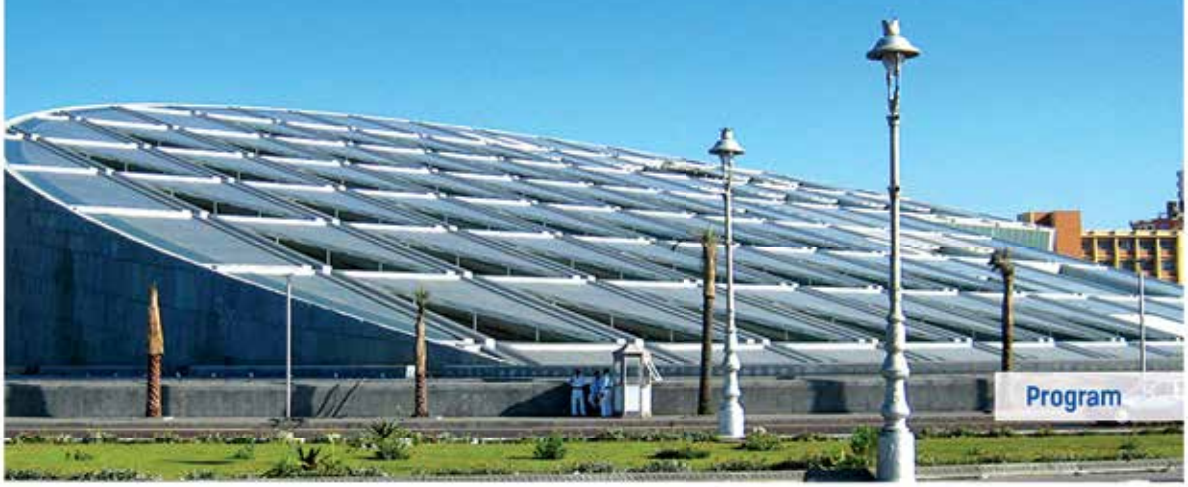
Annual Summer Meeting

of The Egyptian Society of Neurology, Psychiatry and Neurosurgery

In collaboration with

Neuropsychiatry Department, Faculty of Medicine
Alexandria University

11th – 13th June, 2015 Sheraton Hotel – Alexandria



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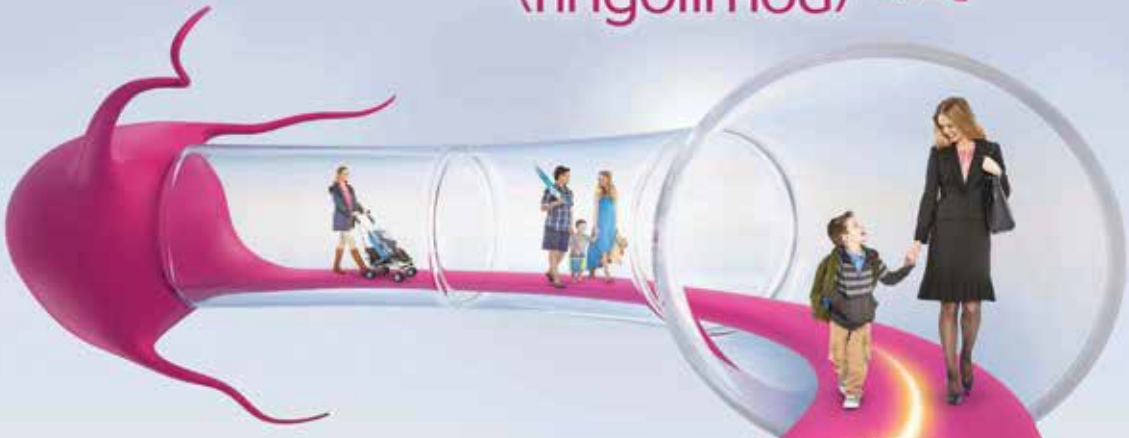

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Welcome Message

On behalf of the Egyptian society of Neurology, Psychiatry and Neurosurgery, I have the pleasure and honor to invite you to actively participate in the ESNPN Conference, which will be conducted in Sheraton Hotel Alexandria, , 11th to 13th June 2015 – Alexandria, Egypt.

The scientific Program is rich, including updated subjects in different fields of Neurology and psychiatry.

Interesting lectures by eminent neurologists and Psychiatrists, as well as free papers in all fields and lastly round table discussion in very interesting topics (Stroke, Epilepsy, MS, Alzheimer Disease, and Neuro-Therapeutics).

My dear Professors and colleagues the scientific activity will be coupled with interesting social activity and the opportunity to relax in such a lovely spot.

