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Cipralex offers:

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- Significantly faster symptom relief than Cipram[1]
- At least the same familiar tolerability profile as Cipram[2]

1. Gorman JM, Korotzer A, Su G. Int J Neuropsychiatric Med. 2002;7(4,suppl 1):40-44
2. Colonna L, Reines EH, and Andersen HF. IPMAD, November 27-30, 2002, Monte Carlo, Monaco.

Lundbeck Cipralex escitalopram

Presentation: Cipralex® tablets 10 mg: each containing 12.77 mg of escitalopram as the oxalate. Indications: Treatment of major depressive episodes. Treatment of panic disorder with or without agoraphobia. Dosage: Treating depression: Adults: Usual dosage is 10 mg once daily. Depending on individual patient response, the dose may be increased to a maximum of 20 mg daily. After the symptoms resolve during initial treatment (usually 2-4 weeks for antidepressant response), a period of treatment for at least 6 months is required for consolidation of the response. Panic disorder with or without agoraphobia: An initial dose of 5 mg is recommended for the first week before increasing the dose to 10 mg daily. The dose may be further increased up to a maximum of 20 mg daily, dependent on individual patient response. Maximum effectiveness is reached after about 3 months. The treatment lasts several months. Elderly (>65 yrs): Initial treatment with half the usually recommended dose and a lower maximum dose should be considered. Children and adolescents (<18 years): Not recommended, since safety and efficacy have not been investigated in this population. Reduced hepatic/renal function: In reduced hepatic function an initial dose of 5 mg daily for the first two weeks of treatment is recommended. Depending on individual patient response, the dose may be increased to 10 mg. Dosage adjustment is not necessary in patients with mild or moderate renal impairment. Caution is advised in patients with severely reduced renal function (CLCR< 30 ml/min). Contra-indications: Hypersensitivity to escitalopram. Escitalopram should not be used in combination with a non-selective, irreversible monoamine oxidase inhibitor (MAOI). Escitalopram may be started 14 days after discontinuing treatment with an irreversible MAOI and at least one day after discontinuing treatment with a reversible MAOI-A (RIMA). At least 7 days should elapse after discontinuing escitalopram treatment, before starting a non-selective MAOI. Pregnancy and Lactation: As safety during human pregnancy and lactation has not been established, careful consideration should be given prior to use in pregnant women. It is expected that escitalopram will be excreted into breast milk. Breast-feeding women should not be treated with escitalopram. Precautions: Driving and operating machinery: No direct impairment of psychomotor function. However, as with other psychotropic drugs, patients should be cautioned about the risk to their ability to drive a car or operate machinery. Similarly, though no pharmacokinetic or pharmacodynamic interactions are expected with concomitant alcohol intake, as with other psychotropic medication, the combination is not advised. Combination with the reversible MAO-A (RIMA) moclobemide is not recommended. Co-administration with serotonergic compounds is not recommended. Insulin and/or oral hypoglycaemic dosage may need to be readjusted in diabetics. Hyponatraemia has been observed with SSRI use. Caution in patients with a history of mania/hypomania and co-administration of ECT in patients on SSRIs. Patients with epilepsy, especially unstable epilepsy, should be carefully monitored. Stop treatment if patient develops serotonin syndrome. Caution in patients taking medicines that will affect clotting of blood, platelet function or patients with bleeding disorders (coagulation monitoring at the beginning & end of therapy recommended with oral anti-coagulant use). Use a low starting dose. For panic disorder to reduce the likelihood of an initial paradoxical anxiogenic effect experienced by some patients. This usually subsides within the first two weeks of continuous therapy. Do not stop treatment abruptly. As with all SSRIs it is advisable to closely monitor patients for suicide risk in the first few weeks of treatment. Drug Interactions: MAO inhibitors (see Contraindications/Precautions): Also advise caution in use with selegiline (MAOI-B). Use lithium and tryptophan with caution. Caution with medicinal products that are capable of lowering the epilepsy seizure threshold. Avoid with St John's Wort (Hypericum perforatum). In known poor metabolisers, with respect to CYP2C19, an initial 5 mg daily dose should be used, which can be increased to 10 mg after assessment. Co-administration with the known enzyme inhibitors omeprazole and high doses of cimetadine may require reduction of the escitalopram dose. Caution is advised with co-administration of drugs metabolised by the enzymes CYP2D6 (e.g. flecainide, propafenone, metoprolol, desipramine, clomipramine, nortriptyline risperidone, thioridazine & haloperidol) or CYP2C19 (weakly inhibited by escitalopram). Adverse Events: With escitalopram, adverse effects are in general mild and transient, and are most frequent during the first or second week of treatment, usually decreasing in intensity and frequency with continued treatment. Most prolonged administration, abrupt cessation of SSRIs may produce withdrawal reactions in some patients. Although withdrawal reactions may occur on stopping therapy, the available pre-clinical and clinical evidence does not suggest that SSRIs cause dependence. Withdrawal reactions have not been systematically evaluated with escitalopram, however the following have been observed with the racemic citalopram: dizziness, headache and nausea. The majority are mild and self-limiting. Abrupt withdrawal of escitalopram should be avoided. Most commonly observed events occurring more frequently with escitalopram than placebo in clinical trials include: nausea, sweating, somnolence, dizziness, insomnia, constipation, diarrhoea, appetite decrease, sexual dysfunction, fatigue, pyrexia, somnitis and yawning. Overdosage: Doses of 340 mg of escitalopram have been taken without any serious symptoms being reported. Symptoms of overdose with racemic citalopram (>600 mg): Dizziness, tremor, agitation, somnolence, unconsciousness, seizures, tachycardia, changes in the ECG with ST-T changes, broadening of the QRS complex, prolonged QT interval, arrhythmias, respiratory depression, vomiting, rhabdomyolysis, metabolic acidosis, hypokalaemia. It is anticipated that overdoses with escitalopram would result in similar symptoms. There is no specific antidote. Treatment is symptomatic and supportive with monitoring of cardiac and vital signs. Early gastric lavage suggested. Legal Category: Product licence holder: H. 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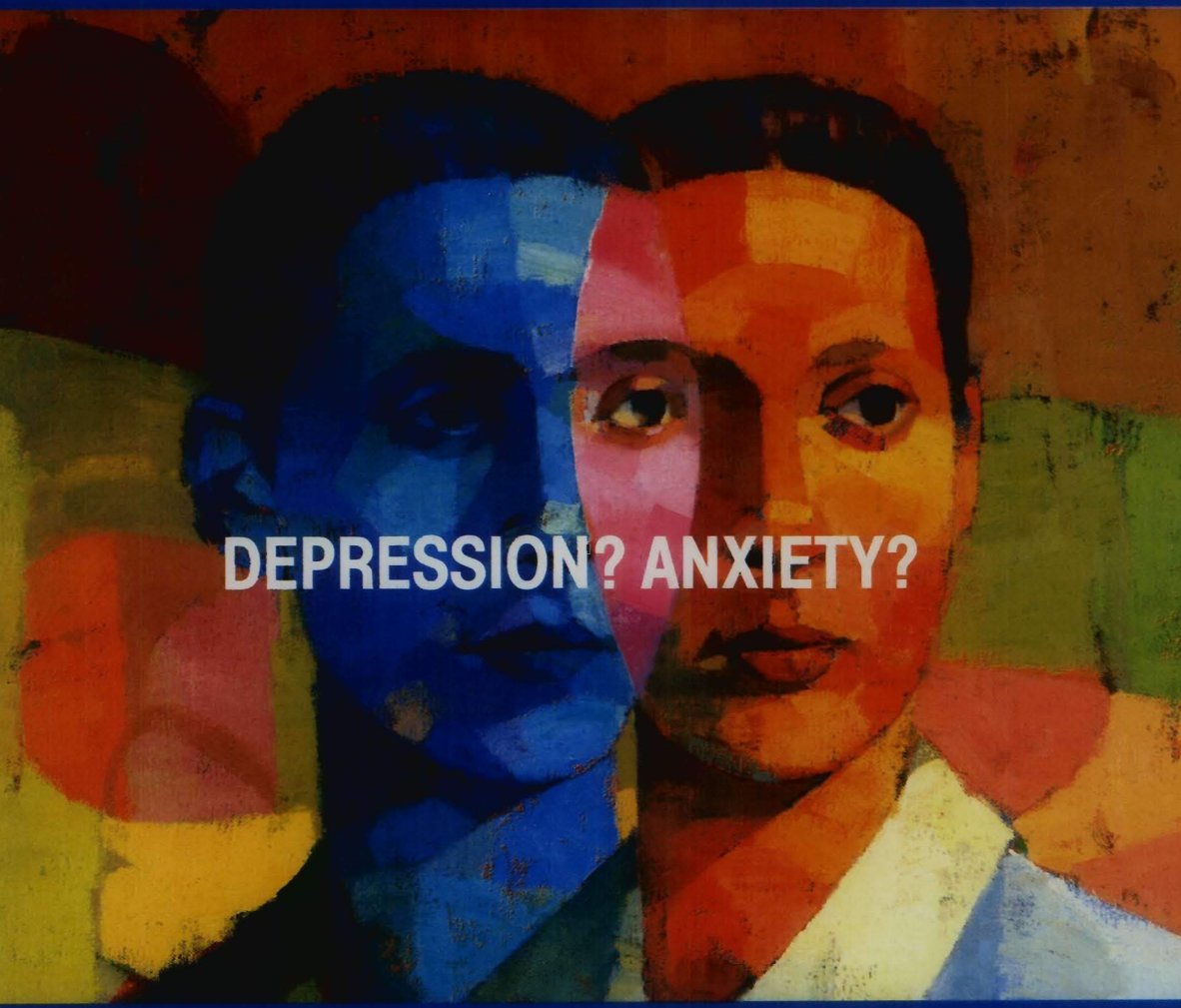

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المجلة العربية للطب النفسي

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معلومات هامة للناشرين

لقد صدرت المجلة العربية للطب النفسي عام 1989 من قبل اتحاد الأطباء النفسانيين العرب، وينشر في المجلة أبحاث علمية أصيلة، مراجعات علمية ومقالات تهتم بالعمل السريري. ويمكن أن تكتب المقالة باللغة العربية أو الإنجليزية مع ملخصين باللغة العربية والانجليزية. ويتم قبول الأوراق العلمية التي تتمشى مع أخلاقيات القوانين المحلية والدولية. ويمكن أن ترسل المقالات إما الى رئيس التحرير أو نائبه أو المحررين المشاركين. وتقيّم كل الأوراق من قبل محكمين دوليين.

المقالة: ترسل بنسختين مطبوعتين بمسافات مزدوجة على صفحات A4 بحواشي 3 سم. ويجب أن لا تزيد العناوين الفرعية عن ثلاث مستويات ويراعى عند كتابة المقال أن تخصص الصفحة الأولى لعنوان الورقة باللغة العربية والإنجليزية مع أسماء المشاركين بها دون ألقاب بما لا يزيد عن 40 حرف.

الصفحة الثانية: ملخص باللغة العربية لا يزيد عن مائتين وخمسين كلمة منظم حسب أهداف الدراسة وطريقتها والنتاج ثم الخلاصة.

الصفحة الثالثة: تحتوى على أسماء المشاركين وعناوينهم وعناوين المراسلة.

يمكن أن تخصص صفحة للشكر للأفراد والمؤسسات التي دعمت البحث.

أما الملخص باللغة الإنجليزية فيفضل أن يكون على صفحة منفصلة بعد المراجع.

الجداول: يجب أن تطبع الجداول بمسافات مضاعفة وعلى صفحات خاصة وترقم وأن يكون لها اسم مختصر.

الإيضاحات: كل الإيضاحات من صور أو رسومات يجب أن تكون ضعف الحجم الذي ستظهر به بالطباعة حتى يمكن تصويرها.

قائمة المراجع: يجب أن يتبع أسلوب فانكوفر بحيث تظهر المراجع حسب الترتيب الذي ظهرت به في المقالة وليس حسب الترتيب الأبجدي. ويفضل كتابة أسماء المشاركين في المرجع إلا إذا زاد العدد عن ستة فيكتفي بكتابة (وجماعته et al).

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ملاحظة: على الكاتب إرسال النسخة النهائية من الورقة أو المخطوطة على البريد الألكتروني للمجلة، أو على CD متوافق مع IBM-Microsoft World

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ابن سينا
وكتاب القانون – الجزء الثاني *

إن الأوجاع الأكّالة التي تخيل أن في رأس الإنسان دبيباً يأكل ، واللذّاعة ، فأنها تدل على مادة حارة ، والضَرَبَانية على ورم حار ، ويؤكد دلالتها لزوم الحمى ، والثقيلة الضاغطة على مادة ثقيلة باردة والممدّة على مادة ريحية ، والانتقال يؤكد ذلك ،والوجع الذي كأنه يطرق بمطرقة يدل على مثل البيضة والشقيقة المزمنة ، والوجع أيضاً يدل بجهته مثل أن الوجع الذي بمشاركة المعدة يكون على وجه ، والذي بمشاركة الكبد على هيئة أخرى كما سنذكره ، وقد يدل مع ذلك بدوامه ، فإن الوجع إذا دام في مقدم الرأس ومؤخره ، أنذر بالعلة المعروفة بقرانيطس .

فصل في دلائل الأمزجة الرديئة الواقعة في الجبّلة :

يرى " جالينوس" أن الحرارة تولد أختلاط العقل والهذيان ، وليلحق بهذا الطيش وسرعة وقوع البذاءات وافتنان العزائم وأن البرودة تولد البلادة ، وسكون الحركة، وليلحق بهذا بطء الفهم وتعذر الفكر والكسل ، وأن اليبوسة تفعل السهر ، ويدل عليها السهر ، وليشترط في هذا ما لم يكن عن الرطوبات البورقية ، ولم يكن مع ثقل في الدماغ ، ودوام استفراغ الفضول أو غير ذلك من دلائل الرطوبة ، فإن الرطوبة المالحة والبورقية بشهادة "جالينوس" نفسه تفعل أرقاً كما في المشايخ، وأما الرطوبة فتفعل النوم المستغرق ، واشترط مع نفسك الشرط المذكور .

ويرى "جالينوس" أن الدلالة على أن مزاجاً غالباً بلا مادة ، هو عدم سيلان الفضول مع دلالة سوء المزاج ، والدلالة على أنه غالب بمادة سيلان الفضول ، ونحن نقول: إن لم يكن سدد أو ضعف من القوة الدافعة، وعلامة ذلك ما ذكرناه وفرغنا منه ، فدلائل حرارة المزاج للدماغ سرعة نبات الشعر في أول الولادة ، أو في البطن وسواده في الإبتداء ، أو تسوده بعد الشقرة سريعاً وجعودته وسرعة الصلع وسرعة امتلاء الرأس وثقله من الأسباب الواقعة مثل الروائح ونحوها ، وتأذية بالروائح الحادة ، وقلة أستعمال النوم مع خفته وظهور عروق العينين ، وذكاء ما وسرعة التقلب في الآراء والعزائم ، كحال الصبيان ، ويدل عليه اللمس وحمرة اللون، ونضج الفضول المنصبة والمنتفضة واعتدالها في القوام بالقياس إلى غيره .

وأما دلائل المزاج البارد ، فزيادة نفض الفضول على ما ذكر من الشرط وسبوطة الشعر وقلة سواده وسرعة الشيب ،

وسرعة الأنفعال من الآفات وكثرة النوازل وعروض الزكام لأدنى سبب ، وخفاء العروق في العينين وكثرة النوم ، وتكون صورته مثل صورة الناعس ، بطئ حركة الأجفان والثبات على العزائم كحال المشايخ .

وأما دلائل المزاج اليابس فنقاء مجاري الفضول وصفاء الحواس والقوة على السهر وقوة الشعر وسرعة نباته لدخانية المزاج في السن الأول وسرعة الصلع وتجعيد الشعر .

وأما دلائل المزاج الرطب ، فسبوطة الشعر وبطء النبات منه ، وبطء الصلع وكدورة الحواس ، وكثرة الفضول والنوازل واستغراق النوم .

وأما دلائل المزاج الحار اليابس ، فعدم الفضول وصفاء الحواس وقوة السهر ، وقلة النوم ، وإسراع نبات الشعر في الأول ، وقوته وسواده وجعودته وسرعة الصلع جداً ، وحرارة ملمس الرأس وجفوفه مع حمرة بينة فيه ، وفي العين ، وتنقل في العزائم وعجلة فيها وقوة الفهم والذكر وسرعة الأفعال النفسية .

وأما دلائل المزاج الحار الرطب فإنه إن كان ذلك المزاج غير بعيد جداً من الاعتدال ، كان اللون حسناً والعروق واضحة والملمس حاراً ليناً ، وكون الفضول أكثر وأنضج ، والشعر أسبط إلى الشقرة غير سريع الصلع ، ويكون التسخن والترطيب سريعين إليه ، وأما إن كان بعيداً منه فيكون مسقاماً قبُولا للنكايات من الحر والبرد والأمراض العفنية في جوهره سريعاً ، وتكون حواس صاحبه ثقيلة كدرة وعيناه ضعيفتان ولا يصبر عن النوم ، ويرى أحلاماً مشوشة .

وأما دلائل المزاج البارد اليابس ، فأن يكون الرأس بارد الملمس ، حائل اللون خفي العروق فيه وفي العينين ، بطئ نبات الشعر أصهبة رقيقه بطئ الصلع ، خصوصاً إن لم يكن يبسه أغلب من برده، ويكون متضرراً بالمبرِّدات على الشرط المذكور وتكون الحواس صافية في الشيبة، فإذا طعن في السن ضعف بسرعة وهَرم، وظهر التشنج والتعفن والتقبض في نواحي رأسه ، ويكون سريع الشيخوخة وتكون صحته مضطربة فتارة يكون خفيف الرأس منفتح المسالك وتارة يكون بالخلاف .

وأما المزاج البارد الرطب فيكون الإنسان فيه كثير النوم مستغرقاً فيه رديء الحواس، كسلان بليداً كثير استفراغ الفضول من الرأس ، ويدل عليه أيضاً بطء الصلع وسرعة وقوع النوازل ، وأما دلائل الأورام فسنقوله في التفصيل .

فصل في قوانين العلاج :

إننا إذا أردنا إن نتقياً مادة ، فإن دلت الدلالة على أن معها دماً وافراً وليس في الدم نقصان أي مادة كانت ، بدأنا بالفصد من القيفال ، ومن عروق الرأس المذكورة في باب الفصد مثل عروق الجبهة والأنف وعروق ناحية الأذن ، ويجب أن يقع فصدها في خلاف جانب الوجع .

فإن كان الأمر عظيماً والدم غالباً ، فصدنا الوداج وإنما نميل إلى الفصد–وإن غلبت الأخلاط الأخرى أيضاً فنبدأ به – لأن الفصد تقيؤه (استفراغ) مشترك للأخلاط ، فإن كانت المادة دماً فقط كفى الفصد التام ، وإن كانت إختلاطاً أخرى نظرنا فإن كان ذلك بشركة البدن كله استفرغنا البدن كله ، ثم فصدنا الرأس وحده واستعملنا الاستفراغات التي تخصه، ولا نُقدِمُ عليها البتة إلا بعد استفراغ البدن إن كان في البدن خِلط ، وذلك إن علمنا أن المادة فيه ناضجة ، وذلك بمشاهدة ما يجلب إليه ، إن لم يكن رقيقاً جداً أو غليظاً جداً ، وإن كان المرض قد وافى المنتهي ، وكنا قد تقدمنا بالإنضاج بالمَرُوخَات والنَّطُولات والضمادات المُنضِجَة استفرغنا من الرأس خاصة بالغرغرة إن لم نَخَفْ آفة في الرئة ، ولم تكن النوازل المستنزلة بالغرغرة من جنس خلط حاد لاذع ، ولم يكن الإنسان قابلاً لأمراض الرئة ، وكان يمكن الاحتراس عن نزول شئ رديء إلى الرئة ، وكان حال الرأس أشد أهتماماً من حال الرئة ، واستعملنا أيضاً المشمومات المُفتِّحة المُعطِّسة والسَّعُوطات والنَّطُولات لتجذب المواد من الرأس .

وربما ضمَّدنا الرأس بعد الحلق بأدوية مسهِّلة لحبس الخلط الذي فيه ، أما الدم فإن كان في البدن كله ، وكان حصل في الرأس مادة وافرة فصدت القيفال ، وإن كان بعد لم يحصل وهو (آخذٌ) في الحصول فصَدت الأكحل ، وإن خفت الحصول قبل أن يأخذ في الحصول ، مثل أن يقع سبب جذاب للأخلاط حول الرأس من حر خارجي أو ضربة أو غير ذلك ، فصَدتَ الباسليق ، وإن شئت أن تجذب أكثر من ذلك فصَدت الصافن وحَجَمتَ الساق فوق الكعب بشبر ، وفصدت عروق الرِّجل ، وإن كان بمشاركة عضو فصَدتَ العرق المشترك لهما ، إن أردت أن تستفرغ منهما جميعاً ، وإن أردت الجذب إلى ناحية من استفراغ العضو المشارك فصَدتَ عِرقاً يشارك العضو المتقدم بالعلة، ويقع في خلاف جهة الرأس ، ثم إذا توجهت نحو الرأس وحده أو كان الدم من أول الأمر وحده فيه ، فما كان واقعاً في الحُجُب الخارجة من القحف على ما سنذكره في الأمراض الجزئية ، أو كان الوجع محسوساً بقرب الشؤون وأردت علاجاً خفيفاً ، فالحجامة عند النقرة ، وإن كان غائراً وكان لا يُرجَى انجذابه إلى خارج القحف ، فصدت عرق الجبهة خاصة إن كان الوجع مؤخراً ، وبعد أخذ الدم يتناول المستفرغات المتخذة من الهليلج وعصارات الفواكه ، إن بقيت حاجة ، ويستعمل الحقن ، وإن كانت العلة صعبة ، مثل سكتة دموية فصدت من الوداج .

