humans. In this review, we summarize the latest findings.

Overeating

- Pathological overeating: an overview.

Authors: K. Gunnar Götestam, Finn Skårderud, Jan H. Rosenvinge, Einar Vedul-Kjelsás

Summary: An eating disorder apart from anorexia nervosa and bulimia nervosa is «binge eating disorder» (BED): eating in a short period of time a large quantity of food and a feeling of lack of control over food intake. There is also an atypical rest category, «eating disorders not otherwise specified» (EDNOS). Diagnostic criteria for BED and EDNOS are incomplete, particularly with respect to the definition of «bingeing» relative to bulimia nervosa. More restrictive criteria for anorexia nervosa and bulimia nervosa skew the diagnostic distribution towards BED and EDNOS, though the total prevalence of eating disorders remains unchanged.

For BED and EDNOS taken together the lifetime prevalence in women is about 6 %. The relationship between BED, EDNOS and overweight has mainly been overlooked; further investigations are needed.

Lasting treatment effects have been found for overweight people with BED. Other eating disorders apart from anorexia nervosa and bulimia are prevalent and clinically important, and research has opened up a potential for effective treatment.

Leptin & Eating Disorders

- The role of leptin in regulating neuroendocrine function in humans.

Authors: Bluher S, Mantzoros CS. - Division of Endocrinology, Department of Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA 02215.

Source: J Nutr. 2004 Sep;134(9):2469S-74S

Summary: Eating disorders are a group of disease states including anorexia nervosa, bulimia nervosa and binge eating on one end as well as episodic or chronic overeating resulting in obesity at the other end of the spectrum. These disorders are characterized by decreased and/or increased energy intake and are frequently associated with hormonal and metabolic disorders. The discovery of leptin, an adipocyte-secreted hormone acting in the brain to regulate energy homeostasis, and its subsequent study in human physiology have significantly advanced our understanding of normal human physiology and have provided new opportunities for understanding and possibly treating disease states, such as anorexia and bulimia nervosa. It has been recently discovered that leptin levels above a certain threshold are required to activate the hypothalamic-pituitary- gonadal and hypothalamic-pituitary-thyroid axes in men, whereas the hypothalamic-pituitary-adrenal, renin-aldosterone, and growth hormone-IGF-1 axes may be largely independent of circulating leptin levels in men. In this review, we summarize the latest findings related to the role of leptin in the regulation of several neuroendocrine axes, such as the hypothalamic-pituitary-gonadal and the hypothalamic-pituitary-thyroid axes in humans and discuss its potential pathophysiologic role in eating disorders.

Nutrition & Eating Disorders

- Nutrition expertise in eating disorders.

Authors: Breen HB, Espelage DL. - Department of Human Nutrition, University of Illinois at Chicago, Chicago, IL, USA. HBBreen@aol.com

Source: Eat Weight Disord. 2004 Jun;9(2):120-5

Summary: Anorexia nervosa (AN) and bulimia nervosa (BN) dominate published reports on disordered eating, although they actually account for a small number of cases. Binge eating disorder (BED) and subclinical syndromes of disturbed eating and distress are far more prevalent. Medical nutrition therapy including education is a cornerstone of therapy, however there has been no evaluation of baseline knowledge of nutrition and diet composition in this population relative to individuals who do not exhibit pathological eating behavior. In addition, previous reports suggest that individuals with clinical eating disorders have above-average knowledge of nutrition. In the present investigation, individuals with subclinical eating disorders did not differ from control participants. Poor scores overall indicate that nutritional counseling may be a useful component of treatment. These results further suggest that nutrition expertise is not an early feature of the disorder and, therefore, does not likely contribute to its development.

CBT & BED

- Cognitive-behavioral therapy with simultaneous nutritional and physical activity education in obese patients with binge eating disorder.

Authors: Fossati M, Amati F, Painot D, Reiner M, Haenni C, Golay A. - Division of Therapeutic Education for Chronic Diseases, University Hospital, Geneva, Switzerland

Source: Eat Weight Disord. 2004 Jun;9(2):134-8

Summary: An important problem with obese patients suffering from binge eating disorders (BED) is to treat their dysfunctional eating patterns while initiating a weight loss. We propose to assess a cognitive-behavioral therapy combined with a nutritional and a physical activity program. Our purpose is to verify that the addition of a nutritional and a physical program leads to a significant weight loss and enables psychological improvement. The patients (n=81) participated in a 12-week sessions group treatment of either a purely cognitive-behavioral therapy, or a cognitive-behavioral therapy associated to a nutritional approach mainly focused on fat restriction, or to a cognitive-behavioral therapy combined with a nutritional and a physical activity approach. The mean weight loss is significant (p<0.01) after the association of the cognitive-behavioral therapy and the nutritional education, but is even more significant (p<0.001) after the combination of a cognitive-behavioral therapy with a nutritional education and a physical activity program. Depression scores decrease in the three approaches, anxiety (p<0.05) results improve only in the combined nutritional, physical activity and cognitive-behavioral approach. Eating disorders improved significantly in all three approaches even if improvements in subscales seem more important in the combined approach. Finally, exercise seems to be a positive addition to the nutritional cognitive-behavioral therapy since it decreases negative mood, improves eating disorders and leads to an effective body weight loss.

Eating Disorders & Laxative Dependency

Arabpsynet Journal: № 4 - October – November – December 2004
**A blinded laxative taper for patients with eating disorders.**

**Authors:** Harper J, Leung M, Birmingham CL. - Department of Pharmacy, University of British Columbia. St. Paul's Hospital. Vancouver, B.C., Canada

**Source:** Eat Weight Disord. 2004 Jun;9(2):147-50

**Summary:** OBJECTIVE: To evaluate a blinded laxative taper, supervised entirely by pharmacists, in eating disorder patients with laxative dependency. METHODS: All subjects received a blinded laxative taper according to a set protocol, in addition to the usual treatment for their eating disorder. No specific treatment was given for laxative dependency other than the pharmacist's supervisions of the blinded taper. RESULTS: Ten patients were enrolled, of whom seven completed the study. Five of the seven patients (71%) decreased their laxative intake by at least 50%. Of these seven patients, three withdraw completely from laxative use. DISCUSSION: A standardized blinded laxative taper shows promise as a treatment option for laxative dependency in patients with eating disorders. The laxative taper may be less costly and more available than inpatient or psychologically based treatment because it can be given on an outpatient basis under the supervision of a pharmacist.

**Orthorexia Nervosa**

**Orthorexia nervosa: a preliminary study with a proposal for diagnosis and an attempt to measure the dimension of the phenomenon**

**Authors:** Donini LM, Marsili D, Graziani MP, Imbriale M, Cannella C. - Istituto di Scienza dell'Alimentazione, Università degli Studi di Roma La Sapienza, Italy. lorenzomaria.donini@uniroma1.it

**Source:** Eat Weight Disord. 2004 Jun;9(2):151-7

**Summary:** AIM: To propose a diagnostic proceeding and to try to verify the prevalence of orthorexia nervosa (ON), an eating disorder defined as "a maniacal obsession for healthy foods". MATERIALS AND METHODS: 404 subjects were enrolled. Diagnosis of ON was based on both the presence of a disorder with obsessive-compulsive personality features and an exaggerated healthy eating behaviour pattern. RESULTS: Of the 404 subjects examined, 28 were found to suffer from ON (prevalence of 6.9%). The analysis of the physiological characteristics, the social-cultural and the psychological behaviour that characterises subjects suffering from ON shows a higher prevalence in men and in those with a lower level of education. The orthorexic subjects attribute characteristics that show their specific "feelings" towards food ("dangerous" to describe a conserved product, "artificial" for industrially produced products, "healthy" for biological produce) and demonstrate a strong or uncontrollable desire to eat when feeling nervous, excited, happy or guilty.

**MED & PEM**

**Integrated medical-psychiatric treatment of the "crisis phase" in severe protein-energy malnutrition secondary to major eating disorders**

**Authors:** Alfano V, Bellini O, De Filippo E, Alfonsi L, Pasanisi F, Contaldo F. - CSIRO, Clinical Nutrition, Department of Clinical and Experimental Medicine, University "Federico II", Napoli, Italy

**Source:** Eat Weight Disord. 2004 Jun;9(2):158-62

**Summary:** C.A., a 23-year old male was admitted in the clinical nutrition medical ward for severe, complicated protein-energy malnutrition (PEM) [body mass index (BMI) 11.08 kg/m2; body weight kg 35.81] due to major eating disorders. C.A.'s personality was narcissistic, with a rigid psychic structure. During hospitalisation (lasted 72 days) two acute episodes (a possibly self-inflicted damage and a persecution feeling) occurred that we consider as part of the "crisis phase", the period in which the patient's restrictive behaviour is no longer able to keep his personality equilibrium stable. The patient was treated by an integrated medical and psychiatric approach, including periods of never forced parenteral nutrition, nutritional and intensive psychoterapeutic interventions. For a short period the patient received also a pharmacological support (aloperidol orally). Treatment was successful and the patient was discharged completely autonomous and followed up on an outpatient basis. After about one year follow-up he is still in good clinical condition and in sufficient psychological equilibrium.

**Chronic/ Recurrent Headache**

**Non-invasive physical treatments for chronic/recurrent headache**

**Authors:** Bronfort G, Nilsson N, Haas M, Evans R, Goldsmith CH, Assendelft WJJ, Bouter LM

**Source:** The Cochrane Library, Issue 3, 2004. Chichester, UK: John Wiley & Sons, Ltd

**Summary:** A substantive amendment to this systematic review was last made on 25 May 2004. Cochrane reviews are regularly checked and updated if necessary. Background: Non-invasive physical treatments are often used to treat common types of chronic/recurrent headache. Objectives: To quantify and compare the magnitude of short- and long-term effects of non-invasive physical treatments for chronic/recurrent headaches. Search strategy: We searched the following databases from their inception to November 2002: MEDLINE, EMBASE, BIOSIS, CINAHL, Science Citation Index, Dissertation Abstracts, CENTRAL, and the Specialised Register of the Cochrane Pain, Palliative Care and Supportive Care review group. Selected complementary medicine reference systems were searched as well. We also performed citation tracking and hand searching of potentially relevant journals. Selection criteria: We included randomized and quasi-randomized controlled trials comparing non-invasive physical treatments for chronic/recurrent headaches to any type of control. Data collection and analysis: Two independent reviewers abstracted trial information and scored trials for methodological quality. Outcomes data were standardized into percentage point and effect size scores wherever possible. The strength of the evidence of effectiveness was assessed using pre-specified rules. Main results: Twenty-two studies with a total of 2628 patients (age 12 to 78 years) met the inclusion criteria. Five
types of headache were studied: migraine, tension-type, cervicogenic, a mix of migraine and tension-type, and post-traumatic headache. Ten studies had methodological quality scores of 50 or more (out of a possible 100 points), but many limitations were identified. We were unable to pool data because of study heterogeneity. For the prophylactic treatment of migraine headache, there is evidence that spinal manipulation may be an effective treatment option with a short-term effect similar to that of a commonly used, effective drug (amitriptyline). Other possible treatment options with weaker evidence of effectiveness are pulsating electromagnetic fields and a combination of transcutaneous electrical nerve stimulation [TENS] and electrical neurotransmitter modulation. For the prophylactic treatment of chronic tension-type headache, amitriptyline is more effective than spinal manipulation during treatment. However, spinal manipulation is superior in the short term after cessation of both treatments. Other possible treatment options with weaker evidence of effectiveness are therapeutic touch; cranial electrotherapy; a combination of TENS and electrical neurotransmitter modulation; and a regimen of auto-massage, TENS, and stretching. For episodic tension-type headache, there is evidence that adding spinal manipulation to massage is not effective. For the prophylactic treatment of cervicogenic headache, there is evidence that both neck exercise (low-intensity endurance training) and spinal manipulation are effective in the short and long term when compared to no treatment. There is also evidence that spinal manipulation is effective in the short term when compared to massage or placebo spinal manipulation, and weaker evidence when compared to spinal mobilization. There is weaker evidence that spinal mobilization is more effective in the short term than cold packs in the treatment of post-traumatic headache.

Reviewers’ conclusions: A few non-invasive physical treatments may be effective as prophylactic treatments for chronic/recurrent headaches. Based on trial results, these treatments appear to be associated with little risk of serious adverse effects. The clinical effectiveness and cost-effectiveness of non-invasive physical treatments require further research using scientifically rigorous methods. The heterogeneity of the studies included in this review means that the results of a few additional high-quality trials in the future could easily change the conclusions of our review.

**Epilepsy & Differential Diagnosis**

**Defining the Problem: Psychiatric and Behavioral Comorbidity in Children**

*Authors*: Pellock JM. Division of Child Neurology, Department of Neurology, VCU, Virginia Commonwealth University, Medical College of Virginia Campus, Richmond, VA 23298, USA.


