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إصدار مؤسسة العلوم النفسية العربية - تونس

عادل صادق ... الكرامة حرية

د. جمال التركي - الطب النفسي / تونس

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لا يهمل الإنسان الحياة فاما إلا وهو على وشك مغادرتها .. فما أن يبدأ في الفهر حتى تهدر خلايا مخه وتصبح غير قادرة على الفهر فيموت الإنسان مثلاً ولد لا يعرف شيئاً .. يولد جاهلاً ويموت جاهلاً .. وكل إنسان في مرحلة حياته مكتوب عليه قدر من الحزن وقدر من الفرح .. ربما بالنسبة للنساء .. تدهب المصائب فيكسب .. وتدهور الذائبة .. وينسى .. وبقيته فرح .. فينشي .. ويعقب فرحه فنور .. يذهب عنه الزهو، وينهاوى الفخار، وهذه حكمة بليغة إذ يجب أن يشعر بضعة ولا مانع أن يشعر أيضاً بالذل .. أي انه لا شيء .. وهذه هي مشاعر المكسب .. مشاعر من الذونية والخطا والنبش والنزق .. ولو أن الإنسان ترك له أن يشعر إلى ما لا نهاية بالقوة والنية والفخار لطغى ويغى وسيطر وتجبر .. ولكن الإنسان في هذه الحياة كالجالس على الأرجوحة مرة إلى الأمام ومرة إلى الخلف .. مرة إلى أعلى .. ومرة إلى أسفل .. لا يبقى في علو دائره ولا قذف به غيرة وتخطى رقبته .. ولا يبقى في دنو دائره ولا سحقه اليأس ودفنه تحت الأرض.

أ. د. عادل صادق: الأهرام الأسبوعي - العدد 123 - السبت 26 يناير 2002

الأسناد الدكتور عادل صادق في ذمة الله ...

و نحن نسعد لإقبال هذا العدد من المجلة فجعنا و الأوساط الطبسية العربية والعلمية بفقد علم من أعلام هذا الاختصاص . فجعنا بأن غيب الموت زميلاً عزيزاً و أسناداً فاضلاً ... رحمه الله عادل صادق رحمة واسعة ... كان وقع الصدمة شديداً على من عرفه كإنسان، كطبيب، كزميل، كأسناد و كمفكر ... فقلناه في زمن الهوان العربي، .. زمن لم تكن حاجتنا له و لا لمتاله في يوم أكش من حاجتنا إليه اليوم ... فقلنا فيه رجلاً حمل المهمل العربي و كابد معاناته في كل خلية من خلاياه ... فقلناه يوم أطلق صرخته "هيا الهضي يا أمة العرب" قبل فترة قصيرة من وفاته، يومها أعلن أن "كثير من الناس كسالى و قابعون، قليل منهم يتقودون عن كون المياه الرأككة، يبعثون الأمل في النفوس الياسته، يشطون الروح في الأجساد الحاملة" أطلق صرخته في زمن أحاطت بنا الخطوب ... زمن القهقري والحدلان والانعكاس، أطلق صرخته و كأنني به لم يعد يقدر أن يفعل ما آل إليه حالنا فكان أن تلقفناه العناية الإلهية رحمة به من هومر أضنه مجباً إياه ما لم يعد يتأى غمله ... إنا و نحن نفقد لا يسعنا إلا أن ندعو الله العلي القدير أن ينغمه برحمته الواسعة و يسكنه فرديس جناته من دفين "أن إلى لقاء" كما ردها أحد عكاشته يوم تأينه و "أن البقاء لله" كما ردها غيبي الخاوي يوم فقهه منسلاً لمن البقاء فعلا بين البشر عبر العالم؟ هل البقاء للأقوى أم للأشطر أم للأضمر سلاحا؟ أم للأخبث عابراً أم للأخلاق إعلاماً؟ ... "ترك عادل صادق دنياه دون ضجيج أو تكرير يلق به المألوف في عقوداً من حياته في العمل الدؤوب لعلاج المرضى النفسيين من أعلى القمة حتى قاع المجمع ولكن لم يذكره أحد عندما رحل .. كل ذنبه أنه طبيب نفسي وهذه وصمة عار في مجتمعاتنا العربية ..!!!!" (د. الحضير). قد يكون هذا جزءاً من حقيقة واقعنا العربي الذي نسعى إلى المساهمة في تطويره و سنعمل كأُسرة في شبكة العلوم النفسية العربية أن نكرمه بطريقنا ... نكرمه بإصدار عدد خاص به من المجلة الإلكترونية، نعرض فيه لأهم محطات مسيرته العلمية و مواقفه الفكرية مع ملخصات أهم أبحاثه (مقالات، أطروحات، عوثر ميدانية) و شهادات من عرفه كإنسان، عرفه كأسناد، عرفه كطبيب و إنني أدعو الزملاء الذين حصل لهم شرف العمل معه أو الذين تلمذوا عليه أن يساهموا في تأسيس هذا العدد بملنا بما لديهم من وثائق و أبحاث و مقالات.

إن تكرير عادل صادق إنجاز فكره عالم فقلناه له حق علينا إلى إعادة الاعتبار للذات. إن هميش الأمانة لعلمائها ازدهاراً لمتوماتها و كيونتها، إن علماء الأمر مصابيح فندى هم مجتمعاتها و إن فكرهم معالم في طريق النهضة و لا نهضة بدون مرد الاعتبار للذات، لا نهضة بدون كرامة و لا كرامة بدون حرية، "الكرامة حرية" هكذا أعلنها عادل صادق يوم تحدث عن الحرية ... الحرية هي ألا يعني أحد من فعل الخير من أجل نفسي وعشيرتي وبلدي، حرية أن أفكر وأن أشعر وأن

أندرك بطريقتي الخاصة... بأسلوب الموروث ومن وحي قرائي وقارئتي وعقائدي وقناعاتي... نعم الكرامة حرية... لا كرامة مع الاستبداد... لا كرامة مع الاحتلال... لا كرامة في ظل الجهل والخلف... نسأل الله الرحمة والمغفرة لعادل صادق. اللهم لا تحرمنا أجره ولا تفتنا بجلده واغفر لنا ولهم.

من مخزونات العدد...

يشارك العدد الثالث مجموعة من أساتذة الطب النفسي و علم النفس بأبحاث علمية مميزة لمسجلات الاختصاص حيث يعرض أحد عكاشات تصنيفات الاضطرابات الثقافية وما شهد هذا الاضطراب مؤخرًا من مراجعات جنسية سواء على مستوى التشخيص أو المقاربة العلاجية وما لأهمية معدلات المزاج من استقرار المرض على المدى الطويل، كما يشترك كل من الرخاوي (مصر)، التركي (تونس)، عبد القوي (مصر)، الخطيب (الأردن)، و رضوان (سوريا) بأبحاث تناولت قراءة في معنى ودلالة لغة ظاهرة الإلحاح، اضطرابات خلل الوجدان السابق للحيض، علاقة أفضلية اليد بالوظائف المعرفية، المراجعات النفسية في الأردن و مرهاب إلهاء عقد العمل، تأتي هذه الأبحاث في زمن قلنى فيه العطاء العلمي العربي نتيجة الضغوطات والرضات التي يمر بها عالمنا. إننا إذ نرفع حقبة قديم و إكبار لهؤلاء الحامضون في أرض صعبة المراس ندعو مختصي العلوم النفسية في أوطاننا للمساهمة بالأبحاث الميدانية والدراسات العلمية أملا في رفع مستوى الباقية النفسية للإنسان العربي التي تشهد أدنى مستوياتها و ما إحصاءات الهيئة الصحية العالمية لا دليلا على ذلك عندما تشير إلى أن نحو 80 في المائة من الشعب العربي يعانون من عقد نفسية، وأن 5 في المائة يخضعون للعلاج. إلهاء أرقام مفرغة، قصدي لها حب الله (لبنان) بالتحريك والتحليل في مقالته "غياب علم النفس عن الفكر العربي" مؤكدا أن على الإنسان العربي أن يعبر كمي يتوصل إلى مستوى من المسؤولية ومن الوعي الذاتي فكم من حل مسائله الشائكة وأول مستويات العصر أن يعبر ذاته من رواسب الموروث الثقافي التي جسي لينقل رسالته إلى محبيه في إطار علمي قابل للجدل، مضافا أنه مطلوب من كل عربي أن يعلن مواقفه من خمس مسائل أساسية: حكم الأكثرية، الحرية، مفهوم الفرد، موضوع المرأة، الخطاب العلمي. و يعالجها فكريا و ثقافيا لأن العربي بعد أن عرف (تبدل الحدود الجغرافية و انفتاح الفضاء على كامل القضايا) لا يستطيع القول إنه لا يعرف، كما أن المعرفة إن أدت إلى "الأسر العقلي" و محو خصائص الهوية الثقافية و غس الذات فلا خير فيها وهو ما ذهب إليه النابلسي في مقالته "مستقبل العلوم الإنسانية في الوطن العربي" عندما أكد أن "العقل الأسير" عقلا مكمكا بين انتمائه الموروث و انتمائه المكتسب و أنه يعاني الرفض المزددج، مرفوض في الثقافة التي تأسر لكونه حاملا بذور "عالمه الأصلي" كما أنه مرفوض في ثقافته لرفض هذه الثقافة مبدأ الأسر و لعجزها عن تأمين شروطه، و هكذا ينحول العقل الأسير إلى مرفوض في حيلة دفاعية لتجنب الاعتراف بكونه مرفوضا، إن رفض "الأسر العقلي" يستوجب أيضا رفض "السلط الفكري" الذي يمارسه ثقافة مسيطرة على أخرى مستضعفة و في هذا الإطار يدعو عيسى الرخاوي في قراءته لأحداث الحادي عشر من سبتمبر إلى ضرورة إعادة قراءة الثقافات المختلفة و التعامل معها كثقافة تستوجب الاحترام بعيدا عن التبئيس و الهميش، إن الحادي عشر من سبتمبر إن لم يدعونا لإعادة قراءة الفكر المخالف بعقلية التكافؤ و الندية فلن يكون إلا بداية صراعات قد يدفع بها ضحايا الإنسان أيتما و جلد و في نطاق قبل الآخر و الاعتراف به كمختلف تعرض غيتا. الخياط لجانب من مسيرة ذي كليميو التي قضى جزءا منها بالمغرب وكيف كان تعامله مع الثقافة العربية المختلفة جندرها عن الثقافة الغربية.

مخزوني هذا العدد في أبوابه الثابتة إلى تعريف بالجمعية العراقية لعلم النفس مصحوبا بالنقاء الذي وجهته إلى المؤسسات و الجمعيات العلمية للموازنة و الدعم و إقامة أواصر التعاون العلمي معها. و إلى عرض أهم مسجلات الاختصاص للتلازمة الثانية لعام 2004. و في باب المؤتمرات تعرض للمؤتمر العربي السادس للوقاية من الإلحاح، للملتقى الكويتي المختص في العلاج النفسي، لشهادة الماجستير في الطب النفسي و لورشات العمل التي يظلمها بالأردن المركز العربي للأبحاث النفسية و التحليلية حول المدرسة و الجامعة: أزمت، عقبات إضافة لأجندة المؤتمرات النفسية العربية و العالمية للتلازمة الرابعة لسنة 2004 هذا و جاءت أبواب مراجعة كتب و مجلات موشحة بعرض كتاب سيكولوجية الشائكة للنابلسي و ملخصات العدد التاسع عشر من مجلة الطفولة العربية (الكويت).

كما تراسعادات أبواب جديدة تمثلت في باب جوائز العلوم النفسية تعرض فيه لجوائز الجمعية العالمية للطب النفسي المتمثلة في جائزة جان دلاي للطب النفسي، مكافأة عكاشة للدول السائرة في طريق النمو و جائزة جينيف لحقوق الإنسان في الطب النفسي و باب المعجم النفسي حيث تعرض بعض المصطلحات النفسية باللغات العربية و الإنجليزية و الفرنسية من الحرف الأبجدي الأول لكل لغة، يأتي أحداث باب المعجم النفسي في نطاق التأسيس لاستشارة موسعة بين المهنيين بترجمة المصطلح سعيا لتوحيد ترجمة و تحت المصطلح النفسي مناجاة للظهور السريع الذي يشهده الاختصاص.

STATE OF THE ART IN THE MANAGEMENT OF BIPOLAR DISORDER *

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Abstract :In this paper there is a revision of the classification and prevalence of mood disorders, with the emphasis on the higher rates of prevalence of Bipolar Disorders in recent studies. The clinical phenomenology is updated with some discussion of the misunderstood classification and inconsistent diagnosis and treatment worldwide. The managements in psychiatry generally and in Bipolar disorder in particular has been discussed. The pharmacotherapy of Bipolar Disorder is reviewed. Lithium, Novel antipsychotics and the management of acute mania or mixed episodes as well as acute depression and rapid cycling also maintenance treatment is clarified, without forgetting the importance of psychosocial intervention. The conclusion calls for more research in the field.

Introduction

An application of DSM-IV criteria (Zurich cohort) produced prevalence for mania and hypomania of 5.5% by age 35. In a recent study, Angst (1998) reported that, while 13 epidemiologic studies since 1980 had persistently found a low lifetime prevalence of mania (0.0-1.7%), the application of DSM-IV criteria by trained clinical psychologists produced a prevalence for mania and hypomania of 5.5% by age 35. A broadening of diagnostic criteria to include other aspects of the bipolar spectrum (hypomania, cyclothymia, and bipolar disorder not otherwise specified) in six studies since 1978 yields a prevalence ranging between 3.0 and 8.8%. Depression usually causes more subjective distress than does mania. Hence, patients are more likely to seek help for depression than for mania. This high prevalence suggests that bipolar disorder is a general health problem with significant social and economic sequel, a fact of which policy makers should be aware.

Clinical Phenomenology of Bipolar Subtypes.

Akiskal and Pinto (1999) broadened the bipolar spectrum and suggested several subtypes as follows:

Bipolar 1: Full - Blown Mania

Bipolar 1 1/2 : Depression with protracted hypomania

Bipolar 2: Depression with hypomania

Bipolar 2 1/2 : Cyclothymic depression

Bipolar 3: Antidepressant associated hypomania.

Bipolar 3 1/2: Bipolarity Masked and unmasked by stimulant abuse.

Bipolar 4: Hyperthymic Depression.

Bipolar V & VI: Not yet defined.

Mixed states

Mixed states, also called dysphoric mania, constitute 5 - 7 % of cases. This category has been recently acknowledged as a distinct entity of bipolar disorder, occupying an intermediate position on a spectrum between mania and depression. It is a severer form of bipolar disorder and its phenomenology consists of a superimposed independent mania and depression

Morbidity and Social Cost

Bipolar disorder is the 6th leading cause of disability worldwide and is a recurrent illness in more than 90% of patients. Suicidal ideation is present in 25-50 % in cases of mixed mania. At least 25% of bipolar patients attempt suicide,

while the actual suicide rate is estimated between 11-19 %. Co morbid substance abuse or other psychiatric diagnoses (McElroy et al., 2001) and/or medical conditions (Strakowski et al., 1994) are highly prevalent in bipolar disorder.

Functional recovery often lags behind symptomatic and syndromal recovery. Recurrent episodes may lead to progressive deterioration in functioning and the number of episodes may affect subsequent treatment response and prognosis, hence the importance of early identification and intervention.

Psychosocial disability from bipolar disorder remains extensive and encompasses multiple domains, including work and social functioning, independent community living, family adjustment, premature mortality, and diminished quality of life.

In an Egyptian retrospective study of a sample of bipolar patients, Okasha et al., (In Press) found that the syndromic recovery was almost double that of functional recovery, including work, domestic and interpersonal relationships.

Goldberg et al. (1995) found at both 2- and 5-year follow-ups that fewer than one-quarter of bipolar patients with affective relapses had steady work performance, and that affective relapse led to impaired work functioning more profoundly among bipolar than unipolar patients. At least one quarter of euthymic bipolar patients show impaired insight and an inability to recognize affective prodromes.

The economic burden of treatment of bipolar disorder is poorly understood. A recent study (Johnstone et al., 2001) discussed the direct cost of care for bipolar disorder in an employer claims database representing the health care experience of approximately 1.6 million covered lives. The study examined the estimated cost of care including expenditures for hospitalisation, hospital outpatient services, outpatient medications, psychiatric day/night facilities, nursing home facilities, office visits, laboratory tests, substance abuse treatment, and other services. The prevalence of bipolar disorder in the population was 5.5 patients per 1000 eligible members. These patients incurred significant annual expenditures, totaling \$ 13,402 in 1995, \$ 11,856 in 1996, and \$ 11,146 in 1997. These expenditures were comparable to the costs of treatment for schizophrenia in the same population during this period. Annual costs for outpatient use of mood stabilizers and antipsychotic medications increased by \$ 168 (42%) over the study interval, totaling \$ 568 in 1997. However, other costs of care for these patients decreased by \$ 2,424 (i.e., more than 80%) during the same period.

Caveats in International Classification

Bipolar disorder, however, remains frequently misunderstood, leading to inconsistent diagnosis and treatment. Bipolar disorder is under diagnosed and under recognized and frequently misdiagnosed as unipolar major depressive disorder, which can increase the burden of the disorder. Reasons for under diagnosis include patients' impaired insight into mania, failure to involve family members in the diagnostic process, and inadequate understanding by clinicians of manic symptoms. Slavish adoption of the DSM- IV and ICD-10 definitions may have hindered research into the etiology of mental disorders and contributed to the high degree of short-term diagnostic instability for many disorders. The concrete use of DSM-IV and ICD-10 entities where they are considered to be equivalent to "diseases" is more likely to obscure rather than elucidate research findings. In recent years, much progress has been made in the diagnosis and treatment of schizophrenia and depression. The work by Berrettini (1996) indicating that three of the putative susceptibility loci associated with bipolar disorder also contributes to the risk of schizophrenia. Bipolar disorder however remains frequently misunderstood, leading to inconsistent diagnosis and treatment. The states of diagnosis and treatment in bipolar disorder are suboptimal. More diagnostic attention to manic criteria is necessary.

Management strategies

Lack of treatment specificity is the rule rather than the exception. The current pattern of use of antidepressants in bipolar disorder needs to change. Antidepressants are probably overused and mood stabilizers underused. Why is this the case? Many fundamental aspects of the therapeutics of bipolar disorders remain remarkably underdeveloped and require further systematic study.

Goals of Management in Psychiatry

Any psychiatric management attempts to fulfill a series of objectives. Initially there is the objective to establish and maintain a therapeutic alliance, which is a prerequisite for any doctor patient relationship. There is also a need to monitor the patient's psychiatric status, provide education regarding bipolar disorder, enhance treatment adherence, and promote regular patterns of activity and sleep. On the preventive side a sound management plan should be able to anticipate stressors, early identify indicators for new episodes and seek to minimize functional impairments.

In the case of bipolar mania, specific goals involve control of dangerous symptoms such as suicide, agitation and psychosis, stabilize mood, i.e. control mania without provoking depression. Treatment should target all faces of mania including depressive, anxious and psychotic elements. The most important guideline in the above process is to monitor overdose and protect patient from harmful side effects such as severe toxicity and teratogenicity. The long-term objective of treatment of bipolar mania is to restore patient's premorbid functioning. This entails a simplification of patient's daily routines, enhancement of compliance through simple dosing, avoiding annoying side effects or dulling and limitation of needs for medical procedures.

To meet the above requirements we are in need of an "Ideal Mood Stabilizer" which is effective over Time and across episodes. Such ideal medication should have a rapid efficacy for mania, treat psychotic symptoms while at the same time have a broad efficacy spectrum (e.g. mixed, rapid cycling). At the same time it should reduce depressive symptoms,

maintain favorable cognitive functions, enjoy long-term usefulness, be easy to use, safe and well tolerated, and, ideally, affordable.

Pharmacotherapy in bipolar disorder

Mood-stabilizing Agents that have been developed or used in an attempt to achieve the above recipe are variable. The most classic is Lithium. But then there are also the anticonvulsants: Carbamazepine (Tegretol), and oxycarbazepine (Trileptal), Valproate (Depakene, Depakote), Lamotrigine (Lamictal), Gabapentin (Neurontin), Topiramate (Topamax), Benzodiazepines such as Clonazepam or Klonopin and finally conventional (e.g. Haloperidol) and novel antipsychotics (e.g. Clozapine, Olanzapine, Risperidone) and others. Valproate is recognized by the American guidelines to be antimanic and can be used as a mood stabiliser. Gabapentin and Topiramate proved to be effective as an add on drug, especially in maintenance treatment. Lamotrigine showed favorable response in resistant bipolar depression.

Lithium

The antimanic effect of lithium is supported by recent evidence. Different authors estimate the range of efficacy of lithium in the treatment and prevention of bipolar disorder between 49-70%. The onset of action of lithium takes 5-21 days as a therapeutic medication and about 6 months as a preventive one. Predictors of lithium responsiveness include a diagnosis of classic mania, manic-depressive illness and fewer numbers of episodes. A drastic reduction of affective morbidity is very frequent in bipolar patients receiving lithium prophylaxis regularly for several years. Lithium does seem to be efficacious also in bipolar disorders with mood- incongruent psychotic features at least in the large majority of patients. It also seems to exert an antisuicidal effect in bipolar patients. Despite its powerful therapeutic and preventive effect, lithium use is hampered by its high side effect profile, which includes neurocognitive, renal, gastrointestinal, endocrinological side effects and weight gain. In addition there is an increased recurrence risk in the months following its discontinuation.

Novel Antipsychotics in Bipolar Disorder

Novel antipsychotics have been recommended both in the treatment of acute mania, depression with psychotic features and as maintenance treatment in recurrent bipolar disorder. Their advantages include a reduced risk of extra pyramidal side effects, reduced risk of Prolactin elevation and reduced risk of tardive dyskinesia. Novel antipsychotics used in the treatment of acute mania include Clozapine (open trials and many double-blind comparison trial), Risperidone (1 add-on comparison trial), Olanzapine (2 double-blind, placebo-controlled trials; 1 lithium comparison trial), Quetiapine (anecdotal reports) and Ziprasidone (preliminary evidence in bipolar schizoaffective patients).

State of the Art in the Management of Bipolar Disorder

I. Acute mania or mixed Episode

Treatment selection should depend on illness severity, associated features such as rapid cycling or psychoses, and where possible, patient preference. For patients not yet in treatment for bipolar disorder and who suffer severe mania or mixed episodes, treatment is best initiated with lithium in combination with an antipsychotic or valproate in combination with an antipsychotic. For less ill patients, monotherapy is recommended. Lithium, Valproate, or an antipsychotic may be sufficient. In the latter case atypical antipsychotics are preferred

over typical ones. Short-term adjunctive treatment with a benzodiazepine may also be helpful. For mixed episodes, valproate may be preferred over lithium. Alternatives include Carbamazepine or Oxcarbazepine, Ziprasidone or Quetiapine. Antidepressants should be tapered and discontinued if possible. For the best effect, pharmacotherapy should be coupled with psychosocial therapies.

"Breakthrough" manic or mixed episode while on maintenance treatment

In the case of a recurrence of a manic or mixed episode while on maintenance treatment, the medication dose should be optimized to achieve a higher serum level. Sometimes it is necessary to resume the use of an antipsychotic. If the breakthrough episode is not adequately controlled within 10 to 14 days of treatment with optimized doses of the first-line medication regimen, another first-line medication should be added. For example adding Carbamazepine or Oxcarbazepine in lieu of an additional first-line medication (Lithium, Valproate, antipsychotic drug) or changing from one antipsychotic to another. Clozapine may be particularly effective in refractory illness. Electro convulsive therapy (ECT) may also be considered for manic patients who are severely ill or whose mania is treatment resistant. For psychoses during a manic or mixed episode, patients should be prescribed an antipsychotic medication and ECT may also be considered.

II. Acute Depression

In the case of bipolar patients who are not yet in treatment for bipolar disorder, medication is best initiated by either lithium or Lamotrigine. Treatment initiation can also be done with both lithium and an antidepressant simultaneously. However, antidepressant monotherapy is not recommended. Again, ECT can be considered. Interpersonal therapy and cognitive behavior therapy may be useful when added to pharmacotherapy.

Breakthrough depressive episode while on maintenance treatment

Again, the medication dosage should be optimized in the case of a breakthrough depressive episode while on maintenance treatment of bipolar disorder. The dose adjustment should target a higher serum level of the drug, which however should be maintained in the therapeutic range. Psychotic features require an antipsychotic medication and ECT might be considered. If the patient fails to respond to optimized maintenance treatment, consider adding Lamotrigine, Bupropion, or Paroxetine. Alternative next steps include adding another newer antidepressant (e.g. another (SSRI) or venlafaxine) or a monoamine oxidase inhibitor (MAOI).

Tricyclic antidepressants may carry a greater risk of precipitating a switch.

III. Rapid Cycling

In the case of rapid cyclers one should start with an attempt to identify and treat medical conditions such as hypothyroidism or drug or alcohol use that may contribute to rapid cycling. If possible, medications (particularly antidepressants) that may contribute to cycling should be tapered. For initial treatment, lithium or valproate should be used. An alternative treatment is Lamotrigine. For many patients, combinations of medications are required. This may be a combination of two of the agents mentioned above or one of them in addition to an antipsychotic.

IV. Maintenance Treatment

The decision on maintenance treatment and even more the tailoring of maintenance treatment is one of the biggest challenges in the fields of psychiatry. Long-term maintenance treatment in bipolar disorder cases should be considered once the patient develops more than two episodes in the first two years of the history of his/her illness.

Although maintenance therapy with atypical antipsychotics may be considered, there is as yet no definitive evidence that their efficacy in maintenance treatment is comparable to that of other agents discussed above. The rule is that antipsychotic medications should be discontinued unless they are needed for control of persistent psychoses or prevention of recurrence of mood episodes. If a patient fails to respond, i.e. continues to experience subthreshold symptoms or breakthrough mood episodes, another maintenance medication could be tried such as an atypical antipsychotic, or an antidepressant. There are insufficient data to support one combination over another. Maintenance ECT may also be considered for patients who respond to ECT during an acute episode.

Psychosocial intervention

Concomitant psychosocial interventions addressing illness management (i.e. adherence, lifestyle changes, and early detection of prodromal symptoms) and interpersonal difficulties are likely to be of benefit. Supportive and psychodynamic psychotherapies are widely used in combination with medication. Group psychotherapy and family therapy may also help. Support groups are helpful in providing participants with useful information.

Finally, we need to encourage a research agenda on bipolar disorder that goes beyond our current ways of thinking to a dimensional approach addressing issues of under diagnosis and misdiagnosis. More emphasis should be made on the bipolar spectrum and more diagnostic attention given to manic criteria.

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LES TROUBLES PSYCHIQUES PÉRIMENSTRUELS : SPM & TDPM

Revue de Littérature Multidisciplinaire

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- Dispose-t-on de **concepts nosographiques** permettant d'encadrer de façon valable les troubles périmenstruels de la femme. ?

Quelle sont les outils fiables pour assurer le **diagnostic** et l'**évaluation** clinique du SPM ?

Quels sont l'**épidémiologie** et l'**impact psychosocial** des différentes formes des troubles prémenstruels de la femme ?

Peut-on formuler des hypothèses structurées concernant les rapports qui pourraient s'établir entre les cycles biologiques, les facteurs de stress et l'agencement des réactions adaptatives chez une femme donnée ?

Faut-il **traiter** le SPM? Et si la réponse est oui, dispose-t-on d'un traitement efficace -?

Notre propos rejoint finalement l'objectif d'établir s'il est justifié et faisable de porter plus d'attention à la **clinique des SPM**, ainsi qu'à l'**investigation gynécologique** et **psychiatrique** des troubles présentés par les femmes qui souffrent de malaise périodique prémenstruel.

Le SPM fait l'objet d'un **regain d'intérêt** car son **étiologie** n'est pas connue, sa **physiopathologie** et sa **psychopathologie** restent très incertaine et les nombreux **traitements** proposés sont contestés. Il paraît donc utile, au vu des publications récentes, de refaire le point sur cette pathologie **très fréquente**. (55)

Mots Clés : Dysphorie, femmes, menstruation, périmenstruel, prémenstruel, SPM

INTRODUCTION

Le **cycle menstruel**, un événement qui ponctue les vies de la plupart des femmes, il peut être associé à divers changements **comportementaux**, **psychologiques**, et **somatique**. Il joue un rôle significatif dans l'équilibre physique et/ou psychique de la femme. Inversement, la cyclicité menstruelle peut être aisément perturbée par toutes affections physiques et/ou psychologique. (4)

Les changements comportementaux, émotionnels et physiques associés au cycle menstruel ont été observés à travers l'histoire. Depuis **1931**, date où le **premier syndrome prémenstruel** était décrit dans la littérature médicale, il a été une source de controverse dans la communauté médicale, certains médecins refusent d'accepter son existence « autre que dans l'esprit de la femme » (Wiches 1988). De même cette Controverse existe encore autour d'une **définition universellement** reconnue de ce concept ; ceci est en rapport avec plusieurs facteurs : la **cause** du SPM qui demeure inconnue ; la **diversité** et la **sévérité** de ces symptômes à travers les femmes et pour la même femme d'un cycle à un autre

Le syndrome prémenstruel (SPM) représente probablement la **gêne fonctionnelle** le plus fréquemment alléguée par la femme. Il s'agit d'un ensemble de manifestations **bénignes** pouvant intéresser tous les appareils, et dont le seul point commun est leur **caractère cyclique**, apparaissant dans les jours qui précèdent les règles pour disparaître au début ou au cours de la menstruation. La **fréquence** de cette pathologie est évidemment difficile à apprécier avec précision, du fait de son caractère essentiellement **subjectif** et il est impossible de déterminer sûrement à partir de quel degré un molimen cataménial banal devient un syndrome prémenstruel ; cette fréquence est en général évalué autour de 35 à 40 % des femmes. Le molimen cataménial est observé dans 80% des cas, mais au cours de la consultation, 30 à 40% des patients signalent une gêne et 3 à 8% environ des femmes en âge de procréer présentent un **SPM sévère**. Pour affirmer le SPM, le degré de modification des symptômes doit être apprécié au cours de la phase Lutéale.

Durant le cycle menstruel 30 % des femmes entre 18 et 45

ans avaient des **changements d'humeur** qualifiés de modérés à sévères, sur lesquelles environ 5 à 10% demandaient un traitement pour leur trouble thymique. 25% des femmes n'avaient aucun changement d'humeur et 3% souffraient de troubles sévères.

Pour **coter** cette sévérité, certains auteurs (15) préconisent de compter le nombre de jours de la phase lutéale sans symptômes et d'en soustraire le nombre de jours avec symptômes maximaux.

Les patientes souffrant de troubles de l'humeur liés aux cycles menstruels, souffrent **du point de vue psychiatrique** de **troubles Thymique cycliques atypiques**. En effet, ces troubles, la plus part du temps dépressif, débutent rapidement après l'ovulation, augmentant en sévérité pour atteindre un maximum pendant les 5 jours précédents les règles et disparaissent ensuite rapidement dès l'arrivée de ces dernières. Les troubles de l'humeur peuvent varier d'un cycle à l'autre chez un même individu.

La documentation exacte des symptômes se fait sur un **calendrier menstruel** permettant l'identification des femmes ayant des modifications cycliques de leurs troubles. La majorité de ces troubles survient pendant la phase lutéale du cycle menstruel.

LE CYCLE MENSTRUEL

La cyclicité menstruel normal nécessite la coordination de : l'**hypothalamus**, la **glande de pituitaire**, et les **ovaires**. La libération du **gonadotrophine-releasing hormone (GnRH)** se fait dans une mode pulsatile par l'hypothalamus. Sa sécrétion est modulée par une variété de neurotransmetteurs, incluant le norepinephrine, la sérotonine, et les opioïdes endogène. La gonadotrophine - releasing hormone stimule la libération du **follicule-stimulating hormone (FSH)** et du **luteinizing hormone (LH)** de glande pituitaire antérieure. (Anté-hypophyse) (5)

La **phase folliculaire**, ou le temps de développement d'ovocyte avant l'ovulation, est marquée par la croissance progressive d'un **follicule d'ovarien**. Cette phase est caractérisée par la **sécrétion d'œstrogène**,

cette sécrétion est graduelle au début, puis devient exponentielle dans les 5 à 6 jours conduisant jusqu'à l'ovulation. Une baisse transitoire et brusque du niveau d'œstrogène coïncident avec l'ovulation. L'élévation du niveau d'œstrogène au cours de la phase Lutéale résulte de la production par le corps Lutéal de l'œstrogène et de progestérone.

Le lifespan du corps Lutéal est évalué approximativement à 12 jours. Si l'ovule n'est fécondé, le corps Lutéal subit une involution, les niveaux d'œstrogène et de progestérone chutent dramatiquement et la menstruation survient. La diminution des hormones ovariennes stimule par rétroaction négative l'hypothalamus et la glande de pituitaire, et ainsi commence un nouveau cycle de stimulation d'ovarien. (5)

▪ DEFINITION ET ASPECT CLINIQUE

Le syndrome Prémenstruel (SPM) regroupe une large variété de symptômes **comportementaux, psychologiques ou physiques** qui peuvent être éprouvés par des femmes pendant la **phase de Lutéale** du cycle menstruel. Les symptômes surviennent 7 à 14 jours avant le début des règles et disparaît de le 1er ou le 2ème jours de la menstruation. Un facteur nécessaire dans le SPM est l'absence de symptôme dans la phase folliculaire du cycle menstruel. L'exacerbation de ces signes perturbe l'activité personnelle, professionnelle et familiale.

Les symptômes les plus communément rapportés du SPM sont :

Les symptômes Physiques

Fatigue, tension mammaire, rétention hydrosodée, ballonnement abdominal, prise du poids, Acné, mal de tête et Constipation.

Les symptômes Psychologiques

Anxiété, colère, dépression, irritabilité, diminution de l'aptitude de concentration, modification de la libido, hypersensibilité au rejet, modification de l'appétit (cravings pour les sucreries) Plusieurs études posent la question de savoir s'il faut classer le SPM en tant que trouble psychiatrique, la question n'est pas réglée à ce stade. Les cas sévères sont actuellement l'objet d'étude international. En 1985, le groupe de travail américain sur les maladies mentales a précisé le syndrome prémenstruel ou "**trouble dysphorique de la phase lutéale tardive**" : **TDPLT** (late luteal phase dysphoric disorder : **LLPDD**) et des questionnaires ont pu être établis.

Actuellement l'**authenticité de ce syndrome**, compte tenu des méthodes récentes de diagnostics, ne fait plus de doute, mais il est nécessaire d'établir la variation cyclique des symptômes sur des critères statistiques. Le caractère cyclique des symptômes est fondamental. Ils apparaissent dans la dernière semaine de la phase lutéale et disparaissent la première semaine des règles (au moins de J 4 à J 12)

On voit alors émerger la notion de syndrome dysphorique prémenstruel, c'est-à-dire la notion d'un syndrome prémenstruel caractérisé par la simultanéité d'un désordre affectif et d'un trouble menstruel et ayant donc une périodicité caractéristique. Rappelons que le **DSM-IV** décrit, une **forme spécifique de SPM**. Cette catégorie diagnostique met au premier plan: L'importance de la périodicité du trouble (pendant la dernière semaine de la phase lutéale et disparition de ces symptômes quelques jours après le début de la phase folliculaire), qui se présente sous forme d'un éventail de symptômes hautement corrélés et dont un nombre minimum doit être présent sans qu'il soit "**une exacerbation prémenstruelle**" d'un autre trouble psychiatrique (peut être surajoutée à une dépression majeure, un trouble panique, une dysthymie ou un trouble de la personnalité) et présentant un

handicap dans les activités sociales pendant la période prémenstruelle. Dans son travail de 1989, *Spitzer (73)* souligne l'importance de la chronologie du syndrome, confirmée prospectivement, pour classer ce dernier comme une exacerbation ou un désordre autonome. Mais il est déjà apparu qu'il n'est pas facile d'établir ce diagnostic différentiel. La phase prémenstruelle couvre en effet le 25% du cycle et il n'est donc pas facile de distinguer le **TDPLT** d'un cas de **dépression brève récurrente**, indépendante du SPM, à moins que cette dernière ne se manifeste toujours à une autre période du cycle. D'autre part, il est difficile d'évaluer l'importance des troubles de l'humeur secondaires à la menstruation (*Ainscough, 1990; Abraham et al., 1990*) (2,3). En effet, il est possible que des conditionnements culturels, social et familiale influencent les modes de représentation de cette dernière.

Le **DSM-IV** définit le **syndrome dysphorique de la phase lutéale tardive** en terme de **symptôme psychiatrique individuel** (trouble de l'humeur, anxiété, etc.), ainsi qu'en terme de **problèmes sociaux, professionnels ou relationnels**. On exclut, la simple exacerbation d'un autre trouble psychiatrique. Par ailleurs, l'**élément cyclique** lié aux menstruations est clairement défini dans le point D.

Certains auteurs (25) notent que les patientes souffrant de **trouble bipolaire à cycle rapide** ont une tendance augmentée à faire des **SPM plus sévères**. De plus, ils concluent que toutes les études semblent d'accord pour définir **deux groupes** différents de patientes souffrant de SPM.

-Le **«SPM pur»** : représente les femmes qui n'ont de symptômes que durant la phase lutéale et ne montrent pas d'affection psychiatrique plus importante que le groupe contrôle dans leur passé ou actuellement. Elles n'ont pas non plus de trouble de personnalité aux tests appropriés.

-Le **«SPM exacerbé»** : représente les femmes ayant des symptômes durant tout le cycle avec majoration dans la phase lutéale, elles ont aussi plus fréquemment des antécédents de troubles psychiatriques et leurs tests de personnalité dévient de la norme.

A noter qu'un **terrain familial** (mère, sœur) est retrouvé dans 45% des cas et **des antécédents de dysménorrhée** dans 50% des cas (6, 9). Par ailleurs il n'y a pas de différence concernant les paramètres suivants : niveau socio-économique, appartenance ethnique, parité, durée de cycle et poids (43). Les nombreux signes observés peuvent faire l'objet d'une évaluation plus objective au moyen de **questionnaire avec modèle mathématique d'évaluation**; le **« COPE »** (calendar of Premenstrual Experiences) comporte 22 items chiffrés de 0 à 4. Il a été testé à *San Diego Californie*.

Les **troubles dysphorique Prémenstruel : TDP (PDD)** est une forme sévère de SPM. Les critères diagnostiques ont été définis par l'association américaine de psychiatrie dans le «manuel de diagnostic et de Statistique des troubles Mentaux, 4th éditions »(**DSM IV**) sont résumées dans La **table suivant** :

A -Au cours de la plupart des cycles menstruels de l'année écoulée, cinq ou plus des symptômes suivants ont été présents la plupart du temps lors de la dernière semaine de la phase lutéale. Ils se sont améliorés au cours des premiers jours de la phase folliculaire et sont demeurés absents pendant la première semaine après les règles. L'un des symptômes doit être : (19,46,32,14) :

1. Humeur dépressive marquée, sentiments de désespoir ou auto-dépréciation (idées de dévalorisation).
2. Anxiété marquée, tensions, impression d'être noué, tendu, nerveux.
3. Labilité émotionnelle marquée (p.ex., brusque sentiment de tristesse, envie de pleurer, hypersensibilité au rejet).

4. Colère ou irritabilité marquée et persistante ou augmentation des conflits interpersonnels.
5. Diminution de l'intérêt pour les activités habituelles (p.ex., travail, école, amis, loisirs).
6. Difficultés subjectives à se concentrer.
7. Léthargie, fatigabilité excessive ou perte d'énergie marquée.
8. Modifications marquées de l'appétit, hyperphagie, envie impérieuse de certains aliments.
9. Hypersomnie ou insomnie.
10. Sentiment d'être débordé ou de perte de contrôle.
11. Autres symptômes physiques tel que tension ou gonflement des seins, céphalées, douleurs articulaires ou musculaires, impression d'« enfler », prise de poids.

N.B.: Au cours du cycle menstruel, la phase lutéale correspond à la période comprise entre l'ovulation et le début des règles, la phase folliculaire débute avec les règles. Chez les femmes non réglées (p.ex., en cas d'hystérectomie), la datation des phases lutéale et folliculaire peut nécessiter le dosage des hormones sexuelles circulantes.

B -La perturbation interfère nettement avec le travail ou l'activité scolaire, les activités sociales habituelles et les relations avec les autres (p.ex., évitement des activités sociales, diminution de la productivité ou de l'efficacité au travail ou à l'école). Ce critère est indispensable pour le diagnostic et fait appel à une confirmation par auto-évaluation prospective pendant au moins deux cycles.

C -La perturbation ne correspond pas seulement à l'exacerbation des symptômes d'un autre trouble comme un Trouble dépressif majeur, un Trouble panique, un Trouble dysthymique ou un Trouble de la Personnalité (bien qu'elle puisse se surajouter à chacun de ces troubles).

D -Des évaluations quotidiennes prospectives réalisées pendant au moins deux cycles symptomatiques consécutifs doivent confirmer la présence des critères A, B et C (avant cette confirmation, le diagnostic peut être porté à titre provisoire).

Il est important de souligner Certains faits des **Conséquences du SPM Sur le plan personnel et professionnelles:**

Perturbations de la vie affective ;

Diminution des performances physiques et sportives, augmentation de la fatigabilité ;

Diminution de la rentabilité ;

Augmentation des épisodes dépressifs.

des arrêts de travail et un absentéisme épisodique fréquent

▪ **DIAGNOSTIC ET CLASSIFICATION**

L'interrogatoire est le temps fondamental du diagnostic de ce syndrome essentiellement subjectif. Il faut préciser **l'apparition prémenstruelle** des troubles, et **leur disparition dès la survenue des règles** et apprécier la **date d'installation** des symptômes ainsi que les éventuelles **circonstances déclenchantes**, ou un **contexte psycho-émotionnel**. Il faut faire une évaluation de l'ensemble des troubles avec véritable **calendrier d'autoévaluation quotidienne**, qui peut par ailleurs être repéré sur la **courbe thermique**. Il permet enfin d'établir un **climat relationnel** qui sera fondamental dans la thérapeutique ultérieure.

L'examen clinique, si possible au cours de la période prémenstruelle, est la plupart du temps **strictement normal**, il doit cependant toujours être pratiqué à la recherche de

facteurs favorisants locaux.

La **courbe pondérale** notamment avec mesure quotidienne du poids au cours de la période prémenstruelle, peut objectiver par ses fluctuations d'un jour à l'autre la réalité de la pathologie et son évolution.

Le diagnostic du SPM se fait habituellement par des symptômes survenus pendant la phase Lutéale du cycle menstruel. Les **questionnaires de symptôme** sont souvent employés comme aide au diagnostic, en plus il est souhaitable de garder un **agenda menstruel de symptôme**, afin de documenter l'évolution et clarifier davantage le modèle de symptôme.

Dans une tentative de bien délimiter les **formes cliniques** du SPM, plusieurs d'experts ont élaboré des systèmes de classification qui identifient les patients ayant un SPM en sous-groupes. Nous évoquant la classification qui partage le SPM en **quatre sous-groupes distincts**. Chaque sous-groupe est relié aux symptômes spécifiques.

Le SPM type A (A = anxiety)

Le SPM **type A** est la catégorie de symptôme la plus commune et il est généralement fortement associé avec un excès d'œstrogène et à un déficit de progestérone pendant la phase prémenstruelle. Les symptômes communs de cette catégorie sont : l'anxiété, l'irritabilité, et l'instabilité émotionnelle

Le SPM type C (C = carbohydrate craving)

Le SPM **type C** est généralement associé à des troubles du conduit alimentaire (appétit excessif, appétit insatiable pour des bonbons), des maux de tête, une fatigabilité, des périodes d'évanouissement et des palpitations. Les tests de tolérance de glucose (Glucose tolerance tests "GTT") pratiqué chez les patients appartenant au SPM **type C** pendant les cinq à dix jours avant leurs règles, montrent un aplatissement de la partie initial de la courbe (qui implique habituellement une sécrétion excessive d'insuline en réponse à consommation importante du sucre), tandis que pendant les autres parties du cycle menstruel, leur GTT est normal. Actuellement, il y a aucune explication claire pour ce phénomène, bien que l'augmentation de capacité cellulaire à lier l'insuline ait été postulée. Cette augmentations de la capacité de fixation de l'insuline paraît être en rapport avec la régulation hormonale, mais d'autres facteurs peuvent être aussi impliqués tels qu'une prise excessive de sel ou une diminution des niveaux de prostaglandines ou de magnésium.

Le SPM type D (D = depression)

Ce type est mal connu et il est relativement rare dans sa forme pure. Son symptôme clé est la **dépression** qui est habituellement associée à des niveaux assez bas des neurotransmetteurs dans le système nerveux central. Pour les patientes présentant un SPM **type D**, il y a fort probable une chute importante des neurotransmetteurs par suite d'une diminution importante du niveau d'œstrogène (par contraste au SPM **type A** qui montre des résultats opposés). La diminution d'œstrogène ovarien a été attribuée au stress qui induit une augmentation de la sécrétion d'adrenaline androgène et/ou de progestérone.

Le SPM type H (H = hyperhydration)

Il est caractérisé par une prise de poids (+ 1.5 kg), un ballonnement abdominal avec lourdeur pelvienne, une tension mammaire avec congestion et, et parfois un gonflement occasionnel du visage, des mains et des chevilles. Ces symptômes sont dus à un accroissement du volume des fluides secondaires à un excès d'aldostérone qui provoque une augmentation de la rétention hydrosodé. L'excès d'aldostérone pendant la phase prémenstruelle chez les patientes ayant un SPM **type H** peuvent être provoqué par le stress, l'excès

d'œstrogène, le déficit en magnésium ou secondaire a un excès de sel.

▪ LES INSTRUMENTS D'ÉVALUATION DU SPM

Le diagnostic de SPM est peu fiable en raison:

1. A) du **caractère changeant et aspécifique** des manifestations cliniques qui caractérisent cette affection

B) du **manque de fiabilité de l'évaluation subjective rétrospective** des troubles prémenstruels.

Un effort considérable de recherche a porté, de ce fait, sur la mise au point d'**instruments d'évaluation clinique (questionnaires)** et de **critères diagnostiques standardisés** afin de résoudre ces difficultés.

De façon schématique, on peut distinguer deux groupes de questionnaires:

2. Les **questionnaires rétrospectifs**: Les instruments destinés à une évaluation rétrospective (Menstrual Symptom Questionnaire, **MSQ**; Premenstrual Assessment Form, **PAF**) tendent à être hyperinclusifs: la quantité des troubles est en général sur-estimée, le jugement de la patiente étant perturbé par l'inévitable transformation du souvenir.

Les **questionnaires prospectifs**: les outils prospectifs, tels le «Menstrual Distress Questionnaire» (**MDQ**) ou les **calendriers d'auto-évaluation** que la femme doit remplir tous les jours du cycle pendant un ou plusieurs mois. Cette méthode est beaucoup plus fiable, mais elle a aussi des inconvénients dont il faut tenir compte (compliance limitée, effet placebo pouvant induire des faux positifs). (1)

LE QUESTIONNAIRE D'ÉVALUATION DU SPM :

Pour ce questionnaire chaque symptôme est apprécié par l'un des chiffres suivant : 0, 1, 2, 3.

'0' Absence de symptôme

'1' Symptômes présents mais discrets

'2' Symptômes Inhibent les activités quotidiennes

'3' Symptômes modifient le rythme de vie (41)

▪ EVALUATION DES SYMPTOMES EMOTIONNELS PRE – MENSTRUELLE :

Degrés des symptômes	Absents	Discrets	Inhibent les activités	Modifient le rythme de vie
Nature des symptômes				
accès soudain de tristesse, d'irritabilité ou de colère	0	1	2	3
Anxiété ou tension importante	0	1	2	3
Diminution d'intérêt pour les occupations habituelles	0	1	2	3
Manque de confiance, étourdissement	0	1	2	3
Humeur dépressive importante	0	1	2	3
Mauvaise relation avec le corps	0	1	2	3
Sentiment de culpabilité	0	1	2	3
Sentiment de désespoir, crise de larmes	0	1	2	3
Hyperémotivité, Impression d'être "sur les nerfs"	0	1	2	3
Désir de solitude	0	1	2	3
Dévalorisation de soi, autodépréciation	0	1	2	3
Labilité de l'humeur, labilité émotionnelle	0	1	2	3
Impression de modification de la personnalité	0	1	2	3
Accès d'explosion violente	0	1	2	3
Colère ou irritabilité importante et persistante	0	1	2	3

Sentiment de stress à l'extérieur	0	1	2	3
Méfiance, sentiment d'insécurité	0	1	2	3
LE TOTAL	E1	E2	E3	E4
SCORE DES SYMPTOMES EMOTIONNELS : SE = E1 + E2 + E3 + E4				

▪ EVALUATION DES SYMPTOMES PHYSIQUES PRE – MENSTRUELLE :

Degrés des symptômes	Absents	Discrets	Inhibent les activités	Modifient le rythme de vie
Nature des symptômes				
Ballonnement abdominal	0	1	2	3
Difficultés à se concentrer sans trouble intellectuel	0	1	2	3
Mal de dos, douleurs musculaires ou articulaires	0	1	2	3
Modification de l'appétit (food cravings, binge eating)	0	1	2	3
hypersensibilité ou gonflement mammaire	0	1	2	3
Constipation	0	1	2	3
Œdème (chevilles gonflées, mains, etc.)	0	1	2	3
Evanouissement, vertige	0	1	2	3
Fatigabilité ou perte importante d'énergie	0	1	2	3
Malaise corporelle général	0	1	2	3
céphalées	0	1	2	3
Hypersomnie ou insomnie	0	1	2	3
Nausée	0	1	2	3
prise de poids	0	1	2	3
LE TOTAL	P1	P2	P3	P4
SCORE DES SYMPTOMES PHYSIQUES : SP = P1 + P2 + P3 + P4				
SCORE TOTAL = SCORE EMOTIONNEL + SCORE PHYSIQUE				
S TOTAL = SP + SE				

Δg positif du SPM si S TOTAL Sup. 20 pts

Le diagnostic du SPM est fort probable pour tout score total (S TOTAL) supérieur à 20 points.

LES EXAMENS COMPLEMENTAIRES

Il est nécessaire d'évaluer prospectivement les critères diagnostiques:

courbe ménothermique et calendriers des symptômes (fonctionnels et poids) sont essentiels. Nous demandons aux femmes de noter quotidiennement sur 3 cycles les symptômes du SPM. Le score quotidien est établi et la variation cyclique facile à évaluer.

On peut de plus demander : un **bilan sanguin complet** avec un **ionogramme**; des **dosages hormonaux** :

Hormones thyroïdiennes (T3-T4- TSH) ; Hormones **stéroïdiennes** (progestérone, œstrogène) et le niveau de prolactine et ce au cours du **21ème jour** du cycle.

S'il n'y a pas d'anomalies apparentes -il n'est pas recommandé de refaire le bilan biologique (dosages hormonaux de base peu contributifs, dosages cinétiques et étude de pulsatilité difficiles à interpréter et coûteux) ;

Une **capillaroscopie** comparatives phases du cycle. (17)

En fait, aucun examen complémentaire ou paramètre biochimique ne peut caractériser le SPM.

▪ EPIDEMIOLOGIE DU SPM

Les études épidémiologiques ont été relativement nombreuses et ont porté sur :

1. la détermination de la prévalence
2. L'étude des associations cliniques du SPM.

Les recherches effectuées ont donné, cependant, des résultats fort discordants en raison de problèmes méthodologiques qui étaient en rapport avec :

1. La définition diagnostique du syndrome.
2. L'appréciation des symptômes.
3. Le type de population étudiée.

Des travaux plus récents ont permis d'estimer que 20 à 90 % des femmes éprouve certains symptômes prémenstruels (Hsia et Long 1990), alors que ces symptômes sont sévères chez de 5 à 10 % des femmes (Mortola 1992) (50).

- 3 à 10% de la population féminine en âge de procréer présente aucun trouble prémenstruel.

- 50% des femmes présenteraient un malaise d'intensité modérée

3. 35% des femmes présenteraient des symptômes qui entraîneraient un certain degré de perturbation de la vie sociale, professionnelle ou familiale.

4. Enfin 5 à 10% des femmes souffriraient d'un syndrome prémenstruel sévère entraînant une perturbation grave de leur vie.

Le SPM survient chez les des femmes à partir de l'âge du début des règles jusqu'à l'âge de la ménopause, cependant plusieurs études montrent que c'est à partir de la **trentaine** que les femmes commencent généralement à chercher un traitement pour LE SPM. L'explication de cette **incidence élevée** pour cette tranche d'âge n'est pas encore connu ; est-ce que vraiment l'incidence du SPM augmente durant cette tranche d'âge ou est-ce que la sensibilité aux symptômes du SPM devient plus exagérée ou tout simplement est-ce que cette **incidence** élevée ne reflète que le résultat d'une accessibilité plus facile aux soins médicaux (Hsia et Long 1990) (78).

Certaines études ont montré une incidence élevée du SPM a la suite de l'accouchement, (mais la littérature est encore controversée sur ce sujet) (Porth 1994) (77). Cependant d'autres facteurs paraissent n'avoir aucune corrélation avec Le SPM comme : le statut Conjugal, l'éducation lors de l'enfance, l'appartenance raciale et ethnique, la culture et le statut socio-économique, (Nader 1991) (52). De même aucune association conséquente n'a été trouvée entre Le SPM et les variables démographiques ou diététiques, l'activité physique et professionnelle, le niveau Psycho-social, le stress, les caractéristiques menstruelles de cycle, le soutien du partenaire et les traits de caractères de la personnalité (Pearlstein 1995) (58).

L'étude des **relations entre SPM et troubles psychiatriques** a commencé à se développer vers 1960. Coppen et Kessel (1963) (18) ont :

1. Confirmé l'importance des associations entre troubles psychiatriques et malaises péri menstruel
2. Montré que la prévalence des troubles menstruels est nettement augmentée chez les femmes ayant reçu un diagnostic de "névrose" et, dans une moindre mesure, chez des patientes présentant des troubles anxieux et dépressifs, alors qu'elle était inchangée ou diminuée chez les schizophrènes (exclusion faite pour les femmes souffrant

simultanément de dépression). Par rapport aux autres groupes diagnostics, les femmes souffrant de "névrose" présentent plus d'irritabilité, de tension, de nervosité et de dépression, ont plus de difficultés sexuelles et consultent plus fréquemment le médecin pendant la période prémenstruelle.

Les Auteurs suggèrent que le SPM puisse se manifester chez des sujets ayant une tendance à développer des **états de "tension"**, tendance qui résulterait elle-même d'une **"anormalité constitutionnelle"**, pouvant entraîner à la fois des altérations menstruelles et des troubles de la personnalité. (1)

▪ PHYSIOPATHOLOGIE ET PSYCHOPATHOLOGIE DU SPM

Le SPM est lié aux modifications cycliques **neurodiencéphalo-hypothalamo hypophysoovariennes**. Il apparaît après la puberté, **disparaissent** lors des cycles anovulatoires, avec l'ovariectomie, avec la ménopause non traitée, et avec le traitement par les agonistes du GnRH. Plusieurs théories tentent d'expliquer les bases physiopathologiques et psychopathologiques de ce syndrome.

1) THEORIES BIOLOGIQUE

Perturbation des hormones stéroïdien ovarien

Du fait que les symptômes du SPM évoluent d'une façon périodique selon la cyclicité des phases menstruelles, plusieurs études concernant son étiologie ont été portées sur le rôle des hormones stéroïdien ovarien vu que :

3. Les femmes qui ont subi une hystérectomie sans ovariectomie, peuvent présenter des symptômes cycliques qui ressemblent au SPM ;

4. Les symptômes du SPM sont rares chez les femmes en période post-ménopausique (Porth 1994) (59).

Le rôle de ces hormones ovarien est scientifiquement confirmé par Muse et al. (1984) qui montre bien que l'induction d'une **"ovariectomie médicale"** en utilisant les agonistes des gonadotropine realising 'hormone (GnRH) provoque une résolution spectaculaire des symptômes du SPM (Mortola 1992) (50)?

Comme la **progestérone** est l'**hormone prédominante pendant la phase Lutéale** du cycle menstruel, et comme le SPM survient, par définition, dans la phase Lutéale, en a posé l'**hypothèse** que le SPM est causé par un **manque de progestérone** (Mèches 1988) (48). Cependant, les niveaux bas de progestérone n'ont pas été confirmés dans les études, alors que bien d'autres études contrôlées utilisant le substitut de progestérone comme thérapie pour les symptômes du SPM, ont échoué de trouver aucun avantage de progestérone versus placebo (Muse 1991) (51). De même cette théorie d'**hyperfolliculinie "relative"** ou **insuffisance lutéale** (la progestérone étant sécrétée par le corps jaune en quantité insuffisante pour s'opposer à l'effet périphérique des œstrogènes en deuxième partie de cycle) ; n'est pas assez valide du fait que plusieurs patientes, ayant des insuffisances lutéales responsables de stérilité ou d'avortements à répétition, ne présentent pas de syndrome prémenstruel. De plus, cette théorie ne peut pas tout expliquer, notamment la persistance de la symptomatologie après la ménopause ou la castration.

Une autre étude récente était conduite par Schmidt et al. (1991) (68), défiait l'hypothèse que la cause immédiate des symptômes du SPM est en rapport avec un niveaux bas de progestérone pendant la phase Lutéale. Dans cette étude, les différentes phases du cycle menstruelle sont annulées chez les femmes ayant un SPM confirmé par l'administration d'un antagoniste de progestérone « **le mifepristone** » (RU 486), en association tantôt avec l'hormone HCG (human chronic gonadotropin) tantôt avec un placebo, sept jours après le

déferlement de LH.

Le **mifépristone** en tant que **bloquant des récepteurs de progestérone** entraîne une diminution rapide de progestérone plasmatique et provoque l'apparition des règles dans 48 à 72 heures. Les sujets qui recevaient le mifépristone avec placebo entraient dans la phase folliculaire juste après l'apparition des règles (qui sont induites par le mifépristone), alors que ceux recevant le mifépristone avec l'HCG avaient des règles relativement normales. Le résultat significatif de cette étude, était que le groupe recevant le mifépristone avec le placebo continuait à éprouver des symptômes psychologiques du SPM à la phase folliculaire malgré la restauration de leur cycle menstruel. Les auteurs interprétaient ces résultats en indiquant que les symptômes du SPM ne résultent pas uniquement de manifestation hormonale précédant la phase Lutéale tardive mais le SPM représente en fait **un trouble cyclique de l'humeur** qui devient synchronisé avec le cycle menstruel (Schmidt et al. 1991) (68).

Dans une tentative de trouver des variations dans les niveaux des hormones ovariennes (ou des variations du rapport **œstrogène / progestérone**) entre des femmes ayant un SPM et les femmes asymptomatiques ; les résultats ont échoué de trouver des résultats conséquents. Mortola (1992) (50) suggère qu'il puisse y avoir **une prédisposition biologique** chez certaines femmes à **être plus susceptible aux niveaux d'hormone ovarienne** à travers le cycle menstruel. Cette **prédisposition** peut être **génétique** ou **réglée écologiquement**

Sur le plan hydro-électrolytique ; Les œstrogènes provoquent **une rétention hydrosodée** en favorisant la perméabilité vasculaire et les progestérone possèdent un **effet natriurétique** en diminuant la liaison de l'aldostérone à son récepteur.