الفصل الأول كلام كُلى في الصدُّاع:

الصداع ألم في أعضاء الرأس ، وكل ألم فسببه تغير مزاج دفعة ، وأختلافه أو تفرق إتصال أو اجتماعهما جميعاً . وتغيُّر المزاج هو أحد الستة عشر المعروفة ،

وإن كان الرطب هو غير مؤثر ألماً إلا أن يكون مع مادة تتحرك ، فتُفَرِّقُ الاتصال معلوم ،وأصنافه بحسب أسبابه معلومة ، واجتماع سببي الألم معاً يكون في الأورام، والأورام كما علمت معدودة الأصناف ، وأصنافها أربعة ، وجميع ذلك قد يكون في جوهر الدماغ نفسه ، وقد يكون في الحجاب المطيف به ، وقد يكون في الجانبَين المطيفَين به ، وقد يكون في العروق ، وقد يكون في الأغشية الخارجة عن القحف لما بينها من العلائق المعروفة في التشريح الموصوف ، وقد يكون بمشاركة غيره له: إما عضوٌ يصل بينه وبين أعضاء الرأس واشجهُ العصب مثل المعدة ، الرحم ، الحجاب وأعضاء أخرى إن كانت ، أو عضوٌ يصل بينه وبين الدماغ واشجة العروق من الأوردة والشرايين مثل القلب ، الكبد والطحال ، وإما عضوٌ يجاوره مجاورةً أخرى مثل الرئة الموضوعة تحته ، فيؤدي إليه آفته ، وإما عضوٌ مشارك لعضو من جهة ، وللدماغ من جهة أخرى مثل مشاركته للكلية في أوجاعها ، وإما بمشاركة البدن كله كما يكون في الحميات ، وإما كان بمشاركة فقد يكون بأدوار ونوائب ، بحسب أدوار ونوائب السبب الذي في العضو المشارك ، مثل ما يكون بمشاركة المعدة إذا كان لانصباب المواد المرارية أو غيرها إليها أدوار ، ومثل ما يكون مع أدوار تزيُّد أصناف الحميات والصُّداع ، فقد ينقسم من جهة أخرى ، فإن منه ما سببه صنف من الأسباب البادية ، مثل صداع الخُمَار ما دام صداع خُمَار ، ولم يرسخ لرسوخ سبب أزيَد من ذلك أو متولد من ذلك ، ومثل صداع أكل شيء حار نحو الثوم وغيره ، ومنه ما سببه سابق قد وصل فهو لابث فيلبث هو لأجله ، وربما كان عَرضاً ثم صار مرضاً ، وإذا بقي مرضاً بعد الحُميات الحارة أنذر بعِلَلٍ دماغية ، ودلَّ على عجز الطبيعة عن دفع المادة بالكمال ، برُعاف أو غيره من العلل التي ينذر بها سبات ، وسكات ، وجنون أو استرخاء ، أو صمم بحسب جوهر المادة وبحسب حركاتها .

والصداع قد ينقسم من جهة مواضعه ، فإنه ربما كان في أحد شِقَّي الرأس وما كان من ذلك معتاداً لازماً ، فإنه يسمى شقيقة ، وربما كان في مقدم الرأس وربما كان في مؤخر الرأس ، وربما كان محيطاً بالرأس كله ، وما كان من ذلك معتاداً لازماً فإنما يُسمَّى : بَيْضَة وخُوْذَة تشبيها ببيضة السلاح التي تشتمل على الرأس كله.

والصداع قد يختلف أيضاً بالشدة والتوسط والضعف ، فمن الصداع ما هو شديد جداً حتى إنه إذا صادف يافوخ صبي لين العظام مزقه وصدع درزه ، ومنه ما هو ضعيف مثل أكثر ما يكون ليثرغس ، ومن الضعف ما هو لازم ، ومنه ما هو غير لازم ، وربما كان الصداع الذي سببه ضعيف يعرض لبعض دون بعض ، فيعرض لمَن حِسُّ دماغه قويّ ، ولا يعرض لمِمَن حِسُّ دماغه ضعيف ، وبالجملة فإن من هو قوي حِسِّ الدماغ

قابل بالتصدع من كل سبب مصدع ، وإن ضعف .

وبالجملة فإن الدماغ يكون سريع القبول للمصُدِّعات : إما لضعفه ، وقد عرف في الكليات أن الضعف تابع لسوء المزاج ، وإما لقوة حِسِّه فيتأذى من كل سببٍ ، وإن خف . وأيضاً فإن من الصداع ، ما لا أعراض له ومنه ما يؤدي إلى أعراض تختفي بنواحي الرأس : مثل أن يُحدث – أعني الصداع – لشدة الوجع– أوراماً في نواحي الرأس ، ومنه ما يؤدي إلى أعراض تتعدى إلى أعضاء أخرى ، مثل أن يتأذى أذاه وأضراره ، أو (إيلامه) إلى أصول الأعصاب ، فيحدث التشنج أو يتعدى شيء من ذلك إلى المعدة ، فيحدث سقوط الشهوة والفُواق والغثيان وضعف الهضم ونحو ذلك . واعلم أن الصداع المزِمن إما أن يكون لبلغم ، أو لسوداء ، أو ضعف رأس أو ورم صلب مُبْتَدأ ، أو حار قد صلب وهو الكثير والصادع ، وجميع الأمراض قد تختلف فربما كان المرض مسلّماً ، والمسلّم هو الذي لا مانع من تدبيره بما يجب له في نفسه ، ومنه ما ليس بمسلم بل هو ذو قرينة ، وربما منعت عن تدبيره بالواجب مثل أن يكون صداع ونزلة فتُعارض النزلة الصداعَ في واجبه من التدبير .

والصداع أيضاً قد ينقسم باعتبار آخر ، فإن من الصداع ما يعرض أحياناً للصحيح لا قلبه به ومنه ما إنما قد يعرض لذي أورام و أوصاب ، ومن الأبدان أبدان مستعدة للصداع وهي : الأبدان الضعيفة الرؤوس الضعيفة الأعضاء الهاضمة ، فتتولد فيها أبخرة وتنصب إلى معدِاتهم أخلاط مرارية ، فتصدِّع ، وأيضاً فإن من المتناولات أشياء مصدِّعة ، قد ذكرت في جداول الأدوية المفردة ، وجميع الأفاوية مصدِّعة ، خصوصاً السليخة والقسط ، الزعفران ، والدارصيني ، والحماما ، وجميع المبخِّرات مصدِّعة حارةً كانت أو باردة ، ولكنها إذا تعاقبت تدافعت ، أعني إذا كان قد تقدم ما آذى بحرارة بخاره ، وعقبه ما يبخر بخاراً بارداً أو بالعكس ، وأما إذا كان الأذى ليس بالكيفية وحدها ، بل وبالكمية فلا ينفع تعاقبها ، بل يضر . وقد يكثر الصداع البارد للاحتقان في الشتاء ، وإذا كان الصيف شمالياً قليل المطر وكان الخريف جنوبياً مطيراً كثر الصداع في الشتاء ، وكثيراً ما يكون الصداع بسبب تأدية الرياح الأبخرة الخبيثة إلى الرأس .

فصل في تدبير كلي للصداع:

وفي علاج الصداع يقول ابن سينا أنه أسوة بغيره من العلل ، في وجوب قطع سببه ومقابلته الضد (أي إعادة التوازن حسب نظرية الأخلاط) وبعد ذلك فإن من الأمور النافعة في إزالة الصداع ، قلة الأكل والشرب وخصوصاً من الشراب (الخمر) وكثرة النوم ، ولا شيء للصداع كالتوديع (العيش في هدوء ودَعَةٍ بعيداً عن كل ما يثير) وتَرك كل ما يحرِّك من الجماع ومن الفكر .

ومن الأشياء القوية في جذب مادة الصداع إلى أسفل دلك الرجلين فإنَّ كثيراً ما ينام عليه المصاب الصداع ، وربما سببنا الماء الحار على أطراف المصاب بالصداع وتُديمُ ذلك ، فيحس بأن الصداع ينزل من رأسه إلى أطرافه .

ويحذر ابن سينا من أن القيء ليس من معالجات الصداع، وهو شديد الضرر ، إلا أن يكون – الصداع – بسبب المعدة وبمشاركتها فينتفع بالقيء .

وربما كان الصداع عَرَضاً – لمرض – ثم صار مرضاً ، وإذا بقي مرضاً بعد الحُمَّيَات الحارة أنذر بعلل دماغية.

والصداع قد يختلف بالشدة والتوسط والضعف ، فمن الصداع ما هو شديد جداً، حتى أنه إذا صادف يافوخ صبي لين العظام مزقه وصدع درزه .وكما أن الصداع يحدث متأثراً من أعضاء أخرى في الجسم ، فإنه أيضاً قد يؤثر في أعضاء أخرى ، فيحدث التشنج أو يتعدى شيء من ذلك إلى المعدة ، فيحدث سقوط الشهوة والفواق والغثيان وضعف الهضم ونحو ذلك .ولبيان سبب الصداع يقول : " فيجب أن تتثبت في مثل هذا ، وتتعرف حال كل واحد من العضوين في نفسه ، فتَحدُسَ السابق من المسبوق " وذلك دلالة على التثبت من كون الصداع في الدماغ أثر على المعدة أو المعدة سَبَّبَت الصداع .

وربما كان الصداع من ضربة أو سقطة أو قطع من خارج ، والذي يكون من داخل فربما لم يلتحم وبقي قرحة تؤذي الرأس .وتديم التصديع، والضربة والسقطة ربما كانت خفيفة فتعالج ، وربما بلغت أن يتقلقل لها الدماغ (ارتجاج دماغ) ويهلك.

ويقول : إن الأعضاء المشاركة للرأس أولها وأولاها المعدة ، ولذلك هناك أسباب غذائية للصداع سببها أكل بعض الأنواع كصداع الثوم والبصل والخردل ، أو شرب الخمر كصداع الخمار وصداع تناول المشروبات أو الأطعمة الباردة ، أو من الرياح والأبخرة كصداع النتن وصداع الطيب ، وهذه كلها العوامل الحافزة لصداع الشقيقة . وقد يكون الصداع نسائياً، أي بعد ولادة أو إسقاط أو احتباس طمث أو قلته ، وهذا ما يحدث في صداع الشقيقة

أما الصداع الناتج عن ضربة الشمس فعلاجه عند ابن سينا التبريد بالإيواء إلى المساكن الباردة واستعمال الضمادات المُبَرِّدة .

أما الصداع الكائن عن مادة حارة دموية فعلاجه بالفصد إذا كانت صحة المريض تسمح بذلك ، وإن لم يكف الفصد في عروق الساعد ، ورأيت في الرأس والوجه والعين امتلاءً واضحاً ، فيجب فصد العروق التي يستفرغ (يتقيأ) فصدها من نفس الدماغ كفصد العروق التي في الأنف من كل جانب ، وفصد العروق التي في الجبهة ، فإنه عرق يستأصل فصده كثيراً من آلام الرأس .

أما الصداع الذي سببه ضربة البرد الشديد فعلاجه عند ابن سينا التكميد بما هو مسخن ، وأن يحسروا عن رؤوسهم في الشمس ، والمصرود يحال بينه وبين

الحركات البدنية والنفسانية والفكرية ويمنع الشراب البارد والبروز للبرد ، ومما يفيد أنواع السعوط .

أما الصداع الحادث عن تعاطي الكحول والخمور ويسميه ابن سينا صداع الخُمَار، فأول ما يجب فعله تنقية المعدة، ويقصد بالقيء وإن لم يجب القيء بإسهال المعدة، وينصح بالنوم فهو الأصل بعلاجه.

أما الصداع الناتج عن ضربة أو سقطة وتدبير من يعرض له زعزعة فيجب أولاً تسكين الألم إما باستفراغ وإما بجذب إلى الخلاف لئلا يرم ، وتعالج الجراحة إن حدثت لتندمل ، وإن أصابت المريض الحمى واختلط العقل فقد أخذ في التورم وهنا يجب اللجوء للفصد لمنع التورم .

ويقول: إنه إذا وصل الألم إلى حُجُب الدماغ كان فيه خطر، وإذا خرج بسبب الضربة دم من الدماغ فيجب أن يسقى صاحبه أدمغة الدجاج ما أمكن ثم يسقى عليه ماء الرمان الحامض.

فصل في الشقيقة :

فنقول هي وجع في أحد جانبي الرأس يهيج ، ويحدُّها جالينوس بأنها الساترة المتوسطة ، وربما كان سببه من داخل القحف وربما كان في الغشاء المجلل للقحف ، وأكثر ما يكون في عضل الصدغ ، وما كان خارجاً فقد يبلغ إلى أن لا يحتمل المس ، وتكون المواد واصلة إلى موضعه ، إما من الأوردة والشرايين الخارجة ، وإما من الدماغ نفسه وحُجُبه ، فيصعد أكثر ذلك من طريق الدروز ، وقد يكون من أبخرة تندفع من البدن كله ، أو عضو من ذلك الشق ، وأكثر ما تكون الشقيقة تكون ذات أدوار ، وإنما تكون على الأغلب عن الأخلاط ، ولا تكون شقيقة لها قدر من سوء مزاج مفرد ، والتي تكون من الأخلاط فقد تكون من أخلاط حارة ، ومن أخلاط باردة ، ومن رياح وأبخرة ، وقد علمت العلامات ، وتجد مع البارد سكوناً بالتسخين وتمدداً قريباً ، ومع الحار سخونة بالملمس وضرباناً في الأصداغ وراحة بالمبرِّدات ، وأيضاً فإن البارد يحس معه ببرد والحار يحس معه بحر وذلك عند اشتداد الوجع .

فصل في السبات والنوم :

يقال سبات للنوم المفرط الثقيل، لا لكل مفرط ثقيل، ولكن لما كان ثقله في المدة والكيفية معاً ، حتى تكون مدته أطول ، وهيئته أقوى ، فيصعب الانتباه عنه ، وإن نُبِّه . فالنوم منه طبيعي في مقداره وكيفيته ، ومنه ثقيل ، ومنه سبات مستغرق ، والنوم على الجملة ، رجوع الروح النفساني عن آلات الحس والحركة إلى مبدأ تتعطل معه آلاتها عن الرجوع بالفعل فيها ، إلا ما لا بد منه في بقاء الحياة وذلك في مثل آلات التنفس . وقد يكون (السبات) من كثرة الديدان وحب القرع، وقد يكون من انضغاط الدماغ نفسه تحت عظم القحف أو صفحه أو قشره إذا أصاب الدماغ ضربة .

والسبات الكائن عن الدم فيعلم من انتفاخ الأوداج ، وحمرة العينين والوجنتين

وحمرة اللسان ، وحس الحرارة في الرأس وما أشبه ذلك مما علمت ، وإن كان الدم أو البلغم مع ذلك مجتمعاً اجتماع الأورام ، رأيت علامات قرانيطس أو ليثرغس أو السبات السهري ، وإن كان السبب فيه أبخرة تجتمع وترتفع من البدن في حُمَّيَات وخاصة عند وجع الرئية والورم فيها المسمى ذات الرئة والأبخرة من المعدة – علمت كلاً بعلاماته ، فإنه إن كان من المعدة تَقَدَّمَهُ سَدَر ودُوار ودَويّ وطنين وخيالات ، وكان يخف مع الجوع ويزيد من امتلاء ، وإن كان منا ناحية الرئة والصدر تقدمه الوجع الثقيل ، أو الوجع في نواحي الصدر وضيق النفس والسعال، وأعراض ذات الجنب ، وذات الرئة ، وكذلك إن كان من الكبد تَقَدَّمَهُ دلائل مرض في الكبد ، وإن كان من الرحم تَقَدَّمَهُ علل الرحم وامتلاؤها ، والذي يكون من ضربة على الهامة أو على الصدغ فيعرف بدليله .

والفرق بين السُّبات وبين السكتة ، أن المَسبُوت يمكن أن يفهم ويُنَبَّه ، وتكون حركاته أسلس من إحساسه ، والمسكوت مُعَطل الحس والحركة .

ونبض المغشيّ عليه أضعف وأصلب ، والغشي يقع يسيراً مع تغير اللون إلى الصفرة وإلى مشاكله لون الموتى وتبرد الأطراف .

وأما السبات فلا يتغير فيه لون الوجه، إلا إلى ما هو أحسن ولا ينحف رقعة الوجه والأنف ولا يتغير عن سحنة النوم إلا بأدنى تهيج وانتفاخ.

والفرق بين المسبوت وبيت المختنقة الرحم، أن المسبوت يمكن أن يفهم ويتكلم بالتكلف ، والمختنقة الرحم تفهم بعسر ولا تتكلم البتة ، وتكون الحركة – خاصة حركة العنق والرأس والرجل – أسهل على المسبوت والحس وفتح الأجفان أسهل على المختنق رحمها ، ويكون اختناق الرحم سبباً يقع دفعة ، ويقضي سلطانه ، وينقضي أو يقتل ، والسبات قد يمتد ويكون الدخول في الستغراق فيه متدرجاً ، ويبتدئ بنوم ثقيل إلا أن يكون سببه برداً يصيب دفعة أو دواء يشرب ، فيعلم ذلك قطعاً

علاج السبات والنوم الثقيل الكائن في الحميات :

أما السبات الذي هو عرض مرض في بعض الأعضاء فطريق علاجه فصد ذلك العضو بالتدبير ليتنقى ويزل ما به ، ويقويه الدماغ حتى لا يقبل المادة ، وذلك بمثل دهن الورد والخل الكثير لئلا ينوم الدهن إذا انفرد وحده وبعصارات الفواكه المقوية ، وبعد ذلك النَّطولات المبردة ، ثم ينتقل إلى المحللة إن كان احتبس في الدماغ شيء ، وقد عرفت جميع ذلك في القانون الذي يكون في الحميات ، وفي ابتداء الأدوار ، فيجب أن يبادر إلى ربط الأطراف وتحريك العطاس دائماً، وإشمام الخل وبخاره ، وتعريق الرأس بدهن الورد والخل الكثير أو ماء الحصرم والرمان ، والقوابض التي تكون لشرب

المخدرات فيعالج بحسب ذلك المخدر وسقي ترياقه .
وأما الكائن لغلة الدم فيجب أن يبادر إلى الفصد من القيفال وحجامة الساق ، أو فصد الصافن ، ويستعمل الحقن المعتدلة .
ومن معالجاته أنه يسمع صاحبه ويرى ما يغمه، فإن الغم في أمثال هذه الأمراض التي يضعف فيها الفكر ويجمد، فهو يحرك النفس ويرده إلى الصلاح.

فصل في الدوار :

الدُّوار : هو أن يتخيل لصاحبه أن الأشياء تدور عليه ، وأن دماغه وبدنه يدور ، فلا يملك أن يثبت بل يسقط ، وكثيراً ما يكره الأصوات ويعرض له من تلقاء نفسه مثل ما يعرض لمن دار على نفسه كثيراً بالسرعة ، فلم يملك أن يثبت قائماً أو قاعداً ، وأن يفتح بصره ، وذلك لما يعرض للروح الذي في بطون دماغه ، وفي أوردته وشراييته من تلقاء نفسه ، ما يعرض له عندما يدور دوراناً متصلاً ، والفرق بين الصرع والدوار ، إن الدوار قد يثبت مدة ، والصرع يكون بغتة ويسقط صاحبه ساكناً ويفيق ، وأما السّدَر ، فهو أن يكون الإنسان إذا قام أظلمت عينه وتهيأ للسقوط ، والشديد منه يشبه الصرع، إلا أنه لا يكون مع تشنج كما يكون الصرع .
وهذا الدوار قد يقع بالإنسان بسبب أنه دار على نفسه فدارت الأبخرة والأرواح فيه كما يدور الفنجان المشتمل على ماءٍ مدةً. ويسكن فيبقى ما فيه دائراً مدة.