*Summary*: A variety of comorbid psychiatric conditions are frequently identified in children and adolescents with epilepsy, including depression, anxiety, psychosis, and attention-deficit hyperactivity disorder. Data regarding the epidemiology and precise prevalence of comorbid disorders in childhood epilepsy are incomplete and just now beginning to be compiled. Psychiatric and behavioral comorbidities are believed to affect approximately 40-50% of children and adolescents with epilepsy. Optimal diagnosis, clinical evaluation, and choice of treatment are predicated on the proper identification of coexisting psychiatric and behavioral disorders. Comorbid conditions in children and adolescents with epilepsy should be evaluated and treated as soon as they are recognized.
behavioral outcome. A growing body of evidence suggests it is important to think about the potential effect on psychiatric and behavioral disorders commonly associated with epilepsy and especially associated with central nervous system damage, family dysfunction, and severe seizures. This article discusses the risk factors to be considered when focusing on the prevalence of behavioral problems, the family factors that influence their incidence, as well as the differential diagnosis of behavioral disorders commonly associated with epilepsy. It also considers the assessment of these behavioral disorders and their treatment with psychotherapy, education, and a variety of psychopharmacological agents.

Epilepsy & Behavioral Comorbidities —

* Effects of epilepsy surgery on psychiatric and behavioral comorbidities in children and adolescents.

Authors: Shields WD. Division of Pediatric Neurology, David Geffen School of Medicine at UCLA, Los Angeles, CA 90095-1752, USA.


Summary: When considering surgery in children, it is important to think about the potential effect on psychiatric and behavioral outcome. A growing body of evidence suggests that the type of epilepsy (benign or catastrophic) and the age of the patient affect developmental outcome. In younger children with catastrophic epilepsy, the primary goal of therapy is not only to control seizures, but also to offer the child the opportunity for the best possible development. In older children with partial seizures, the therapeutic goal is to control seizures, which may allow a more normal life and improve behavior. Epilepsy surgery generally appears to have a positive effect on behavior and development in many younger children with catastrophic epilepsy. Psychiatric and behavioral outcomes in older children with complex partial epilepsy are less clear; many patients improve, but there does appear to be a small risk of developing new psychiatric or behavioral disorders postoperatively.

Epilepsy & Effects of Antiepileptic Medications —

* Effects of antiepileptic medications on psychiatric and behavioral comorbidities in children and adolescents with epilepsy.

Authors: Glauzer TA. Children's Hospital Medical Center, Cincinnati, OH 45229-3039, USA.


Summary: The three goals of this article are (1) to delineate the limitations in determining the actual incidence of antiepileptic drug (AED) psychiatric and behavioral side effects; (2) to summarize existing data on the direct effects of AEDs on psychiatric and behavioral comorbidities and examine the relationship between these direct effects and specific AED mechanisms of action; and (3) to recognize the indirect effects of AEDs on psychiatric and behavioral medications that can result in aggravation of these comorbidities through drug-drug interactions. All of these data are then combined and formatted into a practical algorithm useful in many clinical situations.

Epilepsy & Behavioral Issues —

* Behavioral issues involving children and adolescents with epilepsy and the impact of their families: recent research data.

Authors: Austin JK, Dunn DW, Johnson CS, Perkins SM. Indiana University School of Nursing, Indianapolis, IN 46202, USA.


Summary: OBJECTIVE: Using data from a larger study on new-onset seizures, we reported preliminary findings concerning relationships between family factors and child behavioral problems at baseline and 24 months. We also explored which baseline and changes in family factors were associated with changes in child behavioral problems over the 24-month period. METHODS: Subjects were 224 children and their primary caregivers. Data were collected using structured telephone interviews and analyzed using multiple regression. RESULTS: Deficient family mastery and parent confidence in managing their child's discipline were associated with behavioral problems at baseline and at 24 months; they also predicted child behavior problems over time. Decreasing parent confidence in disciplining their child was associated with increasing child behavior problems. Decreases in parent emotional support of the child were associated with increases in child internalizing problems. CONCLUSION: Child behavior problems, family environment, and parenting behaviors should be assessed when children present to the clinical setting with new-onset seizures.

Epilepsy, Carbamazepine & Valproate Monotherapy —

* Carbamazepine versus valproate monotherapy for epilepsy.

Authors: Manson AG, Williamson PR, Hutton JL, Clough HE, Chadwick DW; on behalf of the epilepsy monotherapy trialists.


Summary: A substantive amendment to this systematic review was last made on 18 April 2000. Cochrane reviews are regularly checked and updated if necessary. Background: Carbamazepine and valproate are drugs of first choice for epilepsy. Despite the lack of hard evidence from individual randomized controlled trials, there is strong clinical belief that valproate is the drug of choice for generalized epilepsies and carbamazepine for partial epilepsies. Objectives: To overview the best evidence comparing carbamazepine and valproate monotherapy.

Search strategy: We searched the Cochrane Epilepsy Group trials register (27 June 2003); the Cochrane Central Register of Controlled Trials (The Cochrane Library issue 2, 2003) and MEDLINE (27 June 2003). In addition, we contacted pharmaceutical companies and colleagues in the field to ascertain any unpublished or ongoing studies. Selection criteria: Randomized controlled trials comparing carbamazepine and valproate monotherapy for epilepsy. Data collection and analysis: This was an individual patient
data review. Outcome measures were time to withdrawal of allocated treatment, time to 12 month remission, and time to first seizure post randomization. Data were analysed using the stratified log rank test with results expressed as hazard ratios (HR) with 95% confidence intervals (CIs), where HR>1 indicates an event is more likely on valproate. A test for an interaction between treatment and epilepsy type (partial versus generalized) was also undertaken.

Main results: Results data were available for 1265 participants from 5 trials, representing 85% of the participants recruited into the 8 trials that met our inclusion criteria. The main overall results (HR) were: time to treatment withdrawal 0.97(95% CI 0.79 to 1.18); 12 month remission 0.87(95% CI 0.74 to 1.02); first seizure 1.09(95% CI 0.96 to 1.25) suggesting no overall difference for these outcomes. The test for an interaction between treatment and epilepsy type was non significant for time to treatment withdrawing and 12 month remission, but significant for time to first seizure. The age distribution of adults classified as having a generalized epilepsy indicate that significant numbers of individuals may have had their epilepsy misclassified.

Reviewers’ conclusions: We have found some evidence to support the policy of using carbamazepine as the first treatment of choice in partial epilepsies, but no evidence to support the choice of valproate in generalized epilepsies, but confidence intervals are too wide to confirm equivalence. Misclassification of people with epilepsy may have confounded our results, and has important implications for the design and conduct of future trials.

Epilepsy & Depression

* Impairment Of Inhibitory Control Of The Hypothalamic Pituitary Adrenocortical System In Epilepsy.

Authors: Zobel A, Wellmer J, Schulze-Rauschenbach S, Pfeiffer U, Schnell S, Elger C, Maier W. - Klinik und Poliklinik für Psychiatrie und Psychotherapie, Universitätsklinikum Bonn, Sigmund-Freud-Strasse 25, 53105, Bonn, Germany, astrid.zobel@ukb.uni-bonn.de


Summary: Excess comorbidity between depression and epilepsy proposes common pathophysiological patterns in both disorders. Neuroendocrine abnormalities were often observed in depression as well as in epilepsy. Lack of inhibitory control of the hypothalamic pituitary adrenocortical (HPA) system is a core feature of depression; main relay stations of this system are located in the amygdala and hippocampus, which are key regions for both disorders. Therefore we explored the feedback mechanism of the HPA system in epilepsy. In order to control for the impact of depression we focused on epilepsies without depression. We compared patients with epilepsy (subdivided by medication with or without hepatic enzyme inducing antiepileptic medication) with 16 healthy controls and 16 patients with unipolar major depression but without epilepsy. We observed a lack of inhibitory control of the HPA system in patients with epilepsy, also in the absence of enzyme inducing medication. An impact of the temporal lobe location of the epileptic focus could not be observed. Thus, epilepsies share with depression the deficiencies in the feedback mechanism of the HPA system, proposing common pathophysiological features of up to now unknown nature.

Sleep Deprivation & Epileptiform Discharges

* Does Sleep or Sleep Deprivation Increase Epileptiform Discharges in Pediatric Electroencephalograms?

Authors: Gilbert DL, DeRoos S, Bare MA. - Division of Neurology, Cincinnati Children’s Hospital Medical Center, ML 2015, 3333 Burnet Ave, Cincinnati, OH 45229-3039, USA. d.gilbert@cchmc.org

Source: Pediatrics. 2004 Sep;114(3):658-62

Summary: Sleep deprivation before obtaining an electroencephalogram (EEG) is believed both to increase the likelihood of sleep during an EEG and to increase the detection of interictal epileptiform discharges. However, depriving a child of sleep poses a burden on both the parent and the child. The objective of this study was to compare the effects of sleep, standard sleep deprivation, partial sleep deprivation, and no sleep deprivation on the odds of an epileptiform abnormality in outpatient pediatric EEGs. METHODS: Data were collected from all pediatric EEGs performed at a busy, university-based neurologic practice during two 2-month periods. During the first period, all EEGs were performed as ordered, either standard sleep-deprived (SSD) or non-sleep-deprived (NSD). During the second 2 months, SSD EEGs were performed per routine. However, non-SSD families were instructed to keep their children awake 2 hours later the night before the EEG. Those who complied were classified as partially sleep-deprived (PSD). Patient characteristics across protocols were compared with chi(2) and analysis of variance tests as appropriate. The odds of epileptiform and abnormal findings associated with sleep, NSD, PSD, and SSD EEGs were calculated using logistic regression. RESULTS: Of 820 eligible EEGs, sleep occurred in 22% of NSD, 44% of PSD, and 57% of SSD EEGs. The sample size of this study allowed for an 85% power, with alpha of.05, to detect an absolute increased EEG yield of 10%. Neither the presence of sleep (odds ratio [OR]: 0.99; 95% confidence interval [CI]: 0.69-1.42) nor the use of PSD (OR: 0.90; 95% CI: 0.50-1.62) or SSD (OR: 0.96; 95% CI: 0.63-1.47) protocols increased the odds of epileptiform EEGs. CONCLUSIONS: Sleep deprivation should not be used routinely to increase the yield of pediatric EEGs.

Cognitive Behaviour Therapy (CBT)

CBT & Becoming a Self-Therapist

* Becoming a self-therapist: Using cognitivebehavioural therapy for recurrent depression and/or dysthymia after completing therapy.

Authors: Glassman D, Finlay WM, Brock D. University of Surrey, UK.

Source: Psychol Psychother. 2004 Sep;77(Pt 3):335-51

Summary: OBJECTIVES: To explore the ways in which people use cognitive-behavioural therapy (CBT) for recurrent depression and/or dysthymia after leaving therapy. DESIGN: A qualitative interview was used in this study. METHOD: Semi-structured interviews were carried out with nine people who had completed a course of CBT at least three months previously. The interviews explored their use of CBT techniques or models outside of therapy and their everyday management of
depression. RESULTS: Eight of the nine participants reported engaging in some self-therapeutic activity, and identified depression, or the threat of depression, as a continuing presence in their lives. They used a range of techniques, either directly transferred from therapy or modified in some way, and identified a number of changes in the way they reacted to difficult situations or negative emotions. These included active responses such as leaving the room, making self-efficacy statements, or remembering what the therapist had said to them. Participants also described situations in which they could not use the things they had learnt in CBT. Finally, a range of factors that influenced the ways in which participants became self-therapists were identified. CONCLUSIONS: A number of implications for clinical practice are described. An understanding of how people modify CBT and use it (or not) in their everyday lives is important to understanding and improving effectiveness.

CBT & Smoking Cessation

**Group behaviour therapy programmes for smoking cessation**

**Authors:** Stead LF, Lancaster T


**Summary:** A substantive amendment to this systematic review was last made on 11 December 2001. Cochrane reviews are regularly checked and updated if necessary.

Background: Group therapy offers individuals the opportunity to learn behavioural techniques for smoking cessation, and to provide each other with mutual support.

Objectives: We aimed to determine the effects of smoking cessation programmes delivered in a group format compared to self-help materials, or to no intervention; to compare the effectiveness of group therapy and individual counselling; and to determine the effect of adding group therapy to advice from a health professional or nicotine replacement. We also aimed to determine whether specific components increased the effectiveness of group therapy. We aimed to determine the rate at which offers of group therapy are taken up.

Search strategy: We searched the Cochrane Tobacco Addiction Group trials register, with additional searches of PsycINFO and MEDLINE, including the terms behavior therapy, cognitive therapy, psychotherapy or group therapy, in December 2001.

Selection criteria: We considered randomised trials that compared group therapy with self-help, individual counselling, another intervention or no intervention (including usual care or a waiting list control). We also considered trials that compared more than one group programme. We included those trials with a minimum of two group meetings, and follow-up of smoking status at least six months after the start of the programme. We excluded trials in which group therapy was provided to both active therapy and placebo arms of trials of pharmacotherapies, unless they had a factorial design.

