and Physiology. Brain Disorders Ther S1:001.
87. Amin ND, Grant P, Zheng Y, Kesavapany S, Pant HC (2012) Septin Phosphorylation and Neuronal Degeneration; Role of Cyclin Dependent Kinase 5 (Cdk5). Brain Disorders Ther S1:002.
88. Hagerman RJ, Pak JS, Ortigas M, Olichney J, Frysinger R, et al. (2012) Case Series: Deep Brain Stimulation in Patients with FXTAS. Brain Disord Ther 1:104.
89. Erdem H, Uzunlar KA, Yildirim U, SAV A, Dosoglu M (2012) Collision Tumor of Meningioma and Non Hodgkin Malignant Lymphoma of Cerebellum. Brain Disorders Ther 1:103.

90. Hayashi Y, Kimura M, Kinoshita A, Hamada JI (2012) Occlusion of an Incidentally-Found Dissecting Aneurysm of the Vertebral Artery at Removal of a Jugular Tubercle Meningioma. Brain Disorders Ther 1:102.
91. Jim HSL, Boyd TD, Booth-Jones M, Pidala J, Potter H (2012) Granulocyte Macrophage Colony Stimulating Factor Treatment is Associated with Improved Cognition in Cancer Patients. Brain Disorders Ther 1:101.
92. Wang H (2012) Rett Syndrome: Translate Medicine from Brain to Heart. Brain Disord Ther 1:e101.
93. Baslow MH, Burlina AP (2012) N-Acetylaspartate Metabolism Underlays the Structural and Functional Units of the Vertebrate Brain: A Bioenergetic Rationale for Clinical Observations of Changes in the Neuronal Biomarker â€œNAAâ€□ in many Human Brain Disorders. Bioenerg Open Access 1:102.