وكلما كان البدن أضعف كان هذا الأنفعال فيه أشد كما في المرضى ، فإنه قد يبلغ المريض في ذلك مبلغاً بعيداً حتى أنه ليُدار به بأدنى حركة منه ، لأنهم يحتاجون في الحركة إلى تكلف شديد يتمكنون به من الحركة لضعفهم ، فيعرض لروحهم أذى وأنفعال وتزعزع .
وقد يكون الدوار إما من أسباب بدنية حاضرة في جوهر الدماغ ، حاصلة فيه من أبخرة جائلة في العروق التي فيه وفي العصب ، وإما من أخلاط محتقنة فيه من كل جنس فيتبخر بأدنى حركة أو حرارة ، فإذا تحركت تلك الأبخرة حركت بحركتها الروح النفساني الذي إنما ينضج ويتقوم في تلك العروق ، ثم يستقر في جوهر الدماغ ، ثم يتفرق في العصب إلى البدن .
وقد يكون من محرك للروح من خارج ، مثل ضارب للرأس أو كاسر للقحف حتى يضغط الدماغ ، والروح الساكن فيتبعه حركات مختلفة دائرة متموجة ، كما يحدث في الماء من وقوع ثقل عليه أو وقوع ضرب عنيف على متنه فيستدير موجه ، ووقوع مثل ذلك في الهواء والجرم الهوائي أولى لكنه لا يحس .
والدوار في المشايخ ينذر بسكتة، وقد يحل الدوار (إلى) صداع عارض، وقد يحل الصداع (إلى) دوار عارض.
ومن مقدماته أوجاع الرأس والدويُّ والطنين والثقل في الرأس ، ويجد ظلمة ثابتة ويجد في الحواس تقصيراً حتى في الذوق والشم ، ويحس في الشرايين المتقدمة ضرباناً شديداً ويصيب ثقلاً في

الشم . وقد يكون بسبب مشاركة أعضاء أخرى من الجسم فإن كان من الرحم تَقدَّمُه اختناق احتباس المني أو الطمث أو أورام فيه وكذلك إن كان من المثانة.

المعالجات:

أما الكائن بسبب دوران الإنسان على نفسه ونظره إلى الدورات أو نظرة من مكان عال ، فيعالج بالسكون والقرار والنوم إن لم يسكن سريعاً ويتناول القوابض الحارة ويكسر لقماً فيها ويتناولها .

وأما الكائن عن دم وأخلاط محتقنة في البدن ، فيعالج بالفصد من القيفال ، ثم من العرق الساكن الذي خلف الأذن ، فإنه أفضل علاج لجميع أصناف الدُّوار المادي.

وربما كوي كياً وخاصة فيما كان سببه صعود أبخرة من البدن في أي الطريق صعدت وتنفع الحجامة على النقرة وعلى الرأس أيضاً.

ويجب أن يجتنب صاحب الدُّوار النظر إلى كل شيء دائر بالعجلة ويجتنب الإشراف من المغارات ومن القُلل والآكام والسطوح العالية .

فصل في الصرع :

الصرع علة تمنع الأعضاء النفسية عن أفعال الحس والحركة والانتصاب منعاً غير تام ، وذلك لسدة تقع ، وأكثره لتشنج كلي يعرض من آفة تصيب البطن المقدم من الدماغ فتحدث سدة غير كاملة ، فيمنع نفوذ قوة الحس والحركة فيه وفي الأعضاء نفوذاً تاماً من غير انقطاع بالكلية ، ويمنع عن التمكن من القيام ولا يمكن الإنسان أن يبقى معه منتصب القامة، لأن كل تشنج كما نبينه ، فإما عن امتلاء وإما عن يبس ، وإما عن قبض بسبب مؤذٍ ، وكذلك الصرع ، لكنه لا يكون عن اليبوسة ، لأن الصرع يكون دفعة ، والتشنج اليابس لا يكون دفعة ، ولأن الدماغ لا يبلغ الأمر منه يبسه أن يتشنج له أو يعطب البدن قبله ، فيبقى أن سببه إما بقبض الدماغ لدفع شيء مؤذٍ هو إما بخار ، وإما كيفية لاذعة أو رطوبة رديئة الجوهر ، وإما خلط يحدث سدة غير كالمه في بطن الدمغ ، أو أصول منابت العصب ، وقد يكون ذلك من الخلط لحلاكة موجبة تقع في الخلط ، أو لغليان من حرارة مفرطة فيما يقع من السدة ، لا تنفذ قوة الحس والحركة نفوذه الطبيعي ، وبما لا تتم ينفذ منه شيء بمقدار ما ، فلا يعدم الأعضاء قوة الحس وقوة الحركة بالتمام ، وإما لريح غليظة تحبس في منافذ الروح على ما يراه الفيلسوف الأكبر "أرسطاطاليس" ويراه أحد أسباب الصرع، وإذا كان هناك خلط ساد فإن الدماغ مع ذلك أيضاً ينقبض لدفع المؤذي ، مثل ما يعرض للمعدة من الفواق والتهوع ، ومثل ما يعرض من الاختلاج إذا كان الانقباض والانعصار أصلاً في دفع الأعضاء ما تدفعه ، وإذا تقبض الدماغ اختلفت حركاته، وتبعه تقبض العصب في الوجه وغيره ، واختلاف حركاته .

وأما الإفاقة فإما أن تقع لاندفاع الخلط أو لتحلل الريح ، أو لاندفاع المؤذي ، وأما التشنج النازل إلى الأعضاء الذي يصحب الصرع فسببه أن المادة التي تغشي الدماغ، أو الأذى الذي يلحقه يلحق العصب أيضاً ، فتكون حالها حاله ، وذلك لعلل ثلاث : إتباعها لجوهر الدماغ ، وتأذيها بما يتأذى به ، وامتلاؤها من الخلط المندفع إليها في بداياتها ليزداد عرضها وينقص طولها ، وإنما كان الصرع يجري مجرى التشنج ليس مجرى الاسترخاء ، فيفعل انقباضاً من الدماغ ويقلصها ، ولا يفعل استرخاءً وانبساطاً لأن الدماغ يحاول في ذلك دفع شيء عن نفسه .

والدفع إنما يتأتى بالانقباض والانعصار ، وكل تشنج مادي فإنه ينتفع بالحمى والصرع تشنج مادي ، فهو ينتفع بالحمى والأورام إذا ظهرت به ، فربما حلته ونقصت مادته ، وكثيراً ما ينتقل المالنخوليا إلى الصرع ، وكثيراً ما ينتقل الصرع إلى المالنخوليا ، وقد ظن بعض الناس أنه قد يكون من الصرع ما ليس عن مادة ، فإن عنى بهذا أن السبب فيه بخاراً ، وكيفية تضر بالدماغ ، فيفعل فيه التقلص المذكور فلقوله معنى ، وإن عنى أن سبب ذلك هو نفس المزاج الساذج إذا كان في الدماغ فيفعل الصرع ، فذلك ما لا وجه له ، لأن تلك الكيفية إذا كانت قد تكيف بها الدماغ ، وجَبَ أن يكون الصرع ملازماً إياها ، ولا يكون مما يزول في الحال ، بل سبب الصرع هو مما يكون دفعة ويزول في الحال ، بل سبب الصرع هو مما يكون دفعة ويزول في الحال أو يغلب فيقتل ، ومثل ذلك لا يكون كيفية حاصلة في نفس الدماغ ، بل مادة وكيفية تؤدي إليه وتنقطع ، وذلك من عضو آخر لا محالة ، والذي يعرض في الصرع لاضطراب حركة النفس لاختناقه وذلك الاضطراب لاضطراب التشنج ، ويعرض في السكتة للاختناق ولا ستكراه التنفس ، فكأن الصرع تشنج يخص أولاً الدماغ ، والتشنج صرع يخص أولاً عضواً ما ، وكان حركة العطاس حركة صرع خفيف ، وكأن الصرع عطاس كبير قوي ، إلا أن أكثر دفع العطاس إلى جهة المقدم لقوة القوة ، وضعف المادة ، ودفع الصرع إلى أي وجه كان أمكن وأسهل ، ويجب أن يحصل مما قيل : إن الصرع إذا كان في الدماغ نفسه ، فالسبب فيه مادة لا محالة تفعل ريحاً محتبسة في مجاري الحس والحركة ، أو تملأ البطنين المقدمين بعض الملء ، وهذه المادة إما دم غالب وكثير ، وإما بلغم وإما سوداء وإما صفراء ، وهو قليل جداً ، وبعده في القلة الدم الساذج .

وقد قال "بقراط" : أن أكثر الغنم التي تصرع إذا شرح عن أدمغتها وجد فيها رطوبة رديئة منتنة ، وكل سبب للصرع دماغي ، فإنه يستند إلى ضعف الهضم فيه فلا يخلو ، إما أن يكون في جوهر الدماغ ومخيته ، وهو أردأ، وإما إن يكون في أغشيته ، وهو أخف ، والصرع السوداوي أردأ ، وإن كان البلغمي أكثر ، فإن السوداوي أسد لمنافذ الروح ،

والمخصوص عند بعضهم بإسم أم الصبيان ، قاتل جداً ، وإذا اتصلت نوائب الصرع قتل .
والقُوَاق نوع من التشنج ، وإذا عرض للدماغ من مثل هذا السبب تشنج وانقباض، فإنه حينئذ انقباض جميع العصب وتشنجه، وحكى "جالينوس " عن نفسه أنه كان يصيبه القُوَاق عند تناوله الفلافلي ، ثم الشرب للشرب بعده لتأذى فم المعدة بالحدة، وقد شاهدنا قريبا من ذلك لغيره ، وقد حكى " جالينوس" وغيره ، وشاهدنا نحن أيضا بعده إن كثيراً ما كان يحس المصروع بشيء يرتفع من إبهام رجله لريح باردة ، ويأخذ نحو دماغه فإذا وصل إلى قلبه ودماغه صُرع . قال " جالينوس": وكان إذا ربط ساقه برباط قوي قبل النوبة امتنع ذلك ، أو خف ، وقد شاهدنا نحن من هذا الباب أموراً عجيبة ، وقد كوي بعضهم على إبهامه وبعضهم على إصبع آخر ، كان البخار من جهته فبرأ (يذكر محقق القانون في الطب سعيد اللحام : أن بين إبهام القدم والإصبع الذي يليه عصب يتصل بالفقرات العليا والأعصاب الرئيسية في النخاع الشوكي ، ودلك هذا العصب يزيل الصداع ، ولا ريب أن إصابته بآفة يمكن أن يصيب نوعاً من الصرع وكيُّه يقطع صلته مع النخاع الشوكي والدماغ).
ومن هذا الباب الصرع الذي يعرض بسبب الديدان أو حب القرع (الدودة الوحيدة) ، وضربٌ من الصرع مركب بالغشي يكاد الأطباء يخرجونه من باب الصرع ، وهو فيه وضربٌ منه ومن قبيلة يسمى اختناق الرحم ، وهو إن المرأة إذا عرض لها أن احتبس طمثها لا في وقته فاحتقن ، أو احتبس مَنيُها لترك الجماع استحال ذلك في رحمها إلى كيفية سمية ، وكان له حركات وتبخيرات ، فيُعرض إن يرتفع بخارها إلى القلب والدماغ فتصرع المرأة ، وكذلك قد يتفق للرجل أن يجتمع في أوعية المني منه كثير ويتراكم ويبرد ويستحيل إلى كيفية سمية فيصيبه مثل ذلك.
كذلك يتفق للمرأة صرع في الحمل ، فإذا وضعت واستفرغت المادة الرديئة الطمثية زال ذلك ، وقد حُكيَ لنا صرع يبتدئ من العمود الفقري ، وصرع يبتدئ من الكتف وغير ذلك ، وإما أن يكون من المعدة ، ومن المراق وبسبب تخم تورث سُدَدًا في العروق ، فلا تقبل الغذاء المحمود ويفسد فيها الخلط ، أو يبقى فيها الغذاء المحمود مختنقاً للسدد ، فيفسد ، وكثيراً ما يتراجع إلى المعدة فاسداً ، فيفسد الغذاء الجديد المحمود الكيموس ، وكثيراً ما يعرض بسبب ذلك القئ للطعام غير المهضوم ، وعلى كل حال كان الصرع بشركة أو بغير شركة فإن مبدأ الصرع القريب هو الدماغ أو البطن المقدم منه ، والبطون الأخُر معه ، لأن أول آفة يُعتَدُّ بها تقع في حس البصر ، والسمع ، وفي حركات عضل الوجه والجفن ، وإن كان سائر الحواس والأعضاء المتحركة في الآفة ، ولولا المشاركة في الآفة لسائر البطون لما بطل الفهم ، ولما تضرروا في النفس ، والصرع في أكثر الأمر يتقدمه التشنج ثم

يكون من بعده الصرع ، وذلك لأنه إذا استحكم التشنج كان الصرع ، فإذا اندفع السبب المؤذي أو تحلل الريح عادت الأفعال الحسية والحركية ، وربما ظهر الخلط المندفع معاينة في المنخر وفي الحلق ، وكثيراً ما يكون الصرع بلا تشنج محسوس ، وذلك لأن المادة الفاعلة له تكون رقيقة وتفعل بالامتلاء لا بالرداءة الشديدة .

والصرع يصيب الصبيان كثيراً بسبب رطوبتهم ، فربما ظهر بهم أول ما يولدون وقد يكن بعد الترعرع ، فإن أصيب في تدبيرهم زال وإلا بقي ، ويجب أن يجتهد أن يزال عنهم ذلك قبل الإنبات (إنبات الشعر على عانتهم أي البلوغ) ، وأبعد الصبيان من ذلك يعرض له من ناحية رأسه قروح وأورام ويكون سائل المنخرين .

وللدماغ رطوبة في أصل الخلقة من حقها أن تنبثق ، فربما في الرحم ، وربما أنبثقت بعد الولادة ، فإن لم تنبثق لم يكن بدٌّ ، من صرع وأكثر الصرع الذي يصيب الصبيان فإنه قد يخف علاجه ويزول بالبلوغ إذا لم يعنه سوء التدبير وترك العلاج ، والصرع قد يصيب الشبان ، فإن كثر بعد خمس وعشرين سنة لعلة في الدماغ وخاصة في جوهره كان لازماً، ولا يفارق ويكون غاية فعل العلاج فيهم تخفيف من عاديته وأبطأ بنوائبه .

وقد قال "بقراط " : إن الصرع يبقى بهم إلى أن يموتوا ، وأما المشايخ فقلما يصيبهم الصرع السددي ، وقد يعين الأسباب المحركة للصرع أسباب من خارج ، مثل التغذي في المطعم والمشرب والتخم ، ومثل التعرض الكثير للشمس ، مما يجذب من المواد إلى الرأس ، وذلك لما يمنع من إنتشار المواد في جهتي البدن فيحركها إلى فوق .

والجماع الكثير من أسبابه ، ومن أسبابه التنعم والسكون وقلة الرياضة ، ومن أسبابة الرياضة على امتلاء كما تتحرك لها الأخلاط إلى تحلل غير تام ، وتملأ التجاويف ، ومن أسبابه ما يضعف القلب من خوف ، أو وقع هدة وصيحة بغتة ، ومن أسبابه الصوم لصاحب المعدة الضعيفة وشرب الشراب الصرف أيضاً لما يؤذي المعدة ، وهذه أسباب بعيدة توجب الأسباب القريبة ، ونحن نجعل لهذه الأسباب باباً مفرداً ، وقيل : إن المصروع إذا لبس سلاخ غنز كما سلخ وشرع في الماء صرع ، وكذلك إذا دخن بقرن الماعز والمر والحاشي ، وكثيراً ما ينحل الصرع بحميات يقاسيها صاحبه ، وخصوصاً ما طال ، والربع خاصة لشدة طوله ولتكوينه المادة السوداوية حتى ينحل والنافض القوي ، فإن النفض يزعج ما تلحج بالدماغ من الفضول والعرق الذي يتبع النافض ينفضه ، وكما أن السكتة تنحلُّ إلى فالج ، فكذلك كثيرٌ من الصرع ينحل إلى فالج ، وقد زعم بعضهم أن البلغمي يصحبه ارتعاش واضطراب ، لأن البلغم لا يبلغ من كثافته أن يسد المجاري سداً تاماً وأما السوداوي فقد يسد سداً تاماً ، فيعرض منه قلة الإضطراب . وزعم

بعضهم أن الذي يكثر معه الإضطراب فبالحري أن يكون سببه الخلط الأقل مقداراً والأقل نفاذاً في المجاري ، فيجعل الأمر بالعكس ، ولا شيء من القولين بمقطوع به .

قال"روفس": إذا ظهر البرص بنواحي الرأس من المصروع دل على انحلال مادة الصرع ، وعلى البرء ، وكثيراً ما ينحل الصرع إلى فالج ومالنخوليا .

فصل في السكتة :

السكتة تَعَطُّل الأعضاء عن الحِس والحركة لانسداد واقع في بطون الدماغ ، وفي مجاري الروح الحساس والمتحرك ، فإن تعطلت معه آلات الحركة والتنفس أو ضعفت فلم تسهِّل النَّفَس كان هناك زَبَد ، وكان ذا فترات كالاختناق ، أو كالغطيط فهو أصعب يدل عل عجز القوة المحركة لأعضاء النَّفَس ، وأصعبه أن لا يظهر النَّفَس ، ولا الزَّبَد ولا الغطيط ، وإن لم تعظم الآفة في التنفس ونفذ في حلقه ما يُوجَر ، ولم يخرج من الأنف فهو وإن كان أرجى من الآخر ، فليس يخلو من خطر عظيم ، وقد قال "بقراط": إن السكتة إذا كانت قوية لم يبرأ صاحبها وإن كانت ضعيفة لم يسهل برؤه .

ويقول ابن سينا بثاقب علمه " وقد يعرض أن يُسكَت الإنسان – تصيبه سكتة – فلا يفرق بينه وبين الميت، ولا يظهر منه تنفس ولا شيء، ثم إنه يعيش ويسلم.. والنبض يسقط تمام السقوط .. ولذلك استحب أن يُؤَخَّر دفن المُشكِل من الموتى إلى أن تستبين حالته ، ولا أقل من اثنتين وسبعين ساعة .

وقد قال " بُقراط": من عرض له _ وهو صحيح _ وجع بغتة في رأسه ثم أُسكِت فإنه يهلك قبل السابع إلا أن يعرض به حُمَّى ، فيرجى _أيّ الحمى يرجى معها _ أن تنحل الفضلة .

ويضيف : والسكتة قد تنحل في أكثر الأمر إلى فالج كما أنه إذا انبسطت مادة الفالج في الجانبين أحدثت سكتة .

ويفرق ابن سينا بين السكتة والسُّبَات ويقول إن السكتة يتقدمها في أكثر الأوقات صداع وانتفاخ الأوداج ودوار وسدر ، وظلمة البصر واختلاج في البدن كله ، وكسل وثقل .

وما كان من ورم فلا يخلو من حمى ما.. ويكون الوجه مُحمَرًّا والعينان مُحمَرَّتين جداً، وتكون الأوداج وعروق الرقبة ممتدة.

ومن علامات الفرق بين السكتة والسبات أن المسكوت يغط وتدخل نَفَسَه آفة ، والمسبوت ليس كذلك ، والمسبوت يتدرج من النوم الثقيل إلى السبات ، والمسبوت يعرض ذلك له دفعة .

وعن أمراض العصب يقول ابن سينا أنها تعرض له أصناف الأمراض الثلاثة، ويعني بها المزاجية والآلية وانحلال الفرد المشترك وتظهر الآفة في أفعاله الطبيعية والحاسة والمحركة.

وعن الفالج يميز ابن سينا بين ما يكون لأحد شقي البدن طولاً، ويكون الوجه والرأس معه صحيحاً، وما يسري في

جميع الشِقّ من الرأس إلى القدم، وإذا عمَّ الفالج الرأس كان سكتة.. ونتيجة ذلك بطلان الحس والحركة .

وقد يكون "من انضغاط شديد كما يعرض عند ضربة أو سقطة ، وإذا مالت الفقرات وانكسرت فتضغط العصب الخارج منها الذي يكون يمنة أو يسرة ، لأن مخارج العصب ليست من جهتي قدام وخلف .

أما ما يكون عن ورم فذلك يعرض في منابت الأعصاب وشُعَبها.

أما القطع الذي يعرض للعصب فما كان طولا ، فلا يضر الحس والحركة ، وما كان عرضاً فيمنع الحس والحركة من الأعضاء التي كانت تستقي من المجاري المتصلة بينه وبين الليف المقطوع .

ويقول إن النخاع مثل الدماغ في أنقسامه إلى قسمين ، وإن كان الحس لا يميزه ، وكيف لا يكون كذلك وهو ينبت أيضاً عن قسمي الدماغ ، فلا يستعبد أن تحفظ الطبيعة إحدى شِقيه .

ويقول: إنه إذا كانت الآفة التي تفعل الفالج في شِقّ من بطون الدماغ، عم شِقّ البدن كله وشق الوجه معه.

فإن كانت عند منيت النخاع كان البدن كله مفلوجاً دون أعضاء الوجه ، وربما وقع مع ذلك خدر في جلدة الرأس لأن جلدة الرأس يأتيها العصب الحاسُّ من العنق ... وإن كان من العصب استرخى ما يخص ذلك العصب . ومن الفالج .. كثيراً ما يبقى معه الحس دون الحركة .

ويذكر ابن سينا "إن من الفالج ما يكون بُحرَاناً للقولنج "، وكثيراً ما يبقى معه الحس ، لأن المادة تكون معه في أعصاب الحركة دون الحس .

وكثيراً ما يبقى معه الحس، لأن المادة تكون معه في أعصاب الحركة دون الحس. ويدلّل ابن سينا على ذلك بقوله :" وذكر بعض الأولين أن القولنج عمم بعض السنين ، فقتل الأكثر ومن نجا نجا بفالج مزمن أصابه كأن الطبيعة نفضت تلك المادة التي كانت تأتي الأمعاء وردتها إلى خارج ، وكانت أغلظ من أن تنفذ بالعرق فلجحت في الأعصاب وفعلت الفالج .