Data collection and analysis: We extracted data in duplicate on the people recruited, the interventions provided to the groups and the controls, including programme length, intensity and main components, the outcome measures, method of randomisation, and completeness of follow-up. The main outcome measure was abstinence from smoking after at least six months follow-up in patients smoking at baseline. We used the most rigorous definition of abstinence in each trial, and biochemically validated rates where available. Subjects lost to follow-up were counted as smokers. Where possible, we performed meta-analysis using a fixed effects (Peto) model.

Main results: A total of fifty-two trials met inclusion criteria for one or more of the comparisons in the review. Sixteen studies compared a group programme with a self-help programme. There was an increase in cessation with the use of a group programme (N=4,395, odds ratio 1.97, 95% confidence interval 1.57 to 2.48). Group programmes were more effective than no intervention controls (six trials, N=775, odds ratio 2.19, 95% confidence interval 1.42 to 3.37). There was no evidence that group therapy was more effective than a similar intensity of individual counselling. There was limited evidence that the addition of group therapy to other forms of treatment, such as advice from a health professional or nicotine replacement produced extra benefit. There was variation in the extent to which those offered group therapy accepted the treatment. There was limited evidence that programmes which included components for increasing cognitive and behavioural skills and avoiding relapse were more effective than same length or shorter programmes without these components. This analysis was sensitive to the way in which one study with multiple conditions was included. There was no evidence that manipulating the social interactions between participants in a group programme had an effect on outcome.

Reviewers' conclusions: Groups are better than self-help, and other less intensive interventions. There is not enough evidence on their effectiveness, or cost-effectiveness, compared to intensive individual counselling. The inclusion of skills training to help smokers avoid relapse appears to be useful although the evidence is limited. There is not enough evidence to support the use of particular components in a programme beyond the support and skills training normally included.

CBT & Depression in Alzheimer

**Cognitive Behavioural Therapy For Depression In A Person With Alzheimer's Dementia**

**Authors:** Deborah A. Walker a1c1 South London & Maudsley NHS Trust, UK

**Source:** Medline query on article authors walker da

**Summary:** The treatment of depression in an older person with Alzheimer's dementia using cognitive behavioural therapy (CBT) is described. Treatment focused on identifying beliefs associated with dementia, behavioural experimenting to test the validity of these beliefs, and increasing pleasurable activities. Results were promising, suggesting that it may be possible to treat depression in people with cognitive impairment.

Key Words: Depression; Alzheimer's dementia; behavioural; cognitive; older adults.

CBT of Depression & BPD

**Cognitive-Behavioral Treatment of Depression with Comorbid Borderline Personality Traits**

**Authors:** Michelle M. Lee - Department of Psychology, Case Western Reserve University, 10900 Euclid Avenue, Cleveland, OH 44106-7123 and James C. Overholser - Department of Psychology, Case Western Reserve University, 10900 Euclid Avenue, Cleveland, OH 44106-7123; overholser@po.cwru.edu

**Source:** Journal of Contemporary Psychotherapy 34 (3): 231-245, Fall 2004

**Summary:** Personality dysfunction can influence the onset and
maintenance of depressive symptoms. When both depression and personality dysfunction are present, it is important to develop an integrated treatment plan that addresses both conditions. A case example is used to illustrate how features of borderline personality disorder can influence the assessment and treatment of major depression. Specific challenges encountered by the therapist include: 1) differentiating borderline personality from depressive symptoms, 2) maintaining the therapeutic alliance, 3) managing impulsivity and self-destructive tendencies, 4) staying focused on long-term therapeutic goals, and 5) coping with noncompliance. Over the course of 27 sessions, the client was able to make positive changes in mood, self-image, and impulsive tendencies. Although the client’s borderline personality traits complicated the course of treatment for depression, neglecting these personality problems would have left the client vulnerable to depressive relapse.

Keywords: depression, borderline personality disorder, cognitive-behavioral treatment

Alcohol Abuse (AA)

Alcohol Dependence & Topiramate

* Uses of topiramate in the treatment of alcohol dependence

Authors: BA Johnson


Summary: Alcohol dependence is a major cause of morbidity and mortality in the USA and throughout the world. Over the last 10 years there has been an intense interest in developing pharmacotherapies that address the neurochemistry of alcohol dependence. Using a novel pharmacological approach to treating alcohol dependence, topiramate (Topamax® Ortho-McNeil Pharmaceutical) has recently been shown to improve the drinking outcomes of alcohol-dependent individuals. This drug profile highlights the scientific concepts and clinical evidence in the development of topiramate for treating alcohol dependence. Alcohol dependence, anticonvulsant, craving, pharmacotherapy, topiramate.

Alcohol Dependence & Topiramate

* Oral topiramate reduces the consequences of drinking and improves the quality of life of alcohol-dependent individuals: A randomized controlled trial.

Authors: Johnson BA, Ait-Daoud N, Akhtar FZ, Ma JZ. Department of Psychiatry, The University of Texas Health Science Center at San Antonio, 78229-3900, USA. bjohnson@uthscsa.edu

Source: Arch Gen Psychiatry. 2004 Sep;61(9):905-12.

Summary: Background: Topiramate, a fructosepyranose derivative, was superior to placebo in improving the drinking outcomes of alcohol-dependent individuals. Objectives: To determine whether topiramate, compared with placebo, improves psychosocial functioning in alcohol-dependent individuals and to discover how this improvement is related to heavy drinking behavior. Design: Double-blind, randomized, controlled, 12-week clinical trial comparing topiramate vs placebo for treating alcohol dependence (1998-2001).

Participants: One hundred fifty alcohol-dependent individuals, diagnosed using the DSM-IV. Interventions: Seventy-five participants received topiramate (escalating dose of 25 mg/d to 300 mg/d), and 75 had placebo and weekly standardized medication compliance management. Main outcome measures: Three elements of psychosocial functioning were measured: clinical ratings of overall well-being and alcohol-dependence severity, quality of life, and harmful drinking consequences. Overall well-being and dependence severity and quality of life were analyzed as binary responses with a generalized estimating equation approach; harmful drinking consequences were analyzed as a continuous response using a mixed-effects, repeated-measures model. Results: Averaged over the course of double-blind treatment, topiramate, compared with placebo, improved the odds of overall well-being (odds ratio [OR] = 2.17; 95% confidence interval [CI], 1.16-2.60; P = .01); reported abstinence and not seeking alcohol (OR = 2.63; 95% CI, 1.52-4.53; P = .001); overall life satisfaction (OR = 2.28; 95% CI, 1.21-4.29; P = .01); and reduced harmful drinking consequences (OR = - 0.07; 95% CI, -0.12 to -0.02, P = .01). There was a significant shift from higher to lower drinking quartiles on percentage of heavy drinking days, which was associated with improvements on all measures of psychosocial functioning. Conclusions: As an adjunct to medication compliance enhancement treatment, topiramate (up to 300 mg/d) was superior to placebo at not only improving drinking outcomes but increasing overall well-being and quality of life and lessening dependence severity and its harmful consequences.

Alcohol Abuse (AA)

Alcohol & Hepatitis C

* Alcohol and hepatitis C.

Authors: Safdar K, Schiff ER. - Center for Liver Diseases, Division of Hepatology, Department of Medicine, University of Miami, Miami, Florida.

Source: Semin Liver Dis. 2004 Aug;24(3):305-15

Summary: Alcohol abuse and hepatitis C virus (HCV) infection coexist with chronic liver disease in many patients. The mechanism of injury in these patients is probably multifactorial and involves, but is not limited to, a combination of diminished immune clearance of HCV, oxidative stress, emergence of HCV quasi-species, hepatic steatosis, increased iron stores, and increased rate of hepatocyte apoptosis. In patients with HCV infection, alcohol consumption is known to cause accelerated progression of liver fibrosis, higher frequency of cirrhosis, and increased incidence of hepatocellular carcinoma (HCC). These patients also have decreased survival as compared with patients with either alcohol abuse or HCV liver injury alone. Alcohol abuse causes decreased response to interferon treatment in HCV patients. It is therefore necessary for patients with HCV infection to abstain from alcohol consumption.

Alcohol Abuse (AA)

Alcoholic Liver Disease

* Diagnosis and therapy of alcoholic liver disease.

Authors: Levitsky J, Maillard ME. - University of Nebraska College of Medicine, Department of Internal Medicine, University of Nebraska Medical Center, Omaha, Nebraska.


Summary: Alcoholic liver disease (ALD) presents considerable challenges to clinicians. Screening for alcohol abuse and maintenance of depressive symptoms. When both depression and personality dysfunction are present, it is important to develop an integrated treatment plan that addresses both conditions. A case example is used to illustrate how features of borderline personality disorder can influence the assessment and treatment of major depression. Specific challenges encountered by the therapist include: 1) differentiating borderline personality from depressive symptoms, 2) maintaining the therapeutic alliance, 3) managing impulsivity and self-destructive tendencies, 4) staying focused on long-term therapeutic goals, and 5) coping with noncompliance. Over the course of 27 sessions, the client was able to make positive changes in mood, self-image, and impulsive tendencies. Although the client’s borderline personality traits complicated the course of treatment for depression, neglecting these personality problems would have left the client vulnerable to depressive relapse.

Keywords: depression, borderline personality disorder, cognitive-behavioral treatment

Alcohol Abuse (AA)

Alcohol Dependence & Topiramate

* Uses of topiramate in the treatment of alcohol dependence

Authors: BA Johnson


Summary: Alcohol dependence is a major cause of morbidity and mortality in the USA and throughout the world. Over the last 10 years there has been an intense interest in developing pharmacotherapies that address the neurochemistry of alcohol dependence. Using a novel pharmacological approach to treating alcohol dependence, topiramate (Topamax® Ortho-McNeil Pharmaceutical) has recently been shown to improve the drinking outcomes of alcohol-dependent individuals. This drug profile highlights the scientific concepts and clinical evidence in the development of topiramate for treating alcohol dependence. Alcohol dependence, anticonvulsant, craving, pharmacotherapy, topiramate.

Alcohol Dependence & Topiramate

* Oral topiramate reduces the consequences of drinking and improves the quality of life of alcohol-dependent individuals: A randomized controlled trial.

Authors: Johnson BA, Ait-Daoud N, Akhtar FZ, Ma JZ. Department of Psychiatry, The University of Texas Health Science Center at San Antonio, 78229-3900, USA. bjohnson@uthscsa.edu

Source: Arch Gen Psychiatry. 2004 Sep;61(9):905-12.

Summary: Background: Topiramate, a fructosepyranose derivative, was superior to placebo in improving the drinking outcomes of alcohol-dependent individuals. Objectives: To determine whether topiramate, compared with placebo, improves psychosocial functioning in alcohol-dependent individuals and to discover how this improvement is related to heavy drinking behavior. Design: Double-blind, randomized, controlled, 12-week clinical trial comparing topiramate vs placebo for treating alcohol dependence (1998-2001).

Participants: One hundred fifty alcohol-dependent individuals, diagnosed using the DSM-IV. Interventions: Seventy-five participants received topiramate (escalating dose of 25 mg/d to 300 mg/d), and 75 had placebo and weekly standardized medication compliance management. Main outcome measures: Three elements of psychosocial functioning were measured: clinical ratings of overall well-being and alcohol-dependence severity, quality of life, and harmful drinking consequences. Overall well-being and dependence severity and quality of life were analyzed as binary responses with a generalized estimating equation approach; harmful drinking consequences were analyzed as a continuous response using a mixed-effects, repeated-measures model. Results: Averaged over the course of double-blind treatment, topiramate, compared with placebo, improved the odds of overall well-being (odds ratio [OR] = 2.17; 95% confidence interval [CI], 1.16-2.60; P = .01); reported abstinence and not seeking alcohol (OR = 2.63; 95% CI, 1.52-4.53; P = .001); overall life satisfaction (OR = 2.28; 95% CI, 1.21-4.29; P = .01); and reduced harmful drinking consequences (OR = - 0.07; 95% CI, -0.12 to -0.02, P = .01). There was a significant shift from higher to lower drinking quartiles on percentage of heavy drinking days, which was associated with improvements on all measures of psychosocial functioning. Conclusions: As an adjunct to medication compliance enhancement treatment, topiramate (up to 300 mg/d) was superior to placebo at not only improving drinking outcomes but increasing overall well-being and quality of life and lessening dependence severity and its harmful consequences.

Alcohol Abuse (AA)

Alcohol & Hepatitis C

* Alcohol and hepatitis C.

Authors: Safdar K, Schiff ER. - Center for Liver Diseases, Division of Hepatology, Department of Medicine, University of Miami, Miami, Florida.

Source: Semin Liver Dis. 2004 Aug;24(3):305-15

Summary: Alcohol abuse and hepatitis C virus (HCV) infection coexist with chronic liver disease in many patients. The mechanism of injury in these patients is probably multifactorial and involves, but is not limited to, a combination of diminished immune clearance of HCV, oxidative stress, emergence of HCV quasi-species, hepatic steatosis, increased iron stores, and increased rate of hepatocyte apoptosis. In patients with HCV infection, alcohol consumption is known to cause accelerated progression of liver fibrosis, higher frequency of cirrhosis, and increased incidence of hepatocellular carcinoma (HCC). These patients also have decreased survival as compared with patients with either alcohol abuse or HCV liver injury alone. Alcohol abuse causes decreased response to interferon treatment in HCV patients. It is therefore necessary for patients with HCV infection to abstain from alcohol consumption.