En plus de l'action antiminéralocorticoïde, la progestérone a des **actions centrales sédatives**. Les substances qui activent le **récepteur GABA type A** ont des actions anxiolytiques. Robel P (InU33) a souligné qu'un métabolite de la progestérone, la **tétrahydroprogestérone (THP)**, en intervenant sur le récepteur de l'acide gammaaminobutyrique de type A, potentialiser la transmission GABA ergique. La THP a une influence sur les troubles de l'humeur accompagnant le syndrome prémenstruel, le post-partum et la ménopause. Plusieurs études ont suggéré la présence d'un **taux bas de progestérone plasmatique** dans le SPM (12). Ces résultats n'ont jamais été confirmés ; des études randomisées comparant l'effet de la prescription de progestérone au placebo (lors du SPM) n'ont pas objectivé de différence. L'administration de **mifépristone** ne réduit pas le syndrome prémenstruel alors qu'il induit des règles et un profil hormonal de phase folliculaire. Les autres **dosages hormonaux comparatifs** (chez les femmes ayant un SPM et les témoins) n'ont pas montré de différence en ce qui concerne le taux des **stéroïdes sexuels**, des gonadotropines et les profils de sécrétion ne sont pas perturbés. Le dosage des métabolites de la progestérone (qui modifient l'action de l'acide gamma-aminobutyrique) ne semble pas perturbé. Les études concernant la **pulsatilité** sont contradictoires.

L'origine **neuroendocrinienne** du SPM est fortement évoquée. La diminution en **bêta-endorphine** du système nerveux central par l'intermédiaire des **neurones du GnRH** pourrait être responsable de la perte de pulsatilité de LH et de progestérone. (55, 45, 33)

- **Perturbation du taux de prolactine** :

Une augmentation du taux de prolactine a été généralement

associée au SPM vu les constatations suivantes :

-1 Son effet direct sur les glandes mammaires qui peut être responsable du symptôme de tension du sein commune dans Le SPM;

-2 Son rapport indirect avec la libération et le métabolisme de la dopamine lors des manifestations de stress

-3 Son effet de rétention d'eau, du sodium, et du potassium.

La prescription de **bromocriptine** (inhibiteur de la libération de prolactine), a montré une réduction de la douleur cyclique du sein, sans avoir aucune amélioration sur les autres symptômes du SPM (O'Brien 1985) (53). Cela suggère bien que seul un excès de prolactine ne puisse être l'unique cause du SPM mais il peut être associé avec d'autres facteurs (45)

Perturbation des neurotransmetteurs

Le rôle actuellement attribué aux neurotransmetteurs est croissant, une baisse du taux plaquettaire de la **sérotonine** et de la **monoamine oxydase** est constatée dans le SPM, ainsi le rôle de la sérotonine est fortement évoqué dans le SPM. Les déficiences du système sérotoninergique sont associées à la dépression, à la sous estimation du moi, à l'anxiété, l'agressivité, les troubles de l'appétit, symptômes observés classiquement dans le SPM ;

Des anomalies au niveau des neurotransmetteurs en particulier la sérotonine ; ont été constatées durant la phase Lutéale chez les femmes souffrant de SPM, et ont été responsables des troubles thymique et anxieux fréquemment retrouvés. Les hormones **stéroïdes ovariens** peuvent avoir un **effet direct** sur la **synthèse**, la **libération**, la **recapture** et de certains neurotransmetteurs, et un **effet indirect** par l'inactivation enzymatique, et par la modification de la sensibilité de certains récepteurs post-synaptique et pré-synaptique. Le mécanisme de cette interaction n'est encore bien élucidé (Pearlstein 1985).

Une étude récente a montré que l'activité de la sérotonine est altérée chez les femmes ayant des TDP. Les taux de sérotonine plasmatique et plaquettaire sont diminués pendant la phase de Lutéale du cycle menstruel (Mortola 1992) (50).

Steiner et al. (1995) (75-74) montrer que l'administration continue du **fluoxétine**, un antidépresseur qui **inhibe sélectivement la recapture de la sérotonine (IRSS)**, réduit certains symptômes psychologiques comme : l'irritabilité, la tension, et la dysphorie chez 52% de femmes recevant ce médicament (Steiner et al. 1995). En ce basant sur ces constatations, il a été suggéré que les symptômes thymiques des TDP puissent être déclenchés par des hormones ovariennes chez les sujets sensibles ou prédisposés à une instabilité de l'humeur, et que ces symptômes thymiques peuvent disparaître en faisant disparaître ces hormones ovariennes (ovariectomie) ou en renversant la sensibilité (avec les inhibiteurs de la recapture de la sérotonine) (Rubinow et Schmidt 1995) (68-67).

Autres modifications biologiques

Le bêta-endorphine :

Les **peptides opioïdes** interviennent non seulement dans l'analgésie mais aussi dans de nombreuses fonctions : Humeur, comportement, appétit, sommeil, régulation thermique et fonction intestinale. Les variations cycliques de la **B-endorphine** joueraient un rôle dans le SPM. La **B-endorphine** plasmatique est plus faible chez les femmes souffrant de SPM (15) mais il n'est pas sûr que les niveaux plasmatiques reflètent les modifications cérébrales.

la mélatonine

Plus récemment, des auteurs ont évoqué des anomalies du rythme circadien de la **mélatonine** comme celles observées dans la dépression. Shafii et al (72).

Proposent en effet d'étudier la **mélatonine** en tant que possible "marker" de dérégulation des rythmes circadiens (sommeil, température, cortisol, etc...) dans la dépression et le SPM.

- la **régulation vasculaire**

La modification de la **régulation vasculaire**, avec redistribution de la répartition hydrique par augmentation du coefficient de filtration capillaire sont fréquemment les femmes souffrant de SPM

- la **TSH** et **TRF** : Des réponses anormales de la **TSH** au **TRF** ont été soulignées par certains mais non retrouvé pour d'autres (46).

- **L'aldostérone** : L'élévation prémenstruelle de l'**aldostéronémie** n'a pas été retrouvée par tous les auteurs.

- La **cortisolémie** n'est pas un bon marqueur

- Le **peptide atrio-natriurétique** et l'activité **rénine plasmatique** ne semblent pas jouer de rôle dans le SPM.

Le **magnésium** : Une baisse de la **magnésémie intra-érythrocytaire** chez les femmes souffrant de SPM

Autres facteurs discutés

- déficiences diététiques de Vitamine B6 et magnésium
- Atténuation des réponses de la GH et du cortisol au L tryptophane ;
- Réduction du **sommeil à ondes lentes**, diminution du **sommeil profond** ;
- l'excès d'aldostérone
- la déficience d'endorphine
- les prostaglandines (l'un ou l'autre dans l'excès ou déficience)
- L'hypoglycémie
- l'acide - base le déséquilibre
- L'infection ovarienne asymptomatique et les candidoses chroniques.

Actuellement ces théories ne peuvent pas être attribuées directement aux causes de SPM (Mèches 1988).

2) THEORIES BIOPSYCHOSOCIALES :

Certains auteurs (*Hsia et Long 1990*) croient que le SPM a un composant psychosocial significatif en plus du composant biologique et que ces facteurs psychosociaux influencent le degré des symptômes émotionnels et physiques pendant la période prémenstruelle. Les **facteurs psychologiques** sont en rapport avec le profil de la personnalité (Un contexte de sensibilité thymique est souligné), les croyances vis à vis du cycle menstruel, les superstitions, les perceptions, les craintes, l'adoption du rôle de malade, ainsi que de la co-existence de troubles psychiatriques (Notons la fréquence du SPM chez les femmes ayant présenté une dépression du post-partum d'où l'intérêt de l'étude des antécédents.). Quant aux **facteurs sociaux** sont en rapports avec les passés et l'état actuel, les attitudes de la famille, des amis et de la société vis à vis des femmes, de la menstruation et du SPM, les attitudes envers la maladie, et le soutien social.

Ce **modèle bio-psychosociale** du SPM peut nous aider à comprendre les variations importantes des symptômes qui existe d'une patiente à une autre (*Mèches 1988*) (48).

La notion de SPM fait de plus en plus appel à l'intervention de facteurs psycho-biologiques capables d'influencer simultanément le SNC et le système génital. Ces facteurs pouvant être semblables à ceux qui sous-tendent la psychobiologie des troubles affectifs, l'étude des rythmes biologiques a un intérêt tout particulier chez la femme qui souffre de ce trouble. Ainsi, *Shafii et al. (1990) (72)* rapportent des observations de cas de **SPM ayant un rythme saisonnier**

(*Tamarkin, 1985; Roy-Byrne et al, 1986*) (66-77). Ces ont développé un modèle de **troubles circadiens**, qui permet d'établir quelques relations entre les altérbiologiques décrites dans le SPM et dans la dépression majeure.

On peut se demander si les vulnérabilités inscrites dans la psycho-biologie du cycle menstruel par les vicissitudes du développement ne constituent pas un problème plus grave dans les sociétés qui confèrent à la femme un rôle plus actif et autonome.

Rose et Abplanalp (1983) (65) ont souligné que la montée de l'intérêt pour le SPM est vraisemblablement en rapport avec l'augmentation de l'impact socio-culturel de ce trouble et le profond changement des habitudes sexuelles des femmes. Certains auteurs ont confirmé, que le nombre de journées de travail perdues en raison de troubles de type prémenstruel pourrait être très élevé et que l'impact psychosocial de ces mêmes troubles diffère dans les pays industrialisés et dans les pays du tiers-monde.

3) THEORIES GENETIQUE :

Certaines études montrent bien une influence de l'hérédité sur les symptômes prémenstruels. La ressemblance des symptômes pour les jumelles ne pourrait pas être en rapport avec l'enfance ou la similarité écologique de l'adulte ni avec la similarité dans la parité ou l'emploi de contraceptif oral.

Les symptômes prémenstruels évalués par l'auto-questionnaires pour des paires de jumelles femelles Britanniques de Londres (364 paires) et de Birmingham (98 paires).

- **Dans le plus grand échantillon de Londres**, ils trouvaient que la prédisposition aux facteurs génétiques familiale aux symptômes prémenstruels représente 30%, mais ils notaient aussi une influence modeste de l'environnement familial.

- **Dans le petit échantillon de Birmingham**, le taux d'hérédité trouvé pour les symptômes prémenstruels est beaucoup plus élevé (80%) avec aucune influence pour l'environnement familiale.

Dans une analyse préalable des données sur les symptômes prémenstruels concernant les jumelles la ressemblance des symptômes était due uniquement aux facteurs génétiques, avec taux d'hérédité estimé à 35%.

- **Dans l'unique étude prospective de jumelle** des symptômes prémenstruels, *Dalton et al. (22)* ont examiné 31 paires de jumelles dont le diagnostic de SPM est confirmé cliniquement, ils trouvent le taux de concordance considérablement élevé pour les paires **monozygotes 93%** (23 de 24) alors qu'il n'est que de **44%** que les paires **dizygotes** (12 de 25).

Dans 300 paires de jumeaux volontaires Australiens, *Condon (17)* trouvait une corrélation dans les "scores globaux du SPM", ils sont presque deux fois plus important pour les paires monozygotes ($r=0.55$) que pour les paires dizygotes ($r=0.28$) paires. La plus part des études sont conséquentes (à l'exception des résultats trouvés sur le petit échantillon de Birmingham) et conclues bien que la présence des facteurs héréditaires soit **certaine** dans le SPM mais modéré et que l'influence **de l'environnement familial**, malgré qu'il soit présent, il reste très **limitée**. (25) Ainsi nous pourrions rejeter l'hypothèse que la ressemblance des jumelles pour des symptômes prémenstruels est due entièrement aux facteurs familiaux et écologiques. Ces résultats ne sont pas concordants avec l'hypothèse que les symptômes prémenstruels sont fortement influencés par des attitudes vers **"le rôle féminin"** **appris** régulièrement par des filles de ces parents, par les

facteurs culturels et/ou social, ou par un milieu religieux assez particulier.

4) THEORIES PSYCHOPATHOLOGIQUE :

Les Auteurs de tendance psychanalytique ont largement contribué à mettre en valeur l'importance clinique des troubles périménstruels. Ils ont ramené le syndrome prémenstruel à l'existence d'hypothétiques processus de **"somatisation"** de **conflits psychiques** entraînant la **conversion** des tensions associées à ces derniers en symptômes physiques plus ou moins symboliques.

Dans son livre **"Maternité et sexualité"**, Marie Langer (1951) décrit les caractéristiques de la menstruation, de même que la qualité de l'expérience émotionnelle associée à la survenue des règles, seraient dépendantes de la structure plus ou moins "névrotique" de la personnalité de la femme.

- Pour une **filles non névrotique**, les règles et le cycle menstruel seraient vécus comme une **réconciliation avec la mère**. Elle ressentirait la **maturité sexuelle comme un cadeau de la mère**, qui lui "permettrait ainsi d'avoir des enfants.

- Par contre, **La femme névrotique** (à cause des complexes dont elle souffre), percevrait, les règles de façon **angoissante et culpabilisante**. " L'enfant imagine la blessure comme quelque chose d'intérieur (vu que le sang sort de l'intérieur du corps) et pense que le corps de la femme est blessée. Et comme elle imagine qu'il y a des enfants à l'intérieur du corps féminin, l'hémorragie devient à ses yeux la preuve que ses futurs enfants aussi ont été endommagés". Il existe déjà l'idée de la **blessure, agression** subie par la femme, et, l'organe qui perd du sang étant le génital, la blessure et ses conséquences (catastrophiques) vont être ressenties comme la conséquence d'un acte génital" (Ibidem).

En conclusion, les auteurs d'orientation **psychodynamique** décrivent le SPM comme l'un des avatars cliniques spécifiques du **complexe d'Œdipe** et du **complexe de castration chez la femme**. Mais la psychobiologie du syndrome prémenstruel n'est pas explicitée par ce point de vue. Les hypothèses psychosomatiques réduisent les troubles pré-périménstruels à des phénomènes de type hystérique alors que les symptômes du SPM évoquent davantage la **névrose d'angoisse**, la **dépression** ou la **maladie somatique** proprement dite que les phénomènes de conversion.

T. Benedek (9) a tenté, par contre, de développer un modèle de la complexité, qui tient compte des vulnérabilités spécifiques pouvant sous-tendre les SPM. Pour B., ces troubles seraient à comprendre comme des **"névroses récurrentes prémenstruelles"**. Elle étudie, à travers l'analyse des rêves, les relations qui s'établissent entre la succession des phases du cycle menstruel et la structure du fonctionnement psychique de la femme. **T. Benedek** (9) décrit **"un cycle Psycho-biologique de la femme"**, à l'intérieur duquel la **dimension biologique** et la **dimension psychologique** du cycle sont liées par l'intermédiaire de la notion freudienne de pulsion (c'est-à-dire, une entité à la limite entre somatique et psychique). Le cycle menstruel est vu ainsi comme un **processus dynamique** caractérisé par des phases périodiques d'intégration (progression) et de désintégration (régression) de la pulsion sexuelle ayant des corrélats psychologiques et biologiques spécifiques.

Le concept de "névrose récurrente" fait appel, finalement, à un point de vue "néo-darwinien" et met en valeur la survie, chez la femme, de **vicissitudes de la pulsion sexuelle** qui hériteraient des phénomènes observables dans l'œstrus de

certain animaux. **Th. Benedek** (9) fait correspondre la **phase folliculaire** avec une tendance pulsionnelle dirigée vers l'objet sexuel et visant la gratification par le coït. Quand la production de progestérone commence, dans la **phase pré-ovulatoire**, on verrait apparaître aussi des tendances passivo-réceptives qui, d'abord, s'intègrent avec la tendance active dirigée vers l'extérieur (au moment de l'ovulation). Ensuite, la pulsion sexuelle rejoindrait le plus haut niveau d'intégration (entre les tendances actives et passives qui la caractérisent) au moment de l'ovulation. On pourrait alors parler d'**œstrus**, au sens de sommet du cycle sexuel de la femme. **Dans la phase progestative**, la vie émotionnelle serait dominée, par contre, par des tendances à but passivo-réceptif et de rétention, qui iraient de pair avec la formation du corps jaune et les transformations de l'utérus, qui préparent la femme à une éventuelle maternité.

Les changements hormonaux de la phase prémenstruelle s'accompagneraient, enfin, d'un **processus de régression** à la phase pré-génitale et donc, aussi, de **désintégration de la pulsion psychosexuelle**. **Mme Benedek**(9) formule l'hypothèse que le déficit relatif d'hormones ovariennes, caractéristique de cette période du cycle, est à la base d'une augmentation de l'irritabilité du système nerveux central favorisant l'apparition de **"névroses récurrentes" prémenstruelles** chez des femmes prédisposées. Le cycle influencerait donc à la fois la réactivité émotionnelle et le profil psycho-neuro-endocrinien spécifique d'une femme donnée. Ce "profil", à la fois **neuro-endocrinien** et **psycho-émotionnel**, serait l'expression finale des caractéristiques particulières du développement psychosexuel de la femme. Chez l'être humain, le cycle menstruel deviendrait en somme un cycle sexuel en suivant un chemin tracé par les avatars des relations entre maturation physiologique et développement psychologique.

■ TROUBLES AFFECTIFS ET SPM :

Au cours de ces dernières années, un accent grandissant a été porté, sur les rapports entre SPM et troubles affectifs. L'identification du prototype TDPLT est le résultat de nombreuses recherches ayant essayé de mieux préciser la nature de cette association. Il existe une corrélation très nette entre l'émergence de troubles psychiatriques graves (crises maniaques, tentatives de suicide, etc.) et la survenue des règles chez des femmes souffrant de troubles affectifs bipolaires. L'existence d'une **vulnérabilité prémenstruelle** aux désordres affectifs aurait une valeur diagnostique et pronostique, permettant d'identifier des sous-groupes de patientes bipolaires caractérisés par une évolution plus grave. Un lien est ainsi établi, qui met en valeur les rapports pouvant s'établir entre vulnérabilité affective et perturbation de la sphère génitale à la période prémenstruelle.

La **période menstruelle** a été identifiée depuis longtemps comme un **moment de crise émotionnelle** caractérisé par des troubles importants de l'humeur et de la vie affective pouvant déboucher sur l'**apparition** et/ou l'**exacerbation** de toutes sortes de manifestations pathologiques.

De nombreux travaux expérimentaux avaient aussi montré qu'il existe une augmentation de la mortalité par suicide pendant la période menstruelle et une **association entre SPM et troubles de l'humeur**. Plus récemment, l'hypothèse a été avancée que les troubles périménstruels pourraient Soit :

- Marquer l'existence d'une **vulnérabilité de type bipolaire**
- Ou représenter une **forme infra-clinique de trouble bipolaire**.

Schuckit et al. (1975) (69) auraient trouvé 11% de troubles bipolaires parmi des étudiantes souffrant de SPM.

Ces résultats sont cependant controversés.

Malgré ces premiers résultats décevants, les rapports entre troubles affectifs et troubles menstruels ont continué de faire l'objet d'un vif intérêt.

A partir des années 80, l'étude des relations entre SPM et troubles de l'humeur a été facilitée par l'introduction des méthodologies d'évaluation clinique et diagnostique standardisées. Il a été alors observé que la **prévalence** du SPM diagnostiqué avec le PAF peut atteindre 62% dans des collectifs de femmes présentant des troubles dépressifs majeurs.

En utilisant le PAF, *Endicott et al (1981) (26)* ont distingué un **sous-groupe spécifique** de SPM en rapport avec l'existence d'un diagnostic présent ou passé de Trouble dépressif majeur. Quatre autres sous-groupes seraient identifiés, respectivement, par la coexistence de **rétenion d'eau, malaise général, impulsivité pathologique et difficultés sociales**. En utilisant cette classification, *Endicott et al (1981)* auraient pu confirmer l'existence d'une association significative entre le **sous-groupe dépressif du SPM** et la **présence de Troubles dépressifs majeurs**. Les résultats de cette investigation prospective (incidence accrue des troubles affectifs dans le groupe avec SPM et dépression majeure par rapport au groupe de contrôle avec trouble affectif seul) sont nuancés, mais suggèrent que le **SPM type dysphorique** pourrait représenter une « **forme atténuée et spécifique** » de troubles de l'humeur.

L'existence d'une association significative entre **clinique de la dépression bipolaire/endogène/majeure et SPM** a été également suggérée (*Halbreich et Endicott, 1985; Endicott et al, 1986*) (34-27) par les résultats d'études prospectives effectuées au moyen de calendriers d'auto-évaluation. Par ailleurs :

- 57% des femmes avec un diagnostic "lifetime" de trouble dépressif majeur présenteraient un SPM de type dysphorique au PAF,

- Alors que seulement 14% de femmes sans pathologie psychiatrique souffriraient de ce trouble.

Réciproquement :

- 84% de femmes avec SPM type dysphorique au PAF souffrent d'un trouble dépressif majeur (RDC)

- Et seulement 9% de femmes souffrant de SPM ne présentent aucune affection psychiatrique (*Halbreich et Endicott, 1985*) (34).

Hypothèse du syndrome affectif lié à la période prémenstruelle : SAPM

De Jong et al. (23) (1985) ont également utilisé des méthodes d'investigations prospectives pour vérifier l'hypothèse qu'il pourrait exister un « syndrome affectif lié à la période prémenstruelle » (**premenstrually related mood syndrom : PRMS**). Ces auteurs ont étudié 57 femmes (d'âge compris entre 22 et 45 ans) qui avaient été dépistées sur la base d'une histoire antérieure de problèmes d'humeur et de changements physiques pendant la période prémenstruelle. Toutes ces femmes ne prenaient pas de médicaments et avaient été répartis en trois groupes selon le diagnostic psychiatrique (pas de troubles structurés, troubles affectifs, autres troubles). Le diagnostic (prospectif) de SAPM était retenu sur la base des critères établis par l'Institut américain de la Santé Mentale (*PMS NIMH research workshop*):

- Augmentation de la gravité des symptômes dysphoriques d'au moins 30% pendant la semaine précédant les règles.

- Le diagnostic (prospectif) de **SAPM type dépressif** fut retenu chez 58% des sujets.

- Quarante-deux femmes (73.6%) n'avaient pas une

aggravation significative des troubles de l'humeur au moment des règles contrairement à ce qui avait été rétrospectivement indiqué au moment de l'entrée dans l'étude.

Les femmes sans confirmation prospective de SAPM étaient plus âgées et avaient, elles aussi, des troubles de l'humeur au cours du suivi, mais ces troubles n'étaient pas liés à la période menstruelle. Contrairement à l'attendu, le diagnostic, à l'entrée dans l'étude, de troubles de l'humeur DSM IV, était plus fréquent chez les femmes sans confirmation prospective de SAPM (58% vs 30%). L'étude démentit donc clairement l'hypothèse que toutes les formes de SPM (selon l'acception SAPM) sont l'expression d'un trouble de l'humeur sous-jacent mais ne contredit pas l'hypothèse que le SPM ou des sous-groupes de SPM, pourraient constituer une forme particulière de trouble affectif.

De Jong et al, (1985) (23) ont souligné que le **SPM pourrait "sensibiliser" une femme présentant une vulnérabilité spécifique à la dépression**. Réciproquement, un **épisode dépressif pourrait "prêter" sa symptomatologie** caractéristique à un trouble menstruel.

L'étude de *De Jong (23)* montre, la **complexité méthodologique** de ce type de recherches permettant de saisir la prévalence des diverses formes de SPM, ainsi que la fréquence de la comorbidité SPM/troubles dépressifs majeurs. Il apparaît déjà, cependant, que nombre de femmes peuvent souffrir, lors de la période menstruelle, d'une espèce de « **dépression brève récurrente** » qui présente, un intérêt clinique et psychobiologique tout à fait considérable sur le plan de l'étude des altérations psychosomatiques et psychoneuroendocriniennes associées aux troubles de l'humeur.

Enfin, *Ascher-Svanum et al (1990) (7)* ont étudié de façon prospective une large population hospitalière, qu'ils ont suivie pendant une année. Ces auteurs trouvent à nouveau une prévalence significativement accrue de SPM chez les patientes souffrant de troubles affectifs. La prévalence observée est, cependant, inférieure (21% par rapport à 65%) à celle trouvée dans le cadre d'études rétrospectives (*Endicott et al, 1981*) (26). Peu de relations sont d'ailleurs trouvées entre le **diagnostic prospectif de SPM et la gravité de la dépression** ou la **présence d'une histoire familiale de dépression**.

En conclusion, il existe sûrement une relation entre SPM et troubles de l'humeur et il est possible que le SPM constitue, lorsqu'il se présente isolé, une forme atténuée ou infra-clinique de trouble de l'humeur.

Gitlin et al (1989) (63) et Hartley Gise et al (1990) (35) ont énuméré les principales opresentées dans la littérature à ce sujet:

- Troubles psycho-émotionnels d'origine organique,

- Troubles hystérde conversion,

- Troubles psychosomatiques issus de phénomènes de somatisation plus complexes,

- Troubles psychiques (*Coppen et Kessel, 1963*) (18).

- Troubles affectifs majeurs atypiques.

- Troubles liés à une sensibilisation de l'axe du stress (*Heilbrun et al, 1989; Schmidt et al, 1990*). (38-68)

- Forme subclinique de troubles affectifs majeurs (*Rubinow et al, 1984; Chisholm et al, 1989*), (67-13)

- SAPM (*De Jong et al. , 1985*) (23)

- Désordre des rythmes biologiques corrélé aux troubles affectifs saisonniers (*Shafii et al, 1990*). (72)

Hartley Gise (36) et al admettent finalement une **étiopathogenèse polyfactorielle** du SPM

■ SPM ET IMPACTE SUR LE COUPLE

Il faut aussi ne pas négliger l'impact sur le couple. Peu d'études s'y sont intéressées. Citons *Cortese et Brown en 1989 (12)* qui ont clairement montré que les partenaires étaient affectés par les symptômes de leurs compagnes ou par la manière dont ces dernières utilisaient leurs symptômes. Ils ont aussi observé une différence de stratégie d'adaptation des hommes dépendant de la sévérité du syndrome. Ainsi, les partenaires de femmes souffrant **sévèrement du SPM** avaient plus tendance à rechercher de l'information et de l'aide et se sentaient plus en colère face à la situation.

Une étude latino-américaine faite en 1995 s'est intéressée à la **communication dans le couple** durant les périodes pré et post-menstruelles (pendant deux cycles consécutifs). Les auteurs observèrent une **baisse de la communication** durant la période prémenstruelle, en comparaison de celle suivant les règles. Par ailleurs, ils ont aussi montré une corrélation négative entre la communication dans le couple et la sévérité de leurs symptômes. Ces corrélations furent retrouvées, mais à un moindre degré, en considérant les questionnaires masculins.

• STRATEGIES THERAPEUTIQUE DU SPM

Compte tenu de l'absence d'étiologie précise, aucun traitement spécifique n'existe actuellement ; la plus part des approches thérapeutiques envisagées restent essentiellement symptomatiques. En outre les traitements placebo apportent 30% d'amélioration **HERAPIES**

1-mesures hygiénodietétiques

-Les formes légère et moyenne du SPM ne nécessitent pas généralement de traitement médicamenteux, mais plutôt des mesures hygiéno- diététiques. Certains conseils sont utiles durant cette période. (21)

1. Eviter l'exposition au stress et aménager au mieux la vie personnelle et sociale en fonction des dates du cycle menstruel;

2. **Limiter la tension nerveuse et la fatigue physique** ; (relaxation, temps supplémentaire de repos).

3. **Alimentation appropriée**: respecter un certain degré de restriction hydro-sodée, éviter les sucres à élimination rapide, ainsi que les excitants du système nerveux (alcool, tabac, boissons contenant de la méthylxanthine: Café, thé, boissons au cola.)

4. Approche nutritionnelle:

Encouragé une alimentation riche en calcium (apport supplémentaire de 1000 mg/j), en magnésium (360 mg d'ion magnésium), en manganèse et en zinc Réduit de façon significative les symptômes du SPM.

-Limité la consommation de sel, de viande rouge, de caféine et du chocolat. ,

- Réduction des graisses d'origine animale, et de la consommation d'acides gras insaturés dans leur forme isomérique " trans"; apport en acides gammalinoléique (huile d'onagre) précurseur des prostaglandines E1, dont le déficit a été invoqué à l'origine de la plupart des manifestations du syndrome prémenstruel

-l'apport multi-vitaminique en vitamine B6, en vitamine E, a aussi fait la preuve de son efficacité (études randomisées contre placebo de London et de *Chakmakjian*)

5. **Organiser une vie calme**, Les patientes devraient être encouragées, par exemple, à obtenir un sommeil adéquat pendant la période prémenstruel et pratiquée certaines exercices physiques, (32). (activité sportive régulière)

6. **Un support de soutien moral et psychologique** devrait être offert à toutes femmes présentant un SPM. L'éducation de ces patientes et de leur famille de la réalité des symptômes

préménstruels permet de réduire les sentiments de honte, de culpabilité, et d'impuissance qui sont généralement présent chez ces patientes et aussi une meilleure compréhension et assistance de leur entourage. Des évaluations quotidiennes des symptômes par des « calendrier d'auto-évaluation » permet a la femme un plus grand sens de prédictabilité et de contrôle de ces symptômes et peuvent l'encourager à réorganiser son horaire et à minimiser au maximum le stress pendant la semaine prémenstruelle.

7. **Expliquer la situation de ces patientes** (souffrant de SPM) à l'entourage.

8. rechercher et **éliminer d'autre facteur déclenchant** (6)

2. Soutien psychologique et Psychothérapies

Il faut **rassurer les patientes sur la bénignité des signes** et assurer leur caractère fonctionnel

-conseiller les patientes des troubles afin de bien maîtriser leurs symptômes

Afin d'améliorer les aspects psychologiques du SPM, plusieurs variétés de méthodes psychothérapiques ont été pratiquées avec succès. Les **thérapies de Relaxation et les psychothérapies individuelles de rétro contrôle biologique à court-terme** (thérapie cognitive) ont été particulièrement efficaces.

-Les thérapies cognitivo-comportementales

L'efficacité des thérapies cognitivo-comportementales est de plus en plus confirmées, soit par la **restructuration cognitive** soit par un **travail d'affirmation de soi**.

ces thérapies réduisent d'une manière assez significative les symptômes du SPM, l'un des avantages de la thérapie cognitive dans le traitement du SPM par rapport à l'usage des antidépresseurs c'est qu'elle permet d'assurer des bons résultats qui seront entretenus avec le temps (28)

Une étude récente (29) (1997), compare des patientes soumises à une **thérapie cognitive** à des patientes sur **liste d'attente**. Il s'agissait de traitements individuels s'étendant sur **12 semaines avec une séance par semaine**. Les résultats démontrent que la thérapie cognitive était de manière significative plus efficace que le fait d'être dans un groupe d'attente. On a pu constater la **rémission quasi complète** des symptômes tant somatiques que psychiques, ainsi que les difficultés de fonctionnement.

Les conclusions sont donc que la thérapie cognitive représente dans l'état actuel l'une des thérapies les plus **«efficace»** pour le SPM en absence d'un traitement étiologique.

Un follow-up à 2 et 4 mois après l'intervention a montré une légère amélioration se poursuivant en comparaison des données à la fin du traitement.

Dans une étude faite en 1995 (25) Les deux thérapies (**cognitivo-comportementales et focalisée sur l'information**) se sont révélées aussi efficaces l'une que l'autre dans la **réduction des traits d'anxiété, de dépression et des pensées négatives automatiques**, ainsi que dans l'**impact des changements physiques**.

-La thérapie focalisée sur l'information

Cette attitude thérapeutique vise à améliorer le soutien social des femmes souffrant de SPM, avec notamment une intervention en groupe durant 4 cycles consécutifs, où l'accent est mis sur le **recadrage positif du vécu**, tant **émotionnel** que **cognitif**, des femmes face à leurs symptômes, en insistant sur l'**identification des changements positifs**. Ce recadrage positif peut être très bénéfique pour des femmes souffrant de SPM en apportant une opportunité de **redéfinir leur image sociale** dans laquelle en générale la menstruation est perçue

comme un élément négatif. Il permet aux femmes d'explorer des phénomènes auparavant non identifiés en raison du contexte socioculturel. Le **recadrage positif** peut par exemple se faire en discutant avec les patientes du fait que les menstruations sont l'affirmation de leur fertilité, de leur normalité en tant que femme. Une large variété de traitements pharmacologiques a été rapportée permettant de réduire la symptomatologie du SPM. Les traitements employés visent généralement une des trois stratégies : le soulagement des symptômes, la modification d'un déséquilibre biochimique possible, et suppression de l'ovulation.

LES PSYCHOTROPES :

Les Anxiolytiques :

La buspirone (Buspar*) et l'Alprazolam (Xanax*) donnés en phase lutéale sont efficaces. La fluoxétine (Prozac*), antidépresseur qui augmente la sérotonine, à la dose de 20 mg/j pendant 3 cycles, est également efficace mais les symptômes récidivent 15 jours à 1 mois après l'arrêt du traitement.

Présentation pharmacologique : (47)

Xanax® Pharmacia & Upjohn Orale Alprazolam Cp. à 0,25-0,5-1,0 et 2,0 mg **posologie** : *0,25 mg 3x/jour en phase lutéale

Les Antidépresseurs : TC et IRSS

Les traitements sérotoninergiques sont révélés efficaces ces dernières années.

De nombreuses études ont démontré l'efficacité des antidépresseurs sérotoninergiques (40), comme la **fluvoxamine** (Floxyfral), la **fluoxétine** (Prozac), la **paroxétine** (Deroxat) ou la **sertraline** (Zoloft). On peut aussi citer la clomipramine (Anafranil) de la classe des tricycliques.

Tous ces traitements sont efficaces, ils se distinguent essentiellement par leurs **demi-vies** plus ou moins longues (la fluoxétine a une demi-vie longue de 2 à 3 jours, les autres ayant une demi-vie de 24 à 30 heures). Ils se distinguent aussi par leurs **effets secondaires**, en général rares et disparaissant après la première semaine de traitement, qui varient d'une patiente à l'autre. Afin de minimiser les effets secondaires, la dose thérapeutique la plus basse sera tentée.

De plus, des études ont aussi montré que **des doses séquentielles** de ISRS à **courte demi-vie** données uniquement durant la phase lutéale, par exemple sertraline (Zoloft), se révélaient efficaces. Ceci paraît étonnant, lorsqu'on pense à la durée d'entrée en action des antidépresseurs varie de 2 à 8 semaines habituellement, pourtant cela semble bien un **traitement possible et élégant**. Plusieurs études contrôlées montrent que la fluoxétine à 20 mg/jour est efficace dans les symptômes dysphoriques prémenstruels (12, 5). D'autres médicaments sérotoninergiques, incluant la paroxétine, la sertraline, et clomipramine, paraissent aussi efficaces dans l'anxiété et dépression prémenstruelle. Pour garantir un maximum d'efficacité certaines études préconisent la prescription des antidépresseurs d'une façon continue durant tout le cycle menstruel, bien que d'autres décrivent des résultats positifs quand les antidépresseurs (essentiellement les IRSS) sont administrés pendant les 12-14 jours prémenstruels. (12)

Présentation pharmacologique : (30)

Deroxat® SmithKline Beecham Orale Paroxétine Cp. à 20 mg **posologie** : *20 mg/jour.

Zoloft® Pfizer Orale Sertraline Cp. à 50 mg **posologie** : *50 à 150 mg/jour

Seropram® Lundbeck Orale Citalopram Cp. à 20 mg **posologie** : *20 mg/jour.

Fluoxetine® Lilly Orale Fluoxétine Gélules et cp. à 20 mg **posologie** : *20 mg/jour

L'HOMONOTHÉRAPIE :

La progestérone et ses dérivés :

L'homonothérapie reste le recours le plus fréquent et le plus utile en cas d'échec des autres moyens.

-La **progestérone naturelle** sous sa forme injectable représentait une thérapeutique très efficace du syndrome prémenstruel, mais elle n'est plus actuellement disponible.

3. La progestérone micronisée sous forme orale a les mêmes propriétés, mais son efficacité est plus irrégulière et son administration entraîne parfois des effets secondaires inconnus avec la molécule endogène; dans ces cas, l'administration vaginale des comprimés apporte une meilleure tolérance.

4. Les **progestérone injectables retard** ont une action beaucoup moins régulière et ne peuvent pas être utilisées dans cette indication.

5. Les **progestatifs de synthèse** sont irrégulièrement efficaces sur le syndrome prémenstruel car l'exacerbation de certaines propriétés progestatives, sur l'endomètre par exemple, n'a aucune relation avec l'ensemble des propriétés biologiques de la progestérone naturelle. C'est pourquoi les meilleurs résultats sont à attendre des progestatifs de synthèse qui ont pratiquement gardé toutes les propriétés biologiques de la progestérone (**démégestone, rétroprogestérone**), à raison de 2 à 3 comprimés par jour en commençant 2 à 3 jours avant la date d'apparition habituelle des symptômes, jusqu'à la veille de la date présumée des règles. Les **norandrostanes** sont moins régulièrement efficaces, mais peuvent avoir de bons résultats en cas d'échec des PS 1 face à certains symptômes (céphalées, asthénie), probablement du fait de leur proximité chimique avec la testostérone. Il semble d'ailleurs que chaque patiente soit mieux soulagée par un progestatif de synthèse que par les autres.

Il est également possible d'utiliser un **estroprogestatif oral** pour mettre l'ovaire au repos et substituer un climat hormonal artificiel fixe au jeu perturbé des hormones endogènes; on choisira une formulation à climat progestatif dominant, ne comportant pas plus de 30 microgrammes d'éthinylestradiol. Lorsqu'il existe un désir contraceptif, il peut être résolu de la même manière; une autre méthode est de prescrire un progestatif à fort pouvoir antigonadotrope sans effet métabolique du 5e au 25e jour du cycle (promégestone, acétate de nomégestrol).

Présentation pharmacologique : (47)

Colpro® Orale **Médrogestone** Cp. à 5 mg 5 à 10 mg 1x/jour du 16e au 25e jour du cycle. **Duphaston®** Orale **Dydrogesterone** Cp. à 10 mg 20 mg 1x/jour du 11e au 25e jour du cycle

Utrogestan® Orale **Progestérone naturelle micronisée** Caps. à 100 mg 2 capsules (200 mg) à 3 capsules (300 mg) pendant 10 jours, habituellement du 17^{ème} au 26^{ème} jour du cycle.

Progestogel® Percutanée **Progestérone** 100 g gel et 1 g progestérone *1 dose (5 g)/jour réparti sur les deux seins (1 réglette de 2,5 g sur chaque sein), du 10e au 25e jour du cycle ou en continu à partir de la fin des règles.

Les agonistes du GnRH :

Ces produits agissent en supprimant le cycle menstruel. Le **danazol** à dose inhibant l'ovulation a été largement utilisée avec un certain succès dans le traitement des symptômes comportementaux et physiques du SPM (Nader 1991). Ces produits sont plus efficaces que le placebo mais leur action est accompagnée d'effets secondaires en augmentant le risque d'ostéoporose qui est généralement associée à la suppression prolongée de l'ovulation ce qui limite généralement son emploi (Pearlstein 1995). L'association GnRH et AddBack thérapie pourrait constituer une solution pour les formes invalidantes.

Le Danazol (21) mais ses effets secondaires limitent son emploi. (6)

Présentation pharmacologique : (47)

Danazol® Orale **Danazol** Gélules à 100 et 200 mg *100-200 mg 2x/jour, en phase lutéale ou en continu.

LA VITAMINOTHERAPIE :

Un apport multi-vitaminique : **Vitamine B6**(100 mg/j), **B complexe**, **Vitamine E** (400-600 IU/J) et **vitamine C** (1000 mg/j); est recommandée pour soulager certaines symptômes (l'irritabilité, la rétention des liquides, les manifestations douloureuse associée, la tension des seins , l'anxiété, la dépression et la fatigue..)

La **pyridoxine (vitamine B6)**, coenzyme du métabolisme de la dopamine, sérotonine et norépinéphrine a été utilisée a une posologie de 100 à 150 mg par jour comme traitement symptomatique du SPM (dépression, irritabilité, fatigue, œdème, céphalée) ;

L'emploi excédentaire de Vitamine B6 peut conduire au développement de neuropathie périphérique et afin d'éviter ces effets secondaires (généralement réversibles a l'arrêt du traitement) l'apport supplémentaire de vitamine B6 ne doit pas dépasser 150 mg par jour

Certains auteurs considèrent que L'administration de 50 à 100 mg de pyridoxine n'est pas plus efficace qu'un placebo. (6, 45)

Présentation pharmacologique : (47)

Vitamine B6 (pyridoxine) Cp. à 100 mg 100 mg/jour environ une sem. avant et jusqu'au 3e jour après le début de la menstruation.

LES SELS MINÉRAUX :

Le calcium et le magnésium ont été essayée pour la dépression prémenstruelle, douleur, et fatigue. Bien que les données sur leur efficacité ne sont concordantes, ces sels sont généralement bénéfique et bien tolérés. Les doses de 300-500 mg/jour de magnésium sont efficaces, et sont indiquées seulement pendant la période prémenstruel (6)

LES INHIBITEURS DE LA PROSTAGLANDINE :

Du fait que les prostaglandines modulent les réactions inflammatoires et augmente la sensibilité à douleur, les inhibiteurs de la prostaglandine peuvent réduire la douleur et l'inflammation. En particulier, l'acide mefenamic (Ponstel) et le naproxen de sodium (*Anaprox*, *Naprosyn*) sont efficaces dans les algies pelviennes prémenstruelles, les myalgies et les céphalées. Pour une meilleure efficacité, ils devraient être commencés avant l'apparition des symptômes (7-10 jours avant la menstruation). Les inhibiteurs de prostaglandines ne semblent pas être efficace sur les des symptômes thymique prémenstruels. (6)

LES INHIBITEURS DE LA PROLACTINE :

Certaines études ont montré l'efficacité des **inhibiteurs de la prolactine (la bromocriptine)** dans certains symptômes algiques associé au SPM (dysménorrhée, tension mammaire (O'Brien 1985). Mais elle s'accompagne souvent d'effets secondaires: hypotension, nausées, vomissements. Les femmes devraient être conseillées a prendre la bromocriptine au cours des repas, pour éviter les effets indésirable (6)

Présentation pharmacologique : (47)

Parlodel® Novartis Pharma Orale

Bromocriptine Cp. à 2,5 mg; caps. à 5 ou 10 mg Commencer le traitement le 14e jour du cycle avec 1,25 mg/jour, en augmentant par paliers de 1,25 mg/jour pour atteindre 2,5 mg 2x/jour, jusqu'à la menstruation. (B 14, 30 et 100 cp. à 2,5 mg; B 100 caps. à 5 mg B 30 et 100 caps. à 10 mg)

Serocriptin® Serono Orale

Bromocriptine Cp. à 2,5 mg ½ cp./jour à partir du 14ème jour du cycle menstruel. Augmenter progressivement de ½ cp./jour

jusqu'à une dose journalière de 2 cp. Jusqu'à la menstruation. (B 30 et 90 cp).

Dopergin® Schering Orale

Lisuride Cp. à 0,2 mg Commencer le traitement le 14e jour du cycle avec 0,1 mg/jour, en poursuivant avec 0,1-0,2 mg 2x/jour jusqu'à la menstruation. Arrêter le traitement au bout de 6 à 12 cycles afin de tester si la poursuite du traitement est nécessaire.

LES DIURÉTIQUES :

Les Diurétiques ont été utilisés pour traiter certains symptômes du SPM qui sont attribuée à une rétention d'eau et sodium (prise de poids, œdème, tension des seins, ballonnement abdominal). Malgré l'absence de données physiopathologique valable de la rétention hydro sodée dans le SPM, les études ont montré que le **spironolactone** (antagoniste d'aldostérone), pris pendant la phase de Lutéale, soulage les symptômes de rétention hydro sodée et améliore aussi les troubles dysphorique associés aux manifestations oedémateux (*Mêches 1988*). Les femmes qui n'éprouvent pas d'œdème prémenstruel ne paraissent pas bénéficier de diurétiques. (48)

Présentation pharmacologique (47)

Aldactone® Searle Orale **Spironolactone** Cp. à 25, 50 et 100 mg *100 mg/jour du 12-14ème jour du cycle menstruel jusqu'à la menstruation.

Hygroton® Novartis Pharma Orale **Chlortalidone** Cp. à 25 et 50 mg Rétention hydrique du syndrome prémenstruel à condition que la prise de poids soit le symptôme dominant et soit bien documentée. L'administration ne se fera que sur des périodes limitées. Les doses ne doivent pas excéder 50 mg/jour.

Spiroctan® Roche Orale **Spironolactone** Dr. à 25 et 50 mg, caps. à 100 mg *100 mg/jour du 12-14ème jour du cycle menstruel jusqu'à la menstruation. D 20 et 100 dr. à 25 mg

Xélonal® Mepha Pharma Orale **Spironolactone** Lactab à 50 et 100 mg *100 mg/jour du 12-14ème jour du cycle menstruel jusqu'à la menstruation.

AUTRES MÉDICAMENTS :

Une variété de divers médicaments a été rapportée réduire certaines symptômes du SPM.

-**Les beta-bloquant** : (atenolol*) peut améliorer l'irritabilité, l'hyper-émotivité et l'excitabilité prémenstruelle

-**La clonidine** : médicament antihypertenseur ; stimulant la production de B endorphine, aurait une action favorable sur certains symptômes psychique du SPM (30) comme l'hostilité, l'irritabilité, l'anxiété prémenstruelle et les troubles dysphorique..

-**L'antagoniste opiacé naltrexone** : peut réduire les symptômes prémenstruels généraux, incluant l'irritabilité, l'anxiété, la dépression, les céphalées et les mastalgies prémenstruel

-**Analgésiques, anti-inflammatoires non stéroïdiens** :indiquée dans les manifestation algique et les tension mammaire associées au SPM :

Ponstan® Parke-Davis Orale, rectal **Acide méfénamique** Caps. à 250 mg, filmtabs à 500 mg, supp. à 500 mg 500 mg 3x/jour en phase lutéale.

LE TRAITEMENT CHIRURGICAL :

Ce n'est que dans les formes sévère du SPM que l'indication d'une castration chirurgicale se pose (12, 13). Soulignons que le recours à ce traitement radical est exceptionnel ; d'où l'intérêt dans ces formes sévères du traitement par les agonistes de la GnRH.

CONCLUSION

Le SPM est éprouvé pendant la phase de Lutéale du cycle menstruel par une proportion significative de femmes. Il est

caractérisé par des symptômes **compo, psychologiques ou physiques de degré et sévérité variable**. Actuellement, **aucune cause spécifique** ne peut être considérée comme l'unique facteur étiologique du SPM. Comme confirment les multiples traitement du SPM. Les hormones ovariennes jouent certainement un rôle important dans son étiologie. L'utilisation des agonistes du GnRH et du danazol, annulent les fluctuations hormonales pendant le cycle menstruel. Le traitement des symptômes psychologiques du SPM chez certaines femmes suggère **une déficience de sérotonine** qui joue certainement un rôle dans la genèse du SPM.

La théorie bio-psychosociale reconnaît l'influence possible **des facteurs sociaux et psychologiques**, ainsi que **des facteurs biologiques** dans l'étiologie du SPM, et peut expliquer pourquoi les variations inter-individuelles des symptômes du SPM sont assez importantes entre les individus. Le SPM ne peut pas être attribué à une seule cause, il résulte certainement d'une **influence génétique considérable** et de l'inter- action de plusieurs facteurs **socio-psycho- neuroendocrinienne** qui s'enchevêtrent entre eux d'une manière assez complexe.

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علاقة أفضلية اليد بالوظائف المعرفية

(دراسة نيوروسيكولوجية مقارنة لدى عينة من طلبة الجامعة)

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ملخص البحث: أجريت الدراسة بهدف التعرف على الفروق بين طلبة الجامعة في الوظائف المعرفية في ضوء كل من الجنس، والخصص الدراسي وأفضلية استخدام اليد. وكذلك التعرف على ما إذا كان هناك تأثير دال للتفاعل بين هذه المتغيرات الثلاثة على الوظائف المعرفية أم لا. وقد تكونت عينة الدراسة من 200 من طلبة الجامعة موزعة إلى مجموعتين متساويتين حسب الجنس، ممن تراوحت أعمارهم بين 18-25 سنة بمعدل قدره 20.43 عاماً، ومن تخصصات دراسية مختلفة، وكان من بين أفراد العينة 144 طالباً وطالبة ممن يستخدمون اليد اليمنى بنسبة 72%، و56 طالباً وطالبة ممن يستخدمون اليد اليسرى بنسبة 28%. وقد استخدمت الدراسة مجموعة من الأدوات لقياس الوظائف المعرفية (اختبار بنون للاحتفاظ البصري، إعادة الأرقام، ترتيب الصور، اختبار التعقب أو توصيل الحلقات)، بالإضافة إلى أداة أعدّها الباحث لقياس أفضلية استخدام اليد. وأشارت نتائج الدراسة إلى وجود فروق دالة بين الجنسين في معظم الوظائف المعرفية لصالح الطلاب، وكذلك بين طلبة الكليات النظرية والكليات العملية لصالح الأخيرة. كما أوضحت النتائج وجود فروق بين أنماط استخدام اليد في الوظائف المعرفية، ووجود تفاعل دال بين كل من الخصص الدراسي وأفضلية استخدام اليد في التأثير على بعض الوظائف المعرفية (الدأكرة اللفظية والتنظيم المكاني وسرعة الاستجابة)، وتفاعل دال بين الجنس واستخدام اليد في التأثير على القدرة على التخطيط والتنظيم البصري، كما تبين وجود أثر دال للتفاعل بين استخدام اليد والتاريخ الأسري لاستخدام اليد اليسرى على الدأكرة البصرية المكانية.

(Haeley et al., 1986; Witelson & Goldsmith,

1991; Holder, 1992; Annett, 2001)

(Broca)

1861

Sinistrals

Bihemispheric

Dextrals

Functional Laterality

*Corpus Callosum

Anomalous Dominance

Neurosciences.

(Witelson &

Goldsmith, 1991; Cornish, (1996; Kathleen & Eliassen, 1998).

(Kolb &

whishaw, 1990; (Bishop, 1990; Hylton & Hartman, 1997; Alworth, 2000)

(Aaron, 1996)

(Padovani et al., 1992)

Reversed laterality

Crossed aphasia

Left handedness

(Springer & Deutsch, 1999, p. 18) Right handedness

(Aaron,

1996)

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) Leading Hemisphere

Function of diversity

:2001)
Beaumont et al., 1984; McCallum, 1981; Spinelli & , 137
Mecacci, 1990; Springer & Deutsch, (1999)

Sequential

Holder, 1994)
 .(1992, Schold, 1998, Springer & Deutsch, 1999

(Rodriguez et al., 1994;

138 :2001

(Witelson, 1985;
Witelson & Goldsmith, (1991; Kolbe & Whishaw, 1990; Holder,
.1992; Kathleen & Eliassen, 1998)

.(Annett, 2000,

.(Jonathan,1998,141 :2001)

(Tan & Kutlu, 1992)

.(1999)
(Annett, 1985)

Dexterous

Recessive	() Dextral () Sinister ()
Parental	Hand Preference P. Broca Handedness
Plenum Temporal	Handedness
Left handedness	
Visuospatial skills	
Autism	Ambidextrous
	Dominant Gene

(Soliman,1989) (200 200) 400		Handedness Discrete variable	
(Annett,1992)		(Annett,2001)	
(John & Martin, 1997)		(Al-Biali, 1993)	
104	86	190	24-19
21.4			
(88) .(102)		-1	
		-2	
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		-5	
(1999)		75	
		56	

مجلة شبكة العلوم النفسية العربية: العدد 3 - جويلية - أوت - سبتمبر 2004

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1999 (Al-Biali, 1993; Soliman, 1989).

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20.43 25-18
() 1.58
1.12

(1994)

(Al-Biali, 1993)

(1999)

(1)

(1)

(Cornish, 1996)

4.

(Jones & Martin, (1997)
()

(Annett, 1992)

5.

		ن = 100		ن = 100		
%	ك	%	ك	%	ك	
72	144	69	69	75	75	اليدين اليمينى
28	56	31	31	25	25	اليدين اليسرى
100	200	100	100	100	100	المجموع
40	80	34	34	46	46	كليات عملية
60	120	66	66	54	54	كليات نظرية
100	200	100	100	100	100	المجموع
30	60	39	39	21	21	تاريخ أ. موجب
70	140	61	61	79	79	تاريخ أ. سالب
100	200	100	100	100	100	المجموع

(%25)

(%31)

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- Edinburgh (1971) Oldfield Handedness Inventory
- Waterlow Handedness Questionnaire
- Annett Hand 12 (Annett, 1985) Preference Questionnaire 6
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- (Annett, 2001)
- 45
- 0.88
- 9
- Benton Visual Retention Test 1974
- 11
- (1989)
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مستوى الدلالة		طالبات ن = 100		طلاب ن = 100		الجنس
						البعد
0.001	4.18	1.31	5.95	1.262	6.68	1 يتنون.
0.001	3.44	0.98	5.76	1.08	6.26	2 إعادة الأرقام للأمام.
0.05	2.43	0.82	4.99	0.88	5.28	3 إعادة الأرقام للخلف.
0.001	3.24	1.63	10.76	1.72	11.54	4 إعادة الأرقام (د.ك.).
0.001	4.61	1.10	15.53	1.26	16.30	5 ترتيب الصور.
غ.د	1.03	4.81	39.75	4.69	39.06	6 التعقب (جزء أ)
0.001	4.13	6.34	62.10	7.04	58.19	7 التعقب (جزء ب)

() .Vigilance
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Visual Mental flexibility
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(Alsworth, 2000, Marnate, 2000, p.