ويقول:إن أكثر ما يعرض الفالج يعرض في شدة برد الشتاء.

ونبض المفلوج ضعيف بطيء متفاوت .. والبول قد يكون فيه على الأكثر بياضاً، وربما أحمر جداً لضعف الكبد عن تمييز الدم عن المائية، أو ضعف العروق عن جذب الدم.

وقد يعرض أن يكون الشق السليم من الفالج مشتعلاً كله في نار ، والآخر المفلوج بارداً كأنه ثلج ، ويكون نبض الشِقين مختلفاً ، فيكون نبض الشق البارد ساقطاً . وما كان من الأعضاء المسترخية والمفلوجة... ليس يصغر ولا يضمر فهو أرجى مما يخالفه.

والفالج الحادث عن زوال الفقار قابل في الأكثر (للشفاء) والذي عن صدمة لم يدق العصب دقاً شديداً ، فقد يبرأ أفرط لم يرج إن يبرأ والذي يرجى منه يجب أن يُبدأ فيه بالفصد .

وإن كان (الفالج) عن التواء أو سقطة أو ضربة أو قطع، فالسبب يدل عليه، وربما

خَفِيَ السبب في القطع إذا كان العصب غائراً فيدل عليه أنه يقع دفعة ولا ينفعه تدبير.
أما الذي يقبل العلاج فهو ما ليس عن قطع، بل ما ورم ونحوه.

المعالجات:
يقول ابن سينا إنه " في أمراض العصب الخمسة ، أعني الخَدَر ، والتشنج والرعشة والفالج والاختلاج " يكون الفصد في مؤخر الدماغ . أما تدبير غذائهم فإنه يجب أن تقتصر بالمفلوج في أول ما يظهر على مثل ماء الشعير وماء العسل ... واجتَهد في تجويعه وإطعامه الأغذية اليابسة ، ثم تعطشه تعطيشاً طويلا .
والماء خير لهم من الشراب، فإن الشراب ربما تَحَمَّضَ في أبدانهم فصار خَلاً، والخل أضرُّ الأشياء بالعصب.
وفي علاج الفالج يقول إن وضع الأدوية على العضو المفلوج نفسه، فمما لا ينفع نفعاً يُعتَدُّ به، وعليك بمنابت الأعصاب.. وعلى المبدأ الذي يخرج منه العصب المتجه إلى العضو المفلوج .
أمَّا (الفالج) الكائن عن القطع فلا علاج له البتة.

في التشنج :
يُعَرِّف ابن سينا التشنج بأنه " علة عصبية تتحرك لها العضل إلى مباديها (بداياتها) فتعصي في الانبساط ، فمنها ما تبقى على حالها فلا تنبسط ومنها ما يسهل عودة إلى الانبساط كالتثاؤب والفواق .

وحسب نظرية الأخلاط الأربعة يعيد ابن سينا أسباب التشنج إلى مادة بلغميه أو سوداوية أو دموية.
ويقول : إنه إذا كان التشنج بلا ورم كانت المادة الفاعلة له مشتملة على العضل كله ، وأما أن تكون حاصلة في موضع واحد ، ويتبعها سائر الأجزاء ، كما تكون عن التشنج الكائن للورم عن مادة منصبة لضربة أو لقطع ، أو لسبب الآخر من أسباب الورم .
ويضيف: أرى أنه (التشنج) مما يعرض (يحدث) كثيراً ويزول في الوقت، وقد يعرض كثيراً.. عقيب الخوانيق وعقيب ذات الجنب وعقيب السرسام .
كما يحدث التشنج – كما يقول ابن سينا _ عن مادة سمية تتأذى إلى الدماغ والعصب، كما تعرض لمن لسعته العقرب على عصبه، وإما عن مادة غير سمية مثل ما يعرض للعصب من برد شديد يجمع العصب والعضل ويكتفه فيتقلص إلى رأسه.
ومن التشنج ما يعرض للمرضعات بمجاورة الثدي .. ومنه ما يعرض للسُكارى .. وللصبيان لرطوبتهم ، وكثيراً ما يعرض لهم في حُمَّيَاتهم الحادة ، وعند اعتقال بطونهم وفي سهرهم وكثرة بكائهم.
وبالجملة فإن الصبيان يسهل وقوعهم في التشنج.. ويسهل خروجهم عنه ، أما البالغون فلا يسهل أحد الأمرين فيهم .
وأما من جاوز سبع سنين فلا يتشنج إلا لحمى صعبة جداً ، ومن التشنج ما يعرض للخوف ، أو بسبب الإعتماد على بعض

الأعضاء وهو منقبض ، وربما عن ضربة أو حمل ثقيل أو نوم على مهاد صلب ، وهذا مما يزول بنفسه .
ويقول: إن التشنج الناتج عن الحُميَات ليس بذلك الصعب الرديء ، إنما الصعب الرديء ما كان في الحُمْيَات المُحْرقة ْ والسرسام الذي يجفف العصب والعضل ويشوي الدماغ ، وما كان في الحميات المزمنة .
كما يقع (التشنج) لمن شرب الأدوية المُخدِّرة كالأفيون.
كما يمكن أن يقع التشنج قبل وبعد الإسهال، وبعد ألقيء أو لمشاركة الدماغ الرَّحم في أمراضها والمثانة، وقد يقع بسبب الديدان.
ومن التشنج الرديء ما كان خاصاً في الشفة والجفن واللسان، فيعلم أن سببه من الدماغ نفسه.. وربما اشتد التشنج حتى يلتوي العنق ، وتصطك الأسنان . ويقول: إن "كل من مات من التشنج مات وندته بعدُ حار " ، وذلك مما يقتل بالخنق ، لأن عضل التنفس تتشنج وتبطل حركتها . وكل تشنج يتبع جراحة فهو قتال وهو من علامات الموت في أكثر الأمر.
وفي علامات التشنج يقول : إن نبض المتشنجين مُتَمَدِّد في الموضع يصعد وينزل كسهام تنقلب من قوس رام ، وتختلف حركات نقراته في السرعة والبطء ، ومن الدلائل الدالة على حدوث التشنج إحمرار الوجه ويظهر بالعينين حَول ومَيَلان ، وفي التنفس انقطاع وانبهار ، وربما عرض ضحك لا على أصل وتعتقل الطبيعة .. والبول أيضاً كثيراً ما يحتبس ويعرض لهم فُوَاق وسَهَر وصُداع ورعشة ووجع تحت مفصل العنق بين الكتفين وعند مفصل القطن والعصعص .
وفي العلاج يصف ابن سينا كما هو متوقَّع كل ما يعيد التوازن إلى الجسم حسب نظرية الأخلاط ْ .

في الرعشة :

يعزو ابن سينا الرعشة إلى " اختلاط حركات إرادية بحركات غير إرادية " وهي آفة في القوة المحركة، كما أن الخدر آفة في (القوة) الحسَّاسة.
ويضرب أمثلة على حدوث الرعشة فإنها تحدث في حالات الخوف أو مخاطبة محتشم مهيب.. أو غمٍّ أو فرح مشوش لنظام حركات القوة... والغضب قد يفعل ذلك، كما تحدث من السكر المتواتر وكثرة شرب الماء البارد أو شربه في غير وقته.
والرعشة ربما كانت في جميع الأعضاء وربما في اليدين أو الرأس وحده.
والرعشة في المشايخ لا تزول بعلاج .
وفي معالجة الرعشة ينصح ابن سينا بالإستحمام بمياه الحَمَّات .

في الخدر :

يُعرِّف ابن سينا الخدر بأنه علة آلية تحدث للحس اللمسي آفة ، وإما بطلاناً وإما نقصاناً مع رعشة .. وقد يوجد أحياناً خدر بلا عسر حركة لاختلاف عصب الحركة والحس.

وسبب الخدر قد يكون في الحُمَّيَات القوية والحادة... أو لبرد شديد أو لسع حيوان كالعقرب المائي أو مسّ الرعّادة المُسمَّى نارقاً ، أو شرب دواء كالأفيون .. أو لسدد من ضغط ورم أو ضغط شد ورباط أو ضغط يلوي العصب.

والخدر غالباً ينذر بسكتة أو صرع أو تشنج أو كزّاز أو فالج عام ، وخدر كل عضو إذا دام واشتد ينذر بفالج أو تشنج يصيبه .

وخدر الوجه ينذر باللقوة ، وكثيراً ما يعقب ذات الرئة وذات الجنب والسرسام البارد خدر.

وعلاج الخدر يكون بالفصد إذا قامت دلالة من امتلاء العروق وإنتفاخ الأوداج ونوم وحمرةِ وجهٍ وعين .

ومن المعالجات النافعة للخدر رياضة ذلك العضو ودوام تحريكه .

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الخلاصة. وجود علاقة واضحة بين العوامل النفسية والإجتماعية ومرض السكري. ولذا فإن التقييم النفسي والإجتماعي ينبغي أن يكون جزءاً رئيسياً من التقييم العـــام . كمـــا أن التدخل النفسي والإجتماعي يعتبر أمرا هاماً إذا ماستدعى الوضع ذلك.

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mal school life for diabetic patients of all ages [2].

Full and correct information should be provided to the school personnel at diagnosis and to be repeated when the child move to another school [2] to facilitate the appropriate care of students with diabetes, school and day care personnel must have an understanding of diabetes and must be trained in its management and in the treatment of diabetes emergencies [9].

The diabetes health care plan in the school and day care setting should address the specific needs of the children and provide specific instructions for each of the following:

1- Blood glucose monitoring, including the frequency and circumstances requiring testing.
2- Insulin administration (if necessary) including doses, injection times and storage of insulin.
3- Meals and snacks including food contents, amount and timing.
4- Symptoms and management of hypoglycemia
5- Symptoms and management of hyperglycemia
6- Testing for ketones and appropriate action to take for abnormal ketones level [9].

Conclusion :

Links between psychosocial factors and diabetes do exist. The psychosocial components of assessment and management should be considered early rather than being kept until the end.

الخلاصه:

الهدف. مراجعة الجوانب النفسية والإجتماعية لمرض السكري لدى الأطفال والمراهقين.

طريقة البحث. مراجعة المقالات المنشورة المتوفرة والمناسبة لهذا البحث من عام 1990 وحتى 2005.

النتائج. تشير معظم الدراسات إلى دور العوامل النفسية والإجتماعية في توقيت ظهور السكري وإزدياد شدته وتعقيد معالجته.كما أن الاصابة بالسكري يمكن أن يؤثر سلباً على الطفل المصاب بل وعلى والديه واخوته. وعند معالجة السكري لابد من الإلتفات لتقييم الطفل أو المراهق نفسيا وإجتماعيا مع التركيز على الجوانب التعليمية لكل ما يخص السكري وطرق التعايش معه كما ينبغي أن تشمل المعالجة العلاج النفسي الفردي والإرشاد الأسري ومعالجة الإضطرابات النفسية المصاحبة وما يتعلق بالتحصيل الدراسي اذا ماستدعى الأمر أياً منها.

chronic disease. Successfully, parents can be educated on diabetes management. It is important that family education involve not only the mother, but also the father and if possibly any other relative who lives with the child [2].

Family education should include the following components:

1- Establishment of a cooperative relationship amongst family and the diabetes team.
2- Emphasis on the promotion of problem solving strategies, and
3- Emotional and psychological support [2].

Managing Psychiatric Co-morbid ity.

Glycemic control can be improved by appropriate treatment of the co morbid psychiatric disorders [64-65].

Treatment of co morbid Psychiatric disorders with medications requires careful monitoring because most antidepressant, mood stabilizers, and atypical antipsychotic may stimulate appetite or affect glucose tolerance, inducing hypoglycemia or hyperglycemia. Beta blockers should be avoided as they mask the early signs of hypoglycemia [3,66].

Care at School

Children with chronic condition like diabetes are at increased risk for school absenteeism due to the physical and psychological consequences of their illness [49, 67].

Increased school absenteeism is associated with more critical illness, including those resulting in activity restrictions and poor psychological adjusted to the condition [49, 67].

Children with diabetes missed approximately 3 more days of school than their siblings and peers. Near normal school attendance therefore is a reasonable expectation for children with diabetes [49].

Diabetes can generates fright and anxiety in teachers and pupils, leading to pity attitudes towards diabetic children [2].

Studies have shown that the majority of school personnel have an in adequate understanding of diabetes and that parents of children with diabetes lack confidence in their teacher's ability to manage diabetes effectively [9,68].

Children in the school and day care setting face discrimination. For example, some day care centers may refuse admission to children with diabetes and children in the classroom may not be provided the assistance necessary to monitor blood glucose and may be prohibited from eating needed snacks.[9].

The diabetic team should assume the maximum effort to allow nor-

Diabetic Education

Adequate knowledge of diabetes predicts a better psychosocial adjustment [1].

Parents and Children should be encouraged to think that diabetes is quite compatible with normal active life [1]. The aim of education is to provide individuals with diabetes and their families with knowledge and skills to perform self care [2].

Education is Comprehensive and mandatory in the following areas:

- Early and repeated full explanation of the illness and its complications[1].
- medical and dietary therapy [2]
- Scheduled and free activities [2]
- Appropriate therapeutic adjustment to daily factors to achieve or maintain optimal glucose control [2]

  Advice on insulin management should include discussion of ways in which children can gradually become responsible for their own injections and urine and blood testing, certainly as they move into their early teens and usually earlier [2].

Education at diabetes camps:

The mission of camps specialized for children and youth with diabetes is to allow for a camping experience in safe environment and to enable children with diabetes to meet and share their experiences with one another while they learn to be more personally responsible for their disease. For this to achieve a skilled medical and camping staff must be available to ensure optimal safety and an integrated camping and educational experience [57].

It is an ideal place for teaching diabetes self-management skill e.g., insulin injection techniques and blood glucose monitoring [57].

Individual Psychotherapy:

Individual Psychotherapy for children with poor diabetic control has been found helpful [1, 58]. Cognitive behavioral psychotherapy [59], behavioral therapy [1, 60] and psychoanalytically based individual psychotherapy [61] have been found to be useful.

Family Counseling

Good results have been found for family therapy in treatment of children with poor diabetic control [62].

A well functioning family can facilitate a child's well-being by providing emotional support, advice and practical help [4].

Family involvement in type 1 diabetes is necessary and inevitable [63]. Initially, the treating team has to overcome the parent's resistance due to sense of guilt, ignorance about the existence of diabetes in children or no acceptance of

Children with diabetes show an increase rate of learning problems [1].
Reading difficulties have been noted occurring mainly in children who were younger at the time of diabetic onset [44, 48].

Siblings

Siblings can be relatively neglected by parents and health care workers.
Reactions of jealousy, over protectiveness and survival guilt are common [10].
It is possible that the impact of diabetes management on the family's day to day functioning may cause siblings of children with diabetes to miss more school days than do their classmates who do not have a sibling with chronic condition [20,49].

Families

The period immediately following a diagnosis is an anxious and distressing time for the whole family [50 - 52]. Parents often find it difficult to come to term with the diagnosis which they may view as the end of normal health and familiar life style[50, 53]. When the child is diagnosed with diabetes, parents experience shock, distress, grief response similar to that normally associated with bereavement [52 - 55].
Family reactions to an ill child are complex and dependent on prior family patterns, coping style and experiences with illness.
Reactions of fear, anger, loneliness and guilt seem universal.
Marital strain seems inevitable. Some researches have found greater rate of divorce and depressive symptoms in mothers of children with chronic illness like diabetes. [10].

Psychosocial Aspects of Management

The goal of medical care should not only aim to alleviate the physical complications of diabetes but also to improve the patients overall quality of life [42, 56].

Assessment

The Psychosocial Components of the diagnostic process should get equal billing with physical and laboratory components rather than withheld as a last resort [10]. Assessment may be carried out by pediatrician, social worker, psychologist or psychiatrist[1]. It should involve a whole family interview, interview with parents and interview with the child or teenager [1].
The focus should mainly be on aspects of family life other than the diabetes and its control.
A home visit especially in families where co-operation is poor will also often be helpful [1].

mote weight loss among young females with diabetes [29].

Given that there is minimal CNS involvement, neuronal dysfunction shall not be considered a major risk factor for psychopathology [16].

Hospitalized group was reported to have lower self-esteem and social competence [41].

A study conducted on Kuwaiti adolescents with diabetes showed no difference in number of close friends or frequency of meeting them, number of hours spent on recreational activity, school performance compared with non-diabetic adolescents [15] people with diabetes often feel challenged by the demand of day to day management of the disease and the fear of developing serious complications. Therefore, the quality of life which includes personal perception of health and satisfaction with life is substantially effected [42].

Children who were at higher risk of developing psychiatric co morbidity were those in the 1st year after diagnosis and those who had preexisting anxiety or mothers with psychopathology [43].

Learning Disabilities

Children with diabetes function lie within the same range of intelligence as the general population but more subtle cognitive deficit may be present. Children with the onset of diabetes early in life, before 5 or 7 years of age have been found in some cases to perform lower on overall intelligence [44-46].

Cognitive difficulties in abstract/visual reasoning subscales of the Stanford Binet Intelligence Scale have been noted in children with relatively mild hyperglycemia if the onset of their illness occurred before 5 years of age. Similarly, problems in cognitive processing speed acquisition of new knowledge and conceptual reasoning ability has been documented 2 years after diagnosis. These problems may be the result of persistent hyperglycemia [46].

It has been noted that children with early onset diabetes tend to have spatial difficulties, whereas children with later onset have difficulties with verbal abilities, school achievement and psychomotor skills. These differences in new psychological symptoms may be related to age and associated risk factors, such as hypoglycemic episodes in children with early disease onset and hyperglycemia and social burden for children with later onset of diabetes [44, 47].

Psychosocial Disturbances associated with Diabetes Mellitus.

Diabetic Individuals

Although some studies found no significant pathology (Psychiatric illness or family dysfunction) in children with diabetes or their families [20,29], several studies show that diabetic individuals have high rates of psychiatric disorders compared to individual without diabetes [20,30,31].

There have been found higher prevalence rates of depression, anxiety and disruptive behavioral disorders in people with diabetes compared with the general population [31-34]. The prevalence of depression or depressive symptoms in children and adolescents with diabetes ranges from 2 to 3 times that of peer without diabetes [35-36].

It has been reported that the prevalence of depressive symptoms in young people with diabetes was 12% children age 8-12 years and 18% in adolescents [37].

Poor diabetic control might cause or exacerbate depression via direct effects on brain functions or indirectly through complications, functional impairment or decreased quality of life [38].

A special risk in depressed diabetic individuals is attempted suicide by method of insulin overdose [16].

It has been reported relatively high level of anxiety in children related to their fear of hypoglycemic and hyperglycemic coma as well as later vascular complications [16].

Problems in adjustment to the diagnosis of diabetes in children have also been reported [16].

Some moodiness and feeling of isolation and of loss or grief as well as mild anxiety about future are to be expected as normal response to diabetes [30]

Repeated episodes of severe diabetic ketoacidosis (DKA) would be stressful [20]. The specific psychiatric disorders associated with DKA were anxiety, depression and attention / disruptive behavioral disorders. Social phobia, simple phobia and separation anxiety disorder are important as they have mutually influencing relationship with episodes of repeated illness and subsequent hospitalization [20].

Eating disorders are relatively common. A study of 91 adolescent girls, age 12-18 years followed for 5 years, found highly or moderately eating disorders in 29% at baseline and 37% at follow-up [40]. When Diabetes and eating disorder and combined, treatment is more difficult. Insulin abuse is common to pro-

fects very early parents child relationship because of its usual later onset. Risk for relation-related disorders should not be elevated [16].

Preschoolers: Preschoolers are verbal and increasingly active. Chronic illness and repeated hospitalization cause restriction of activities and may impair early needed socialization experience [10]. Because of the preschooler's egocentrism, illness may be psychologically associated with a sense of wrong doing or punishment. Parents at the same time may become confused as to the degree of strictness that is required.

Misbehavior which is normally emerges in this stage may become extreme or the wish to be naughty may be overly controlled in the perfect child. In both cases the normal, balanced internalization of family rules has been interrupted [10].

Latency age: Latency age children can employ with causal thinking, and are more observant of their own bodily reactions. Therefore, they can understand their illness in some logical detail. Dramatic misconception can still exist if professionals are not careful to use developmentally appropriate words.

If the illness interferes with school performance or is disruptive of normal activities, it can damage self esteem [10].

Adolescence: Adolescence can be a difficult time for those with diabetes. The rise in the incidence of poor metabolic control may be partially attributed to some of the hormonal changes occurring during this development period [15]. Diabetes can have impact on their struggle for autonomy, their physical-sexuality development and their peer relationship [10, 24]

Compliance problems often result when the illness and its treatment become involved in a teenager's conflictual struggle for independence and personal identity [10, 25].

It is also influenced by their self-efficiency [4].

If diabetes started during adolescence, the compliance can be more difficult than if it started during early childhood, because the adolescent is not mature enough to take responsibility for his diabetes but finds it hard to let parents do it [26, 27].