Alcoholic Liver Disease

* Diagnosis and therapy of alcoholic liver disease.

Authors: Levitsky J, Maillard ME. - University of Nebraska College of Medicine, Department of Internal Medicine, University of Nebraska Medical Center, Omaha, Nebraska.


Summary: Alcoholic liver disease (ALD) presents considerable challenges to clinicians. Screening for alcohol abuse and...
alcoholism should be routine and repeated annually with close attention to signs and symptoms of liver disease. In patients with evidence of liver dysfunction or injury, consideration should be given to performance of liver biopsy for diagnosis and prognosis and prior to initiation of medication with the potential for significant side effects. Therapy depends on the spectrum of pathological liver injury: alcoholic fatty liver, alcoholic hepatitis, and cirrhosis. Abstinence is the foundation of therapy for an alcohol problem. Alcoholic fatty liver should improve with abstinence, but the similarity to the pathogenesis of nonalcoholic fatty liver and potential for progressive injury merits consideration of lipotropic agents. The continuing mortality, poor acceptance of corticosteroids, and identification of tumor necrosis factor-alpha (TNF-alpha) as an integral component has led to studies of pentoxifylline and, recently, anti-TNF antibody to neutralize cytokines in the therapy of severe alcoholic hepatitis. Antioxidant therapy of alcoholic cirrhosis has significant promise but will require large clinical trials.

Transplantation & Alcoholic Liver Disease (ALD)

* Transplantation in the Alcoholic Patient.

Authors: Watt KD, McCashland TM. - Departments of Internal Medicine and Hepatology, University of Nebraska Medical Center, Omaha, Nebraska.


Summary: Alcoholic liver disease (ALD) is the second leading indication for transplantation in the United States. Most transplant programs in the United States require a minimum of 6 month's abstinence before transplantation is performed. Most studies have shown a recidivism rate of between 20 and 30% by 2 years after orthotopic liver transplantation (OLT). Higher rates of recidivism are reported if pre-OLT abstinence was < 6 months. The impact of recidivism on patient and graft survival is required in the early postoperative setting, whereas monitoring for recidivism and malignancy are late postoperative issues.

Immunology & Alcoholic Liver Disease (ALD)


Authors: Thiele GM, Freeman TL, Klassen LW. - Professor, University of Nebraska Medical Center, Department of Internal Medicine, Section of Rheumatology and Immunology, Nebraska Medical Center and Department of Veterans Affairs Alcohol Research Center, Omaha Veterans Administration Medical Center, Omaha, Nebraska.


Summary: Clinically, the association of alcoholic liver disease ALD with circulating autoantibodies, hypergammaglobulinemia, antibodies to unique hepatic proteins, and cytotoxic lymphocytes reacting against autologous hepatocytes strongly suggests altered immune regulation with an increased activity toward normal self-proteins (loss of tolerance). Experimentally, there are several immune responses generated specifically recognizing self-proteins that are modified by metabolites of alcohol. These data strongly suggest that immune reactions may play a significant role in inducing and sustaining an inflammatory cascade of tissue damage to the liver. Additional support for this comes from the observation that the histological appearance of livers with ALD is that of a chronic active hepatitis-like inflammatory disease. Therefore, the hypothesis that immune mechanisms are involved in recurrent alcoholic hepatitis, although not summarily proven, is reasonable, supported by clinical and experimental evidence, and the subject of this article.

Nutrition & Alcoholic Liver Disease (ALD)

* Nutrition and Alcoholic Liver Disease.

Authors: Halsted CH. - Professor, Departments of Internal Medicine and Nutrition, University of California Davis, Davis, California.


Summary: Malnutrition is a common finding in chronic alcoholics, and protein calorie malnutrition (PCM) is universal and predictive of survival in patients with established alcoholic liver disease (ALD). These patients also demonstrate frequent deficiencies of folate, thiamine, pyridoxine, and vitamin A, which enhance the likelihood of anemia, altered cognitive states, and night blindness. The etiologies of malnutrition in ALD patients are multiple and interactive and include anorexia with inadequate dietary intake, abnormal digestion of macronutrients and absorption of several micronutrients, increased skeletal and visceral protein catabolism, and abnormal interactions of ethanol and lipid metabolism. Numerous, and mostly inadequately controlled, studies have evaluated the potential efficacies of oral, enteral, and parenteral nutrition approaches to treatment of ALD, with mixed results on liver function, clinical improvements, and short- or long-term survival. Targeted metabolic treatments include supplementation with S-adenosylmethionine (SAM) or phosphatidylcholine derivatives, each with promising experimental bases but inconclusive clinical trials.

Alcohol & ICP

* Different CFTR mutational spectrum in alcoholic and idiopathic chronic pancreatitis?


Summary: Cystic fibrosis transmembrane conductance regulator (CFTR) mutations are responsible for cystic fibrosis (CF) and have been postulated as a predisposing risk factor to chronic pancreatitis (CP), but controversial results demand additional support. We have therefore investigated the role of the CFTR gene in a cohort of 68 CP patients. METHODS: We have performed the CFTR gene analysis using 2 screening techniques. Fragments showing abnormal migration patterns were characterized by sequencing. Patients were classified in RESULTS: Sixteen mutations/variants were identified in 27 patients (40%), most of them (35%) presenting a single CFTR
mutant gene. The 1716G/A variant showed the highest frequency accounting for 22% in ICP and 5% in ACP, in contrast with other more common mutations such as F508del found in 8% of ACP and the 5T variant identified in 7% of patients. Acute pancreatitis, abdominal pain, tobacco, pancreatic calcifications, and pancreatic pseudocysts showed significant higher values in ACP than ICP patients. No significant differences were found between patients with and without CFTR mutations. CONCLUSIONS: Apart from reinforcing previous findings our data highlight the increased susceptibility of CFTR heterozygous to developing CP. Heterozygosity, combined with other factors, places these individuals at greater risk.

**Alcohol & BAP**

- **Exocrine pancreatic function after alcoholic or biliary acute pancreatitis.**

  **Authors:** Migliori M, Pezzilli R, Tomassetti P, Gullo L. - Institute of Internal Medicine, University of Bologna, Sant'Orsola Hospital, Bologna, Italy.

  **Source:** PubMed

  **Summary:** There have been various studies of exocrine pancreatic function after acute pancreatitis, but few have examined the relationship between this function and the etiology of the pancreatitis. The aim of this work was to study pancreatic function in patients who had had acute alcoholic or acute biliary pancreatitis.

  **METHODS:** Seventy-five patients who had had a single attack of acute pancreatitis were studied. The etiology was alcohol in 36 and cholelithiasis in 39. Pancreatic function was studied between 4 and 18 months after pancreatitis by duodenal intubation in 18 patients (8 alcohol, 10 lithiasis) and by the amino acid consumption test (AACT) in the remaining 57 (28 alcohol, 29 lithiasis). For those who underwent AACT, the test was repeated 1 year after the first examination.

  **RESULTS:** Among the 36 patients with alcoholic pancreatitis, most had impaired pancreatic function at both duodenal intubation (8/8, 100%) and at AACT (22/28, 78.6%); at the second test, the AACT remained pathological (18/23, 82.1%). Of the 39 patients with biliary pancreatitis, only 4 of the 10 (40%) who underwent duodenal intubation and only 5 of the 29 (17.2%) who performed AACT had pancreatic insufficiency; at the second test, only 4 of the 26 (15.4%) who repeated the AACT were pathological. The differences in the frequency and degree of pancreatic insufficiency between patients with alcoholic and those with biliary pancreatitis were statistically significant.

  **CONCLUSIONS:** The results show that after alcoholic acute pancreatitis, the pancreatic insufficiency was significantly more frequent and more severe than after biliary pancreatitis. These findings together with the fact that the insufficiency was also more persistent suggest that acute alcoholic pancreatitis may occur in a pancreas that already has chronic lesions.

**GAD & Somatic Symptoms**

- **Somatic symptoms and physiologic responses in generalized anxiety disorder and panic disorder: an ambulatory monitor study.**

  **Authors:** Hoehn-Saric R, McLeod DR, Funderburk F, Kowalski P. Department of Psychiatry, The Johns Hopkins Medical Institutions, Baltimore, MD 21287-7113, USA. rhoehn@mail.jhmi.edu

  **Source:** Arch Gen Psychiatry. 2004 Sep;61(9):913-21.

  **Summary:** BACKGROUND: Physiologic responses of patients with anxiety disorders to everyday events are poorly understood. OBJECTIVE: To compare self-reports and physiologic recordings in patients with panic disorder (PD), patients with generalized anxiety disorder (GAD), and nonanxious controls during daily activities. DESIGN: Participants underwent four 6-hour recording sessions during daily activities while wearing an ambulatory monitor. Physiologic and subjective data were recorded every 30 minutes and during subject-signal periods of increased anxiety or tension or panic attack. SETTING: Participants’ everyday environment. PARTICIPANTS: Twenty-six patients with PD and 40 with GAD, both without substantial comorbidity, and 24 controls. INTERVENTIONS: Recordings obtained during everyday activities. MAIN OUTCOME MEASURES: Recordings of heart interbeat intervals, skin conductance.

**General Anxiety Disorder (GAD)**

**GAD & Comorbidity**

- **Comorbidity of Anxiety and Depressive Disorders: Issues in Conceptualization, Assessment, and Treatment.**

  **Arabpsynet @Journal: N° 4 - October - November - December 2004**
levels, respirations, motion, and ratings of subjective somatic symptoms and tension or anxiety. RESULTS: Patients with anxiety disorders rated higher on psychic and somatic anxiety symptoms than did controls. Common to both anxiety disorders was diminished autonomic flexibility that manifested itself throughout the day, accompanied by less precise perception of bodily states. The main differences between patients with PD and GAD were a heightened sensitivity to body sensations and more frequent button presses. There also was a trend toward heightened basal arousal in patients with PD, manifesting itself in a faster heart rate throughout the day. CONCLUSIONS: Patients with PD or GAD are more sensitive to bodily changes than nonanxious individuals, and patients with PD are more sensitive than those with GAD. Patients with PD experience more frequent distress than those with GAD and controls, but their physiologic responses are comparable in intensity. The findings suggest that the perception of panic attacks reflects central rather than peripheral responses. The diminished autonomic flexibility observed in both anxiety conditions may result from dysfunctional information processing during heightened anxiety that fails to discriminate between anxiety-related and neutral inputs.

GAD & Venlafaxine XR —

- **Pooled analysis of venlafaxine XR efficacy on somatic and psychic symptoms of anxiety in patients with generalized anxiety disorder.**

**Authors:** Meoni P, Hackett D, Lader M. - Wyeth Research, Paris, France.

**Source:** Depress Anxiety. 2004;19(2):127-32.

**Summary:** We evaluated the relative efficacy of venlafaxine XR on the psychic versus somatic symptoms of anxiety in patients with generalized anxiety disorder as determined by the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition. Data were pooled and analyzed from 1,841 patients with generalized anxiety disorder who participated in five short-term (8-week) double-blind, multicenter, placebo-controlled studies, two of which had long-term (6-month) extensions. Somatic and psychic anxieties were studied using the Hamilton rating scale for anxiety (HAM-A) factor scores. We examined response rates (> or =50% improvement over baseline severity score) in the overall population and in patients with mainly somatic symptomatology at baseline (somatizers). Venlafaxine XR significantly reduced factor scores for both psychic and somatic HAM-A factors compared with placebo, from the first and second weeks of treatment, respectively. Patients treated with venlafaxine XR had significantly higher rates of response than patients receiving placebo on the psychic (58% vs. 38%, P<.001 at week 8; 66% vs. 35% at week 24, P<.001) and somatic (56% vs. 43%, P<.001 at week 8; 67% vs. 47% at week 24, P<.001) factors of the HAM-A. There was a TreatmentxFactor interaction (P<.027) in response rates: Patients treated with venlafaxine showed similar psychic and somatic anxiety response rates, whereas placebo-treated patients showed higher somatic compared with psychic response rates. Somatizers showed similar rates of response to the total population for the somatic factor of the HAM-A in either treatment group. Patients with generalized anxiety disorder treated with venlafaxine XR showed similar absolute rates of response on somatic and psychic symptoms, but relative to patients treated with placebo, more improvement in psychic than somatic symptoms.

Anxiety disorders & Progesterone

- **Role of progesterone and other neuroactive steroids in anxiety disorders.**

**Authors:** J-M Le Mellédo & GB Baker


**Summary:** It remains unexplained why a greater prevalence of anxiety disorders exists in women than in men, and how female hormone-related events (i.e., menstrual cycle and postpartum) can influence the course of anxiety disorders. It would appear logical that female hormones and their derivatives play a major role in these observations. The abundance of preclinical data demonstrating a role for sex hormones and their derivatives in anxiety-like behavior is in contrast to the relative paucity of experimental clinical data on the role of female hormones and neuroactive steroids in anxiety disorders. There is a dramatic potential for therapeutic anxiolytic activity of pharmacological compounds derived from powerful anxiolytic agents, such as the progesterone derivative, allopregnanolone. As a result, there is currently tremendous interest from the pharmaceutical industry in developing and testing such agents in anxiety disorders.

