

Abstracts for oral sessions

5 April 2008 CME Course: Suicide and risk management in depressed pediatric patients

C18.01

CME course: Suicide and risk management in depressed pediatric patients

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The proposed course targets a major international public health issue: the cause of death of about 100, 000 children and adolescents worldwide each year, suicide. It is the second leading cause of death in that age group in many European countries. There are over three million suicide attempts made by adolescents annually. Although genetic and family risk factors are highly associated with both suicide and with suicide attempts, the specific genetic alleles that transmit this vulnerability between generations have yet to be identified.

Some risk factors for teen suicide attempts and completion have been identified and will be discussed, including an Axis I psychiatric disorder (e.g. mood disorder), family discord, aggressive-impulsive traits and physical and sexual abuse. One key factor consistently associated with suicide and suicidal behavior and will get a special attention in the course is a family history of suicidal behavior. This is as strong a risk factor as major depression, and stronger than environmental factors such as abuse. Suicidal behavior runs in families, independently of axis I or II diagnosis. Gene-environment interaction models in children and families will be presented and discussed. We will propose a stress-diathesis model of suicidal behavior and a practical tool for risk assessment for the clinician.

6 April 2008 Core Symposium: Integrated treatment of sexual dysfunctions

CS01.01

Integrated treatment of male and female desire disorders

R.T. Segraves, K.B. Segraves. *Psychiatry Department, Metrohealth, Cleveland, OH, USA*

Sexual behavior is determined by a complex interplay of psychosocial and biological factors. Evaluation of sexual disorders includes an evaluation of psychosocial factors such as cultural influences, life history, individual psychodynamics, couple interaction patterns, life stage as well as biological factors such as current medications, medical illness, psychiatric illness and endocrinological status. Sexual functioning usually declines in woman during the menopausal transition and this decline is related to declining estradiol levels. In spite of this, the major factor predicting postmenopausal sexual function is premenopausal sexual function and relationship with the sexual partner. There has been minimal study of psychological factors influencing male sexual desire. Intervention requires an integrated approach with equal consideration to numerous possible etiological factors.

CS01.02

Integrated treatment of female and male sexual arousal disorders

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Impaired arousal has been defined, in physiological terms, as lack of or impaired erection in males, and lack of or impaired lubrication/swelling in females. However, these criteria lack any correlation with the subjective experience of arousal

Treatment of sexual arousal disorders has been the most successful area of treatment of sexual dysfunctions. Nevertheless, the initial enthusiasm about the efficacy of phosphodiesterase-5 inhibitors in male erectile disorder has been tempered by studies demonstrating that pharmacological agents alone do not address all the complexities of the causative factors or treatment-subsequent psychological issues. It is also obvious that the effectiveness of pharmacological treatments is lower than their efficacy, especially in long term treatment. The results of treatment trials of female arousal disorder with pharmacological agents (oral or topical) suggest that no agent alone has been really consistently efficacious in this indication.

The interplay of physiological and psychological factors in the etiology of female and male sexual arousal disorder and the results of treatment trials underscore the need for an integrated approach to the treatment of this disorder.

Several trials suggest that the combination of pharmacological and psychological treatments may be the most suitable approach. For instance, one study demonstrated higher rates of success of the combination of sildenafil and cognitive-behavior sex therapy over sildenafil alone in male erectile disorder.

This presentation discusses the advantages of integration of pharmacological, psychological and other treatment modalities in the management of impaired sexual arousal and proposes a stepwise integrated approach to this disorder in both males and females.