However, having type 1 diabetes does not heighten the usual developmental conflicts of adolescents with their parents overall [28].

and to quality of child's dietary intake [17]. However, adherence and dietary intake may also be related to family variables. Higher level of family stress and lower level of resources were significantly associated with poor metabolic control [18]

Studies of poorly controlled diabetic children show high divorce rates in the parents, frequent family conflict, living with one parent family or in a family with the father had low level of education and generally inadequate parental Care [1, 19].

Ketoacidosis was linked to either dietary indiscretion or insulin mismanagement in only a minority of cases (5-10%). However, in majority of these cases family difficulties were felt to be associated with less adequate control. [16].

There was a strong association (OR 2.2) between hospital admission rates with poor diabetic control and the absence of one or both parents from home [12]. Younger age, single parent family and poor glycemic control were associated with more days of rehospitalization [19].

Psychiatric Disorder and Diabetes

Diabetic individuals with poor metabolic control have been found to have a higher rate of psychiatric disorders than diabetic subjects with adequate control [20-21].

The Presence of Psychiatric co morbidity can result in difficult clinical course, because it may affect adherence to medication and self care regimens [6].

Depression has been shown to decrease adherence to diabetic diet and predict drop out from weight loss programs and to be associated with diabetic complications [22-23].

Emotional reactions such as anxiety and depression directly affect the secretions of various hormones that regulate glucose and fat metabolism[15].

The Influence of Diabetes on different development level.

The child level of psychosocial development will influence reactions to a new illness or the progression of chronic disorder like diabetes mellitus.

Infancy: In infancy, severe illness may produce intense emotional reactions in new parents at the very time they are trying to become a family.

Predictable guilt, horror, disbelief associated with illness in an infant can damage parental child attachment leading to chronic dysfunction[10]. However, diabetes rarely af-

spective evidence that children who have had adverse experiences in the 1st two year of life are prone to develop diabetes later on. [11].

There have also been suggestions that external stress factors, either of a non-specific nature or specifically related to loss of a parent, might precipitate the illness. It does seem established that there is an excess of life events in the few months preceding the onset of the condition, particularly in older children [1].

Some reports have found a stronger association with increasing social advantages, although other have reported an increased risk with mothers having a lower educational level, with fathers in manual occupations and with increasing level of deprivation and yet others have found no association with social class [12-14].

Diabetes Control:

Although the majority of children with diabetes achieve very satisfactory control with minimum inconvenience to themselves and their families, a minority present control problems and, in a smaller proportion still, these difficulties produce a major disruption in the child's life, requiring frequent out-patient visits, hospitalizations, and absence from school [1] Such brittle or labile cases of diabetes may be due to specific metabolic or endocrine factors, but they are more likely to be due to the direct or indirect influence of stressful factors operating on the child :

Direct factors: Laboratory studies have demonstrated that the production of pituitary hormones and catecholamine induced by stress can lead to decrease in insulin production and an increase in free fatty acids in the blood [1-15].

Indirect factors: The success of diabetic control depends on the compliance of the patient and family with treatment, diet, injections and urine-testing. Compliance can be affected by the quality of organization of care in the home. It is likely to be poor in those families where, for reasons of financial hardship or personality conflicts between the parents or a combination of the two, the home care is relatively disorganized [1, 10,16]. The confidence and self-esteem of the parents are important [1]. Some parents react to their own anxiety about the diabetes by becoming more over controlling and rigid or rejecting and neglectful [1]

Glycemic control is most strongly related to adherence to treatment

Introduction :

Definition:

Diabetes Mellitus is a condition in which there is a failure of production of insulin, the hormone responsible for clearing glucose and ketones from the blood. This type is called juvenile onset-diabetes or type I diabetes. The patient is dependent on insulin for survival. The onset of this condition is usually between 3 and 15 years of life, but it may occur later[1]. Type 1 diabetes represents 80-85% of all cases of diabetes in children and adolescents[2]. It must be treated with exogenous insulin [3]. In maturity onset diabetes, which usually comes on much later in life, a different pathological mechanism, independent of insulin production is involved [1].

Background Features:

Diabetes Mellitus ranks as the 2nd most common chronic disease of childhood. Its prevalence in the school-aged population is exceeded only by asthma [2,4,5].

It is considered one of the most psychologically demanding of chronic medical illness. It is a life – altering condition for children, adolescents and their families. It is a stressful chronic illness with life-threatening aspects, demands for Life style modifications and complex treatment requirements which are all threats to personal control[4,6,7].

The world incidence of type 1 diabetes varies greatly from 2 per 100,000 per year in Japan to 35 per 100,000 in some Scandinavian countries. The European incidence also varies greatly from 5 per 100,000 per year in Turkey to 18 per 100,000 per year in United Kingdom and 25.8 in Sweden.

The prevalence of diabetes in those younger than age 20 is about 1.7 per 1000 in the United States [3,5,8,9].

Links with Psychosocial Factors :

Psychosocial factors and etiology of diabetes

Various people have reported a possible association between the etiology of childhood diabetes and psychosocial factors. They may precipitate the onset of a disorder or may influence the timing of symptom presentation [10]. It has been suggested that psychological stress could trigger the onset of diabetes mellitus, given the ability of stress in normal control subjects to stimulate hormones that oppose insulin's action but this relationship remains uncertain [10]. There is weak retro-

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Review Article

Psychosocial Aspects of Diabetes Mellitus in Children and Adolescents:

Fatima Al-Haidar

الجوانب النفسية والإجتماعية لمرض السكري لدى الأطفال والمراهقين مقالة نقدية

فاطمة الحيدر

Abstract

Objective: To review the links between psychosocial factors and diabetes mellitus in children and adolescents, trying to look for these factors whether they play a role in the etiology of diabetes or not, and whether there are psychosocial consequences of diabetes or not.

Method: Reviewing the available and relevant published articles, from 1990 to 2005

Results: Psychosocial factors could precipitate the onset of diabetes and may influence the timing of symptoms presentation.

Stress and family problems may affect children and adolescents compliance with diabetic regimen leading to more medical, psychological and social complications. Presence of psychiatric co morbidity can result in difficult clinical course of diabetes.

Diabetes can affect children negatively at different phases of development from infancy to adolescence. Diabetic children have high rates of depression, anxiety, adjustment disorders and disruptive behavioral disorders.

Their siblings and families are affected negatively as well.

Management should include psychosocial assessment of diabetic children and adolescents and their families, diabetic education, individual psychotherapy, family counseling, management of psychiatric co morbidity and issues related to school attendance and achievement.

Conclusion: Links between psychosocial factors and diabetes do exist. The psychosocial components should be considered early in the process of assessment, management and subsequent rehabilitation.

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Conclusion

In summary, the results of this study suggest a potential role for physical trauma in the etiology of medically unexplained neurological symptoms. This idea is not new but had fallen into obscurity during the 20th century. These findings emphasize the need to review the current DSM-IV [2] and ICD-10 [18] diagnostic criteria of conversion disorder.

الملخص

أهداف الدراسة: تهدف هذه الدراسةِ إلى التدقيق في الأدلة ِ الحاليةِ للعوامل المرسبة لظهور إضطرابات الهراع (الهستيريا) التحويلية (الحركية و الحسية) كغيرها من الأمراض غير معروفة الأسباب.

طرق البحث: لقد تمت هذه الدراسة بواسطة التحليل المنهجي المنظم لكل الدراسات السابقة منذ عام 1965 بفحص نسبة العوامل المحفزة لظهور هذه الأمراض كالإصابات الجسدية و الضغوطات النفسية الحادة و إضطراب الهلع و الإمراض العضوية و الجراحية.

النتائج: تبين بأن الإصابات الجسدية الاكثر شيوعا كعامل محفز لظهور مثل هذه الإضطرابات حيث بلغت نسبتها6. 31% مقارنة مع الضغوطات النفسية الحادة حيث بلغت نسبتها 25.4%.

الخلاصة: تفيد الدراسة بأن الاصابات الجسدية هي الأكثر شيوعا كعوامل محفزة لهذا الإضطراب النفسي إلا أن هذه الأستنتاجات تجريبية و تحتاج إلى المزيد من الدراسات المستقبلية حيث أن الإصابات الجسدية غالبا ما يصاحبها أعراض الخوف و القلق النفسي.

إن نتائج هذه الدراسة لها إنعكاسات على المعايير التشخيصية لهذا المرض في نظام التصنيف العالمي ICD10 وكذلك الأمريكي DSMIV والذي يصر على الضغوطات النفسية كمعيار تشخيصي دون التطرق الي العوامل الأخرى كالأصابات الجسدية مثلاً.

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injuries that developed from slight shocks and mild blows inflicted directly upon the back. He introduced the notion of invisible damage at the molecular level, and gave credence to the notion that the injury did not have to be visible to be compensable. In fact, although he held organic view he was struck by the disproportion that occurred between an apparently minor accident and the severe consequences.

This debate goes in line with scientific evidence and clinical experience, which suggests that an exclusively psychological paradigm is viewed negatively by patients, some of whom are actively hostile to it. The emerging evidence base documenting biological factors in the development of MUS, recently reviewed by Clauw and Chrousos [12], suggests that some of patients' concerns may be justified. Indeed the father of psychoanalysis himself (Freud) did not deny the pathophysiological correlate of MUS and acknowledged that certain types of MUS may be caused by organic factors. He stated-

"I will take the word functional or dynamic lesion in its proper sense: "alteration in function of mechanism". Such an alteration, for example, would be a diminution in excitability or in a physiological quality which in the normal state remains constant or varies within fixed limits.....[15]. On addition, Trimble wrote: -

"Thus it will be recalled that originally the term 'functional disorder' used in association with the concept of organic disorder, implied disease attributed to altered function, rather than structure, of neuronal processes" [15].

Accordingly, there is a considerable body of evidence in the literature, and from clinical practice which notes an excess in physical attribution for MUS. If this has occurred in our sample, we may have overestimated the importance of physical trauma (recall bias).

Finally, it is difficult to comment on how representative this sample was. Many were case reports and few were from consecutive case series. It is likely that the sample contains significant heterogeneity and is prone to bias, which cannot be foreseen. The findings of this review need to be considered cautiously owing to the many confounding factors possible. Nonetheless, it seems desirable to emphasize the need for better-designed studies that aim to examine this interaction between physical and emotional factors.

of conversion disorder is a major confounding factor in the present results. Few of the studies actually used validated measures for examining life events. There are problems with extracting data from studies that do not specify the nature of the life events that they have reported. Further, even clearly described data needs careful analysis; shrapnel wounds sustained in a war or road traffic accident injuries may be interpreted as both physical trauma and stressful life events.

Taking history from patients with such symptoms, it must always include a search for physical trauma, however minor, along with details of their state of mind at onset. In the golden age of hysteria research, just before the ascendance of psychoanalysis, the role of physical trauma was recognized and MUS are accepted as reversible functional disturbances of the nervous system. The accepted treatment included attention to psychological aspects of medical management. Thus, Jelliffe commented: -

"I talk of "reversible" and "irreversible" organic changes, never of organic changes. Organic changes are always taking place in all functioning organs. Anyone should know that, whether within the physiological range, or whether reversible or not is the important consideration'' [15]. The authors of the various papers studied were from differing disciplines, including general medicine, neurology, orthopedic, dental surgery, rehabilitation, physiotherapy, and psychiatry. It may be that the histories taken by each would be different. Given that most diagnostic criteria stipulate psychological factors, it is possible that many investigators have simply not looked for or asked about minor or trivial physical trauma leading to an underestimation of its true prevalence. Equally, many non-psychiatrists are unaware of operationalised criteria in their practice, which may be completely uninfluenced by them or even lead to an under emphasis on psychological factors.

The present findings and suggestions are not new. The prevailing model through history has been toward the organic paradigms of the MUS, from uterine theories through "wondering womb" to irritated toxins and vapors, reflex theory, animal spirits, physical trauma and traumatic nervous disorder to functional nervous shift. John Eric Erichsen introduced the term "spinal concussion" describing railways

severity of the physical injury was less significant than the suddenness of the event, pointing out that MUS could follow a minor physical injury or emotional problems [6]). He also noted the importance of fear, "The nervous shock or commotion" that accompanied the event. Thus despite emphasizing the role of physical trauma, he appears to have believed that fear "panic" associated with the trauma was the potentially pathogenic feature. Furthermore, Herbert W. Page stressed the importance of fear "panic" in precipitating MUS following minor or trivial trauma in railway accidents, calling them "nervous mimicry" which allowed a functional and curable disorder to mimic an incurable structural lesion. He stated-

"the suddenness of the accident, which comes without warning, or with a warning which only reveals the utter helplessness of the traveler, the loud noise, the hopeless confusion, the crises of those who are injured; these in themselves and more especially if they occur at night or in the dark are surely adequate to produce a profound impression on the nervous system" [15].

In this study 18 cases (2.1%), reported panic symptoms just before the onset of Medically Unexplained Paresis and Anesthesia. This percentage is low in comparison to other stressful life events, which stands at 25.4% (214 patients).

Our findings must be interpreted in the knowledge of limitations to the study. Slater [16-17] famously stated in a follow-up study that hysteria had been misdiagnosed in over 60% of cases. However, more recent studies, which are of better quality but less strident authorship, have demonstrated rates of 5-10%. More recently, Carson [18] has shown that current rates are extremely low, around 5%. He criticized Slater's research on hysteria that it was, by modern standards, a fatally flawed study. The lack of any real definitions for entry criteria, sampling frames or outcome measures make it too difficult to interpret the relevance of the data actually is. Curiously, in the Slater study "hysteria" was often being used to describe a histrionic reaction to organic disease (functional overlay). Given that the patients had identified neurological disease at entry to the study, it is really no surprise that they still had identified disease at follow up. The Slater study is perhaps best regarded as a warning that clarity of terminology is essential in medicine. It is unlikely that misdiagnosis

onstrate causality. The direction of potential causation is not in doubt (i.e. the future onset of symptoms is unlikely to be the cause of trauma) but it is not clear as to whether this association is of etiological significance or an epiphenomenon. Furthermore, in some articles [10], it is clear that both factors (physical and psychological) have a role in precipitating hysterical symptoms; however, it is not clear which was the more important.

In emphasizing the role of physical trauma it can be seen that the role of psychological process in pathogenesis is not discounted. Simply one would caution against the emphasis on life events and stress in existing diagnostic criteria. For example, a patient with a non-organic motor gait disorder displaying a positive Hoover's sign but no evidence of psychological trauma is best described as having conversion disorder despite not fitting current criteria. In response to this problem, Wessely [11] proposed to drop the classic psychoanalytic –derived criteria for conversion disorder.

The failure of science to define the precise physiologic basis for the symptoms seen in these disorders should not necessarily lead to labeling an individual with a psychiatric rather than medical diagnosis. There is evidence of a genetic predisposition to MUS and that different types of environmental triggers (emotional, physical and immune stresses) can either initiate or exacerbate MUS [12]. Such trigger factors include physical trauma as demonstrated in this review. However, the results of the present study may not be readily generalizable to other functional symptoms. Wessely [13] has demonstrated that physical factors such as viral infection are commonly implicated in chronic fatigue syndrome (CFS). Sixteen percent of our cases revealed organic diseases associated with the onset of MUS. This is in support of the current thinking that organic factors could precipitate the onset of functional disorders [1-2, 4, 10, and 14]. There are multiple reports of pathophysiological abnormalities noted in individuals with MUS such as fibromyalgia, CFS and other allied syndromes that might explain MUS although it must be appreciated that many such findings may relate to experimental error and unknown confounding factors.

Trauma is a heterogeneous concept, and can allow for a range of quantitatively and qualitatively differing insults. Charcot proposed that the

Table 1 – Percentage Of Patients Describing Specific Trigger Factors Prior To Onset Of The Symptoms (No. 841 Patients).

Trigger factor	No of patients	Percentage
Physical Trauma	266	31.6%
Stressful life events	214	25.4%
Panic	18	2.1%
Organic disease	135	16%
Surgery	50	5.9%

Note: - patients who had experienced physical precipitating factors (physical trauma, organic disease and surgery) rose to 53. 5 % and those who had experienced psychological factors (psychological stress and panic) fell to 27. 5%.

Discussion

This is the first systematic review of precipitating factors in medically unexplained paresis and anesthesia. The results suggest that physical trauma is the most commonly associated factor preceding the onset of such Medically Unexplained Symptoms (MUS). This is in stark contrast to current diagnostic frameworks of DSMIV and ICD 10, which insists on the presence of life/stressful events and credits them with etiological significance.

Physical trauma may often be far more psychologically and emotionally traumatic, than appears to be the case at first sight. Furthermore, the personal significance of the trauma to the individual may be an important factor even if the trauma is mild and "trivial". This may be influenced by the circumstances in which the trauma occurs. Pilowsky [8] placed an emphasis on the need for pains-taking analysis of the accident in all its details, since patients tend not to offer the information spontaneously. He tried to draw attention to the fact that the true nature of the accident from the patients' perspectives is often overlooked. Furthermore, recent studies [9] showed that conversion disorder patients were more prone to minimize their trauma history than other dissociative disorders.

Although the findings suggest that physical trauma may be an important part of the pathogenesis of MUS, an association does not dem-

5. Following surgery.

The data were recorded on an access database and then analyzed using a status direct software package. The analysis was performed using simple descriptive statistics.

Result

13089 titles and abstracts were checked among which 1345 articles were found. Of these, 147 articles met the study entry criteria (see appendix 1). They contained descriptions of 841 patients. Most articles illustrated a series of cases: we incorporated only those cases that met our criteria, and excluded those that did not. We also included one study that described onset details as percentages: it was apparent that all patients met entry criteria and data from this study were pooled with those from individual case reports.

Of the 841 we analyzed, 266 patients or 31.6% CI. (28-34%) were described as having experienced recent physical trauma preceding, and relevant to, the onset of their functional symptoms (Table 1). By contrast, just 214 patients or 25.4% (CI. 22-28%) were recognized to have experienced recent stressful events preceding symptom onset. Interestingly, patients who had experienced physical precipitating factors (physical trauma, organic disease and surgery) rose to 53.5 % and those who had experienced psychological factors (psychological stress and panic) fell to 27. 5%. Eight papers, although containing some onset data on 125 patients, did not actually examine for physical trauma. When we excluded these, the proportion of the 716 remaining patients, who had experienced recent physical trauma, preceding onset, rose to 37.1% CI (34-41%) and the percentage of those with preceding stressful life events fell to 12.4% CI (9-15 %).

All studies were included if the following criteria were met; -

1. Studies were included if the symptoms characterized were described as hysterical, functional, psychogenic, nonorganic, conversion, unexplained, dissociative or psychosomatic.
2. The following symptoms were included; paralysis, paresis, motor, weakness, gait, tremor, dystonia, myoclonus, or sensory disturbance.
3. Data on the onset of a symptom could be extracted.
4. They reported on at least one patient.

Reports of the following were excluded: -

1. Complex regional pain,
2. Reflex sympathetic dystrophy.
3. Pseudoseizures,
4. Factitious, Munchausen's disorder.
5. Mixed organic/non-organic disease,
6. Other conversion symptoms where visual loss, deafness, globus, dysphonia, fugue, pain were the primary symptoms.
   When a study described functional motor or sensory symptoms associated with pain, they were included; the exclusion was of studies where unexplained pain was the sole symptom. Studies reporting motor symptoms as part of reflex sympathetic dystrophy and complex regional pain syndromes were also excluded.
   The papers were reviewed by the author using a structured proforma. Results were then checked by another psychiatrist and any discrepancies resolved by a team of adjudicators.

The structured Proforma in an electronic Access Spread Sheet recorded the following: -

- The reference journal including the author and year of publication.
- Demographic data.
- Inclusion and exclusion criteria.
- The type and nature of medically unexplained symptoms.
- Circumstances of the incidence including both organic and psychological factors preceding the onset of these symptoms.
- Factors precipitating the onset of MUS such as: -
  1. Any physical trauma,
  2. Recent stressful life events (bereavement, divorce, redundancy etc),
  3. Whether precipitated by panic symptoms.
  4. precipitated by organic disorder,

DSM IV [2]. Therefore, it is clear from the definition that psychic trauma is a diagnostic criterion associated with their onset or exacerbation. In addition, multifactorial stress model was suggested in one of the recent studies [3] in the evolution of conversion disorder, showing the significant association between the recent life events and the severity of conversion symptoms. By contrast, the role of physical trauma has been minimized and become obsolete.

The psychoanalytic schools insisted on psychogenesis of conversion hysteria, facilitating its transfer from neurology as "functional" to psychiatry as a "conversion" disorder. These constructs are not empirically testable and Freud's arguments in support of his theories were unreliable or even misleading [4]. Caution should be in place against the validity of existing diagnostic criteria.

Physical trauma is *historically* reported as precipitating the onset of such MUS. Charcot "the father of neurotic hysteria" introduced the term of "traumatic hysteria", emphasizing the role of physical trauma as a trigger factor to the onset of functional symptoms. Erichsen, Benjamin Brodie, Russell Reynolds, and James Paget published case reports of nervous afflictions and paralyses that were precipitated by physical injury and occurred without apparent underlying pathology [5,6]. These views received support from contemporary neuropathologists [7]. By the end of 19-th and beginning of the 20-th century, the common view was that a combination of psychological and physical mechanisms was accepted as etiologically significant in the development of this malfunction.