GAD & Sertraline —

- **Efficacy of sertraline in a 12-week trial for generalized anxiety disorder.**

**Authors:** Allgulander C, Dahl AA, Austin C, Morris PL, Sogaard JA, Fayyad R, Kucher SP, Clary CM. - Karolinska Institutet, Neurotect Department, Section of Psychiatry at Huddinge, University Hospital, 141 86 Huddinge, Sweden. Christer.Allgulander@neurotek.ki.se

**Source:** Am J Psychiatry. 2004 Sep;161(9):1642-9

**Summary:** OBJECTIVE: Sertraline’s efficacy and tolerability in treating generalized anxiety disorder were evaluated. METHOD: Adult outpatients with DSM-IV generalized anxiety disorder and a total score of 18 or higher on the Hamilton Anxiety Rating Scale were eligible. After a 1-week single-blind placebo lead-in, patients were randomly assigned to 12 weeks of double-blind treatment with placebo (N=188, mean baseline anxiety score=25) or flexible doses (50-150 mg/day) of sertraline (N=182, mean anxiety score=25). The primary outcome measure was baseline-to-endpoint change in the Hamilton anxiety scale total score. A secondary efficacy measure was the Clinical Global Impression (CGI) improvement score; response was defined as a score of 2 or less. RESULTS: Sertraline patients had significantly greater improvement than placebo patients on all efficacy measures at week 4. Analysis of covariance of the intent-to-treat group at endpoint (with the last observation carried forward) showed a significant difference in the decrease from baseline of the least-square mean total score on the Hamilton anxiety scale between sertraline (mean=11.7) and placebo (mean=8.0). Significantly greater endpoint improvement with sertraline than placebo was obtained for mean scores on the Hamilton anxiety scale psychic factor (6.7 versus 4.1) and somatic factor (5.0 versus 3.9). The rate of responders, based on CGI improvement and last observation carried forward, was significantly higher for sertraline (63%) than placebo (37%). Sertraline was well tolerated.
tolerated; 8% of patients versus 10% for placebo dropped out because of adverse events. CONCLUSIONS: Sertraline appears to be efficacious and well tolerated in the treatment of generalized anxiety disorder.

**COMT & Phobic Anxiety**

* Association between catechol-O-methyltransferase & phobic anxiety.  
  **Authors**: Am J Psychiatry. 2004 Sep;161(9):1703-5  
  **Source**: McGrath M, Kawachi I, Ascherio A, Colditz GA, Hunter DJ, De Vivo I. Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, 181 Longwood Ave., Boston, MA 02115. nhmcc@channing.harvard.edu  
  **Summary**: OBJECTIVE: The authors assessed the association between the catechol-O-methyltransferase (COMT) Val158Met polymorphism and scores on the phobic anxiety scale of the Crown-Crisp Experimental Index. METHOD: A total of 1,234 women completed the Crown-Crisp Experimental Index phobic anxiety scale and were genotyped for the COMT polymorphism. The authors used unconditional logistic regression to compute odds ratios and 95% confidence intervals (CIs) to evaluate the association between the COMT genotype and phobic anxiety. RESULTS: The mean scores for the three genotypes were statistically significantly different. Compared to the COMT Met/Met genotype, the age-adjusted odds ratio for scoring >/=6 compared to scoring 0 or 1 were 1.15 (95% CI=0.71-1.85) and 1.99 (95% CI=1.17-3.40) for the COMT Val/Met and COMT Val/Val genotypes, respectively; a significant gene dosage effect was observed. CONCLUSIONS: Our results suggest that the functional COMT polymorphism is associated with the development of phobic anxiety.

**OCD & Trichotillomania**

* Dissociative experiences in obsessive-compulsive disorder and trichotillomania: Clinical and genetic findings.  
  **Authors**: Lochner C, Seedat S, Hemmings SM, Kinneer CJ, Corfield VA, Niehaus DJ, Moolman-Smook JC, Stein DJ.  
  **Source**: Compr Psychiatry. 2004 Sep-Oct;45(5):384-91  
  **Summary**: A link between dissociation proneness in adulthood and self-reports of childhood traumatic events (including familial loss in childhood, sexual/physical abuse and neglect) has been documented. Several studies have also provided evidence for an association between dissociative experiences and trauma in patients with various psychiatric disorders, including post-traumatic stress disorder, borderline personality, dissociative identity and eating disorders. Based on the relative paucity of data on dissociation and trauma in obsessive-compulsive disorder (OCD) and trichotillomania (TTM), the primary objective of this study was to examine the relationship between trauma and dissociative experiences (DE) in these two diagnostic groups. Furthermore, the availability of clinical and genetic data on this sample allowed us to explore clinical and genetic factors relevant to this association. A total of 110 OCD and 32 TTM patients were compared with respect to the degree of dissociation (using the Dissociative Experiences Scale [DES]) and childhood trauma (using the Childhood Trauma Questionnaire [CTQ]). Patients were classified on the DES as either "high" (mean DES score >/= 30) or "low" (mean DES score < 30) dissociators. Additional clinical and genetic factors were also explored with chi-square and t tests as appropriate. A total of 15.8% of OCD patients and 18.8% of TTM patients were high dissociators. OCD and TTM groups were comparable on DES and CTQ total scores, and in both OCD and TTM groups, significant positive correlations were found between mean DES scores and mean CTQ subscores of emotional anxiety, physical abuse, sexual abuse, and physical neglect. In the OCD group, high dissociators were significantly younger than low dissociators, and significantly more high dissociators than low dissociators reported a lifetime (current and past) history of tics (P <.001), Tourette's syndrome (P =.019), bulimia nervosa (P =.003), and borderline personality disorder (P =.027). In the TTM group, significantly more high dissociators than low dissociators reported (lifetime) kleptomania (P =.005) and depersonalisation disorder (P =.005). In the Caucasian OCD patients (n = 114), investigation of genetic polymorphisms involved in monoamine function revealed no significant differences between high and low dissociator groups. This study demonstrates a link between childhood trauma and DE in patients with OCD and TTM. High dissociative symptomatology may be present in a substantial proportion of patients diagnosed with these disorders. High dissociators may also be differentiated from low dissociators on some demographic features (e.g., lower age) and comorbidity profile (e.g., increased incidence of impulse dyscontrol disorders). Additional work is necessary before conclusions about the role of monoaminergic systems in mediating such dissociation can be drawn.

**Biography (B)**

**Biology & Endophenotype**

* Endophenotype--a new concept for biological characterization of psychiatric disorders  
  **Authors**: Zobel A, Maier W. Klinik und Poliklinik fur Psychiatrie und Psychotherapie, Universitat Bonn. Astrid.Zobel@ubk.uni-bonn.de  
  **Summary**: In the field of psychiatric genetics, the concept of endophenotypes is presently receiving high popularity. Current difficulties in the search for susceptibility genes of complex diseases have been attributed to the etiological heterogeneity of the clinical, psychopathologically defined phenotype of the disease. Neurobiological correlates of the disease which are genetically influenced and stable over time are considered to be more promising targets of phenotypes. They are more directly influenced by gene effects and presumably defined by a genetic determination which is less complex than the phenotype of the disorder. The endophenotype strategy has already been successful in complex disorders of other areas in medicine, including alcoholism and late-onset Alzheimer's disease. For schizophrenia, a series of endophenotypes for molecular/genetic investigation has been suggested, but the concept "endophenotype" also provides a basis for biological classification of mental disorders.

**Biological & Clinical Studies**

* Clinical study: Atypical depression in growth hormone  
  **Authors**: Zobel A, Maier W. Klinik und Poliklinik fur Psychiatrie und Psychotherapie, Universitat Bonn. Astrid.Zobel@ubk.uni-bonn.de  
  **Summary**: In the field of psychiatric genetics, the concept of endophenotypes is presently receiving high popularity. Current difficulties in the search for susceptibility genes of complex diseases have been attributed to the etiological heterogeneity of the clinical, psychopathologically defined phenotype of the disease. Neurobiological correlates of the disease which are genetically influenced and stable over time are considered to be more promising targets of phenotypes. They are more directly influenced by gene effects and presumably defined by a genetic determination which is less complex than the phenotype of the disorder. The endophenotype strategy has already been successful in complex disorders of other areas in medicine, including alcoholism and late-onset Alzheimer's disease. For schizophrenia, a series of endophenotypes for molecular/genetic investigation has been suggested, but the concept "endophenotype" also provides a basis for biological classification of mental disorders.

**Psychiatric New Papers**

Arabpsynet Journal: N° 4-October - N°01-December-2004
DEFICIENT ADULTS, AND THE BENEFICIAL EFFECTS OF GROWTH HORMONE TREATMENT ON DEPRESSION AND QUALITY OF LIFE.

Authors: Tripti Mahajan1, Anna Crown1, Stuart Checkley2, Anne Farmer2 and Stafford Lightman1

Henry Wellcome Laboratories for Integrative Neuroscience & Endocrinology, University of Bristol 1 and Institute of Psychiatry, London 2

Source: (Correspondence should be addressed to S Lightman)

Summary: Some growth hormone deficient adults (GHDAs) have an impaired quality of life, which may improve with growth hormone (GH) treatment. The objective of our study was to make an in depth psychiatric evaluation of patients with adult-onset (AO) and childhood-onset (CO) GHD, and to assess the time course of changes in their quality of life and symptoms of depression in response to GH treatment.

Design: The study design was a 4 month, double-blind, crossover, placebo-controlled trial of GH therapy.

Methods: We used a detailed psychiatric interview to characterise 25 patients with proven GH deficiency at baseline. They were reassessed at monthly intervals during treatment with GH or placebo, using the Nottingham Health Profile and two well-recognised depression rating scales.

Results: 11 / 18 (61%) of the patients with AO GH deficiency, but 0 / 7 of the patients with CO GH deficiency, were found to have atypical depression at baseline. There were significant improvements in the depression rating scale scores after 2 months of GH therapy, with significant improvements in emotional reaction and social isolation scores from 1 month, and in energy levels and sleep disturbance from 2 and 3 months respectively.

Conclusions: The results of our study confirm that a large proportion of GHDAs have unequivocal psychiatric morbidity, and suggest that a response to treatment can be seen after a short trial of GH therapy. We hypothesise that this rapid improvement of symptoms of atypical depression represents a direct central effect of GH therapy.

Heschl’s Gyrus & SLLI, MDD, BP

* CELL DENSITY AND CORtical THICKNESS IN HESCHL’S GYRUS IN SCHIZOPHRENIA, MAJOR DEPRESSION AND BIPOLAR DISORDER.

Authors: Cotter D, Mackay D, Frangou S, Hudson L, Landau S - Beaumont Hospital, Dublin 9, Ireland. d.cotter@rcsi.ie

Source: Br J Psychiatry. 2004 Sep;185:258-9

Summary: There is evidence that the superior temporal gyrus and Heschl’s gyrus within it are implicated in schizophrenia. We investigated neuronal and glial cell density and cortical thickness within Heschl’s gyrus, using the optical disector to estimate cell density within cortical layers 3 and 5 in tissue derived postmortem from people with diagnoses of major depressive disorder, schizophrenia and bipolar disorder, compared with normal controls (n=15 per group). No significant difference in neuronal or glial cell density or in cortical thickness was observed between the groups; our findings therefore provide no support for the presence of cellular pathology within Heschl’s gyrus in schizophrenia.

FRAGILE X SYNDROME

* NEUROPSYCHIATRIC SYMPTOMS OF FRAGILE X SYNDROME: PATHOPHYSIOLOGY AND PHARMACOTHERAPY.