Prof. Hassan Farwiz
Hassan Farwiz
President of the Society & Congress

Prof. Mohamed Fouad Boraey
Mohamed Fouad Boraey
Head of Department of Neuropsychiatry,
University of Alexandria

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Thursday
11th June 2015

Registration — 01:00 -03:30 PM

01:00 – 03:30 PM	Registration
02:00 – 03:00 PM	Lunch (Hotel)

Thursday
11th June 2015

Opening Ceremony

03:30 – 04:00 PM

Prof. Hassan Farweez
(ESNPN President)

Prof. Mohamed Fouad Boraey
(Head of Department of Neuropsychiatry, University of Alexandria)

Prof. Maged Abdel Naseer
(ESNPN Secretary General)

Thursday
11th June 2015

Plenary Session

04:00 – 05:45 PM

Chairpersons

(Alphabetically)

Ammar El-Taher
Mohamed S. El-Tamawy
Osama Abdulghani

It is Myasthenia Gravis
Ammar El-Taher 30 MIN

The selection of AED for the treatment of epilepsy
Farouk Talaat 45 MIN

Mental state changes in dementai patients
Fathy Afifi 30 MIN

Thursday
11th June 2015

New Bridge Symposium

05:45 – 06:15 PM

Chairpersons

(Alphabetically)

Ahmed Deif
Azza Abbas
Hassan Farawiz

Drug Management in Parkinson's disease
Maged Abdel Naseer 30 MIN

06:15 – 06:45 PM Coffee Break

Thursday
11th June 2015

Merck Symposium

06:45 – 07:45 PM

Chairpersons

(Alphabetically)

Mohamed Fouad
Samia Ashour
Sayed Tag El-Din

Smokes and Mirrors
Magd Fouad Zakaria 20 MIN

Delaying Disability Progression, where experience counts
Ayman Ezz El-Din 20 MIN

Impact of stopping B. Interferon on relapsing remitting multiple sclerosis
Mabroka Agheila 20 MIN

Thursday
11th June 2015

Novartis Symposium

07:45 – 08:45 PM

NOVARTIS

Chairpersons

(Alphabetically)

Mohamed Ramadan
Mohamed Saad
Nabil El-Agouz

Defining the clinical course of multiple sclerosis
Ismail Ramadan 20 MIN

Fatigue and depression in multiple sclerosis
Ayman Nassef 20 MIN

Implementation of 'NEDA-4' in multiple sclerosis decision model. Uncover timely treatment failure
Hatem samir 20 MIN

Thursday
11th June 2015

Bayer Symposium

08:45 – 09:15 PM

Bayer HealthCare
Bayer Schering Pharma

Chairpersons

(Alphabetically)

Abdallah Maamoun Sarhan
Ashraf Abdou
Ismail Montaser

Axonal loss in Multiple Sclerosis

Tarek Tawfik — 30 MIN

09:15 – 11:00 PM Dinner (Hosny Restaurant)

Friday
12th June 2015

Session I

10:30 –12:00 AM

Chairpersons

(Alphabetically)

Nadia Hafez
Obsis Madkour
Smaiha Abd El-Moneim

Endocrine myopathies
Samir Asaad 30 MIN

The Carpal Tunnel Syndrome and the Double Crush Syndrome Hypothesis: Revisited
Heba Raafat 15 MIN

Bone Marrow Transplantation (Neurological indications and complications)
Hany Mohammed El Deeb 15 MIN

Early predictors of subclinical atherosclerosis in epilepsy patients
Mostafa Mohamed Magdy 15 MIN

Effect of Natalizumab on clinical activity/radiological and EDSS of relapsing remitting multiple sclerosis
Mabroka Agheila 15 MIN

12:00 – 01:00 PM Gomaa Prayer

Friday
12th June 2015

Session II

01:00 – 02:00 PM

Chairpersons

(Alphabetically)

Gamal Azab
Wael Fadel
Yousria El-Taweel

Challenges in the care of stroke survivors in Zagazig university hospitals
Wafaa S. Mohamed 15 MIN

Cerebral venous thrombosis
Amr El-Fatatry 15 MIN

Quantitative Assessment of Shoulder Proprioception in Patients with Stroke
Abdelaziz A. Elsherif 15 MIN

Transcranial Direct Current Stimulation (tDCS) in Stroke Rehabilitation
Islam F. Halawa 15 MIN

Friday
12th June 2015

Session III

02:00 – 03:00 PM

Chairpersons

(Alphabetically)

Amira Zaky
Eman Khedr
Magdi Aidaros

Endocrine myopathies
Samir Asaad 30 MIN

The different classification of personality disorders.
Tarek Molokhia 15 MIN

Insomnia
Jaidaa Mekky 15 MIN

Anti-angiogenic Therapy for malignant glioma
Sherine El Mously 15 MIN

Gamma knife Radiosurgery for Peri-optic Meningiomas
Hossam Maaty 15 MIN

03:00 – 05:00 PM Lunch (Hosny Restaurant)

09:00 PM Gala Dinner (Hotel)