In this study, a systematic analysis of the scientific literature since 1965 was undertaken to examine the extent of the existing evidence describing the precipitating factors for onset of medically unexplained paresis and anaesthesia.

Methods

The literature was searched from 1965 to June 2004 by using the following databases: - Medline (from January 1966), Cinahl (from1982), Embase (from 1980) and Psychlit (from 1965) and BIDS ISI (from January 1981). All studies that described the precipitating factors for onset of unexplained motor or sensory symptoms among patients were eligible for inclusion.

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Meta-Analytic Review

Precipitating Factors Relating to Onset of Medically Unexplained Paresis and Anesthesia.

Meta- Analytic Review

Mohammad Zaubi

العوامل المرسبة لبدء ظهور أعراض ضعف وخدر غير مفسر طبياً

محمد الزعبي

Abstract

Objective: The aim of this study is to examine the extent of the existing evidence describing the precipitating factors relating to onset of medically unexplained paresis and anaesthesia

Method: *A* systematic analysis of the available scientific literature since 1965 was undertaken to examine the rate of recorded trigger factors for onset of medically unexplained paresis and anesthesia. These factors examined were physical trauma, stressful life events, panic state, organic illness and surgery.

Result: Physical trauma was the most frequent precipitating factor 31.6% (confidence interval CI (28-34%) compared to stressful life events 25.4% CI (22-28%).

Conclusion: Physical trauma was a more likely precipitant for medically unexplained paresis and anesthesia than purely psychological events. The conclusions are however tentative as many physically traumatic events are associated with fear and panic. Nonetheless these results have implications for current DSM IV and ICD-10 diagnostic criteria of conversion disorder which insist upon a purely psychological triggering event.

Key Word: Medically unexplained symptoms (MUS), Chronic Fatigue Syndrome (CFS), Diagnostic & Statistical Manual (DSM1V), American Psychiatric Association (APA), Functional and Psychogenic.

Introduction

Hysterical symptoms are common in neurological practice, accounting for about 1% of neurological diagnoses [1]. Such unexplained motor and sensory symptoms were considered as Conversion Disorder in

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ملخص :

الأهداف : هناك دعم واضح في عدد من الدراسات القديمة والحديثة للرأي بإرتباط أحداث الحياة بالإضطرابات النفسية والأمراض العضوية . هذه المقالة تراجع الدراسات الحديثة حول العوامل المساعدة والمسببه ، الأساليب الجديدة وصعوبات البحث ، والذي لها أهميه في التخطيط لأساليب الوقاية والعلاج الفعالة .

النتائج: إن المعدلات العالية لإرتباط أحداث الحياة والإضطرابات النفسية والأمراض العضوية تشير إلى أنها مرتبطة وظيفياً مع بعضها .

الخلاصة : إن إدخال المرضى الذين تعرضوا لأحداث الحياة والإضطرابات النفسية والأمراض العضوية في البحوث العصبية والدراسات السريرية ، سيكون أساسي لتطوير طرق علاجيه لهذه المجموعة من المرض التي تعاني من أعراض شديدة

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lows: poor reliability; poor validity; the vague and ambiguous nature of many of the event descriptions; inclusion of events which may be indicators of, or caused by, the disorder under study; the muddling of events with ongoing difficulties; and insensitivity to the subtleties of events in terms of meaning, such that all events of a specific type are treated the same (the dictionary) nature of check-lists.

Gorman and Brown [163] summarized how some of these problems can be dealt with by introducing refinements to the check-list approach: reliability and validity, along with the problem of vagueness of questions, can be improved to some extent by making event descriptions more precise and by employing aids to recall such as calendars and discussing at some length the dating of particular events and contaminated events can be removed from the check-list.

In contrast to the `respondent-based` approach of the check-list method, the `investigator-based` technique developed by the London group (23, 53, 164) make use of an intensive semi-structured interview to collect data, and assesses the significance and meaning of events through a system of contextual rating by the investigator rather than relying on respondents` subjective reports.

Conclusion:

An increasing number of reports support an association between life events and both physical and psychological disturbances. This seems to be a complex phenomenon and research in this field often encounters serious methodological problems and limitations. Research is offering important information and with new development in investigation tools we hope to have an even better understanding of the problem. This is expected to result in developing more effective preventive and treatment approaches. These approaches are more likely to succeed if they take into account the complexity and multifactorial nature of the problem.

ences in cognitive ability have a role in the experience of and subsequent coping with life events [159].

c. Adverse environmental factors (lack of social support, low social status, ongoing difficulties)

Much attention has been devoted to parental separation/divorce (with resulting lack of child care in early years) and to the effects of physical and sexual abuse in childhood as possible risk factors for depression in adult life. Poor social support is related to onset and relapse of depression either through a direct effect unrelated to levels of concurrent adversities or through a buffering action which adds to the effect of adversities [160]. However, levels of social support do not contribute to gender differences in depression. Research on the association between social support and depression in male and female has provided controversial findings, with some studies reporting that social support is equally important in males and females as a predictor of recovery from depression, and others showing greater beneficial effects or even a detrimental action in either of the two genders [161].

Clinical Implications

Results of this review indicate that the occurrence of life events has an important role in the development of both psychiatric and physical disorders. However, may be it is not a direct cause of it. In this situation it will be recommended that:

1. Life events are very important aspect in the history taking in patients with both physical and psychiatric disorders.
2. Addressing the issue of life events in childhood and its effects in later life is very important aspect of management in different types of diseases.
3. Counselling and psychotherapy are crucial in reducing symptoms and stabilisation of social life.

Limitations:

The study of life events and both psychiatric and physical disorder face a paradoxical situation. While the popularity of this approach to aetiology has grown enormously, there has been increasing confusion and uncertainty about the validity of the measures employed [162].

In terms of collecting data about life events there is a fundamental division between the use of check-lists and in depth interviews [163]. Many authors have pointed to the shortcomings of the check-list approach [53, 162,164-167]. Brown and Harris [168] summarized these problems as fol-

on such personality variables as `hard working`, `ambitious`, `self reliant`, `feeling capable and accepted` and high scores on being `untrustworthy`, `rebellious`, `unsociable` and impulsive` and low scores on socialization during early adolescence predicted frequent and early use of cigarettes, alcohol, marijuana and other illicit drugs during high school [141-143].

Some of the risk factors for depression are also risk factors for substance use. Current depressive symptoms, poor coping skills, conflict with parents, and dissatisfaction with school are examples [144]. Depressive mood in early adolescence is a significant risk factor and be a mediating mechanism for drug use. It was suggested that certain depressed adolescents might begin to use marijuana in order to relieve depression [145]. Deykin et al [146] found an association between alcohol abuse and major depressive disorder but not with other diagnoses. They also found that the onset of major depressive disorder predated alcohol and drug abuse suggesting the possibility of self-medication as an initiating factor. However, Henry et al [147] in a prospective study showed that pre-adolescent depressive symptoms were found to predict multiple drug use 4 years later in boys but not in girls. This suggests that this mediating mechanism does not necessarily apply to all adolescents.

Peer influences seem, by most accounts, to have the biggest impact on adolescent substance using behaviour [148-151]. Peer influence has universally been identified as the single factor most likely to predict current drug use [152]. Drug use seems to serve a social purpose for some adolescents. It seems to provide asocial environment and a setting favourable to drug use. Such a setting increases the risk of initiation as well as continued use [153, 154].

Homelessness has also been implicated as a mediator factor in different psychological and physical problems [155-157]. However, we have to consider the bi–directional model where mental illness may be a risk factor for homelessness.

Cognitive ability is another possible factor mediating person environment relationships. Lower childhood cognitive ability has been identified as an independent developmental risk factor for both childhood and adult onset affective and neurotic disorder in two separate births cohort studies [158]. It has been suggested that individual differ-

stress may play a role in the evolution of depression.

b) Personal vulnerability (low self-esteem, helplessness, external locus of control, poor coping strategies)

There is some evidence that genetic effects on the likelihood of experiencing stressful life events are mediated by heritable traits such as cognitive ability and neuroticism [128]. The likelihood of experiencing stressful life events is partly under genetic control [42, 129]. Evidence suggests that around 10-15% of genetic effects on liability for depression may in fact be mediated by a mechanism whereby individuals select themselves into high-risk environments [41]. This mechanism is referred to as gene-environment correlation or genetic control of exposure to the environment [130-132].

Furthermore, Farmer et al [133, 134] found that neuroticism and extraversion were correlated with mood changes and life events. The mechanisms that mediate the effects of life events as risk factors are often referred to as `mediators` [135]. Mediating mechanisms have gained some attention in recent studies. However, the relationship between life events as risk factors and their mediators can be very complex in nature. Cloninger [136] first described certain personality characteristics of individuals at risk of developing early onset alcoholism. In a subsequent study Cloninger et al[137] supported this theory in a study of 431 Swedish males. Although Cloninger`s theory addressed alcohol abuse, it has been empirically tested in other substances [138]. Other research found that difficult temperamental characteristics in early childhood increased the possibility of tobacco, alcohol and marijuana use in adolescence [139]. Blackson and Tarter [140] studied individual, family, and peer affiliation factors predisposing to early-age onset of alcohol and drug use. They found that parents tend to take out their anger more often on children with difficult temperaments. They concluded that in a dysfunctional family, children are likely to disengage prematurely from the parental sphere of influence to peer influence. In the presence of strained parent-child relationships, this disengagement may increase involvement in unconventional activities including substance abuse.

Childhood personality attributes seem to be risk factors for early initiation into substance use. A series of studies showed that low scores

is that drug use is a response to stress [117, 119], and the second is that drug use is functional in reducing stress [120, 121]. O`Doherty concluded that heroin and alcohol use act as stress ``buffers``, in that they reduce awareness of ongoing stressful life events. But in so doing they introduce a lot of new stresses, which possibly maintain the drug use and ultimately increase stress.

Pathways to Co-morbidity:

Elevated rates of Co-morbid mental and physical disorders in people with history of negative life events complicate any effort to develop a model of the relationship between these variables. High rates of Co-morbidity could suggest that these factors are functionally related to one another. Several pathways have been described to explain these high rates.

a) **biological mechanisms (dysregulation of the hypothalamic-pituitary-adrenal axis)**
In humans and animals, acute stress elicits a cascade of neurohormonal events, including increased turnover of nor epinephrine in terminal projection regions of the locus ceruleus and liberation of hypothalamic corticotrophin-releasing hormone (CRH) into the pituitary portal system, which stimulates release of ACTH from the pituitary, which in turn triggers release of cortisol (human) or corticosterone (rat) from the adrenals. Animal and human research has implicated this cascade in the pathophysiology of both substance use disorders and PTSD [122, 123].

Recent studies have suggested that elevated cortisol levels, probably caused by stressful life events, may themselves lower brain 5-HT function and this in turn leads to the manifestation of the depressive state [124, 125]. It seems from this theory that cortisol is the key biological mediator through which life stress lowers brain 5-HT function, thereby causing depression in vulnerable individuals [126]. However, Strickland et al [127] found that elevated cortisol levels in a subgroup of participants who had experienced recent severe life events whether or not they were currently depressed. They concluded that the hypothalamic-pituitary-adrenal axis is sensitive to social stress but does not mediate vulnerability to depression. Exaggerated 5-HT2 receptor responsiveness to

year) for the comparison of event rates. Brown et al [64] studied the influence of family life on the course of schizophrenic illness. The authors found that they could predict relapse of schizophrenia during a nine months period following discharge by using an index of the expressed emotion (EE) shown by the relative during an interview shortly after the patient was admitted to hospital. This study was replicated and extended by Vaughn and Leff,[65]. Nonetheless, there is evidence from family, twin and adoption studies of a substantial genetic contribution to schizophrenia. The mode of transmission is complicated and very rarely if ever involves a single gene. Rather schizophrenia results from multiple genes of small effect and their interplay with the environment [66].

In addition life events have been found to have an effect on nearly every psychiatric disorders and psychological phenomenon including: the **onset of phobia**[67] , **trichotillomania** [68]; **age of first onset of bipolar disorder** [69]; **suicide attempts** [69- 74]; **drug induced deaths** [75]; of **substance abuse, depression overdose and suicide** [76- 78], Co-morbidity [79]; **schizophrenia and suicide**[80]; **personality disorders**[81- 87]; **panic disorder and anxiety disorder** [88- 92, 94, 95]; **somatoform disorders**[96- 98]; **sleep disturbance** [99]; **Obsessive compulsive disorders** [100, 10]; **Co-morbid post-traumatic stress disorder and other psychological disturbance** [101-106]; and even was found affecting the compliance to treatment and utilizing the mental health system [107- 110]. Finely patients with chronic fatigue syndrome reported significantly more negative life events prior to the onset of their illness than healthy controls [111- 113].

Another important area of research in the Co-morbidity of life events is the problem of **substance misuse**. Prior research has reported a greater frequency of life events for drug users prior to the onset of drug use, compared to matched psychiatric and controls groups. There are also reports on higher incidence of life events after onset when comparing people who inject drugs to a control group [114].

The literature focusing on risk factors involved in addiction has explored a number of variables e.g. constitutional predisposition; environmental factors (family and peers) and life events [115, 116].

The theory that life events can have an impact on drug use arises from two assumptions. The first of these

that, about one third of the association between stressful life events and onsets of depression is non causal. Using database derived from the Virginia Twin Registry, the authors provided two basic results to support their conclusion:

1) Dependent stressful life events (resulting from subject's behaviour) are more strongly associated with depression than are independent events (unrelated to a subject's behaviour) and 2) the risk for depression associated with personal stressful life events appears to be higher in a sample of female twins than in a sub sample of monozygotic twins.

Michalak et al [58] assessed the association between a range of demographic and psychosocial factors, ``seasonality and ``casesness`` in a community sample in the UK. They found that four factors (having experienced more numerous negative life events, having low levels of social support, being a woman and being non-native) were predictive of higher seasonality.

The situation in old age depression is different, in that Ormel et al [59] investigated the risk of stressful life events on old age population. They found that stressful life events did not increase risk. High neuroticism and difficulties increased risk, even without a stressful life event. The authors demonstrated the usefulness of a **dynamic stress-vulnerability model** for understanding late life depression. However, Butler et al [60] provided some evidence in their study that having two or more stressful and negative life events may reduce survival in older people with and without dementia.

Schizophrenia is one of the corner stones in this area of research. The major controlled and systematic studies of life events and schizophrenia are those of Brown and Birley [61] in Camberwell, London and Jacobs and Myers [62] in New Haven, Connecticut. The Camberwell study [61, 63] investigated 50 schizophrenic patients who had been admitted consecutively to a hospital in Southeast London. It was found that there was a significantly greater rate of events in the three weeks before the onset of schizophrenia than in the comparable period for the controls: 60% of the schizophrenic group experienced an `independent` or `possibly independent` event in this period, as opposed to 19% of the controls. In the New Haven study [62] the results obtained were inconclusive, probably because the authors selected an over-long antecedent period (one

ings. First, numerous studies have now shown that exposure to stressful life events is substantially influenced by **genetic factors** [37-39]. Individuals do not experience stressful life events at random; rather, some individuals have a stable tendency to select themselves into situations with a high probability of producing stressful life events. Second, the genetic risk factors for stressful life events are positively correlated with the genetic risk factors for major depression [40,41]. That is, a genetically influenced set of traits both increases individual's probability of selecting themselves into high-risk environments likely to produce stressful life events and increases their vulnerability to major depression [42].

Patten [43] found that associations between certain variables (traumatic life events, recent life events, and social support) and major depressive disorders in community populations might reflect non-specific associations with mental ill health.

Hays et al [44] found that social support and physical health were more relevant to chronicity of major depressive illness than were severity of illness or family history.

Finlay-jones & Brown[45] investigated the frequency of life events reported by three types of cases (**depression; anxiety; and mixed depression/anxiety**) as occurring in the year before the onset of their disorder was compared with the frequency of events in the same time period reported by a group of women without severe psychiatric disorder. The results were used to argue that severe loss was a causal agent in the onset of depressive disorder and severe danger was a causal agent in the onset of Anxiety State in this sample. Cases of **mixed depression /anxiety** were more likely to report both a severe loss and severe danger before onset.

A prospective study of 400 largely working-class women with children living at home has demonstrated the major importance of long-term severe threatening life events in provoking case ness of depression [46].

Numerous studies supported the Co-morbidity of depression/anxiety and its correlation with childhood adversities particularly **physical and sexual abuse** [47-49]. Nonetheless, a few studies have failed to confirm the expected relationships [50-52] and cautions have been sounded [53-57].

However, Kendler et al [33] addressed the important issue of the extent to which stressful life events cause the onset of depression and concluded

Chronic adversities were common[20]. The role of life events as precipitating factor of **bulimia nervosa** has been studied by Welch et al, [21]. Their study showed a higher incidence of life events in the year prior to onset in bulimia group when compared to the control group. Significant physical illness, pregnancy, sexual or physical abuses were more often reported by women in bulimia group than women in the control group.

Schmidt et al [22] also investigated the impact of life events on the onset of eating disorders using Brown and Harris`s [23] life Events Difficulties Schedule. They found that 22% of women with anorexia nervosa, 34% of those with bulimia, and 4% of the control group had endured at least one major difficulty in 12 months before onset. Threat of divorce or actual separation; problems with a parent; health problems; death of a relative; work or school problems were among some of the major difficulties. Troop and Treasure [24] measured life events prior to onset of eating disorders. They found that 58% of anorexia nervosa and 77% of bulimia nervosa experienced a severe event or marked difficulty-provoking onset of their eating disorder.

Life events and psychiatric disorders:

There is evidence of a link between stressful life events and problems of psychological health. Negative life events are common in patients with different psychiatric disorders [25 - 27]. This part of the article reviews clinical, epidemiological and neurological studies relevant to the problem of Co-morbid life events and psychiatric disorders and discusses the clinical implications of these findings.

Numerous investigations have found a correlation between the occurrence of stressful life events and the subsequent onset of an episode of **major depression** [28- 32]. However, there is less certainty about the nature of the relationship between major depression and stressful life events. In particular, it remains unclear to what extent stressful life events cause subsequent onsets of depression and to what extent the occurrence of stressful life events and onsets of depression are correlated for other reasons. [33].

Although concerns about a non-casual association between stressful life events and major depression have been expressed for a long time [34-36] the salience of this problem has been increased by two sets of find-

changes in life circumstance, and the additive effect of Co-morbid life events, is significant in producing impaired and extended coping during subsequent ischemic events. Interestingly Melamed et al [16] study revealed a striking disparity in the outcomes. Life events were negatively associated with systolic (p=0.001) and diastolic (p=0.038) **blood pressure, triglycerides** (p=0.011), and **uric acid** (p=0.05), even after controlling for job strain and other possible confounders. In contrast, life events were positively associated with somatic complaints (p<0.0001), anxiety (p<0.0001), irritability (p<0.0001), and depression (p<0.0001). In addition there was a linear trend between intensity level of life events and low exercise (p=0.006), smoking (p=0.007), and alcoholic intake (p=0.035). The authors explained the disparity of the result as a product of powerful biases, such as repressive coping and negative affectivity disposition.

In **gastrointestinal system**, Burke et al [17] reviewed the clinical findings on **irritable bowel syndrome** in adults and **recurrent abdominal pain** in children and its relationship to the Co-morbidity and life events. They found evidence of striking similarities between the disorders in prevalence, course, medical and psychiatric history, and association with life events. In another review North and Alpers [18] looked for a significant relationship between **Crohn`s disease** and stressful life events or psychiatric symptoms or disorders. Published data indicate that Crohn`s disease, unlike ulcerative colitis, may be statistically associated with lifetime psychiatric disorders. This association appears to be more modest than in irritable bowel syndrome, in which far higher rates of psychiatric disorders are reported than in Crohn`s disease. Stanghellini [19] in the International Gastroenterology Surveillance Study (DIGEST) investigated the prevalence and quality of life impact of **upper gastrointestinal (GI) symptoms**. The most notable risk factors for the occurrence of upper GI symptoms were found to be various indicators of psychological stress (particularly recent life events) and psychiatric disease.

Eating disorder is another area of several works to investigate the relationship with life events.

As part of a prospective outcome study, subjects with **anorexia nervosa** reported intermediate rates of negative life events between community and psychiatric controls.

number of diseases at the same time or in a limited time period and others do not [3]. Recently there is an increasing evidence that exposure to traumas was correlated with number of physical illness and mental disorders [4,5].

Studies on the effect of life events on co-morbidity of both physical and psychiatric disorders were identified for review by means of computerized and manual searches. In this review article this co-morbidity are examined in details.

Life events and physical illnesses:

There is evidence of a link between stressful life events and risk of different physical diseases, including the onset and progression of Creutzfeldt-Jakob disease [6]; infection and chronic fatigue syndrome [7]; Alopecia areata[8]; Altered pituitary-adrenal axis [9]; in the rare syndrome of burning mouth[10]; and even in the congenital malformations (particularly of the cranial neural crest [11].

Although there is evidence that stress is associated with alterations in immunity, the role of emotional factors in the onset and course of **immune-based diseases** such as cancer and **AIDS** has not been established till recently. Evans et al[12] reported the first evidence from a prospective research study that severe life event stress is associated with an increased rate of early **HIV** disease progression. Faster progress to AIDS was associated with higher cumulative average stressful life events [13].