Authors: Tsouris JA, Brown WT. - George A. Jervis Clinic, New York State Institute for Basic Research in Developmental Disabilities, Staten Island, New York, USA


Summary: Fragile X syndrome is the leading inherited form of mental retardation, and second only to Down's syndrome as a cause of mental retardation attributable to an identifiable genetic abnormality. Fragile X syndrome is caused by a defect in the fragile X mental retardation 1 gene (FMR1), located near the end of the long arm of the X chromosome. FMR1 normally synthesises the fragile X protein (FMRP), but mutations in FMR1 lead to a lack of FMRP synthesis, resulting in fragile X syndrome. While the specific function of FMRP is not yet fully understood, the protein is known to be important for normal brain development. The physical, cognitive and behavioural features of individuals with fragile X syndrome depend on gender (females have two X chromosomes, one active and one inactive) and the molecular status of the mutation (premutation, full mutation or mosaic). Features of the behavioural profile of individuals with fragile X syndrome include hypersensitivity to stimuli, overarousability, inattention, hyperactivity and (mostly in men) explosive and aggressive behaviour to others or self. Social anxiety, other anxiety disorders, depression, impulse control disorder and mood disorders are the most common psychiatric disorders diagnosed in individuals with fragile X syndrome, although no formal studies have been undertaken. There have been very few psychopharmacological studies of the treatment of behaviours associated with fragile X syndrome. These limited studies and surveys of psychotropic drugs used in individuals with fragile X syndrome suggest that stimulants are helpful for hyperactivity, that alpha(2)-adrenoceptor agonists and beta-adrenoceptor antagonists help to control overarousability, impulsivity and aggressiveness, and that SSRIs can control anxiety, impulsivity and irritability, alleviate depressive symptoms and decrease aggressive and self-injurious behaviour. Typical and atypical antipsychotics in combination with other psychotropics have been used for control of psychotic disorders and severe aggressive behaviours. Mood stabilisers have been found to be useful when mood dysregulation or mood disorders are present with or without aggressive behaviour. Folic acid and L-acetylcarnitine (levacecarnine) have not been found to improve deficits or behaviours. As there is no specific psychotropic drug for any of the deficits or behaviours associated with fragile X syndrome, clinicians are advised to diagnose any psychiatric syndromes or disorders present and treat them with the appropriate psychotropic drug. If no psychiatric disorder can be diagnosed and the patient's challenging behaviours cannot be controlled with environmental manipulation or behaviour modification techniques, the most benign psychotropic drug should be used. Antipsychotics should be reserved for psychotic disorders, for impulse control disorders (used in combination with other psychotropics), or when challenging behaviours constitute an emergency. In the future, new medications targeting molecules implicated in the modulation of anxiety, fear and fear responding will be useful for treating the social anxiety and overarousability exhibited by individuals with fragile X syndrome.
Mood Stabilisers (MS)

**MS & VALPROATE**

- **Valproic acid, valproate and divalproex in the maintenance treatment of bipolar disorder**

**Authors:** Macritchie K.A.N., Geddes J.R., Scott J, Haslam D.R.S., Goodwin G.M.

**Source:** The Cochrane Library, Issue 3, 2004. Chichester, UK: John Wiley & Sons, Ltd

**Summary:** A substantive amendment to this systematic review was last made on 19 March 2001. Cochrane reviews are regularly checked and updated if necessary.

**Background:** Although lithium has been the most commonly used maintenance treatment in bipolar disorder for several decades, valproate is being used increasingly - especially in the United States of America. There is a need to clarify whether the increasingly prominent prophylactic role of valproate in bipolar disorder is justified.

**Objectives:** To review the effectiveness of valproate, relative to placebo, other mood stabilisers and antipsychotics, in the prevention and/or attenuation of acute episodes of bipolar disorder. The effectiveness of valproate was considered in terms of mood symptoms, mortality, general health, social functioning, adverse effects and overall acceptability to patients.

**Search strategy:** The CCDAN group search strategy was used.

**Main results:** One trial of 12 months duration with 372 patients taking divalproex who left the study because of adverse effects and overall acceptability to patients.3. The prevalence of side-effects.4. Mortality on valproate treatment. Outcomes concerning relapse/recurrence were analysed excluding data from discontinuation studies, which were to be analysed separately. Sub-group analyses were to be performed to examine the effects of valproate treatment in rapid cycling bipolar disorder and previous mood stabiliser non-responders. Data were analysed excluding data from discontinuation studies, which were to be analysed separately. Sub-group analyses were to be performed to examine the effects of valproate treatment in preventing or attenuating further episodes of bipolar disorder, including its effectiveness in rapid cycling disorder.2. The acceptability of valproate treatment to patients.3. The prevalence of side-effects.4. Mortality on valproate treatment. Outcomes concerning relapse/recurrence were analysed excluding data from discontinuation studies, which were to be analysed separately.

**Reviewers’ conclusions:** In view of the equivocal findings of this review, conclusions about the efficacy and acceptability of valproate compared to placebo and lithium cannot be made with any degree of confidence. With current evidence, patients and clinicians would probably wish to use lithium before valproate for maintenance treatment. At present, the observed shift of prescribing practice to valproate is not based on reliable evidence of efficacy.

**Valproic Acid & Hyperammonemia**

- **Valproic Acid-induced hyperammonemia: a case report.**

**Authors:** McCall M, Bourgeois JA. - Department of Psychiatry and Behavioral Sciences, University of California, Davis Medical Center, Sacramento CA.

**Source:** J Clin Psychopharmacol. 2004 Oct;24(5):521-6

**Summary:** The authors present a case of a patient treated with valproic acid for seizure disorder who presented with acute mental status changes consistent with encephalopathy. Notably, her serum ammonia level was 3 times the upper limit of normal, despite an only mildly elevated aspartate aminotransferase and normal bilirubin. Her serum valproic acid level was in the therapeutic range. Her symptoms resolved with discontinuation of valproic acid and supportive care. The authors review the possible mechanisms of valproic acid-associated hyperammonemia with encephalopathy and propose clinical practice modifications to minimize the incidence of this adverse reaction to this generally well-tolerated and clinically important psychotropic medication.

**Sleep Disorders**

- **Sleep-related breathing disorders: impact on mortality of cerebrovascular disease.**

**Authors:** Parra O, Arboix A, Montserrat JM, Quinto L, Bechich S, Garcia-Eroles L. - Dept of Pneumology, Hospital del Sagrat Cor, Barcelona, Spain. 22515opo@comb.es

**Source:** J Clin Psychopharmacol. 2004 Oct;24(5):521-6

**Summary:** The authors present a case of a patient treated with valproic acid for seizure disorder who presented with acute mental status changes consistent with encephalopathy. Notably, her serum ammonia level was 3 times the upper limit of normal, despite an only mildly elevated aspartate aminotransferase and normal bilirubin. Her serum valproic acid level was in the therapeutic range. Her symptoms resolved with discontinuation of valproic acid and supportive care. The authors review the possible mechanisms of valproic acid-associated hyperammonemia with encephalopathy and propose clinical practice modifications to minimize the incidence of this adverse reaction to this generally well-tolerated and clinically important psychotropic medication.
Sleep-Disordered Breathing

Performance vigilance task and sleepiness in patients with sleep-disordered breathing.

**Authors:** Sforza E, Haba-Rubio J, De Bilbao F, Rochat T, Ibanez V. - Sleep Laboratory, Dept of Psychiatry, Geneva University Hospital, Geneva, Switzerland. Emilia.Sforza@hcuge.ch

**Source:** Eur Respir J. 2004 Aug;24(2):279-85

Summary: Altered vigilance performance has been documented in patients with sleep-related breathing disorders (SRBDs). Sleep fragmentation, sleepiness, respiratory disturbances and nocturnal hypoxemia have been suggested as the pathogenesis of these deficits, yet it remains difficult to find a good correlation between performance deficits and the above factors. In the present study, which performance measure better characterised SRBD patients and the main factors implicated in these disturbances were examined. The study group consisted of 152 patients and 45 controls, all examined using a performance vigilance task and subjective sleepiness assessment. Speed and accuracy in the psychomotor vigilance task (PVT) were measured in patients and controls. Objective daytime sleepiness was assessed in the patient group using the maintenance of wakefulness test. In comparison with controls, PVT accuracy rather than speed seems to be affected in SRBD patients, with lapses and false responses significantly greater in patients with more severe objective sleepiness and higher apnoea/hypopnoea index. Although slowing and increased variability in reaction time were associated with shorter sleep latency in the maintenance of wakefulness test, subjective sleepiness, sleep fragmentation, nocturnal hypoxemia and apnoea/hypopnoea index influenced mainly PVT accuracy. It is concluded that vigilance impairment, sleep fragmentation and severity of disease may partially and differentially contribute to the diurnal performance consequences found in sleep-related breathing disorders.

Since the psychomotor vigilance task worsening is more marked in accuracy that in speed, measurement of lapses and false responses would better characterise the degree of diurnal impairment in these patients.

Obstructive Sleep Apnea & Neuropsychological Sequelae

Neuropsychological sequelae of obstructive sleep apnea-hypopnea syndrome: a critical review.

**Authors:** Aloia MS, Arnedt JT, Davis JD, Riggs RL, Byrd D. - Brown Medical School, Providence, Rhode Island 02906, USA. Mark.Aloia@Brown.edu

**Source:** J Int Neuropsychol Soc. 2004 Sep;10(5):772-85

Summary: Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a well-recognized clinical sleep disorder that results in chronically fragmented sleep and recurrent hypoxemia. The primary daytime sequelae of the disorder include patient reports of excessive daytime sleepiness, depression, and attention and concentration problems. It has been well established that OSAHS negatively impacts certain aspects of cognitive functioning. The primary goals of this article are to (1) clarify the pattern of cognitive deficits that are specific to OSAHS; (2) identify the specific cognitive domains that improve with treatment; and (3) elucidate the possible mechanisms of cognitive dysfunction in OSAHS. At the conclusion of the paper, we propose a potential neurofunctional theory to account for the etiology of cognitive deficits in OSAHS. Thirty-seven peer-reviewed articles were selected for this review. In general, findings were equivocal for most cognitive domains. Treatment, however, was noted to improve attention/vigilance in most studies and consistently did not improve constructional abilities or psychomotor functioning. The results are discussed in the context of a neurofunctional theory for the effects of OSAHS on the brain.

Parkinson Disease (PD)

Therapies for depression in Parkinson's disease.

**Authors:** Shabnam Ghazi-Noori, TH Chung, KHO Deane, H Rickards, CE Clarke


Summary: A substantive amendment to this systematic review was last made on 21 February 2003. Cochrane reviews are regularly checked and updated if necessary. Background: Depression is one of the most common neuropsychiatric disturbances in Parkinson’s disease. 40% of observed variation in quality of life is due to depression. However, there is little hard evidence of the efficacy and safety of antidepressant therapies in Parkinson’s disease. Objectives: To assess the efficacy and safety of antidepressant therapies in idiopathic Parkinson's disease. Safety refers to both the direct side-effects of the therapy and also the therapy's interactions with the symptoms of Parkinson's disease and with the antiparkinsonian medications. Search strategy: Relevant clinical trials were identified by electronic searches the Cochrane Controlled Trials Register MEDLINE(1996-2001),
Suicide

Suicide & Seasonal differences

Seasonal differences in psychopathology of male suicide completers.

Authors: Kim CD, Lesage AD, Seguin M, Chawky N, Vanier C, Lipp O, Turecki G.


Summary: Suicide is known to vary according to season, with peaks in the spring and troughs in the winter. The presence of psychopathology is a significant predictor of suicidality, and it is possible that the seasonal variation of suicide completion may be related to seasonality in the manifestation of psychiatric disorders common to suicide completers. In the current study, we evaluated 115 French-Canadian male suicide completers from the Greater Montreal Area for DSM-IV psychiatric disorders using proxy-based diagnostic interviews. Subjects were assessed for seasonal differences in the prevalence of DSM-IV psychiatric diagnoses just before their deaths. Diagnoses of major depressive disorder (MDD) without comorbid cluster B personality disorders, and schizophrenia were differently distributed between seasons. Most (63.4%) subjects with MDD committed suicide in the spring/summer (P = 0.038). However, closer examination revealed that depressed suicides with comorbid cluster B personality disorders did not show seasonality, while 83.3% of depressed suicides without comorbid cluster B personality disorders committed suicide in the spring/summer (P = 0.019). 87.5% of those suicides with schizophrenia committed suicide in the fall/winter (P = 0.026), and the only suicide with schizophrenia who died in the spring/summer was also the only one without positive symptomology. Our study is limited to male suicide completers, and results should not be generalized to women. We conclude that seasonal variation in suicide manifests itself differently in patients with different psychopathology. These findings indicate that assessment of suicide risk may need to include consideration of possible seasonal effects, depending on psychopathology.

Attempted Suicide & Personality

Personality and attempted suicide in depressed adults 50 years of age and older: A facet level analysis.

Authors: Useda JD, Duberstein PR, Conner KR, Conwell Y.


Summary: We examined the contribution of personality traits to attempted suicide, the number of suicidal attempts, and suicidal ideation in a sample of depressed inpatients. Personality was assessed via the Revised NEO Personality Inventory (NEO-PI-R). Bivariate analyses showed that suicide attempters were more self-conscious, self-efficacious, impulsive, and vulnerable to stress, and less warm, gregarious, and inclined to experience
positive emotions. Multivariate regression analyses controlling for age, gender, severity of depression, and psychiatric comorbidity showed that patients with a lifetime history of attempted suicide were less inclined to experience positive emotions and be more self-efficacious. Patients with more severe suicidal ideation were less warm and more self-efficacious. Results indicated that specific personality traits confer risk for suicidal behaviors in middle age and older adults. Interventions tailored to specific personality profiles in this high-risk group should be developed, and their efficacy examined.

Narcolepsy Susceptibility

- A narcolepsy susceptibility locus maps to a 5 Mb region of chromosome 21q.

Authors: Dauvilliers Y, Blouin JL, Neidhart E, Carlander B, Eliaou JF, Antonarakis SE, Billiard M, Tafti M. Service de Neurologie B, Hopital Gui-de-Chauliac, Montpellier, France.