237)

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: Tukey (Post HOC)

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Three way ANOVA

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المتغيرات	مصدر التباين	مجموع المربعات	درجات الحرية	متوسط مجموع المربعات	قيمة (ف)	الدلالة
اختبار بنتون	الجنس (أ)	12.87	1	12.87	11.19	0.001
	التخصص	3.00	1	3.00	2.61	غ.د
	الدراسي (ب)	89.93	4	22.47	19.53	0.001
	استخدام اليد (ج)	0.33	1	0.33	0.28	غ.د
	أ x ب	6.59	4	1.65	1.43	غ.د
	ب x ج	4.92	4	1.23	1.07	غ.د
	أ x ب x ج	4.28	3	1.43	1.30	غ.د
	الخطأ	208.24	181	1.151		
إعادة الأرقام للأمام	الجنس (أ)	2.55	1	2.55	3.30	غ.د
	التخصص	15.82	1	15.82	20.52	0.001
	الدراسي (ب)	36.56	4	9.14	11.85	0.001
	استخدام اليد (ج)	1.35	1	1.35	1.75	غ.د
	أ x ب	3.51	4	0.88	1.140	غ.د
	ب x ج	9.20	4	2.30	2.98	0.05
	أ x ب x ج	1.69	3	0.231	0.299	غ.د
	الخطأ	139.57	181	0.771		
إعادة الأرقام للخلف	الجنس (أ)	1.123	1	1.123	1.89	غ.د
	التخصص	6.07	1	6.07	10.33	0.001
	الدراسي (ب)					

التخصص	طلاب 100 = ن	طالبات 100 = ن	مستوى الدلالة
1 اختبار بنتون.	6.13	1.32	0.05
2 إعادة الأرقام للأمام.	5.78	1.09	0.001
3 إعادة الأرقام للخلف.	4.96	0.89	0.001
4 إعادة الأرقام (د.كلية).	10.74	1.78	0.001
5 ترتيب الصور.	15.78	1.22	غ.د
6 التعقب (جزء أ)	40.13	4.90	0.01
7 التعقب (جزء ب)	61.26	7.25	0.01

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15 28 16 101 40

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One way ANOVA

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الأدوات	مصدر التباين	د.ج	مجموع المربعات	متوسط المربعات	قيمة (ف)	الدلالة
اختبار بنتون	بين المجموعات	4	91.77	22.24	16.93	0.001
	داخل المجموعات	195	264.23	1.36		
	المجموع الكلي	199	356.00			
إعادة الأرقام للأمام	بين المجموعات	4	41.53	10.38	11.22	0.001
	داخل المجموعات	195	180.45	0.92		
	المجموع الكلي	199	221.98			
إعادة الأرقام للخلف	بين المجموعات	4	21.83	5.46	8.62	0.001
	داخل المجموعات	195	123.53	0.63		
	المجموع الكلي	199	145.36			
إعادة الأرقام الدرجة الكلية	بين المجموعات	4	123.80	30.95	12.58	0.001
	داخل المجموعات	195	479.70	2.46		
	المجموع الكلي	199	603.50			
ترتيب الصور	بين المجموعات	4	10.51	2.63	1.74	غ.د
	داخل المجموعات	195	295.05	1.51		
	المجموع الكلي	199	305.56			
التعقب (جزء أ)	بين المجموعات	4	1900.86	475.22	35.73	0.001
	داخل المجموعات	195	2593.33	13.30		
	المجموع الكلي	199	4494.19			
التعقب (جزء ب)	بين المجموعات	4	3568.29	892.07	28.90	0.001
	داخل المجموعات	195	6084.51	31.20		
	المجموع الكلي	199	9652.80			

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Two way ANOVA

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المتغيرات	مصدر التباين	مجموع المربعات	درجات الحرية	متوسط مجموع المربعات	قيمة (ف)	الدلالة
الذاكرة البصرية	استخدام اليد (أ)	68.642	1	68.642	47.17	0.001
	التاريخ الأسري (ب)	1.859	1	1.859	1.278	غ.د.
	أ x ب	9.881	1	9.881	6.791	0.01
	الخطأ	285.20	196	1.455		
إعادة الأرقام للأمام	استخدام اليد (أ)	25.178	1	25.178	25.70	0.001
	التاريخ الأسري (ب)	0.373	1	0.373	0.381	غ.د.
	أ x ب	0.0001	1	0.0001	0.006	غ.د.
	الخطأ	191.97	196	0.979		
إعادة الأرقام للخلف	استخدام اليد (أ)	15.417	1	15.417	23.44	0.001
	التاريخ الأسري (ب)	0.00001	1	0.00001	0.011	غ.د.
	أ x ب	0.233	1	0.233	0.355	غ.د.
	الخطأ	128.91	196	0.658		
إعادة الأرقام الدرجة الكلية	استخدام اليد (أ)	80.483	1	80.483	30.78	0.001
	التاريخ الأسري (ب)	0.522	1	0.522	0.200	غ.د.
	أ x ب	0.287	1	0.287	0.110	غ.د.
	الخطأ	512.38	196	2.614		
ترتيب الصور	استخدام اليد (أ)	2.114	1	2.114	1.421	غ.د.
	التاريخ الأسري (ب)	9.653	1	9.653	6.427	0.05
	أ x ب	0.833	1	0.833	0.560	غ.د.
	الخطأ	291.64	196	1.488		
التعقب (جزء أ)	استخدام اليد (أ)	1612.3	1	1612.329	121.2	0.001
	التاريخ الأسري (ب)	3.629	1	3.629	0.273	غ.د.
	أ x ب	1.788	1	1.788	0.134	غ.د.
	الخطأ	2606.6	196	13.299		
التعقب (جزء ب)	استخدام اليد (أ)	2997.3	1	2997.357	96.38	0.001
	التاريخ الأسري (ب)	32.015	1	32.015	1.029	غ.د.
	أ x ب	0.00001	1	0.00001	0.000	غ.د.
	الخطأ	6095.3	196	6095.355		

تابع جدول (5)

تابع جدول (5)						
المتغيرات	مصدر التباين	مجموع المربعات	درجات الحرية	متوسط مجموع المربعات	قيمة (ف)	الدلالة
	استخدام اليد (ج)	19.93	4	4.98	8.40	0.001
	أ x ب	0.23	1	0.23	0.39	غ.د.
	أ x ج	1.21	4	0.303	0.50	غ.د.
	ب x ج	1.59	4	0.40	0.17	غ.د.
	أ x ب x ج	0.538	3	0.179	0.301	غ.د.
	الخطأ	107.36	181	0.593		
إعادة الأرقام الدرجة الكلية	الجنس (أ)	6.97	1	6.97	3.34	غ.د.
	التخصص الدراسي (ب)	41.23	1	41.23	19.77	0.001
	استخدام اليد (ج)	110.85	4	27.71	13.29	0.001
	أ x ب	2.77	1	2.77	1.33	غ.د.
	أ x ج	7.41	4	1.85	0.891	غ.د.
	ب x ج	16.63	4	4.16	1.99	غ.د.
	أ x ب x ج	2.34	3	0.78	0.374	غ.د.
	الخطأ	377.43	181	2.085		
ترتيب الصور	الجنس (أ)	24.63	1	24.63	19.01	0.001
	التخصص الدراسي (ب)	0.52	1	0.52	0.401	غ.د.
	استخدام اليد (ج)	18.50	4	4.62	3.59	0.01
	أ x ب	1.04	1	1.04	0.81	غ.د.
	أ x ج	15.29	4	3.82	2.97	0.05
	ب x ج	12.25	4	3.06	2.38	غ.د.
	أ x ب x ج	1.373	3	0.458	0.355	غ.د.
	الخطأ	233.31	181	1.289		
التعقب (جزء أ)	الجنس (أ)	0.00001	1	0.00001	0.00	غ.د.
	التخصص الدراسي (ب)	317.79	1	317.79	27.96	0.001
	استخدام اليد (ج)	1622.3	4	405.60	35.69	0.001
	أ x ب	1.27	1	1.27	0.122	غ.د.
	أ x ج	89.01	4	22.25	1.95	غ.د.
	ب x ج	147.49	4	36.87	3.24	0.01
	أ x ب x ج	27.557	3	9.186	0.808	غ.د.
	الخطأ	2057.2	181	11.36		
التعقب (جزء ب)	الجنس (أ)	269.17	1	269.17	10.49	0.001
	التخصص الدراسي (ب)	359.01	1	359.01	13.99	0.001
	استخدام اليد (ج)	3202.7	4	800.70	31.21	0.001
	أ x ب	18.26	1	18.26	0.71	غ.د.
	أ x ج	41.86	4	10.47	0.408	غ.د.
	ب x ج	216.63	4	54.158	2.11	غ.د.
	أ x ب x ج	79.831	3	26.611	1.307	غ.د.
	الخطأ	4644.1	181	25.658		

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(1993 Al-Biali, 1993)

(Cornish, 1996; Annett, (1992)

(Soliman, 1982)

1989; Al-Biali, 1993

(Aaron, 1996)

Cognitive presentations

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(Mehta & Newcomb, 1991; McKlevie & Aikins, 1993; Richardson, 1993; Cornish, 1996)

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(Aaron, 1996)

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(Levy,1985)

(Reuter et al., 1990; Bryden et al., 1994; Springer & Deutsch, (1999; Rotenberg & Weinberg, 1999; Marnat, 2000,p. 402; McManus, 2002,p. 178)

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(Jonides et al., 1993; Reuter et

.al., (2000)

(Annett, 1992; Jones & Martin, 1997;

Sinclair, 2001)

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(Kathleen & Eliassen, 1998)

(Mehta & Newcomb, 1991, (McKlevie & Aikins, 1993; Richardson, 1993; Cornish, 1996)
(McManus, 2002)

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Reversed laterality

.Familial Sinistrality

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(Witelson & Goldsmith, 1991; Cornish, 1996; Kathleen & Eliassen, 1998)
(Annett, 1992)

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(McManus, 2002)

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Footedness

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(Aaron, 1996)

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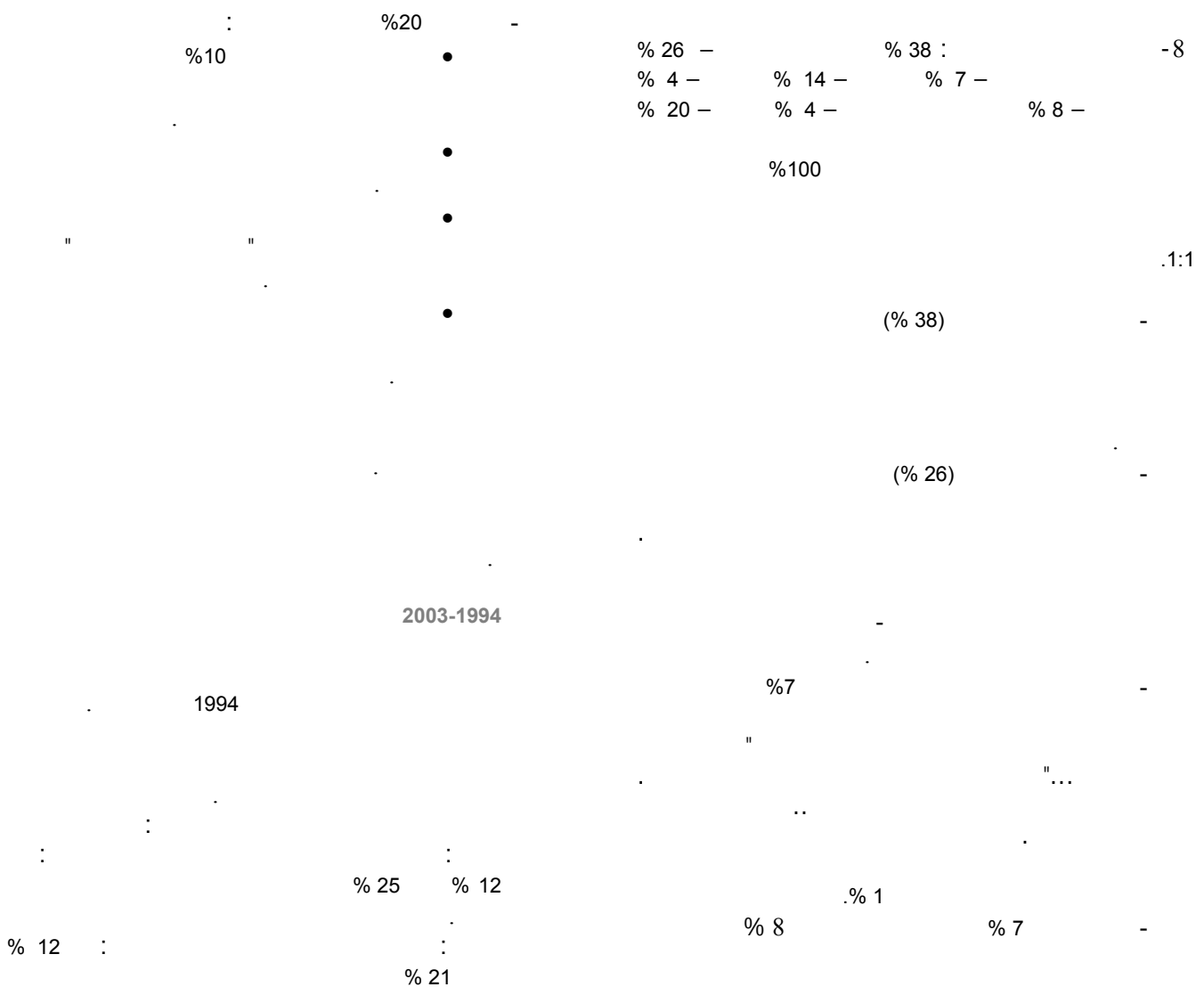
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الإيمان : نذير على مسار التطور البشري - المعنى والدلالة (قراءات في لغة الظاهرة)

أ.د. يحيى الرخاوي - الطب النفسي - القاهرة / مصر

بريد إلكتروني : email@rakhawy.com

قبل المقدمة : ظاهرة الإيمان هي ظاهرة بيولوجية، نفس-اجتماعية، مرتبطة بخصيكية الوعي (الفردى والجماعي)، وهي مرتبطة أيضا بالفترة التاريخية التي تظهر فيها، وبالظروف الاجتماعية والاقتصادية التي تميز تلك الفترة، كما أن لها علاقة مباشرة وغير مباشرة بالاستعداد الوراثي (ليس قاصرا على الاستعداد للإيمان بالذات)، ولها دلالات ومعان مختلفة باختلاف الأفراد والثقافات.

أصبحت الظاهرة تمثل "ثقافة فرعية" احتجاجية في ظاهرها، وإن كانت محلها سلبية، احتجاجية ربما في مواجهة الاغتراب المنمادى في الحياة الكمية التي تروج لها الشركات العملاقة العابرة فوق-المركزية الأعلى، المناهضة للسلطات التقليدية.

الإيمان هو لعب ضامر بالوعي البشري، لعب متحيز مصطنع، يحقق تغييرا نوعيا، مؤقتا، سرعان ما يتقلب إلى احتياج لحوج للكرام، ومن ثمّ النماذى فيه، على حساب مسنوبات الوعي المنفلق المنتج المنكامل .

ظاهرة الإيمان ليست مرادفة لنعاطى المخدرات، حيث ألها مرتبطة بنعاطى أى مادة توثر في الدماغ فتغير الوعي سواء كانت محلبة أمر متيرة .

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غياب علم النفس عن الفكر العربي ؟

د. عدنان حب الله - رئيس المركز العربي للأبحاث النفسية والتحليلية / لبنان

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ماذا يعني الحوار؟ مع من نتحاور؟ هل الحوار هو بين رجال السياسة، أو بين رجال الدين أو ما بين المفكرين أنفسهم؟ حتى لو سلمنا في ذلك هل نتائج هذا الحوار سواء كانت وفاقية، أم خلافية، تؤدي إلى ترجمة عملية في مسار الفكر العربي؟

كلها أسئلة لا يمكن الإجابة عنها إذا أخذنا في الاعتبار مفهوم النفس - النفس العربية - التي تحمل في طياتها ما هو قابل للتطور، وما هو من فوض ليس لهدم قناعة العقل، وإنما لأسباب موروثية جعلت من الكبت جداراً منيعاً يحول دون أي تطور. والجدار النفسي هو كما نعرف لم يعد محصوراً بعلم النفس - بل أصبحت هذه الكلمة، مندأولة بين الذين يعاطون الشؤون السياسية، انطلاقاً من السادات عندما تكلم عن الجدار النفسي وصولاً إلى تناحر الطوائف وإقامة الحوار لحيي يعترفوا في النهاية، بأن إزالة الجدار الأرضي الذي بدأ بزوال جدار برلين لا بد من أن تسبقه، أو تتبعه إزالة الجدار النفسي. وإذا كان الأول سهلاً ويبدأ بهدم الجدار حجراً حجراً فالثاني أصعب لأنه يتطلب عملاً وجهداً نفسياً كبيراً يبدأ بالذات قبل أن يطالب الآخر بالمقابل.

وعلى رغم اعتراف القائلين بهذه الحقيقة، إلا أنهم كما يبدو، يفتقدون إلى المعرفة والوسائل التي توفر تحرير النفس من عقلاها لكي تتوجه وتتصلح مع الآخر الكبير.

وحين لا يبقى هذا النقاش محصوراً في التطوير، لا بد من الإشارة إلى بعض المسائل العالقة والتي تتطلب منا التفكير وإعادة النظر كي نستطيع أن نواصل إلى مرافقة حضارة بدلاً من العودة إلى سلفية كانت صالحة لاسلافنا. وإذا لم نستطع الإجابة، فما لا شك فيه، كما أثبتت الأحداث وسشتت لاحقاً أن العنف سيكون نصيبنا، لأنه سيكون بمثابة الإجابة الوحيدة.

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حول مستقبل العلوم الإنسانية في الوطن العربي

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تعرض العلوم الإنسانية مراراً لمراجعات نقدية مكثفة. وهذه المراجعات ليست من نوع الترف النظري-الفكري كسابقتها. فهي مطروحة كضرورة ملحة، وحيوية للحفاظ على الفعالية الإجرائية لهذه العلوم. إذ أن استمراريتها مرتبطة بالتعديلات المنهجية والفكرية التي يمكن لهذه المراجعات إدخالها لتحديث هذه العلوم وتخليصها من شوائب تكاد تقضي على فعاليتها. ولهذا يمكن لهذه العلوم الاستمرار في البرهان على فعاليتها وقدرتها على تحسين مستوى اللياقة النفسية والفكرية للإنسان. حيث الإنسان هو طموح هذه العلوم وموضوعها. والواقع أن ثورة الاتصالات والمعلومات قد تسببت في إخراجات كثيرة ومتعددة الصعد لمناهج العلوم الإنسانية وأيديولوجياتها. مما اقتضى المراجعة.

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أ.د. محمد أحمد النابلسي

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AFTER 11 SEPTEMBER EVENTS. : REVIVAL OF THE NEED TO LISTEN TO EACH OTHER

PR. YAHIA RAKHAWY – PSYCHIATRIST – EGYPT
EGYPT, J. PSYCHIAT. 25: 1&2 JANUARY & JULY 2002

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The relation of culture to psychiatry is not simply like the relation of culture to other branches of medicine. Psychiatry is a medical profession that deals with the structural basis of existence, the goal of life and the how of re-channeling suffering of handicapped individuals to live up to their potentialities and aspirations. Cultural studies in psychiatry should neither be restricted to comparing epidemiological figures of specific disorders in a particular territory nor to delineating some different delusional contents or particular outcome of certain syndromes. Such activities are simply describing superficial differences rather than making use of profound diversities.

To establish some genuine converging synthesis between cultures (transversely) and civilizations (longitudinally), what is needed is to uncover basic structural differences in the how of conceptualization along the way towards complementary synthetic integration between different groups of people sharing common human interest. This lies very deeply in the structural and biological make up of different cultures and individuals. Besides, this is basically bound to both language and religion as the most significant and available bio-existential structures of human beings. Language is the basic biological structural configuration that judges our perception to ourselves and the world in health and disease. Religious stands and attitudes are unconscious structures that could not be identified or managed simply through some declared beliefs influencing certain overt behaviour. 'Islam,' for instance, is not a system of beliefs with different unfamiliar rituals as a western man may perceive. **It represents another way of being in the world.** This could not be judged at a distance or properly evaluated through a casual accident, biased claims or emotional prejudice. This file has been opened lately and could have its negative influence on whatever discipline dealing with cultural differences. Psychiatric practice would come among the top ones.

Reading September 11th's (2001) incidence, in teleological language, could reveal indispensable cues to know better about the current status of human race. It could be perceived as some real, serious, **protest against dominant discipline.** Simultaneously, it could indicate a definite **threat to such dominant level of existence** (including dominant brain organization). On the other hand, the so called 'new world order' proved to be either: 'in the make' or but an 'illusion'. Human race is passing a critical evolutionary impasse. Power system is getting more and more blind, chaotic and deleterious. The recent technological devices proved to be at least double edged.

The ongoing consequences of September's events are proving to be more and more negative than positive. For instance, the international regulations and laws have been overlooked, put aside or neglected; the dominant partner is becoming much more dominant, hence more blind. More and more prejudice has been cultivated on both sides and the influence of secret authorities (Lobbies and Mafia) seems to be augmented.

On the other hand, the hopeful positive results depend on the how of management of such consequences. There is a real chance for revision of current human illusions including

the mutilation and solidification of old religions, as well as the risky, false and new 'pseudo-religions'. The idealized pseudo-freedom called democracy as well as the literal human rights would be reevaluated. Creative search for better alternatives would be activated. The so-called 'quality of life', as well as the ill-defined common goal of human existence would be re-defined or at least re-considered. Promoting human growth at individual level and human evolution at race level **would find better systems apt to fulfill better achievements that are essential for promotion of human evolution.**

All such hazards, as well as hopes, have their direct or indirect influence on psychiatric practice. Perhaps globalization of psychiatry has preceded globalization at large. The world wide invasion by the vocabulary innovated by the DSM-IV, and to a less extent by the ICD -10, has been going on throughout the last three decades. The side-effects of such tendencies have resulted in nullifying cultural differences, leading to more and more hazardous effects that devalue the claimed benefits of agreeing upon common diagnostic labels. Psychiatrists have succeeded to agree much upon what they do not really know.

Four terms may interfere with each other when dealing with the issue of globalization, they are not the least synonymous. These are: globalization, internationalism, totalitarianism and trans-nationalism (up to trans-continental). 'To Globalize' means to make worldwide, in scope or application. 'internationalism' refers to what is across nations (two or more). 'totalitarianism' relates to central control (extremely centralized authority), 'trans-nationalism' refers to extensions above or beyond nations and 'trans-continental' is what transcend even continents.

Perhaps it is necessary to identify two types of globalization: authoritarian globalization, (what mighty authorities are after, becoming a sort of totalitarianism) and humanistic globalization aiming at objective synthetic convergence of diversities. The latter is essentially related to the degree of orientation of cultural differences, hence working through them and converging towards new synthesis.

In spite of the many alternative meanings of the word 'culture' and the occasional equating it with 'civilization' as synonym, it is necessary to delineate one from the other. Culture is an unconscious network of a holistic mode of existence of a particular group of people at a particular moment of history. Civilization is not simply a relatively high level of cultural and technological development or refinement of thought, manner or taste. For instance, to consider a specific culture to be

considered as civilization, it should represent a specific different existential stand in life. Revelation of such stand in everyday life by most individuals related to this culture, a tendency to export (to market) it to others (all over the world) and efficient methods and tools to fulfill such goal should be considered in defining 'civilization'. Besides, test of efficacy and test of time would prove that such stand and tendency are promising something better to human beings which is apt to propagate worldwide.

Moslems, nowadays, do not fulfill such criteria to make their hundreds of sub-cultures a unified competitive civilization. Islamic civilization is both a historical fact and possible alternative potential. Genuine basic principles of Islam emphasize practicing monotheism in everyday life all the time as a tool for cultivating genuine creative personal freedom. Overvaluing self control and judgment, in spite of, and along with all written rights and disciplines, are essential to be a Moslem. The direct relation to nature and macrocosms in a biorhythmic open ended harmony is coloring most praying and religious practices.

As such, nobody, including psychiatrists, could claim that he is dealing with what he labels 'Islamic Civilization'. The above mentioned principles do not currently or predominantly exist. What is available or presenting all over the world are but sporadic different subcultures having the same label 'Al-Islam'. What is needed is to find out current real differences between cultures in order to get use of whatever positive aspect of each that may be useful for all others.

In psychiatry, for instance, through respect of other's cultures (and civilizations), methodologies, and clinical results, observations and special art of healing, some possible integration of knowledge and experiences could be achieved. In our practice, especially that of relatively senior psychiatrists, some possible hypotheses re-emerge once and again. They need verification by different methodologies. For example, controlled studies (including double blind techniques) could prove to be more illusive than narrating clinical results. Clinical experience does not go parallel with the results of the so called scientific research. Individual variations between psychiatrists are wide enough extending to their use of drugs. The so called algorithm is becoming more handicapping for creative management of individual patients. Diagnostic labels are more and more replacing unique individual identification of each patient. Most practitioners are coming back to emphasize that it is essential to know the state of the disorder than to label it by an agreed upon particular term (name or label). It is more important to know the how of the disorder than to know the why of it. It is more useful to know the pathology of the biorhythmic pulsations (periodical, intermittent, remittent) than to know the possible micropathology of limited neurotransmitters. It is more important to follow-up by clinical monitoring denoting which brain organization is dominant than by the blood level.

It is more courageous to expect, assimilate and make use of the relapse rather than to follow a strict 'prevention program' with the possible deadening of affective resonance and creative potentialities.

We are in due need to listen to each other instead of claiming destructive conflicts between civilizations, while declaring pseudo-equality and unification.

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موافق

LE 11 SEPTEMBRE 2001 DES ARABES

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Le monde a changé depuis le 11 septembre 2001.

J'ai eu la surprise indescriptible de regarder en direct les attentats grâce au décalage horaire et j'ai vu en direct non seulement le deuxième immeuble des deux tours se faire encastrier par le deuxième avion mais une heure ou un peu plus après, la chute des deux constructions, dans des images d'apocalypse.

Je ne suis pas allée travailler ce jour-là et j'ai passé des heures à réfléchir et à méditer devant ces scènes depuis Casablanca ; j'étais envahie par des émotions, des sentiments et des idées aussi nombreuses que contradictoires et prégnantes.

L'incroyable et l'inattendu s'était produit sous les yeux du monde entier, ahuri, stupéfait ou content, bouleversé ou traumatisé.

Pour avoir pris bien longtemps auparavant un déjeuner au sommet de l'une des tours, j'étais dans un connu qui me donna fort à penser. Dans les explications offertes aux visiteurs, il était décrit comment les deux tours oscillaient, chacune sur son axe, d'un mètre par jour pour tenir l'équilibre de ce colossal ensemble architectural, le plus en vue dans le monde. J'avais, par ce jour de beau temps, éprouvé des sensations vertigineuses dans le ciel, ivresse à être dans une réalisation aussi extraordinaire, hommage à ce que l'habilité humaine peut arriver à faire.

Vertige et aussi émotion devant le nombre de morts en accident du travail ; je ne perdais pas de mémoire cette fonction aussi admirable de l'humain à sa tâche livrée pour réaliser ce qui est la gloire et la fierté de tous les autres hommes. Je me souvenais que c'était les Indiens américains qui sautaient comme des chats en montant les structures métalliques de plus en plus haut en vissant des milliers et de milliards de vis pour arriver à cette hauteur absolument extraordinaire au-dessus du niveau de la mer...

Les ambivalences et les contradictions s'emparèrent de moi pour tenter de mettre de l'ordre dans les pensées qui s'imposaient à moi...

J'avais donné à Paris deux mois auparavant à une maison d'édition un manuscrit d'un texte intitulé « Lettre ouverte à l'Occident » traité par le plus grand mépris car les vérités qu'il contenait n'étaient pas dicibles en Europe...

Ce texte était « né » dès octobre 2000, avait été amendé après les Sommet de Gênes et celui de Durban qui n'avait pas été soulevé car le livre était parti avant la tenue de cette dernière rencontre sur le racisme. Il ne faudra jamais oublier que le sommet de Durban en Afrique du Sud s'est tenu quelques jours seulement avant le 11 septembre 2001...

Je me retrouvais en situation de révolte, de découragement et de questionnements assez insolubles. Ayant vécu l'impérieuse loi des éditions occidentales et les positions des journalistes les plus en vue en France concernant ce qu'ils attendaient que je leur dise, je me retrouvais vaincue une fois de plus par la force de l'Occident seulement dans et ma faiblesse à exprimer ce que je voulais

dire non le monde arabo-musulman mais à l'Europe et à tous les Occidentaux. J'ai dit non à tous les poncifs traînant par exemple dans toutes les soi-disant études sur le monde arabe et musulman et concernant la vision occidentale de la femme arabe, à tous les clichés en psychopathologie qui veulent considérer l'hystérie comme la principale maladie psychique des femmes arabo-musulmanes, à tous les schémas réducteurs qui empêchent justement le tissage de liens différents mais égalitaires entre les mondes orientaux et occidentaux.

Mais mes propos sont très difficiles à faire entendre pour le moment. Si Noam Chomsky (« Cette Amérique qui n'apprend rien » Le Monde, 22 novembre 2001p. VIII) peut les écrire, c'est parce qu'il est Américain et que cela lui donne la puissance, curieusement même pour se dresser contre son propre pays. Moi, je suis trop faible, aussi faible que le monde dont je proviens et dans lequel fut inventer la bombe humaine, elle se fait se sauter car l'Occident refuse d'entendre ce que nous avons tous à lui dire, Arabes, musulmans, intellectuels du tiers monde, Américains du Sud, pauvres, opprimés, femmes tuées pour être nées à la mauvaise place, vies laminées et détruites par l'absence de liberté et des libertés dans nos pays qui ont horreur de la liberté.

Comme exemple on peut prendre mon ouvrage « Une psychiatrie moderne pour le Maghreb » resté au fond d'un tiroir d'éditeur à Paris pendant plusieurs années car le sujet n'intéresserait pas, d'après le responsable de collection, les lecteurs français. On aurait voulu que je traite de l'hystérie féminine au Maroc et au Maghreb... jusqu'à la mort du professeur de psychiatrie Mustapha Boucebsi tué car propageant en Algérie des idées trop modernistes. Faisant valoir que nous allions être tués jusqu'au dernier si nos idées ne se propageaient pas, le livre a enfin été édité et doit sa sortie à la mort de Boucebsi. Ainsi, on voit que les mauvaises qualités de la compréhension entre les individus et les peuples ont opéré des désastres d'abord dans les champs de la cognition, du savoir, de la culture, de la capacité des intellectuels à établir des relations utiles et fécondes entre les sociétés et les peuples.

Ce lent travail d'accumulation de la négligence des pays du nord vers les pays du sud a provoqué la catastrophe du 11 septembre 2001, et c'est une catastrophe anthropologique car elle n'a fait que se faire creuser un fossé plus démentiel entre des cultures trop différentes qui ne trouveront peut être plus les moyens humains du rapprochement. Hutton, Américain lui aussi, a écrit un livre que les Occidentaux s'accordent à trouver remarquable, « Le choc des cultures »

Edward Saïd, encore un Américain par l'adoption, Palestinien d'origine, en a fait une critique remarquable (in « Le Monde » octobre 2001) et ce spécialiste de la relation entre l'Orient et l'Occident, nous fait apercevoir, une fois de plus l'euro-péo et l'américanocentrisme. On ne peut négliger non plus son ouvrage fondamental paru en 1978 " L'Orientalisme,

L'Orient vu par l'Occident ") un classique qui abordait les impossibilités de rencontre entre engeances arabes et orientales et Occidentaux attachés en premier lieu à rêver sur des créations fantasmagoriques sorties plus de leur imagination que de la réalité des déserts, des oasis et des yeux noirs des femmes lascives dansant dans leurs sept voiles. En général, les points de vue et les opinions des uns et des autres sont radicalement différents voire opposés. Pour l'Occidental, je suis Marocaine donc Orientale mais Maroc en arabe veut dire Occident, ponant, couchant et je me vois affublée de qualificatifs aussi absurdes que si je traitais une Portugaise de Lapone ou d'Ukrainienne blanche !

Cette cécité des Occidentaux concernant les autres cultures est tout simplement un mépris pour l'Autre et une indifférence dans les meilleurs cas. Connaissant six langues uniquement parce que j'ai été colonisée et ayant appris la géographie de la France à la place de celle de mon pays, le Siècle Classique au lieu des oeuvres complètes de Jalal Dine Roumi ou de la grandeur des Abbassides, sachant tout de l'Europe et des États-Unis, puis du monde grâce à cet éclatement qui a fait que je ne pouvais rester bi-culturée seulement et uniquement cela, ce n'était pas ou plus assez, j'ai investigué dans toutes les cultures du monde. Et bien m'en a pris sous la férule du très grand maître Georges Devereux, lui-même un déchiré entre les cultures comme moi : Devereux était le créateur mondial de la discipline dite « Ethnopsychiatrie » et de cela m'est resté un projet de « re » connaissance de tous les Autres et une revendication égalitariste entre tous les peuples et toutes les culturocivilisations, c'est ainsi que je voudrais télescoper les deux concepts car une culture correspond obligatoirement à une civilisation et réciproquement. Les cultures ne sont pas univoques et les civilisations ne sont ni étanches ni parfaitement isolées et autarciques.

Quelque chose entre les êtres humains a circulé d'un bout à l'autre de la planète et si l'Amazonien et l'Aborigène semblent très différents voire autres, si le cannibale et le Zoulou, le Pygmée et le Peuhl sont noyés dans la même ignorance égalisatrice, ils ont à voir avec l'Indien des Plaines si cher à Devereux, celui qui a été exterminé par les Américains pour qu'ils occupent tout le sol mais surtout, surtout, circule en eux tous une âme humaine, la même pour tout le monde. C'est le même Indien des Plaines qui a grimpé au mépris de sa vie les hauteurs vertigineuses des deux tours du World Trade Center pour les construire. Et Colin Powell oublie qu'il est noir et ne s'embarrasse pas d'états d'âme pour décider, en consensus, de la mort des « soft targets » ou cibles molles ou douces dans les « side effects » ou effets latéraux de la guerre contre l'Afghanistan qui tue des milliers de civils directement par les armes et des millions par la faim et toutes les sortes de déprivation. Mais les anciens esclaves et opprimés ne deviennent-ils pas les plus féroces oppresseurs, les plus zélés et les plus déterminés ? Ce sont des processus psychologiques inconscients. Ceux qui opèrent en madame Condoleezza Rice, conseillère de G. W Bush, elle qui avait été victime des mesures d'ostracisme exercées contre les Noirs quand elle était enfant. Sa mère la consolait en lui disant de rester à sa place mais de travailler surtout pour devenir présidente des États-Unis, ce que la sage petite fille fit et on connaît son extraordinaire réussite. Elle en a oublié qu'elle fut aussi méprisée que les femmes arabes, que les femmes afghanes, que les esclaves, ses ancêtres pas si lointains...

L'Occident va de mal en pis dans son « misunderstanding » ou « mécompréhension » des autres s'il ne change

radicalement d'« entendement » des Autres, de tous les Autres.

Que l'on ne s'y trompe pas : aux États-Unis et en Europe, Oussama Ben Laden est un terroriste, le terroriste le plus recherché de la planète. Mais aux yeux des deux cent cinquante millions d'Arabes et du milliard deux cent millions de musulmans, dans leur écrasante majorité, Ben Laden n'est pas un terroriste mais le héros et le sauveur de la nation et du peuple arabo-musulman écrasé par le racisme des pays riches, par le mépris des pays démocratiques, par la majeure partie des individus qui peuplent le monde dit libre.

Les Occidentaux l'ont porté au pouvoir de ce qu'ils appellent le terrorisme quand les Américains l'armaient contre l'ex-U.R.S.S. pour la chasser d'Afghanistan. Ils en ont fait un immense personnage contre lequel toute la planète blanche et riche s'arme et de notre point de vue arabe et musulman, nous avons beaucoup de pitié et de mépris vis-à-vis d'une coalition contre un individu terré au fond de grottes et de cavernes qui fait trembler le monde libre par sa seule volonté, paranoïaque.

Ben Laden, pour moi, femme arabe, ne peut être porteur de projets de société valables s'il arrivait même à anéantir tout l'Occident. Je refuse de vivre comme moitié d'un être humain, dégradée et anéantie par les hommes de ma propre engeance. Ce projet de déchéance de la femme est principalement agissant dans la cosmogonie anthropologique de Oussama Ben Laden qui ne craint d'épouser une enfant quand bien même c'est son père, le mollah Omar, que la lui a offerte et réciproquement, il offre sa fille, une enfant, au même mollah Omar créant des cercles d'inceste, d'homophilie et de pédophilie entre eux que personne n'a seulement songé à expliquer et divulguer.

Ces filles de quinze ou seize ans ont continué à payer pour l'aberration des hommes arabes et musulmans qui ne conçoivent leurs rapports aux femmes que dans une intégrale domination des femmes par les hommes ; cela doit changer et si des fous furieux comme Ben Laden prennent le pouvoir, cela en est fait de la nation arabe et musulmane pendant des siècles. Les Arabes et les musulmans pensent que l'évolution de la condition des femmes n'est qu'une errance de type occidentale et une aventure impulsée par le colonialisme qui doit s'arrêter pour que les femmes retournent sous le voile, dans la maison, sous la tutelle de tous les hommes, de la famille, de la société et de l'état.

Il ne faut pas oublier et, un monsieur comme George W. Bush est inapte à le comprendre, (Ses conseillers ignorent superbement l'arabe comme langue, la culturocivilisation arabo-musulmane comme un système tout juste bon autrefois à faire des films hollywoodiens dans lesquels des Américaines brunes dansent lascivement les fameuses danses des sept voiles...) Oussama Ben Laden a créé une idéologie et un système de pensée et de comportements qui lui survivra. Les Arabes et les musulmans tragiquement révélés à leur misère et à leur insatisfaction ont appris à se suicider en se transformant en bombes humaines et kamikazes capables de se jeter dans n'importe quelle aventure en harmonie avec leurs croyances.

Il y a aujourd'hui dans le monde arabo-musulman une série de Ben Laden car l'Occident a tué Mahdī Ben Barka, Marocain, Patrice Lumumba, Congolais, Machel Samoa, Mozambicain, Hamilcar Cabral, de Guinée-Bissau, Sira Wiwa, Nigérien, Che Guevara, Colombien, Salvador Allende, Chilien, tous gauchistes qui rêvaient d'un monde plus vrai, plus juste, plus égalitaire entre les hommes et les femmes sur toute la planète.

Ils ont été tués car l'Europe et les États-Unis avaient peur du réveil des peuples et leurs leaders les plus adéquats ont été éliminés un par un tandis que les tyrans, les dictateurs, les

satrapes et les personnages les plus corrompus se sont arrogés les pays, les gens, les richesses et le pouvoir de vie et de mort sur les êtres les plus courageux et les plus à même de sauver humainement leurs contrées et une idée juste du monde et de la vie. G d'Estaing donnait du « mon cousin » à l'horrible Jean Bedel Bokassa, et Papa Doc martyrisait Haïti avec sa milice Les Tontons Macoutes sous les yeux des Américains et à leur porte... tandis que son fils, le repoussant Baby Doc est toujours réfugié en France. Ces mêmes « puissances » et c'est ainsi qu'elles se revendiquent, ont causé la mort de 11 millions d'Africains entre le 15 et le 19^e siècles dans l'horrible absurdité de l'esclavage pour onze millions de déportés de plus, les ancêtres de ceux que l'on nomme aujourd'hui les Afro-Américains. Pour que la justice soit faite et que tout soit dit, un jour, il ne faut pas oublier que les Arabes aussi, furent de rudes négriers. Les génocides se sont faits avec la complicité de l'Amérique dont le président Clinton est allé présenter des excuses pour le génocide du Rwanda qu'il a délibérément laissé faire, 900 000 à deux millions de morts, d'avril à juin 1994. Mais l'Occident n'a pas demandé pardon à l'Afrique au Sommet de Durban pour l'esclavage car il ne voulait rien payer, ce qui aurait pu se produire s'il reconnaissait l'atrocité des massacres et des traitements subis par les Africains lors de la traite des Noirs.

Les Occidentaux ont privilégié les royaumes les plus pourris de la création, en Péninsule Arabique, et ces mêmes chantages de la démocratie et du progrès humain traitent avec la famille des Al Sabah au Koweït qui possèdent ce pays, gouvernent comme au moyen âge florentin sans l'esprit de la Renaissance et sans l'art et le raffinement des cours italiennes, interdisant aux femmes de voter et de se présenter aux élections. En somme des bêtes ou des serves.

Ce sont ces fameux Al Sabah qui ont été défendus en 1990-91 lors de la Guerre du Golfe, soi disant parce que ce pays – une petite excroissance autrefois province de l'Irak-, dépecée par les Anglais quand ils occupaient la région et faisaient ce qu'ils voulaient, était un état souverain et inviolable.

Ben Laden exécute ces pays et a juré leur ruine et on est obligé de se rendre à l'évidence : il a raison.

Mais là où le bât blesse cruellement, c'est que, ce qu'il préconise à la suite de la chute éventuelle de ces pays, c'est pire, peut être. Asservissement complet des femmes, réaction fasciste musulmane d'extrême droite, inhibition, interdiction de l'art et de la culture émancipationniste et libertaire ou libératoire. Ceci est un résultat tragique de l'histoire depuis un siècle ou deux pour la cuturocivilisation arabo-musulmane.

Tous nos espoirs de vie meilleure ses sont effondrés avec le 11 septembre 2001 car nous serons pris en étau entre l'Occident qui n'a que la réaction du corps blessé, celle de se défendre par tous les moyens, qui va ignorer les exigences et les nécessités du tiers monde et spécifiquement des Arabes et des musulmans et l'incapacité des chefs arabes et musulmans de dicter une politique démocratique et valable car ils ne se sont tous maintenus au pouvoir que par le meurtre et la rapine depuis cinquante ans.

Si les états arabes et musulmans s'affilient à la tendance internationale dominante et à la ligne de conduite des instances onusiennes, les Arabes et les musulmans, dans leur immense majorité, ne sont pas d'accord avec leurs dirigeants et vivent avec un narcissisme blessé et une personnalité amoindrie et dévalorisée à travers l'histoire, à travers les aberrations de l'histoire récente depuis les luttes d'indépendance et les libérations factices des pays du tiers monde totalement dominés économiquement par les pays riches et puissants. Les périodes de colonisation sont

prolongées par le fait de la guerre toujours perdue de la Palestine contre Israël et depuis la manière effroyable dont a été menée la destruction systématique de l'Irak où Saddam Hussein est vécu lui aussi comme un héros et un grand de la nation arabe. Qui sait en Occident que Sabra et Chatila sont deux camps palestiniens où deux mille civils, en majeure partie femmes et enfants ont été massacrés sans représailles par les Israéliens ? Il faut le spécifier le lendemain de deux attentats qui font trente morts en Israël, le 1^{er} décembre 2001, au grand dam de la communauté internationale qui crie au scandale.

Le problème atroce est que la peau d'un Noir, d'un Arabe ou d'un musulman ce n'est rien, ça ne vaut rien, même pas un papier dans la presse avec la rubrique des chiens écrasés. Deux bombes humaines se sont encore sacrifiées pour faire entendre au monde le drame du peuple palestinien. Le calcul des morts de la deuxième Intifada donne, pour une année, deux cents morts israéliens pour huit cents palestiniens. Les chiffres parlent seuls ; on oublie aussi que cette seconde Intifada, « La Guerre des Pierres » a débuté quand un enfant palestinien a été tiré comme un canard par les soldats israéliens...

L'acharnement de l'Amérique à détruire l'Irak, par ailleurs, est immonde et effroyable. Cette destruction massive a été justifiée par une désinformation absolue des masses occidentales et des instances aptes à défendre des causes justes. Encore une fois l'Irak n'est pas ce que la propagande américaine a voulu nous faire croire à travers C.N.N. et ses affabulations.

C'était l'état arabe le plus avancé, le plus laïc, le plus nanti en chances de progrès. L'Irak a fait la guerre avec l'Iran à la place de tous les états arabes de la région en raison d'une haine légendaire entre Arabes et Persans et pour des problèmes actuels d'hégémonie, de main mise sur les lieux saints de l'islam par la seule Arabie saoudite, de pétrole et de relations avec l'Occident que l'Iran islamiste a appelé le Grand Satan. Quand la guerre fut finie, millions de morts, les paiements promis pas les royaumes arabes ne furent pas faits à l'Irak qui avait supporté seule l'effort de guerre. On sait la suite. C'est un règlement de compte entre Arabes dans lequel les U.S.A. ont mis le nez pour des raisons économiques et de pétrole, parce que l'Irak était au bord de la fabrication de la bombe nucléaire, parce que le niveau scientifique du pays était très avancé pour un pays arabe et menaçait Israël, parce que les Arabes ne peuvent être aux yeux des Occidentaux que ceux qui donnent leur pétrole, encaissent quelques dollars par baril et se taisent en laissant leurs capitaux ainsi volés à la nation arabe et musulmane fructifier en Occident.

Aux dernières nouvelles, les magnats et nababs arabes sont en train de faire fuir leurs capitaux vers... la Suisse qui se frotte les mains. Les Américains ont cru bon de confisquer les avoirs de tout le monde arabe en arguant de masses d'argent servant à la structuration du terrorisme. Ils oublient seulement le terrorisme des états du Golfe Arabique sur leurs populations et l'état qui y est fait aux femmes, cet état monstrueux dont tout le monde se moque, au fond.

Nous contestons, nous Arabes libres, que l'argent du pétrole et des recettes du pèlerinage à la Mecque ne soit pas redistribué entre tous les Arabes et les musulmans. Si ! Les Saoudiens, très généreux, envoient les dépouilles des moutons sacrifiés par deux ou trois millions de pèlerins pendant l'accomplissement de leurs rites aux pauvres d'entre les pauvres, au Bengla Desh et au Pakistan.

Certains trésors de guerre de nos pays sont gelés par la mort des leaders qui partaient faire ce qu'ils voulaient en Occident et sont décédés en emportant en eux les chiffres des combinaisons des comptes suisses comme certains hommes

d'affaires maghrébines et arabes qui n'avaient pas prévu les cancers et les crises cardiaques et ont laissé des trésors au Panama et dans les paradis fiscaux du monde entier, en fait l'argent de la nation arabe et musulmane. Nous réclamons ces sommes colossales pour bâtir nos hôpitaux, lycées et universités, stades et maisons de jeunes, crèches et orphelinats, maison de la jeunesse et jardins publics, musées et aéroports, laboratoires de recherche, observatoires d'astrophysique –il n'y en a pas un seul pour l'immense territoire arabo musulman, ports et maternités, pour électrifier nos campagnes et dessaler l'eau de mer pour l'immensité aride de nos vingt et un pays arabes ou pour la centaine de pays musulmans...

Ces éléments de la réalité ne sont ici rapportés que pour expliquer ce que les Occidentaux ignorent et ne veulent même pas comprendre pour que les choses changent dans le monde.

Le 11 septembre c'est l'instauration à jamais de la Croisade entre musulmans et Occidentaux comme lors des siècles que l'on croyait finis dans leurs haines et leur agressivité. Les millions d'innocents qui meurent en Afghanistan et en Irak - qui sait en Occident qu'un million d'enfants irakiens est mort en dix ans des bombardements massifs de tout le pays, de faim, de maladies, d'absence de soins car tout manque et surtout les médicaments ? - les millions d'Africains qui n'ont pas de quoi soigner les vingt huit millions de sidéens d'entre eux, les laissés pour compte de toute la planète ne peuvent qu'entrer en guerre contre le gendarme du monde, cette Amérique arrogante qui dans une autre position paranoïaque ne comprend pas que l'on puisse la haïr ?

Chelsea Clinton est très triste de savoir que l'on hait l'Amérique et n'a pas pensé, pauvre petite fille riche, étudiante en Angleterre où elle a fait sa déclaration, qu'elle a bénéficié dans son enfance de deux mandats de son père et a vécu les meilleures conditions au monde qu'un enfant puisse avoir entre dix et dix-huit ans. Pendant ce temps qu'elle a passé à la Maison Blanche combien de petites filles ont été excisées, combien ont été légalement violées à son âge dans le mariage le plus légal, combien sont mortes fautes de soins, de nourriture, de tendresse et de confort ? Son père n'a reconnu que quatre après qu'il est le responsable du génocide du Rwanda et a demandé pardon comme si demander pardon allait ressusciter un ou deux millions de morts ?

Ces drames ont amené des intellectuels comme moi à désespérer de l'Occident et de leurs pays dans une attitude de repli et de suicide scientifique et idéique sachant que rien ne peut changer, les uns nous ignorant ou cherchant à acheter nos capacités et les autres, les nôtres, tuant en nous jusqu'à la volonté de créer encore et de s'exprimer. Pour ma part, je demande justice tant à mon pays et à ma civilisation qu'à l'Occident pour le fait que je n'utiliserai chez moi que le dixième de ce que j'ai consenti à apprendre : il s'agit d'un meurtre commis sur ma Personne, sur mes possibilités, sur mes désirs, sur mon Être philosophique, sur mon sexe saccagé en terre d'islam. Cette tuerie commise sur moi, qui suis un être libre et inviolable au même titre que tout individu arrivé sur la terre, est un scandale affreux et aujourd'hui, je ne peux plus le taire et je redoute en même temps que la revue SUD/NORD qui m'a demandé à deux reprises, la deuxième fois, à la suite de mon hésitation due à l'humeur pessimiste et aux éléments précédemment évoqués, ne soit d'accord pour livrer in extenso ce que j'ai écrit. Cela ne serait qu'habituel entre l'Occident et moi... mais l'heure est grave. Le drame est cette haine forcenée que les Américains sont arrivés à faire

naître chez les Arabes et les musulmans : il n'y a aucun espoir aujourd'hui d'une quelconque possibilité de rapports d'estime et d'amitié entre Arabes et Américains.

C'est une horreur de le dire et c'est une horreur que cela existe. Les tiers mondistes ne veulent pas des Américains, de leur civilisation qui gagne partout et de leur désir de régenter le monde. Arabes et musulmans sont en train de démontrer qu'ils sont les seuls capables de se dresser contre l'hégémonie américaine avec les moyens du faible et du pauvre contre le riche et le puissant.

Les Occidentaux doivent redouter que les futurs Oussama, (le prénom se donne à une infinité de petits garçons qui viennent au monde ! et en arabe il signifie « Le Lion » -qui le sait en Occident ?-) que les futurs rebelles, donc, ne réussissent là où Ben Laden risque d'échouer. Il n'a pas échoué à entrer dans l'histoire.

La radicalisation de l'islam politique dans le monde y compris en Occident (cinq millions de musulmans en France, l'Angleterre base de l'action islamique dans le monde, les Noirs américains, Black Muslims...) conduira à l'édification d'une énorme nation musulmane, un milliard et deux cents millions d'individus, à un projet de société en complète opposition avec le monde occidental.

Ses valeurs, ses buts, ses méthodes sont radicalement opposés à ceux des pays chrétiens, riches et prospères. La bombe atomique de la nation musulmane c'est le ventre de leurs femmes qui repeuplent l'Occident et lui donne les bras dont l'économie a besoin pour continuer à être prospère.

Travaillant en Italie dans le monde de l'émigration, je peux déjà dire que l'autre bombe c'est la présence des migrants en Occident et, il ne faut pas s'y tromper, les descendants de ces gens qui ont quitté la pauvreté, l'absence de démocratie, l'esclavage déguisé, ne sont pas plus bêtes que d'autres. Leurs enfants produiront aussi des génies, des chercheurs, des savants, des artistes, des personnalités de renom. Il faut redouter que la blessure très profonde de leur être ne se transforme en ce que les Occidentaux appellent le terrorisme basé sur tous les sentiments de frustration qui le déclenche et l'explique. Ceux-là font partie de l'Occident sur sa terre.

Moi, je me demande où va cette humanité si elle se complait dans la haine et le mépris. On est là pour l'instant. La solution est l'aisance du monde arabe et musulman, aisance matérielle, morale, et humaine et on en est très très loin. Il faut vivre dans la peur que la radicalisation ne continue encore.

Je suis celle qui dit à l'Occident : les bombes humaines ont aussi des mères qui crèvent de chagrin quand leurs fils explosent. Je suis celle qui dit : la solution n'est plus entre les mains des politiques du monde entier. Elle est entre les circonvolutions cérébrales des penseurs, des philosophes, des psychiatres, psychanalystes, écrivains et poètes. Personne ne les écoute.

Le désespoir est celui des penseurs, philosophes, psychiatres, psychanalystes, écrivains et poètes du tiers monde auxquels leurs collègues occidentaux n'ont pas donné la main pour sauver l'humanité, démolie dans les tours de ses désirs et de ses idéaux.

Ces désirs et ces idéaux sont sous les gravats et sous les décombres des tours jumelles américaines et des taudis d'Afghanistan, sous les tentes des camps palestiniens dressées depuis cinquante ans où vivent ceux qui n'ont plus de terre et de maisons, sous mon crâne livré au vertige des injustices provoquant des révoltes impuissantes, à la vindicte des tyrans et des dictateurs qui ont fait de ma nation un champ de détresse sans lendemain qui chante !

Qui des Occidentaux sait que nous ne vivons pas et encore moins, nous les femmes, les sacrifiées des sacrifiés...

Casablanca, 2 décembre 2001

الجمعية النفسية العراقية

”من أجل عراق ينعم فيه الإنسان بالطمأنينة و يستثمر طاقاته في تطوير شخصيته و المجتمع“

بريد إلكتروني : iraqipa@hotmail.com

2003 / 8 / 19

iraqipa@hotmail.com :

2003 / 10 / 5

2003/8/19 (

2003/10

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THE 6th ARAB CONFERENCE FOR PREVENTION OF S.A

"TOWARDS A BETTER STRATEGY FOR DEALING WITH SUBSTANCE ABUSE PREVENTION"

"THE ARAB FEDERATION FOR NGOS FOR PREVENTION OF SUBSTANCE ABUSE (AFNDA)"

24-25 NOVEMBER, 2004 - GRAND HYATT CAIRO

Website : www.substanceabuse2004.com

المؤتمر العربي السادس للوقاية من الإدمان

نحو استراتيجية أفضل للتعامل مع مشاكل الإدمان

الإتحاد العربي للوقاية من الإدمان

24-25 نوفمبر 2004 - القاهرة - فندق جراند حياة

الموقع على الويب : www.substanceabuse2004.com

Congress Sponsorship

"His Excellency of Prof. Dr. **Amr Moussa** – Secretary General of Arab League

His Excellency of Prof. Dr. **Awad Tag El-Deen** - Minister of Health and Population

His Excellency Prof. Dr. **Hussein El-Gazyry** – Regional Director of World Health Organization of the Eastern Mediterranean

Second Announcement

The Arab Federation of NGOs for Prevention of Substance Abuse (AFNDA) has the pleasure of inviting interested psychiatrist, psychologist, social workers, scientists and NGOs to participate in the conference.

It is regretting to say that the problem of substance abuse is increasing, the age of onset of abuse is getting early in life, the attitude against substance abuse is getting low and the involvement of girls and mothers in smoking and substance abuse is reflecting badly on the overall situation of the drug abuse problem. The governmental and ungovernmental effort needs more partnership that made us think about organizing this conference on

"Towards a better strategy for substance abuse".

Congress Committee

Pr. Dr. Ahmed Abou El-Azayem - Conference President

Pr. Dr. Khaled A. Al-Saleh – Secretary General

Pr. Dr. Saud Al-Duhayan - President of Scientific Committee

Goals of the Conference

- 1- To present the update situation of substance abuse problems in the Arab World. -1
- 2- Illustrate the recent strategies around the Arab world. -2
- 3- Reviewing and assessing substance abuse treatment and rehabilitation strategies around the Arab World. -3

Conference Axes

1- First Prevention axis:

To know the statistics of substance, its kinds and the ways of presenting it in the arab world.

2- Second Prevention axis:

- a- Universal Prevention policies.
- b- Selective prevention programme.
- c- Urgent Prevention program

3- Third Treatment of prevention axis:

It is for the selection of the best programme to treat substance abuse.

4- Forth Recovery and Rehabilitation axis.**Congress Topics:****A- Prevention Strategies:**

- 1- Research for identifying at risk groups.
- 2- Prevention programmes to raise protective factors.
- 3- Programs for promotion social and psychological skills to prevent substance abuse.
- 4- Hot lines and counselling programs.
- 5- Priority in prevention programs.

B- Therapeutic Strategies:

- 1- Early identification of addiction
- 2- Role of school health authority
- 3- Role of Primary health care
- 4- Relapse prevention strategies.
- 5- Recovery strategies and self help group.

C- Strategies of Damage Control:

- 1- Prevention of AIDS and Hepatitis infection
- 2- Prevention of crime in addict population.

D- Strategies for Training in the Prevention Programme and the Treatment from Substance Abuse.**E- Strategies of Dealing with the Drug Addict after going out from Prison.****Tours information**

Ground Handler & Travel Agent: Escapade Travel

Website : www.escapadetravel.com.eg

www.escapade.travel.com.eg :

Daily Tours

Tour 1: Half day tour to the Egyptian Museum (28\$)	(\$ 28) :	: 1
Tour 2: Half day tour to the Pyramids & sphinx (28\$)	(\$ 28) :	: 2
Tour 3: Half day tour to the Pharaonic Village (27\$)	(\$ 27) :	: 3
Tour 4: Half day Coptic Cairo incl. Churches (27\$)	(\$ 27) :	: 4
Tour 5: Half day tour to Khan Khalil Bazaars (17\$)	(\$ 17) :	: 5
Tour 6: Half day tour to Islamic Cairo (27\$)	(\$ 27) :	: 6
Tour 7: Sound & Light show (27\$)	(\$ 27) :	: 7
Tour 8: Cruising Dinner incl.show (50 \$)	(\$ 50) :	: 8
Tour 9: Overday tour to Alex incl. Lunch (75 \$)	(\$75):	: 9
Tour 10: Overday tour to Luxor incl. Lunch (350 \$)	(\$350) :	:10

Pre & Post Conference tours:

Tour 11 : 4 night cruise Luxor/Aswan	/ -	: 11
Monday 15/11 or Monday 22/11/2004	2004/11/22 11/15	
- Per person in Double (660\$)	(\$ 660)	-
- Per person in Single (780\$)	(\$ 780)	-
Tour 12: 3 night cruise Aswan / Luxor	/ -	: 12
Friday 19/11 or Friday 26/11/2004	2004/11/26 11/19	

- Per person in Double (600\$) (\$ 600) -
- Per person in Single (700\$) (\$ 700) -

Rates include:

Transfer from Hotel in Cairo to Cairo Airport -
 Airport transfers in Luxor & Aswan -
 4 or 3 night accommodation on full board basis -

Classical sightseeing with English peaking guide -
 Domestic flight Cairo/Luxor -Aswan/Cairo or v.v. on Y-
 class

Tour 13 : Hurdagha : 3 nights accommodation on BB

At 5 stars Hotel:

- Per person in Double (525\$) (\$ 525) :
- Per person in Single (625\$) (\$ 625) :

At 4 stars Hotel:

- Per person in Double (450\$) (\$ 450) :
- Per person in Single (495\$) (\$ 495) :

Tour 14 : Charm Esheikh

At 5 stars Hotel:

- Per person in Double (525\$) (\$ 625) :
- Per person in Single (725\$) (\$ 725) :

At 4 stars Hotel:

- Per person in Double (545\$) (\$ 545) :
- Per person in Single (595\$) (\$ 595) :

General Information

EGYPT: the mystic land of the Pharaohs. A land unequalled for its majestic monuments and authentic treasures of diversified Pharaonic, Greco-Roman, Coptic and Islamic blend of cultures. This blend of the old and the new is perfectly found on both sides of the famous Nile. On the Western side, at the edge of the desert plateau stands the Pyramids of Giza. Designated as one of the Seven Wonders of the World, they stand today as they did thousands of years ago, still guarded the Great Sphinx. On the Eastern side, high in the centre of the old city, lies the Saladin Citadel. Visiting this area, one may plunge into a living museum of Moslem society that has changed little over the centuries. A touch of the Middle Ages can be found in Khan El Khalili. This is also the site situated in the Centre of Cairo, the Egyptian Museum houses the wealth of artefacts discovered as a result of the wave of enthusiasm for Egyptology. Thus, Modern Egypt with its variety of settings, Mediterranean Beaches, Res-sea fascinating underwater life, mountains and deserts, memorable landscapes alongside the Nile provides a choice of holiday resorts for everyone from all over the world.

- **Venue of the conference** : the Grand Hyatt Hotel in Cairo. :
- **Conference Languages** : Arabic & English :
- **Climate** : In November, temperature ranges from 25C° in day time to 18C° at night 25-22 :
- **Shopping** : Stores open in Cairo every day from 10 a.m. till 10 p.m. Some shops close on Sunday. 10 18 :
- **Technical exhibition** : Pharmaceutical companies, manufactures of technical equipment and software, : 10

and publishers of scientific books are invited to display their products at an exhibition to be organized in the conference centre.

Instructions for Submission of Abstracts:

- Abstract texts should be type written within the rectangle.
- Please do not exceed 200 words written within the rectangle.

Official Languages: Arabic / English

Dead line for submission of Abstracts is 31 October, 2004.

Application:

Please fill Abstract form

Registration Fees

Before 30/09/2004 :

- Non members: USD 100
- Members USD 50
- Accompanying USD 50

2004/09/30 :
(100 : - \$ 100 :) -
(50 : - \$ 50 :) -
(50 : - \$ 50 :) -

After 30/09/2004 :

- Non members USD 150
- Members USD 100
- Accompanying USD 50

2004/09/30 :
(150 : - \$ 150 :) -
(100 : - \$ 100 :) -
(50 : - \$ 50 :) -

Registration fees include:

Opening ceremony / Coffee breaks / Admission to scientific sessions / Admission to technical exhibition / Certificate of attendance and badge / Conference kits (name tag, bag, programme, abstracts book).

/ / /
/ () /

For accompanying person:

- Opening ceremony / Coffee breaks

/ -

Payment:

Please address your method of payment in: Favour of:
The Arab Federation of NGOs of Substance Abuse

Acc. No 01906010047079

Bank : Cairo Bank – Nasr City Branch

Application : Please fill Registration form

01906010047079 :

0190510079701 :

- :

Hotels

Grand Hyatt Cairo Hotel (5*) : Single 120 USD - Double 130 \$

\$ 130 - \$ 120 : (5)

Helnan Hotel (5*) : Single 100 \$ - Double 110 \$

\$ 110 - \$ 100 : (5)

Pyramisa Hotel 5* : Single 80 \$ - Double 90 \$

\$ 90 - \$ 80 : (5)

Pharaons Hotel 3* : Single 40 \$ - Double 50 \$

\$ 50 - \$ 40 : (3)

Above rates are including service charge, governmental taxes& buffet breakfast

Transfer :

Meet, assist and transfer by private car from CAIRO Airport to Hotel in : "One way" Per person 20 \$ - Per 2 person 26 \$

() :
\$ 26 : 2 - \$ 20

To Contact Us

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Medical Design Center (MDC)
PO Box 106 Panorama October, 11811 Cairo – EGYPT

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-4

ورشة عمل : المدرسة و الجامعة – أزمات ، عقبات و فشل

تنظيم : المركز العربي للأبحاث النفسية و التحليلية

السبت 20 نوفمبر 2004

مدرسة المشرق الدولية ، الهاشمية – دابوق- عمان – الأردن

يشرف المركز العربي للأبحاث النفسية و التحليلية بدعوةكم الى المشاركة في ورشة عمل حول المدرسة و الجامعة- أزمات ، عقبات و فشل

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5412203 :

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961-1-810811 :

ahabalah@idm.net.lb :

MASTERE EN PSYCHIATRIE LÉGALE ET EXPERTALE

شهادة ماجستير : الطب النفسي الشرعي

UNIVERSITÉ TUNIS - FACULTÉ DE MÉDECINE

جامعة تونس المنار - كلية الطب - تونس

COORDINATEUR : PR. FAKHREDDINE HAFFANI

المنسق : أ.د. فخر الدين الحفاني

Objectifs de La Formation

- Former des médecins capables :
- D'utiliser des instruments législatifs de base du droit médical et pénal dans leur pratique médicale et expertale.
 - De pratiquer les différentes missions d'expertise psychiatrique.
 - De prévenir et de prendre en charge des malades mentaux dangereux en se basant sur les connaissances actuelles en criminologie clinique et psychiatrie légale.
 - D'exercer la psychiatrie en milieu pénitentiaire.