CS01.03

Integrated treatment of female and male orgasmic disorders

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For about a century male ejaculatory disorders were considered to be caused mainly by psychological disturbances. However, since the 1990s, daily SSRI treatment has become very popular to delay ejaculation in men with lifelong PE. In addition, research has shown that it is likely that the persistent occurring very short ejaculation times of less than 1 minute in lifelong PE are related to neurobiological dysfunction in the central nervous system. On the other hand, epidemiological studies have shown that “complaints” of PE may not only occur in men with very short intravaginal ejaculation latency times (IELTs) but also in men with normal and even long ejaculation latency durations of, for example, 20 minutes. As their complaints are probably highly psychologically determined, treatment by counseling, psychotherapy or other non-medical interventions have been suggested. Integration of drug treatment, psycho-education, counseling and psychotherapy increases the chances for better coping mechanism in men affected by ejaculatory and orgasm problems. For female orgasmic disorders, particularly anorgasmia, medication is not yet available. Primary female anorgasmia is difficult to treat as multiple factors are involved in its pathophysiology. Neurobiological and pharmacological research is needed to develop drug treatment for those women who would like to alter this state. But with or without drug treatment, counseling may be of great value and contribute to better coping styles.

Symposium: Animal models of CNS disorders. Effects of drug treatments

S06.01

Gene-environment interaction in an animal model of depression

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Background: Both genes and environment play a role in depression. Data indicate that in addition to monoamines, other endogenous compounds, such as neuropeptides, as well as hippocampal cell loss/neurogenesis may be important in pathophysiology and treatment of depression. Finally, it is not clear whether early intervention could alleviate or prevent the disorder. Consequently, we studied neuropeptides in animal models: (i) a genetic model, the Flinders Sensitive Line (FSL) rat and their controls, FRL line, (ii) an environmental model, early maternal separation that mimics early life trauma in humans - experiences that predict adult life psychopathology, and (iii) maternal separation superimposed on the genetic FSL model

Methods: Behavior was studied when the animals reached adulthood, and brain neurochemistry and cell proliferation postmortem. On postnatal days (PND) 2–14, FSL and FRL pups were maternally

separated for 180. Escitalopram or vehicle were started on PND 44. Porsolt swim test was done on PND 64–65.

Results: baseline FSL-FRL differences were found in the Porsolt swim test and in brain neuropeptides, in particular NPY and CGRP in selected brain regions. Cell proliferation was also affected. Moreover, maternal separation and escitalopram also differentiated between the strains.

Conclusions: Both genes and environment play a role in “depression” but the consequences of early life events are more deleterious in genetically vulnerable individuals. Neurochemical, in particular NPY and CRH, and cell proliferation changes indicate that we may have identified some biological correlates of depression. potential strategy to alleviate adult life psychopathology.

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S06.02

High and low anxiety rat model: Emotionality, neuropeptides and aggression^{*}

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The selective breeding of Wistar rats for high (HAB) versus low (LAB) anxiety-related behaviour resulted in two rat strains which have been validated as a suitable animal model for studying neurochemical and genetic mechanisms underlying anxiety- and depression-related disorders. The robust differences in the anxiety phenotype are accompanied by alterations in neuroendocrine and neuronal stress-responsiveness to various stimuli, and in relevant brain neurotransmitter systems including arginine vasopressin (AVP), CRF and serotonin, and by impaired hippocampal neurogenesis. Manipulation of the endogenous vasopressin or oxytocin systems reveals their significant involvement as neuromodulators of anxiety behaviour in HAB rats.

HAB and LAB rats also provide an excellent model for studying interactions between early environmental factors (i.e. early life stress: prenatal stress, maternal separation) and the genetic predisposition for either high or low stress susceptibility. Thus, differential, partly opposite effects of prenatal or postnatal stress on adult emotionality, stress coping, neuropeptide expression patterns within the hypothalamus or hippocampal cell survival have been found in adult HAB and LAB rats.

Finally, selection for low trait anxiety in LAB rats goes along with the development of high intermale aggression during the resident-intruder test, and with a generally high neuroendocrine and neuronal response to social stimuli. Therefore, LAB males may develop as a promising animal model for studying neurobiological mechanisms of pathological aggression and its link to the genetically determined level of anxiety.

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S06.03

The nitric oxide pathway in anxiety and stress-related disorders

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Affective disorders are widely distributed disorders with severe social and economic effects. Strong evidence underlines that effective treatment helps to restore function and quality of life. Unfortunately,