Summary: The genetic basis of human narcolepsy remains poorly understood. Multiplex families with full-blown narcolepsy-cataplexy are rare, whereas families with both narcolepsy-cataplexy and excessive daytime sleepiness without cataplexy are more common. We performed a genomewide linkage analysis in a large French family with four members affected with narcolepsy-cataplexy and 10 others with isolated recurrent naps or lapses into sleep. Only three regions showed logarithm of odds (LOD) scores greater than 1 in two-point linkage analysis (D6S1960, D11S2359, and D21S228). Genotyping additional markers provided support for linkage to 9 markers on chromosome 21 (maximum two-point LOD score, 3.36 at D21S1245). The multipoint linkage analysis using SimWalk2 provided further evidence for linkage to the same region (maximum parametric LOD score, 4.00 at 21GT26K). A single haplotype was shared by all affected individuals and informative crossovers indicated that the elusive gene that confers susceptibility to narcolepsy is likely to be located between markers D21S267 and ABCG1, in a 5.15 Mb region of 21q.

Narcolepsy & Glucose Hypometabolism

- Glucose hypometabolism of hypothalamus and thalamus in narcolepsy.

Authors: Joo EY, Tae WS, Kim JH, Kim BT, Hong SB. Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, 50 Irwon-Dong, Gangnam-Gu, Seoul, Korea.


Summary: It has been hypothesized that hypothalamus is involved in narcolepsy. The relative difference between cerebral glucose metabolism of 24 narcoleptic patients and 24 normal controls was studied using 18F-fluorodeoxy glucose positron emission tomography. Patients with narcolepsy showed significantly reduced cerebral glucose metabolism in bilateral rectal and subcallosal gyri, the medial convexity of right superior frontal gyrus, bilateral precuneus, right inferior parietal lobule, and in left supramarginal gyrus (uncorrected p < 0.001). Bilateral posterior hypothalami and mediodorsal thalamic nuclei showed hypometabolism with significance at the level of corrected p < 0.05, with small volume correction. This study showed cerebral glucose hypometabolism of the hypothalamus-thalamus-orbitofrontal pathways in the narcoleptic brain.

Psychotherapy & Diabetes Mellitus

* Integrative treatment of unstable diabetes mellitus: education or psychotherapy?


Summary: The term "brittle diabetes" describes a subtype of unstable type-I diabetes, characterized by high variations of blood sugar without any evident cause and despite careful clinical management. Clear guidelines for a precise definition of the condition are still lacking; this fosters insecurities concerning diagnosis and therapy of the disease. Psychosocial influences, triggering these conditions, were discussed. The patient-doctor-relationship appears to be tensed due to an often missing compliance. Using a paradigmatic case study as background, the specific diagnostic and therapeutic problems in brittle diabetes were presented. Brittle diabetes advocates a close cooperation between internal and psychosomatic medicine units and a combination of patient education and psychotherapy. Seen under a psychosomatic paradigm, brittle diabetes can be detected early and effective treatment may avoid further complications in these young patients.

Psychotherapy & Diabetes Mellitus

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Borderline Personality Disorder & Psychotherapy —

* Strategies for Securing Commitment to Treatment from Individuals Diagnosed with Borderline Personality Disorder

Authors: Denise D. Ben-Porath - Assistant Professor of Psychology, John Carroll University, 20700 North Park Blvd., University Heights, OH 44118; dbenporath@jcu.edu

Source: Journal of Contemporary Psychotherapy 34 (3): 247-263, Fall 2004

Summary: Individuals diagnosed with borderline personality disorder (BPD) are notoriously difficult to engage in treatment. The purpose of this paper is to highlight therapeutic strategies that are likely to facilitate early alliance in therapy with individuals diagnosed with BPD. The seven strategies include, collaborative assessment, the use of contracts, motivational interviewing, linking treatment targets to client goals, commitment strategies, validation, and the use of metaphors. Clinical vignettes are presented to elucidate the concepts described and demonstrate their use in clinical practice.

Keywords: premature termination, borderline personality disorder, psychotherapy, engagement.

BPD & Psychotherapy

* Avoiding Patient Distortions in Psychotherapy with Borderline Personality Disorder Patients

Authors: David M. Allen - Department of Psychiatry, University of Tennessee Health Science Center, 135 N. Pauline, Sixth Floor, Memphis, Tennessee; DMAAllen@utmem.edu and Stephanie Whitson - Department of Psychiatry, University of Tennessee Health Science Center, 135 N. Pauline, Sixth Floor, Memphis, Tennessee

Source: Journal of Contemporary Psychotherapy 34 (3): 211-229, Fall 2004

Summary: Patients with borderline personality disorder (BPD) have a reputation among psychotherapists for distorted thinking and misleading reports about their interpersonal relationships. This article discusses the difficulty in ascertaining whether seeming distortions are caused by true cognitive deficiencies or are instead caused by purposeful or subconscious manipulation of relationships. The tendency of BPD patients to use an impressionistic cognitive style that leaves out important details and leads to seemingly distorted reports may be related to the phenomenon of family invalidation. Empirical research into BPD distortions is reviewed. The article then describes techniques useful in psychotherapy for obtaining more factual and objective accounts of current relationship episodes. Transcripts from a therapy session are used to illustrate the techniques.

Keywords: borderline personality disorder, psychotherapy, cognitive distortion, Unified Therapy.

S.L.E

Neuropsychiatric Damage & SLE

* Predictors of neuropsychiatric damage in systemic lupus erythematosus: data from the Maryland lupus cohort.

Authors: Mikdash J, Handwerger B. - University of Maryland School of Medicine, Baltimore, MD, USA.

Source: Rheumatology (Oxford). 2004 Sep 1

Summary: Objective. To identify factors predictive of significant neuropsychiatric (NP) damage in systemic lupus erythematosus (SLE). Methods. One hundred and thirty patients with SLE were followed at the University of Maryland Lupus Clinic from 1992 until 2003. NP manifestations were defined according to the revised American College of Rheumatology (ACR) nomenclature and case definitions for NP-SLE syndromes. Disease activity was measured using the SLE Disease Activity Index (SLEDAI), organ damage using the Systemic Lupus International Collaborating Clinics Damage Index SLICC/ACR (SDI); NP damage (NPDI) was measured with the corresponding domain of the SDI. At end of study period, 64 patients exhibited no NP damage (NPDI = 0) and 66 patients developed significant NP damage (defined as NPDI >=1). The baseline features for these two patient groups were compared, and variables found to be significantly different were examined by multivariable analyses to determine their contribution to NP damage. Results. Significant NP damage is common in SLE; mortality is infrequent and the cause of death is unrelated to NP damage. Independent predictors of significant NP damage were disease activity, Caucasian ethnicity and the presence of antiphospholipid antibodies and anti-Ro/SSA antibody. Certain clinical features at baseline predicted specific NP damage. For example, higher disease activity at baseline was predictive of psychosis and cognitive impairment, anti-dsDNA was predictive of polynuropathy, and antiphospholipid antibodies were predictive of seizures and cerebrovascular accidents. Conclusions. In this longitudinal SLE cohort, significant cumulative NP damage occurred. Early aggressive therapy targeted towards NP manifestations may prevent the occurrence of NP damage.

Nocturnal Enuresis (NE)

* Complex behavioural and educational interventions for nocturnal enuresis in children

Authors: Giazenor CMA, Evans JHC, Peto RE


Summary: A substantive amendment to this systematic review was last made on 26 November 2003. Cochrane reviews are regularly checked and updated if necessary. Background: Nocturnal enuresis (bedwetting) is a socially disruptive and stressful condition which affects around 15-20% of five year olds, and up to 2% of young adults. Objectives: To assess the effects of complex behavioural and educational interventions on nocturnal enuresis in children, and to compare them with other interventions.

Search strategy: We searched the Cochrane Incontinence Group trials register (December 2002) and the reference lists of relevant articles. Date of the most recent searches: December 2002.

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Neuroleptic reduction and/or cessation and neuroleptics as specific treatments for tardive dyskinesia

**Authors:** McGrath JJ, Soares-Weiser KVS
**Summary:** A substantive amendment to this systematic review was last made on 25 February 1998. Cochrane reviews are regularly checked and updated if necessary.

**Background:** Since the 1950s neuroleptic medication has been extensively used to treat people with chronic mental illnesses, such as schizophrenia. These drugs, however, have been also associated with a wide range of adverse effects, including movement disorders such as tardive dyskinesia (TD). Various strategies have been examined to reduce a person’s cumulative exposure to neuroleptics. These strategies include dose reduction, intermittent dosing strategies, such as drug holidays, and neuroleptic cessation.

**Objectives:** To determine whether, for those people with both schizophrenia (or other chronic mental illnesses) and tardive dyskinesia (TD), a reduction or cessation of neuroleptic drugs was associated with reduction in TD symptoms. A secondary objective was to determine whether the use of specific neuroleptics for similar groups of people could be a treatment for already established TD.

**Search strategy:** Electronic searches of Biological Abstracts (1982-1997), Cochrane Schizophrenia Group’s Register of trials (1997), EMBASE (1980-1997), LILACS (1982-1996), MEDLINE (1966-1997), PsycLIT (1974-1997), and SCISEARCH (1997) were undertaken. References of all identified studies were searched for further trial citations.

**Principal authors of trials were contacted.**

**Selection criteria:** Reports were included if they assessed the treatment of neuroleptic-induced tardive dyskinesia in people with schizophrenia or other chronic mental illnesses and already established TD, who had been randomly allocated to (a) neuroleptic cessation (placebo or no intervention) versus neuroleptic maintenance; b. neuroleptic reduction (including intermittent strategies) versus neuroleptic maintenance; or c. specific neuroleptics for the treatment of TD versus placebo or no intervention.

**Data collection and analysis:** The reviewers extracted the data independently and the Odds Ratio (95% CI) or the average difference (95% CI) were estimated. The reviewers assumed that people who dropped out had no improvement.

**Main results:** Two trials were able to be included in this review. Sixty two were excluded and 16 are awaiting assessment. Seven trials are still pending classification. No randomised controlled trial-derived data were available to clarify the role of neuroleptics as treatments for TD. This includes the atypical antipsychotics including clozapine. Despite neuroleptic cessation being a frequently first-line recommendation, there were no RCT-derived data to support this. Two studies (Cookson 1987, Kane 1983) found a reduction in TD associated with neuroleptic reduction.

**Reviewers’ conclusions:** The lack of evidence to support the efficacy of neuroleptic cessation as a treatment for TD, combined with the accumulating evidence of an increased risk of relapse should antipsychotic drugs be reduced, makes this intervention a hazardous treatment for TD. Dose reduction may offer some benefit as a treatment for TD compared to standard levels of neuroleptic use. There is a need to evaluate the utility of clozapine and the ‘atypical’ antipsychotics as treatments for established TD.

**Neuroleptic reduction and/or cessation and neuroleptics as specific treatments for tardive dyskinesia**

**TD & Neuroleptic**

**TD & Benzozezepines**

**Authors:** Walker P, Soares KVS
**Summary:** A substantive amendment to this systematic review.
Chronic Fatigue Syndrome (CFS) was last made on 06 February 2003. Cochrane reviews are regularly checked and updated if necessary.

**Background:** Tardive dyskinesia (TD) is a potentially disfiguring movement disorder of the orofacial region often caused by the use of neuroleptic drugs. A wide range of strategies have been used to help manage tardive dyskinesia and, for those who are unable to have their antipsychotic medication stopped or substantially changed, the benzodiazepine group of drugs has been suggested as a useful adjunctive treatment.

**Objectives:** To determine the effects of benzodiazepines for people with neuroleptic-induced tardive dyskinesia and schizophrenia or other chronic mental illnesses.


**Selection criteria:** All randomised clinical studies focusing on people with both schizophrenia or other chronic mental illnesses and neuroleptic-induced tardive dyskinesia and comparing benzodiazepines with placebo or no intervention.

**Data collection and analysis:** Studies were reliably selected, quality assessed and data extracted. Data were excluded where more than 50% of participants in any group were lost to follow up. For binary outcomes a fixed effects risk ratio (RR) and its 95% confidence interval (CI) was calculated. Where possible, the weighted number needed to treat/harm statistic (NNT/H), and its 95% confidence interval (CI), was also calculated. For continuous outcomes, endpoint data were preferred to change data. Non-skewed data from valid scales were synthesised using a weighted mean difference (WMD). If statistical heterogeneity was found by Mantel-Haenszel chi-square test, random effects models were used.

**Main results:** Two small trials (total n=32) were included. Using benzodiazepines as adjunctive treatment did not result in any clear changes for a series of tardive dyskinesia medium term outcomes (RR not improved to a clinically important extent 1.08 CI 0.57 to 2.05, n=30, 2 RCTs; RR not improved at all 1.91 CI 0.3 to 5.3, n=30, 2 RCTs; RR deterioration 1.85 CI 0.3 to 10.1, n=30, 2 RCTs). Adverse effects were not reported.

**Reviewers’ conclusions:** The 2002 update has added almost no extra data. This is clearly not an area of active research. Benzodiazepines may have something to contribute to the care of people with tardive dyskinesia but the use of this group of compounds should be considered experimental. Large definitive studies are indicated.