Programme

Le mastère spécialisé de psychiatrie Légale et Expertale est programmé sur quatre semestres (deux années).

Enseignement Theorique

- Volume horaire : 120 heures
- Présence obligatoire (assiduité exigée : 75% au minimum)
- Avec le concours d'enseignants et d'experts en psychiatrie légale, psychiatrie pénitentiaire, médecine légale et dans le domaine judiciaire, selon les thèmes suivants (7 modules).
- **Module I** : Organisation sanitaire de la psychiatrie en Tunisie
- **Module II** : Législation Tunisienne et psychiatrie
- **Module III** : Criminologie clinique
- **Module IV** : Prise en charge des malades mentaux dangereux et médico-légaux
- **Module V** : Eléments de Droit pénal et expertise psychiatrique
- **Module VI** : L'expertise en psychiatrie : généralités, pratique et missions
- **Module VII** : La psychiatrie en milieu pénitentiaire

Lieu de l'enseignement

Complexe d'enseignement « Sleim Ammar » à l'hôpital RAZI, tous les vendredi de 14heures à 17heures.

Enseignement pratique et dirigé

La présence aux enseignements dirigés et aux stages pratique est obligatoire.

Enseignements dirigés

- Sous forme d'ateliers
- Volume horaire : 24 heures
- Une séances bimensuelle de deux heures, à partir du troisième semestre
- Analyse et initiation à la rédaction d'expertises psychiatriques
- Analyse de cas cliniques et initiation à l'évaluation de

la dangerosité des malades mentaux.

■ Stage pratique

Visites d'institutions prenant en charge les malades mentaux médico-légaux, les patients dangereux et les toxicomanes :

- le service de psychiatrie légale de l'hôpital Razi
- le centre de traitement des toxicomanes « El Amal »
- la consultation de psychiatrie de la prison civile 9 Avril (après confirmation).

Validation Du Diplome

Un diplôme sera délivré après validation de l'écrit, des stages et du mémoire par la Faculté de Médecine de Tunis.

Inscriptions

Peuvent demander leur inscription à cet enseignement les candidats :

- Médecins spécialistes en psychiatrie
- Médecins spécialistes en médecine légale
- Résidents en psychiatrie
- Résidents en médecine légale

L'inscription définitive est tributaire de l'accord de la commission de sélection du **Mastère Spécialisé de Psychiatrie Légale et Expertale**

Renseignements Pratiques

- Faculté de Médecine de Tunis
Service de Scolarité – Bureau du 3ème Cycle
Tel : 71 563 709 / 710
Fax : 71 569 427
- Service de Psychiatrie Légale
Hôpital Razi, 2010 La Mannouba
Tel : 71 600 339 poste 279
Fax : 71 601 555
Email : rym.haffani@ms.tn

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Arabpsynet JOURNAL: N°3 – July – August – September 2004

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FORMATION SPÉCIALISÉE EN PSYCHOTHÉRAPIE

ملتقى تكويني مختص في العلاج النفسي

Organisée par :

L'association des médecins psychiatres du Québec
Comité de développement professionnel

En collaboration avec :

La Société Tunisienne de Psychiatrie
L'association Tunisienne des psychiatres privé

HAMMAMET, TUNISIE 2 / 10 OCTOBRE 2004

:
جمعية الأطباء النفسيين بكيبك - كندا
:
الجمعية التونسية للطب النفسي
الجمعية التونسية للأطباء النفسيين الخواص

تونس - الحمامات : 10/02 أكتوبر 2004

Programme scientifique préliminaire

L'association des médecins psychiatres du Québec et son comité de développement professionnel, en collaboration avec la Société Tunisienne de Psychiatrie et l'association Tunisienne des psychiatres d'exercice privé, sont heureux de vous inviter à la quatrième édition de leur formation spécialisée en psychothérapie.

C'est avec plaisir que nous vous offrons cette année un programme scientifique renouvelé, vous proposant six atelier d'apprentissage hautement spécialisés empreints d'échanges avec nos collègues tunisiens.

En espérant vous y voir.

Anne-Marie Bouchard, M.D.

Comité organisateur :

Mark-P. Adams, M.D./ Saida Douki, Pr. / Anne-Marie Bouchard, M.D. / Raja Labbane, Pr. Agr. / Afif Bousetta, Pr. Agr. / Marie-Eve Picard, M.D. / Dominique Croteau, M.D. / Annick Vincement, M.D. / André Delorme, M.D. / Sofiane Zribi, M.D. / Chebil Ben Dhia, M.D.

Atelier I

Introduction à l'approche systémique en psychiatrie / Guy Ausloos, M.D.

Objectifs :

- Récapituler les principes de base de l'approche systémique
- Appliquer ceux-ci à la pratique du psychiatre
- Envisager les situations particulières telles que l'urgence, l'hospitalisation, la gestion du suicide.

Atelier II

La thérapie interpersonnelle de la dépression majeure et ses autres indication / Simon Patry, M.D. / Ch Robert-Giffard

Objectifs :

- Connaître les bases théorique et clinique de la PTI.
- Appliquer la PTI dans le traitement de la dépression majeure.
- Connaître les autres indications de la PTI.

Atelier III

Prise en compte des spécificités culturelles dans les thérapies cognitivo-comportementales. / Sofiane Zribi, M.D.

Objectifs :

- Identifier les facteurs culturels pouvant empêcher

Arabpsynet Journal: N°3 - July - August - September 2004

مجلة شبكة العلوم النفسية العربية: العدد 3 - جويلية - أوت - سبتمبر 2004

ou influencer sur la prise en charge des troubles en thérapie cognitivo-comportementale.

- Savoir intégrer certains facteurs culturels utiles dans la prise en charge des TCC.
- Corriger les dysfonctions cognitives d'origine culturelle.

Atelier IV

Traitement de la psychose : ce que la psychothérapie cognitivo-comportementale peut offrir / Amal AbdelBaki, M.D.
– Luc Nicole, M.D.

Objectifs :

- Expliquer les troubles psychotiques à partir du modèle cognitif.
- Savoir différencier les principales approches du TCC pour la psychose :
 - Selon les objectifs et leurs buts
 - Selon les techniques utilisées
- Discuter des études démontrant l'efficacité des TCC pour la psychose
- S'initier aux différentes technique de TCC pour la psychose.

Atelier V

Thérapie cognitivo-comportementale des troubles anxieux (atelier avancé) / Nicole Thibodeau, M.D.

Objectifs :

- Approfondir la conceptualisation cognitivo-comportementale des troubles anxieux suivants : phobie sociale, trouble panique (avec ou sans agoraphobie), trouble obsessionnel compulsif et trouble anxieux généralisé.
- Mettre en pratique les stratégies cognitivo-comportementales correspondant à chacun de ces troubles anxieux au moyen de vignettes cliniques et d'exemples vidéos.

Atelier VI

La prise en charge des victimes de violence conjugale / Saïda Douki, Professeur

Objectifs :

- Evaluer l'importance et les répercussion sanitaires du problème
- Identifier les obstacles culturelles à la prise en charge.
- Connaître les principes et la spécificité de la prise en charge.

Formulaire de participation

- Choix du programme scientifique (lundi, mardi, jeudi, vendredi , 8 h à 12h)
- Veuillez identifier votre choix de cours, par ordre de préférence, de 1 à 6 (étant votre premier choix).
- Nous respectons votre premier choix jusqu'à la limite maximale, soit 15 participants par cours.

Ateliers : 1 | 2 | 3 | 4 | 5 | 6

Inscription sur place : Coûts 50 dinars

Information personnelles :

Nom :
Prénom :
Lieu et adresse de pratique :

- - 30 - - 2
8 1 5 ()
(514) 350-5128 :
(514) 350-5198 :

— 8050 . . —
(216) 72-27-95-50 :
(216) 72-27-99-72 :

(514) 350-5108

mdaneau@fmsg.org

XIII WORLD CONGRESS OF PSYCHIATRY EGYPT – Cairo September 10-15/2005		
Dates to Remember	DESCRIPTION	How to Contact Us
July 1, 2004	Deadline for submission of proposals Symposia, Workshops and Courses	Important Addresses Please address all correspondence concerning the congress to: ✱ XIII World Congress of Psychiatry Scientific and Technical Secretariat TILESA OPC, S.L. c. Londres, 17 - 28028 Madrid (Spain) Tel.: +34 913 612 600 Fax: +34 913 559 208 e-mail: secretariat@wpa-cairo2005.com ✱ Travel Agent EMECO Travel Accommodation, Tourist Services e-mail: accommodation@wpa-cairo2005.com For the latest information, please visit our web site: http://www.wpa-cairo2005.com
November 1, 2004	Deadline for submission of Abstracts for Lectures, Papers and Posters	
January 1, 2005	Deadline for Fellowship program and award application	
April 1, 2005	Deadline for reduced registration fees. Notification of acceptance of Abstracts and Posters.	

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Psy CONGRESS AGENDA

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أجندة المؤتمرات النفسية

الثلاثية الرابعة 2004

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ARAB Psy CONGRESS AGENDA

Title: Diagnostic Treatment of Attention Deficit & Hyperactivity Disorders**Date:** December 07, 2004 - December 08, 2004**Country:** Saudi Arabia - **City:** Riyadh**Contact:** Pamela Page**Phone:** 966-1-442-7238 - **Fax:** 966-1-442-4153**E-Mail:** web_symposia@kfshrc.edu.sa**Title:** Specialized Formation in Psychotherapy**Date:** October 02, 2004 - October 10, 2004**Country:** TUNISIA - **City:** Hammamet**Contact:** Mr. Manon Daneau**Phone:** (514) 350 5128 / 350 5108 - **Fax:** (514) 350 5198**E-Mail:** mdaneau@fmsq.org**Website:** www.arabpsynet.com/Congress/TrainingPsy-CaTn.pdf**Title:** The 6th Arab Conference for Prevention of S.A**Date:** November 24, 2004 - November 25, 2004**Country:** Egypt - **City:** Cairo**Tel/Fax:** (202) 4148089 - **Fax:** (202) 4183175**E-Mail:** mdc@medical-design.net**Website:** www.substanceabuse2004.comwww.arabpsynet.com/Congress/6thCong.Prev.againstDrugs.pdf

INTERNATIONAL Psy CONGRESS AGENDA

Title: Alzheimer's Disease: From Molecular Mechanisms to Drug Discovery**Date:** December 11, 2004 - December 17, 2004**Country:** Mexico - **City:** Cancun**Contact:** Nico Stanculescu**Phone:** 1-773-784-8134 - **Fax:** 1-208-575-5453**E-Mail:** meetings@worldeventsforum.com**Title:** Work Shop : Arab Center of Psychological & Psychoanalytical Research**Date:** November 20, 2004**Country:** JORDAN - **City:** Amman**Contact:** Mrs. Tamara Bassam Molhess**Phone:** 5412203**E-Mail:** ahabalah@idm.net.lb**Website:** www.arabpsynet.com/Congress/Wshop.CAEP.pdf**Title:** 1st Middle East Medical Students Congress**Date:** December 10, 2004 - December 12, 2004**Country:** Iran - **City:** Mashhad**Contact:** Congress Secretariat**E-Mail:** iaems@erh.ir**Title:** 23rd Franco Maghrebine Congress of Psychiatry**Date:** October 07, 2004 - October 08, 2004**Country:** TUNISIA - **City:** Monastir**Contact:** Pr. Lotfi GAHA**Phone:** 00216 73 461 965 - **Fax:** 00216 73 460 678**E-Mail:** gaha.lotfi@rns.tn**Website:** www.arabpsynet.com/Congress/Cong-FrancoMaghreb.pdf**Title:** Visiting Fellowship in ECT (Electroconvulsive Therapy)**Date:** December 06, 2004 - December 10, 2004**Country:** United States - **City:** Durham**State/Province:** NC**Contact:** Peg Musser**Phone:** 919-681-8742 - **Fax:** 919-681-7462**E-Mail:** pmusser@duke.edu**Title:** Update in Neurology and Psychiatry**Date:** October 05, 2004 - October 09, 2004**Country:** Maldives - **City:** Bolifushi Resort**Contact:** Mrs. Hanna Lahat**Phone:** 972-4-954-1870 - **Fax:** 972-4-954-1872**E-Mail:** hannal@netvision.net.il**Title:** Phase-Oriented Treatment of Psychological Trauma**Date:** December 03, 2004 - December 04, 2004**Country:** United States - **City:** Berlin**State/Province:** MA**Contact:** Office of Continuing Education**Phone:** 617-384-8600 - **Fax:** 617-384-8686**E-Mail:** hms-cme@hms.harvard.edu

Title: Buddhism and Psychotherapy Conference 2004**Date:** November 19, 2004 - November 21, 2004**Country:** Australia - **City:** Sydney**State/Province:** NSW**Contact:** Meeting Organiser**Phone:** 0-295-196-054 - **Fax:** 0-295-193-402**E-Mail:** info@buddhistlibrary.org.au**Title: Medical Student Congress, Young Researchers Club****Date:** November 17, 2004 - November 19, 2004**Country:** Iran - **City:** Najafabad**Contact:** Meeting Organiser**Phone:** 98-9-133-100-141 - **Fax:** 98-3-312-649-936**E-Mail:** 3nms@jaun.ac.ir**Title: A Psychiatric Update 2004****Date:** November 17, 2004 - November 17, 2004**Country:** Canada - **City:** London**State/Province:** ON**Contact:** Sandra Dunbar**E-Mail:** Sandra.Dunbar@sjhc.london.on.ca**Title: Australian Psychoanalytical Society Annual Conference - Psychoanalysis and Symbolisation****Date:** November 15, 2004 - November 16, 2004**Country:** Australia - **City:** Adelaide**Contact:** Mrs Janet King, Acting Honorary Secretary**Phone:** 61-398-828-628**E-Mail:** king@hyp.com.au**Title: Psychiatric Update****Date:** November 12, 2004 - November 13, 2004**Country:** United States - **City:** Madison**State/Province:** WI**Contact:** Cathy Means**Phone:** 608-263-6637 - **Fax:** 608-262-8421**E-Mail:** cjmeans@wisc.edu**Title: Advanced Course: Treatment of Mental Disorders during Pregnancy and Post-partum****Date:** November 11, 2004 - November 11, 2004**Country:** Italy - **City:** Florence**Contact:** Pr Mario Maj**Phone:** 39-0-815-666-502 - **Fax:** 39-0-815-666-523**E-Mail:** secretariat@wpa2004florence.org**Title: International Congress of the WPA - Treatments in Psychiatry: an Update****Date:** November 10, 2004 - November 13, 2004**Country:** Italy - **City:** Florence**Contact:** Pr Mario Maj**Phone:** 39-0-815-666-502 - **Fax:** 39-0-815-666-523**E-Mail:** secretariat@wpa2004florence.org**Title: 7th World Congress of Bioethics****Date:** November 09, 2004 - November 12, 2004**Country:** Australia - **City:** Sydney**State/Province:** NSW**Contact:** Kimberley Hatchett**Phone:** 61-293-853-503 - **Fax:** 61-293-136-185**E-Mail:** k.hatchett@unsw.edu.au**Title: Turkish Sleep Research Society Congress****Date:** November 07, 2004 - November 09, 2004**Country:** Turkey - **City:** Cesme**Contact:** Dr. Ibrahim Oztura**Phone:** 90-2-324-124-062 - **Fax:** 90-2-322-777-721**E-Mail:** ibrahim.oztura@deu.edu.tr**Title: Psychiatry Review Course Celebrity Cruise****Date:** November 06, 2004 - November 13, 2004**Country:** Barbados - **City:** Bridgetown**Contact:** Course Organiser**Phone:** 416-237-1427**E-Mail:** info@psychiatryreviewcourse.com**Title: Treating Bipolar Disorder: From Childhood to Adulthood****Date:** November 05, 2004 - November 06, 2004**Country:** United States - **City:** Boston**State/Province:** MA**Contact:** Office of Continuing Education**Phone:** 617-384-8600 - **Fax:** 617-384-8686**E-Mail:** hms-cme@hms.harvard.edu**Title: 21st Annual Mental Health Aspects of Developmental Disabilities: Promising Treatment & Service****Date:** October 27, 2004 - October 30, 2004**Country:** United States - **City:** Philadelphia**State/Province:** PA**Contact:** Cynthia Johnson**Phone:** 215-842-7180**E-Mail:** cme@drexel.edu / Cynthia.Johnson@drexel.edu

Title: XVIII World Congress of Social Psychiatry
Date: October 24, 2004 - October 27, 2004
Country: Japan - **City:** Kobe
Contact: The Japanese Society of Social Psychiatry
Phone: 81-473-720-141 - **Fax:** 81-783-026-485

Title: Psychopharmacology
Date: October 21, 2004 - October 23, 2004
Country: United States - **City:** Boston
State/Province: MA
Contact: Office of Continuing Education
Phone: 617-384-8600 - **Fax:** 617-384-8686
E-Mail: hms-cme@hms.harvard.edu

Title: Psychiatry for General Practice
Date: October 16, 2004 - October 17, 2004
Country: South Africa - **City:** Stellenbosch
Contact: Charmaine Hugo
Phone: 27-219-389-229 - **Fax:** 27-219-314-172
E-Mail: [mhc@sun.ac.za](mailto:mhic@sun.ac.za)

Title: Psychopharmacology Update 2004
Date: October 16, 2004 - October 17, 2004
Country: United States - **City:** Houston
State/Province: TX
Contact: Office
of Continuing Medical Education, Baylor College of Medicine,
1709 Dryden, Suite 1218, Houston Texas, 77030
Phone: 713-798-8237 - **Fax:** 713-798-7955
E-Mail: cme@bcm.tmc.edu

Title: 20th International Conference of the Alzheimer's Disease International
Date: October 15, 2004 - October 17, 2004
Country: Japan - **City:** Kyoto
Contact: Miyake Yoshio MD
Phone: 81-75-811-8195 - **Fax:** 81-75-811-8195
E-Mail: adiconference@alzheimer.or.jp

Title: 5th International Congress of Neuropsychiatry. 1st Mediterranean Regional Congress of the World Federation of Societies of Biological Psychiatry
Date: October 14, 2004 - October 18, 2004
Country: Greece - **City:** Athens
Contact: Dora Gondicas
Phone: 30-2103609442/30-2103615201 - **Fax:** 30-2103625572
E-Mail: easytravel@hol.gr
secretariat@ina-wfsbp-dualcongress.gr

Title: 54th Canadian Psychiatric Association Annual Meeting
Date: October 14, 2004 - October 17, 2004
Country: Canada - **City:** Montreal
State/Province: QC
Contact: The Canadian Psychiatric Association, 260-441 MacLaren Street, Ottawa, Ontario, K2P-2H3
Phone: 613-234-2815 - **Fax:** 613-234-9857
E-Mail: cpa@cpa-apc.org / agm@cpa-apc.org

Title: 3rd Indonesian Conference on Schizophrenia
Date: October 09, 2004 - October 14, 2004
Country: Indonesia - **City:** Denpasar
Contact: Diana
Phone: 62-213-107-741 - **Fax:** 62-2-139-899-128
E-Mail: psikiatri.fkui@link.net.id

Title: 12th World Congress on Psychiatric Genetics
Date: October 09, 2004 - October 13, 2004
Country: Ireland - **City:** Dublin
Contact: Dr Michael Gill
Phone: 35-312-802-641 - **Fax:** 35-312-802-665
E-Mail: mgill@tcd.ie / wcp2004@ovation.ie

Title: 8th Congress of the International Association for the Treatment of Sexual Offenders: Prevention of Sexual Abuse
Date: October 06, 2004 - October 09, 2004
Country: Greece - **City:** Athens
Contact: Dr Orestis Giotakos
Phone: 30-6-945-464-619 - **Fax:** 30-2-106-994-225
E-Mail: giotakos@tri.forthnet.gr

Title: Mental Health Conference
Date: October 06, 2004 - October 06, 2004
Country: United States - **City:** Rootstown
State/Province: OH
Contact: Northeastern Ohio Universities College of Medicine, Continuing Medical Education, 4209 State Route 44, Rootstown, OH 44272-0095
Phone: 330-325-6575 - **Fax:** 330-325-5929
E-Mail: cme@neoucom.edu

Title: The 1st National Conference on Addiciton Medicine
Date: October 01, 2004 - October 03, 2004
Country: Malaysia - **City:** Kuala Lumpur
Contact: Ms. YM Kong
Phone: 60-320930100 /60-320930200 - **Fax:** 60-320930900
E-Mail: acadmed@streamyx.com

أ.د. محمد أحمد النابلسي - الطب النفسي - لبنان

بـسرید الـكترونـی : nabulsy@cyberia.net.lb

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Some psychological and behavioral characteristics among children with learning disabilities / Prof. Aman Mahmoud , Dr. Samia Saber

Abstract: The aim of the study is to investigate the relationship between anxiety, rating behavior characteristics and psychological centrality concept among children with learning problems disabilities.

The children who participated in the study were 50 (boys, girls) selected from a primary and elementary schools, in Kuwait. The ages of children ranged between (8 - 12). They were divided into two group (special class, regular class). The results obtained were as follows: Children with learning disabilities reported more anxiety (emotional, cognitive, physiology and (physical). They, also, reported less (psychological) centrality concept and rating behavior such as (personal, motor coordination, orientation, spoken language, auditory comprehension) than normal children. Children with learning disabilities (Male - Female) differed from each other, where male have repotted more rating behavior (personal), psychological centrality concept (physical). One other hand female reported more anxiety. Children with learning disabilities whom were drawn from different educational level and attended (special - regular) class did not differ in reporting psychological traits.

A Diagnostic of Cognitive Competences and their Development / Prof. El Ghali Aharchaou - University of Sidi Mohammed Bin Abdullah Fez

Abstract : This work aims to provide succinct sorts of evidence to back up the belief that cognitive competencies are likely to lie improved and analyzed, assuming, hence, that modern psychology's task is to find feasible solutions for issues that have stimulated research in acquisition, adaptability and aptitude to everyday life practices. It has become a trite truism that children's cognitive faculty is flexible to admit development via interference, as proved by the multifaceted but convenient programs and procedures.

The idea that children must enjoy high cognitive faculty and mental competencies for them to acquire and successfully realize given tasks has been largely transcended. Rather, they have to be trained to apply successfully those faculties on all aspects of real life. This reiterates the importance and the efficiency of cognitive diagnostic sways and switches along with cognitive educational programs viz à viz of the reforming of children's cognitive faculty and its development. Equally important is children's sensitizing towards an efficient learning strategy to improve and enrich their learning practices.

The work offers a wealth of illuminating information bringing to mind that any potential failure cannot be traced to a weak cognitive faculty but rather to children's inability to start out their cognitive force, their weak personal efforts and their bound feelings of an acquired failure.

Phonological Encoding & Cognitives Processes of Arabic language Production / Dr. Mostafa BOUANANI – University of Sidi Mohammed Bin Abdullah Fez

Abstract : This article main objective to determine the cognitive processes of spoken language production (In Arabic children speakers). We will start first by presenting the different stages of Arabic language production, namely, the conceptualization, the lexicalization (based on two types of encoding : syntaxico-semantic and morpho-phonologic), as well as the articulation. Second, we will try to show the importance of phonological encoding in the lexical access. We will prove that the lexical access (used in every language production) is done through an obligatory use of the phonological component of the language. Finally, we will refer to some empirical evidences to support the phonological mediation via the presentation of some production phenomena that we can understand and examine without referring to the phonological information specific to the Arabic language. This will also prove that all correct didactic and pedagogic strategies of Arabic language instruction should take into consideration these parametrical specifications of Arabic language organization and functioning.

Key-words : Arabic language production, Cognitive processes used in language production, phonological encoding.

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مجلة شبكة العلوم النفسية العربية: العدد 3 - جويلية - أوت - سبتمبر 2004

PREMENSTRUAL Dysphoric Disorder (PMDD)

PMDD & WOMEN CASABLANCA

▪ ASSESSMENT OF PREMENSTRUAL DYSPHORIC DISORDER SYMPTOMS: POPULATION OF WOMEN IN CASABLANCA

Authors : McHichi alami Kh, Tahiri SM, Moussaoui D, Kadri N. Centre Psychiatrique Universitaire Ibn Rochd, Casablanca, Maroc..

Summary : Menstruation is a biological phenomenon that has been subject of myths and taboos within and among various cultures. These myths distort the reality surrounding menstruation and create ambivalent feelings about the value and usefulness of this function outside of its necessity as mean of reproduction. Thus studies concerning menstruation need to take into account cultural and psychosocial factors that define the meaning, values and behavior associated with this biological phenomenon. According to several studies, 70% of women experience psychological faintness during this menstrual phase, 40% of them have these symptoms at each menstruation and between 3 to 8% of them suffer severely reacquiring medical support. This entity called premenstrual dysphoric disorder is defined by the presence of several symptoms (distress, tension, irritability, moodiness...) with a significant impairment in work or social functioning beginning during the week before and ending within a few days after the onset of menses. Several studies conducted over the past few years suggested that selective serotonin reuptake inhibitors (SSRIs) and serotoninergic tricyclic drugs may be more effective than other types of antidepressants in treating PMS symptoms. Two protocols are proposed; a continuous treatment or intermittent use during few days during premenstrual and menstrual phase for several cycles. The objective of the current study was to evaluate the prevalence of a potential premenstrual dysphoric disorder (PMDD) during one menstrual cycle, in a representative sample of general population of Casablanca, according the DSM IV criteria. On the other hand, a questionnaire, available from the authors, was used to explore socio-demographic data. Among 618 women interviewed, 310 met the criteria of a potential PMDD (50.2%). The mean age of the population with PMDD was 32.2 8 years ranging from 20 to 50 years; 54.8% of them were married, 33.9% of them were single and 66.5% of them had between 1 to 4 children. Two third of them were without a professional activity. During this premenstrual phase the following symptoms were found among the whole sample: marked depressive mood, feeling of hopelessness, or self-depreciation thoughts (77.7%, n=241); difficulty of concentration (65%, n=201); marked change in appetite, overeating or specific food craving (82.8%, n=256); marked affective lability, with sadness tearful and increased sensitivity to rejection (65.8%, n=204); hypersomnia or insomnia (59.7%, n=185); subjective sense of being overwhelmed or out of control (55.7%, n=172); lethargy, excessive fatigability (91.6%, n=283); physical symptoms including breast tenderness, swelling, headache, joint or muscular pain, and a sensation of bloating and weight gain (81.9%, n=253). The most severe symptoms were fatigue and irritability. On the other hand, 73.9% of the sample had a disturbance in their socio-professional lives as a consequence to the psychological disturbances. Half of these women consulted a physician mostly a general practitioner. These data are in accordance with the literature, confirming that this disorder is common and

has a bad impact on mental health and on quality of life of the women suffering from PMDD.

PMDD & PAROXETINE

▪ PAROXETINE CONTROLLED RELEASE

Authors : Bang LM, Keating GM . Adis International Limited, Auckland, New Zealand.

Summary : A controlled-release (CR) formulation of the SSRI paroxetine has been developed. This CR formulation delays the release of paroxetine until the tablet has passed through the stomach; the drug is then released over 4-5 hours. In well designed placebo-controlled trials in patients with major depressive disorder (including a study in the elderly), social anxiety disorder or premenstrual dysphoric disorder (PMDD), paroxetine CR was consistently superior to placebo with regards to primary endpoints (i.e. mean Hamilton Rating Scale for Depression total score [major depressive disorder], Liebowitz social anxiety scale total score and Clinical Global Impressions-Global Improvement score [social anxiety disorder] and Visual Analogue Scale-Mood score [PMDD]). The duration of treatment was 12 weeks or, in PMDD, over three menstrual cycles (intermittent or continuous administration). Paroxetine CR also demonstrated efficacy in three well designed studies in patients with panic disorder with or without agoraphobia. Paroxetine CR was generally well tolerated in clinical trials, with an adverse-event profile typical of SSRIs, although recipients of paroxetine CR experienced significantly less nausea than recipients of immediate-release paroxetine in the first week of treatment.

PMDD (SEVERE PMS) & Citalopram

▪ CITALOPRAM IN PMS PATIENTS WITH PRIOR SSRI TREATMENT FAILURE : A PRELIMINARY STUDY

Authors : Freeman EW, Jabara S, Sondheim SJ, Auletto R. Departments of Obstetrics/Gynecology, University of Pennsylvania Medical Center, Philadelphia, Pennsylvania, USA.

Summary : OBJECTIVES: Evidence shows that the selective serotonin reuptake inhibitors (SSRIs) effectively reduce the symptoms of severe premenstrual syndrome (PMS). A placebo-controlled study of citalopram, the most selective SSRI, demonstrated that half-cycle dosing (luteal phase) was effective for DSM-IV-defined premenstrual dysphoric disorder (PMDD), a severe form of PMS. This study examined the effectiveness of half-cycle dosing of citalopram in PMS patients who did not respond to previous SSRI treatment.

METHODS: Seventeen women with no improvement in symptoms after two menstrual cycles on an SSRI were given open-label citalopram (20-40 mg/day). Eleven subjects received half-cycle dosing, and 6 subjects received full-cycle dosing. Scores on the 17-item daily symptom report (DSR) and on each of five DSR symptom clusters were used to measure citalopram efficacy. **RESULTS:** Total premenstrual DSR scores were significantly improved ($p < 0.001$) in both half-cycle and full-cycle dosing groups. The half-cycle group reported lower DSR scores throughout treatment compared with the full-cycle group, but the difference did not reach statistical significance in this small sample. All DSR factor scores (mood, behavioral, pain, physical symptoms, and appetite) significantly improved. Clinical improvement ($> 50\%$ decrease from baseline DSR) was reported by 76% of the subjects overall. Forty-one percent of the subjects

experienced symptom remission, defined as a decrease in symptoms to postmenstrual levels. **CONCLUSIONS:** These results from a small number of subjects with open-label treatment must be viewed as preliminary but suggest that citalopram treatment is effective for PMS patients who failed previous SSRI treatment.

PMDD & Venlafaxine

▪ EFFECTIVE OPEN-LABEL TREATMENT OF PREMENSTRUAL DYSPHORIC DISORDER WITH

Authors : Hsiao MC, Liu CY.

Department of Psychiatry, Chang Gung Memorial Hospital and Chang Gung University School of Medicine, Tao-Yuan, Taiwan. liucy752@cgmh.org.tw.

Summary : Various studies have demonstrated the efficacy of selective serotonergic re-uptake inhibitors in the treatment of premenstrual dysphoric disorder (PMDD). But the effectiveness of novel antidepressant, venlafaxine, in PMDD has been reported in only one Western study. The purpose of the present open-label study was to provide preliminary data on the effectiveness of venlafaxine for Asian women with PMDD. Thirty women with PMDD were enrolled and treated with a flexible dosage of venlafaxine for two menstrual cycles. Responses were assessed every 2 weeks. Outcome measures included the scores of the Prospective Record of the Impact and Severity of Menstrual Symptomatology (PRISM) calendar, self-rating Zung Depressive Scale (Zung), State and Trait Anxiety Inventory (STAI), Hamilton Rating Scale for Depression/Anxiety (HAM-D/HAM-A), and the Clinical Global Impression scale (CGI). Twenty patients completed the trial. All patients had significant improvement of the mood and behavior components in the PRISM calendar. The effects of active treatment were marked by the first active cycle of menstruation. Venlafaxine at a mean dose of 60.1 +/- 29.1 mg per day was effective in reducing PMDD symptoms. The results of the present open trial indicated that venlafaxine is effective in the treatment of ethnic Taiwanese women with PMDD.

PMDD, PMS & SSRI

▪ SELECTIVE SEROTONIN REUPTAKE INHIBITORS FOR PREMENSTRUAL SYNDROME

Authors : Wyatt KM, Dimmock PW, O'Brien PMS

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 08 October 2001. Cochrane reviews are regularly checked and updated if necessary.

Background: Severe premenstrual syndrome affects between three to five per cent of women of reproductive age. Such severe PMS is classified under the Diagnostic and Statistical Manual of Mental Disorders as premenstrual dysphoric disorder, PMDD. Selective serotonin reuptake inhibitors (SSRIs) are increasingly being used as a front-line therapy for premenstrual syndrome (PMS). A systematic review was undertaken on the efficacy of SSRIs in the management of severe PMS/PMDD, to assess the evidence for this treatment option.

Objectives: The objective of this review was to evaluate the effectiveness of SSRIs in reducing premenstrual syndrome symptoms in women diagnosed with severe premenstrual

syndrome.

Search strategy: Electronic searches for relevant randomised controlled trials of the Cochrane Menstrual Disorders and Subfertility Group specialised register of controlled trials, Cochrane Controlled Trials Register, MEDLINE, EMBASE and PsycLIT were undertaken. References were searched interactively to identify missed trials. Where insufficient data were presented original authors were contacted for further details.

Selection criteria: All trials were considered in which women with a prospective diagnosis of PMS/ PMDD were randomised to receive SSRIs or placebo in a double blind trial for the treatment of premenstrual syndrome.

Data collection and analysis: 31 randomised controlled trials were identified which reported the use of SSRI s in the management of PMS. 16 trials were excluded, 15 trials were included in the systematic review, and ten trials were included in the main analyses. The reviewers extracted the data independently and standardised mean differences for continuous outcomes were estimated from the data.

Main results: The primary analysis of reduction in overall symptomatology included data on 844 women with premenstrual syndrome. SSRIs were found to be highly effective in treating premenstrual symptoms. Secondary analysis showed that they were as effective in treating physical as well as behavioural symptoms. There was no significant difference between trials funded by pharmaceutical companies and those independently funded. Withdrawals due to side effects were 2.5 times more likely to occur in the treatment group, particularly at higher doses.

Reviewers' conclusions: There is now very good evidence to support the use of selective serotonin reuptake inhibitors in the management of severe premenstrual syndrome.

Major Depression Disorder (MDD)

MDD & Obesity

▪ OBESITY AMONG OUTPATIENTS WITH MAJOR DEPRESSIVE DISORDER.

Authors : Papakostas GI, Petersen T, Iosifescu DV, Burns AM, Nierenberg AA, Alpert JE, Rosenbaum JF, Fava M. Depression Clinical and Research Program, Massachusetts General Hospital Harvard Medical School, Boston, MA, USA.

Source : Int J Neuropsychopharmacol. 2004 Sep 13;1-5.

Summary : Studies focusing on the prevalence of obesity in Major Depressive Disorder (MDD), or the impact of excess body fat on the treatment of MDD are lacking. The aim of the present work is to systematically study obesity in MDD outpatients. A total of 369 MDD outpatients enrolled in an 8-wk trial of 20 mg fluoxetine had height and weight measured at baseline. We then examined: (1) the prevalence of being overweight or obese, (2) the relationship between obesity and a number of demographic and clinical variables, and, (3) the relationship between relative body weight and obesity with clinical response. We found that more than 50% of patients were overweight body mass index (BMI) 25 kg/m², while 20% were obese (BMI 30 kg/m²). Obese patients presented with worse somatic well-being scores than non-obese MDD patients, but they did not differ with respect to depression severity, anxiety, somatic complaints, hopelessness or hostility. Greater relative body weight, but not obesity, predicted non-response. In conclusion, greater relative body weight was found to place MDD outpatients at risk for

fluoxetine resistance.

MDD & Clinician-Rated Measures

■ COMPARISON OF SELF-RATED AND CLINICIAN-RATED MEASURES OF DEPRESSIVE SYMPTOMS: A NATURALISTIC STUDY

Authors : Dorz S, Borgherini G, Conforti D, Scarso C, Magni G. Affective Disorders Unit, Casa di Cura Parco dei Tigli, Padova, Italy.

Source : Psychol Psychother. 2004 Sep;77(Pt 3):353-61.

Summary : In order to assess the concordance between self-rating and clinician's assessment tools of depression, as well as factors involved in the differences between auto and hetero evaluation, 198 depressed in-patients were assessed at admission and at discharge using the Montgomery Asberg Depression Rating Scale (10-item version, MADRS) and the self-rating scale Symptoms CheckList (90-item version, SCL-90). We found that about 18% of patients overestimated and about 15% underestimated their depressive symptomatology (SCL-90 depression subscale) relative to the psychiatrist's assessment. Logistic regression analysis showed that the presence of personality disorders and previous history of psychiatric disorders predicted the overestimating group. Discriminant analysis showed that approximately 75% of participants were correctly classified when previous history of psychiatric disorders, presence of personality disorders and age were entered separately into the equation.

MDD & Reasons for Depression

■ THE REASONS FOR DEPRESSION QUESTIONNAIRE (RFD): UK STANDARDIZATION FOR CLINICAL AND NON-CLINICAL POPULATIONS

Authors : Thwaites R, Dagnan D, Huey D, Addis ME. North Cumbria Mental Health and Learning Disabilities NHS Trust, UK.

Source : Psychol Psychother. 2004 Sep;77(Pt 3):363-74

Summary : Recent research into reason giving for depression has illustrated the importance of client beliefs about the cause of their depression. Reasons given have been found to be associated with level of depression, perceived credibility of treatments and therapy outcome. It has been suggested that giving reasons for depression is a form of rule-governed behaviour and as such can cause the depression to be harder to treat (i.e. the reasons become functionally true for the individual). This study investigates the reliability and validity of the Reasons for Depression Questionnaire (RFD; Addis, Truax, & Jacobson, 1995), a 48-item self-report measure developed to measure explanations for the causes of depression. The study provides preliminary normative data for both clinical (n = 123) and non-clinical (n = 105) UK samples. The data indicate high reliability for all subscales including a further subscale (biological) added since the measure was initially developed. Certain subscales correlate significantly with level of depression and specific aspects of self-esteem. This supports the validity of the measure and suggests that it is measuring a distinct concept rather than significantly overlapping with individuals' general beliefs about themselves.

MDD & High Dose L-Thyroxine

■ HIGH DOSE L-THYROXINE IN

THERAPY REFRACTORY DEPRESSION. CASE ANALYSIS AND CATAMNESIS AS QUALITY CONTROL

Authors : Pfeiffer H, Scherer J, Albus M. Bezirkskrankenhaus Haar, Haar. pfeiffer@krankenhaus-haar.de

Source : Nervenarzt. 2004 Mar;75(3):242-8.

Summary : In a depression unit in a state hospital, 28 patients who had failed in six antidepressant strategies were treated with L-thyroxine at an average dose of 350 micro g/die. Outcomes were moderate in 39.3% and very good in 21.5%, corresponding to 21-item HAMD scores of < or =16 and < or =8 and clinical judgement. Of all patients, 39.3% had to stop treatment due to nonresponse or side effects. Follow-up of all responders to treatment was conducted 45.2 weeks after discharge. Those 28.6% patients who had stopped treatment had significantly more readmissions, i.e., 62.5%, vs none in those who continued, whereas subjective clinical ratings did not differ between the two groups. In contrast to the literature not finding serious side effects in 70 mainly bipolar patients, we found cardiac arrhythmia in 10.7% of inpatients and 7.1% of follow-up patients that was serious enough to discontinue treatment. In conclusion, systematic investigation of high-dose L-thyroxine treatment in treatment-resistant depression seems promising and necessary.

MDD & Review of Studies

■ A REVIEW OF STUDIES OF THE HAMILTON DEPRESSION RATING SCALE IN HEALTHY CONTROLS: IMPLICATIONS FOR THE DEFINITION OF REMISSION IN TREATMENT STUDIES OF DEPRESSION.

Authors : Zimmerman M, Chelminski I, Posternak M. Department of Psychiatry and Human Behavior, Brown University School of Medicine, Rhode Island Hospital, Providence, RI.

Source : J Nerv Ment Dis. 2004 Sep;192(9):595-601.

Summary : The Hamilton Rating Scale for Depression (HRSD) is the most commonly used symptom severity scale to evaluate the efficacy of antidepressant treatment. On the basis of an expert consensus panel, an HRSD score of ≤ 7 was recommended as a cutoff to define remission. Since that recommendation, little empirical work has been conducted to confirm the validity of this threshold. One approach toward determining a cutoff score for defining remission is to establish the range of values for healthy controls. We therefore conducted a literature review of studies of the HRSD in healthy controls to determine the normal range of values. Studies of the HRSD in healthy control groups were identified in two ways. First, a MEDLINE search for the years 1966 to 2002 was conducted using the key words Hamilton, depression, and controls, and articles were reviewed. Second, the 69 studies included in two review articles written by the authors were examined. We identified 27 studies that included data on the HRSD for 1014 healthy controls. Across all studies, the weighted mean (SD) HRSD score, adjusting for sample size, was 3.2 (3.2; 95% CI, 3.0 to 3.4). HRSD scores were similar in geriatric and nongeriatric samples, and in men and women. Because HRSD scores in healthy controls are more likely to follow a skewed than a normal distribution, based on a mean of 3.2 and a SD of 3.2, at least 84% of healthy controls scored 7 or less on the HRSD, and 97.5% scored 10 or less. Thus, these results can be taken as support for the recommended cutoff of 7 on the HRSD to define

remission. The results can also be used for normative comparisons in which posttreatment group mean scores are compared with mean scores from normative samples.

MDD, Antidepressant & Benzodiazepine

■ ANTIDEPRESSANT AND

BENZODIAZEPINE FOR MAJOR DEPRESSION

Authors : Furukawa TA, Streiner DL, Young LT

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 04 April 2001. Cochrane reviews are regularly checked and updated if necessary.

Background: Anxiety frequently coexists with depression. Adding benzodiazepines to antidepressants is commonly used to treat people with depression, although there has been no convincing evidence to show that such a combination is more effective than antidepressants alone and that there are suggestions that benzodiazepines may lose their efficacy with long-term administration and that their chronic use carries risks of dependence.

Objectives: To determine whether, among adult patients with major depression, adding benzodiazepines to antidepressants brings about any benefit in terms of symptomatic recovery or side-effects in the short term (less than 8 weeks) and long term (more than 2 months), in comparison with treatment by antidepressants alone.

Search strategy: We searched MEDLINE (1972 to September 1997), EMBASE (1980 to September 1997), International Pharmaceutical Abstracts (1972 to September 1997), Biological Abstracts (1984 to September 1997), LILACS (1980 to September 1997), PsycLIT (1974 to September 1997), the Cochrane Library (issue 3, 1997) and the trial register of the Cochrane Depression, Anxiety and Neurosis Group (last searched March 1999), combined with hand searching, reference searching, SciSearch and personal contacts.

Selection criteria: All randomised controlled trials that compared combined antidepressant-benzodiazepine treatment with antidepressant alone for adult patients with major depression. Exclusion criteria are: antidepressant dosage lower than 100 mg of imipramine or its equivalent daily and duration of trial shorter than four weeks.

Data collection and analysis: Two reviewers independently assessed the eligibility and quality of the studies. Two reviewers independently extracted the data. Standardized weighted mean differences and relative risks were estimated with random effects model. The dropouts were assigned the least favourable outcome. Two sensitivity analyses examined the effect of this assumption as well as the effect of including medium quality studies. Three a priori subgroup analyses were performed with regard to the patients with or without comorbid anxiety and with regard to the type.

Main results: Aggregating nine studies with a total of 679 patients, the combination therapy group was less likely to drop out than the antidepressant alone group (relative risk 0.63, 95% confidence interval 0.49 to 0.81). The intention-to-treat analysis (with people dropping out assigned the least favourable outcome) showed that the combination group was more likely to show improvement in their depression (defined as 50% or greater reduction in the depression scale from baseline) (relative risk 1.63, 95% confidence interval 1.18 to 2.27 at one week and relative risk 1.38, 95% confidence interval 1.15 to 1.66 at four weeks). The difference was no longer significant at six to eight weeks. None of the included

RCTs lasted longer than eight weeks. The patients allocated to the combination therapy were less likely to drop out from the treatment due to side effects than those receiving antidepressants alone (relative risk 0.53, 95% confidence interval 0.32 to 0.86). However, these two groups of patients were equally likely to report at least one side effect (relative risk 0.99, 95% confidence interval 0.92 to 1.07).

Reviewers' conclusions: The potential benefits of adding a benzodiazepine to an antidepressant must be balanced judiciously against possible harms including development of dependence and accident proneness, on the one hand, and against continued suffering following no response and drop-out, on the other.

MDD, SSRIs & Tricyclic

■ SELECTIVE SEROTONIN REUPTAKE

INHIBITORS (SSRIs) VERSUS OTHER

ANTIDEPRESSANTS FOR DEPRESSION

Authors : Geddes JR, Freemantle N, Mason J, Eccles MP, Boynton 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 15 July 1999. Cochrane reviews are regularly checked and updated if necessary.

Background: The relatively new class of antidepressant, the selective serotonin reuptake inhibitors (SSRIs), may be better tolerated than the older tricyclic antidepressants. This review compares the efficacy of SSRIs with other antidepressants.

Objectives: To examine the relative efficacy of selective serotonin reuptake inhibitors (SSRIs) compared to other antidepressants.

Search strategy: The search strategy included a search of (a) Electronic bibliographic databases (MEDLINE, EMBASE); (b) reference lists of related reviews (c) reference lists of all located studies (d) contact with the manufacturer and (e) the Cochrane Group register of controlled trials

Selection criteria: Randomised controlled trials comparing selective serotonin reuptake inhibitors with other kinds of antidepressants in the treatment of patients with depressive disorders. The outcome measures assessed included measures of the severity of depression.

Data collection and analysis: Data were collected from each study the main outcome measure from each study. These included: mean Hamilton depression rating scale, mean Montgomery & Asberg depression rating scale, Clinical Global Impression rating scale. An analysis of standardised mean difference of these scales was performed using Review Manager 3.1 software. The presence of heterogeneity of treatment effect was assessed

Main results: Ninety-eight trials contributed data to the analysis of the relative efficacy of SSRIs and related drugs with comparator antidepressants (Figure 3 & Appendix 3). Analysis of efficacy was based upon 5044 patients treated with an SSRI or related drug, and 4510 treated with an alternative antidepressant. The standardised effect size for SSRIs and related drugs together versus alternative antidepressants using a fixed effects model was 0.035 (95% CI -0.006 to 0.076; $Q = 149.25$, $df = 97$, $p < 0.001$).

Reviewers' conclusions: There are no clinically significant differences in effectiveness between selective serotonin reuptake inhibitors and tricyclic antidepressants. Treatment decisions need to be based on considerations of relative patient acceptability, toxicity and cost.

MDD & TMS

TRANSCRANIAL MAGNETIC STIMULATION FOR TREATING DEPRESSION

Authors : Martin JLR, Barbanof MJ, Schlaepfer TE, Clos S, Perez V, Kulisevsky J, Gironell, A

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 12 July 2001. Cochrane reviews are regularly checked and updated if necessary.

Background: Transcranial magnetic stimulation can either excite or inhibit cortical areas of the brain, depending on whether the speed of the repetitive stimulation is applied at high or low frequencies. It has been used for physiological studies and it has also been proposed as a treatment for depression.

Objectives: To assess the clinical efficacy and safety of transcranial magnetic stimulation for treating depression.

Search strategy: An electronic search was performed including the Cochrane Collaboration Depression, Neurosis and Anxiety Review Group trials register (last searched June, 2001), the Cochrane Controlled Trials Register (Issue 2, 2001), MEDLINE (1966-2001), EMBASE (1974-2001), PsycLIT (1980-2001), and bibliographies from reviewed articles. Unpublished data and grey literature were searched through personal communications with researchers.

Selection criteria: Randomised controlled trials assessing the therapeutic efficacy and safety of transcranial magnetic stimulation for depression.

Data collection and analysis: All reviewers independently extracted the information and verified it by cross-checking. Disagreements were resolved through discussion. Continuous data: When similar studies were grouped, the overall standardised mean difference was calculated under a fixed effect model weighted by the inverse variance method with 95% confidence intervals. (In the presence of statistical heterogeneity, a random effects model was to be used.)

Main results: Sixteen trials were included in the review and fourteen contained data in a suitable form for quantitative analysis. Most comparisons did not show differences between rTMS and other interventions. No difference was seen between rTMS and sham TMS using the Beck Depression Inventory or the Hamilton Depression Rating Scale, except for one time period (after two weeks of treatment) for left dorsolateral prefrontal cortex and high frequency; and also for right dorsolateral prefrontal cortex and low frequency, both in favour of rTMS and both using the Hamilton scale. Comparison of rTMS (left dorsolateral prefrontal cortex and high frequency) with electroconvulsive therapy showed no difference except for psychotic patients after two weeks treatment, using the Hamilton scale, which indicated that electroconvulsive therapy was more effective than rTMS.

Reviewers' conclusions: The information in this review suggests that there is no strong evidence for benefit from using transcranial magnetic stimulation to treat depression, although the small sample sizes do not exclude the possibility of benefit.

MDD & Stroke

INTERVENTIONS FOR PREVENTING DEPRESSION AFTER STROKE

Authors : Anderson CS, Hackett ML, House AO

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 17 November 2003. Cochrane reviews are regularly checked and updated if necessary.

Background: Abnormal mood is an important consequence of stroke and may affect recovery and outcome. However, depression and anxiety are often not detected or inadequately treated. This may in part be due to doubts about whether antidepressant treatments commenced early after the onset of stroke will prevent depression and improve outcome.

Objectives: To determine if pharmaceutical or psychological interventions can prevent the onset of depression, including depressive illness and abnormal mood, and improve physical and psychological outcomes, in patients with stroke.

Search strategy: We searched the Cochrane Stroke Group trials register (June 2003). In addition we searched the following electronic databases: Cochrane Central Register of Controlled Trials (The Cochrane Library, Issue 3, 2002), MEDLINE (1966 to September 2002), EMBASE (1980 to September 2002), CINAHL (1982 to September 2002), PsychINFO (1967 to September 2002), Applied Science and Technology Plus (1986 to September 2002), Arts and Humanities Index (1991 to September 2002), Biological Abstracts (1969 to September 2002), General Science Plus (1994 to September 2002), Science Citation Index (1992 to September 2002), Social Sciences Citation Index (1991 to September 2002), and Sociofile (1974 to September 2002). Reference lists from relevant articles and textbooks were searched, and authors of known studies and pharmaceutical companies who manufacture psychotropic medications were contacted.

Selection criteria: Randomised and quasi-randomised controlled trials comparing different types of pharmaceutical agents (eg selective serotonin reuptake inhibitors) with placebo, or various forms of psychotherapy against standard care (or attention control), in patients with a recent clinical diagnosis of stroke, where the treatment was undertaken with the explicit intention of preventing depression.

Data collection and analysis: The primary analyses focussed on the proportion of patients who met the standard diagnostic criteria for depression applied in the trials at the end of follow-up. Secondary outcomes included depression or mood scores on standard scales, disability or physical function, death, recurrent stroke, and adverse effects.

Main results: Twelve trials involving 1245 participants were included in the review. Data were available for nine trials (11 comparisons) involving different pharmaceutical agents, and three trials of psychotherapy. The time from stroke onset to entry ranged from a few hours to six months, but most patients were recruited within one month of acute stroke. The duration of treatments ranged from two weeks to one year. There was no clear effect of pharmacological therapy on the prevention of depression or on other measures. A significant improvement in mood was evident for psychotherapy, but this treatment effect was small and from a single trial. There was no effect on diagnosed depression.

Reviewers' conclusions: This review identified a small but significant effect of psychotherapy on improving mood, but no effect of either pharmacotherapy or psychotherapy on the prevention of depressive illness, disability, or other outcomes. More evidence is therefore required before any recommendations can be made about the routine use of such treatments to improve recovery after stroke.

MDD, Escitalopram & Venlafaxine

▪ A DOUBLE-BLIND COMPARISON OF ESCITALOPRAM AND VENLAFAXINE EXTENDED RELEASE IN THE TREATMENT OF MAJOR DEPRESSIVE DISORDER.

Authors : Bielski RJ, Ventura D, Chang CC. - Summit Research Network, Okemos, Mich. (Dr. Bielski); and Forest Laboratories, Inc., New York, N.Y. (Drs. Ventura and Chang).

Source : J Clin Psychiatry. 2004 Sep;65(9):1190-6

Summary: Escitalopram is the most selective serotonin reuptake inhibitor (SRI) antidepressant available. Venlafaxine is a non-selective SRI that also inhibits noradrenergic reuptake. This study compared escitalopram and venlafaxine extended release (XR) in depressed outpatients at the highest doses recommended in the United States. **METHOD:** In this randomized trial, patients (diagnosis of DSM-IV-defined major depressive disorder; baseline Hamilton Rating Scale for Depression score of ≥ 20) received 1 week of single-blind placebo treatment, followed by 8 weeks of double-blind, fixed-dose treatment with either escitalopram or venlafaxine XR (rapidly titrated to 20 mg/day and 225 mg/day, respectively, in accordance with prescribing information). The primary efficacy variable was change from baseline to week 8 in Montgomery-Asberg Depression Rating Scale (MADRS) total score. Data were collected from May to December 2002. **RESULTS:** Mean baseline MADRS scores for the escitalopram (N = 97) and venlafaxine XR (N = 98) groups were 30.7 and 30.0, respectively. There were no significant differences in measures of efficacy between the 2 antidepressants. Mean changes from baseline to endpoint in MADRS total score for escitalopram and venlafaxine XR were -15.9 and -13.6, respectively. Remission (MADRS score of ≤ 10) rates at endpoint were 41.2% for escitalopram and 36.7% for venlafaxine XR. Response ($\geq 50\%$ reduction from baseline MADRS score) rates for the escitalopram and venlafaxine XR groups were 58.8% and 48.0%, respectively. Tolerability measures favored escitalopram over venlafaxine XR treatment. The venlafaxine XR group had a higher incidence than the escitalopram group of treatment-emergent adverse events (85.0% vs. 68.4%) and discontinuation due to adverse events (16.0% vs. 4.1%; $p < .01$). **CONCLUSION:** Results of this study indicate that, when titrated rapidly to their maximum recommended doses, escitalopram is at least as effective as venlafaxine XR and significantly better tolerated. These results do not support the hypothesis that nonselective SRIs have greater efficacy than selective SRIs.

MDD & Emotional Information

▪ EMOTIONAL INFORMATION PROCESSING IN FIRST AND RECURRENT MAJOR DEPRESSIVE EPISODES.

Authors : Nandrino JL, Dodin V, Martin P, Henniaux M. - Department of Psychology, UPRES 2453, Domaine Universitaire du pont de Bois, University of Lille 3, F-59653 Villeneuve d'Ascq cedex, France

Source : J Psychiatr Res. 2004 Sep-Oct;38(5):475-84

Summary: Depressive states are classically associated to increased sensitivity to negative events. However this hypersensitivity may not be stable in time, being absent in remission periods or further reinforced with recurrent depressive episodes, or may concern positive stimuli instead, e.g. in young depressive patients. To study the evolution of the

processing of emotional information in depression we recorded late components of evoked potentials in first-episode and recurrent depressed patients before and after recovery. We used a visual attentional paradigm manipulating the processing of emotional information. Subjects first counted words with positive valence, and then words with negative valence from lists of usual words. The results showed that recurrent patients had increased P300 amplitudes for negative words selection only in negative words counting situation, while first-episode patients had decreased P300 amplitudes for positive words selection. After clinical improvement, the negative biases in recurrent patients group disappeared but P300 amplitudes of first-episode patients remained significantly low for positive words. First-episode depressed patients show a selective impairment for positive stimuli, with decreased response to pleasant stimuli, while recurrent depressive subjects show signs of hyperesthesia for negative stimuli. These results suggest that responses to emotional stimuli in word processing are related to the duration of the mood disorders.

MDD & Electroencephalographic Sleep

▪ ELECTROENCEPHALOGRAPHIC SLEEP PROFILES IN TREATMENT COURSE AND LONG-TERM OUTCOME OF MAJOR DEPRESSION: ASSOCIATION WITH DEX/CRH-TEST RESPONSE

Authors : Hatzinger M, Hemmeter UM, Brand S, Ising M, Holsboer-Trachsler E. - Psychiatric University Hospital, Depression Research Unit, Wilhelm Klein-Str. 27, CH-4025 Basel, Switzerland

Source : J Psychiatr Res. 2004 Sep-Oct;38(5):453-65

Summary: Altered electroencephalographic (EEG) sleep patterns are among the most prominent neurobiological findings in depression. Several of these alterations have been suggested to be associated with an unfavorable long-term outcome. However, the impact of pathological sleep parameters on a more recurrent course of illness or vice versa still warrants clarification. Underlying mechanisms may involve systems known to be related to both sleep regulation and long-term course of depression such as the hypothalamic-pituitary-adrenocortical (HPA) axis. Thus, EEG sleep profiles of patients with depression were examined to determine whether (1) the retrospective clinical course of depression, and (2) the prospective long-term outcome in follow-up are associated with EEG sleep parameters. To elucidate related mechanisms HPA system functioning was evaluated by using the combined DEX/CRH test. Fifteen patients with affective disorders who participated in an earlier controlled antidepressant treatment study over 6 weeks were consecutively enrolled in an exploratory follow-up study. The retrospective analysis revealed that during the acute state of depression predominantly sleep continuity measures were associated with the number of previously experienced episodes. While this relation disappeared during treatment and did not correlate with the prospective course, decreased slow wave sleep variables especially in the first sleep period and increased rapid eye movement density were predictive for the occurrence of recurrences in follow-up and, hence, probably reflect more trait-like markers. Additionally, EEG sleep variables unfavorable for long-term outcome were related to excessive stress hormone response in the DEX/CRH-test. These disturbances may reflect important mechanisms responsible of causing and maintaining the disease process of

depression.

MDD & Medical Illness

■ COMORBID MEDICAL ILLNESS AND RELAPSE OF MAJOR DEPRESSIVE DISORDER IN THE CONTINUATION PHASE OF TREATMENT.

Authors : Iosifescu DV, Nierenberg AA, Alpert JE, Papakostas GI, Perlis RH, Sonawalla S, Fava M. - Depression Clinical and Research Program, Psychiatry Department, Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA. diosifescu@partners.org

Source : Psychosomatics. 2004 Sep-Oct;45(5):419-25

Summary: The authors examined the impact of comorbid medical illness on the rate of relapse of major depressive disorder during continuation therapy. Subjects (N = 128) with major depressive disorder (according to DSM-III-R criteria) achieved clinical remission (a 17-item Hamilton Depression Rating Scale score ≤ 7) after 8 weeks of treatment with fluoxetine and entered the continuation phase of antidepressant treatment. They used the Cumulative Illness Rating Scale to measure the severity of comorbid medical illness. Eight patients (6.3%) relapsed during the 28-week continuation phase. With logistic regression, the total burden and the severity of comorbid medical illness significantly predicted the relapse of major depressive disorder during continuation therapy with fluoxetine. Greater medical comorbidity was also associated with higher increases in self-reported symptoms of depression, anxiety, and anger during the follow-up.