In addition Bowman and Markand [14] found that events is associated with the occurrence of pseudoseizure and common current psychiatric diagnoses included **somatoform disorders** (89%), **dissociative disorders** (91%), **affective disorders** (64%), **Personality disorders** (62%), **posttraumatic stress disorder** (PTSD) (49%), and other **anxiety disorders** (47%). **Trauma** was reported by 84% of subjects: **sexual abuse** by 67%, **physical abuse** by 67% and **other trauma** by 73%. This study showed that pseudo seizures often appear to express distress related to abuse reports. The clinicians should screen pseudo seizure patients for adult and childhood trauma. Furthermore, there is evidence of a link between stressful life events and risk of cardiovascular disease, but the pathway has not been fully explored.

Alonzo [15] review article found growing evidence that traumatogenic potential of **acute myocardial infarction** (AMI) with its sudden and unexpected onset, dramatic

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Review Article

Life events, Co-morbidity of Psychiatric Disorders and Physical illnesses:

A Review of the Literature

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الإرتباط بين أحداث الحياة، الإضطرابات النفسية والأمراض العضوية

مراجعة للأدبيات

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Abstract:

Objective: The view that co-morbidity of life events, psychiatric disorders and physical illnesses enjoys considerable support by a number of older and recent publications. This article reviews recent studies on possible etiological and contributing factors, new approaches and research difficulties, which are of particular interest for planning more effective preventive and treatment strategies.

Method: studies were identified by means of computerized and manual searches.

Results: high rates of Co-morbidity suggest that life events, psychiatric disorders, and physical illnesses are functionally related to one another.

Conclusion: inclusion of patients with Co-morbid life events, psychiatric and physical disorders in neurobiological research and in clinical trials will be critical for development of effective treatment for this severely symptomatic patient's population.

Key wards: co-morbidity, life events, physical, psychiatric disorders.

Life events, Co-morbidity of Psychiatric Disorders and Physical illnesses: A Review of the Literature

Introduction:

Many people are suffering from multiple diseases. This health problem called multi-morbidity [1] or co-morbidity. The term co-morbidity refers to the presence or co-occurrence of two different medical conditions [2]. Generally it is not clear why some patients have a

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control probands were similar to the cases in most aspects other than the presence of disgnosis of panic disorder .

Third : not all the interviews in the study were direct and in person . we conducted these interviews at the hospital , we adopted the family-history study in about a third of the cases in which we interviewed a key person in the family who was most knowledgable about other family member's psychiatric history .

الملخص :

إضطراب الهلع من الأمراض النفسية الشائعة ، ويكون عادة بنسبة أعلى لدى النساء مقارنة بالرجال ، وأن معدل متوسط العمر للإصابة بهذا المرض هو ما بين 22-44 سنة ، وهناك إحتمالية إصابة أقارب الدرجة الأولى بنسبة 17.3% .

الأهـــــداف : تهدف هذه الدراسة إلى معرفة نسبة إضطراب الهلع في أقارب الدرجة الأولى للمرضى والأصحاء ضمن عينة مختارة ومعرفة الخصائص الإجتماعية والديموغرافية لهؤلاء المرضى .

طريقة البحث : تم جمع عينة من ستين مريضاً مع (342) من أقارب الدرجة الأولى لهؤلاء المرضى مع ستين من الأصحاء و(314) من أقارب الدرجة الأولى لهؤلاء الأصحاء بعد تطابق العمر والجنس وتم دراسة إنتشار إضطراب الهلع في أقارب الدرجة الأولى للمرضى والأصحاء بإستخدام طريقة التاريخ العائلي ، وكذلك جمع معلومات إجتماعية وديموغرافية عامة ومن ثم تطبيق المحاورة شبه المنظمة المبنية على أساس خصائص إضطراب الهلع المثبتة في الكراس التشخيصي – المراجعة الرابعة .

النتائج : أظهرت الدراسة أن نسبة إضطراب الهلع كان أكثر عند أقارب الدرجة الأولى للمرضى (14.9%) مقارنة بأقارب الدرجة الأولى للأصحاء (3.1%) وكانت نسبة الإصابة عند الإناث مقارنة بالذكور 2 : 1 وكان معظم المرضى من المتزوجين ويسكنون في المناطق الحضرية وكان المستوى التعليمي جيد .

الإستنتاج : تبين من الدراسة أن إضطراب الهلع كان أعلى عند أقارب الدرجة الأولى للمرضى المصابين بإضطراب الهلع مقارنة مع مجموعة الأصحاء وهذا يعني أن العامل الوراثي يلعب دوراً في نسبة الإحتمال بالإصابة بهذا المرض .

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Table 4 . summary of findings of other researchers on the rate of PD among FDR's of case and control groups .

Source, year	Diagnostic criteria	Rate of PD in case relatives	Rate of PD in control relatives
Crowe et al 1983	DSM – III-	17.3%	1.8%
Maier et al 1987	DSM– III-R	7.9%	1.3%
Vondenheuvel1997	DSM- IV	20.2%	4.2%
Gormood et al 1999	DSM- IV	10.7%	10.7%
This study 2004	DSM- IV	14.9%	3.1%

Discussion

This study reports that Panic disorder is familial , the FDR's of probands diagnosed as suffering with PD according to DSM- IV had five fold higher rate of PD as compared to the control group , and this finding is generally in accord with the most recent family studies of panic disorder .This result in comparison with the finding of other similar studies dating back to the eighties showed some similarities and differences because of different methodological approaches used and different settings in which those studies were done. There are several methodoligical limitations to our study :

First : all case probands were in treatment and attending the O/P clinic regularly for follow up . a community sample would have been more desirable, but practically it is difficult to screen hundreds to obtain sufficient numbers of panic disorder probands to conduct this studey .

Second: control probands may have agreed to participate in the study based on their personal or family concerns about emotional difficulties , so we made every effort possible to ensure that the

Table 1. Demographic Characteristics of Cases and Control Probands

sex	Case Probands (n = 60 No. (Percentage)	Control Probands (n = 60) No. (Percentage)	Total
Males	20 (33.34 %)	20 (33.34 %)	40
Females	40 (66.66 %)	40 (66.66 %)	80
Age Groups			
20-27	16 (26.6 %)	17 (28.4 %)	33
28-35	21 (35 %)	22 (36.6 %)	43
36-43	9 (15 %)	12 (20 %)	21
44-51	14 (23.4 %)	9 (15 %)	23
Total	**60**	**60**	**120**

Table 2 . Demographic Characteristics of Cases and Control First Degree Relatives

	Case Relatives (n= 342)	Control Relatives (n= 314)
Sex	No. (Percentage)	No. (Percentage)
Male	178 (52.05 %)	145 (46. 18)
	164 (47.95 %)	169 (53.82)
Types of Relatives		
Parents	112 (32.75 %)	117 (37.26 %)
Siblings	169 (49.42 %)	176 (56.05 %)
Children	61 (17.83 %)	21 (6. 7 %)

X=18.6 , df= 2 , p = 0.0001

Table 3. Rate of P.D among FDR's of both Case & Control Relatives

Relatives	Case Relatives n = 342	Control Relatives n = 314
	No . (%)	No. (%)
Positive Cases of Panic Disorder	51 (14.91 %)	10 (3.18 %)

P= < 0.0001 highly significant $X^2 = 25.32$ Df =1

Data were collected and analyzed statistically, chi-square and (t) test were applied whenever indicated.

Patients excluded from the study were those who had history of psychiatric illness other than PD and those who were unwilling to participitate in the study .

Control Group: A group of Sixty people matched for age and sex were selected from the community, and 314 of their first degree relatives were interviewed using semi structured interview based on DSM IV criteria for Diagnosis of PD.

Results: The results are summarized in tables 1-4 .

As presented in Table 1 : the sex distribution in both case probands and control group is 33.3% for males and 66.6% for females, the age distribution of both groups show that the numbers of the sample in age group 28-35yr. was higher in relation to other age groups.

As presented in Table 2 : The sex distribution of FDRs of both case probands and control probands shows the males in case proband group are over represented 52.4% while in control group the female are over represented 53.8% , the distribution of the types of FDRs studied in both groups shows that the siblings were over represented in both groups 49.41% versus 56.05% .

Table 3 shows that the rate of PD in FDRs of case probands is higher than in control group (14.9% Versus 3.18%) which is statistically highly significant finding .

Table 4 presents summary of findings of other researchers on the rate of PD among FDR's of case and control groups .

compared with (41) control group and their 262 FDRs in which the rate of PD among FDRs of proband were 17.3% while in FDRs of control group the rate was 1.8% [10,11].
Maier et al studied the rate of PD in FDRs by using DSM-111-R criteria in sample of 40 probands and their families compared with 80 control and their families in which the rate of PD in FDRs of proband group was 7.9% while the rate of PD in the FDRs of control group was 1.8% [12].
Von Den Heuvel studied the rate of PD in FDRs by using DSM 1V criteria among sample of 80 probands and their 612 FDRs with 80 control and their 723 FDRs, the rate of PD in FDRs of probands was 25% while in FDRs of control group was 4.2%[13].
Gorwood et al studied the rate of PD in FDRs by using DSM 1V criteria, among sample of 780 probands and their 3700 FDRs and the rate of PD was 10.7% in the FDRs while in 720 control group and their 3400 FDRs, the rate of PD was 1.4% [14].

Objectives : to study the rate of PD among FDR's of both case probands and control probands .

Methods Sample : Sixty patients diagnosed as panic disorder according to the Diagnostic and Statistical Manual 4th revision (DSM – IV) , were randomly selected from those who were referred by senior psychiatrist at two psychiatric out patient clinics in Baghdad .
The two psychiatric out patient clinics were in Baghdad , namely Ibn Rushd Teaching Psychiatric hospital and Al-kadimyah teaching hospital.
342 first degree relatives of those patients were interviewed using semistructed interview based on DSM IV criteria for diagnosis of PD after taking their verbal consent to participate in the study .
The duration of the study was seven months between June 1st 2004 and December 31st 2004 .

Design: The patients were examined in a cross sectional approach one day a week for six months and a special form was used to gather information about the socio demographic characteristics .
Semi – structured Psychiatric interview schedule based on DSM IV diagnostic criteria of PD was applied to diagnose PD, that schedule was translated into Arabic and the translation was subjected to expert opinions from seven qualified Psychiatrists .

treatment and cognitive behaviour therapy [2].

There is high correlation beteween PD and Hypochondriasis and this intricate relationship requires more extensive research , since the current management of both conditions are not alike , whether it is pharmocological or psycho-therapeutic [3].

The preponderance of evidence suggests there is a genetic contribution to the predisposition to develop panic attacks and agoraphobia , there are increased rates of PD in FDRs with 7.9 – 41% versus 8% or less in control populations . overall increased risk in FDR ranges From 2-20 folds , the increased familial aggregation is specific for PD , these findings are consistent with a modest genetic transmission with relatively high specificity [4].

Evidence from family studies suggest that PD involves modest inheritability of around 30- 40 % . The best model suggests 50 % genetic and 50% environmental influences , recent linkage studies to confirm these hypotheses have been contradictary but do suggest that single gene transmission is unlikely , this leaves the possibilities of either heterogeneity across families and / or a polygenic inheritance .

PD takes its name from the Greek god Pan , god of flocks , Pan was known for suddenly frightening animals and humans out of the blue, **These sponstaneous out of the blue**: characteristic of panic attack is the principal identifying factor of PD and central to its recognition and diagnosis ,we know the syndrome that currently called PD has probably existed since the beginning of recorded history . Hippocrate presented a case of obvious phobic avoidonce around 400 B.C [5] .

In 1870 Da Costa ,a distinguished Philadelphia physician, described another syndrome of chest pain which he called the irritable heart syndrome consisting of palpitation, cardiac pain of various types, rapid pulse, headache, sweating, shortness of breath, this was the first description of panic attack [6,7] . Hecker (1893) and Freud (1894) delineated the psychological and the somatic aspects of the "anxiety neurosis" and gave the first clear description of panic attacks [8,9] .

Crow et al in 1983 studied the rate of PD among FDRs by using DSM111 criteria among sample of (41) probands with their 278 FDRs

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A family study of Panic disorder in Iraq

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دراسة عائلية لاضطراب الهلع في العراق

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Abstract

Summary: Panic disorder is a common psychiatric disorder and is slightly higher in women than men, the age range is between 22-44 years, there is increasing evidence of familial transmission of panic disorder with morbidity risk in first degree relative of 17.3%.

Objectives: To determine the rate of PD among first degree relatives of both probands and control group among a sample of psychiatric out patients with PD and the sociodemographic characteristics of such patients.

Methods: A sample of sixty probands with their 342 FDRs and a control group of sixty people matched for age and sex with their 314 FDRs were assessed by the use of semi structured psychiatric interview schedule based on diagnostic and statistical manual fourth revision (DSM IV) criteria for diagnosing PD .

Results: The rate of PD was high among FDRs of probands (14.9 %) than FDRs of control group (3.1%) with female to male ratio of 2:1 , most patients were married and living in urban area and were of good educational status.

Conclusion: PD was higher among FDRs of case probands than FDRs of control group which suggest a genetic aetiology for PD.

Introduction and Review of Literature

PD is one of the anxiety disorders in which the essential features are the presence of recurrent unexpected panic attacks followed by at least one month or more of persistent concern a bout having another panic attack , worry about the possible implications or consequences of panic attack or significant behavioral change related to the attack [1].

Panic disorder is one of the common psychiatric disorders with prevalence rate of 0.6-1% and is slightly higher in women than men , the age range is 25-44 years lowers in persons over the age of 62 year . Available data suggests that Panic disorder responds well to both drug

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ملخص

المقدمه: إن التقدم فى مفهوم التوحد (Autism)هو من بين التطورات ذات المغزى الكبير فى التاريخ الحديث للطب النفسى فأدبيات الطب النفسى فى العالم العربى بحاجه أن تضع تأكيداً أكبر وتشجع البحث العلمى المحلى فى هذا المجال وهذه الدراسه التجريبيه تتبنى المفهوم الأوسع لمنظور الإضطرابات التوحديه (ASD)

الاهداف: تهدف هذه الدراسه إلى تقديم استفتاء جديدا وهو The Autistic Quotient Questionnaire ويرمز اليه بـــ (AQ) إلى أدبيات الطب النفسى فى العالم العربى. وقد تمت ترجمه هذا الإستفتاء إلى اللغه العربيه وإستخدام على مشاركين مصريين ولا يمكن إثبات مصداقيه هذا الإستفتاء بطريقه رسميه لأن هذا خارج مجال هذه الدراسه التجريبيه كما أن الدراسه قد صممت لإكتشاف عما اذا كان الطلاب المصريين الذين يعملون بدرجه عاليه قادرين أن يكون لديهم بعض السمات التوحديه وعما إذا كانت هذه السمات لها أى مغزى طبيّ.

الطريقه: فقد أكمل 202 طالبا من كليات مختلفه فى جامعه الزقازيق بطريقه كامله الاستفتاء The Autistic Quotient Questionnaire كما أكمل الطلاب كذلك النسخه العربيه لإستفتاء الصحه العامه (GHQ-30) كمقياس عام للعلل النفسيه

النتائج: كأن متوسط إجمالى درجات (AQ) هو 22.72 (SD 4.44) وكذلك يتفق مع التوزيع الطبيعي ، كما كان متوسط إجمالى درجات إستفتاء الصحه العامه (GHQ) هو 34.43 (SD13.9). ولقد وجد إرتباط إيجابى بين إجمالى درجات الـــ (AQ) والدرجات الإجماليه لإستفتاء الصحه العامه (GHQ) (إرتباط إختبار Pearson الاول المعدل هو (P=0.36

وتوحى النتائج أن السمات التوحديه كما تعبر عنها درجات الـــ AQ موجوده بمدى مختلف لدى الطلاب المصريين ولا تختلف عن الدراسات البريطانيه واليابانيه. كما أن السمات التوحديه ارتبطت ايجابياً بالعلل النفسيه كما عبر عنها درجه إستفتاء الصحه العامه (GHQ)

الإستنتاج: الدراسه توحى بأن مقياس الـــ (AQ) مفيد بدرجه كبيره لقياس السمات التوحديه فى الأفراد ذوي الكفائة العاليه فى الحالات المصريه. وتتبع مثل هذه السمات فى مثل هؤلاء الأفراد يمكن أن يكون له مغزاً طبياً هاماً وسيكون إضافه ذات مغزى لطرق القياس الطبى والذى سيثرى معرفتنا بالسلوك البشري.

version of the AQ questionnaire is a complicated process beyond the scope of this research. The sample does not contain control groups of autistic patients. Obviously the sample in this study is neither big enough, nor representative of the general public and not randomly selected.

The AQ questionnaire is a new scale which will probably develop further in the future. It has its limitations which need further work on. One limitation is that it is a self report open to bias of subjectivity. There is probably a need for developing scales which are still easy to use and are more objective in the future.

The GHQ is also a fairly reliable but simple test. The study results could have become much more valid and indicative if a bigger and more elaborate scale was used. However such a pilot study is necessary to reassure researchers that such line of inquiry is worth investigating.

However, it is a fairly new thinking, to use such application of the wider concept of "Autistic Spectrum" and look for autistic traits in highly functioning individuals and trace its clinical significance. This study is hopefully a new line of research in the Arabic and Egyptian literature in both psychiatry and psychology.

Bishop and colleagues [53] used the AQ scale on parents of Autistic Spectrum Disorders patients, concluding that The AQ scales can be combined to give an index of broad phenotype. The authors found that the probands parents scored higher on the social skills (SS) and communication (C) subscales. This seems to be consistent with the fact that the social skills (SS), Imagination (I) and communication (C) subscales have been the most correlating subscales with the AQ total score (Atot). Saying that it is important to refer to the following observations; first, it might be too assumptive to think of the Egyptian student with high AQ total score (Atot) as equivalent to relatives with Broad Autistic Phenotype (BAP).

While the first group share a high score on the AQ scale, the second have something different which is the shared genetic factor/s with autistics relative. The student sample might have many false positive results either due to the limitations of the scale or due to environmental factors which made them behaves like or look like people with significant autistic traits. This factor is much less prominent in the Broad Autistic Phenotype (BAP) relatives.

It is also significant to mention that while this study looked for a broad and non-specific evidence of psychological discomfort (as indicated by GHQ) and related high AQ score, the Broad Autistic Phenotype (BAP) studies looked for diagnosable psychiatric disorders following much more stringent criteria for making such diagnoses. It is quite possible that those relatives could have scored high on the GHQ too and still not diagnosed with formal psychiatric disorders.

It is also possible that the students have been relatively functioning on a higher level compared with the Broad Autistic Phenotype (BAP) relatives and enjoyed higher level of cognitive functions. This could possibly make them more aware of their problems and more responsive to assessment. It is also a highly interested group of students who were keen on participating in the research which is a source for selection bias.

Limitations of the study

This is a pilot study in more than one aspect. It is a limited study in its scope, tools and numbers. For example the Arabic version of the AQ questionnaire is not standardized. Standardisation of the Arabic

the two groups are different in academic abilities, level of stress related studying environments, socioeconomic background and probably the general level of stress in life. GHQ total score (Gtot) in science colleges = 35.24 (SD = 13.36) & (Gtot) in theoretical colleges = 29.36 (SD = 10.03). This suggest that the Egyptian results agree with the conclusion made by Baron-Cohen et al [62] in that the AQ questionnaire is not influenced by IQ or socioeconomic status. It might be also not easily influenced by the level of stress affecting subjects.

On the other hand the Egyptian males scored more on the AQ total score (Atot) = 23.11 & SD = 4.5) than the female (Atot) = 22.03 & SD = 4.3) This is not a statistically significant difference. Descriptively it is however, consistent with Baron-Cohen et al study [62].

This study provides more data about the psychological distress, in terms of the total score of GHQ (Gtot), and relates them to the AQ total score in Egyptian students. This type of comparison is neither in Baron-Cohen et al study [62] nor in Wakabayashi et al studies [64]. In this study the AQ total score (Atot) is found to have a statistically significant correlation to GHQ total score (Gtot) (p = 0.036) (1-tailed).

The GHQ total score (Gtot) has also been found to be significantly different between types of colleges; (Gtot = 35.24) (SD = 13.36) in science colleges while (Gtot = 29.36) (SD = 10.34) in theoretical colleges (P = 0.027). The GHQ total score (Gtot) has also been found to be different between sexes though not statistically significant (Gtot = 33.33) (SD = 11.8) in males while (Gtot = 36.4) (SD = 15.02) in females. This could be a reflection of the stress related to being a female studying in a highly demanding and challenging subjects, like medical schools, in the Egyptian culture. Taking all these results in consideration together it might be acceptable to think that high AQ score can increase the vulnerability to already present social stresses like those related to demands of studies and building career especially in females.

The positive correlation between AQ total score (Atot) and GHQ total score (Gtot) in this study could seem to be in contradiction with earlier studies which failed to find a correlation between psychiatric disorders specially depression and Broad Autistic Phenotype (BAP)[54-58].

Discussion

The sample of this study is mainly young university students from Egypt. Such specific sample can still be relevant to the progress of the research idea. The sample is similar to samples in other researches which have used the AQ Questionnaire. This group of students is a good example of highly functioning adults of average or above average intelligence. Finding relevant use of AQ concept in such group proves the point of the research.