**Chronic Fatigue Syndrome (CFS) & Exercise Therapy**

**CFS & Exercise Therapy**

**Authors:** Edmonds M, McGuire H, Price J


**Summary:** A substantive amendment to this systematic review was last made on 08 May 2004. Cochrane reviews are regularly checked and updated if necessary. Background: Chronic fatigue syndrome (CFS) is an illness characterised by persistent medically unexplained fatigue. CFS is a serious health-care problem with a prevalence of up to 3%. Treatment strategies for CFS include psychological, physical and pharmacological interventions.

**Objectives:** To investigate the relative effectiveness of exercise therapy and control treatments for CFS.

**Search strategy:** CDDANCTR-Studies and CENTRAL were searched using “Chronic Fatigue” and Exercise. The Journal of Chronic Fatigue Syndrome and CFS conferences were handsearched. Experts in the field were contacted. Clinicaltrials.gov and controlled-trials.com were searched.

**Selection criteria:** Only Randomised Controlled Trials (RCT) including participants with a clinical diagnosis of CFS and of any age were included.

**Data collection and analysis:** The full articles of studies identified were inspected by two reviewers (ME and HMG). Continuous measures of outcome were combined using standardised mean differences. An overall effect size was calculated for each outcome with 95% confidence intervals. One sensitivity analysis was undertaken to test the robustness of the results.

**Main results:** Nine studies were identified for possible inclusion in this review, and five of those studies were included. At 12 weeks, those receiving exercise therapy were less fatigued than the control participants (WMD -0.77, 95% CIs -1.26 to -0.28). Physical functioning was significantly improved with exercise therapy group (WMD -0.64, CIs -0.96 to -0.33) but there were more dropouts with exercise therapy (RR 1.73, CIs 0.92 to 3.24). Depression was non-significantly improved in the exercise therapy group compared to the control group at 12 weeks (WMD -0.58, 95% CIs -2.08 to 0.92). Participants receiving exercise therapy were less fatigued than those receiving the antidepressant fluoxetine at 12 weeks (WMD -1.24, 95% CIs -5.31 to 2.83). Participants receiving the combination of the two interventions, exercise + fluoxetine, were less fatigued than those receiving exercise therapy alone at 12 weeks, although again the difference did not reach significance (WMD 3.74, 95% CIs -2.16 to 9.64).When exercise therapy was combined with patient education, those receiving the combination were less fatigued than those receiving exercise therapy alone at 12 weeks (WMD 0.70, 95% CIs -1.48 to 2.88).

**Reviewers’ conclusions:** There is encouraging evidence that some patients may benefit from exercise therapy and no evidence that exercise therapy may worsen outcomes on average. However the treatment may be less acceptable to patients than other management approaches, such as rest or pacing. Patients with CFS who are similar to those in these trials should be offered exercise therapy, and their progress monitored. Further high quality randomised studies are needed.

**Family, Conduct Disorder & Delinquency Aged**

**Family, Conduct Disorder & Delinquency Aged**

**Authors:** Woolfenden SR, Williams K, Peat J

**Source:** The Cochrane Library, Issue 3, 2004. Chichester, UK: John Wiley & Sons, Ltd

**Summary:** A substantive amendment to this systematic review was last made on 28 February 2001. Cochrane reviews are
regularly checked and updated if necessary.

Background: Conduct disorder and delinquency are significant problems for children and adolescents and their families, with the potential to consume much of the resources of the health, social care and juvenile justice systems. A number of family and parenting interventions have been recommended and are used for these conditions. The aim of this review was to determine if these interventions are effective in the management of conduct disorder and delinquency in children and adolescents, aged 10-17. Objectives: To determine if family and parenting interventions improve the child/adolescent's behaviour; parenting and parental mental health; family functioning and relations; and have an effect on the long term psychosocial outcomes for the child/adolescent.

Search strategy: Randomised controlled trials were identified through searching the Cochrane Controlled Trial Register (CCTR), databases (MEDLINE, EMBASE, PsycINFO, CINAHL, Sociofile, ERIC, Healthstar), reference lists of articles and contact with authors.

Selection criteria: Randomised controlled trials with a major focus on parenting and/or family functioning were eligible for inclusion in the review. Trials needed to include at least one objective outcome measure (e.g. arrest rates) or have used a measure that had been published in peer review publications and validated for the relevant purpose. Studies were required to have a control group, which could be a no intervention group, a wait list group or a usual intervention group (e.g. probation). Trials in children and adolescents aged 10 to 17 years with conduct disorder and/or delinquency and their families were considered. Conduct disorder was defined by a standardised psychological assessment (for example, using a child behaviour checklist), or a psychiatric diagnosis. Delinquency was defined by a referral from a juvenile justice or another legal system for a child/adolescent who has committed a serious crime e.g assault and/or offended on at least two occasions.

Data collection and analysis: Two reviewers independently reviewed all eligible studies for inclusion, assessed study quality (allocation concealment, blinding, follow up, clinically important outcomes) and extracted data. Heterogeneity was assessed using the Chi squared test of heterogeneity along with visual inspection of the data. A significance level less than 0.1 was interpreted as evidence of statistically significant heterogeneity. For data where heterogeneity was found the reviewers looked for an explanation. If studies with heterogeneous results were thought to be comparable the statistical synthesis of the results was done using a random effects model. This model takes into account within-study sampling error and between-studies variation in the assessment of uncertainty and will give wider confidence intervals to the effect size and hence a more conservative result. Sensitivity analysis was performed to explore the effects of the varying quality of the studies included on the results.

Main results: Of the nine hundred and seventy titles initially identified through the search strategy, eight trials met the inclusion criteria. A total of 749 children and their families were randomised to receive a family and parenting intervention or to be in a control group. In seven of these studies the participants were juvenile delinquents and their families and in only one the participants were children/adolescents with conduct disorder who had not yet had contact with the juvenile justice system. At follow up, family and parenting interventions significantly reduced the time spent by juvenile delinquents in institutions (WMD 51.34 days, 95% CI 72.52 to 30.16). There was also a significant reduction in the risk of a juvenile delinquent being re arrested (RR 0.66, 95% CI 0.44 to 0.98) and in their rate of subsequent arrests at 1-3 years (SMD -0.66, 95% CI -1.100 to -0.03). For both of these outcomes there was substantial heterogeneity in the results suggesting a need for caution in interpretation. At present there is insufficient evidence that family and parenting interventions reduce the risk of being incarcerated (RR=0.50, 95% CI 0.20 to 1.21). No significant difference was found for psychosocial outcomes such as family functioning, and child/adolescent behaviour.

Reviewers’ conclusions: The evidence suggests that family and parenting interventions for juvenile delinquents and their families have beneficial effects on reducing time spent in institutions. This has an obvious benefit to the participant and their family and may result in a cost saving for society. These interventions may also reduce rates of subsequent arrest but at present these results need to be interpreted with caution due to the heterogeneity of the results.

**Dysthymia**

**Dysthymia & Pharmacotherapy**

**Authors**: Lima MS, Hotopf M


**Summary**: A substantive amendment to this systematic review was last made on 27 April 2003. Cochrane reviews are regularly checked and updated if necessary.

**Background**: Many drug treatments have been proposed for the treatment of dysthymia, but with so many potential comparisons it is not possible at the present time to determine which is the treatment of choice. There is a need to know whether the different classes of antidepressants have similar efficacy. In addition, the tolerability of treatments may be even more important, since dysthymia is a chronic condition characterised by less severe symptoms than major depression. Objectives: To conduct a systematic review of all randomised controlled trials comparing two or more active drug treatments for dysthymia.

**Search strategy**: Electronic searches of Cochrane Library, EMBASE, MEDLINE, PsycLIT and LILACS, Biological Abstracts; reference searching; personal communication; unpublished trials from pharmaceutical industry.

**Selection criteria**: Only randomised and quasi-randomised controlled trials were included. Trials had to compare at least two active drug treatments in the treatment of dysthymia. Exclusion criteria were: non-randomised studies, studies which included patients with mixed major depression/dysthymia and studies on depression/dysthymia secondary to other disorders (e.g. substance abuse).

**Data collection and analysis**: The reviewers extracted the data independently and odds ratios, weighted mean difference and number needed to treat were estimated. The reviewers assumed that people who died or dropped out had no subsequent benefit. At present there is insufficient evidence that family and parenting interventions reduce the risk of being incarcerated (RR=0.50, 95% CI 0.20 to 1.21). No significant difference was found for psychosocial outcomes such as family functioning, and child/adolescent behaviour.

**Reviewers’ conclusions**: The evidence suggests that family and parenting interventions for juvenile delinquents and their families have beneficial effects on reducing time spent in institutions. This has an obvious benefit to the participant and their family and may result in a cost saving for society. These interventions may also reduce rates of subsequent arrest but at present these results need to be interpreted with caution due to the heterogeneity of the results.
conclusions: The conclusion is that the choice of drug must be made based on consideration of drug-specific side effect properties.

**Somatoform Disorders (SD)**

**SD & Pharmacotherapy**

- **Pharmacotherapy of somatoform disorders.**
  
  **Authors:** Fallon BA, Department of Psychiatry, Division of Therapeutics, Somatic Disorders Program, New York State Psychiatric Institute, Columbia University, 1051 Riverside Drive, Unit 69, New York, NY 10032, USA. balf@columbia.edu
  
  **Source:** J Psychosom Res. 2004 Apr;56(4):455-60.
  
  **Summary:** OBJECTIVE: This paper reviews the published literature on the pharmacologic management of somatoform disorders. METHODS: Using Medline, the author identified all articles published between 1970 and 2003 on this topic, selecting the best-designed studies for inclusion. RESULTS: The review reveals that patients with the obsessional cluster of somatoform disorders (hypochondriasis and body dysmorphic disorder [BDD]) respond well to serotonin reuptake inhibitors (SRIs). Less is known about the pharmacologic responsiveness of patients with the primarily somatic cluster of somatoform disorders (somatization, pain), a patient group that is common in the health provider's office. CONCLUSIONS: Improvements in the design of future clinical trials are needed. A particular focus needs to be applied to study the neglected area of the pharmacologic treatment of syndromal and subsyndromal somatization and pain disorders. Copyright 2004 Elsevier Inc.

**Electroconvulsive Therapy (ECT)**

**ECT & Value of Diagnostic**

- **Value of diagnostic imaging in evaluation of electroconvulsive therapy [Article in German].**
  
  **Authors:** Frodl T, Meisenzahl EM, Moller HJ. Psychiatrische Klinik, Ludwigs-Maximillians-Universitat, Munchen, Germany
  
  **Source:** Nervenarzt. 2004 Mar;75(3):227-33.
  
  **Summary:** Magnetic resonance imaging (MRI), positron emission tomography (PET), and single photon emission computed tomography (SPECT) may be able to investigate the clinical efficacy and underlying neuronal processes of electroconvulsive therapy (ECT). The following review focuses on neuroimaging in ECT. Neuroimaging findings support that ECT does not result in significant macroscopic structural changes. However, in patients with subtle structural changes such as subcortical lesions and dilatation of lateral ventricles before ECT, the possibilities of poor therapeutic outcome, increased incidence of delirium, and longer-lasting cognitive deficits should be considered. Functional studies show reduced blood flow and glucose metabolism during the first days after ECT. Afterwards, their normalization can be observed, which seems to correlate to clinical improvement. The importance of this suppression effect needs to be further elucidated. Future studies of receptor systems and longitudinal studies will open new perspectives in future imaging research.

**Huntington Chorea (HC)**

**HC & Neuropsychiatric Aspects**

- **Neuropsychiatric aspects of Huntington chorea. Presentation of 2 cases and review of the literature (Article in German).**
  
  **Authors:** Tost H, Schmitt A, Brassen S, Wendt CS, Braus DF. NMR-Forschung am Zentralinstitut fur Seelische Gesundheit Mannheim, Universitat Heidelberg.
  
  **Source:** Nervenarzt. 2004 Mar;75(3):258-66.
  
  **Summary:** Huntington's disease (HD) is an autosomal, dominant, inherited disorder of the central nervous system with characteristic neurodegenerative alterations in the basal ganglia and cortex. Dependent on the individual CAG expansion load, disease onset occurs between the third or fourth decade of life, entailing an invariably lethal progression within 10 to 20 years. Although the clinical picture is characterized equally by cognitive and psychiatric disturbances, the apparent neurodegenerative alterations and presentation as a choreatic movement disorder account for the traditional link of Huntington's disease to the field of neurology. In contrast to the traditionally emphasized core features of chorea and dementia, recent empirical evidence points to the frequent emergence of nonchoreic motor signs and subtle cognitive and psychiatric complaints, especially in asymptomatic gene carriers and early disease stages. The case studies presented here emphasize the spectrum of neuropsychiatric phenomena associated with HD and illustrate the resulting difficulties of differential diagnosis in clinical settings. Furthermore, current scientific knowledge of HD pleiotropy is reviewed and the diagnostic power of specific neuropsychological approaches is explained.