MDD, Venlafaxine & SSRI

■ COST AND EFFECTIVENESS OF VENLAFAXINE EXTENDED-RELEASE AND SELECTIVE SEROTONIN REUPTAKE INHIBITORS IN THE ACUTE PHASE OF OUTPATIENT TREATMENT FOR MAJOR DEPRESSIVE DISORDER

Authors : Trivedi MH, Wan GJ, Mallick R, Chen J, Casciano R, Geissler EC, Panish JM. - University of Texas Southwestern Medical Center, Dallas, TX; daggerWyeth Research, Global Health Outcomes Assessment, Collegeville, PA and double daggerThe Analytica Group, New York, NY.

Source : J Clin Psychopharmacol. 2004 Oct;24(5):497-506

Summary: The purpose of this retrospective analysis was to estimate the cost and effectiveness of venlafaxine extended-release (VXR) compared with selective serotonin reuptake inhibitors in the outpatient treatment of major depressive disorder. **METHODS:** Pooled data from 8, 8-week, randomized, double-blind studies comparing treatment of major depressive disorder with venlafaxine/venlafaxine XR (n = 851), selective serotonin reuptake inhibitors (fluoxetine, paroxetine, fluvoxamine; n = 748), or placebo (4 studies; n = 446) were retrospectively analyzed to determine the economic implications of symptom remission from the perspective of a US third party payer and that of an employer. A decision modeling approach was used to determine cost and effectiveness ratios. **RESULTS:** Patients on VXR were associated with 22.8 depression-free days versus 18.6 depression-free days with the studied selective serotonin reuptake inhibitors, based on the decision model. Productive and quality-adjusted days were also expected to increase for

VXR patients (22.06 vs. 19.34 and 4.56 to 9.36 vs. 3.72 to 7.63), as was the percentage of patients achieving full activity (25.9% vs. 19.6%). The expected cost per patient achieving remission of symptoms was US\$1303.94 and US\$1514.96, and the cost per depression-free days was US\$25.66 and US\$28.25, for the VXR and selective serotonin reuptake inhibitors groups, respectively. **CONCLUSIONS:** Treatment with VXR is not only expected to increase the rate of remission of symptoms but is also associated with achievement of full activity, higher number of depression-free days, productive days, and quality-adjusted days. VXR is a cost-effective treatment option for major depressive disorder.

MDD, Fluoxetine & Psychosocial Functioning

■ PSYCHOSOCIAL FUNCTIONING DURING THE TREATMENT OF MAJOR DEPRESSIVE DISORDER WITH FLUOXETINE.

Authors : Papakostas GI, Petersen T, Denninger JW, Tossani E, Pava JA, Alpert JE, Nierenberg AA, Fava M. - Depression Clinical and Research Program, Massachusetts General Hospital, Harvard Medical School, Boston, MA.

Source : J Clin Psychopharmacol. 2004 Oct;24(5):507-11

Summary: Major depressive disorder (MDD) is associated with significant disability, having a profound impact on psychosocial functioning. Therefore, studying the impact of treatment on psychosocial functioning in MDD could help further improve the standard of care. **METHODS:** Two hundred twenty-two MDD outpatients were treated openly with 20 mg fluoxetine for 8 weeks. The self-report version of the Social Adjustment Scale was administered at baseline and during the final visit. We then tested for the relationships between (1) self-report version of the Social Adjustment Scale scores at baseline and clinical response, (2) nonresponse, response and remission status and overall psychosocial adjustment at end point, (3) the number/severity of residual depressive symptoms and overall psychosocial adjustment at end point in responders, and (4) the time to onset of response and overall psychosocial adjustment at end point. **RESULTS:** An earlier onset of clinical response predicted better overall psychosocial functioning at end point (P = 0.0440). Responders (n = 128) demonstrated better overall psychosocial adjustment at end point than nonresponders (P = 0.0003), while remitters (n = 64) demonstrated better overall psychosocial adjustment at end point than nonremitted responders (P = 0.0031). In fact, a greater number/severity of residual symptoms predicted poorer overall psychosocial adjustment at end point in responders (P = 0.0011). Psychosocial functioning at baseline did not predict response. **CONCLUSIONS:** While MDD patients appear equally likely to respond to treatment with fluoxetine, regardless of their level of functioning immediately before treatment, the above results stress the importance of achieving early symptom improvement then followed by full remission of depressive symptoms with respect to restoring psychosocial functioning in MDD.

MDD & Treatment

■ DEPRESSED PATIENTS MAY NEED TREATMENT FOR BOTH PHYSICAL AND EMOTIONAL SYMPTOMS

Source : Blackwell Publishing Ltd

Summary: Physical symptoms (such as headache, back pain, stomach problems, joint or muscle pains, and dizziness) are

nearly as common in depression as emotional symptoms and are the predominant complaint depressed patients present with in the primary care setting.

A study published in the Journal of General Internal Medicine examined the prevalence, impact on quality of life, and outcome of physical symptoms in depressed patients during nine months of antidepressant therapy. While physical symptoms showed, on average, some improvement with antidepressant treatment, the improvement was typically less than for emotional symptoms. The physical symptoms showed the greatest improvement during the initial month of treatment. In contrast, depression continued to show gradual improvements over the 9-month period. Unlike depression, however, improvement in physical symptoms typically plateaus with minimal resolution in subsequent months.

Therefore, it is important to recognize the physical symptoms that commonly co-exist with depression and, if they fail to improve during the first month of treatment, to consider additional therapies. Corresponding author, Dr. Kroenke states, "It is important to ask patients with depression about physical symptoms at the start of treatment and when assessing improvement ask about physical as well as emotional symptoms."

MDD & Milnacipran

▪ PREDICTION OF ANTIDEPRESSANT RESPONSE TO MILNACIPRAN BY NOREPINEPHRINE TRANSPORTER GENE POLYMORPHISMS.

Authors : Yoshida K, Takahashi H, Higuchi H, Kamata M, Ito K, Sato K, Naito S, Shimizu T, Itoh K, Inoue K, Suzuki T, Nemeroff CB. - Department of Psychiatry, Akita University School of Medicine, 1-1-1 Hondo, Akita 010-8543, Japan. cxw01076@nifty.com

Source : Am J Psychiatry. 2004 Sep;161(9):1575-80

Summary: OBJECTIVE: With a multitude of antidepressants available, predictors of response to different classes of antidepressants are of considerable interest. The purpose of the present study was to determine whether norepinephrine transporter gene (NET) and serotonin transporter gene (5-HTT) polymorphisms are associated with the antidepressant response to milnacipran, a dual serotonin/norepinephrine reuptake inhibitor. METHOD: Ninety-six Japanese patients with major depressive disorder were treated with milnacipran, 50-100 mg/day, for 6 weeks. Severity of depression was assessed with the Montgomery-Asberg Depression Rating Scale. Assessments were carried out at baseline and at 1, 2, 4, and 6 weeks of treatment. The method of polymerase chain reaction was used to determine allelic variants. RESULTS: Eighty patients completed the study. The presence of the T allele of the NET T-182C polymorphism was associated with a superior antidepressant response, whereas the A/A genotype of the NET G1287A polymorphism was associated with a slower onset of therapeutic response. In contrast, no influence of 5-HTT polymorphisms on the antidepressant response to milnacipran was detected. CONCLUSIONS: The results suggest that NET but not 5-HTT polymorphisms in part determine the antidepressant response to milnacipran.

DEPRESSION IN MEN

▪ DEPRESSION IN MEN ATTENDING A RURAL GENERAL PRACTICE: FACTORS ASSOCIATED WITH PREVALENCE OF

DEPRESSIVE SYMPTOMS AND DIAGNOSIS.

Authors : Shiels C, Gabbay M, Dowrick C, Hulbert C. - Department of Primary Care, Whelan Building, University of Liverpool, Liverpool L69 3GB, UK. cs50@liv.ac.uk

Source : Br J Psychiatry. 2004 Sep;185:239-44

Summary: BACKGROUND: Doctors are less likely to diagnose depression in men than in women. Little research has been conducted to explore the underlying reasons for this in rural settings, or to compare primary care doctors' and male patients' ratings of perceived depression. AIMS: To identify symptomatic and socio-demographic correlates of depression in men attending a rural practice, and to compare and contrast general practitioners' and patients' assessments of depression. METHOD: All male patients of working age attending a rural general practice over a 12-month period were invited to participate. RESULTS: Men reporting recent "chest pain" or "feeling tired/little energy", expressing low job enjoyment or with a previous diagnosis of depression were more likely to be scored above threshold on the Hospital Anxiety and Depression Scale-Depression sub-scale. There was little agreement between the doctors and their male patients about the degree of perceived depression. CONCLUSIONS: Educational interventions aimed at addressing the diagnosis of depression in men should take greater account of factors within a particular social setting.

Bipolar Disorder (BD)

BD & Bipolar Spectrum Disorder

▪ BIPOLAR SPECTRUM DISORDER: A PILOT STUDY

Authors : Ghaemi SN, Hsu DJ, Ko JY, Baldassano CF, Kontos NJ, Goodwin FK. Bipolar Disorder Research Program, Department of Psychiatry, Cambridge Health Alliance, Cambridge, Mass., USA.

Source : Psychopathology. 2004 Sep 7;37(5):222-226. Epub 2004 Sep 07

Summary : Objective: To assess depressive features of a proposed definition of bipolar spectrum disorder (BSD). Methods: Thirty-six patients with bipolar disorder type I or II were compared to 37 patients with unipolar major depressive disorder through patient interview and chart review. Results: Univariate analysis suggests that 7 of 12 (recurrent major depressive episodes, brief major depressive episodes, atypical depressive symptoms, early age of onset, family history of bipolar disorder, antidepressant tolerance, and antidepressant-induced mania) features of major depressive episodes were more likely to occur in bipolar versus unipolar patients. After adjustment in a multivariable regression model, however, the five most powerful predictors of bipolar disorder were brief major depressive episodes, early age of onset, antidepressant-induced mania, postpartum depression, and atypical depressive symptoms. Conclusions: This preliminary study supports the idea that bipolar disorder is characterized by some depressive features less likely to be found in unipolar depression. Further prospective study needs to be conducted comparing BSD with unipolar depression.

BD & Depression With Versus

▪ DEPRESSION WITH VERSUS WITHOUT MANIC FEATURES IN RAPID-CYCLING BIPOLAR DISORDER.

Authors : Goldberg JF, Wankmuller MM, Sutherland KH. *Bipolar Disorders Research Program, Department of Psychiatry Research, Zucker Hillside Hospital, North Shore Long Island Jewish Health System, Glen Oaks, NY; daggerDepartment of Psychiatry, Weill Medical College of Cornell University, New York, NY; double dagger Department of Psychiatry, New York Presbyterian Hospital-Payne Whitney Clinic, New York, NY; and section signDepartment of Psychology, Long Island University, Brooklyn, NY.

Source : J Nerv Ment Dis. 2004 Sep;192(9):602-606.

Summary : Depression has been identified as a hallmark feature of rapid-cycling bipolar disorder, although less attention has been paid to the presence of manic features accompanying depression in rapid cyclers. To provide greater information about the extent to which depression arises with or without salient manic features in rapid cycling, we conducted a preliminary study of rapid cycling in outpatients seeking treatment at an academic specialty center for bipolar disorder. Forty DSM-IV affectively symptomatic bipolar outpatients with past year DSM-IV rapid cycling underwent systematic evaluation of symptoms and illness characteristics. Manic and depressive symptoms, treatments, and clinical features were rated by standardized scales. Major depression was present in most rapid cyclers (85%), but salient manic features were also evident in half of all depressed rapid cyclers. A lifetime history of suicide attempts was significantly more common in rapid cyclers who presented with major depression plus salient manic features than in those who presented with pure depression or pure mania ($p = .033$).

Antidepressants were being prescribed for approximately one third of depressed rapid cycling patients regardless of the presence of concomitant manic features, whereas mood stabilizers tended to be used less often when manic features accompanied depression. Depression in conjunction with manic symptoms, rather than pure depression alone, may be more common among rapid-cycling bipolar patients who seek treatment. Lifetime suicide risk may be greater among rapid cycling patients whose depression occurs in tandem with manic symptoms. Prescribing habits in the community that favor antidepressants over mood stabilizers may promote further mood destabilization in this population. Further studies with larger sample sizes are needed to affirm these provisional findings.

BD & Affective Disorders

▪ AFFECTIVE DISORDERS SPECIFIC TO AGEING

Authors : Pellerin J. Service de psychiatrie du sujet age, groupe hospitalier Charles Foix, 94205 Ivry-sur-Seine Cedex. jerome.pellerin@cfx.ap-hop-paris.fr

Source : Rev Prat. 2004 Apr 15;54(7):717-24.

Summary : With time, affects evolution can lead old people to a pathological organisation of their own mental universe. A general feeling of ill-being (syndrome of ageing badly) may appears and must be differentiated from an usual depressive syndrome. Post-traumatic disorders indicate a current or an old inability to metabolise painful life events. The "syndrome de glissement" (failure-to-thrive) generate not only bedridden state often irreversible but also guilt or depressive symptoms in caregivers. Those particular forms of ageing determine the relation with the practitioner and can induce feeling of dissatisfaction or hostility.

BD, VALPROATE & ACUTE MANIA

▪ VALPROATE FOR ACUTE MOOD EPISODES IN BIPOLAR DISORDER

Authors : Macritchie K, Geddes JR, Scott J, Haslam D, de Lima M, Goodwin G.

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 08 September 2002. Cochrane reviews are regularly checked and updated if necessary.

Background: Valproate has become a leading adjunctive and alternative treatment to lithium in bipolar disorder.

Objectives: To determine the efficacy and acceptability of valproate in acute episodes of bipolar disorder.

Search strategy: Registers and databases: Cochrane Collaboration Depression, Anxiety and Neurosis Controlled Trials Registrar (version 2-2002) Cochrane Controlled Clinical Trials Register (3-2002) Medline (1966-January 1999) PsychLit (1996-June 1999) Embase (1980-January 1999) Reference lists of relevant papers/books. Trial authors, experts and pharmaceutical companies. Handsearches (specialist journals and conference proceedings, listed below)

Selection criteria: Randomised controlled trials comparing valproate with placebo and other medications for any acute episode. Bipolar patients, male and female, of all ages were included.

Data collection and analysis: Data extraction and methodological quality assessment were each performed independently by two reviewers. For analysis, relative risk was used for binary efficacy outcomes and the weighted mean difference or standardised mean difference used for continuously distributed outcomes.

Main results: Ten trials compared valproate with other interventions in mania. None examined depression or mixed affective episodes. Data were extracted on 'failure to respond by the end of the study' (i.e. less than 50% reduction in Young Mania Rating Scale or SADS-S mania scale). Three trials (316 participants) compared valproate with placebo. Three trials (158 participants) compared valproate with lithium. Two trials (363 participants) compared valproate with olanzapine. One trial (36 participants) compared valproate with haloperidol. Two trials (59 patients) compared valproate with carbamazepine. Treatment acceptability was estimated by the 'total number withdrawing from the study'. Three trials (321 patients) compared valproate and placebo, two (144 patients) compared valproate with lithium. One study (30 patients) compared valproate and carbamazepine. Pooled relative risks (95% confidence intervals) were calculated using fixed effect. Valproate was more efficacious than placebo (RRR 38%; RR 0.62; 95% C.I. 0.51 to 0.77) in the treatment of mania. There was no significant difference between valproate and lithium (RRI 5%; RR 1.05; 95% C.I. 0.74-1.50) or between valproate and carbamazepine (RRR 34%; RR 0.66; 95% C.I. 0.38 to 1.16). Valproate was less effective than olanzapine (failure to achieve clinical response; RRI 25%; RR 1.25, 95% C.I. 1.01 to 1.54; average of 2.8 point less change on the Mania Rating Scale (95% CI 0.83 to 4.79). There were no significant differences in those withdrawing from the study. Reviewers' conclusions: There is consistent, if limited, evidence that valproate is an efficacious treatment for acute mania. Valproate may be less efficacious than olanzapine. More, rigorously designed, trials over the full range of acute affective episodes are required.

Bipolar Disorder

■ IMPACT OF FAMILY BURDEN AND AFFECTIVE RESPONSE ON CLINICAL OUTCOME AMONG PATIENTS WITH BIPOLAR DISORDER.

Authors : Perlick DA, Rosenheck RA, Clarkin JF, Maciejewski PK, Sirey J, Struening E, Link BG. the Yale University School of Medicine in New Haven, Connecticut.

Source : Psychiatr Serv. 2004 Sep;55(9):1029-35

Summary: This study evaluated the direct and indirect effects of family burden and affective response on medication adherence and outcome among patients with bipolar disorder. **METHODS:** Data were examined for 126 patients who were consecutively admitted to the psychiatric service at a university-affiliated hospital and who met research diagnostic criteria for bipolar I or II disorder or for schizoaffective disorder, manic type, and their family caregivers. A total of 101 pairs of patients and family caregivers (80 percent) completed 15 months of study and were included in the analyses. Patients and their identified caregivers were assessed within two weeks of either discharge from the index inpatient admission or initiation of outpatient treatment (baseline assessment). Patients and caregivers were also assessed seven and 15 months after the baseline assessment. Structural equation modeling was used to evaluate caregivers' influences on patients' medication adherence seven months after baseline and on clinical outcome 15 months after baseline. **RESULTS:** The indexes of overall fit for the path model confirmed the a priori measurement model. Significant paths were found from the caregiver's perceived burden at baseline to the caregiver's emotional overinvolvement at baseline, from the caregiver's emotional overinvolvement at baseline to the patient's medication adherence at the seven month follow-up, and from the patient's medication adherence at the seven-month follow-up to the patient's outcome at the 15-month follow-up. The paths from the caregiver's perceived burden at baseline to the patient's medication adherence seven months after baseline and the patient's outcome 15 months after baseline were not significant. **CONCLUSIONS:** When caregivers of patients with bipolar illness experience a high burden, patient outcome is adversely affected. This relationship is mediated through families' affective response and patients' medication adherence.

Bipolar I Disorder & Olanzapine

■ USE OF OLANZAPINE IN THE TREATMENT OF BIPOLAR I DISORDER

Authors : DZ Lieberman & FK Goodwin

Source : Expert Review of Neurotherapeutics 4(5),759-767 (2004)

Summary: Olanzapine (Zyprexa®, Eli Lilly & Co.) is an atypical antipsychotic medication with once-daily dosing that was originally developed for the treatment of schizophrenia. It has shown broad efficacy in the treatment of bipolar mixed and manic episodes, but is less effective in the treatment of bipolar depression. Double-blind studies have demonstrated a rapid onset of action in acute bipolar mania, significantly greater rates of response compared with placebo, and a remission rate of 88.3% in a 49-week open-label study. Diverse presentations of the illness responded well to olanzapine including patients with rapid-cycling bipolar disorder, mixed episodes, as well as psychotic and

nonpsychotic manias. Olanzapine monotherapy improved symptoms of depression related to its sedating and appetite-enhancing profile, but core symptoms such as depressed mood did not improve significantly. However, in combination with fluoxetine, bipolar depressed patients responded without an increased risk of mania. Weight gain and sedation are prominent adverse effects, and it has been associated with atherogenic dyslipidemia and glucose intolerance. bipolar disorder, depression, divalproex, fluoxetine, lithium, mania, manic depressive illness, olanzapine atypical antipsychotics.

BD & Antidepressants

■ ANTIDEPRESSANTS FOR BIPOLAR DEPRESSION: A SYSTEMATIC REVIEW OF RANDOMIZED, CONTROLLED TRIALS.

Authors : Gijsman HJ, Geddes JR, Rendell JM, Nolen WA, Goodwin GM. - Scutari Clinic, St. Thomas' Hospital, Lambeth Palace Rd., London, U.K. harm.gijsman@doctors.net.uk

Source : Am J Psychiatry. 2004 Sep;161(9):1537-47

Summary: **OBJECTIVE:** This study reviewed the evidence from randomized, controlled trials on the efficacy and safety of antidepressants in the short-term treatment of bipolar depression. **METHOD:** The authors performed a systematic review and meta-analysis of randomized, controlled trials. They searched the Cochrane Collaboration Depression, Anxiety, and Neurosis Controlled Trials Register, incorporating results of searches of MEDLINE, EMBASE, CINAHL, PsycLIT, PSYINDEX, and LILACS. The main outcome measures were the proportion of patients who clinically responded to treatment and the rate of switching to mania. **RESULTS:** Twelve randomized trials were included, with a total of 1,088 randomly assigned patients. Five trials compared one or more antidepressants with placebo: 75% of these patients were receiving a concurrent mood stabilizer or an atypical antipsychotic. Antidepressants were more effective than placebo. Antidepressants did not induce more switching to mania (the event rate for antidepressants was 3.8% and for placebo, it was 4.7%). Six trials allowed comparison between two antidepressants. The rate of switching for tricyclic antidepressants was 10%, and for all other antidepressants combined, it was 3.2%. **CONCLUSIONS:** Antidepressants are effective in the short-term treatment of bipolar depression. The trial data do not suggest that switching is a common early complication of treatment with antidepressants. It may be prudent to use a selective serotonin reuptake inhibitor or a monoamine oxidase inhibitor rather than a tricyclic antidepressant as first-line treatment. Given the limited evidence, there is a compelling need for further studies with longer follow-up periods and careful definition and follow-up of emerging mania and partial remission.

BD & STED

■ STRATEGIES FOR IMPROVING TREATMENT OF BIPOLAR DISORDER: INTEGRATION OF MEASUREMENT AND MANAGEMENT.

Authors : Sachs GS.

Source : Acta Psychiatr Scand Suppl. 2004 Sep;(422):7-17

Summary: Bipolar disorder is a common and complex condition associated with high rates of disability and high health care costs. The aim of this article is to provide an

overview of the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD). Method: The Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD) was conceived in response to an NIMH request for proposals to study the effectiveness of treatments for Bipolar Disorder. Aspects of this program have been adapted and enriched for presentation in this paper. Result: Designed for implementation in routine practice across a variety of settings, STEP-BD offers a disease management program in which standardized assessments are linked to critical decision points in clinical management pathways. Conclusion: This paper describes strategies used in STEP-BD to improve the treatment of Bipolar disorder: a simple conceptual model, which integrates assessments and management, and several specialized elements, used in the STEP-BD assessment package.

BD & New Medication

■ NEW MEDICATION TREATMENT OPTIONS FOR BIPOLAR DISORDERS.

Authors : Ketter TA, Wang PW, Nowakowska C, Marsh WK. - Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Stanford, California, USA

Source : Acta Psychiatr Scand Suppl. 2004 Sep;(422):18-33

Summary: Objective: To assess new treatment options for bipolar disorders. Method: Controlled studies of new treatments for bipolar disorders were identified by computerized searches and reviews of scientific meeting proceedings, and were compiled by drug category. Results: Two main categories of medications, newer anticonvulsants and newer antipsychotics, are yielding emerging new treatment options for bipolar disorders. Newer anticonvulsants have diverse psychotropic profiles, and although not generally effective for acute mania, may have utility for other aspects of bipolar disorders (e.g. lamotrigine for maintenance or acute bipolar depression), or for comorbid conditions (e.g. gabapentin for anxiety or pain, topiramate for obesity, bulimia, alcohol dependence, or migraine, and zonisamide for obesity). In contrast, newer antipsychotics generally appear effective for acute mania, and some may ultimately prove effective in acute depression (e.g. olanzapine combined with fluoxetine, quetiapine) and maintenance (e.g. olanzapine). Conclusion: Emerging research is yielding new treatment options for bipolar disorders and comorbid conditions.

BD & Psychological Interventions

■ PSYCHOLOGICAL INTERVENTIONS IN BIPOLAR DISORDER: FROM WISHFUL THINKING TO AN EVIDENCE-BASED APPROACH.

Authors : Vieta E, Colom F. - Bipolar Disorders Program, Hospital Clinic, University of Barcelona, IDIBAPS, Stanley Medical Research Center, Barcelona, Spain.

Source : Acta Psychiatr Scand Suppl. 2004 Sep;(422):34-8

Summary: Objective: We aimed to examine the historical and current relevance of psychosocial approaches to bipolar illness by conducting a systematic review of prospective studies assessing the effectiveness of psychological interventions for bipolar disorder. Method: A systematic literature search was conducted using EMBASE, MedLine and PsychLIT and reference sections of papers were scrutinized for further relevant reports. Only four trials met the criteria of a

prospective study and achieved the necessary methodological standards. Results: The studies showed benefits for patients in terms of relapse prevention and the reduction of hospitalization rates. Psychoeducation (delivered in groups or as part of a family intervention) and cognitive behavioural therapy were also found to be effective prophylactic treatments for bipolar disorder in medicated patients. Other interventions do not appear to be supported by sufficient evidence. Conclusion: Psychological approaches, and particularly psychoeducation and cognitive-behavioural therapies, are evidence-based prophylactic therapies for bipolar patients receiving pharmacotherapy. They should be used as adjuncts to medication where possible in the prevention of bipolar disorder.

BD & SCALE MATTERS

■ SCALE MATTERS: THE NEED FOR A BIPOLAR DEPRESSION RATING SCALE (BDRS).

Authors : Berk M, Malhi GS, Mitchell PB, Cahill CM, Carman AC, Hadzi-Pavlovic D, Hawkins MT, Tohen M. - Barwon Health and The Geelong Clinic, Geelong, Australia.

Source : Acta Psychiatr Scand Suppl. 2004 Sep;(422):39-45

Summary: Objective: To briefly review the clinical and biological distinctions between unipolar and bipolar depression critiquing in particular currently available depression rating scales and discuss the need for a new observer-rated scale tailored to bipolar depression. Method: Relevant literature pertaining to the symptomatic differences between bipolar disorder and unipolar disorder as well as their measurement using existing assessment scales was identified by computerized searches and reviews of scientific journals known to the authors. Results: Bipolar depression is distinct from unipolar depression in terms of phenomenology and clinical characteristics. These distinguishing features can be used to identify bipolarity in patients that present with recurrent depressive episodes. This is important because current self-report and observer-rated scales are optimized for unipolar depression, and hence limited in their ability to accurately assess bipolar depression. Conclusion: The development of a specific bipolar depression rating scale will improve the assessment of bipolar depression in both research and clinical settings and assist the development of better treatments and interventions.

Bipolaroids

■ BIPOLARIDS: FUNCTIONAL IMAGING IN BIPOLAR DISORDER.

Authors : Malhi GS, Lagopoulos J, Owen AM, Yatham LN. - School of Psychiatry, The University of New South Wales, Australia

Source : Acta Psychiatr Scand Suppl. 2004 Sep;(422):46-54

Summary: Objective: To evaluate the literature pertaining to the use of functional magnetic resonance imaging (fMRI) in bipolar disorder research. Method: A search for papers published in English in journals from 1984 onwards was conducted using MedLine and EMBASE with the following terms: functional neuroimaging or fMRI and depression or bipolar disorder. In addition, retrieved papers and literature known to the authors was also scrutinized for further relevant reports. Results: The research findings from 26 articles are tabulated and the results from 10 articles dealing specifically with bipolar disorder are discussed in detail. Conclusion: fMRI

is a useful tool for investigating bipolar disorder. Preliminary studies point to trait and state abnormalities involving structures known to be associated with the generation and modulation of emotion. The patterns of fMRI activation are different to those found in healthy subjects and patients with major depression. FMRI studies are likely to provide valuable insights into the pathophysiology of bipolar disorder.

POST NATAL DEPRESSION (PND & PPD)

PND, OESTROGENS & PROGESTOGENS

■ OESTROGENS AND PROGESTOGENS FOR PREVENTING AND TREATING POSTNATAL DEPRESSION

Authors : Lawrie TA, Herxheimer A, Dalton K

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 13 January 1999. Cochrane reviews are regularly checked and updated if necessary.

Background: Postnatal depression, with a prevalence of at least 10%, is probably the most common complication of the puerperium. A deficiency or imbalance of sex hormones has repeatedly been suggested as a cause.

Objectives: The objective of this review was to evaluate the role of oestrogens and progestogens in the prevention and treatment of postnatal depression.

Search strategy: The register of clinical trials maintained and updated by the Cochrane Pregnancy and Childbirth Group.

Selection criteria: All trials were considered in which pregnant or postpartum women (up to 18 months) were randomised to receive postpartum oestrogen or progestogen or placebo for the treatment or prevention of postnatal depression. **Data collection and analysis:** Two published randomised placebo controlled trials were identified for inclusion in the analyses for this review. One study was excluded. **Main results:** Depot norethisterone enanthate given within 48 hours of delivery and lasting 8-12 weeks was associated with significantly higher postpartum depression scores than placebo. Oestrogen therapy in severely depressed women was associated with a greater improvement in depression scores than placebo. **Reviewers' conclusions:** There is no place for synthetic progestogens in the prevention or treatment of postnatal depression. Long-acting norethisterone enanthate is associated with an increased risk of postnatal depression. It and other long-acting progestogen contraceptives should be used with caution in the postnatal period, especially in women with a history of depression. The role of progesterone in the prevention and treatment of postnatal depression has yet to be evaluated in a randomised placebo-controlled trial. Oestrogen therapy may be of modest value at a late stage of severe postnatal depression. Its role in the prevention of recurrent postnatal depression has not been evaluated. Further research on its value is unlikely for ethical reasons.

PND & Antidepressant

■ ANTIDEPRESSANT TREATMENT FOR POST-NATAL DEPRESSION

Authors : Hoffbrand S, Howard L, Crawley H

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 12 January 2001. Cochrane reviews are regularly checked and updated if necessary.

Background: Postnatal depression is a common disorder, which can have profound short and long term effects on maternal morbidity, the new infant and the family as a whole. Social factors appear to be particularly important in the aetiology and prognosis of postnatal depression and treatment is often largely social support and psychological interventions. It is not known whether antidepressants are an effective and safe choice for treatment of this disorder.

Objectives: To evaluate the effectiveness of different antidepressant drugs and compare their effectiveness with other forms of treatment. To assess any adverse effects of antidepressants in the mother or the nursing baby.

Search strategy: The registers of clinical trials maintained and updated by the Cochrane Depression, Anxiety and Neurosis Group and the Cochrane Pregnancy and Childbirth Group were searched. Contact was made with pharmaceutical companies and experts in the field.

Selection criteria: All trials were considered in which women with depression in the first six months postpartum were randomised to receive antidepressants alone or in combination with another treatment, or to receive any other treatment including placebo.

Data collection and analysis: Data was extracted independently from the trial reports by the reviewers. Missing information was requested from investigators wherever possible. Data was sought to allow an "intention to treat" analysis.

Main results: Only one trial could be included in this review, leaving most of the objectives of the review unfulfilled. Appleby et al (1997) reported that Fluoxetine was significantly more effective than placebo and, after an initial session of counselling, as effective as a full course of cognitive-behavioural counselling in the treatment of postnatal depression. There was no interaction between medication and counselling.

Reviewers' conclusions: Women with postnatal depression can be effectively treated with fluoxetine, which is as effective as a course of cognitive-behavioural counselling in the short-term. However, more trials with a longer follow-up period are needed to compare different antidepressants in the treatment of postnatal depression, and to compare antidepressant treatment with psychosocial interventions. This is an area that has been neglected despite the large public health impact described above.

POSTPARTUM DEPRESSION (PPD)

■ TREATMENT OF POSTPARTUM DEPRESSION, PART 2: A CRITICAL REVIEW OF NONBIOLOGICAL INTERVENTIONS.

Authors : Dennis C.L.- University of Toronto, Toronto, Ontario, Canada

Source : J Clin Psychiatry. 2004 Sep;65(9):1252-65

Summary: While postpartum depression is a common mental condition with significant burden, it often remains undiagnosed and untreated. The objective of this article is to critically review the literature to determine the current state of scientific knowledge related to the treatment of postpartum depression from a nonbiological perspective. **DATA SOURCES:** Databases searched for this review included MEDLINE, PubMed, CINAHL, PsycINFO, EMBASE, ProQuest, the Cochrane Library, and the WHO Reproductive Health Library

from 1966 to 2003. The search terms used were postpartum/postnatal depression and randomized controlled/clinical trials. Published peer-reviewed articles in English from 1990 to 2003 were included in the review, although select earlier studies were also included based on good methodological quality and/or the absence of more recent work. **METHOD:** The criteria used to evaluate the interventions were based on the standardized methodology developed by the U.S. Preventive Services Task Force and the Canadian Task Force on Preventive Health Care. **RESULTS:** Twenty-one studies that met inclusion criteria were examined. These studies included interpersonal psychotherapy, cognitive-behavioral therapy, peer and partner support, nondirective counseling, relaxation/massage therapy, infant sleep interventions, infant-mother relationship therapy, and maternal exercise. Although some of these interventions have been better studied for depression unrelated to childbirth, methodological limitations render their efficacy equivocal for postpartum depression. **CONCLUSIONS:** Definite conclusions cannot be reached about the relative effectiveness of most of the nonbiological treatment approaches due to the lack of well-designed investigations. Randomized controlled trials are needed to compare different treatment modalities, examine the effectiveness of individual treatment components, and determine which treatments are most useful for women with different risk factors or clinical presentations of postpartum depression.

PRD & PPD

■ POSTPARTUM DEPRESSION RECURRENCE VERSUS DISCONTINUATION SYNDROME: OBSERVATIONS FROM A RANDOMIZED CONTROLLED TRIAL.

Authors : Sunder KR, Wisner KL, Hanusa BH, Perel JM. Departments of Psychiatry (Drs. Sunder and Wisner), Obstetrics and Gynecology and Reproductive Sciences, and Epidemiology (Dr. Wisner), and Women's Behavioral HealthCARE, Western Psychiatric Institute and Clinic, University of Pittsburgh Medical Center (Drs. Sunder, Wisner, and Perel); and the Center for Research on Health Care, Division of General Medicine, Department of Medicine, University of Pittsburgh (Dr. Hanusa), Pittsburgh, Pa.

Source : J. Clin Psychiatry. 2004 Sep;65(9):1266-1268.

Summary: To differentiate characteristics of a discontinuation syndrome from a recurrence of major depressive disorder in the context of a randomized trial. **METHOD:** We performed a randomized clinical trial to compare the efficacy of sertraline versus placebo for the prevention of recurrent postpartum DSM-IV major depressive disorder. Women whose depression did not recur in the initial 17-week active treatment trial were followed through the taper phase (weeks 18-20). At week 17, 3 women assigned to placebo and 8 assigned to sertraline remained in the trial. Nine symptoms that characterize discontinuation syndrome were extracted from the 25-item Asberg Rating Scale for Side Effects (ASE) and assessed weekly during the taper phase. The 21-item Hamilton Rating Scale for Depression was used to evaluate depressive symptoms. **RESULTS:** In the taper phase, there were no significant differences between the sertraline- and placebo-treated women on the sum of the ASE-derived symptoms. Both groups had low levels of symptoms on the ASE during the weeks of taper. None of the 3 women assigned to placebo and 2 of the 8 women assigned to sertraline suffered a depressive recurrence within 6 weeks of the end of the study.

CONCLUSIONS: A gradual taper of sertraline (75 mg) over 3 weeks did not lead to discontinuation syndrome; however, the systematic dissection of symptoms resulted in our conclusion that the duration of preventive therapy should be extended to 26 weeks (about 6 months) in subsequent randomized trials, consistent with the treatment guidelines for a single episode of depression.

PPD & TREATMENT

■ TREATMENT OF POSTPARTUM DEPRESSION, PART 1: A CRITICAL REVIEW OF BIOLOGICAL INTERVENTIONS.

Authors : Dennis CL, Stewart DE. - University of Toronto, Toronto, Ontario, Canada.

Source : J Clin Psychiatry. 2004 Sep;65(9):1242-51

Summary: While postpartum depression is a major health issue for many women from diverse cultures, this affective condition often remains undiagnosed and untreated. The objective of this article is to critically review the literature to determine the current state of scientific knowledge related to the treatment of postpartum depression from a biological perspective. **METHOD:** Databases searched for this review included MEDLINE, PubMed, CINAHL, PsycINFO, EMBASE, ProQuest, the Cochrane Library, and the WHO Reproductive Health Library from 1966 to 2003. The search terms used were postpartum/ postnatal depression and randomized controlled/ clinical trials in various combinations. Published peer-reviewed articles in English from 1990 to 2003 were chosen for review, although select earlier studies were also included based on good methodological quality and/or the absence of more recent work. The criteria used to evaluate the interventions were based on the standardized methodology developed by the U.S. Preventive Services Task Force and the Canadian Task Force on Preventive Health Care. **RESULTS:** Nine studies that met study criteria were examined. The interventions studied included antidepressant medication, estrogen therapy, critically timed sleep deprivation, and bright light therapy. Although some of these interventions have been better studied for depression unrelated to childbirth, methodological limitations render their efficacy equivocal for postpartum depression with limited strong evidence available to guide practice or policy recommendations. **CONCLUSIONS:** Despite the recent upsurge of interest in this area, many questions remain unanswered, resulting in diverse research implications. In view of the lack of randomized controlled trials, psychiatrists who are experts in the treatment of postpartum mood disorders have developed consensus guidelines. These guidelines will require regular updating as better and stronger evidence emerges.

PPD, PAROXETINE & CBT

■ THE USE OF PAROXETINE AND COGNITIVE-BEHAVIORAL THERAPY IN POSTPARTUM DEPRESSION AND ANXIETY: A RANDOMIZED CONTROLLED TRIAL.

Authors : Misri S, Reebye P, Corral M, Milis L. - Department of Psychiatry (Drs. Misri, Reebye, and Corral) and the Department of Obstetrics and Gynecology (Dr. Misri), Faculty of Medicine, University of British Columbia and Reproductive Mental Health Programs, St. Paul's Hospital and BC Women's Hospital and Health Centre (Drs. Misri and Corral and Ms. Milis), Vancouver, British Columbia, Canada.

Source : J Clin Psychiatry. 2004 Sep;65(9):1236-1241

Summary: Approximately 10% to 16% of women experience a major depressive episode after childbirth. A significant proportion of these women also suffer from comorbid anxiety disorders. The purpose of this study was to evaluate whether the addition of cognitive-behavioral therapy (CBT) to standard antidepressant therapy offers additional benefits in the treatment of post-partum depression with comorbid anxiety disorders. **METHOD:** Thirty-five women referred to a tertiary care hospital outpatient program with a DSM-IV diagnosis of postpartum depression with comorbid anxiety disorder were randomly assigned to 1 of 2 treatment groups-paroxetine-only monotherapy group (N = 16) or paroxetine plus 12 sessions of CBT combination therapy group (N = 19)-for a 12-week trial. Progress was monitored by a psychiatrist blinded to treatment group, using the Hamilton Rating Scale for Depression, Hamilton Rating Scale for Anxiety, Yale-Brown Obsessive Compulsive Scale, Clinical Global Impressions scale, and Edinburgh Postnatal Depression Scale. Data were analyzed using 2-tailed statistical tests at an alpha level of .05. The study was conducted from April 1, 2002, to June 30, 2003. **RESULTS:** Both treatment groups showed a highly significant improvement ($p < .01$) in mood and anxiety symptoms. Groups did not differ significantly in week of recovery, dose of paroxetine at remission, or measures of depression, anxiety, and obsessive-compulsive symptoms at outcome. **CONCLUSION:** Antidepressant monotherapy and combination therapy with antidepressants and CBT were both efficacious in reducing depression and anxiety symptoms. However, in this sample of acutely depressed/anxious postpartum women, there were no additional benefits from combining the 2 treatment modalities. Further research into the efficacy of combination therapy in the treatment of moderate-to-severe depression with comorbid disorders in postpartum women is recommended.

PPD & SEROTONERGIC FUNCTION

■ ALTERATIONS IN PLATELET SEROTONIN TRANSPORTER BINDING IN WOMEN WITH POSTPARTUM ONSET MAJOR DEPRESSION.

Authors : Newport DJ, Owens MJ, Knight DL, Ragan K, Morgan N, Nemeroff CB, Stowe ZN. -Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, 1365 Clifton Road NE, Suite B6100, Atlanta, GA 30322, USA.

Source : J Psychiatr Res. 2004 Sep-Oct;38(5):467-73

Summary: There is considerable debate as to whether postpartum depression (PPD) is biologically distinct from other depressive syndromes. Although abnormalities in serotonergic neural systems have repeatedly been reported in depression, few such studies have been conducted in PPD. In the present study, platelet serotonin transporter (SERT) binding was assessed using [3 H]paroxetine in 14 depressed pregnant women, 31 normal healthy pregnant women, 39 depressed postpartum women, and 27 normal healthy postpartum women; all of the subjects were drug-free. Significant differences were detected among the 4 groups with respect to the dissociation constant (K_d) of platelet binding sites for [3 H]paroxetine with the highest K_d values among those with PPD. The density (B_{max}) of platelet binding sites for [3 H]paroxetine did not differ between the study groups. These data suggest that PPD may be associated with unique

alterations in serotonergic function that are specific to the puerperium.

WOMEN MENTAL HEALTH (WMH)

BREASTFEEDING & SSRI

■ BREASTFEEDING DURING MATERNAL ANTIDEPRESSANT TREATMENT WITH SEROTONIN REUPTAKE INHIBITORS: INFANT EXPOSURE, CLINICAL SYMPTOMS, AND CYTOCHROME P450 GENOTYPES.

Authors : Berle Jy J, Steen VM, Aamo TO, Breilid H, Zahlisen K, Spigset O. - Centre for Child and Adolescent Mental Health (Dr. Berle) and the Dr. Einar Martens Research Group for Biological Psychiatry and Locus on Neuroscience, Center for Medical Genetics and Molecular Medicine (Drs. Steen and Breilid), University of Bergen, Bergen; the Center for Medical Genetics and Molecular Medicine, Haukeland University Hospital, Bergen (Drs. Steen and Breilid); and the Department of Clinical Pharmacology, St. Olav's University Hospital, Trondheim (Drs. Aamo, Zahlisen, and Spigset), Norway.

Source : J Clin Psychiatry. 2004 Sep;65(9):1228-1234

Summary: The aims of the study were to quantify the drug exposure in breastfed infants of antidepressant-treated mothers, to identify possible adverse events, and to correlate these variables to maternal and infant drug metabolism-relevant genotypes and milk triglyceride content. **METHOD:** The study included 25 lactating women treated with citalopram (N = 9), sertraline (N = 6), paroxetine (N = 6), fluoxetine (N = 1), or venlafaxine (N = 3) and their 26 breastfed infants. Drug concentrations in maternal and infant serum and milk were analyzed using liquid chromatography mass spectrometry methods; milk triglyceride levels were measured with a commercial kit. Cytochrome P450 (CYP) 2D6 and CYP2C19 activity was determined by polymerase chain reaction-based genotyping of the mothers and infants. An infant adverse event questionnaire was completed by the medication-treated mothers as well as by a control group of medication-free breastfeeding mothers of 68 infants. **RESULTS:** Sertraline and paroxetine were not detected in any of the drug-exposed infants. The infant serum level of citalopram was either undetectable (N = 4) or low (N = 6). All venlafaxine-exposed infants had measurable drug concentrations. We identified a paroxetine-treated mother and her infant who were both CYP2D6 poor metabolizers, as well as a citalopram-treated mother with CYP2C19 poor metabolizer status, but the serum drug levels of their infants were still either undetectable (paroxetine) or low (citalopram). There was no evidence of adverse events in the drug-exposed infants. **CONCLUSION:** Serum drug levels in breastfed infants of antidepressant-treated mothers were undetectable or low. This study adds further evidence to previously published data indicating that breastfeeding should not be generally discouraged in women using serotonin reuptake inhibitor anti-depressants.

MATERNAL DEPRESSED MOOD

■ IN THE SHADOW OF MATERNAL DEPRESSED MOOD: EXPERIENCES OF PARENTHOOD DURING THE FIRST YEAR AFTER CHILDBIRTH.

Authors : Seimyr L, Edhborg M, Lundh W, Sjogren B. -

Department of Woman and Child Health, Karolinska Institutet, Stockholm, Sweden. Louise.Seimyr@kbh.ki.se

Source : J Psychosom Obstet Gynaecol. 2004 Mar;25(1):23-34

Summary: To study the period and point prevalence of maternal depressive mood at three occasions before and after childbirth, and the relationship to the parents' psychosocial conditions and experiences of parenthood during the first year after childbirth. In a longitudinal community-based study, 434 pregnant women were invited to complete the Edinburgh Postnatal Depression Scale (EPDS) (cut-off score 9/10) at three time points. The parents' psychosocial conditions and experiences of parenthood were enquired at two months and at one year after childbirth, when the form Experience of Motherhood/Fatherhood Questionnaire (EMQ/EFQ) was applied. Three times measurement responses from both men and women were analyzed using non-parametric statistical methods and path-analysis. About 75% of the parents responded to the questionnaires. The period prevalence was 28%, and the point prevalence found on the three time points was EPDS I 21%, EPDS II 17% and EPDS III 12%. Correlations between antenatal and postnatal depressive symptoms were found, $r = 0.61$ and $r = 0.45$, respectively. Women, who experienced financial worries, lack of social support and losses and strains after childbirth showed more symptoms of depressed mood. The maternal depressive mood influenced negatively on breastfeeding and experiences of motherhood, but not on experiences of fatherhood. The partners of depressed women were neither more involved in childcare nor did they utilize paternal leave more than the other men. Both men and women reported the sexual life as negatively influenced by the women's depressed mood.

DEPRESSION & SEX HORMONES IN ELDERLY WOMEN

DEPRESSION AND SEX HORMONES IN ELDERLY WOMEN.

Authors : Erdinciler D, Bugay G, Ertan T, Eker E. - Geriatric Unit of the Internal Medicine Department, Cerrahpas? a Medical School, Istanbul University, Istanbul, Turkey.

Source : Arch Gerontol Geriatr. 2004 Nov-Dec;39(3):239-44

Summary: We aimed to study the relation between sex hormones and depression among elderly women. The study was carried out on 74 volunteered female subjects above 60 years of age. Each subject was asked to fulfill the geriatric depression scale (GDS) questionnaire and further evaluated for clinical depression by a psychiatrist using the DSM IV diagnostic criteria. For statistical analysis, subjects were later divided in two groups, according to the presence of clinical depression. Cognitive functions were assessed with the standardized mini mental test (SMMT).

Disability in the activities of daily living was assessed with instrumental activities of daily living (IADL) scale. Plasma levels of estrogen, testosterone, progesterone, and dehydroepiandrosterone sulfate (DHEA-S) were measured with chemiluminescent methods, and plasma levels of androstenedione were measured with radioimmunoassay. Among 74 subjects, 34 (39%) had clinical depression. Age, number of years spent in education, SMMT scores, and IADL scores did not differ between the depressive and non-depressive groups. Plasma sex hormone levels were not found to be different between the two groups.

EMOTIONAL PROBLEMS & LOW GESTATIONAL AGE

BEHAVIORAL AND EMOTIONAL ADJUSTMENT OF TEENAGERS IN MAINSTREAM SCHOOL WHO WERE BORN BEFORE 29 WEEKS' GESTATION.

Authors : Gardner F, Johnson A, Yudkin P, Bowler U, Hockley C, Mutch L, Wariyar U; Extremely Low Gestational Age Steering Group. - University of Oxford, Department of Social Policy and Social Work, 32 Wellington Square, Oxford, OX1 2ER, United Kingdom. frances.gardner@socres.ox.ac.uk

Source : Pediatrics. 2004 Sep;114(3):676-82

Summary: To investigate behavioral and emotional problems and positive adjustment of 15-to 16-year-olds who were born at extremely low gestational age (ELGA), from the perspective of parents, teachers, and teenagers. **METHODS:** Prospective follow-up was conducted of birth cohorts, with classroom control subjects. All infants who were born before 29 weeks in 1983-1984 (mean gestational age: 27 weeks) to mothers who resided in 3 regions of the United Kingdom were studied. A total of 82% (179 of 218) of survivors were traced at age 15 to 16. The 150 in mainstream school were compared with age- and gender-matched classroom control subjects ($n = 108$). Behavioral and emotional problems, delinquency, peer relations, self-esteem, and hobbies, were assessed by standardized, well-validated instruments, including the Strengths and Difficulties Questionnaire, administered by mail to parents, teenagers, and teachers. **RESULTS:** Parents were more likely to rate ELGA teenagers than control subjects as in the "abnormal" range for hyperactivity (8% vs 1%; difference: 7%; (95% confidence interval [CI]: 2-12), peer relationship problems (19% vs 5%; difference: 14%; 95% CI: 6-21), and emotional problems (18% vs 7%; difference: 11%; 95% CI: 3-19), but not conduct problems (10% vs 5%; difference: 5%; 95% CI: -1 to 12)). Teachers reported a similar pattern. In contrast, compared with control subjects, ELGA teenagers did not rate themselves as having more problems with peers, hyperactivity, conduct, depression, or low self-esteem. They reported more emotional problems but less delinquency, alcohol, cannabis, and other drug use. **CONCLUSIONS:** Compared with mainstream classmates, children who are born extremely early continue to have higher levels of parent- and teacher-reported emotional, attentional, and peer problems well into their teens. However, despite these problems, they do not show signs of more serious conduct disorders, delinquency, drug use, or depression.

POST-MISCARRIAGE PSYCHIATRIC MORBIDITY

SCREENING FOR POST-MISCARRIAGE PSYCHIATRIC MORBIDITY.

Authors : Lok IH, Lee DT, Yip SK, Shek D, Tam WH, Chung TK. - Department of Obstetrics and Gynecology, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong SAR, China. ingridlok@cuhk.edu.hk

Source : Am J Obstet Gynecol. 2004 Aug;191(2):546-50

Summary: The purpose of this study was to evaluate 12-item General Health Questionnaire (GHQ-12) in screening for psychiatric morbidity after miscarriage. **STUDY DESIGN:** A prospective cohort study was carried out involving 222 patients. Six weeks after miscarriage, the GHQ-12 was applied. Psychiatric "case" or "non-case" was diagnosed by the psychiatrist with use of the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders-III-R.

The patients were computer randomized into Groups A or B. A receiver operating characteristic (ROC) curve was constructed for Group A. The optimal cutoff value of GHQ-12 was determined, and this value was applied to Group B. The test characteristics were assessed. **RESULTS:** Twenty-seven patients were found to be psychiatric cases. An ROC with area under curve of 0.93 (95% CI 0.87-0.99, $P < .001$) was constructed. The best GHQ-12 cutoff score was $> \text{or} = 4$ in detecting psychiatric caseness. A sensitivity of 83%, specificity of 90%, positive predictive value of 50%, and negative predictive value of 98% were obtained. **CONCLUSION:** GHQ-12 is an effective screening tool in detecting psychiatric morbidity after miscarriage.

MATERNAL DEPRESSION & Offspring Dysfunction

▪ PSYCHIATRIC DISORDERS AMONG OFFSPRING OF DEPRESSED MOTHERS: ASSOCIATIONS WITH PATERNAL PSYCHOPATHOLOGY.

Authors: Marmorstein NR, Malone SM, Iacono WG. - Department of Psychology, Rutgers University, 311 N. 5th St., Camden, NJ 08102. marmorst@camden.rutgers.edu

Source: Am J Psychiatry. 2004 Sep;161(9):1588-94

Summary: **OBJECTIVE:** The association between maternal depression and offspring dysfunction is well documented; however, little attention has been paid to psychopathology in the partners of these depressed mothers or to how paternal psychopathology might influence the relationship between maternal depression and offspring dysfunction. The purpose of this study was to explore whether major depression and/or antisocial behavior tended to occur more frequently among partners of depressed mothers (compared to partners of nondepressed mothers) and to examine how these paternal disorders related to offspring psychopathology. **METHOD:** Participants were drawn from the Minnesota Twin Family Study, a community-based study of twins and their parents. Depressed and nondepressed mothers, their partners (the biological fathers of the twins), and their 17-year-old offspring were included. Structured interviews were used to assess participants for the presence of major depression, conduct disorder, and adult antisocial behavior. **RESULTS:** Depressed mothers tended to partner with antisocial fathers. Depression in mothers and antisocial behavior in fathers were both significantly and independently associated with offspring depression and conduct disorder. No interactions of the parental diagnoses with each other or with the gender of the offspring were found. **CONCLUSIONS:** Many offspring of depressed mothers experience the additional risk of having an antisocial father. The implications of these findings for risk among the offspring of depressed mothers are discussed.

Children & Adolescent Mental Health (CAMH)

Tricyclic Drugs, Depression, Children & Adolescents

▪ TRICYCLIC DRUGS FOR DEPRESSION IN CHILDREN AND ADOLESCENTS

Authors: Hazell P, O'Connell D, Heathcote D, Henry D

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 26 February 2002. Cochrane reviews are regularly checked and updated if necessary.

Background: There is a need to identify effective and safe treatments for depression in children and adolescents. While tricyclic drugs are effective in treating depression in adults, individual studies involving children and adolescents have been equivocal.

Objectives: To assess the effects of oral tricyclic antidepressants compared to placebo in the treatment of child and adolescent depression.

Search strategy: We searched MEDLINE (1966-1997), EMBASE, Excerpta Medica (June 1974-1997), the Cochrane Collaboration Depression, Anxiety and Neurosis Group trials register (most recent search 25/1/2000) and bibliographies of previously published reviews and papers describing original research were cross-checked. Current Contents was screened for recent publications. We contacted authors of relevant abstracts in conference proceedings of the American Academy of Child and Adolescent Psychiatry, and we hand searched the Journal of the American Academy of Child and Adolescent Psychiatry (1978-1999).

Selection criteria: Randomised controlled trials comparing the efficacy of orally administered tricyclic medication with placebo in depressed people aged 6-18 years.

Data collection and analysis: Most studies reported multiple outcome measures including depression scales and clinical global impression scales. For each study the best available depression measure was taken as the index measure of depression outcome. Predetermined criteria were established to assist in the ranking of measures. Where authors reported categorical outcomes we calculated individual and pooled odds ratios for the odds of improvement in treated compared with control subjects. For continuous outcomes pooled effect sizes were calculated as the number of standard deviations by which the change in depression scores for the treatment group exceeded those for the control groups.

Main results: Thirteen trials (involving 506 participants) were included. No overall improvement with treatment compared to placebo was seen for children or adolescents (odds ratio = 0.84, 95% confidence interval 0.56 to 1.25). A statistically significant but small benefit of treatment over placebo was seen in reducing symptoms (effect size (standardised mean difference) = -0.31, 95% confidence interval -0.62 to -0.01). Subgroup analyses suggest a larger benefit among adolescents (effect size = -0.47, 95% confidence interval -0.92 to -0.02), and no benefit among children (effect size = 0.15, 95% confidence interval -0.34 to 0.64). Treatment with a tricyclic antidepressant caused more vertigo (odds ratio = 4.38, 95% confidence interval 2.33 to 8.25), orthostatic hypotension (odds ratio = 6.78, 95% confidence interval 2.06 to 22.26), tremor (odds ratio 6.29, 95% confidence interval 1.78 to 22.17) and dry mouth (odds ratio = 5.17, 95% confidence interval 2.68 to 29.99) than did placebo, but no statistically significant difference was found for other possible adverse effects.

Reviewers' conclusions: Data suggest tricyclic antidepressants are not useful in treating depression in pre pubertal children. There is marginal evidence to support the use of tricyclic antidepressants in the treatment of depression in adolescents, although the magnitude of effect is likely to be moderate at best.

Children & Mental Health CARE

▪ TREATMENT RETENTION AMONG

CHILDREN ENTERING A NEW EPISODE OF MENTAL HEALTH CARE.

Authors : Harpaz-Rotem I, Leslie D, Rosenheck RA. - department of psychiatry of Yale University School of Medicine, 25 Park Street, Suite 617, New Haven, Connecticut 06519. ilan. harpaz-rottem@yale.edu

Source : Psychiatr Serv. 2004 Sep;55(9):1022-8

Summary: This study examined use of mental health services among children and adolescents with private insurance who were entering treatment. Variations in service use were examined by age, gender, diagnosis, recent psychiatric hospitalization, and type of insurance. Differences between children who received treatment from mental health professionals and those who were treated by primary care physicians were also examined. **METHODS:** Drawn from a large database, the sample comprised 11,659 new users of mental health services. Service use was defined as the total number of days children were retained in treatment and the total number of mental health contacts recorded. **RESULTS:** The overall mean number of visits within a six-month period was 3.9. The average duration of treatment was 75.36 days. Children who were treated by a mental health specialist were less likely to drop out of treatment and had a larger number of visits. Severity of illness, psychiatric hospitalization, and managed care insurance coverage were also associated with lower risk of dropout and greater intensity of care. **CONCLUSIONS:** Children's access to services does not guarantee sustained involvement in treatment. To more fully address the nature of service use among children, a closer look at specific barriers to continued involvement in services is needed.

BEHAVIORAL HEALTH DISORDERS

■ DIAGNOSIS AND TREATMENT OF BEHAVIORAL HEALTH DISORDERS IN PEDIATRIC PRACTICE.

Authors : Williams J, Klinepeter K, Palmes G, Pulley A, Foy JM. - Department of Pediatrics, Wake Forest University Health Sciences, Winston-Salem, North Carolina 27157-1060, USA. janewill@wfubmc.edu

Source : Pediatrics. 2004 Sep;114(3):601-6

Summary: There has been a strong push toward the recognition and treatment of children with behavioral health problems by primary care pediatricians. This study was designed to assess the extent to which a sample of primary care pediatricians diagnose and treat behavioral health problems and to identify factors that may contribute to their behavioral health practice. **METHODS:** A standard interview was conducted with 47 pediatricians who work in primary care settings in a predominantly urban setting in North Carolina. Pediatricians' responses to questions about the estimated percentage of children in their practice with a behavioral health disorder, tools used to make diagnoses, frequent and infrequent diagnoses made, comfort level with making a diagnosis, reasons for not making a diagnosis, use of psychotropic medications, types of nonmedication interventions provided, educational background, and needs involving behavioral health issues were evaluated. **RESULTS:** Pediatricians estimated that the average percentage of children in their practices with a behavioral health disorder was 15%. The study did not find significant differences in perceptions related to time in practice or gender of the pediatric provider. The most frequent behavioral health

diagnosis was attention-deficit/hyperactivity disorder (ADHD), and the majority incorporated behavioral questionnaires, expressed a high level of comfort with the diagnosis, and frequently or occasionally prescribed stimulants. Variability was noted in both practice and comfort for other behavioral health disorders. Slightly fewer than half of the pediatricians frequently diagnosed anxiety and depression. Those who make these diagnoses commonly incorporated questionnaires and reported frequent or occasional use of selective serotonin reuptake inhibitors. Comfort in making the diagnosis of anxiety was highly associated with use of selective serotonin reuptake inhibitors. The vast majority (96%) of pediatricians provided nonmedication interventions, including supportive counseling, education for coping with ADHD, behavior modification, and/or stress management. Diagnosis and treatment of severe behavioral health disorders were infrequent throughout the pediatric practices. Areas of greatest educational interest included psychopharmacology, diagnosis and treatment of depression and anxiety, and updates on ADHD. The majority of pediatric providers did not identify a need for education about several high-prevalence disorders that they do not frequently diagnose or treat, including conduct disorder and substance abuse. **CONCLUSIONS:** Pediatricians in this sample frequently diagnosed and treated ADHD. For all other behavioral health disorders, pediatricians reported variability in both comfort and practice. They frequently provided both pharmacologic and nonpharmacologic treatments for children and adolescents with mild to moderate behavioral health disorders but not for severe disorders. Although they identified needs for additional education for anxiety and depression, the majority did not identify educational needs for several high-prevalence behavioral health disorders, including conduct disorder and substance abuse.