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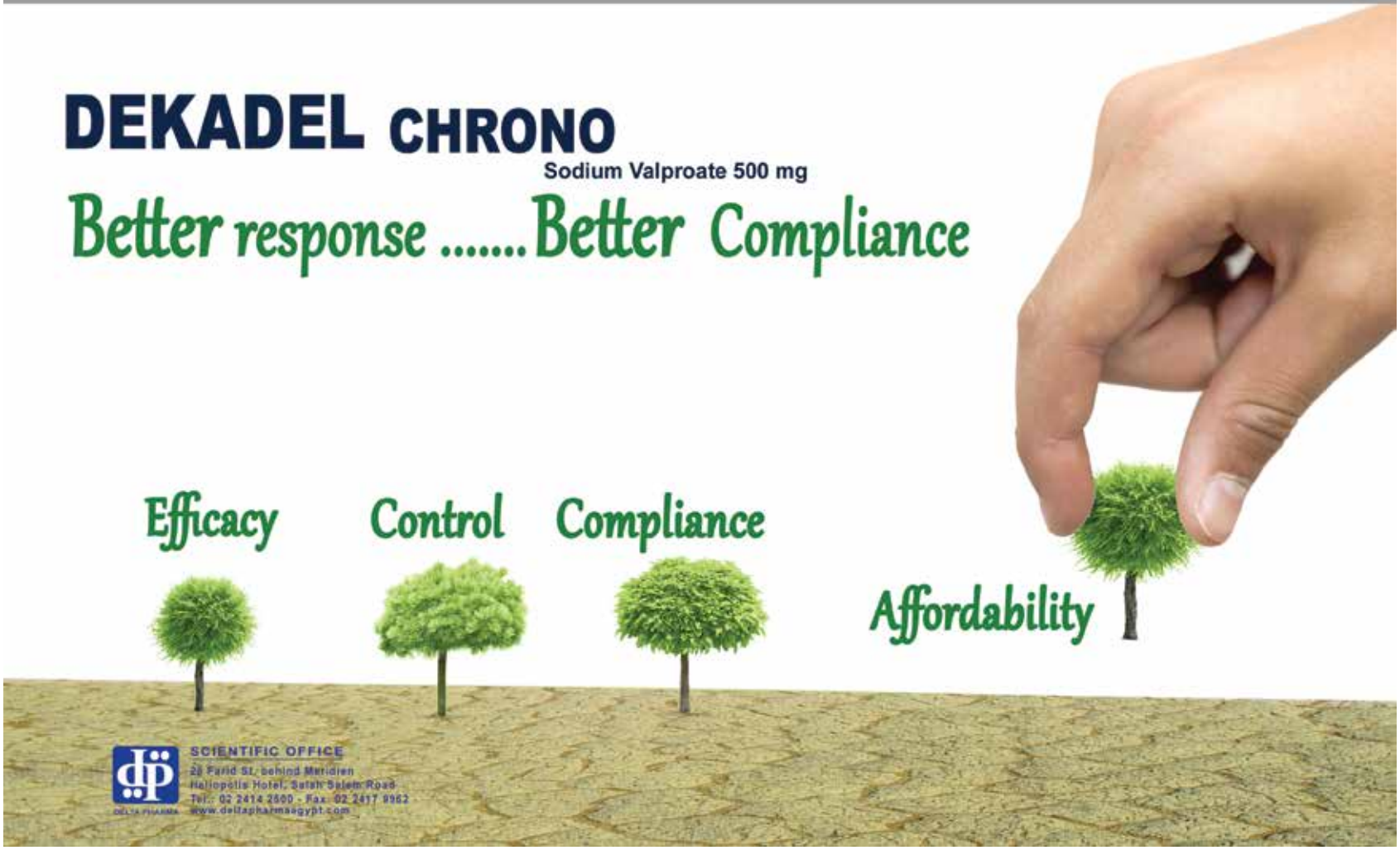

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Abstracts

E.S.N.P.N
2015

Defining the clinical course of multiple sclerosis

Ismail Ramadan

Accurate clinical course descriptions of MS are important for communication,design and recruitment of clinical trials and treatment decision making The descriptions published in 1996 were lacking imaging and biological correlates So the need for new definitions of the clinical course of MS was mandatory.

Fatigue and depression in multiple sclerosis

Ayman Nasef

In this presentation we will discuss the following
1-How common is depression in people with MS?
2- Is depression in MS associated with lesions in
specific regions of the central nervous system?
3- Is there an increased risk of suicide in MS?
4- Is there a higher than expected incidence of anxiety disorders in MS?
5- Are fatigue and depressed mood related in MS?
6- Is there a relation between depression and cognitive impairment in MS?
7- Which psychosocial variables affect the development of depression in MS?
8-Does treatment with interferon increase the risk of depression?
9- How effective are treatments for MS patients with depression?

ENDOCRINE MYOPATHIES

Samir Naim
Assaad, MD, MRCP(UK), FRCP(Edin), FRCP(Lond)
Professor of Medicine & Endocrinology
Faculty of Medicine, University of Alexandria

Secondary limb myopathy is more frequent than primary or hereditary forms. The most common forms of endocrine myopathies are related to thyroid disorders and hypercortisolemia.. Hypothyroidism is commonly associated with proximal myopathy and increased CPK levels. Hyperthyroidism induces proximal muscle weakness and occasionally complicated by periodic hypokalemic muscular paralysis. Cushing syndrome, whether exogenous or endogenous, is characterized by proximal myo-

pathy which is considered as a cardinal feature of this syndrome.
Other endocrinopathies as acromegaly, hyperparathyroidism, Addison's disease can also be associated with myopathy. Diabetes mellitus uncommonly can be complicated with proximal motor neuropathy which presents with painful proximal muscle weakness. Statin use for treatment of diabetic dyslipidemia is rarely linked to myopathy and rhabdomyolysis.
Endocrinopathies can occur with other immune disorders involving muscles as dermatomyositis.
Vitamin D is now considered as a true hormone rather than a classical vitamin.
Vitamin D deficiency is not uncommon in our area. It can present with myopathy with characteristic elevation of serum alkaline phosphatase and parathormone.
It should be excluded in any patient presenting with myopathy.