Methylin & ADHD

■ METHYLIN CHEWABLE TABLETS AND METHYLIN ORAL SOLUTION FOR TREATMENT OF ATTENTION DEFICIT HYPERACTIVITY DISORDER LAUNCHED IN US ATLANTA, GA, --SEPTEMBER 7, 2004

Authors : Alliant Pharmaceuticals, Inc., a leading pediatric specialty pharmaceutical company

Source : InView Communications

Summary: Alliant Pharmaceuticals, Inc., a leading pediatric specialty pharmaceutical company, today announced FDA approval of Methylin Chewable Tablets and Methylin Oral Solution for the treatment of ADHD. The two products are the first, and only, chewable tablet and oral solution for ADHD to gain FDA acceptance.

Compliance with any medical therapy is important. With children, the issue is pronounced, as swallowing pills can be a significant problem. Says Dr. Lyndon Waugh, M.D., Clinical Assistant Professor of Child and Adolescent Psychiatry at Emory University, "The release of this effective, well established medication for ADHD in liquid and chewable tablet form will allow physicians another alternative to carefully fine-tune a dosing strategy for each individual patient. And, it will help diminish family conflict and the child's anxiety when they have difficulty swallowing capsules or tablets."

Currently 26% of the total population has difficulty in swallowing tablets and capsules. This percentage is considered to be higher in the pediatric patient population.

ADHD is a disorder of the brain in which the individual repeatedly exhibits inappropriate impulsivity and/or inattention. According to the Attention Deficit Disorder Association, an estimated 3 to 7 percent of school-age children, and an estimated 4 percent of adults suffer from ADHD. The Attention Deficit Disorder Association was instrumental in convincing the U.S. Senate to sign a resolution designating September 7, 2004, "National Attention Deficit Disorder Awareness Day" in hopes of better educating the public about ADHD.

"As with any medication, compliance is critical," adds Mark Pugh, President and CEO of Alliant Pharmaceuticals. "Methylin Chewable Tablet and Methylin Oral Solution were designed specifically to help improve efficacy in treatment." Mr. Pugh adds that Alliant plans to release additional information on these products at the upcoming American Academy of Adolescent and Child Psychiatry meeting October 20 - 23rd in Washington D.C.

Methylin Chewable Tablets are available in a 2.5mg, 5mg and 10mg dose. The Methylin Oral Solution is available in a 5mg / 5mL and 10mg / 5mL dose.

Children & Behavioral Health Disorders

▪ PEDIATRICIANS TREATING MORE CHILDREN WITH BEHAVIORAL HEALTH DISORDERS

Authors : WINSTON-SALEM, NC -- September 7, 2004

Source : Wake Forest University Baptist Medical Center

Summary: Pediatricians are diagnosing and treating a growing number of children with behavioral health problems. However, they do not always feel comfortable or sufficiently trained to fill this new role, according to a study from Wake Forest University Baptist Medical Center. The study involved interviews with community pediatricians who estimated that an average of about 15 percent of the children they see have behavioral health problems, said Jane Williams, Ph.D., lead author of the study. The report was published in the September issue of Pediatrics.

Attention Deficit Hyperactivity Disorder (ADHD) is the most common behavioral health disorder seen by pediatricians, she said. The pediatricians "expressed a high level of comfort with the diagnosis and frequently or occasionally prescribed stimulants" to treat it. But when a child is suffering from anxiety or depression, the pediatricians felt they were on shakier grounds. Fewer than half the pediatricians said they diagnosed anxiety and depression frequently. Those that did typically used questionnaires in making the diagnosis and prescribed drugs from a class that includes Prozac, Zoloft and Paxil. The study found a "strong interest in diagnosing and treating behavioral health disorders within their perceived limits and level of comfort," Williams and her Wake Forest Baptist colleagues reported. "They were very concerned about the correctness of these diagnoses and consider the impact on both the child and family." The researchers said pediatricians are treating more children with psychiatric problems in part because of chronic underfunding of the public mental health system. Only about 2 percent of the children who need treatment are seen by mental health specialists. In fact, the diagnosis and treatment of ADHD has shifted primarily to pediatricians, the results showed. "The diagnosis of anxiety and depression appeared to be shifting more gradually to pediatric providers," Williams said. But many pediatricians felt they were not prepared in medical school and their residency training programs to treat these children, leading to a scramble to find continuing medical education

courses to fill that gap. They often felt unprepared to treat depression and anxiety and to choose appropriate drugs for these diagnoses.

"Perhaps the most important and most generalizable findings of this study involve the need for increased training and for continuing medical education in behavioral health," Williams said.

That's of special concern because depression often leads to both suicide and substance abuse. "As primary care settings may be the only environment in which adolescents are seen, their high mortality rate from accidents, homicide and suicide would suggest the critical need for pediatricians to recognize and inquire about these symptoms," she said.

Along with Williams, other researchers included Jane M. Foy, M.D. and Kurt Klinepeter, M.D., both of Brenner Children's Hospital, Guy Palmes, M.D., of the Department of Psychiatry at Wake Forest Baptist and Anita Pulley of Northwest Area Health Education Center. The study was sponsored by a grant from the Duke Endowment as part of its Primary Care-Children's Mental Health Initiative.

Adolescents & ADHD

▪ RESPONSE INHIBITION IN ADOLESCENTS DIAGNOSED WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER DURING CHILDHOOD: AN EVENT-RELATED FMRI STUDY.

Authors : Schulz KP, Fan J, Tang CY, Newcorn JH, Buchsbaum MS, Cheung AM, Halperin JM. - Department of Psychiatry, Box 1230, Mount Sinai School of Medicine, One Gustave Levy Place, New York, NY 10029. kurt.schulz@mssm.edu

Source : Am J Psychiatry. 2004 Sep;161(9):1650-7

Summary: OBJECTIVE: Frontostriatal neural abnormalities have been implicated in the response inhibition impairments that are characteristic of attention deficit hyperactivity disorder (ADHD). However, reports of such abnormalities in adolescents are inconsistent. The present study used behavioral and functional neuroimaging techniques to examine inhibitory control processes in adolescents who had been diagnosed with ADHD during childhood.

METHOD: The authors used functional magnetic resonance imaging (fMRI) during performance of a Go/No-Go task to scan 10 male adolescents who were diagnosed with DSM-III-R ADHD when they were 7 to 11 years old and nine age-, sex-, and IQ-matched comparison subjects with no history of ADHD. Response inhibition was tested by contrasting neural activation during No-Go trials with that during Go trials.

RESULTS: The inhibition of a prepotent tendency to respond produced markedly greater activation of the left anterior cingulate gyrus, bilateral frontopolar regions, bilateral ventrolateral prefrontal cortex, and left medial frontal gyrus in the adolescents with childhood ADHD than in the adolescents with no history of ADHD. Activity in the first two regions was inversely related to task performance across the study group.

CONCLUSIONS: Compared with adolescents who had no history of ADHD, adolescents who were diagnosed with ADHD during childhood exhibited enhanced responses during inhibition in ventrolateral prefrontal cortical areas that subserve response inhibition, as well as in anterior cingulate and frontopolar regions implicated in other executive functions.

BDNF & Early Adolescent Bipolar Disorder

▪ **LINKAGE DISEQUILIBRIUM OF THE
BRAIN-DERIVED NEUROTROPHIC FACTOR
VAL66MET POLYMORPHISM IN CHILDREN
WITH A PREPUBERTAL AND EARLY
ADOLESCENT BIPOLAR DISORDER
PHENOTYPE.**

Authors : Geller B, Badner JA, Tillman R, Christian SL, Bolhofner K, Cook EH Jr. - Department of Psychiatry, Washington University School of Medicine, 660 S. Euclid Ave., St. Louis, MO 63110. gellerb@medicine.wustl.edu

Source : Am J Psychiatry. 2004 Sep;161(9):1698-700

Summary: OBJECTIVE: Transmission of the brain-derived neurotrophic factor (BDNF) Val66 allele in children with a prepubertal and early adolescent bipolar disorder phenotype was examined. METHOD: The prepubertal and early adolescent bipolar disorder phenotype was defined as current DSM-IV bipolar I disorder (manic or mixed phase) with at least one cardinal mania criterion (i.e., euphoria and/or grandiosity) to ensure differentiation from attention deficit hyperactivity disorder. Probands (mean age=10.7 years, SD=2.7) were obtained by consecutive new case ascertainment from designated pediatric and psychiatric venues. Parents and probands were interviewed separately by research nurses who were blind to the probands' diagnoses. Genotyping was done with TaqMan Assay-on-Demand. Analysis was done with the Family Based Association Test program. RESULTS: There were 53 complete, independent trios. The BDNF Val66 allele was preferentially transmitted (Family Based Association Test: $\chi^2(2)=6.0$, $df=1$, $p=0.014$). CONCLUSIONS: This finding in child bipolar disorder is consistent with data for adults with bipolar disorder that show preferential transmission of the Val66 allele.

Childhood Stroke & Neuropsychological Lesion

▪ **EFFECT OF SIDE OF LESION ON
NEUROPSYCHOLOGICAL PERFORMANCE IN
CHILDHOOD STROKE**

Authors : Max JE. - University of California and Children's Hospital and Health Center, San Diego, California, USA. jmax@ucsd.edu

Source : J Int Neuropsychol Soc. 2004 Sep;10(5):698-708

Summary: The purpose of the current study was to examine the effect of side of lesion on neuropsychological performance in childhood stroke. While laterality effects have been shown fairly consistently in adults who have experienced stroke, results from studies on children who have experienced childhood stroke are not as clear. Numerous methodological differences between previous studies on laterality effects in childhood stroke make it difficult to draw overall conclusions regarding laterality findings. The current study aimed to study a single group of children who experienced stroke in childhood across a number of cognitive domains. The participants were 13 children/adolescents with left hemisphere lesions and 16 children/adolescents with right hemisphere lesions, with a range of onset from prenatal to 13 years. All participants were administered a broad battery of neuropsychological tests including tests of intelligence, achievement, language skills, visuospatial skills, memory, and executive functioning. No significant differences were found between the groups on any of the measures and the calculated effect sizes were small for all but one of the measures examined. These results have

implications for a greater understanding of the ability of the young brain to reorganize after childhood stroke.

Bimanual Coordination & Alcohol-Exposed Children

▪ **BIMANUAL COORDINATION IN ALCOHOL-
EXPOSED CHILDREN: ROLE OF THE CORPUS
CALLOSUM.**

Authors : Roebuck-Spencer TM, Mattson SN, Marion SD, Brown WS, Riley EP. - National Rehabilitation Hospital, Washington, DC 20010, USA

Source : J Int Neuropsychol Soc. 2004 Jul;10(4):536-48

Summary: The corpus callosum (CC) is one of several brain structures affected in children prenatally exposed to alcohol. This structure plays a major role in coordinating motor activity from opposite sides of the body, and deficits in bimanual coordination have been documented in individuals with agenesis of or damage to the CC, particularly when the task is performed without visual feedback. The Bimanual Coordination Test was used to assess speed and accuracy on a task where both hands must coordinate to guide a cursor through angled pathways providing measures of interhemispheric interaction or the ability of the two hemispheres to coordinate activity via the corpus callosum. Twenty-one children with fetal alcohol spectrum disorders (FASD) and 17 non-exposed control children (CON), matched closely in age, sex, and ethnicity were tested. For trials with visual feedback (WV), children with FASD were slower than CON children but were equally accurate. Although statistically significant group differences were not observed on most trials completed without visual feedback (WOV), accuracy of the FASD group on WOV trials was highly variable. Group differences in accuracy on WOV angles approached significance after accounting for performance on the WV angles, and children with FASD were significantly less accurate on an individual angle believed to be particularly sensitive to interhemispheric interaction. These results indicate that children with FASD are slower than CON children but equally accurate on basic visuomotor tasks. However, as task complexity and reliance on interhemispheric interaction increases, children with FASD demonstrate variable and inaccurate performance.

Psychotic Disorder (PD)**PD & Hallucinatory Experiences**

▪ **HALLUCINATORY EXPERIENCES AND
ONSET OF PSYCHOTIC DISORDER:
EVIDENCE THAT THE RISK IS MEDIATED BY
DELUSION FORMATION.**

Authors : Krabbendam L, Myin-Germeys I, Hanssen M, Bijl RV, De Graaf R, Vollebergh W, Bak M, Van Os J. Department of Psychiatry and Neuropsychology, South Limburg Mental Health Research and Teaching Network, EURON, Maastricht University, Maastricht, The Netherlands.

Source : Acta Psychiatr Scand. 2004 Oct;110(4):264-72.

Summary : Objective: To examine the hypothesis that the risk for onset of psychotic disorder in individuals with self-reported hallucinatory experiences (HE) would be higher in those who developed delusional ideation (DE) than in those who did not. Method: A population sample of 4673 individuals were interviewed with the Composite International Diagnostic

Interview at baseline and 1 and 3 years later. At year 3, clinical re-interview took place to identify onset of psychotic disorder. Results: Given the presence of HEs at baseline, the increase in risk of having the psychosis outcome at year 3 was much higher in those with DE at year 1 than in those without DE (risk difference between individuals with and without DE: 18.72%, 95% CI: 2.22-35.23, $\chi^2(2) = 4.94$, $df = 1$, $P = 0.026$). Conclusion: The results are in line with current psychological theories stating that clinical outcome of psychosis-like experiences is related to the development of secondary beliefs and appraisals.

PD & PLEASURABLE AUDITORY HALLUCINATIONS

PLEASURABLE AUDITORY HALLUCINATIONS

Authors : Sanjuan J, Gonzalez JC, Aguilar EJ, Leal C, Os J. Department of Medicine, Psychiatric Unit, Valencia University, Valencia, Spain

Source : Acta Psychiatr Scand. 2004 Oct;110(4):273-8.

Summary : Sanjuan J, Gonzalez JC, Aguilar EJ, Leal C, van Os J. Pleasurable auditory hallucinations. Acta Psychiatr Scand 2004; 110: 273-278. Copyright Blackwell Munksgaard 2004. Objective: The focus in auditory hallucination (AH) research is usually on the negative impact of the experience itself. There are practically no studies on whether voices can be perceived as pleasurable. The aim of the present study was to assess the frequency of voices as a pleasurable experience in a psychotic patient population. Method: A total of 160 patients with AHs (89 schizophrenia and 17 other psychoses) were assessed with the psychotic symptom rating scale (PSYRATS) for AHs, including an added item on whether the experience was pleasurable. Results: Twenty-eight patients (26%) reported the voices as a pleasurable experience and 10 of them did so frequently. Pleasurable hallucinations showed negative associations with amount and intensity of distress, degree of negative content and loudness. Positive associations were apparent with chronicity and perceived control over the voices. Conclusion: Pleasurable hallucinations can be detected in a substantial proportion of patients, and cross validated with existing instruments.

PD & SERUM LIPIDS IN SCHIZOPHRENIA

SERUM LIPIDS IN SCHIZOPHRENIA AND OTHER FUNCTIONAL PSYCHOSES : A GENERAL POPULATION NORTHERN FINLAND 1966 BIRTH COHORT SURVEY

Authors : Saari K, Jokelainen J, Veijola J, Koponen H, Jones PB, Savolainen M, Jarvelin MR, Lauren L, Isohanni M, Lindeman S. Department of Psychiatry, University of Oulu, Finland

Source : Acta Psychiatr Scand. 2004 Oct;110(4):279-85.

Summary : Objective: To compare fasting serum lipid concentrations of subjects with schizophrenia with a comparison group. Method: The study sample consists of 5654 members of the northern Finland 1966 birth cohort who participated in the field study with blood samples after overnight fasting and clinical examination in 1997-98. Total cholesterol (TC), high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides (TG) and glucose were analyzed. Analysis of variance were used for comparing differences in lipids means between diagnostic categories. Results: Mean fasting TC in subjects with schizophrenia was 20 mg/dl higher than in the comparison group. TC and TG

levels in the group of other psychoses resembled the schizophrenia group. Conclusion: Blood lipid levels in subjects with schizophrenia and other functional psychoses were high. As these persons are at special risk for hyperlipidemia their lipid levels should be regularly monitored, and cholesterol lowering diet, as well as medication, should be considered.

PD & NEUROPSYCHOLOGICAL FUNCTIONING

NEUROPSYCHOLOGICAL FUNCTIONING IN FIRST-BREAK, NEVER-MEDICATED ADOLESCENTS WITH PSYCHOSIS.

Authors : Brickman AM, Buchsbaum MS, Bloom R, Bokhoven P, Paul-Oudouard R, Haznedar MM, Dahlman KL, Hazlett EA, Aronowitz J, Heath D, Shihabuddin L. *Department of Psychiatry, Mount Sinai School of Medicine, New York, NY; daggerTaub Institute for Research on Alzheimer Disease and the Aging Brain, Columbia University, College of Physicians and Surgeons, New York, NY; double daggerDepartment of Psychiatry and Behavioral Medicine, Brown Medical School, Providence, RI; and section signDepartment of Veterans Affairs, Bronx VA Medical Center, Bronx, NY.

Source : J Nerv Ment Dis. 2004 Sep;192(9):615-622.

Summary : The purpose of the current study was to examine neuropsychological functioning in a group of never-medicated first-break adolescents with psychosis. It is the first report of cognition in a sample of adolescents with psychosis in which all patients were drug-naïve. Twenty-nine adolescent patients (mean age = 16.07; SD = 2.00; 15 male and 14 female patients) experiencing their first psychotic episode and 17 age-matched and sex-matched normal volunteers (mean age = 16.88; SD = 2.39; 9 male and 8 female subjects) were recruited and assessed with a neuropsychological battery. Measures of attention, memory, language, executive functioning, perceptual motor processing, and motor speed were obtained. Psychiatric symptomatology, estimated verbal IQ, and parental socioeconomic status were also determined. Patients with psychosis were significantly more impaired than normal volunteers; effect sizes were greatest in the areas of executive functioning, attention, and memory, and significantly smaller in areas of language, perceptual motor processing, and motor speed. The pattern was not altered when differences in verbal IQ and parental socioeconomic status were controlled. Sex and age interactions indicated that younger male patients were particularly impaired. The findings demonstrate neuropsychological deficits in adolescents with psychosis and suggest that cognitive deficits are core symptoms in psychotic disorders.

NEW TREATMENTS & AGITATION

NEW TREATMENTS FOR AGITATION.

Authors : Citrome L. - S. Kline Institute for Psychiatric Research, Orangeburg, NY 10962, USA. citrome@nki.rfmh.org

Source : Psychiatr Q. 2004 Fall;75(3):197-213

Summary : Acute agitation is a frequent reason for emergency psychiatric intervention. It is important to intervene early to avoid escalation of agitation to aggression. Reducing risk by using effective treatments will result in fewer instances of seclusion and restraint, and fewer injuries to staff and patients. This paper will first review the epidemiology of aggressive behavior and mental disorders, followed by a discussion of assessment and diagnostic considerations. The pathophysiology of safety risk is discussed within the context

of the model of the "triune brain." Pharmacological treatment strategies for acute episodes of agitated behavior will be discussed in detail. This includes newer formulations of novel antipsychotics such as liquids and rapidly disintegrating tablets, as well as intramuscular preparations.

ENTORHINAL CORTEX & First-Episode Psychotic

■ THE ENTORHINAL CORTEX IN FIRST-EPI- SODE PSYCHOTIC DISORDERS: A STRUCTURAL MAGNETIC RESONANCE IMAGING STUDY.

Authors : Prasad KM, Patel AR, Muddasani S, Sweeney J, Keshavan MS. - University of Pittsburgh Medical Center, Western Psychiatric Institute and Clinic, 3811 O'Hara St., Pittsburgh, PA 15213. keshavanms@upmc.edu

Source : Am J Psychiatry. 2004 Sep;161(9):1612-9

Summary: OBJECTIVE: Neuropathological findings regarding the entorhinal cortex in schizophrenia are conflicting. The authors used structural magnetic resonance imaging to examine the entorhinal cortex volumes of healthy subjects and medication-naïve patients experiencing their first episode of psychotic illness. METHOD: The study included 33 patients with schizophrenia and related disorders, 11 patients with nonschizophrenic disorders, and 43 matched healthy subjects. All subjects were rated on the Scale for the Assessment of Positive Symptoms and the Scale for the Assessment of Negative Symptoms, and volumetric measurements of the entorhinal cortex were obtained for all subjects. The authors examined differences across the groups as well as clinical correlations of entorhinal cortex volumes adjusted for intracranial volume. RESULTS: A significant diagnosis effect was seen in the left entorhinal cortex: patients with schizophrenia and related disorders and patients with nonschizophrenic psychotic disorders had smaller left entorhinal cortex volumes than healthy subjects. The mean entorhinal cortex volume of patients with schizophrenic disorders did not differ from that of patients with nonschizophrenic psychotic disorders. In patients with schizophrenic disorders, the entorhinal cortex volume positively correlated with severity of delusions. The mean entorhinal cortex volume of patients with nondelusional psychotic disorders was significantly smaller than that of patients with delusional psychotic disorders and healthy subjects. CONCLUSIONS: Smaller entorhinal cortex volume in first-episode, neuroleptic-naïve psychotic disorders may not be a confound of the effects of illness chronicity or antipsychotic treatment. Entorhinal cortex pathology appears to have a significant association with positive symptoms, specifically delusions. The impairment of functions in which the entorhinal cortex participates-such as novelty detection, associative learning, and processing episodic, recognition, and autobiographical memory-could be responsible for its association with psychotic disorders and delusions.

Psychotic disorders & Early victimisation experiences

■ PSYCHOSIS, VICTIMISATION AND CHILDHOOD DISADVANTAGE: EVIDENCE FROM THE SECOND BRITISH NATIONAL SURVEY OF PSYCHIATRIC MORBIDITY.

Authors : Bebbington PE, Bhugra D, Brugha T, Singleton N, Farrell M, Jenkins R, Lewis G, Meltzer H. - Department of Mental Health Sciences, 48 Riding House Street, London W1N 8EY, UK. p.bebbington@ucl.ac.uk

Source : Br J Psychiatry. 2004 Sep;185:220-6

Summary: BACKGROUND: Adverse early circumstances may be more common in people who later develop psychotic disorders. AIMS: To use data from the second British National Survey of Psychiatric Morbidity to examine associations between psychotic disorders and a number of early victimisation experiences. METHOD: Psychiatric disorders were identified through structured assessment of adults resident in private households in Britain (n=8580). Respondents were asked whether they had experienced selected events displayed on cards. RESULTS: Compared with respondents with other psychiatric disorders or with none, the prevalence of every experience bar one was significantly elevated in those with definite or probable psychosis. The largest odds ratio was for sexual abuse. Controlling for depressed mood somewhat reduced the odds ratios for the individual experiences. CONCLUSIONS: In people with psychosis, there is a marked excess of victimising experiences, many of which will have occurred during childhood. This is suggestive of a social contribution to aetiology.

Schizophrenia

Schizophrenia & Aripiprazole

■ ARIPIPAZOLE: A NEW ATYPICAL ANTIPSYCHOTIC DRUG

Authors : Fischer B, Davids E, Gastpar M. Rheinische Kliniken Essen, Klinik für Psychiatrie und Psychotherapie der Universität Duisburg-Essen (Direktor: Prof. Dr. M. Gastpar).

Summary : Aripiprazole is an atypical antipsychotic agent with an intrinsic dopamine agonist activity of 30 %. Aripiprazole exerts additional partial agonist action on 5-HT (1A) receptors and has antagonist properties at 5-HT (2A) receptors. Controlled studies demonstrated an effectiveness in acute relapse of schizophrenic psychosis, chronic schizophrenic and schizoaffective disorders. Aripiprazole was effective in the treatment of productive psychotic and negative symptoms. Compared to other antipsychotics aripiprazole demonstrated a favourable profile of side effects: only slight changes of body weight, mild extrapyramidal symptoms, no prolactin elevation and no significant changes in QTc interval. The efficacy in the long term treatment of schizophrenia seems to be similar to other antipsychotics (e. g. olanzapine). The first evaluations of studies with patients with bipolar disorders showed a significant efficacy in the treatment of mania.

Schizophrenia, clozapine & olanzapine

■ THE EFFECTS OF CLOZAPINE AND HIGH- DOSE OLANZAPINE ON BRAIN FUNCTION IN TREATMENT-RESISTANT SCHIZOPHRENIA

Authors : Robert R. Conley, Deanna L. Kelly, Lori L. Beason-Held, Henry H. Holcomb and Charles M. Richardson, Maryland Psychiatric Research Center, University of Maryland, Baltimore, MD, USA; Maryland Psychiatric Research Center, University of Maryland, Baltimore, MD, USA; Maryland Psychiatric Research Center, University of Maryland, Baltimore, MD, USA; Maryland Psychiatric Research Center, University of Maryland, Baltimore, MD, USA; Maryland Psychiatric Research Center, University of Maryland, Baltimore, MD, USA.

Source : Volume 18 Issue 03 - Publication Date: 09/2004

Summary : Although demonstrating superior efficacy in people with treatment-resistant schizophrenia, clozapine may cause serious side effects, requires blood monitoring and is costly to administer. Olanzapine is similar to clozapine in molecular structure and pharmacologic action but has not demonstrated as robust results as clozapine at routine doses (10-25 mg). Here we present a case study measuring blood flow by positron emission tomography (PET) imaging for a patient treated sequentially with a high dose of olanzapine (50 mg/day) followed by clozapine each for 8 weeks in a double-blind design. During a task, clozapine produced more brain activation patterns than during treatment with olanzapine or during the drug free condition (2 week washout). Clozapine resulted in recruitment of frontal, parietal and cingulate regions that did not appear to be active during olanzapine in this 44 year old right handed male. Additionally, a more robust decrease in symptoms was noted on the Brief Psychiatric Rating Scale (BPRS) score than with olanzapine treatment. These findings suggest that high doses of olanzapine do not produce similar brain activation patterns as clozapine in people with treatment-resistant schizophrenia.

SCHIZOPHRENIA, AMISULPRIDE & CLOZAPINE

AMISULPRIDE AUGMENTATION OF CLOZAPINE: AN OPEN NON-RANDOMIZED STUDY IN PATIENTS WITH SCHIZOPHRENIA PARTIALLY RESPONSIVE TO CLOZAPINE.

Authors : Munro J, Matthiasson P, Osborne S, Travis M, Purcell S, Cobb AM, Launer M, Beer MD, Kerwin R. Section of Clinical Neuropharmacology, Division of Psychological Medicine, Institute of Psychiatry, London, UK.

Source : Acta Psychiatr Scand. 2004 Oct;110(4):292-8.

Summary : Objective: Treatment options are very limited for individuals with schizophrenia resistant to clozapine. We tested the hypothesis that amisulpride augmentation would lead to an improvement in these patients. Method: This was an open non-randomized study. Thirty-three patients with sub-optimal response to clozapine were commenced on amisulpride in addition to clozapine. Clinical status was evaluated at baseline, 3 and 6 months using the Positive And Negative Syndrome Scale (PANSS), Scale for the Assessment of Negative Symptoms (SANS), Global Assessment Scale (GAS), Calgary Depression Scale, Calgary Anxiety Scale and various side effect rating scales. Results: Twenty-eight subjects completed 6 months treatment on clozapine and amisulpride. There was a statistically significant improvement in the mean scores for PANSS, SANS and GAS at follow-up and no significant changes in side effect ratings. Conclusion: Co-administration of amisulpride, in a group of patients partially or non-responsive to clozapine, may lead to a substantial improvement in positive and negative symptoms, without worsening the side effect burden.

SCHIZOPHRENIA & CYTOKINE

CYTOKINE NETWORK IN PATIENTS WITH SCHIZOPHRENIA AND ITS SIGNIFICANCE FOR THE PATHOPHYSIOLOGY OF THE ILLNESS

Authors : Schuld A, Hinze-Selch D, Pollmacher T. Max-Planck-Institut für Psychiatrie München, Germany.

Source : Nervenarzt. 2004 Mar;75(3):215-26.

Summary : Experimental findings from psychoimmunologic

research in humans and epidemiological data suggest that alterations in cytokine networks may induce acute psychopathologic symptoms and may be involved in the pathogenesis and pathophysiology of schizophrenia by influencing brain development. However, there is insufficient evidence from genetic, post-mortem, and cerebrospinal fluid studies to demonstrate this in the CNS of schizophrenic patients. In contrast, there are quite robust findings from peripheral blood that interleukin (IL)-2, IL-6, tumor necrosis factor-alpha, and interferon cytokine systems in patients are regulated differently than in controls. However, these findings are not specific to schizophrenia, they are confounded by numerous intervening variables such as stress, smoking, and medication, and their pathophysiologic relevance for processes in the CNS is undetermined. Therefore, future research on the involvement of cytokines in the pathogenetics, pathophysiology, and treatment of schizophrenia is needed.

SCHIZOPHRENIA & HEART DISEASE

HEART DISEASE IN SCHIZOPHRENIA

Authors : Wobrock T, Sittlinger H, Kindermann M, Behrendt B. Universitätsnervenklinik-Psychiatrie und Psychotherapie, Universitätskliniken des Saarlandes.

thomas.wobrock@uniklinik-saarland.de

Source : Nervenarzt. 2004 Mar;75(3):267-72.

Summary : A 63-year-old male patient diagnosed with chronic schizophrenia and characterized by formal disorders of thought with neologism and paranoid ideation, especially grandiose delusions and feelings of being influenced by radiation, is presented. At admission to psychiatric hospital, the patient reported attacks of weakness and dizziness, which he attributed to his feelings of alien influence. The diagnosis of cardiac disease with severe bradycardia could already be established by basic physical examination. Further diagnostic procedures (e.g., ECG) revealed symptomatic atrioventricular conduction defects (atrioventricular block III). After implantation of a cardiac pacemaker, the somatic symptoms vanished and the patient recovered completely in terms of physical condition.

SCHIZOPHRENIA & HYPOFRONTALITY

HYPOFRONTALITY IN SCHIZOPHRENIA: A META-ANALYSIS OF FUNCTIONAL IMAGING STUDIES.

Authors : Hill K, Mann L, Laws KR, Stephenson CM, Nimmo-Smith I, McKenna PJ. Fulbourn Hospital, Cambridge, UK.

Source : Acta Psychiatr Scand. 2004 Oct;110(4):243-56.

Summary : Objective: Hypofrontality is not a well-replicated finding in schizophrenia either at rest or under conditions of task activation. Method: Studies comparing whole brain and frontal blood flow/metabolism in schizophrenic patients and normal controls were pooled. Voxel-based studies were also combined to examine the pattern of prefrontal activation in schizophrenia. Results: Whole brain flow/metabolism was reduced in schizophrenia to only a small extent. Resting and activation frontal flow/metabolism were both reduced with a medium effect size. Duration of illness significantly moderated resting hypofrontality, but the moderating effects of neuroleptic treatment were consistent with an influence on global flow/metabolism only. Pooling of voxel-based studies did not suggest an abnormal pattern of activation in schizophrenia. Conclusion: Meta-analysis supports resting hypofrontality in schizophrenia. Task-activated hypofrontality is also supported,

but there is little from voxel-based studies to suggest that this is associated with an altered pattern of regional functional architecture.

Schizophrenia & Maternal Care

POOR MATERNAL CARE AND HIGH MATERNAL BODY MASS INDEX IN PREGNANCY AS A RISK FACTOR FOR SCHIZOPHRENIA IN OFFSPRING.

Authors : Kawai M, Minabe Y, Takagai S, Ogai M, Matsumoto H, Mori N, Takei N. Department of Psychiatry and Neurology, Hamamatsu University School of Medicine, Hamamatsu, Japan.

Source : Acta Psychiatr Scand. 2004 Oct;110(4):257-63.

Summary : Objective: We investigated whether antenatal factors in mothers would increase the risk of schizophrenia in the offspring, and also examined any relationship between these factors and histories of obstetric complications (OCs). Method: Using the Mother and Child Health Handbooks of 52 patients with schizophrenia and 284 healthy subjects, we evaluated the risk-increasing effects of the frequency of antenatal care visits and mothers' body mass index (BMI) at both early and late pregnancy. Results: In logistic regression analysis, there was a significant association between the number of antenatal care visits and the risk of the disorder; an increase in a unit of visits corresponds to a reduction of the risk by 12%. We also found a 24% increase in the risk with a one-unit increase of BMI at the early pregnancy, and a 19% increase at the late pregnancy. These antenatal factors were found to contribute, in part, to an excess of OCs in individuals with schizophrenia. Conclusion: Poor maternal care during pregnancy and comparatively high maternal BMI especially at early pregnancy may cause a predisposition to schizophrenia in the offspring.

Schizophrenia & Cognitive Enhancement

COGNITIVE ENHANCEMENT THERAPY FOR SCHIZOPHRENIA: EFFECTS OF A 2-YEAR RANDOMIZED TRIAL ON COGNITION AND BEHAVIOR.

Authors : Hogarty GE, Flesher S, Ulrich R, Carter M, Greenwald D, Pogue-Geile M, Kechavan M, Cooley S, DiBarry AL, Garrett A, Parepally H, Zoretich R. University of Pittsburgh Medical Center, Western Psychiatric Institute and Clinic, Pittsburgh, PA 15213, USA. hogartyje@upmc.edu

Source : Arch Gen Psychiatry. 2004 Sep;61(9):866-76.

Summary : BACKGROUND: Deficits in social cognition and neurocognition are believed to underlie schizophrenia disability. Attempts at rehabilitation have had circumscribed effects on cognition, without concurrent improvement in broad aspects of behavior and adjustment. OBJECTIVE: To determine the differential effects of cognitive enhancement therapy (a recovery-phase intervention) on cognition and behavior compared with state-of-the-art enriched supportive therapy. DESIGN: A 2-year, randomized controlled trial with neuropsychological and behavioral assessments completed at baseline and at 12 and 24 months. SETTING: An outpatient research clinic housed in a medical center's comprehensive care service for patients with severe mental illness. PATIENTS: A total of 121 symptomatically stable, non-substance-abusing but cognitively disabled and chronically ill patients with schizophrenia or schizoaffective disorder. INTERVENTIONS: Cognitive enhancement therapy is a

multidimensional, developmental approach that integrates computer-assisted training in neurocognition with social cognitive group exercises. Enriched supportive therapy fosters illness management through applied coping strategies and education. MAIN OUTCOME MEASURES: Six highly reliable summary measures--Processing Speed, Neurocognition, Cognitive Style, Social Cognition, Social Adjustment and Symptoms--were tested using analysis of covariance and linear trend analysis. RESULTS: At 12 months, robust cognitive enhancement therapy effects were observed on the Neurocognition and Processing Speed composites ($P < .003$), with marginal effects observed on the behavioral composites. By 24 months, differential cognitive enhancement therapy effects were again observed for the 2 neuropsychological composites and for Cognitive Style ($P = .001$), Social Cognition ($P = .001$), and Social Adjustment ($P = .01$). As expected, no differences were observed on the residual Symptoms composite. Effects were unrelated to the type of antipsychotic medication received. Enriched supportive therapy also demonstrated statistically significant within-group effect sizes, suggesting that supportive psychotherapy can also have positive, although more modest, effects on cognitive deficits. CONCLUSION: Many cognitive deficits and related behaviors of patients with stable schizophrenia are improved when sufficient exposure to relevant rehabilitation is provided.

Schizophrenia & Disability Reduction

DISABILITY REDUCTION IN ELDERLY PATIENTS WITH SCHIZOPHRENIA.

Authors : SCHIMMING, CORBETT MD; HARVEY, PHILIP D. PhD.

Source : Journal of Psychiatric Practice. 10(5):283-295, September 2004.

Summary : This article reviews a frequently overlooked subject, the topic of schizophrenia in late life. By examining the available literature on schizophrenia in this particular population, we hope to provide clinicians with a better understanding of the distinguishing characteristics, course, and optimal treatments of this disease in elderly patients. The validity of the concept of symptom "burn out" is discussed and the cognitive changes seen in schizophrenia as patients age are examined. Similarities and differences between late-onset schizophrenia and early-onset schizophrenia in aging patients are compared. The similarities and differences between schizophrenia and dementia in the elderly are also discussed. Finally, treatments for the illness, including both typical and atypical antipsychotic treatments, as well as nonpharmacological intervention strategies, along with their advantages and disadvantages, are reviewed.

Schizophrenia & Sulpiride

SULPIRIDE FOR SCHIZOPHRENIA

Authors : Soares BGO, Fenton M, Chue P

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 24 November 1998. Cochrane reviews are regularly checked and updated if necessary.

Background: The antipsychotic drug sulpiride was formulated over 20 years ago and was marked as having a low incidence of adverse effects and an effect on the negative symptoms of schizophrenia. This relatively inexpensive antipsychotic drug has a similar neuropharmacological profile to several novel

atypical drugs.

Objectives: To estimate the clinical efficacy and tolerability of sulpiride.

Search strategy: Electronic searches of Biological Abstracts (1982-1997), CINAHL (1982-1998), Cochrane Schizophrenia Group's Register (March 1998), Cochrane Library (Issue 1, 1998), EMBASE (1980-1998), MEDLINE (1966-1998), PsycLIT (1974-1997), SIGLE (1994-1998), and Sociofile (1974-1997) were supplemented by reference searching, contacting authors and the manufacturers of sulpiride.

Selection criteria: All randomised or quasi-randomised clinical trials focusing on the use of different doses of sulpiride or comparing sulpiride to (i) placebo; (ii) typical antipsychotic drugs; or (iii) atypical antipsychotic drugs, for those with schizophrenia or serious mental illness were selected.

Data collection and analysis: Trials were reliably selected and quality rated. Data were independently extracted, by two reviewers (BGOS, MF), and analysed on an intention-to-treat basis. It was assumed that people who did not complete the follow up had no improvement. Authors of trials were contacted for additional and missing data. Relative risk (RR) and 95% confidence intervals (CI) of dichotomous data were calculated with the random effects model and weighted mean difference (WMD) was calculated for continuous data.

Main results: The review currently includes 18 studies (30 citations). Studies are generally small and of poor quality. Limited evidence suggests that there is little difference between sulpiride and other drugs although the incidence of side effects may be less for sulpiride. There are no clear findings relating to negative symptoms.

Reviewers' conclusions: Sulpiride may be an effective antipsychotic drug but evidence is limited and data relating to claims for its value against negative symptoms is not trial-based.

SCHIZOPHRENIA & QUETIAPINE

■ QUETIAPINE FOR SCHIZOPHRENIA

Authors : Srisurapanont M, Maneeton B, Maneeton N

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 21 January 2004. Cochrane reviews are regularly checked and updated if necessary.

Background: Quetiapine is an atypical antipsychotic with, theoretically, a low propensity for movement disorder adverse effects. It is used for the treatment of schizophrenia and other psychoses.

Objectives: To determine the effects of quetiapine for schizophrenia in comparison to placebo, and other antipsychotics.

Search strategy: Electronic searches of the Cochrane Schizophrenia Group's Register of Trials (February 2003), Biological Abstracts (1982-2000), CINAHL (1982-2000), the Cochrane Library (2000, Issue 1), EMBASE (1980-2000), MEDLINE (1966-2000), PsycLIT (1974-2000), SIGLE on CD (1980-1997), SocioFile (1974-1997) and many conference proceedings and hand searches of specific journals were undertaken. We contacted AstraZeneca Pharmaceuticals for information regarding unpublished trials. The review was updated in February 2003.

Selection criteria: All randomised controlled trials where adults with schizophrenia or similar illnesses were assigned to quetiapine, placebo or other neuroleptic drugs and where clinically relevant outcomes were reported.

Data collection and analysis: Citations and, where possible, abstracts were inspected independently by reviewers, papers ordered, re-inspected and quality assessed. We independently extracted data. We analysed data using fixed effects relative risk (RR) and estimated the 95% confidence interval (CI). Only homogeneous data were interpreted as favouring treatment or control. Where possible we calculated the number needed to treat (NNT) or number needed to harm statistics (NNH). We calculated relative risk (RR) for dichotomous data, and weighted mean differences (WMD) for continuous data.

Main results: Despite the fact that 3443 people were randomised in 12 quetiapine studies, there are almost no data on service utilisation, economic outcomes, social functioning and quality of life. Over half of those within the quetiapine versus placebo comparison were lost to follow up (53% quetiapine vs 61% placebo, $n=716$, 4 RCTs, RR 0.84 CI 0.7 to 0.9, NNT 11 CI 7 to 55) so it is impossible to interpret any ratings of global or mental state within this comparison with confidence. People allocated quetiapine, however, did not have more movement disorders than those given placebo ($n=395$, 2 RCTs, RR needing medication for EPSE 0.62 CI 0.3 to 1.2). The same applies to the comparison of ≥ 250 mg/day quetiapine with < 250 mg/day quetiapine (49% dropout ≥ 250 mg/day vs 58% < 250 mg/day, $n=1066$, 3 RCTs, RR 0.84 CI 0.8 to 0.9, NNT 11 CI 7 to 29). It should be noted that two deaths occurred in the higher dose group ($n=618$, 1 RCT, RR 0.1 CI 0.0 to 2.1). When quetiapine was compared with typical antipsychotics, about 36% of both groups failed to complete the short-term studies ($n=1624$, 6 RCTs, RR 0.87 CI 0.8 to 1.0). Average change in global state was heterogeneous and equivocal ($n=762$, 3 RCTs, WMD in short term 0.19 CI 0.00 to 0.38, I^2 76%). Mental state measures were also equivocal ($n=1247$, RR not improved 0.97 CI 0.9 to 1.1) including specific measures of negative symptoms ($n=305$, 1 RCT, MD change in SANS short term 0.94 CI -0.2 to 2.0). Movement disorders were less prevalent for those allocated quetiapine ($n=1117$, 4 RCTs, RR needing medication for extrapyramidal adverse effects 0.47 CI 0.4 to 0.6, NNT 4 CI 4 to 5, I^2 88%). Dry mouth ($n=649$, 2 RCTs, RR short term 2.85 CI 1.5 to 5.6, NNH 17 CI 7 to 65) and sleepiness ($n=959$, 3 RCTs, RR 1.51 CI 1.1 to 2.2, NNH 18 CI 8 to 181) may also be more prevalent for people given quetiapine compared with the older drugs. In the quetiapine versus risperidone comparison, over 30% of people left the study before completion ($n=728$, 1 RCT, RR 0.94 CI 0.7 to 1.2). Four people, all treated with quetiapine, died during the study ($n=728$, 1 RCT, RR 2.86 CI 0.2 to 52.8). Continuous mental state measures did not show clear differences between the two drugs ($n=637$, 1 RCT, MD PANSS 1.2 CI -2.0 to 4.4). However, considerably fewer people given quetiapine needed medication for extrapyramidal side effects compared with those allocated to risperidone ($n=712$, 1 RCT, RR 0.27 CI 0.2 to 0.5, NNT 11 CI 10 to 16). Quetiapine caused more dizziness ($n=728$, 1 RCT, RR 1.85 CI 1.0 to 3.3, NNH 18 CI 7 to 487), more dry mouth ($n=728$, 1 RCT, RR 2.11 CI 1.2 to 3.8, NNH 14 CI 6 to 82) and more sleepiness than risperidone ($n=728$, 1 RCT, RR 2.03 CI 1.4 to 2.9, NNH 7 CI 4 to 17).

Reviewers' conclusions: Quetiapine is effective for the treatment of schizophrenia, but it is not much different from first-generation antipsychotics and risperidone with respect to treatment withdrawal and efficacy. In comparison to first-generation antipsychotics and risperidone, quetiapine has a lower risk of movement disorders but higher risks of dizziness, dry mouth and sleepiness. More clearly reported pragmatic randomised controlled trials should be carried out to determine

its position in everyday clinical practice. Studies of medium and long-term effects, including cost-effectiveness, quality of life, social functioning and service utilisation, in comparison with the effects of typical and atypical antipsychotics should be priority areas.

Schizophrenia & Olanzapine

■ OLANZAPINE FOR SCHIZOPHRENIA

Authors : Duggan L, Fenton M, Dardennes RM, El-Dosoky A, Indran S

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 27 October 1999. Cochrane reviews are regularly checked and updated if necessary.

Background: Olanzapine is an atypical antipsychotic that is reported to be effective without producing the disabling extrapyramidal side effects associated with the older, typical antipsychotic drugs.

Objectives: To determine the clinical effects and safety of olanzapine as compared with placebo, typical and other atypical antipsychotic drugs for schizophrenia and schizophreniform psychoses.

Search strategy: The reviewers undertook electronic searches of Biological Abstracts (1980-1999), The Cochrane Library (Issue 2, 1999), The Cochrane Schizophrenia Group's Register (September 1999), EMBASE (1980-1999), MEDLINE (1966-1999), and PsycLIT (1974-1999). References of all identified studies were searched for further trials, and the reviewers contacted relevant pharmaceutical companies and authors of trials.

Selection criteria: All randomised clinical trials comparing olanzapine to placebo or any antipsychotic treatment for those with schizophrenia or schizophreniform psychoses.

Data collection and analysis: Data were independently extracted. For homogeneous dichotomous data the random effects relative risk (RR), the 95% confidence intervals (CI) and, where appropriate, the number needed to treat (NNT) were calculated on an intention-to-treat basis. For continuous data the reviewers calculated weighted mean differences.

Main results: Twenty trials are included. Attrition from olanzapine versus placebo studies was so great (olanzapine - 61%, placebo - 73% by 6 weeks, RR 0.85 CI 0.7-0.98, NNT 8 CI 5-40) that interpretation of results is problematic. Olanzapine appeared superior to placebo at six weeks for the outcome of 'no important clinical response' (RR 0.88 CI 0.8-0.98, NNT 8 CI 5-27), but trial data regarding negative symptoms are equivocal for this comparison. Dizziness and dry mouth were more common in the olanzapine-treated group and, although not statistically significant, the olanzapine group gained more weight. Data from several small trials are incomplete; but, for the short term outcome of 'no important clinical response', olanzapine seem as effective as typical antipsychotics (n=2778, RR 0.9 CI 0.76-1.06). Brief Psychiatric Rating Scale (BPRS) data tended to be equivocal but Positive and Negative Syndrome Scale (PANSS) rating of total score and negative and positive symptom sub-scores favoured olanzapine. With high attrition in both groups (olanzapine - 36%, typical drug - 49% by 6 weeks, n=2738, RR 0.85 CI 0.66-1.1; olanzapine - 83%, typical drug - 90% by 1 year, n=2738, RR 0.9 CI 0.86-1.02), the assumptions included in all continuous data are considerable. Participants allocated olanzapine experienced less extrapyramidal side effects than people given haloperidol. Weight change data for

the short term are not conclusive (n=2455, WMD 0.8kg CI -0.6-2.2) but the three to 12 month results suggest an average gain of four kilograms (n=233, WMD 4 CI 0.3-7.8). It is difficult to distinguish between olanzapine and other atypical drugs, although it may cause less extrapyramidal side effects than risperidone (n=339, RR 0.6 CI 0.4-0.9, NNT 8 CI 4-29). Olanzapine did cause more weight gain than its comparators but current data are not statistically significant (3-12 months, n=535, WMD 2.2kg CI -0.6-5). One study (n=180) found no clear differences between olanzapine and clozapine for people with treatment resistant illness.

Reviewers' conclusions: For people with schizophrenia olanzapine may offer antipsychotic efficacy with less extrapyramidal side effects than typical drugs but more weight gain. The large proportions of participants leaving the studies early, in the large multi-centre trials makes it difficult to draw firm conclusions on clinical effects. Large, long-term randomised trials with participants, interventions and primary outcomes that are familiar to those wishing to help those with schizophrenia are long overdue.

Schizophrenia & Haloperidol

■ HALOPERIDOL DOSE FOR THE ACUTE PHASE OF SCHIZOPHRENIA

Authors : Warchick PS, Adams CE, Roque M, Hamill KM, Marti 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 06 February 2002. Cochrane reviews are regularly checked and updated if necessary.

Background: Haloperidol is a benchmark, accessible antipsychotic against which the effects of newer treatments are gauged.

Objectives: The primary goal of this review is to determine the best range of doses for haloperidol for the treatment of people acutely ill with schizophrenia.

Search strategy: The reviewers searched Biological Abstracts (1980-1999), CINAHL (1982-1999), The Cochrane Library (1999, Issue 2), The Cochrane Schizophrenia Group's Register (December 1999), EMBASE (1980-1999), MEDLINE (1966-1999) and PsycLIT (1887-1999). They also inspected all references of all identified trials and included studies sought as a citation on SCISEARCH database (1980-1999). Authors of identified studies and pharmaceutical companies were also contacted.

Selection criteria: Studies were selected if they involved people being treated for acute schizophrenia, randomised to two or more dose ranges of non-depot haloperidol, and if they reported clinically meaningful outcomes.

Data collection and analysis: The reviewers independently and blindly inspected citations (10% reliability check), they ordered papers, and reliably re-inspected and quality assessed the full reports. The reviewers, again working independently, also extracted data. For homogeneous dichotomous data the relative risk (RR), 95% confidence intervals (CI) were calculated on an intention-to-treat basis. Reviewers assumed that people who left the study early or were lost to follow-up had a negative outcome. Weighted mean differences (WMD) were calculated for continuous outcomes that reported intention to treat (ITT), last observation carried forward (LOCF) data. Data was excluded if loss to follow-up was greater than 50%.

Main results: Sixteen trials with nineteen different randomised dose comparisons were included. No studies

reported data on relapse rates, quality of life and none compared >1.5-3.0 mg/day haloperidol to higher dose ranges. Using low doses (>3-7.5mg/day) did not clearly result in loss of efficacy (no clinically important improvement in global state, versus >7.5-15mg/day n=48, 1 RCT, RR 1.09 CI 0.7 to 1.8; versus >15-35mg/day n=81, 2 RCTs, 0.95 CI 0.8 to 1.2). Doses of haloperidol in the range of >3-7.5 mg/day had a lower rate of development of clinically significant extrapyramidal adverse effects than higher doses (clinically significant extrapyramidal adverse effects, versus >7.5-15mg/day n=64, 2 RCTs, RR 0.12 CI 0.01 to 2.1; versus >15-35mg/day n=144, 3 RCTs RR 0.59 CI 0.5 to 0.8, NNH 3 CI 2 to 6; versus >35mg/day n=86, 2 RCTs, RR 0.70 CI 0.5 to 1.1). All other comparisons between dose ranges did not yield statistically significant differences, but several, particularly with lower dose ranges, were underpowered to detect clinically meaningful differences.

Reviewers' conclusions: No results are conclusive and all are based on small, short, studies. It would be understandable, however, if clinicians were cautious in prescribing doses in excess of 7.5 mg/day of haloperidol to a person with uncomplicated acute schizophrenia, and if people with schizophrenia were equally reticent to take greater doses. Further research is needed regarding the efficacy and tolerability of the >1.5-3.0 mg/day dose range.

Schizophrenia & Family

FAMILY INTERVENTION FOR SCHIZOPHRENIA

Authors : Pharoah FM, Rathbone J, Mari JJ, Streiner D

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 12 May 2003. Cochrane reviews are regularly checked and updated if necessary.

Background: It has been found that people with schizophrenia from families that express high levels of criticism, hostility, or over involvement, have more frequent relapses than people with similar problems from families that tend to be less expressive of their emotions. Psychosocial interventions designed to reduce these levels of expressed emotions within families now exist. These interventions are proposed as adjuncts rather than alternatives to drug treatments and their main purpose is to decrease the stress within the family and also the rate of relapse.

Objectives: To estimate the effects of family psychosocial interventions in community settings for the care of those with schizophrenia or schizophrenia-like conditions compared to standard care.

Search strategy: We updated previous searches of the Cochrane Schizophrenia Group Register (June 1998), MEDLINE (1966-1995), the Cochrane Library (Issue 2 1998), EMBASE (1981-1995) and MEDLINE (1966-1995) by searching Cochrane Schizophrenia Group Register (November 2002). References of all identified studies were searched for further trial citations and authors of trials were contacted.

Selection criteria: Randomised or quasi-randomised studies were selected if they focused primarily on families of people with schizophrenia or schizoaffective disorder and compared community-orientated family-based psychosocial intervention of more than five sessions with standard care.

Data collection and analysis: Data were reliably extracted, and, where appropriate and possible, summated. Relative risk

(RR) with 95% confidence intervals (CI) and number needed to treat (NNT) were estimated. The reviewers assume that people who died or dropped out had no improvement and tested the sensitivity of the final results to this assumption.

Main results: Family intervention may decrease the frequency of relapse (n=721, 14 RCTs, RR 0.72 CI 0.6 to 0.9, NNT 7 CI 5 to 16). These data are statistically heterogeneous, the trend over time of this finding is towards the null and some small but negative studies may not have been identified by the search. Family intervention may also encourage compliance with medication (n=369, 7 RCTs, RR 0.74 CI 0.6 to 0.9, NNT 7 CI 4 to 19) but does not obviously affect the tendency of individuals/families to drop out of care (n=327, 4 RCTs, RR attrition at 3 months 0.86 CI 0.3 to 2.1). It may improve general social impairment and the levels of expressed emotion within the family. This review provides no data to suggest that family intervention either prevents or promotes suicide.

Reviewers' conclusions: Clinicians, researchers, policy makers and recipients of care cannot be confident of the effects of family intervention from the findings of this review. Further data from already completed trials could greatly inform practice and more trials are justified as long as their participants, interventions and outcomes are applicable to routine care.

Schizophrenia, Schizoaffective & Carbamazepine

CARBAMAZEPINE FOR SCHIZOPHRENIA AND SCHIZOAFFECTIVE PSYCHOSES

Authors : Leucht S, McGrath J, White P, Kissling W

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 09 April 2002. Cochrane reviews are regularly checked and updated if necessary.

Background: Many people with schizophrenia do not achieve a satisfactory treatment response with ordinary antipsychotic drug treatment and various additional medications are used to promote additional response. The antiepileptic carbamazepine is one such drug.

Objectives: To review the effects of carbamazepine and its derivatives for the treatment of schizophrenia and schizoaffective psychoses.

Search strategy: We searched Biological Abstracts (1980-2001), The Cochrane Library (Issue 3, 2001), The Cochrane Schizophrenia Group's Register of Trials (December 2001), EMBASE (1980-2001), MEDLINE (1966-2001), PsycLIT (1886-2001) and PSYINDEX (1974-2001). Citations from included trials were also inspected and relevant companies and authors contacted for additional data.

Selection criteria: All randomised controlled trials comparing carbamazepine or compounds of the carbamazepine family to placebo or no intervention, whether as sole treatment or as an adjunct to antipsychotic medication for the treatment of schizophrenia and/or schizoaffective psychoses.

Data collection and analysis: Citations and, where possible, abstracts were independently inspected by reviewers, papers ordered, re-inspected and quality assessed. Data were extracted independently by at least two reviewers. Dichotomous data were analysed using relative risks (RR) and the 95% confidence interval (CI) estimated. Where possible the number needed to treat (NNT) or number needed to harm statistics were calculated.

Main results: Ten studies with a total of 258 participants were included. One study comparing carbamazepine with placebo

as the sole treatment for schizophrenia (n=31) was stopped early due to high relapse rate. No effect of carbamazepine was evident (RR relapse 4.1 CI 0.8 to 1.5). Another study (n=38) compared carbamazepine with antipsychotics as the sole treatment for schizophrenia. No differences in terms of mental state were found (RR 50% BPRS reduction 1.2 CI 0.8 to 1.9). More people who received the antipsychotic (perphenazine) had parkinsonism (RR 0.03 CI 0.00 to 0.04, NNH 1 CI 0.9 to 1.4). Eight studies compared adjunctive carbamazepine plus antipsychotics versus placebo plus antipsychotics. Adding carbamazepine was as acceptable as adding placebo (n=182, RR leaving the study early 0.5 CI 0.2 to 1.4). Carbamazepine augmentation of antipsychotics was superior compared with antipsychotics alone in terms of overall improvement, but participant numbers were low (2 RCTs, n=38, RR 0.6 CI 0.4 to 0.9, NNT 2 CI 1 to 5). There were no differences for mental state outcomes (6 RCTs n=147, RR 50% BPRS reduction 0.9 CI 0.7 to 1.1). Less people in the carbamazepine augmentation group had movement disorders than those taking haloperidol alone (1 RCT, n=20, RR 0.4 CI 0.1 to 1.0). The effects of carbamazepine on subgroups of people with schizophrenia and aggressive behaviour, negative symptoms or EEG abnormalities or with schizoaffective disorder are unknown. Reviewers' conclusions: Based on currently available randomised trial-derived evidence, carbamazepine cannot be recommend for routine clinical use for treatment or augmentation of antipsychotic treatment of schizophrenia. At present large, simple well-designed and reported trials are justified especially if focusing on those with violent episodes and people with schizoaffective disorders or on those with both schizophrenia and EEG abnormalities.

Schizophrenia & Beta-blocker

■ BETA-BLOCKER SUPPLEMENTATION OF STANDARD DRUG TREATMENT FOR SCHIZOPHRENIA

Authors : Cheine M, Ahonen J, Wahlbeck K

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 15 May 2001. Cochrane reviews are regularly checked and updated if necessary.

Background: Many people with schizophrenia or similar severe mental disorders do not achieve a satisfactory treatment response with ordinary antipsychotic drug treatment. In these cases, various add-on medications are used, among them beta-adrenergic receptor antagonists (beta-blockers).

Objectives: To evaluate the clinical effects of beta-blockers as an adjunct to antipsychotic medication in schizophrenia or similar severe mental disorders.

Search strategy: Publications in all languages were searched from the following databases: Biological Abstracts (1982-2000), The Cochrane Library (Issue 3, 2000), The Cochrane Schizophrenia Group's Register (November 2000), EMBASE (1980-2000), LILACS (1982-1996), MEDLINE (1966-2000) and PsycLIT (1974-2000). Reference sections of included papers were screened.

Selection criteria: All randomised controlled trials comparing beta-blockers with placebo as an adjunct to conventional antipsychotic medication for those with schizophrenia.

Data collection and analysis: Studies were selected and then data extracted, independently, by at least two reviewers. Odds ratios (OR) and 95% confidence intervals (CI) of

homogeneous dichotomous data were calculated using the Peto method. A random effects model was used for heterogeneous dichotomous data. Weighted mean differences were calculated for continuous data.

Main results: Currently the review includes five studies but data are poorly presented and there is no evidence of any effect of beta-blockers as an adjunct to conventional antipsychotic medication.

Reviewers' conclusions: At present beta-blockers cannot be recommended in the treatment of schizophrenia. Any possible benefit of adjunctive beta-blockers is obscured by poor reporting within included studies. Existing data on beta-blockers as adjunctive medication to antipsychotics for those with schizophrenia should be collected and re-analysed in order to allow confident conclusions about the effect of this treatment or the need for further trials.

Schizophrenia & Long-Acting Risperidone

■ PRACTICAL APPLICATION OF PHARMACOTHERAPY WITH LONG-ACTING RISPERIDONE FOR PATIENTS WITH SCHIZOPHRENIA.

Authors : Keith SJ, Pani L, Nick B, Emsley R, San L, Turner M, Conley R, Scully P, Chue PS, Lachaux B. - the University of Stellenbosch in Cape Town, South Africa.

Source : Psychiatr Serv. 2004 Sep;55(9):997-1005

Summary: It is now generally accepted that the use of second-generation, or atypical, antipsychotics for schizophrenia represents an advance over conventional antipsychotic agents. However, adherence continues to be a problem, as with other medications for chronic disorders. Long-acting formulations of conventional antipsychotics partly address adherence problems, but their use is limited by tolerability issues. This article provides practical advice to physicians on the characteristics of patients who would benefit from treatment with long-acting atypical antipsychotic agents and offers suggestions on how to initiate treatment. **METHODS:** A literature search for studies published between 1980 and 2003 that evaluated the treatment of patients with schizophrenia with long-acting atypical agents was conducted by using MEDLINE and EMBASE. The primary search parameters were "schizophrenia," "atypical," "antipsychotic," and "long-acting." As expected, long-acting risperidone was the only long-acting atypical agent identified; thus this article focuses on practical advice and suggestions on how to initiate therapy with long-acting risperidone. **RESULTS AND DISCUSSION:** From the results of the literature search and the discussion of a panel of experts at a meeting held in Dublin in 2003 and supported by Johnson & Johnson, it is possible to conclude that long-acting risperidone has demonstrated efficacy and tolerability, even among patients who are considered clinically stable on other antipsychotics. Most patients can switch safely and effectively to long-acting risperidone if appropriate strategies are applied. Long-acting risperidone provides a new and promising therapeutic option for the treatment of schizophrenia.

Schizophrenia & The Independent Living Scales

■ THE INDEPENDENT LIVING SCALES AS A MEASURE OF FUNCTIONAL OUTCOME FOR SCHIZOPHRENIA.

Authors : Revheim N, Medalia A. - Bronx, New York

Source : Psychiatr Serv. 2004 Sep;55(9):1052-4

Summary: The Independent Living Scales (ILS) measures cognitive skills required for independent living and is intended to provide guidelines for appropriate supervision requirements for persons in residential placement. To assess the validity of the ILS among persons with schizophrenia, the instrument was administered to 162 individuals with schizophrenia who were living in three gradations of care: maximum supervision, moderate supervision, and minimal supervision. Scores on the ILS differed significantly across the three levels of care, whereas scores on the Global Assessment of Functioning (GAF) that were provided by clinicians discriminated only two levels of care. The ILS can be used among patients with schizophrenia to measure cognition as it affects functional outcome.

Schizophrenia & Tobacco Dependence

A CASE SERIES OF NICOTINE NASAL SPRAY IN THE TREATMENT OF TOBACCO DEPENDENCE AMONG PATIENTS WITH SCHIZOPHRENIA.

Authors : Williams JM, Ziedonis DM, Foulds J. - the University of Medicine and Dentistry of New Jersey-Robert Wood Johnson Medical School, 671 Hoes Lane, D339, Piscataway, New Jersey 08855. jill.williams@umdnj.edu

Source : Psychiatr Serv. 2004 Sep;55(9):1064-6

Summary: A retrospective case series of 12 smokers with schizophrenia or schizoaffective disorder who had not successfully quit smoking with previous treatments for tobacco dependence were treated with nicotine nasal spray. All but one patient (92 percent) tolerated the nasal spray well, and nine (75 percent) used it at maximal doses for prolonged periods. After treatment five patients (42 percent) were abstinent from smoking for more than 90 days, and four patients (33 percent) substantially reduced the amount that they smoked. Ten patients (83 percent) used the spray in combination with other medications, and all received psychosocial support. Nicotine nasal spray was found to be well tolerated.

Schizoaffective Disorder

IS SCHIZOAFFECTIVE DISORDER A STABLE DIAGNOSTIC CATEGORY: A RETROSPECTIVE EXAMINATION.

Authors : Averill PM, Reas DL, Shack A, Shah NN, Cowan K, Krajewski K, Kopecky C, Guynn RW. - University of Texas Health Science Center at Houston, Department of Psychiatry and Behavioral Sciences and the Harris County Psychiatric Center, Houston, Texas, USA. patricia.averill@uth.tmc.edu

Source : Psychiatr Q. 2004 Fall;75(3):215-27

Summary: Debate continues about whether clear nosologic boundaries can be drawn between schizoaffective disorder (SA), schizophrenia (SP), and bipolar disorder (BPD). This study attempted to clarify these boundaries. A retrospective review of the records of adult psychiatric inpatients with DSM-IV diagnoses of SA (n = 96), SP (n = 245), and BPD (n = 203) was conducted. Patients were assessed at admission and discharge using standardized rating scales (completed by physicians and nurses) and self-report inventories. Differential improvement over time also was examined. Significant differences were found for gender, legal status at admission, age, LOS, episode number, and ethnicity. Overall, SA was rated by clinicians as intermediate between SP and BPD,

although SA rated themselves as the most severe. SA was similar to SP on positive symptoms, intermediate on negative symptoms, and similar to BPD on mood- and distress-related symptoms. Independent of diagnosis, differences in change scores from admission to discharge were related to severity level at admission. Although several differences were found in symptom severity across domains, no syndrome was identifiable associated with the diagnosis of SA and the diagnosis was unstable over time, thereby bringing into question the validity of SA as a diagnostic entity.

Schizophrenia & CBT

COGNITIVE BEHAVIORAL THERAPY IN THE TREATMENT OF SCHIZOPHRENIA

Authors : D Turkington & R Dudley

Source : Expert Review of Neurotherapeutics 4(5),861-868 (2004)

Summary: This review outlines the role that cognitive behavioral therapy can play in specifically addressing the distress associated with the symptoms of schizophrenia, such as hallucinations and delusions. Some of the features that are given greater emphasis (or are a feature of working with people with psychotic illness), engagement, understanding the onset of the illness and work with hallucinations and delusional beliefs are outlined. The evidence base for the utility of cognitive behavioral therapy is considered, and the development and further application of cognitive behavioral therapy for schizophrenia and related disorders are outlined. cognitive behavioral therapy, psychosis, schizophrenia.

Schizophrenia Refractory & Clozapine

EQUIVALENT OCCUPANCY OF DOPAMINE D1 AND D2 RECEPTORS WITH CLOZAPINE: DIFFERENTIATION FROM OTHER ATYPICAL ANTIPSYCHOTICS.

Authors : Tauscher J, Hussain T, Agid O, Verhoeff NP, Wilson AA, Houle S, Remington G, Zipursky RB, Kapur S. - Department of General Psychiatry, Medical University of Vienna, Austria, Wahringerturle 18-20, A-1090 Vienna, Austria. johannes.tauscher@meduniwien.ac.at

Source : Am J Psychiatry. 2004 Sep;161(9):1620-5

Summary: OBJECTIVE: Clozapine, the prototype of atypical antipsychotics, remains unique in its efficacy in the treatment of refractory schizophrenia. Its affinity for dopamine D(4) receptors, serotonin 5-HT(2A) receptor antagonism, effects on the noradrenergic system, and its relatively moderate occupancy of D(2) receptors are unlikely to be the critical mechanism underlying its efficacy. In an attempt to elucidate the molecular/synaptic mechanism underlying clozapine's distinctiveness in refractory schizophrenia, the authors studied the in vivo D(1) and D(2) receptor profile of clozapine compared with other atypical antipsychotics. METHOD: Positron emission tomography with the radioligands [(11)C]SCH23390 and [(11)C]raclopride was used to investigate D(1) and D(2) receptor occupancy in vivo in 25 schizophrenia patients receiving atypical antipsychotic treatment with clozapine, olanzapine, quetiapine, or risperidone. RESULTS: Mean striatal D(1) occupancies ranged from 55% with clozapine to 12% with quetiapine (rank order: clozapine > olanzapine > risperidone > quetiapine). The striatal D(2) occupancy ranged from 81% with risperidone to 30% with quetiapine (rank order: risperidone > olanzapine >

clozapine > quetiapine). The ratio of striatal D(1)/D(2) occupancy was significantly higher for clozapine (0.88) relative to olanzapine (0.54), quetiapine (0.41), or risperidone (0.31). **CONCLUSIONS:** Among the atypical antipsychotics, clozapine appears to have a simultaneous and equivalent occupancy of dopamine D(1) and D(2) receptors. Whether its effect on D(1) receptors represents agonism or antagonism is not yet clear, as this issue is still unresolved in the preclinical arena. This distinctive effect on D(1)/D(2) receptors may be responsible for clozapine's unique effectiveness in patients with schizophrenia refractory to other typical and atypical antipsychotics.

Schizophrenia & Stress in Adult Students

STRESS IN ADULT STUDENTS WITH SCHIZOPHRENIA IN A SUPPORTED EDUCATION PROGRAM.

Authors : Ponizovsky A, Grinshpoon A, Sasson R, Levav I.

Source : Compr Psychiatry. 2004 Sep-Oct;45(5):401-7

Summary: The successful integration of former psychiatric inpatients into the community requires innovative programs of psychosocial rehabilitation, including supported education. This article examines psychological distress as an outcome variable, and social support and coping strategies as mediating variables among 70 service-user students (SUS) with schizophrenia and a comparison group of 55 adult students (AS) with no psychiatric diagnosis. Both groups were participants in a supported education program. The study variables were assessed by standardized research instruments: the Talbich Brief Distress Inventory (TBDI), the Multidimensional Scale of Perceived Social Support (MSPSS), and the Coping Inventory for Stressful Situations (CISS). Univariate and multivariate analyses were used. Compared with the control subjects, SUS reported higher emotional distress and the utilization of emotion-oriented coping strategies, and a lesser availability of social support from family and friends. These variables explained 46.3%, 24.5%, and 22.5%, respectively, of the total variance in psychological distress scores. The findings provide the basis for interventions geared to reduce distress and, as a result, to enable students with severe mental illness to fully utilize the supported education program.

Schizophrenia & Diabetes

UNDERSTANDING SCHIZOPHRENIA AND DIABETES.

Authors : Dinan T, Peveler R, Holt R. - University College Cork, National University of Ireland.

Source : Hosp Med. 2004 Aug;65(8):485-8

Summary: Alongside other risk factors for the development of type 2 diabetes, the presence of severe mental illness is often overlooked. A person with schizophrenia has a two to four times greater risk of developing diabetes than the general population and the prevalence of type 2 diabetes is between 15 and 18% in the schizophrenia population. A full understanding of this issue is vital.

Schizophrenia & Neural Circuitry Function

CHANGES IN DISTRIBUTED NEURAL CIRCUITRY FUNCTION IN PATIENTS WITH FIRST-EPISODE SCHIZOPHRENIA.

Authors : Mendrek A, Laurens KR, Kiehl KA, Ngan ET, Stip E, Liddle PF. - Department of Psychiatry, University of Montreal, Centre de recherche Fernand-Seguin, 7331 Hochelaga, Montreal, Quebec H1N 3V2, Canada. amendrek@crfs.umontreal.ca

Source : Br J Psychiatry. 2004 Sep;185:205-14

Summary: **BACKGROUND:** A number of functional brain abnormalities have been reported in schizophrenia, but it remains to be determined which of them represent trait and state markers of the illness. **AIMS:** To delineate regional brain dysfunctions that remain stable and those that fluctuate during the course of schizophrenia. **METHOD:** A cohort of patients with first-episode schizophrenia and a matched group of control participants underwent functional magnetic resonance imaging on two occasions 6-8 weeks apart during performance of a working memory task. The patients' disease was in partial remission at the second scan. **RESULTS:** Relative to control participants, the function of the left dorsolateral prefrontal cortex, left thalamus and right cerebellum remained disturbed in the people with schizophrenia, whereas the dysfunction of the right dorsolateral prefrontal cortex, right thalamus, left cerebellum and cingulate gyrus normalised, with significant reduction in symptoms. **CONCLUSIONS:** These results suggest that dysfunction of the left fronto-thalamo-cerebellar circuitry is a relatively stable characteristic of schizophrenia, whereas disturbance of the right circuitry and cingulate gyrus is predominantly a state-related phenomenon.

Psychotropic Drugs (PD)

Benzodiazepines & Akathisia

BENZODIAZEPINES FOR NEUROLEPTIC-INDUCED ACUTE AKATHISIA

Authors : Lima AR, Soares-Weiser K, Bacaltchuk J, Barnes TRE

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 11 August 1999. Cochrane reviews are regularly checked and updated if necessary.

Background: Neuroleptic-induced akathisia is one of the most common and distressing early-onset adverse effects of antipsychotic drugs, being associated with poor compliance with treatment, and thus, ultimately, to an increase risk of relapse. This review assesses the role of benzodiazepines in the pharmacological treatment of this problem.

Objectives: To determine the effects of benzodiazepines versus placebo for people with neuroleptic-induced acute akathisia.

Search strategy: Biological Abstracts (January 1982-March 1999), The Cochrane Library (Issue 3 1999), The Cochrane Schizophrenia Group's Register (May 2001), EMBASE (January 1980-March 1999), LILACS (January 1982-March 1999), MEDLINE (January 1964-March 1999), PsycLIT (January 1974-March 1999), and SCISEARCH were searched. Further references were sought from published trials and their authors.

Selection criteria: All randomised clinical trials comparing benzodiazepines with placebo for people with antipsychotic-induced acute akathisia.

Data collection and analysis: Two reviewers, working independently, selected, quality assessed and extracted data.

These data were then analysed on an intention-to-treat basis. For homogeneous dichotomous data the fixed effects relative risk (RR), the 95% confidence intervals (CI) and, where appropriate, the number needed to treat (NNT) were calculated on an intention-to-treat basis. For continuous data, reviewers calculated weighted mean differences.

Main results: Two small (total N=27) randomised controlled trials were included. By seven to 14 days, there was a reduction in symptoms for those patients receiving clonazepam compared with placebo (2 RCTs, N=26, RR 0.09 CI 0.01 to 0.6, NNT 1.2 CI 0.9 to 1.5). No significant difference was found for adverse events (2 RCTs, N=26, RR 3.00 CI 0.2 to 62) or the need for anticholinergic medication (2 RCTs, N=26, RR 1.56 CI 0.9 to 2.7). No one left the two studies early. Data on mental, social and family outcomes could not be pooled and there was little or no data on user satisfaction, deaths, violence, criminal behaviour and costs.

Reviewers' conclusions: Over a short follow-up period, the use of benzodiazepines may reduce the symptoms of antipsychotic-induced acute akathisia. This review highlights the need for well designed, conducted and reported clinical trials to address the claims of open studies.

Antidepressants & Smoking Cessation

■ ANTIDEPRESSANTS FOR SMOKING CESSATION

Authors : Hughes JR, Stead LF, Lancaster T

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 08 January 2003. Cochrane reviews are regularly checked and updated if necessary.

Background: There at least two reasons to believe antidepressants might help in smoking cessation. Depression may be a symptom of nicotine withdrawal, and smoking cessation sometimes precipitates depression. In some individuals, nicotine may have antidepressant effects that maintain smoking. Antidepressants may substitute for this effect.

Objectives: The aim of this review is to assess the effect of antidepressant medications in aiding long-term smoking cessation. The drugs include bupropion; doxepin; fluoxetine; imipramine; moclobemide; nortriptyline; paroxetine; selegiline; sertraline, tryptophan and venlafaxine.

Search strategy: We searched the Cochrane Tobacco Addiction Group trials register which includes trials indexed in MEDLINE, EMBASE, SciSearch and PsycINFO, and other reviews and meeting abstracts, in December 2002.

Selection criteria: We considered randomized trials comparing antidepressant drugs to placebo or an alternative therapeutic control for smoking cessation. For the meta-analysis, we excluded trials with less than six months follow-up.

Data collection and analysis: We extracted data in duplicate on the type of study population, the nature of the drug therapy, the outcome measures, method of randomization, 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, expressed as an odds ratio (OR). We used the most rigorous definition of abstinence for each trial, and biochemically validated rates if available. Where appropriate, we performed meta-analysis using a fixed effects model.

Main results: There was one trial each of moclobemide, sertraline and venlafaxine, two of fluoxetine, five of

nortriptyline, and twenty trials of bupropion. In the bupropion trials, 18 had a placebo arm, two of which tested long-term use to prevent relapse. Nine of the bupropion trials have been published in full. Nortriptyline (five trials, OR 2.80, 95% CI 1.81 - 4.32) and bupropion (16 trials, OR 1.97, 95% CI 1.67 - 2.34) both increased the odds of cessation. In one trial the combination of bupropion and nicotine patch produced slightly higher quit rates than patch alone, but this was not replicated in a second study. Two trials of extended therapy with bupropion to prevent relapse after initial cessation have failed to detect a long-term benefit.

Reviewers' conclusions: The antidepressants bupropion and nortriptyline can aid smoking cessation but selective serotonin reuptake inhibitors (e.g. fluoxetine) do not.

Anticonvulsant Drugs & Migraine

■ ANTICONVULSANT DRUGS FOR MIGRAINE PROPHYLAXIS

Authors : Chronicle E, Mulleners W

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 21 May 2004. Cochrane reviews are regularly checked and updated if necessary.

Background: Anticonvulsant drugs seem to be useful in clinical practice for the prophylaxis of migraine. This might be explained by a variety of actions of these drugs in the central nervous system that are probably relevant to the pathophysiology of migraine.

Objectives: To describe and assess the evidence from controlled trials on the efficacy and tolerability of anticonvulsants for preventing migraine attacks in adult patients with migraine.

Search strategy: We searched MEDLINE (from 1966 on) and the Cochrane Central Register of Controlled Trials (CENTRAL). Date of most recent search: April 2003. Additional information was gained from hand-searching specialist headache journals; correspondence with pharmaceutical companies, authors of reports, and experts in the field; and a wide variety of review articles and book chapters.

Selection criteria: Studies were required to be prospective, controlled trials of self-administered drug treatments taken regularly to prevent the occurrence of migraine attacks and/or to reduce the intensity of those attacks.

Data collection and analysis: Studies were selected and data extracted by two independent reviewers. For migraine frequency data, standardized mean differences (SMDs) were calculated for individual studies and pooled across studies. For dichotomous data on significant reduction in migraine frequency, odds ratios (ORs) and numbers-needed-to-treat (NNTs) were similarly calculated. Adverse events were analyzed by calculating numbers-needed-to-harm (NNHs) for studies using similar agents.

Main results: Fifteen papers were included in the review. Of these, 14 reported trials comparing anticonvulsants with placebo, as follows: four trials of divalproex sodium, three trials of topiramate, two trials of sodium valproate, two trials of gabapentin, and one trial each of carbamazepine, clonazepam, and lamotrigine. One paper reported a trial of sodium valproate versus an active comparator, flunarizine, and one trial of divalproex sodium versus placebo included a comparison against propranolol, also an active comparator. Data from 2024 patients were considered. Analysis of data

from eight trials ($n = 841$) demonstrates that anticonvulsants, considered as a class, reduce migraine frequency by about 1.4 attacks per 28 days as compared to placebo (SMD -0.60; 95% confidence interval [CI] -0.93 to -0.26). Data from 10 trials ($n = 1341$) show that anticonvulsants, considered as a class, also more than double the number of patients for whom migraine frequency is reduced by 50% or more, relative to placebo (OR 3.90; 95% CI 2.61 to 5.82; NNT 3.8; 95% CI 3.2 to 4.6). For seven trials of sodium valproate and divalproex sodium, NNHs for five clinically important adverse events ranged from 6.6 to 16.3. For the three trials of topiramate, NNHs for eight adverse events (100-mg dose) ranged from 2.4 to 32.9.

Reviewers' conclusions: Anticonvulsants appear to be both effective in reducing migraine frequency and reasonably well tolerated. There is noticeable variation among individual agents, but there are insufficient data to know whether this is due to chance or variation in true efficacy. Neither clonazepam nor lamotrigine was superior to placebo (one trial each). Relatively few robust trials are available for agents other than sodium valproate/divalproex sodium. Two recently published and large trials of topiramate demonstrated reasonable efficacy, and one further trial of this agent is anticipated in the near future.

Anticonvulsant Drugs & Chronic Pain

■ ANTICONVULSANT DRUGS FOR ACUTE AND CHRONIC PAIN

Authors : Wiffen P, Collins S, McQuay H, Carroll D, Jadad A, Moore A

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 23 May 2000. Cochrane reviews are regularly checked and updated if necessary.

Background: Anticonvulsant drugs have been used in the management of pain since the 1960s. The clinical impression is that they are useful for chronic neuropathic pain, especially when the pain is lancinating or burning.

Objectives: To evaluate the analgesic effectiveness and adverse effects of anticonvulsant drugs for pain management in clinical practice and to identify a clinical research agenda. Migraine and headache studies are excluded in this revision.

Search strategy: Randomised trials of anticonvulsants in acute, chronic or cancer pain were identified by Medline (1966-1999), Embase (1994-1999), SIGLE (1980-1999) and the Cochrane Controlled Trials Register (CENTRAL/CCTR) (Cochrane Library Issue 3, 1999). In addition, 40 medical journals were hand searched. Additional reports were identified from the reference list of the retrieved papers, and by contacting investigators. Date of most recent search: September 1999.

Selection criteria: Randomised trials reporting the analgesic effects of anticonvulsant drugs in patients, with subjective pain assessment as either the primary or a secondary outcome.

Data collection and analysis: Data were extracted by two independent reviewers, and trials were quality scored. Numbers-needed-to-treat (NNTs) were calculated from dichotomous data for effectiveness, adverse effects and drug-related study withdrawal, for individual studies and for pooled data.

Main results: Twenty-three trials of six anticonvulsants were considered eligible (1,074 patients). The only placebo-controlled study in acute pain found no analgesic effect of

sodium valproate. Three placebo-controlled studies of carbamazepine in trigeminal neuralgia had a combined NNT (95% confidence interval (CI)) for effectiveness of 2.5 (CI 2.0-3.4). A single placebo-controlled trial of gabapentin in post-herpetic neuralgia had an NNT of 3.2 (CI 2.4-5.0). For diabetic neuropathy NNTs for effectiveness were as follows: (one RCT for each drug) carbamazepine 2.3 (CI 1.6-3.8), gabapentin 3.8 (CI 2.4-8.7) and phenytoin 2.1 (CI 1.5-3.6). Numbers-needed-to-harm (NNHs) were calculated where possible by combining studies for each drug entity irrespective of the condition treated. The results were, for minor harm, carbamazepine 3.7 (CI 2.4-7.8), gabapentin 2.5 (CI 2.0-3.2), phenytoin 3.2 (CI 2.1-6.3). NNHs for major harm were not statistically significant for any drug compared with placebo. Phenytoin had no effect in irritable bowel syndrome, and carbamazepine little effect in post-stroke pain. Clonazepam was effective in one study of temporomandibular joint dysfunction.

Reviewers' conclusions: Although anticonvulsants are used widely in chronic pain surprisingly few trials show analgesic effectiveness. No trial compared different anticonvulsants. Only one studied considered cancer pain. There is no evidence that anticonvulsants are effective for acute pain. In chronic pain syndromes other than trigeminal neuralgia, anticonvulsants should be withheld until other interventions have been tried. While gabapentin is increasingly being used for neuropathic pain the evidence would suggest that it is not superior to carbamazepine.

Modafinil & SSRI

■ AN OPEN-LABEL STUDY OF ADJUNCTIVE MODAFINIL IN PATIENTS WITH SEDATION RELATED TO SEROTONERGIC ANTI-DEPRESSANT THERAPY.

Authors : Schwartz TL, Azhar N, Cole K, Hopkins G, Nihalani N, Simionescu M, Husain J, Jones N. - Department of Psychiatry, State University of New York Upstate Medical University, Syracuse, N.Y.

Source : J Clin Psychiatry. 2004 Sep;65(9):1223-7

Summary: In patients with major depressive disorder (MDD), excessive sleepiness and fatigue not only are major components of the disorder, but also may occur as side effects of antidepressant therapy. In addition, sedation may be a consequence of antidepressant regimens. The novel wake-promoting agent modafinil improves wakefulness and reduces fatigue across a variety of clinical disorders. This study assessed the use of modafinil as an adjunctive treatment in patients with MDD who reported sedation related to serotonergic antidepressant therapy. **METHOD:** Data were collected between September 2001 and December 2003. Twenty men and women with DSM-IV-defined MDD were enrolled in this 3-week, open-label, single-center study. In addition to ongoing and stable treatment with selective serotonin reuptake inhibitors (SSRIs), clinic patients received modafinil once daily. Efficacy assessments were conducted at 1-week intervals. **RESULTS:** Sixteen patients (80%) completed the study. Modafinil plus SSRIs significantly improved overall depressive symptoms, as shown by reductions in mean Hamilton Rating Scale for Depression total scores ($p < .001$ vs. baseline). Adjunctive modafinil significantly improved subjective estimates of wakefulness on the Epworth Sleepiness Scale ($p < .001$, all weeks) and reduced fatigue on the Fatigue Severity Scale ($p = .009$). At the final visit, modafinil had improved overall health status and health-

related quality of life, as shown by significant improvements in mean Medical Outcomes Study Short-Form 12-Item Health Survey total scores ($p = .007$) and in physical health ($p = .04$) and mental health ($p = .006$) subscores. **CONCLUSION:** In patients with MDD who experience sedation as a side effect of antidepressant therapy, adjunctive modafinil improved wakefulness and reduced fatigue. Modafinil plus SSRIs also improved mood and quality of life.

Thyroid Hormones & SSRI

PERIPHERAL THYROID HORMONES AND RESPONSE TO SELECTIVE SEROTONIN REUPTAKE INHIBITORS

Authors : Michael Gitlin, MD; Lori L. Altshuler, MD; Mark A. Frye, MD; Rita Suri, MD; Emily L. Huynh, BA; Lynn Fairbanks, PhD; Michael Bauer, MD; Stanley Korenman, MD

Source : J Psychiatry Neurosci 2004;29(5):383-6

Summary: To examine the relation between baseline measurements of thyroid function and response to selective serotonin reuptake inhibitors (SSRIs) and to consider the effect of these antidepressants on thyroid hormone levels. **Methods:** Nineteen subjects with major depression, but without a history of thyroid treatment or lithium treatment, were treated openly with either sertraline or fluoxetine in a university-affiliated tertiary care hospital. Hamilton Depression Rating Scale (Ham-D) scores were measured before and after treatment. Clinical Global Impressions (CGI) scores were measured at study end. Thyroid data, consisting of values for thyroid-stimulating hormone (TSH), triiodothyronine (T3, measured by radioimmunoassay [RIA]), thyroxine (T4, measured by RIA) and free T4, were collected before and after treatment. Complete thyroid data were available for 17 subjects. Data were collected during 1997-1999. **Results:** Baseline TSH correlated strongly with response to treatment as measured by change in Ham-D scores ($r = 0.64$, $p = 0.003$). Low TSH values correlated with greater improvement in depressive symptoms. Thyroid hormone levels decreased with treatment, but these decreases did not correlate with clinical improvement. **Conclusion:** Baseline thyroid function, as measured by serum TSH, may predict a patient's response to antidepressant treatment with SSRIs. Optimal thyroid function, beyond simply being within the normal laboratory values, may be necessary for an optimal response to antidepressants.

Weight & Antidepressants

EARLY PREDICTION OF CHANGES IN WEIGHT DURING SIX WEEKS OF TREATMENT WITH ANTIDEPRESSANTS.

Authors : Himmerich H, Schuld A, Haack M, Kaufmann C, Pollmacher T. - Max Planck Institute of Psychiatry, Kraepelinstrasse 10, Munich 80804, Germany

Source : J Psychiatr Res. 2004 Sep-Oct;38(5):485-9

Summary: Weight gain is a frequent and important side effect of psychotropic treatment. We sought to determine weight change predictors during treatment with antidepressant drugs. In 24 patients weight, plasma levels of leptin, tumor necrosis factor-alpha (TNF- [Formula: see text] and soluble TNF receptors were determined longitudinally and a multiple linear regression analysis was used to predict weight change from baseline to the sixth week of treatment. Changes of weight during the first week of treatment, but no other parameter, strongly predicted weight change until endpoint (adjusted [Formula: see text], [Formula: see text], [Formula: see text],

[Formula: see text]). Very early changes in weight during treatment with psychotropic drugs might be a simple and clinically useful predictor of future weight development.

Tricyclic Antidepressants

TRICYCLIC ANTIDEPRESSANTS, QT INTERVAL PROLONGATION, AND TORSADE DE POINTES.

Authors : Vieweg WV, Wood MA. - Department of Psychiatry, Cardiology Division, Medical College of Virginia, Virginia Commonwealth University, Richmond, VA 23238-5414, USA. vvieweg@vcu.edu

Source : Psychosomatics. 2004 Sep-Oct;45(5):371-7

Summary: The authors postulate mechanisms linking tricyclic antidepressants, QT interval prolongation, torsade de pointes, and sudden cardiac death. Case reports identify amitriptyline and maprotiline as the tricyclic antidepressants most likely to provoke torsade de pointes. Risk factors of family history of congenital long QT syndrome, age, female sex, metabolic and cardiovascular disease, metabolic inhibitors, hypokalemia, drug overdose, and co-prescription of drugs associated with QT interval prolongation were found in cases of torsade de pointes associated with tricyclic antidepressants. Clinicians should be cautious when prescribing tricyclic antidepressants with other drugs, such as thioridazine, that are capable of prolonging the QT interval.

Antipsychotic Medication & Diabetes

RELATIONSHIP BETWEEN ANTIPSYCHOTIC MEDICATION TREATMENT AND NEW CASES OF DIABETES AMONG PSYCHIATRIC INPATIENTS.

Authors : Citrome L, Jaffe A, Levine J, Allingham B, Robinson J. - New York University School of Medicine in New York City.

Source : Psychiatr Serv. 2004 Sep;55(9):1006-13

Summary: This study examined data on patients with serious and persistent mental illness in a large state hospital system to determine whether patients who took second-generation antipsychotics were more likely to develop diabetes mellitus than patients who took first-generation antipsychotics. **METHODS:** A case-control study design was used. A new prescription of an antidiabetic medication was used to identify new cases of diabetes mellitus. Odds ratios were calculated for exposure to second-generation antipsychotics (clozapine, risperidone, olanzapine, quetiapine, and multiple second-generation antipsychotics) compared with exposure to first-generation antipsychotics. Cases and controls were identified by using a database that contained drug prescription information from the inpatient facilities that were operated by the New York State Office of Mental Health. Data from January 1, 2000, to December 31, 2002, were examined. Among 13,611 unique patients who received antipsychotics, 8,461 met entry criteria of being hospitalized for at least 60 days and not having an antidiabetic medication prescribed in the past. A total of 181 of these inpatients received prescriptions for an antidiabetic medication at least 30 days after their admission. Eight controls ($N=1,448$) for each case ($N=181$) were matched by calendar year, length of observation period, race, age group, and diagnosis, giving a total sample of 1,629 patients. **RESULTS:** Statistically significant elevations in risk were seen among patients who received more than one second-generation antipsychotic or clozapine or quetiapine,

compared with patients who received first-generation antipsychotics alone. Although not statistically significant, odds ratios for olanzapine and risperidone were also elevated. Conditional logistic regression adjusting for gender and age did not change the results. **CONCLUSIONS:** Exposure to multiple second-generation antipsychotics or clozapine or quetiapine significantly increased the risk of treatment-emergent diabetes mellitus.

Atypical Antipsychotic & Diabetes

■ INCIDENCE OF NEWLY DIAGNOSED DIABETES ATTRIBUTABLE TO ATYPICAL ANTIPSYCHOTIC MEDICATIONS.

Authors : Leslie DL, Rosenheck RA. - Northeast Program Evaluation Center/182, 950 Campbell Ave., West Haven, CT 06516. douglas.leslie@yale.edu

Source : Am J Psychiatry. 2004 Sep;161(9):1709-11

Summary: **OBJECTIVE:** The purpose of the study was to determine the proportion of patients with schizophrenia with a stable regimen of antipsychotic monotherapy who developed diabetes or were hospitalized for ketoacidosis. **METHOD:** Patients with schizophrenia for whom a stable regimen of antipsychotic monotherapy was consistently prescribed during any 3-month period between June 1999 and September 2000 and who had no diabetes were followed through September 2001 by using administrative data from the Department of Veterans Affairs. Cox proportional hazards models were developed to identify the characteristics associated with newly diagnosed diabetes and ketoacidosis. **RESULTS:** Of the 56,849 patients identified, 4,132 (7.3%) developed diabetes and 88 (0.2%) were hospitalized for ketoacidosis. Diabetes risk was highest for clozapine (hazard ratio=1.57) and olanzapine (hazard ratio=1.15); the diabetes risks for quetiapine (hazard ratio=1.20) and risperidone (hazard ratio=1.01) were not significantly different from that for conventional antipsychotics. The attributable risks of diabetes mellitus associated with atypical antipsychotics were small, ranging from 0.05% (risperidone) to 2.03% (clozapine). **CONCLUSIONS:** Although clozapine and olanzapine have greater diabetes risk, the attributable risk of diabetes mellitus with atypical antipsychotics is small.

Stimulant Drugs Psychosis

■ STIMULANT PSYCHOSIS: SYSTEMATIC REVIEW.

Authors : Curran C, Byrappa N, McBride A. - Pendine Community Mental Health Trust, 124-126 Cowbridge Road West, Ely, Cardiff CF5 5BT, Wales, UK. Cath.Curran@CardiffandVale.wales.nhs.uk

Source : Br J Psychiatry. 2004 Sep;185:196-204

Summary: **BACKGROUND:** Psychosis associated with stimulant use is an increasing problem, but there is little research evidence about the nature of the problem and its management. **AIMS:** To critically review the literature on stimulant psychosis and sensitisation. **METHOD:** Systematic review of studies that have investigated stimulant use and psychosis in humans. The main outcome measures were increases in psychosis with stimulant use, and differences between stimulant users and non-users. **RESULTS:** Fifty-four studies met the inclusion criteria. Experimental studies show that a single dose of a stimulant drug can produce a brief increase in psychosis ratings (a "response") in 50-70% of participants with schizophrenia and pre-existing acute

psychotic symptoms, unaffected by the presence of antipsychotic medication. Those with schizophrenia who do not have acute psychotic symptoms respond, but less frequently (30%). There has been little research into the longer-term effects of use. **CONCLUSIONS:** Compliance with antipsychotic medication by someone with schizophrenia will not prevent a relapse or worsening of psychotic symptoms if stimulants are used. Low-dose antipsychotic treatment may be beneficial in stimulant users, to prevent sensitisation.

ISSR & Cardiovascular Effects

■ CARDIOVASCULAR EFFECTS OF SELECTIVE SEROTONIN REUPTAKE INHIBITORS

Authors : Jean-Sébastien Hulot, Ivan Berlin

Source : Sang Thrombose Vaisseaux. Number 16, volume 6, 302-8, Juin 2004, Mini-revue

Summary: Unlike other antidepressants such as tricyclic agents, selective serotonin reuptake inhibitors (SSRIs) have shown no evidence of cardiac toxicity even in patients with heart disease. They thus represent first line treatment in individuals with cardiopathies (notably of ischemic origin) with coexisting depression. SSRIs display some cardiovascular effects that may be beneficial in the management of such patients. Because they block reuptake of serotonin in platelets, some SSRIs may inhibit platelet activation even in patients receiving conventional anti-agregant therapy. This effect, considered as an adverse effect in other situations, may be beneficial in some cardiovascular disorders. Furthermore, SSRIs may reduce increased sympathetic activity, a common feature of depression and some cardiac disorders and re-establish the sympathetic-parasympathetic balance. These mechanisms may be involved in the reduction of cardiovascular morbidity and mortality in SSRI-treated patients reported in recent studies.

Keywords : depression, platelets, serotonin, cardiac autonomic function, cardiovascular disorders

GERIATRIC PSYCHIATRY (GP)

GP & DEPRESSION IN THE ELDERLY PATIENT

■ DEPRESSION IN THE ELDERLY PATIENT

Authors : Clement JP. Pole de psychiatrie du sujet age, centre hospitalier Esquirol, 87025 Limoges Cedex. jean-pierre.clement@chello.fr

Source : Rev Prat. 2004 Apr 15;54(7):725-33.

Summary : Depression is the most usual mental disorder in the elderly, but underdiagnosed and undertreated. Its prevalence is variable and depends on type and severity of episode. Nevertheless, even subsyndromic depression needs to be correctly treated. Depressive symptomatology observed in the elderly is often similar to adult presentation, but it can be masked and difficult to recognise. The different clinical features are described with underlining their particularities. Secondary depressions are also evoked with individualisation of "vascular" depression and its etiopathogenic hypotheses in relationship with observations given by cerebral neuroimaging. Risk factors of depression in old age are known, but recent studies have reviewed some of them, particularly by distinguishing late onset depression and early onset depression. According to therapeutic response and prognosis, it appears necessary to better discriminate them. Risk of

dementia after depression seems to be related with type of depressive episode and with the treatment efficacy. Finally, the problem of detection of depression in old age is discussed with a suggestion to use assessment instruments as the mini-GDS in all medical practices, to optimise diagnosis and management.

GP & TREATMENT OF Depression in the Elderly

TREATMENT OF DEPRESSION IN THE ELDERLY

Authors : Robert PH. Centre Memoire de ressources & de recherche, CHU, Universite de Nice-Sophia Antipolis--Pavilion M, hopital Pasteur, 06002 Nice.

PHILIPPE.ROBERT15@wanadoo.fr

Source : Rev Prat. 2004 Apr 15;54(7):734-8.

Summary : Depression is the most common mental health problem of later life. There is effective treatments for depression in primary care. Recommendation based on current evidence are: in primary care treatment there is no evidence that one class of antidepressant is anymore effective than others; although newer antidepressants are not more effective than older ones, they are better tolerated in healthy older people and in patients with medical co-morbidity and are safer especially in overdose. Lower dose antidepressant treatment is not recommended for older depressed patients.

GP & Antidepressant Efficacy

ANTIDEPRESSANT EFFICACY AND COGNITIVE EFFECTS OF REPETITIVE TRANSCRANIAL MAGNETIC STIMULATION IN VASCULAR DEPRESSION: AN OPEN TRIAL.

Authors : Fabre I, Galinowski A, Oppenheim C, Gallarda T, Meder JF, De Montigny C, Olie JP, Poirier MF. Sainte-Anne Hospital, University Department of Psychiatry, Paris, France.

Source : Int J Geriatr Psychiatry. 2004 Sep;19(9):833-42.

Summary : BACKGROUND: Beneficial effects of repetitive transcranial magnetic stimulation (rTMS) were demonstrated by many controlled studies in major depression. Moreover, this promising and non invasive therapeutic tool seems to be better tolerated than electroconvulsive therapy. Vascular depression is a subtype of late-life depression, associated with cerebrovascular disease and means a poorer response to antidepressant treatment. We employed rTMS over the left prefrontal cortex in 11 patients with late-onset resistant vascular depression. The primary purpose of this two-week open study was to examine antidepressant efficacy of rTMS in vascular depression. The secondary aim was to evaluate cognitive effects of rTMS in our sample. METHODS: Clinical status, as measured with the Hamilton Depression Rating Scale (HDRS), and cognitive effects, as evaluated by neuropsychological tests, were assessed at baseline and after two weeks of rTMS. Brain measurements to obtain an index of prefrontal atrophy were performed at both the motor cortex and prefrontal cortex. RESULTS: Five out of 11 resistant patients with late-onset vascular depression were responders. They showed a clinically meaningful improvement in HDRS scores, with a decrease of 11, 4 points ($p < 0.01$). Antidepressant response is correlated to the relative degree of prefrontal atrophy ($p = 0.05$). After two weeks, verbal fluency and visuospatial memory improved. No cognitive performance deteriorated except for verbal memory, as the delayed recall decreased significantly in the responders' group.

CONCLUSIONS: Our preliminary observations prompt to perform a subsequent controlled study to examine if rTMS may constitute an alternative to electroconvulsive therapy.

GP & Risperidone & Elderly Patients

EFFICACY AND SAFETY OF LONG-ACTING RISPERIDONE IN ELDERLY PATIENTS WITH SCHIZOPHRENIA AND SCHIZOAFFECTIVE DISORDER.

Authors : Lasser RA, Bossie CA, Zhu Y, Gharabawi G, Eerdeken M, Davidson M. Janssen Medical Affairs, LLC, Titusville, New Jersey, USA.

Source : Int J Geriatr Psychiatry. 2004 Sep;19(9):898-905.

Summary : BACKGROUND: Elderly patients are often an underserved population in terms of optimizing treatment outcomes. Long-acting risperidone, the first long-acting injectable atypical antipsychotic, can improve outcomes through continuous medication delivery. OBJECTIVE: To assess the efficacy and safety of long-acting injectable risperidone in elderly patients with psychotic disorders. METHODS: This is a subanalysis of 57 patients aged ≥ 65 years enrolled in an open-label study of long-acting risperidone that included 725 symptomatically stable patients with schizophrenia or schizoaffective disorder. Patients were assigned to receive 25, 50, or 75 mg of long-acting risperidone every 2 weeks for up to 50 weeks. RESULTS: Fifty-seven elderly patients (mean $\pm$ SE age, 70.9 $\pm$ 0.7 years) were enrolled. Mean Positive and Negative Syndrome Scale (PANSS) total scores improved significantly throughout the study and at endpoint ($p < 0.001$). The PANSS factor scores (positive symptoms, negative symptoms, disorganized thoughts, uncontrolled hostility/excitement, and anxiety/depression) also significantly improved ($p < 0.01$). Clinical improvement ($\geq 20\%$ reduction in PANSS total scores) was achieved by 49% of these stable patients, and 55% improved on the Clinical Global Impressions scale. Severity of movement disorders (Extrapyramidal Symptom Rating Scale scores) was reduced significantly. Adverse events reported in $>10\%$ of patients were insomnia (14%), constipation (12%), and bronchitis (12%). CONCLUSIONS: Long-acting risperidone was associated with significant symptom improvements in stable elderly patients with schizophrenia or schizoaffective disorder. Treatment was well tolerated.

Depression & Sex Hormones

DEPRESSION & SEX HORMONES IN ELDERLY WOMEN.

Authors : Erdinciler D, Bugay G, Ertan T, Eker E.- Geriatric Unit of the Internal Medicine Department, Cerrahpas a Medical School, Istanbul University, Istanbul, Turkey.

Source : Arch Gerontol Geriatr. 2004 Nov-Dec;39(3):239-44.

Summary : We aimed to study the relation between sex hormones and depression among elderly women. The study was carried out on 74 volunteered female subjects above 60 years of age. Each subject was asked to fulfill the geriatric depression scale (GDS) questionnaire and further evaluated for clinical depression by a psychiatrist using the DSM IV diagnostic criteria. For statistical analysis, subjects were later divided in two groups, according to the presence of clinical depression. Cognitive functions were assessed with the standardized mini mental test (SMMT). Disability in the

activities of daily living was assessed with instrumental activities of daily living (IADL) scale. Plasma levels of estrogen, testosterone, progesterone, and dehydroepiandrosterone sulfate (DHEA-S) were measured with chemiluminescent methods, and plasma levels of androstenedione were measured with radioimmunoassay. Among 74 subjects, 34 (39%) had clinical depression. Age, number of years spent in education, SMMT scores, and IADL scores did not differ between the depressive and non-depressive groups. Plasma sex hormone levels were not found to be different between the two groups.

VENLAFAXINE & LATE-LIFE ATYPICAL DEPRESSION

AN OPEN TRIAL OF VENLAFAXINE FOR THE TREATMENT OF LATE-LIFE ATYPICAL DEPRESSION.

Authors : Roose SP, Miyazaki M, Devanand D, Seidman S, Fitzsimmons L, Turret N, Sackeim H. - New York State Psychiatric Institute, and the College of Physicians and Surgeons of Columbia University, New York, USA

Source : Int J Geriatr Psychiatry. 2004 Sep 27;19(10):989

Summary: The atypical subtype in patients with major depressive disorder is characterized by mood reactivity, significant weight gain or increase in appetite, hypersomnia, leaden paralysis and a long-standing pattern of interpersonal rejection sensitivity. Though atypical depression is well documented in younger patients, little attention has been paid to the atypical subtype in samples of late-life depressed patients. This study reports the patient characteristics and treatment results of an eight-week open-label trial of venlafaxine in a sample of older depressed patients with atypical subtype. **METHODS:** Patients received fixed dosing schedule (up to 300 mg/day) of venlafaxine (Effexor XR) for 8 weeks. **RESULTS:** In this sample of 17 patients, the mean age was 65.6 years and 77% were female. Most strikingly, 53% of patients presented with late-onset atypical depression defined as first episode after the age of 50. Fifteen of the 17 patients (88%) completed the eight-week treatment trial. The mean score on the HRSD 24-item decreased from 22.2 +/- 5.1 at baseline to 11.8 +/- 8.9 ($p < 0.001$), and the mean total atypical item score decreased from 6.2 +/- 1.6 to 2.8 +/- 2.0 ($p < 0.001$). Remission was defined as a final HRSD ≤ 10 and a 50% reduction in baseline HRSD score. The intent-to-treat remission rate was 65% and the completer remission rate was 73%. **CONCLUSIONS:** In this sample of late-life patients with atypical depression venlafaxine treatment was reasonably effective and well tolerated. However, the effectiveness of venlafaxine in this study must be considered in the context that this was an open trial of antidepressant medication. Insufficient attention has been given to the atypical subtype in late-life depression. Whether late-onset atypical depression is significantly different from early-onset atypical depression, and whether late-onset patients with atypical depression are significantly different from late-onset patients with other depressive subtypes are questions of compelling interest.

DEMENTIA

DEMENTIA & DIAGNOSIS & TREATMENT

EFFECTIVENESS OF A CLINICAL PATHWAY FOR THE DIAGNOSIS AND TREATMENT OF DEMENTIA AND FOR THE

EDUCATION OF FAMILIES.

Authors : Kazui H, Hashimoto M, Nakano Y, Matsumoto K, Yamamura S, Nagaoka K, Mori E, Endo H, Tokunaga H, Ikejiri Y, Takeda M. Psychiatry and Behavioral Science, Osaka University Graduate School of Medicine, Japan.

Source : Int J Geriatr Psychiatry. 2004 Sep;19(9):892-7.

Summary : AIMS: Clinical pathways (CPs) are rarely used in the treatment of dementia. We established a CP for a series of medical practices (diagnosis, treatment, establishment of a care system, and caregiver education) for patients with dementia hospitalized for a three-week period, and evaluated its usefulness. **METHODS:** The length of hospital stay and hospital costs were compared between 23 consecutive patients with dementia hospitalized and treated using a CP and 20 controls treated by conventional medical practice without using a CP in a special ward for dementia patients. In the CP group, at the time of discharge, primary caregivers, physicians, and nurses were given a questionnaire to obtain their comments about the impression of treatment with the CP. **RESULTS:** The questionnaire survey indicated that the CP deepened the caregiver's understanding of the sequence of medical practices for the inpatient, the disorders of the inpatient, the treatment methods, and the methods for coping with the disorder. The CP was also useful for facilitating inpatient medical practice and promoting the establishment of a care system after discharge. The use of the CP significantly shortened the length of hospital stay and decreased hospital costs during hospitalization but increased the amount of work per day and made the medical staff feel that their freedom to choose medical procedures had been restricted. **CONCLUSIONS:** The CP was useful for execution of inpatient medical practices for patients with dementia.

DEMENTIA & ANTIDEPRESSANTS

ANTIDEPRESSANTS FOR TREATING DEPRESSION IN DEMENTIA

Authors : Bains J, Birks JS, Denning TR

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 17 July 2002. Cochrane reviews are regularly checked and updated if necessary.

Background: The use of antidepressants for patients with dementia accompanied by depressive symptoms is widespread, but their clinical efficacy is uncertain. This uncertainty is due to the difficulties of interpreting the results of clinical trials. Many of the individual trials of antidepressants have been too small to provide precise estimates of the moderate benefits that might realistically be expected. Combining the information from all appropriate trials may provide a better estimate of the likely effects of treatment.

Objectives: This review aims to determine whether antidepressants are clinically effective and acceptable for the treatment of patients diagnosed as having depression and also diagnosed as having dementia.

Search strategy: The CDCIG Specialized Register which includes records from all major medical databases and many trial databases was searched on 21 January 2001. The (long) list of search terms can be found in the main body of the review. Medical information departments of pharmaceutical companies were asked to search their databases for any relevant clinical trials. Where necessary authors of trials were approached with requests for additional information.

Selection criteria: All relevant unconfounded, double-blind, randomized trials comparing any antidepressant drug (as defined by the British National Formulary) with placebo, for patients diagnosed as having dementia and diagnosed as having a depression, according to established criteria.

Data collection and analysis: Data were extracted independently by two of the reviewers and any differences settled by agreement.

Main results: There were six included studies with a total 1077 subjects, of whom 739 met inclusion criteria. Of these, four studies (including a total of 234 subjects) reported results in sufficient detail to enter into meta-analyses. One study's results were limited to adverse results data, therefore the meta-analysis concerning efficacy was limited to three studies (Lyketsos 2000, Petracca 1996, Reifler 1989), with a total of 107 subjects. Of these three studies, two (Petracca 1996, Reifler 1989) investigated the properties of tricyclic antidepressants, drugs not commonly used in this population, and only one study investigated the properties of the more commonly used selective serotonin reuptake inhibitors (Lyketsos 2000). One of these studies (Lyketsos 2000) produced two significant differences in favour of treatment, the Cornell Scale for Depression in Dementia at 6-9 weeks (VMD -7.1, 95% CI -13.05, -1.15) and the psychiatrists' global rating (Peto OR (95% Fixed) 8.17 (1.58, 42.09)). However, the Cornell Scale for Depression in Dementia was not used in any of the other studies and no statistical differences were found with the other measures used in the meta-analysis. The meta-analysis of the number of patients suffering at least one adverse event at 6-9 weeks, using the Peto-odds ratio, showed a significant difference in favour of placebo. There were no other significant results.

Reviewers' conclusions: Available evidence offers weak support to the contention that antidepressants are an effective treatment for patients with depression and dementia. However, only three studies are included in the meta-analysis relating to efficacy, and sample sizes are small. Moreover, only one of the studies included in the analysis of efficacy data investigated the properties of the more commonly used selective serotonin reuptake inhibitors and no studies investigated the properties of newer classes of antidepressants (e.g. selective noradrenergic reuptake inhibitors). This review draws attention to the paucity of research and evidence in this area. It is not that antidepressants are necessarily ineffective but there is not much evidence to support their efficacy either. Given that they may produce serious side-effects clinicians should prescribe with due caution.

DEMENTIA & Music Therapy

MUSIC THERAPY FOR PEOPLE WITH DEMENTIA

Authors : Vink AC, Birks JS, Bruinsma MS, Scholten RJS

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 22 June 2003. Cochrane reviews are regularly checked and updated if necessary.

Background: Dementia is a clinical syndrome with a number of different causes which is characterised by deterioration in cognitive functions. Research is pursuing a variety of promising findings for the treatment of dementia. Pharmacological interventions are available but have limited

ability to treat many of the syndrome's features. Little research has been directed towards non-pharmacological treatments. In this review the evidence for music therapy as a treatment is examined.

Objectives: To assess the effects of music therapy in the treatment of behavioural, social, cognitive and emotional problems of older people with dementia.

Search strategy: The Cochrane Dementia and Cognitive Improvement Group (CDCIG) Specialised Register was searched on 30 June 2003 using the term "music*". This Register contains records from all major health care databases and many ongoing trial databases and is updated regularly. The principal reviewer conducted additional searches to retrieve randomised controlled trials (RCTs) concerning the effect of music therapy on older people with dementia.

Selection criteria: Randomised controlled trials that reported clinically relevant outcomes associated with music therapy in treatment of behavioural, social, cognitive and emotional problems of older people with dementia.

Data collection and analysis: Two reviewers screened retrieved studies independently for methodological quality using a checklist. Data from accepted studies were independently extracted by the reviewers.

Main results: Five studies were included. The methodological quality of the studies was generally poor and the study results could not be validated or pooled for further analyses.

Reviewers' conclusions: The methodological quality and the reporting of the included studies were too poor to draw any useful conclusions.

DEMENTIA & Haloperidol

HALOPERIDOL FOR AGITATION IN DEMENTIA

Authors : Lonergan E, Luxenberg J, Colford 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 27 February 2002. Cochrane reviews are regularly checked and updated if necessary.

Background: Agitation occurs in up to 70% of demented patients. Haloperidol has been used for decades to control agitation in dementia, but its effectiveness remains unclear. Previous meta-analyses examined only English language publications or compared haloperidol with other drugs rather than with placebo. To study the effectiveness of haloperidol a more widely based review was performed.

Objectives: To determine whether evidence supported the use of haloperidol in agitated dementia.

Search strategy: The trials were identified from a last updated search of the Specialized Register of the Cochrane Dementia and Cognitive Improvement Group on 10 July 2003 using the terms halop*, aloperid*, haldol, galoperidol. This register is updated regularly and contains records from all major health care databases and many ongoing trial databases.

Selection criteria: Randomized, placebo-controlled trials, with concealed allocation, where subjects' dementia and agitation were assessed.

Data collection and analysis: 1. Two reviewers extracted data from included trials 2. Data were pooled where possible, and analysed using appropriate statistical methods 3. Odds ratios of average differences were calculated 4. Only 'intention to treat' data were included 5. Analysis included haloperidol treated patients, compared with placebo.

Main results: The five included trials led to the following results: 1. There was no significant improvement in agitation among haloperidol treated patients, compared with controls. 2. Aggression decreased among patients with agitated dementia treated with haloperidol; other aspects of agitation were not affected significantly in treated patients compared with controls. 3. Although two studies showed increased drop-outs due to adverse effects among haloperidol patients, there was no significant difference in drop-out rates, comparing all haloperidol treated patients with controls. 4. The data were insufficient to examine response to treatment in relation to length of treatment, degree of dementia, age or sex of patients, and cause of dementia.

Reviewers' conclusions: 1. Evidence suggests that haloperidol was useful in reducing aggression, but was associated with adverse effects; there was no evidence to support the routine use of this drug for other manifestations of agitation in dementia. 2. Similar drop-out rates among haloperidol and placebo treated patients suggested that poorly controlled symptoms, or other factors, may be important in causing treatment discontinuation. 3. Variations in degree of dementia, dosage and length of haloperidol treatment, and in ways of assessing response to treatment suggested caution in the interpretation of reported effects of haloperidol in the management of agitation in dementia. 4. The present study confirmed that haloperidol should not be used routinely to treat patients with agitated dementia. Treatment of agitated dementia with haloperidol should be individualized and patients should be monitored for adverse effects of therapy.

WOMEN DEMENTIA, HORMONE & COGNITIVE FUNCTION —

■ HORMONE REPLACEMENT THERAPY TO MAINTAIN COGNITIVE FUNCTION IN WOMEN WITH DEMENTIA

Authors : Hogervorst E, Yaffe K, Richards M, Huppert F

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 27 May 2002. Cochrane reviews are regularly checked and updated if necessary.

Background: As estrogens have been shown to have several potentially beneficial effects on the central nervous system, it is biologically plausible that maintaining high levels of estrogens in postmenopausal women by means of estrogen replacement therapy (ERT) could be protective against cognitive decline and the development of Alzheimer's disease (AD) or other dementia syndromes.

Objectives: To investigate the effects of ERT (estrogens only) or HRT (estrogens combined with a progestagen) compared with placebo in randomized controlled trials (RCTs) on cognitive function of postmenopausal women with dementia.

Search strategy: The CDCIG Specialized Register, which contains up-to-date records from many medical databases was searched using the terms ORT, PORT, ERT, HRT, estrogen*, oestrogen*, progesteron* and Alzheimer* on 16th of May 2002. In addition, MEDLINE (1966-2002/01); EMBASE (1985-2002/01); and PsycINFO (1967-2002/01) were searched. **Selection criteria:** All double-blind randomized controlled trials (RCTs) into the effect of ERT or HRT for cognitive function with a treatment period of at least two weeks in postmenopausal women with AD or other types of dementia. **Data collection and analysis:** Abstracts of the references retrieved by the searches were read by two reviewers (EH and KY) independently in order to discard those that were clearly

not eligible for inclusion. The two reviewers studied the full text of the remaining references and independently selected studies for inclusion. Any disparity in the ensuing lists was resolved by discussion with all reviewers in order to arrive at the final list of included studies. The selection criteria ensured that the blinding and randomization of the included studies was adequate. The two reviewers also assessed the quality of other aspects of the included trials. One reviewer (EH) extracted the data from the studies, but was aided and checked by JB from Cochrane.

Main results: A total of five trials including 210 women with AD were analysed. Meta-analyses showed that there was a limited positive effect from low dosage of conjugated equine estrogens (CEE, 0.625 mg once a day) but not from higher dosage (1.25 mg of CEE once a day) on the Mini-Mental Status Examination after 2 months (WMD=1.28, 95% C.I.=0.26 to 2.30, $z=2.45$, $p<0.01$) and the effect disappeared after 3, 6 and 12 months of treatment. This effect was small and not clinically relevant as there was only a difference of 1 point on average in comparison with the placebo users (the scale range is 0-30). There were also short-term effects of 1.25 mg of CEE on tests of concentration and executive function, namely the Trail Making Test-B (WMD=-40.90, 95% C.I.=-79.29 to -2.51, $z=2.09$, $p<0.05$) and Digit Span backward (WMD=0.67, 95% C.I.=-0.01 to 1.34, $z=1.94$, $p<0.05$). With regard to memory, only cued delayed recall of a word list was positively affected by 2 months of transdermal diestradiol (E2) (WMD=6.50, 95% C.I.=4.04 to 8.96, $z=5.19$, $p<0.0001$). No HRT effects were seen on other word lists, Paragraph Recall or Paired Associate Learning. In addition, no effects were seen on visual memory, language functions, most speeded tests, clinical rating scales or depression. Controls had better performance on the delayed recall of the Paragraph Test (overall WMD=-0.45, 95% C.I.=-0.79 to -0.11, $z=2.60$, $p<0.01$) after 1 month and on Finger Tapping after 12 months (WMD=-3.90, 95% C.I.=-7.85 to 0.05, $z=1.93$, $p<0.05$). Clinicians also gave controls a better score on a dementia rating scale (CDR, overall WMD=0.35, 95% C.I.=0.01 to 0.69, $z=1.99$, $p<0.05$). Positive findings in favour of treatment or placebo could have been random effects caused by multiple analyses. After correction for multiple testing, only the short-term positive treatment effect of E2 on memory remained.