The Carpal Tunnel Syndrome and The Double Crush Syndrome Hypothesis: Revisited

Heba Raafat[1], Mye A. Basheer[1],Radwa Azmy[1] , Amira A. Labib[1]

Clinical Neurophysiology Unit, Faculty of Medicine, Cairo University, Egypt Background: The commonest application of the double crush hypothesis is its association to median nerve entrapment at the wrist. The double crush (DC) concept has gained popularity because it provides a rationale for evaluating the cervical spine and roots when treating carpal tunnel syndrome (CTS). The double crush syndrome (DCS) and CTS co-occur more than would be likely of CTS to occur alone.
Up to 90% of CTS patients are misdiagnosed as only 10% have the problem at theirwrist.
Objective: To examine the validity of Double Crush Syndrome hypothesis in CTS patients to support or disregard the theory.
Methods: This study was conducted on 80 patients, 40 patients presenting with brachialgia (Group I) and 40 claiming failed CT release operation (Group II) .
Diagnostic work up included neurological examination, MRI of cervical spine, Phalen test and Tinel sign, electromyographic examination (EMG) and motor and sensory nerve conduction studies (NCS).
Results: EMG and NCS showed 10 cases with CTS (25%), 20 (50%) with double crush syndrome and 10 (25%) with cervical radiculopathy in group I, while group II
patients showed 10 cases (25%) with CTS and 30 (75%) with DCS. In all patients, 50 cases (62.5%) showed DCS. When comparing the first NCS of Group II pre and postoperative, 32 cases (80%) showed improvement of the NCS results while 8 (20%) deteriorated.
Conclusion: In this study, DC hypothesis was supported while the concept of frequent failure of CT release surgery was not supported for the benefit of DC hypothesis.
Keywords: Carpal tunnel syndrome, double crush syndrome, electrophysiological studies, cervical radiculopathy.

Bone Marrow Transplantation (Neurological indications and complications)

Hany Mohammed El Deeb
Assistant lecturer of neuropsychiatry
Alexandria University

Bone Marrow Transplantation involves the use of hematopoietic cells for many hematological and non-hematological indications. There are many neurological complications that may occur during different stages of transplant of special concern is graft versus host disease that affects nervous system in its chronic form. Bone Marrow Transplantation has been tested in many inherited as well as acquired neurological disorders. It proved success in some leukodystrophies, lysosomal disease and some acquired autoimmune diseases.

Early predictors of subclinical atherosclerosis in epilepsy patients

Hala Abd Elmageed Shaheen, Sayed Sobhy Sayed, Lamiaa Ibrahim Abdel Azeem, Mostafa Mohamed Magdy
Neurology department, Fayoum University.

Background: Patients with epilepsy are at higher risk for atherosclerosis which may be due to the epilepsy itself and/or antiepileptic drugs use (AED). This work aimed to detect the impact of epilepsy itself and the antiepileptic drugs on developing subclinical atherosclerotic changes and to correlate atherosclerosis in patients with epilepsy to clinical and laboratory data.
Patients and Methods: Ninety patients with idiopathic epilepsy and 30 age, sex matched healthy controls subjected to neurological examination, extracranial carotid duplex, and measurement of lipid profile, uric acid and CRP levels.
Results: The level of HDL was significantly lower in all patients with epilepsy and those treated with enzyme inducer antiepileptic drugs than the control subjects.
Level of serum uric acid was statistically significantly higher in all patients with epilepsy including untreated patients and those treated with non enzyme inducer AEDs and polytherapy AEDs than control subjects. The mean common carotid artery intima media thickness (CCA IMT) was significantly higher in all patients with epilepsy including untreated and treated patients than control. There was a significant positive correlation between the CCA IMT and age of the patients, duration of illness and duration of the antiepileptic drugs.
Conclusion: The epilepsy itself could result in subclinical atherosclerotic changes in the patients with epilepsy, which could be exacerbated by the antiepileptic drugs,particularly enzyme inducer drugs.
Key words: Epilepsy, AEDs, atherosclerotic risk factors, CCA-IMT.

CHALLENGES IN THE CARE OF STROKE SURVIVORS IN ZAGAZIG UNIVERSTY HOSPITALS

Yosria A. Altaweel, Amal S E. El-Motayam, Wafaa S. Mohamed, Amany wafa
Neurology Department, Faculty of Medicine, Zagazig University.