Reviewers' conclusions: Currently, HRT or ERT for cognitive improvement or maintenance is not indicated for women with AD. As we did not have data on women with other types of dementia (e.g. vascular dementia) this remains to be investigated. As most studies only used CEE and our earlier review in healthy women found effects only after a bolus injection of E2, it remains possible that different preparations or types of ERT or HRT could have a different effects. Several questions are raised in this review, including whether factors such as age, dementia onset (early AD), or the use of a particular preparation for a longer duration of treatment could have different effects. Perhaps the most important question is whether ERT or HRT can delay the time of onset of dementia. For answers to these questions, we have to await the results of the large RCTs currently in progress in the UK, USA, and Canada.

DEMENTIA & DONEPEZIL —

■ DONEPEZIL FOR DEMENTIA DUE TO ALZHEIMER'S DISEASE

Authors : Birks JS, Harvey R

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 27 May 2003. Cochrane reviews are regularly checked and updated if necessary.

Background: Alzheimer's disease is the most common cause of dementia in older people. One of the aims of therapy is to inhibit the breakdown of a chemical neurotransmitter, acetylcholine, by blocking the relevant enzyme. This can be done by a group of chemicals known as cholinesterase inhibitors. However, some (like tacrine) are associated with adverse effects such as hepatotoxicity, but donepezil (E2020, Aricept) is safer.

Objectives: The objective of this review is to assess whether donepezil improves the well-being of patients with dementia due to Alzheimer's disease.

Search strategy: The Cochrane Dementia and Cognitive Improvement Group's Specialized Register was searched using the terms 'donepezil', 'E2020' and 'Aricept' on 9 October 2002. This Register contains up-to-date records of all major health care databases and many ongoing trial databases. Members of the Donepezil Study Group and Eisai Inc were contacted.

Selection criteria: All unconfounded, double-blind, randomized controlled trials in which treatment with donepezil was compared with placebo for patients with mild, moderate or severe dementia due to Alzheimer's disease.

Data collection and analysis: Data were extracted by one reviewer (JSB), pooled where appropriate and possible, and the weighted mean differences or Peto odds ratios (95%CI) estimated.

Main results: Sixteen trials are included, involving 4365 participants. The trials were of 12, 24 or 52 weeks duration in selected patients. Available outcome data cover domains including cognitive function and global clinical state, but data on several important dimensions of outcome are unavailable. For cognition there is a statistically significant improvement for both 5 and 10 mg/day of donepezil at 24 weeks compared with placebo (-2.02 points on the ADAS-Cog scale WMD, 95%CI -2.77 to -1.26, $p < 0.00001$; -2.92 points on the ADAS-Cog scale WMD 95% CI -3.74 to -2.10, $p < 0.00001$) and for 10 mg/day donepezil compared with placebo at 52 weeks (1.84MMSE points, 95% CI, 0.53 to 3.15, $p = 0.006$). The results show some improvement in global clinical state (assessed by an independent clinician) in people treated with 5 and 10 mg/day of donepezil compared with placebo at 12 and 24 weeks. Benefits of treatment were also seen on measures of activities of daily living and behaviour. There were significantly more withdrawals before the end of treatment from the 10 mg/day (but not the 5 mg/day) donepezil group compared with placebo which may have resulted in some overestimation of beneficial changes at 10 mg/day. A variety of adverse effects were recorded, with more incidents of nausea, vomiting, diarrhoea and anorexia in the 10 mg/day group compared with placebo and the 5 mg/day group, but very few patients left a trial as a direct result of the intervention.

Reviewers' conclusions: People with mild, moderate or severe dementia due to Alzheimer's disease treated for periods of 12, 24 or 52 weeks with donepezil experienced benefits in cognitive function, activities of daily living and behaviour. Study clinicians rated global clinical state more positively in treated patients, and measured less decline in measures of global disease severity. Although no significant changes were measured on a patient-rated quality of life scales, the instrument used was crude and possibly unsuited to the task. The additional data now available confirm the findings of

the previous issue of this review and extend the evidence for the effectiveness of treatment to at least 52 weeks and to those with severe dementia. More evidence is still needed for the economic efficacy of donepezil, but clinical efficacy is confirmed.

Eating Disorders

EATING DISORDERS & PERSONALITY DISORDERS

■ EATING DISORDERS AND PERSONALITY DISORDERS: AWARENESS OF POSSIBLE INTERACTIONS & THEIR THERAPEUTIC IMPLICATIONS.

Authors : Kristine Godt

Summary: Personality disorders are defined by a characteristic and enduring pattern of behaviour and inner experience that deviates distinctly from culturally defined norms. Examples include social insecurity or even distrust leading to withdrawal, or patterns of impulsiveness, affective instability or, possibly, self-harm. Personality disorders are distinct from other psychiatric disorders primarily by their enduring character. +

The prevalence of personality disorders among patients with eating disorders depends on population characteristics. In a specialised outpatient clinic that serves a defined catchment area, nearly one third of patients will fulfill the diagnostic criteria for a personality disorder. Hospitalized patients or patients in more specialised units will surely show a co Several modes of interaction between eating disorders and personality disorders could be hypothesized. The treatment of patients with eating disorder should take into account the subgroup with comorbid personality pathology and evaluation and treatment should be planned accordingly, though this requires time and expertise. The issue should also be acknowledged in primary health care and attention be paid to symptoms of eating disorder also in cases in which symptoms of a personality disorder are more pronounced.

Eating Disorders

■ SOMATIC INVESTIGATION AND TREATMENT OF EATING DISORDERS.

Authors : Stein Frostad

Summary: Eating disorders are associated with several medical complications. Growth retardation and osteoporosis can cause permanent sequelae if treatment is delayed. Severe eating disorders are associated with significant mortality. Cardiac arrhythmias are the most common somatic cause of death. Hypokalaemia is a common complication and is associated with increased risk of cardiac arrhythmias. Occasionally, overzealous refeeding may induce a potentially life-threatening condition, the refeeding syndrome. In any patient with severe eating disorder, a physician should perform diagnostic evaluation including assessment of possible somatic complications. This is necessary in order to determine where and how the patient should be treated. Most of the somatic complications of eating disorders are partly or completely reversible if the patient receives adequate treatment in time.

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مكافحة عكاشة الدول السائرة في طريق النمو
جائزة جينيف لحقوق الانسان في الطب النفسي

أ.د. أحمد عكاشة - رئيس الجمعية العالمية للطب النفسي

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The Geneva Prize, awarded by the Geneva Foundation for

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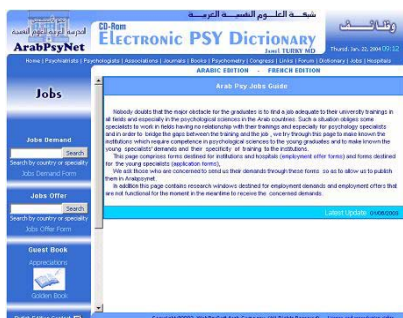
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www.arabpsynet.com/Archives/OP/OP.Naboulsi.PsyArSchool.htm

اللامح المميزة للمدرسة العربية للعلوم النفسية
أ.د. علي زيعور - لبنان

www.arabpsynet.com/Archives/VP/VP.Zayour.PsyArSchool.htm

الطب النفسي عبر الحضاري
أ.د. محمد فاروق السنديوني

www.arabpsynet.com/Archives/OP/OP.SendiyouniPsy.InterCult..htm

الخصائص المعرفية للمحاولات السيكولوجية العربية
د. الغالي أحروشاو / علم النفس - ظهر المهراز / المغرب

www.arabpsynet.com/Archives/OP/OP.Ahroucha.PsyLangage.htm

منتدى شبكة العلوم النفسية العربية - الإصدار العربي



www.arabpsynet.com/Homepage/Psy-Forum.Ar.asp

شارك برأيك: www.arabpsynet.com/propositions/PropForm.htm

انطباعات : أساتذة علم النفس

أ.د. عبد الستار إبراهيم / مصر - السعودية

أ.د. سامر جميل رضوان / سوريا - عمان

أ.د. فيصل خير الزراد / سوريا - الإمارات

المركز العربي للدراسات النفسية / طرابلس - لبنان

أ.د. يوسف طبّاخ / سوريا - السويد

أ.د. نزار عيون السود / عمان - سوريا

أ.د. بشير معمرية / باتنة - الجزائر

د. أحمد المعمري / الحديدة - اليمن

The

" Contemporary who's who

أ.د. سوسن الجليبي / بغداد - العراق

APPRECIATIONS : PROFESSORS of Psychology & Psychologists

PR. CHARLES HARB : BEIRUT - LEBANON

I was pleasantly surprised by the internet portal you forwarded, and am immensely grateful for your efforts in that regard. I have taken the liberty of forwarding the url to several Arab academics in the Arab world. I am sure that the future of the portal is assured. Warmly from. (Social and Behavioral Sciences Department / American University of Beirut)

MAYSAA BURAIK , PH.D : VIRGINIA, USA

This is a wonderful start for Arab mental health professional. I have waited very long for something like this. I use the available dictionary to translate some of the assessments i administer and it has proved to be very useful. I look forward to being a contributing member. My suggestions concern adding the admittance of counselors into your web page. It is my belief that their contributions would be greatly appreciated and useful. After all, isn't this web page intended to improve the science of psychology? I believe there are more Arab counselors out there that may have more experience with the Arab population (whether in the USA or the Middle East). As an Arab psychology student i have received most of my input and help regarding working with Arab clients from Arabic counselors (community, school, family..etc). In addition, I have researched the available literature in the USA regarding working with Arab and Muslim clients and most of the contributions were from counselors.

LYNN WILCOX, PH.D : CALIFORNIA - USA

The website is very impressive! Thank you very much. (Counseling Psychology University of Missouri-USA / Lifetime Member, American Psychological Association. / Asst. Professor, Georgia State University, Atlanta, 1968-69 / Professor, California State University, Sacramento - USA)

DR. REDA ABOUSERE : CAIRO - EGYPT

Many thanks and I am very glad to reach this level of professional website. I am always keen to surve the site and happy to receive your email about it. Please Keep working on it and I have already mentioned about it in the latest conference in Dubai. / With my best wishes.

SAMAR ZEBIAN, PH.D. : BEIRUT - LEBANON

I would like to congratulate you, your colleagues and students on Arabpsynet. Although we recently learned about the site here at AUB in the psychology program (in Beirut) we have already made good use of it.

PR. FAROUK MAJZOUB : BEIRUT - LEBANON

You have achieved, up till now, in the realm of Psychological Sciences in the Arab World. Congratulation

DR. MANDY BRAUER : CAIRO - EGYPT

I have just glanced at this wonderful new site and I look forward to getting to know it and you more. The site is much needed and I am glad it is in 3 languages. How about a section so that readers can search for specific subjects? I, for example, am most interested in children and adolescents since I am a child psychologist. It would be convenient to be able to just write the word children and have further areas about children at my fingertips. Unfortunately, I am so computer-timid that you may well have this feature within the site and I have not yet seen it. Do you also have specific papers on the mental health of the Palestinians currently ? This is of great concern to me. Thanks.

NEHAD AHMED PH.D : OHIO - USA

I am happy for having me as a member of the Arabpsynet. , I do have international experiences in Egypt, Germany, Puerto Rico, and USA. At this course of my life, I am very interested to learn more about the psychological needs for our Arabic and Islamic communities. I appreciate allowing me to be part of the growing Arabic Psychological Society.

DR. N. RAZIK : QUÉBEC - CANADA

Excellente initiative qu'est ce forum sur la psy version monde arabe. Sans être en marge des nations, le monde arabe est un champ fertile à l'exploration (pas seulement le pétrole) intellectuelle. Entre un passé glorieux et un présent douloureux, c'est aux intellectuels et aux professionnels de participer à la renaissance. Je serai curieux de connaître l'impact de cette stigmatisation en cours sur les enfants arabes et musulmans. / Merci et Bravo. (Criminologue / Protection et réadaptation juvénile)

DR. MUSAED AL-NAJJAR : KUWAIT CITY

I just would like to say congratulation on the successful of making the Homepage of arabpsynet.com, it is well designed, got a lot of information. / Congratulation one more time. / Best Regards.

MRS. MAYADA AKRAWI: IRAQ - SWITZERLAND

Thank you very much for your site . It is thanks to the information provided on it that I was able to attend the first regional congress on middle east and north africa psychology in Dubai - It was most interesting and it is thanks to your efforts that this information was dissiminated / With regards.

MR. NAOUFAL BEN ABDELJALIL: MOROCCO

I hope the best for your website to help all the poeple looking for knowledge all over the world especially in the arab countries

المعجم الإلكتروني للعلوم النفسية العربية

مصطلحات عربية : عربي - إنكليزي - فرنسي

بريد إلكتروني: turky.jamel@gnet.tn

الدكتور جمال التركي الطب النفسي - تونس

réaction de soumission	submission reaction
réaction de relaxation	relaxation reaction
réaction d'indépendance	independence reaction
réaction de réveil	awakening reaction
réaction des prisonniers	prisoners reaction
réaction de persécution	persecution reaction
réaction d'approche	approach response
réponse proximale	proximal response
réaction dépressive	depressive reaction
réaction dépressive	neurotic depressive
névrotique	reaction
réaction psychalgique	psychalgic reaction
réaction d'abstinence	abstinence reaction
réponse alpha	alpha response
réaction d'émergence	emergency reaction
réaction de sursaut	startle reaction
réaction d'involution	involution reaction
réaction de retrait	retreat reaction
réaction de stress	stress reaction
réaction de séparation	separation reaction
réaction de la ménopause	menopause reaction
réaction d'émergence	emergency reaction
réaction de pleurer	crying reaction
réaction d'adaptation	adaptation reaction
réaction d'évitement	avoidance reaction
réaction de conversion	conversion reaction
réaction de peur	fear reaction
réaction associative	associative reaction
réaction de précipitation	precipitation reaction
réponse anatomique	anatomy response
réaction de poursuite	pursuit reaction
réponse de diffusion	diffusion response
réaction de	rapprochement reaction
rapprochement	
réaction d'imitation	mimetic reaction
réaction d'adaptation	adaptation reaction
réaction d'adaptation	adjustment reaction
réponse de stigmatisation	stigmatization response
réaction de confirmation	confirming reaction
réaction du système	nervous system reaction
nervous	
réaction de privation	affective privation
affective	reaction

إستجابة - إستحواذ
إستخبار - إستدعاء
إستدلال - إستدماج
إسترخاء

reaction, reponse	reaction, response	()
réaction sociale	social reaction	
réaction d'évitement	avoidance reaction	
réaction tardive	delayed reaction	
réaction uniphasique	uniphasic reaction	
réponse de frustration	frustration response	
réaction kinesthétique,	kinesthetic reaction,	
réaction stotokinétique	stotokinetic reaction	
réaction de choix	choice reaction	
réponse instrumentale	instrumental response	
réaction adrénérergique	adrenergic reaction	
réponse anticipée	anticipatory response	
réaction relaxante	relaxant reaction	
réaction d'éveil	awakening reaction	
réponse conditionnée,	conditioned response,	
réaction conditionnée	conditioned reaction	()
réponse originale	original response	
réaction de persécution	persecution reaction	
réponse arbitraire	arbitrary response	
réponse préparatoire	preparatory response	
réaction dépressive	depressive reaction	
réponse alpha	alpha response	
réponse extravertie	extraverted response	
réponse élective	elective response	
réponse sélective	selective response	
réponse d'involution	involution response	
réaction dissociée	dissociated reaction	
réaction dissociée	dissociated reaction	
réponse d'introversion	introversion response	
réponse réflexive	reflection response	
réaction dissociative	dissociative reaction	
réaction émotionnelle	emotional reaction	
réaction d'involution	involutional reaction	
réaction primaire	primary reaction	
réaction d'involution	involution reaction	
réaction positive	positive reaction	
réaction de frustration	frustration reaction	
réaction du choix	choice reaction	

réaction au stimulant	stimulant reaction
réaction du prévu	provided reaction
réaction hypothétique	hypothetical reaction
réaction de confrontation	confrontation reaction
réponse d'aversion	aversion response
réaction incompatible	incompatible response
réaction d'attaque	attack reaction
réaction d'hystérie	hysteria reaction
réaction de fuite	escape reaction
réaction de panique	panic reaction
réaction maniaque	maniac reaction
réaction d'identité	identity reaction
réponse de position	position response
réaction asthénique	asthenic reaction
réaction d'éveil	awakening reaction
réaction substituée	substitute reaction
réaction visuelle	visual reaction
réponse structurale	texture response
réaction à l'environnement	environment reaction
réaction d'affirmation	affirmation reaction
réaction d'évitement	avoidance reaction
réaction de conversion	conversion reaction
réponse confabulée	confabulated response
réaction catatonique	catatonic reaction
réaction phobogène	phobogenous reaction
réaction de précipitation	precipitation reaction
réponse anatomique	anatomy response
réaction compensatoire	compensatory reaction
réaction différentielle	differential reaction
réaction dissociative	dissociative reaction
réaction dissociée	dissociated reaction
réaction d'adaptation	adjustment reaction
réaction spontanée	spontaneous reaction
réaction génitale	genital reaction
réaction genito-urinaire	genito-urinary reaction
réaction respiratoire	respiratory reaction
réaction dégénérative	degeneration reaction
réaction adaptative	adjustment reaction
réaction fantasmatique	fantasmatic reaction
réaction hypocondriaque	hypochondriac reaction
réaction secondaire	secondary reaction
réaction de délinquance	delinquency reaction
réaction nouvelle	new-reaction
réaction corporelle	corporeal reaction
réaction de somatisation	somatization reaction
réaction dermique	galvanic reaction
galvanique	
réponse galvanique	galvanic response
réaction psycho-	psycho-galvanic
galvanique	reaction

réaction de tristesse	sadness reaction
réaction d'angoisse	anguish reaction
réaction anesthésique	anaesthetic reaction
réaction de soumission	submission reaction
réaction de peur	fright reaction
réaction cachectique	cachectic reaction
réaction d'angor	angina reaction
réaction d'effroi	fright reaction
réaction d'envie	envy reaction
réaction phobique	phobic reaction
réaction sadique	sadistic reaction
réaction au cancer	cancer reaction
réaction de stress	stress reaction
réaction du mutisme	muteness reaction
réaction au stress	stress reaction
réaction du membre fantôme	phantom limb reaction
réaction des urgences	emergency reaction
réaction d'hostilité	hostility reaction
réaction nauséuse	nausea reaction
réaction de colère	anger reaction
réponse du vide	vacuum response
réaction schizophrénique	schizophrenic reaction
réaction de perte	loss reaction
réaction de combat	combat reaction
réaction d'anxiété	anxiety reaction
réaction de catastrophe	reaction
réaction inhibée	inhibited reaction
réaction de latence	latency reaction
réaction de non-identité	non identity reaction
réaction de plaisir	pleasure reaction
haptotaxie	haptotaxis, thigmotaxis
réaction masochiste	masochistic reaction
réaction de poursuite	pursuit reaction
réponse à un stimulus	stimulus response
réaction des mourants	dying reaction
réaction du contenu	content reaction
réaction cérébrale organique	organic cerebral reaction
réaction adolescente	adolescent reaction
réponse d'espace blanc	white-space response
réaction de suceur	sucker reaction
réaction de catastrophe	catastrophe reaction
réaction du thérapeute	therapist
réaction du combat	combat reaction
réaction de résistance	resistance reaction
réaction immunitaire	immune reaction
réaction au stimulus	stimulus reaction

réaction explicite	explicit reaction
réaction aggressive	aggressive reaction
réaction transitoire	transitory reaction
réaction névrotique	neurotic reaction
réaction nerveuse	nervous reaction
réaction musculaire	muscular reaction
réaction musculo-squelettique	musculo- skeleton reaction
obsession, compulsion	obsession, compulsion
obsession morale	moral obsession
obsession impulsive	impulsive obsession
compulsion de la pensée	thinking compulsion
obsession de contamination	contamination obsession
obsession du compte	counting obsession
obsession somatique	somatic obsession
obsession sexuelle	sexual obsession
phobie obsédante	haunting phobia
possession démoniaque	demoniacal possession
obsession masquée	masked obsession
obsession idéologique	ideological obsession
obsession masquée	masked obsession
obsession psychasthénique	psychasthenic obsession
anankastie-anancastie, compulsion obsessionnelle	anankastia-anancastia, obsessional compulsion
questionnaire, test, inventaire	questionary, testing, inventory
questionnaire des intérêts	interest inventory, interest test
questionnaire de classification	questionary sort
questionnaire de personnalité	personality inventory
auto-inventaire	self inventory
questionnaire névrotique	neurotic questionnaire
questionnaire sociométrique	sociometric questionnaire
questionnaire psychonévrotique	psychonevrotique questionnaire
évocation, invitation, convocation, appel	evocation, invitation, () recollection, getting
appel à l'attention	attention getting
évocation des expériences	experiences evocation
association des pensées	thought association
évocation mnésique	mnesic evocation
évocation des mémoires	memory evocation
évocation des mots	words evocation
évocation du refoulé	repressed calling
appel sexuel	sexual appeal

réaction homosexuelle	homosexual reaction
réaction aiguë	acute reaction
réponse libre	free response
réacteur critique	critical reaction
réaction kinétique	kinetic reaction
réaction sensitive	sensitive reaction
réponse cinesthétique	kinesthetic response
réaction viscérale	visceral reaction
réponse animale	animal response
réaction biologique	biological reaction
réaction spéciale	special reaction
réaction fausse	false reaction
réaction phobique	phobic reaction
réaction de peur	fright reaction
réaction de frayeur	fright reaction
réaction de défense	defence reaction
réaction cyclique	cyclic reaction
réponse autonome	autonomic response
réaction d'effroi	panic reaction
réaction psychotique	psychotic reaction
réaction mentale	mental reaction
réaction stuporeuse	stuporous reaction
réaction phobique	phobic reaction
réaction phobogène	phobogenous reaction
réaction paranoïde	paranoid reaction
réaction antécédente	antecedent reaction
réaction sadique	sadistic reaction
réaction akinétique	still reaction
réaction passive	passive reaction
réaction de colère	anger reaction
réponse statique	static response
réponse stato-kinétique	statokinetic response
réponse négative	negative response
réaction comportementale	behaviour reaction
réaction auditive	auditory reaction
réponse populaire	popular response
réaction perverse	perverse reaction
pathergie	pathergia
réaction orgasmique	orgasmic reaction
réaction zombie-like	zombie-like reaction
réaction conditionnelle	conditioning reaction
réponse orale	oral response
réaction épileptique	epileptic reaction
réponse autophonique	autophonic response
réaction dyssociale	dyssocial reaction
réponse implicite	implicit response
réaction urgente	emergency reaction
réaction distale	distal response
réaction infantile	infantile reaction

relaxation des muscles	muscle relaxation
relaxation corporelle	bodily relaxation
relaxation analytique	analytical relaxation
relaxation progressive	progressive relaxation
relaxation progressive	progressive relaxation
hypnose de détente	relaxation hypnosis
relaxation corporelle	corporeal relaxation
relaxation assise	seated relaxation
relaxation en groupe	relaxation in group
relaxation d'autodéfense	relaxing of ego defence
relaxation dynamique	dynamic relaxation
training autogène, auto-relaxation	autogenous training, self-relaxation
relaxation mentale	mental relaxation
relaxation	behavioural relaxation
comportementale	
relaxation générale	general relaxation
relaxation de Schultz	Schultz relaxation
relaxation générale	general relaxation
relaxation classique	classic relaxation
relaxation musculaire	muscular relaxation
relaxation mentale	mental relaxation
relaxation thérapeutique	therapeutic relaxation
relaxation	psychotherapeutic relaxation
psychothérapique	
relaxation différentielle	differential relaxation
relaxation individuelle	individual relaxation
relaxation totale	total relaxation
relaxation progressive	progressive relaxation
relaxation ascendante	ascending relaxation
relaxation allongée	lengthened relaxation
relaxation généralisée	generalized relaxation
relaxation brève	brief relaxation
relaxation harmonieuse	harmonious relaxation
relaxation incomplète	incomplete relaxation
relaxation psychique	psychic relaxation
relaxation spécifique	specific relaxation

évocation sensitive	sensitive evocation
évocation psychique	psychic evocation
inférence, raisonnement	inference, reasoning
inférence fiduciaire	fiduciary inference
raisonnement syncretique	syncretic reasoning
inférence statistique	statistical inference
raisonnement par induction	inductive reasoning
raisonnement déductif	deductive reasoning
raisonnement	hypothetico-deductive -
hypothético-déductif	reasoning
raisonnement par	recurrence reasoning
récurrence	
raisonnement par défaut	default reasoning
symbolie	symbolia ()
raisonnement circulaire	circular reasoning
raisonnement	mathematical reasoning
mathématique	
raisonnement valide	valid reasoning
raisonnement invalide	invalid reasoning
raisonnement intellectuel	intellectual reasoning
déduction immédiate	immediate deduction
ratiocination	ratiocination
raisonnement affectif	emotive reasoning
incorporation, introjection	incorporation, introjection) (
incorporation du sein	breast incorporation
incorporation de l'image	imago incorporation
incorporation du pénis	penis incorporation
identification à la mère	mother identification
incorporation de l'objet d'amour	love object incorporation
incorporation délirante	delirious incorporation
relaxation, relâchement	relaxation
chalastodermie	chalastodermia
relaxation physique	physical relaxation
relaxation du couple	couple relaxation

المعجم الإلكتروني المبرمج للعلوم النفسية

ePsydict EF – English - French Edition (CD)
English French - English French



تنزيل النسخة التقييمية من الإصدار الإنكليزي الفرنسي
www.arabpsynet.com/HomePage/ePsyEFs.exe

ePsydict C – COMPLETE Edition (CD)

Arabic English French - French English Arabic - English Arabic French



تنزيل النسخة التقييمية من الإصدار الكامل
www.arabpsynet.com/HomePage/ePsyCs.exe

E.DICTIONARY of Psychological Sciences

English PSY TERMINOLOGIES (English - French - Arabic)

JAMEL TURKY - TUNISIA

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A

Acute - Affect
Affective - Affectivity
Aggression - Aggressive
Aggressivity

Acute	aigu
Acute agitation	agitation aiguë
Acute akathisia	akathisie aiguë
Acute alcoholic delirium	délire alcoolique aigu
Acute alcoholic encephalitis	encéphalite alcoolique aiguë
Acute alcoholic hallucinosis	hallucinoïse alcoolique aiguë
Acute alcoholic intoxication	intoxication alcoolique aiguë
Acute alcoholic myopathy	myopathie alcoolique aiguë
Acute alcoholism	alcoolisme aigu
Acute anguish	angoisse aiguë
Acute anxiety	anxiété aiguë
Acute anxiety attack	crise d'anxiété aiguë
Acute anxiety reaction	réaction d'anxiété aiguë
Acute ataxia	ataxie aiguë
Acute azotemic psychosis	psychose azotémique aiguë
Acute confrontation	confrontation aiguë
Acute confusion	confusion aiguë
Acute Confuso-catatonic syndrome	syndrome confuso-catatonique aigu
Acute crisis depressive	crise dépressive aiguë
Acute degenerative psychosis	psychose dégénérative aiguë
Acute delirious disorder	trouble délirant aigu
Acute delirious episode	épisode délirant aigu
Acute delirious psychosis	psychose délirante aiguë
Acute delirious puff	bouffée délirante aiguë
Acute delirious state	état délirant aigu
Acute delirium	délire aigu
Acute delirium	délire aigu du

(post- partum-)	post – partum
Acute delusional psychosis	psychose délirante aiguë
Acute dementia	démence aiguë
Acute depression	dépression aiguë
Acute drunkenness	ivresse aiguë
Acute dystonia	dystonie aiguë
Acute hallucinatory mania	manie hallucinatoire aiguë
Acute hallucinosis	hallucinoïse aiguë
Acute imaginative psychosis	psychose imaginative aiguë
Acute Korsakoff's psychosis	psychose aiguë de Korsakoff
Acute mania	manie aiguë
Acute paranoia	paranoïa aiguë
Acute paranoid schizophrenia	schizophrénie paranoïde aiguë
Acute pharmacopsychosis	pharmacopsychose aiguë
Acute poisoning	intoxication aiguë
Acute porphyria	porphyrie aiguë
Acute psychosis	psychose aiguë
Acute psychotic depression	dépression psychotique aiguë
Acute psychotic reaction	réaction psychotique aiguë
Acute reflex	réflexe aigu
Acute schizophrenia	schizophrénie aiguë
Acute schizophrenic reaction	réaction schizophrénique aiguë
Acute situation	situation aiguë
Acute situational reaction	réaction situationnelle aiguë
Acute stress	stress aigu
Acute tic	tic aigu
Acute toxic psychosis	psychose toxique aiguë
Acute violence	violence aiguë
Affect	affect, affectivité
Affect (blunted-)	affect émoussé
Affect (depressive-)	affect dépressif
Affect (detached-)	affection détachée

Affect (epilepsy-)	affect épileptique
Affect (incongruity-)	affect incongruité
Affect (pathology-)	affect pathologique
Affect (pulsional-)	affect pulsionnel
Affect block	blocage affectif
Affect disharmony	dissociation affective
Affect energy	énergie d'affect
Affect fantasy	fantaisie d'affect
Affect fixation	fixation d'affect
Affect flooding	inondation des affects
Affect hunger	faim affective
Affect incongruity	incongruité affective
Affect inversion	inversion d'affect
Affect liberation	libération des affects
Affect quantum	quantum d'affect
Affect report	rapport affectif
Affect retention	rétenion des affects
Affect reversal	retournement de l'affect
Affect structure	structure affective
Affect transposition	transposition d'affect

Affective	affectif
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Affective accident	accident affectif
Affective action	action affective
Affective activity	activité affective
Affective alternation	alternation affective
Affective ambivalence	ambivalence affective
Affective amnesia	amnésie affective
Affective anaesthesia	anesthésie affective
Affective apathy	apathie affective
Affective assimilation	assimilation affective
Affective atony	atonie affective
Affective avidity	avidité affective
Affective body	corps affectif
Affective cathexis	cathexie affective
Affective character	caractère affectif
Affective climate	climat affectif
Affective coldness	froideur affective
Affective communication	communication affective
Affective confinement	confinement affectif
Affective congruency	congruence affective
Affective consolidation	consolidation affective
Affective contact	contact affectif
Affective deficiency	carence affective
Affective dementia	démence affective
Affective dependence	dépendance affective
Affective discharge	décharge affective

Affective disharmony	dysharmonie affective
Affective disinterest	désintérêt affectif
Affective disorder	trouble affectif ()
Affective dissociation	dissociation affective
Affective dysthymia	dysthymie affective
Affective environment	milieu affectif
Affective epilepsy	épilepsie affective
Affective eudemonia	eudemonie affective
Affective exaltation	exaltation affective
Affective excitation	excitation affective ()
Affective experience	expérience affective
Affective expression	expression affective
Affective extortion	chantage affectif
Affective failure	défaillance affective ()
Affective feeble-mindedness	débile affectif
Affective feeling	sentiment affectif
Affective fixation	fixation affective
Affective flattening	indifférence affective

Affective folding up	repliement affectif
Affective force	force affective
Affective hallucination	hallucination affective
Affective harmonisation	harmonisation affective
Affective heat	chaleur affective
Affective heterophobia	hétérophobie affective
Affective hunger	faim affective
Affective illusion	illusion affective
Affective impregnation	imprégnation affective
Affective inappropriateness	ambivalence affective
Affective incontinence	incontinence affective
Affective infantilism	infantilisme affectif
Affective inhibition	inhibition affective
Affective insanity	folie affective
Affective interaction	interaction affective
Affective intuition	intuition affective
Affective investment	Investissement affectif
Affective isolation	isolation affective
Affective knowledge	connaissance affective
Affective lability	labilité affective
Affective lack	manque affectif
Affective learning	étude affective
Affective load	charge affective
Affective logic	logique affective
Affective maturation	maturation affective

Affective maturity	maturité affective
Affective mechanism	mécanisme affectif
Affective melancholia	mélancolie affective
Affective memory	mémoire affective
Affective mimetism	mimétisme affectif
Affective monomania	monomanie affective
Affective need	besoin affectif
Affective objective	objectif affectif
Affective oligomania	oligomanie affective
Affective outcome	résulte affectif
Affective paradox	paradoxe affectif
Affective paraphrenia	paraphrénie affective
Affective penetration	pénétration affective
Affective pledging	engagement affectif
Affective position	position affective
Affective poverty	pauvreté affective
Affective privation	privation affective
Affective privation effect	effet de privation affective
Affective problem	problématique affectif
Affective psychology	psychologie affective
Affective psychosis	psychose affective
Affective quest	quête affective

Affective reaction	réaction affective
Affective recess	retrait affectif
Affective regression	régression affective
Affective rejection	rejet affectif
Affective relation	relation affective
Affective repercussion	retentissement affectif
Affective retardation	arriération affective
Affective retired	retiré affectivement
Affective revendication	revendication affective
Affective rigidity	rigidité affective
Affective schizophrenia	schizophrénie affective
Affective sclerosis	sclérose affective
Affective separation	séparation affective
Affective shock	choc affectif
Affective sideration	sidération affective
Affective sleep	sommeil affectif
Affective solitude	solitude affective
Affective state	état affectif
Affective stimulation	stimulation affective
Affective stupor	stupeur émotionnelle
Affective suffering	souffrance affective
Affective suggestion	suggestion affective
Affective symbiosis	symbiose affective

Affective tendency	tendance affective
Affective tension	tension affective
Affective tone	ton émotionnel
Affective traumatism	traumatisme affectif
Affective trouble	trouble affectif
Affective tuning	accordage affectif
Affective unstable	instable affectif
Affective vacuum	vide affectif
Affective value	valeur affective
Affective violence	violence affective
Affective withdrawal	retrait affectif

Affective-arousal theory théorie de stimulation affective

Affective-habit habitude affective

Affectivity affectivité

Affectivity (forgotten-)	affectivité oubliée
Affectivity (human-)	affectivité humaine
Affectivity (paradoxical-)	affectivité paradoxale
Affectivity (parental-)	affectivité parentale
Affectivity (repressed-)	affectivité refoulée
Affectivity deterioration	détérioration de la affectivité
Affectivity disorder	trouble de l'affectivité
Affectivity exhausting	épuisement de l'affectivité

Aggression agression

Aggression (anger-)	agression de colère
Aggression (anti- social-)	agression antisociale
Aggression (asocial-)	agression asociale
Aggression (covert-)	agression couverte
Aggression (direct-)	agression directe
Aggression (displaced-)	agression déplacée
Aggression (dominance-)	agression de dominance
Aggression (evident-)	agression évidente
Aggression (frank-)	agression franche
Aggression (indirect-)	agression indirecte
Aggression (induced -)	agression induite
Aggression (masked-)	agression masquée
Aggression (maternal-)	agression maternelle
Aggression (neurotic-)	agression névrotique
Aggression (physical-)	agression physique
Aggression (predatory-)	agression prédatrice
Aggression (prosocial-)	agression prosociale
Aggression (psychic-)	agression psychique
Aggression (sadistic-)	agression sadique
Aggression (sexual-)	agression sexuelle
Aggression (vengeance-)	agression de vengeance
Aggression (verbal-)	agression verbale

Aggression antipredatory	agression antiprédatrice
Aggression delirium	délire d'agression
Aggression pulsion	pulsion d'agression

Aggressive **agressif**

Aggressive (asocial-)	agressif-asocial
Aggressive (dominant-)	agressif dominant
Aggressive action	acte agressif
Aggressive behaviour	comportement agressif
Aggressive character	caractère agressif
Aggressive charge	charge aggressive
Aggressive child	enfant agressif
Aggressive confrontation	confrontation aggressive
Aggressive desire	désir agressif
Aggressive disorder	désordre agressif
Aggressive emotion	émotion aggressive
Aggressive energy	énergie aggressive
Aggressive expression	expression aggressive
Aggressive fantasy	fantasme agressif
Aggressive father	père agressif
Aggressive impetus	impulsion aggressive
Aggressive imprint	empreinte aggressive ()
Aggressive instinct	instinct d'agressivité
Aggressive intention	intention aggressive
Aggressive mechanism	mécanisme agressif
Aggressive nature	nature aggressive
Aggressive obsession	obsession aggressive
Aggressive opposition	opposition aggressive
Aggressive oppositionism	oppositionnisme agressif
Aggressive personality	personnalité aggressive
Aggressive pleasure	plaisir agressif
Aggressive practice	pratique aggressive
Aggressive predatory	prédateur agressif
Aggressive psychopath	psychopathe agressif

Aggressive pulsion	pulsion aggressive
Aggressive reaction	réaction aggressive
Aggressive sadistic	sadique aggressive
Aggressive sexual act	acte sexuel agressif
Aggressive sexual behaviour	comportement sexuel agressif
Aggressive symptom	symptôme agressif
Aggressive tendency	tendance aggressive
Aggressive type	type agressif
Aggressive wife	épouse aggressive
Aggressive-passive	passive-aggressive
Aggressive-solitary	solitaire-agressif
Aggressiveness	agressivité
Aggressiveness inhibited	agressivité inhibée

Aggressivity **agressivité**

Aggressivity	agressivité
(constitutional-)	constitutionnelle
Aggressivity (direct-)	agressivité directe
Aggressivity (evident-)	agressivité évidente
Aggressivity (imaginary-)	agressivité imaginaire
Aggressivity (morbid-)	agressivité morbide
Aggressivity (sadistic-)	agressivité sadique
Aggressivity (substitute-)	agressivité substituée
Aggressivity (unconscious-)	agressivité inconsciente
Aggressivity (verbal-)	agressivité verbale
Aggressivity access	accès d'agressivité
Aggressivity crisis	crise d'agressivité
Aggressivity energy	énergie d'agressivité
Aggressivity expression	expression de l'agressivité
Aggressivity feelings	sentiments d'agressivité
Aggressivity forecasting	prévision de l'agressivité
Aggressivity	désir inconscient
unconscious desire	d'agressivité

الكتاب الإلكتروني لمعجم العلوم النفسية

المعجم الإلكتروني النفسي الإنجليزي " PDF doc "

إنجليزية - فرنسية - عربية

نموذج : تنزيل كامل مصطلحات حرف A الإنكليزي (Ko 1024)

www.arabpsynet.com/eDictBooks/A.afe.exe

المعجم الإلكتروني النفسي الفرنسي " PDF doc "

فرنسية - إنجليزية - عربية

نموذج : تنزيل كامل مصطلحات حرف A الإنكليزي (Ko 942)

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المعجم الإلكتروني النفسي العربي - المجلد 1 " PDF doc "

عربية - إنجليزية - فرنسية

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عربية - فرنسية - إنجليزية

نموذج : تنزيل كامل مصطلحات حرف أ العربي (Ko 1271)

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مجلة شبكة العلوم النفسية العربية : العدد 3 - جويلية - أوت - سبتمبر 2004

E.DICTIONNAIRE DES SCIENCES Psychologiques

TERMINOLOGIES PSY FRANÇAISE (FRANÇAIS - ANGLAIS - ARABE)

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A

Activité - Adaptation
 Agitation - Amnésie
 Anesthésie - Angoisse
 Anorexie - Antécédent

Activité	activity
Activité (cage d'-)	activity box
Activité académique	academic activity
Activité affective	affective activity
Activité analytique	analytic activity
Activité antisociale	antisocial activity
Activité au hasard	random activity
Activité autonome	autonomous activity
Activité catalytique	catalytic activity
Activité cérébrale	cerebral activity
Activité cognitive	cognitive activity
Activité collective	collective activity
Activité compensatoire	compensatory activity
Activité compulsive	compulsive activity ()
Activité conditionnelle	conditional activity
Activité confuse	confusion activity
Activité corticale	cortical activity
Activité créative	creative activity
Activité criminelle	criminal activity
Activité de distraction	amusement activity
Activité de fond	background activity
Activité de perception	perception activity
Activité de récréation	recreation activity
Activité de rêverie	reverie activity
Activité de stimulation	stimulant activity
Activité des flux nerveux	nervous flux activity
Activité discursive	discursive activity
Activité électrique	cerebral electric
cérébrale	activity
Activité électro-dermique	electrodermal activity
Activité endocrinienne	endocrinian activity
Activité érotique	erotic activity
Activité fantasmatique	fantastical activity
Activité féminine	female activity

Activité fonctionnelle	functional activity
Activité hétérosexuelle	heterosexual activity
Activité homosexuelle	homosexual activity
Activité hypnagogique	hypnagogic imagery
Activité hypothalamique	hypothalamic activity
Activité intellectuelle	intellectual activity
Activité masculine	masculine activity
Activité mentale	mental activity
Activité motrice	motive activity
Activité musculaire	muscular activity
Activité noétique	noetic activity
Activité onirique	oniric activity
Activité opérationnelle	operational activity
Activité perceptive	perceptual activity
Activité phasique	phasic activity
Activité pragmatique	pragmatic activity
Activité productrice	productive activity
Activité psycho-affective	psycho-affective () activity
Activité psychomotrice	psychomotor activity
Activité quotidienne	daily activity
Activité ralentie	slow activity
Activité relationnelle	relational activity
Activité rituelle	ritual activity
Activité rythmique	rhythmical activity
Activité sexuelle	sexual activity
Activité sociale	social activity
Activité socio-professionnelle	socio-professional activity
Activité spontanée	spontaneous activity
Activité symbolique	symbolic activity
Activité sympathique	sympathetic activity
Activité symptomatique	symptomatic activity
Activité thalamique	thalamic activity
Activité végétative	vegetative activity
Activité verbale	verbal activity
Activité vigile	vigil activity
Activité volontaire	voluntary activity
Activité-passivité	activity-passivity

Adaptation **adaptation**

Adaptation (conservation de l')	adaptation conservation
Adaptation (difficulté d')	adaptation difficulty
Adaptation (maladie de l'-)	adaptation illness
Adaptation (niveau d'-)	adaptation level
Adaptation (syndrome d'-)	adaptation syndrome
Adaptation à l'obscurité	dark adaptation
Adaptation à la lumière	light adaptation
Adaptation à la réalité	real adaptation
Adaptation alloplastique	alloplastic adaptation
Adaptation anxieuse	anxious adaptation
Adaptation atypique	atypical adaptation
Adaptation au couleur	color adaptation
Adaptation au réalité	reality adaptation
Adaptation autoplastique	autoplastic adaptation
Adaptation avec l'autre	adaptation with the other
Adaptation avec la situation	adaptation with situation
Adaptation avec le milieu	conducting adaptation
Adaptation biologique	biological adaptation
Adaptation clinique	clinical adaptation
Adaptation colérique	color adaptation
Adaptation corporelle	corporal adaptation
Adaptation créative	creative adaptation
Adaptation croisée	crossed adaptation
Adaptation culturelle	cultural adaptation
Adaptation de la naissance	birth adjustment
Adaptation de la personnalité	personality adaptation
Adaptation de la personne	person adaptation
Adaptation déficiente	deficient adaptation
Adaptation dépressive	depressive adaptation
Adaptation des conduites	conduct adaptation
Adaptation difficile	difficult adaptation
Adaptation du migrant	migrant adaptation
Adaptation émotionnelle	emotional adaptation
Adaptation endommagée	damaged adaptation
Adaptation ergonomique	ergonomic adaptation
Adaptation familiale	family adaptation
Adaptation fermée	closed adaptation
Adaptation fonctionnelle	functional adaptation
Adaptation générale	general adaptation
Adaptation illusionnelle	illusional adaptation
Adaptation lumineuse	light adaptation

Adaptation mauvaise	bad adaptation
Adaptation mentale	mental adaptation
Adaptation négative	negative adaptation
Adaptation nerveuse	neural set
Adaptation non spécifiée	non-specific adaptation
Adaptation ontogénétique	ontogenetic adaptation
Adaptation photopique	photopic adaptation
Adaptation physiologique	physiologic adaptation
Adaptation positive	positive adaptation
Adaptation posturale	posture adaptation
Adaptation précoce	precocious adaptation
Adaptation professionnelle	vocational adaptation
Adaptation psychique	psychic adaptation
Adaptation psychologique	psychological adaptation
Adaptation psychosociale	psychosocial adaptation
Adaptation rationnelle	rational adaptation
Adaptation réactionnelle	reactional adaptation
Adaptation reproductrice	reproductive adaptation
Adaptation schizophrénique	schizophrenic adaptation
Adaptation scolaire	scholastic adaptation
Adaptation secondaire	secondary adaptation
Adaptation sensorielle	sensory adaptation
Adaptation situationnelle	situational adaptation
Adaptation sociale	biological adaptation
Adaptation spontanée	spontaneous adaptation
Adaptation tendre	tender adaptation
Adaptation verbale	verbal adaptation
Adaptation visuelle	visual adaptation

Agitation	agitation
Agitation (conduite d'-)	agitation leading
Agitation (état d'-)	agitation state
Agitation aiguë	acute agitation
Agitation anxieuse	anxious agitation
Agitation atypique	untypical agitation
Agitation catatonique	catatonic agitation
Agitation confuse	confused agitation
Agitation désordonnée	disordered agitation
Agitation forcée	forced agitation
Agitation hystérique	hysterical agitation
Agitation incessante	unceasing agitation
Agitation maniaque	maniac agitation
Agitation mélancolique	melancholic agitation
Agitation mentale	mental agitation
Agitation motrice	motive agitation
Agitation nocturne	nocturnal agitation
Agitation onirique	oniric agitation

Agitation psychasthénique	psychasthenic agitation
Agitation psychologique	psychological agitation
Agitation psycho-motrice	psychomotor agitation
Agitation psychotique	psychotic agitation
Agitation schizophrénique	schizophrenic agitation
Agitation turbulente	turbulent agitation

Amnésie	amnesia	
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Amnésie affective	affective amnesia
Amnésie alcoolique	alcohol amnesia
Amnésie antérieure	anterior amnesia
Amnésie antérograde	anterograde amnesia

Amnésie antéro- rétrograde	anteroretrograde amnesia
Amnésie aphasie	aphasia amnesia
Amnésie audio- verbale	audio-verbal amnesia
Amnésie auditive	auditory amnesia
Amnésie auto- hypnotique	autohypnotic amnesia

Amnésie axiale	axial amnesia
Amnésie catathymique	catathymic amnesia
Amnésie chiffrée	number amnesia
Amnésie circonscrite	circumscribed amnesia
Amnésie continue	continuous amnesia
Amnésie corticale	cortical amnesia
Amnésie d'enregistrement	registration amnesia
Amnésie d'évocation	retrograde amnesia
Amnésie d'identité	identity amnesia
Amnésie d'intégration	integration amnesia
Amnésie de conversion	conversion amnesia
Amnésie de fixation	anterograde amnesia
Amnésie de lecture	reading amnesia
Amnésie de mémoration	memoration amnesia
Amnésie de remémoration	rememoration amnesia
Amnésie des événements	events amnesia
Amnésie des formes	form amnesia
Amnésie des mots	words amnesia

Anesthésie	anesthesia	
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Anesthésie affective	affective anesthesia
Anesthésie cérébrale	cerebral anesthesia
Anesthésie chirurgicale	surgical anesthesia
Anesthésie cutanée	cutaneous anesthesia
Anesthésie de la ceinture	belt anesthesia
Anesthésie douloureuse	painful anesthesia
Anesthésie du bassin	basin anesthesia
Anesthésie du pied	foot anesthesia

Anesthésie du poignet	wrist anesthesia
Anesthésie électrique	electric anesthesia
Anesthésie en chaussette	sock anesthesia
Anesthésie en gant	glove anesthesia
Anesthésie générale	general anesthesia
Anesthésie hypnotique	hypnotic anesthesia
Anesthésie hystérique	hysterical anesthesia
Anesthésie mentale	mental anesthesia
Anesthésie morale	moral anesthesia
Anesthésie nerveuse	nervous anesthesia
Anesthésie organique	organic anesthesia
Anesthésie pelvienne	girdle anesthesia
Anesthésie psychique	psychic anesthesia
Anesthésie psychogène	psychogenic anesthesia
Anesthésie sensorielle	sensorial anesthesia
Anesthésie sexuelle	sexual anesthesia
Anesthésie spirituelle	spiritual anesthesia
Anesthésie stocking	stocking anesthesia

Angoisse	anguish
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Angoisse (attaque d'-)	anguish attack
Angoisse (crise d'-)	anguish crisis
Angoisse (extinction de l'-)	anguish extinction
Angoisse (hystérie d'-)	anguish hysteria
Angoisse (névrose d'-)	neurosis anguish
Angoisse (rêve d'-)	anguish dream
Angoisse (signal d'-)	signal anguish
Angoisse actuelle	actual anguish
Angoisse aiguë	acute anguish
Angoisse anale	anal anguish
Angoisse anticipatoire	anticipatory anguish
Angoisse archaïque	archaic anguish
Angoisse automatique	automatic anguish
Angoisse chronique	chronic anguish
Angoisse d'abandon	abandon anguish
Angoisse d'anéantissement	annihilation anguish
Angoisse d'anticipation	anticipation anguish
Angoisse de castration	castration anguish
Angoisse de déféminisation	defeminization anguish
Angoisse de démasculinisation	demasculinization anguish
Angoisse de dépersonnalisation	depersonalization anguish
Angoisse de l'enfant	child anguish
Angoisse de la naissance	birth anguish
Angoisse de	masculinity anguish

Angoisse schizo- paranoïde	schizo-paranoid anguish
Angoisse somatogène	somatogenetic anguish
Angoisse vitale	vital anguish
Anorexie	anorexia
Anorexie dépressive	depressive anorexia
Anorexie électorale	elective anorexia
Anorexie hystérique	hysterical anorexia ()
Anorexie masculine	masculine anorexia
Anorexie matinale	morning anorexia
Anorexie mentale	anorexia nervosa
Anorexie névrotique	neurotic anorexia
Anorexie précoce	precocious anorexia
Anorexie primaire	primary anorexia
Anorexie psychogène	psychogenic anorexia
Anorexie psychotique	psychotic anorexia
Anorexie réactionnelle	reactional anorexia
Anorexie réversible	reversible anorexia
Anorexie secondaire	secondary anorexia
Anorexie sociale	social anorexia
Anorexie tardive	tardy anorexia
Anorexie transitoire	transitory anorexia
Antécédent	antecedent
Antécédent constant	constant antecedent
Antécédent de dépression	depression antecedent
Antécédent familial	familial precedents
Antécédent pathologique	pathologic precedent
Antécédent personnel	personal precedents
Antécédent psychiatrique	psychiatric antecedent
Antécédent psychique	psychic precedent
Antécédent psychologique	psychological antecedent
Antécédent psychopathologique	psychopathologic antecedent
Antécédent schizophrénique	schizophrenic precedent
Antécédent somatique	somatic antecedent

المعجم الشبكي للعلوم النفسية

ARABPSYNET DICTIONARIES

[illegible]

www.arabpsynet.com/HomePage/Psy-Dict.htm

قواعد النشر بمجلة شبكة العلوم النفسية العربية

تعمل "مجلة شبكة العلوم النفسية العربية" على الإحاطة بمسجلات الاختصاص في كافة فروع العلوم النفسية، محاوياً بذلك الاستجابة لحاجات المخصصين والمهتمين خصوصاً بعد تداخل تطبيقات الاختصاص مع مختلف فروع العلوم الإنسانية. وذلك من خلال اطلاع المصنف على اتجاهات البحوث العالمية وتعريفه بأخبار ومسجلات هذه البحوث عبر بعض الترجمات للأبحاث الأصلية. أما بالنسبة للبحوث العربية فإن المجلة تسعى لتقديم الدراسات والبحوث الرصينة المساهمة للمسجلات وللمحاجات الفعلية لمجتمعنا العربي.

تقبل للنش الأبحاث بإحدى اللغات الثلاث العربية، الفرنسية أو الإنكليزية.

- 1- الأبحاث الميدانية والتجريبية
 - 2- الأبحاث والدراسات العلمية النظرية
 - 3- عرض أو مراجعة الكتب الجديدة
 - 4- التقارير العلمية عن المؤتمرات المعنية بدراسات الطفولة
 - 5- المقالات العامة المختصة
- المجلة مفتوحة أمام كل الباحثين العرب من أطباء، فنانين و أساتذة علم النفس داخل الوطن العربي وخارجة وهي ترحب بكل المساهمات الملتزمة بشروط النشر التي حددها الهيئة العلمية للموقع على الشكل التالي:

■ قواعد عامة

- الالتزام بالقواعد العلمية في كتابة البحث.
- الجودة في الفكرة والأسلوب والمنهج، والتوثيق العلمي، والخلو من الأخطاء اللغوية والصوتية
- إرسال البحث بالبريد الإلكتروني APNjournal@arabpsynet.com أو بواسطة قرص مرن (لا تقبل الأبحاث الورقية).
- إرسال السيرة العلمية المختصة بالنسبة للكاتب اللذين لم يسبق لهم النشر في مجلة الشبكة.

■ قواعد خاصة

- 1- كتابة عنوان البحث واسم الباحث ولقبه العلمي والجهة التي يعمل لديها مع الملخصات والكلمات المفتاحية باللغات الثلاث العربية، الفرنسية أو الإنكليزية.
- 2- يراعى في إعداد قائمة المراجع ما يلي: تسجيل أسماء المؤلفين والمترجمين منبوعة بسنة النشر بين قوسين ثم بعنوان المصدر ثم مكان النشر ثم اسم الناشر.
- 3- استيفاء البحث لمطلوبات البحوث الميدانية والتجريبية بما يتضمنه من مقدمة والإطار النظري والدراسات السابقة ومشكلة البحث وأهدافه وفرضه وتعريف مصطلحاته.
- 4- يراعى الباحث توضيح أسلوب اختيار العينة، وأدوات الدراسة وخصائصها السيكومترية وخطوات إجراء الدراسة.
- 5- يقوم الباحث بعرض النتائج بوضوح مستعينا بالجدول الإحصائية أو الرسومات البيانية متى كانت هناك حاجة لذلك.
- 6- خضوع الأعمال الطبفسية المعروضة للنشر لفحص اللجنة الاستشارية الطبفسية للمجلة، كما خضوع الأعمال العلمفسية لفحص اللجنة الاستشارية العلمفسية وذلك وفقاً للنظام المعتمد في المجلة ويبلغ الباحث في حال اقتراحات تعديل من قبل المحكمين.
- 7- توجه جميع المراسلات الخاصة بالنشر إلى رئيس الموقع على العنوان الإلكتروني للمجلة.
- 8- الأراء الواردة في المجلة تعبن عن رأي كاتبها وجهات نظرهم.
- 9- لا تعاد الأبحاث المرفوضة لأصحابها.
- 10- لا تدفع مكافآت مالية عن البحوث التي تنشر.

قواعد التوثيق:

عند الإشارة إلى المراجع في نص البحث يذكر الاسم الأخير فقط للمؤلف أو الباحث وسنة النشر بين قوسين مثل (عكاشة، 1985) أو (Sartorius, 1981) وإذا كان عدد الباحثين من اثنين إلى خمسة تذكر أسماء الباحثين جميعهم للمرة الأولى مثل (دسوقي، النابلسي، شاهين، المصري، 1995)، وإذا تكررت الاستعانة بنفس المراجع يذكر الاسم الأخير للباحث الأول وآخرين مثل (دسوقي وآخرون، 1999) أو (Sartorius et al., 1981) وإذا كان عدد الباحثين ستة فأكثر يذكر الاسم الأخير للباحث الأول وآخرين مثل (الدعاش، وآخرون، 1999) أو (Skinner, et al., 1965)، وعند الاقتباس يوضع النص المقتبس بين قوسين صغيرين " " وذلك أرقام الصفحات المقتبس منها مثل: (أبو حطب، 1990: 43)

وجود قائمة المراجع في نهاية البحث يذكر فيها جميع المراجع التي أشير إليها في متن البحث وترتب ترتيباً أبجدياً. دون ترقيم وسلسل. حسب الاسم الأخير للمؤلف أو الباحث وتأتي المراجع العربية أولاً ثم المراجع الأجنبية بعدها وذلك بيانات كل مرجع على النحو الآتي:

-عندما يكون المرجع كتاباً:

اسم المؤلف (سنة النشر) عنوان الكتاب (الطبعة أو المجلد) اسم البلد: اسم الناشر، مثال: مراد، صلاح أحمد، (2001) الأساليب الإحصائية في العلوم النفسية والربوية والاجتماعية، القاهرة: الأجلو المصرية

-عندما يكون المرجع غثاً في مجلة:

اسم الباحث (سنة النشر) عنوان البحث، اسم المجلة، المجلد الصفحات، مثل: القطامي، نايبة (2002). تعليم التفكير للطفل الخليجي، مجلة الطفولة العربية، 12،

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-ج- عندما يكون المرجع غثاً في كتاب:

اسم الباحث (سنة النشر) عنوان البحث، اسم معد الكتاب، عنوان الكتاب، اسم البلد: الناشر، الصفحات التي يشغلها البحث

1- الإشارة إلى الهوامش بأرقام متسلسلة في متن البحث ووضعها مرتمة على حسب السلسل في أسفل النص التي وردت لها مع مراعاة اختصار الهوامش إلى أقصى قدر

ممكّن، وتذكر المعلومات الخاصة بمصدر الهوامش في نهاية البحث قبل الجزء الخاص بالمصادر والمراجع

2- وضع الملاحق في نهاية البحث بعد قائمة المراجع

■ الدراسات والمقالات العلمية النظرية:

تقبل الدراسات والمقالات النظرية للنشر إذا ملست من المراجعة الأولية أن الدراسة أو المقالة تعالج قضية من قضايا الطب النفسي أو علم النفس بمنهج فكري واضح يتضمن المقدمة وأهداف الدراسة ومناقشة القضية ومروية الكاتب فيها، هذا بالإضافة إلى التزامها بالآصول العلمية في الكتابة وتوثيق المراجع وكتابة الهوامش التي وردت في قواعد التوثيق

■ عرض الكتب الجديدة ومراجعتها:

تنشر المجلة مراجعات الباحثين للكتب الجديدة وتقدمها إذا توافرت الشروط الآتية:

1- الكتاب حديث النشر، ويعالج قضية تخص أحد مجالات الطب النفسي، علم النفس، العلاج النفسي أو التحليل النفسي

2- استعراض المراجع لمحتويات الكتاب وأهم الأفكار التي يطرحها وإيجابياته وسلبياته

3- غنى العرض على اسم المؤلف وعنوان الكتاب والبلد التي نشر فيها واسم الناشر، وسنة النشر، وعدد صفحات الكتاب.

كتابة تقرير المراجعة بأسلوب جيد

■ التقارير العلمية عن الندوات والمؤتمرات المعنية بقضايا الطفولة:

تنشر المجلة التقارير العلمية عن المؤتمرات والندوات والحلقات الدراسية في مجال علم النفس والطب النفسي التي تعقد في البلاد العربية أو غير العربية بشرط أن يغطي التقرير بشكل كامل ومنظماً أخبار المؤتمر أو الندوة أو الحلقة الدراسية وتصنيف الأبحاث المقدمة وذا نتجها وأهم القرارات والنوصيات كما تنشر المجلة محاضرات الحوارات في الندوات التي تشارك فيها لمناقشة قضايا تتعلق بالاختصاص.



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