Background : stroke is a global health-care problem with a life-changing event that affects not only the patients, but also their families, caregivers and the society Aim of the work: To identify challenges in the care of stroke survivors in our culture and to assess their effect on health related quality of life (HRQL).
Methods: an epidemiological cross –section study was done including 21 males and 24 females with ages ranged from 30-88 years (mean age 59.5 ± 12.7 years) with first stroke , confirmed by CT and /or MRI brain. They were recruited from Neurology Department, Zagazig University, 1-3 months post stroke onset. Every patient was subjected to thorough history taking, general, neurological examination and routine laboratory investigations. The following scales were applied: modified Rankin scale, Barthel Index and Stroke Impact Scale (SIS).
Results : studying socio-demographic data revealed that older ages > 60 years had a statistically significant poorer HRQL, while low socioeconomic level, lack of social support, poor treatment compliance and absence of rehabilitation were highly statistically significance.
Stroke survivors with comorbid illness had non statistically significant poor HRQL Motor, sensory deficits and sphincteric disturbances had highly statistically significant effect on SIS while aphasia, cranial nerve affections, incoordination ,and hemianopia had statistically significant effect, larger size lesion had highly statistically significant effect. The patients with unfavourable functional outcome and severe disability were associated with poor HRQL. The lowest scores of SIS were in social participation, mobility,and hand function (39.7, 40.6, 41.5 % respectively).
Conclusion: Many factors affected HRQL of stroke survivors as socio-demographic factors, comorbid illnesses, neurological sequelaes and size of the lesion.
Key words: Health related quality of life, stroke survivors, challenges of post stroke care, stroke impact scale.

Cerebral venous thrombosis

Amr El-Fatatry

Cerebral venous thrombosis is sometimes a confusing clinical condition. It hasnonspecific clinical presentation, subtle imaging findings. Although these findings are often present on initial scans, they are frequently detected only in retrospect. So this presentation aims at identifying the pathogenesis, specific clinical features and different imaging modalities used in the diagnosis of cerebral venous thrombosis.

Quantitative Assessment of Shoulder Proprioception in Patients With Stroke

Moshera H. Darwish[1], Sandra M. Ahmed[2], Mohammed S. Eltamawy[2], Abdelaziz A. Elsherif[1]

Department of Physical Therapy for Neuromuscular Disorder and Its Surgery, Faculty of Physical Therapy, Cairo University1; Department of neurology, Faculty of Medicine, Cairo University2 Background: Impairment of proprioception in the upper extremity may impede activities of daily living and limit motor gains after stroke.

The aim of this study was to assess and evaluate objectively shoulder proprioception (joint position sense) in affected (contralesional) and unaffected (ipsilesional) side of stroke patients.

Methods: Thirty stroke patients from both sexes (study group, G1) and thirty normal matched subjects (age, sex, weight and height) (control group, G2) participated in this study.

The age of the patients ranged from 48 to 63 years and the duration of illness was more than six months post stroke. Joint position sense (JPS) was assessed by the Biodex system 3 Isokinetic dynamometer through determining angular displacement error of active and passive angle repositioning of shoulder external and internal rotation .

Assessment procedures applied on both shoulders in patients' group (G1) and on the shoulder of the dominant arm only of normal subjects in control group (G2).

The results: revealed a significant increase in the mean values of errors in the affected arm in all tested movements (active and passive external rotation, active and passive internal rotation) comparing the mean values of errors in the affected arm (G1) with the mean values of errors of both the dominant arm of normal subject (G2) and the unaffected arm (G1) ($p<0.05$). A non-significant difference of all tested movements between the mean values of errors in the unaffected arm (G1) and the dominant arm of normal subject (G2). A significant increase in the mean values of errors in passive external and internal

rotation in the affected arm and passive internal rotation in the unaffected arm when the lesion was cortical rather than subcortical ($p<0.05$). Concerning the side of the lesion there was only a tendency to significantly higher error in the passive internal rotation of the affected arm if the lesion was on the right rather than the left or there was a bilateral brain lesion. Conclusion: Proprioceptive deficit is evident in the affected shoulder in hemiparetic patients. Physical therapy programs must focus on proprioceptive training for better functional outcome. Key Words: Stroke, Shoulder, Proprioception, Joint position sense, Isokinetic dynamometer.

Transcranial Direct Current Stimulation (tDCS) in Stroke Rehabilitation

Islam F. Halawa, Msc.
Assistant researcher National Research center Clinical Neurophysiology

Transcranial direct current stimulation (TDCS) is an emerging technique of noninvasive brain stimulation that has been found useful in examining cortical function in healthy subjects and in facilitating treatments of various neurologic disorders. A better understanding of adaptive and maladaptive poststroke neuroplasticity and its modulation through noninvasive brain stimulation has opened up experimental treatment options using TDCS for patients recovering from stroke. We review the role of TDCS as a facilitator of stroke recovery, the different modes of TDCS, and the potential mechanisms underlying the neural effects of TDCS.

Key word: Transcranial direct current stimulation (TDCS); Stroke; Anodal; Cathodal; stimulation; neuroplasticity; neuromodulation; rehabilitation.

The different classification of personality disorders.

Tarek molokhia
Professor of psychiatry Alexandria University
Head of the psychiatric unit Alexandria University

Also will enlighten the dilemma of personality disorder in Axis I versus Axis II in the American classification. The presentation will discuss the dichotomies versus the description classification of the personality disorders.

At the end will be examples of this dilemma of classification concerning "borderline and narcissistic personality disorder "

Insomnia

Jaidaa Mekky
Lecturer of Neurology
Department of Neuropsychiatry, Alexandria University

Subjective patient complaint of difficulty falling asleep, difficulty staying asleep, poor quality sleep, or inadequate sleep despite adequate opportunity. Hereby we will highlight some of the causes, health consequences, how to investigate and treatment options of insomnia

Anti-angiogenic Therapy for malignant glioma

Sherine El Mously
MBBCH; MD, Lecturer of Neurology, Fayoum University

Malignant gliomas encompassing both WHO grade III and grade IV (Glioblastoma Multiforme; GBM), are the most common malignant primary brain tumors in adults. Despite advances in our understanding about how these tumors develop and proliferate, they present a therapeutic challenge that are physiologic related to anatomy and tissue sensitivity to therapy, as well as immunologic related to immunosuppression in the neoplasm microenvironment.

Malignant gliomas are not curable and the aim of the treatment is to delay the time to recurrence at which treatment responses are very limited.

Malignant gliomas are likely to be one of the most angiogenic cancers. They express specific angiogenic and tumorigenic markers that are useful in predicting therapeutic responses, grading of tumor and prognosis. Factors involved in angiogenesis are targets for multiple clinical trials. Though antiangiogenic therapy has not yet been shown to extend overall survival in this patient population, there is likely substantial benefit by reducing vasogenic edema, allowing for temporary improvement in neurologic function, and minimizing the side effects of prolonged corticosteroid use. A trial of bevacizumab should be considered in those with worsening vasogenic cerebral edema such as recurrent malignant gliomas, radiation necrosis, or progressive brain metastases.

Gamma knife Radiosurgery for Peri-optic Meningiomas

Nabil A, Elnos F, Maaty H, Aboulfetouh I, Reda W, Alshahaby A, and Abdelkarim K

Gamma knife centre – Nasser institute – Cairo – Egypt, and Neurosurgery Dep.- Banha University

Object: Perioptic meningioms pose considerable therapeutic challengesbecause of their proximity to important cranial nerves, vasculature, and endocrine tissue at the anterior cranial base. This retrospective study aims at evaluating gamma knife radisurgery as a treatment modality for management of benign meningiomas in direct contact with the anterior visual pathway, and assessment of its long term effect over tumor control and visual outcome.

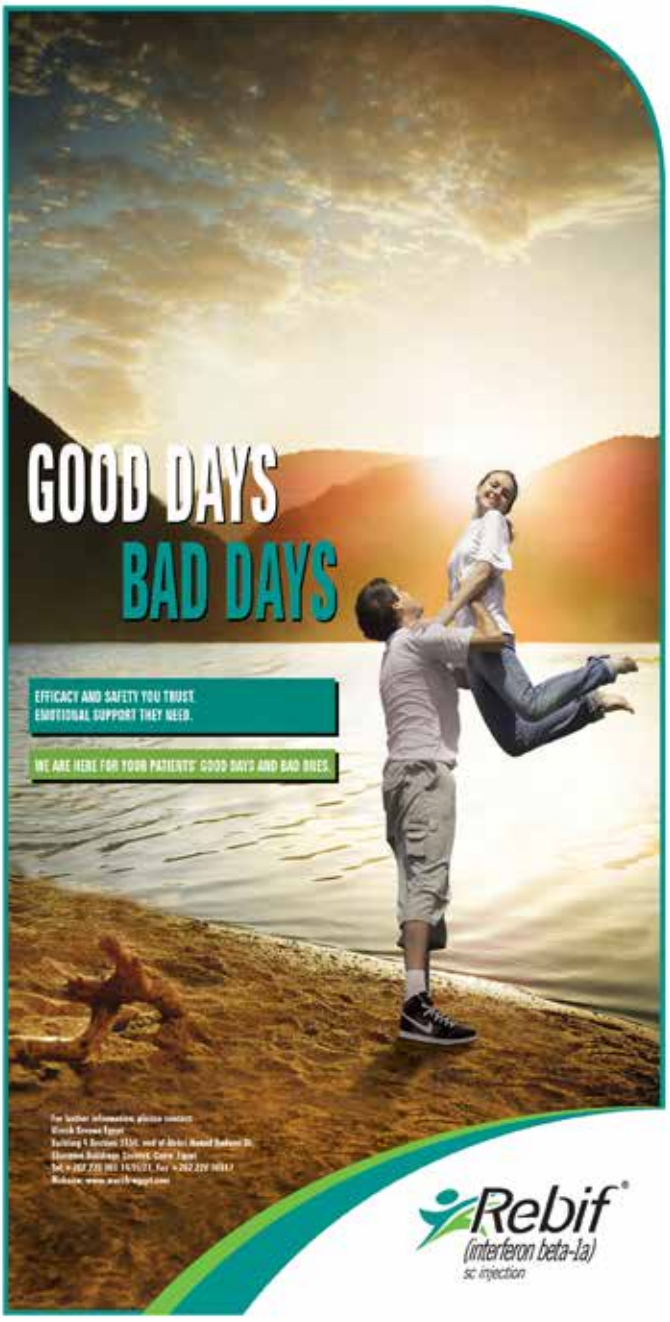
Methods: This is a retrospective analysis of a prospectively maintained,institutional data-base in the Cairo Gamma Knife Centre in Nasser Institute. The study material included 233 consecutive patients with benign skull base meningiomas in direct contact or displacing the anterior visual pathway treated by single session gamma knife radiosurgery during the period between July 2001 & July 2011 (10 years).

Results: Patients were assessed and with neuroimaging and visual field at routine intervals following GKRS. There were s (81%) females and (19%) males with age range (16-80 years). 67 patients (29%) had undergone at least one resection before GKRS, The mean follow-up after GKRS was 47 months (range 23–136 months).

At the last follow-up, tumor volumes remained stable or decreased in 94.8% of patients. Actuarial progression-free survival rates at 3, 5, 8, and 10 years were 99%, 94%, 87%, and 62%, respectively.

At the last clinical follow-up, 42% of patients demonstrated improvement visual outcome, 52% were stable, and 6% hod wors visual outcome. 51% of patients that had pretreatment ocular nerve palsy were improved. perifocal brain oedema was the most common complication after treatment (10.7%).

Conclusions.: Single session SRS with the GK is an effective and minimally invasive option for the treatment of perioptic meningiomas offering a reasonable rate of tumor control with a considerable rate of tumor shrinkage and a low incidence of complications.



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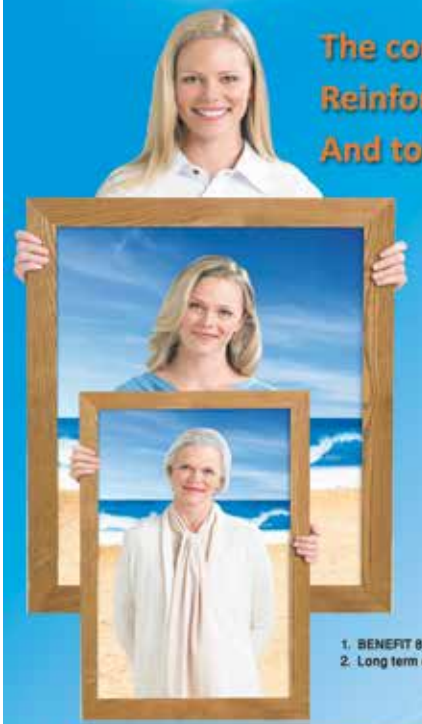

The confident choice in early initiated treatment in MS[1]
Reinforced by proven well established long term safety
And tolerability profile[2]

25 YEARS OF CLINICAL DATA
OVER 1.3 MILLION PATIENT-YEARS OF CLINICAL EXPERIENCE

1. BENEFIT 8 years J Neurol Neurosurg Psychiatry-2014-Eden-118- 39
2. Long term effect of early treatment 5 year treatment extension of the phase 3 BENEFIT trial Lancet Neurol 2009

Abbreviated package insert: Betaferon 250 microgram/ml. Composition: The active substance is interferon beta-1b, 250 microgram per millilitre when reconstituted.The other ingredients are in the powder: mannitol and human albumin, in the solvent (sodium chloride solution 5.4 mg/ml (0.54 % (w/v)) sodium chloride, water for injections. The Betaferon powder is provided in a 3-millilitre vial, containing 300 microgram (9.6 million IU) interferon beta- 1b per vial. After reconstitution each millilitre contains 250 microgram (8 million IU) interferon beta-1b. The solvent for Betaferon is provided in a 2.25-millilitre pre-filled syringe and contains 1.2 ml sodium chloride solution 5.4 mg/ml (0.54%). Indications: Patients who have experienced symptoms for the first time which indicate a high risk of developing multiple sclerosis, who suffer from relapsing-remitting multiple sclerosis, with at least two relapses within the last two years. who suffer from secondary progressive multiple sclerosis with active disease shown by relapses. Contraindications: pregnancy, hypersensitive to natural or recombinant interferon beta, human albumin or any of the other ingredients of Betaferon. severe depression and/or suicidal thoughts, severe liver disease. There is no information on the use of Betaferon in children under 12 years of age. Therefore Betaferon should not be used in this population. Warnings and Precautions: monitoring of gammagraphy, depression or depressed or previous thoughts of suicide. seizures or if taking medicines to treat epilepsy, severe kidney problems, symptoms such as itching all over the body, swelling of the face and/or the tongue or sudden shortness of breath. These may be symptoms of a serious allergic reaction (hypersensitivity), which may become life threatening. If the patient feel noticeably more sad or hopeless than before the treatment with Betaferon, or if he develop thoughts of suicide. If the patient become depressed while on Betaferon, he may need special treatment the doctor will closely monitor him and may also consider stopping his treatment. If he suffer from severe depression and/or suicidal thoughts, he will not be treated with Betaferon, any unusual bruising, excessive bleeding after injury or if he is catching a lot of infections. These may be symptoms of a fall in his blood cell count or in the number of platelets in his blood (cells, which help the blood to clot). He may need extra monitoring by the doctor. loss of appetite, fatigue, feeling sick (nausea), repeated vomiting, especially if the patient notice widespread itching, yellowing of the skin, or of the whites of the eyes or easy bruising. These symptoms may suggest problems with liver. Changes in the liver function values occurred in patients treated with Betaferon during clinical studies. As for other beta interferons, severe liver damage, including cases of liver failure, have been reported rarely in patients taking Betaferon. The most serious were reported in patients taking other medicines or who were suffering from diseases that can affect the liver (e.g. alcohol abuse, severe infection). experiencing symptoms like irregular heart beat, swelling such as of the ankles or legs, or shortness of breath. This may suggest a disease of the heart muscle (cardiomyopathy) which has been reported rarely in patients using Betaferon. notice of pain in the belly which is radiating to the back, and/or he feel sick or have a fever. This may suggest an inflammation of the pancreas (pancreatitis), which has been reported with Betaferon use. This is often associated with an increase of certain blood fats (triglycerides). The doctor should be informed immediately if patient get some or all of these symptoms: foamy urine, fatigue, swelling, particularly in the ankles and eyelids, and weight gain, as they may be signs of a possible kidney problem. The patient will need blood tests to measure the number of his blood cells, blood chemistry and liver enzymes. This will be done before the patient start using Betaferon, regularly after treatment with Betaferon has been initiated and periodically whilst the patient is on it, even if there is no particular symptoms. These blood tests will be in addition to the tests, which are normally done to monitor the MS. If the patient has a heart disease, the flu-like symptoms, which often occur at the start of treatment, may prove stressful to him. Betaferon must be used with caution, and the doctor will monitor the patient for worsening of the heart condition, particularly during the start of treatment. Betaferon itself does not affect the heart directly. The doctor will have a check of the function of the thyroid gland, regularly or whatever thought necessary by the doctor for other reasons. Betaferon contains human albumin and therefore carries a potential risk for transmission of viral diseases. A risk of transmission of Creutzfeld Jacob disease (CJD) cannot be ruled out. During treatment with Betaferon the body may produce substances which are called neutralising antibodies, which may react with Betaferon (neutralising activity). It is not yet clear whether those neutralising antibodies reduce the effectiveness of the treatment. Neutralising antibodies are not produced in all patients. Currently it is not possible to predict which patients belong to this group. During treatment with Betaferon, kidney problems that may reduce the patient kidney function, including scarring (glomerulosclerosis), may occur. The doctor may perform tests to check the kidney function of the patient. Blood clots in the small blood vessels may occur during treatment of the patient. These blood clots could affect his/her kidneys. This might happen several weeks to several years after starting Betaferon. The doctor may want to check the blood pressure of the patient, blood (platelet count) and the function of his/her kidney. Injection site reactions: During Betaferon treatment the patients are likely to experience injection site reactions. Symptoms include redness, swelling, change in the skin colour, inflammation, pain, and hypersensitivity. Dead skin and tissue around the injection site (necrosis) are reported less frequently. Injection site reactions usually become less frequent over time. Injection site skin and tissue breakdown can result in scars forming. If this is severe a doctor may have to remove foreign matter and dead tissue (debridement) and, less often, skin grafting is required and healing may take up to 6 months. To reduce the risk of getting injection site reactions the patient must: use a sterile (aseptic) injection technique, rotate the injection sites with each injection. Injection site reactions may occur less frequently. If the patient use an auto-injector device. If the patient experience any break in the skin, which may be associated with swelling or fluid leaking out from the injection site: Stop injections with Betaferon and doctor should be informed. If the patient have only one sore injection site (lesion) and the tissue damage (necrosis) is not too extensive the patient may continue using Betaferon. If the patient have more than one sore injection site (multiple lesions) he must stop using Betaferon until his skin has healed. The doctor will regularly check the way the patient inject himself, particularly if he experienced injection site reactions. Important information about some of the ingredients of Betaferon: The inactive ingredients of Betaferon include small amounts of mannitol, a naturally occurring sugar and human albumin, a protein. If the patient know that he is allergic (hypersensitive) to any of the ingredients or if he become so, he must not use Betaferon. Interactions: Using Betaferon with other medicines that modify the immune system response is not recommended, except anti-inflammatory medicines called corticosteroids or the adrenocorticotropic hormone (ACTH). Betaferon should be used with caution with: medicines which need a certain liver enzyme system (known as cytochrome P450 system) for their removal from the body, for example medicines used to treat epilepsy (like phenytoin). medicines which affect the production of blood cells. Breast-feeding: It is not known whether interferon beta-1b passes into human breast milk. However, it is theoretically possible that a breast-fed baby could experience serious side effects to Betaferon. Driving or using machines: Betaferon may cause side effects in the central nervous system if the patient is especially sensitive, this might affect his ability to drive or use machines. Administration: Treatment with Betaferon should be started under the supervision of the doctor who is experienced in the treatment of multiple sclerosis. The usual dose is: every other day (once every two days), 1.0 ml of the prepared Betaferon solution This equals 250 microgram (8 million IU) interferon beta-1b. When starting treatment with Betaferon it is tolerated best by gradually increasing the dose, i.e. starting with just 0.25 ml of the medication and then increasing, after every 3rd injection to 0.5 ml, 0.75 ml and then finally to the full (1 ml) dose of Betaferon. The doctor may decide, together with the patient, to change the time interval between increases in the dose depending on side effects the patient may experience at the start of treatment. To easily increase the dosage during the first 12 injections, the patient may be given a special titration pack, containing four differently coloured packs with specially started syringes and with detailed instructions on the separate introductory leaflet for titration pack. Preparing the injection: Before injection, the Betaferon solution has to be prepared from a vial of Betaferon powder and 1.2 ml of liquid from the pre-filled solvent syringe. This will either be done by the doctor or his/her assistant or by the patient after he have been carefully trained.

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