In Baron-Cohen et al study [62], 2% of the control group have scored 32 or more on the AQ score compared to 3% in the Japanese study [64] and 1.5% in the Egyptian sample. The Cambridge students mean total score of AQ was 17.6 (SD = 6.4) compared to Egyptian students mean 22.72 (SD = 4.44). In the Japanese study [64], the Japanese students mean score on AQ was 20.7 (SD = 6.38).

It is difficult to explain the differences in the means between the three cultures. However, the mean AQ by the mathematics Olympiad students was 24.5 (SD = 5.7) in Baron-Cohen et al study [62]. This might suggest an environmental factor. It could be that certain forms of education encourage quantitative accumulation of knowledge at the expense of imagination and also at the expense of normal social activities. This seems to be the case in the Egyptian students. In such case, students would be of higher AQ total score compared to groups who are identified by their creativity and use of imagination.

There is also a possibility that the AQ is culturally biased towards normalising some British patterns of behaviour which might make behaviours from other cultures not typically normal. This could be the case especially that the Japanese student (Mean = 20.7 & SD = 6.38) in Wakabayashi et al study[64] scored differently from the British students (mean = 17.6 & SD = 6.4), as well as the Japanese normal adult control group (Mean = 18.5 & SD = 6.21) compared to the British similar group (Mean = 16.4 & SD = 6.3).

In the mean time, the AQ scores in the Egyptian sample have been found to be similar in the two different student groups. AQ total score (Atot) in science colleges = 22.72 (SD = 4.6) & (Atot) in theoretical colleges = 22.75 (SD = 3.2)). It is probably acceptable to say that

Graph (2) Scree plot for principle component analysis for GHQ 30 questions

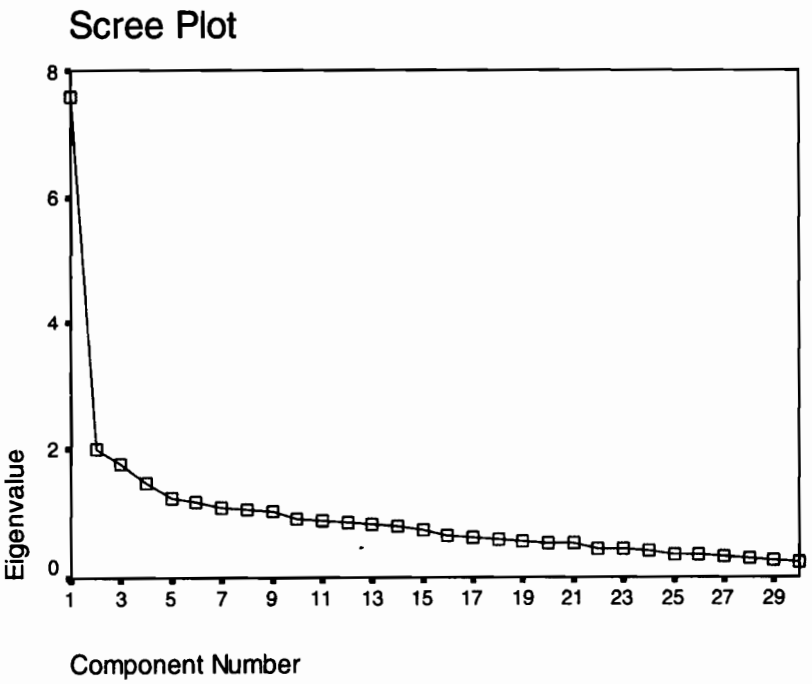
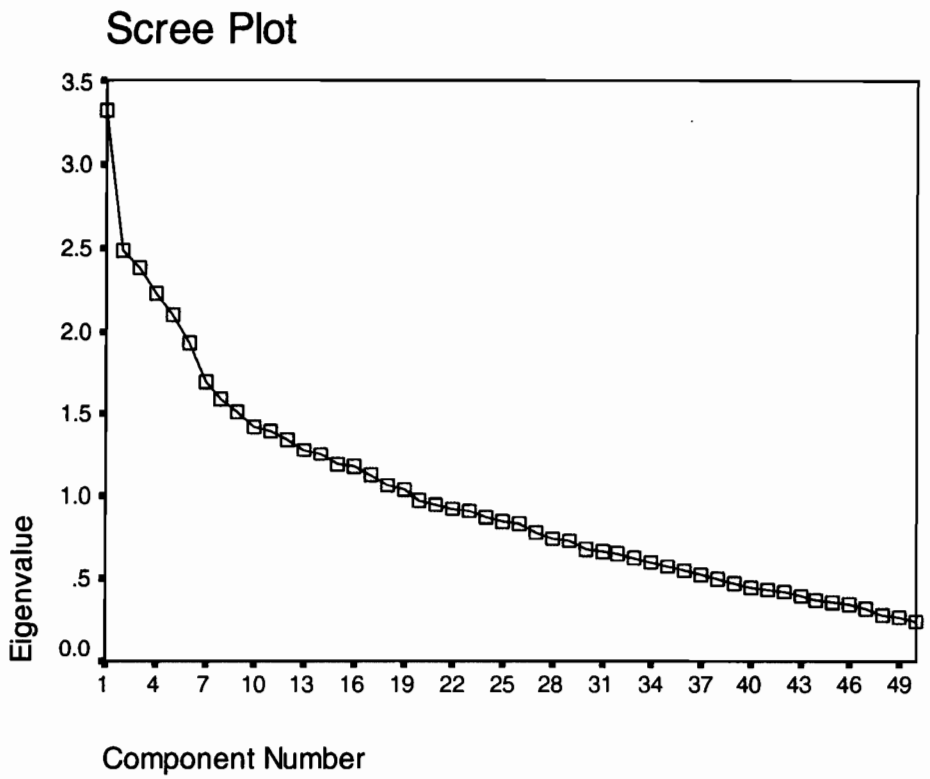


Table (8) Summary of Principle Component Analysis of GHQ 30 Questions

Principle component	**Eigenvalue**	**Main contributing items**	**Variance explained (%)**	**Cumulative variance explained (%)**
1	7.59	G10, G11, G26, G27, G28, G29, G30	25.3	25.3
2	1.99	G5, G18, G19	6.6	31.9
3	1.77	G4, G8, G15	5.9	37.8
4	1.48	G1, G13	4.9	42.8
5	1.22	G12	4.1	46.8
6	1.19	G13, G23, G25	3.9	50.8
7	1.08	G2, G21	3.6	54.4
8	1.07	G3	3.6	57.9
9	1.02	G24	3.4	61.4

Graph (1) Scree plot for principle component analysis for AQ 50 questions



(III) FACTOR ANALYSIS

Table (7) Summary of Principle Component Analysis of AQ 50 questions

Principle component	Eigenvalue	Main contributing items	Variance explained (%)	Cumulative variance explained (%)
1	3.31	A14, A17, A38	6.6	6.6
2	2.48	A31, A43	5.0	11.6
3	2.38	A6, A19, A23	4.8	16.4
4	2.23	A11, A15, A44	4.5	20.8
5	2.10	A12	4.2	25.0
6	1.93	A40, A50	3.9	28.9
7	1.70	A22	3.4	32.3
8	1.60	A27, A48	3.2	35.5
9	1.52	A9, A37	3.0	38.5
10	1.42	A7, A18	2.9	41.4
11	1.41	A36	2.8	44.2
12	1.35	A21	2.7	46.9
13	1.29	A24	2.6	49.4
14	1.27	A45, A46	2.5	52.0
15	1.20	A3, A8	2.4	54.4
16	1.19	A1	2.4	56.7
17	1.13	A33	2.3	59.0
18	1.07	A42	2.1	61.1
19	1.04	A34	2.1	63.2

(II) CORRELATION TESTS

Table (5) Correlation between AQ total score (Atot) and GHQ total score (Gtot):

Correlations

		ATOT	GTOT
ATOT	Pearson Correlation	1	.12*
	Sig. (1-tailed)	.	.03
	N	20	20
GTOT	Pearson Correlation	.12*	1
	Sig. (1-tailed)	.03	.
	N	20	20

*. Correlation is significant at the 0.05 level (1-tailed).

Tables (6) Correlation between AQ subscales and GHQ total score (Gtot):

Correlations

		ATOT	SS	AS	ATD	C	I	GTOT
ATOT	Pearson Correlation	1	.624**	.326**	.444**	.510**	.514**	.126*
	Sig. (1-tailed)	.		.000	.000	.000	.000	.036
	N	202	202	202	202	202	202	202
SS	Pearson Correlation	.624**	1	.169**	-.059	.237**	.141*	.223**
	Sig. (1-tailed)	.000	.	.008	.204	.000	.023	.001
	N	202	202	202	202	202	202	202
AS	Pearson Correlation	.326**	.169**	1	-.072	-.012	-.182**	.196**
	Sig. (1-tailed)	.000	.008	.	.156	.433	.005	.003
	N	202	202	202	202	202	202	202
ATD	Pearson Correlation	.444**	-.059	-.072	1	-.085	.138*	-.072
	Sig. (1-tailed)	.000	.204	.156	.	.114	.025	.155
	N	202	202	202	202	202	202	202
C	Pearson Correlation	.510**	.237**	-.012	-.085	1	.173**	.109
	Sig. (1-tailed)	.000	.000	.433	.114		.007	.061
	N	202	202	202	202	202	202	202
I	Pearson Correlation	.514**	.141*	-.182**	.138*	.173**	1	-.134*
	Sig. (1-tailed)	.000	.023	.005	.025	.007	.	.029
	N	202	202	202	202	202	202	202
GTOT	Pearson Correlation	.126*	.223**	.196**	-.072	.109	-.134*	1
	Sig. (1-tailed)	.036	.001	.003	.155	.061	.029	.
	N	202	202	202	202	202	202	

**. Correlation is significant at the 0.01 level (1-tailed).

*. Correlation is significant at the 0.05 level (1-tailed).

Table (3) Cross-tables of GHQ total scores (Gtot) by sex by college:

SEX	COLLEGE	Mean	Std. Deviation	N
Male	Science colleges	34.3036	11.95927	112
	Theoretical colleges	27.2778	8.85707	18
	Total	33.3308	11.80421	130
Female	Science colleges	36.9355	15.53196	62
	Theoretical colleges	33.1000	11.38664	10
	Total	36.4028	15.01610	72
Total	Science colleges	35.2414	13.35766	174
	Theoretical colleges	29.3571	10.03407	28
	Total	34.4257	13.08628	202

Table (4) Summary statistics for AQ total score (Atot) and GHQ total score (Gtot) by sex and college type

	Practical college			**Theoretical college**		
	N	**Mean**	**Std. dev**	**N**	**Mean**	**Std. dev**
ATOT						
Male	112	23.1	4.64	18	23.1	3.47
Female	62	22.0	4.52	10	22.2	2.78
GTOT						
Male	112	34.3	11.96	18	27.3	8.86
Female	62	36.9	15.53	10	33.1	11.39

ATOT (Kolmogorov-Smirnov test: $Z = 1.198$, $P > 0.05$) and GTOT (Kolmogorov-Smirnov test: $Z = 1.299$, $P > 0.05$) were normally distributed ., summary statistics are given in
Table (7 &13).

I. DESCRIPTIVE SUMMARY

Table (1) Summary statistics for age by college by sex:

	Practical				Theoretical			
	Count	Mean	Error Of Mean	Standard Deviation	Count	Mean	Error Of Mean	Standard Deviation
Male	112	23.16	0.45	4.81	18	20.06	0.55	2.34
Female	62	21.39	0.48	3.78	10	23.50	2.49	7.86

Table (2) Cross-table of AQ total score (Atot) by college by sex

SEX	COLLEGE	Mean	Std. Deviation	N
Male	Science colleges			
	Theoretical colleges		3.47211	18
	To-		4.48295	130
Female	Science colleges		4.51591	62
	Theoretical colleges	22.2000	2.78089	10
	Total		4.30189	72
Total	Science colleges		4.61288	174
	Theoretical colleges	22.7500	3.21599	28
	Total		4.43890	202

ing a student in a college like medical school can increase the level of stress due to the nature and demands of studying such subject.

In the mean time the correlation study between (Gtot) and AQ subscales indicated that GHQ total score (Gtot) was positively correlated to Social Skills (SS), Attention Switching (AS) and Imagination (I) (1-tailed). This meant that the GHQ totals scale was not statistically correlated to Attention to Detail items (ATD) and Communication (C) subscales (Tables 6)

Principle Component Analysis was used to examine and reduce the number of variables in each questionnaire (AQ total score (Atot) & GHQ total score (Gtot)). The Principle Component analysis for AQ questionnaire is presented in table 7 and Graph 1 (Scree plot). Nineteen Principle Components were extracted but there were no strong influences among these Principle Components with the First and second principal Components only accounting for 11.6 of the explained variance. In the correlation table it is noticed that AQ total score (Atot) correlate positively most with subscales of Social Skills (SS) (Pearson Correlation = 0.624), Imagination (I) (Pearson Correlation = 0.514) and Communication (C) (Pearson Correlation = 0.510) (all 1-tailed).

The Principle Component analysis of GHQ questionnaire is presented in table 8 and graph 2 (Scree plot). Nine Principle Components were identified and of these the first three components accounted for 37.8% of the explained variance. The first Principle Component was correlated with seven questions and accounted for 25.3% of the explained variance. Generally there was a weak influence from other Principle Components on this questionnaire. The component is made of 7 questions: G10: *finding it not easy to get on with other people?* G 11: *spent less time chatting with people?* G 26: *feeling not hopeful about own future?* G 27: not *feeling happy, all things considered?* G 28: *been feeling nervous and strung-up all the time*? G29: *felt that life isn't worth living?* G30: *found at times you couldn't do anything because your nerves were too bad?* This component is related to social incompetence, anxiety and depressive symptomatology.

highly competitive and highly technological. The second group belong to colleges which are not that competitive, and based mainly on lectures and theoretical studies rather than laboratories or modern technology. It is usually acceptable to say that students in the first group are usually considered more academically capable and of higher socio economic class.

Results were divided into three main groups; the (I) descriptive summary and the analytical studies including (II) Correlation tests and (III) the factor analysis test. The statistical comparison of the means has been included in the descriptive summary.

Subjects of this study are 130 males and 72 females. Average age is 22.35 (SD 4.64). Subjects from the science colleges are 175 and the theoretical studies subjects are 28 (table 1).

The average Autistic-Spectrum Quotient (AQ) total score (Atot) is 22.72 (SD 4.44) which followed normal distribution. There have been no cut off points used in the analysis, taking in consideration that both questionnaires (AQ and GHQ) are not diagnostic and that they are best used as indications of trends rather than being exact measures of a phenomena. There have been no significant differences in Atot among different groups in the sample (table 2).

The General health Questionnaire (GHQ) average total score (Gtot) is 34.43 (SD 13.09) and of normal distribution. The Gtot has been found to be significantly higher among sciences students compared with the theoretical studies students. It also higher in the females compared to the males, though not statistically significant (Table 3)

A positive correlation has been found between the AQ total scores (Atot) and GHQ total scores (Gtot) (Pearson's correlation one tailed test: $n = 202$, $r = 0.126$, $P = 0.036$) (table 5). However this significant correlation is found only when 1-tailed analysis is used. This is based on the assumption that the relationship between autistic spectrum traits, as represented by (Atot), and psychological distress, as represented by (Gtot), is basically a one direction relationship. This mean that higher autistic traits (higher Atot) is associated with higher psychological distress (higher Gtot) but not necessarily the opposite. People with less autistic traits can have other reasons for psychological distress. For example in this study be-

able. The scale asks whether the respondent has experienced a particular symptom or behaviour recently. Each item is rated on a four-point scale (less than usual, no more than usual, rather more than usual, or much more than usual); and for example when using the GHQ-12 it gives a total score of 36 or 12 based on the selected scoring methods. The most common scoring methods are bi-modal (0-0-1-1) and Likert scoring styles (0-1-2-3). The HGQ-30 was translated, standardised and validated in Al-Ain University, UAE [66]. The simple Likert scoring method (0-1-2-3) rather than the Bi-modal method (0-0-1-1) was used. Using the simple Likert scoring method, the best cut-off point of the GHQ-30 was 31/32 with a sensitivity of 83% and specificity of 83%. The total discriminatory power of the GHQ-30 was approximately 93%.

Data were analysed using the SPSS version 13.0. The underlying distributions were analysed for normality (Kolmogorov-Smirnov test) and summary statistics were generated for questionnaire totals and sub-groupings. Pearson's correlation one tailed test was used to examine relationships between questionnaire scores and between sub-scores. Principle Component Analysis was used to examine and reduce the number of variables in each questionnaire (AQ & GHQ). Independent T-Test has been used to compare between means of scores of a scale in different groups.

Results

By the end of time allowed to collect data, 206 responses were available. Most of the responses were from a limited number of colleges in the first group of colleges i.e. the practical sciences colleges. Most of the second group colleges (theoretical studies colleges) were in the middle of their exam season at the time and the distribution of questionnaires and it had to be put on hold.

Among the 206 responses 4 were not valid and 202 only were processed. The responses have been divided into two groups of colleges; the "practical sciences colleges" (mainly include medical school, school of sciences and technology, school of dentistry, and school of engineering). The other group usually called the "theoretical studies colleges" (mainly school of arts and literature, law and school of economics). The first group belong to top colleges in Egypt which are

cords the abnormal or autistic-like behaviour either mildly or strongly. Approximately half the items were worded to produce a 'disagree' response, and half worded to produce an 'agree' response, in a high scoring person with Asperger Syndrome (AS). This was to avoid a response bias either way. Following this, items were randomized with respect to both the expected response from a high-scorer, and with respect to their domain. The term 'quotient' is not used in the arithmetic sense (the result of dividing one quantity by another) but as derived from the Latin root quotients (how much or how many).

AQ is not diagnostic test, but it can serve as a useful instrument in identifying the extent of autistic traits shown by an adult of normal intelligence. A score of 32+ is used to be a useful cut- off or distinguishing individuals who have clinically significant levels of autistic traits. Such a high score on the AQ however, does not mean an individual has AS, since a diagnosis is only merited if the individual is suffering a clinical level of distress as a result of their autistic traits. The questionnaire has been standardised and validated by authors [53, 62], although their properties have not yet been systematically evaluated [67].

In a validation study of the AQ, Baron-Cohen and colleagues [62] showed that it does discriminate high-functioning people with autism from unaffected adults. It is also sensitive to differences in mild autistic-like features in the normal population, supporting the notion that it is possible to regard autism as the end of a quantitative continuum which start with normality. Men from the general population fell closer to the autistic end of the continuum than did women. Likewise, those engaged in careers in science and engineering scored closer to the autistic end of the continuum than those working in the arts [62].

The Arabic version of General health Questionnaire-30 [66,68]:

The General Health Questionnaire (GHQ) is a measure of current mental health and since its development by Goldberg in the 1970s it has been extensively used in different settings and different cultures. The questionnaire was originally developed as a 60-item instrument but at present a range of shortened versions of the questionnaire including the GHQ-30, the GHQ-28, the GHQ-20, and the GHQ-12 is avail-

This Research

It is of both theoretical and clinical importance to explore the milder forms of Autism. This can take place in two form, the mild forms of illness and the sub-clinical forms and expressions of such phenomenon. One form of sub-clinical expressions of this phenomenon is the "Broader Autism Phenotype" (BAP) which is a name for such sub-clinical forms of autism identified in relatives of autistic patients. Another way to study that is to trace such traits in "ordinary" and adequately functioning individuals. It seems that such traits can lead for discovering a new unexplored aspect of human development and to discover the implications on both behaviour and coping abilities of affected individuals. This research aims at further testing the usefulness of the concept of autistic mild traits as expressed in AQ on a sample of adequately functioning individuals with normal intelligence. It also aims to identify the presence or absence of psychological discomfort which could be associated with higher scores of AQ as detected by the Arabic Version of the General Health Questionnaire (AGHQ-30)[40].

This research also tries to introduce the AQ questionnaire to a new culture and language through using it on an Arabic (Egyptian) sample and introduce the concepts of AQ as a broader spectrum (from normality to severe autism) to the Egyptian literature and Arab scientific communities.

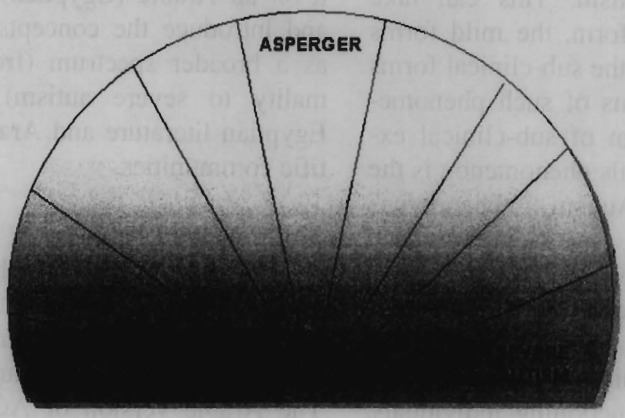
Methods And Material

A pack of questionnaires has been prepared. The package has included both the AQ questionnaire translated into Arabic by the author and The Arabic version of AGHQ-30. The packs were then distributed to the Zagazig university students by university officials.

Tools Used

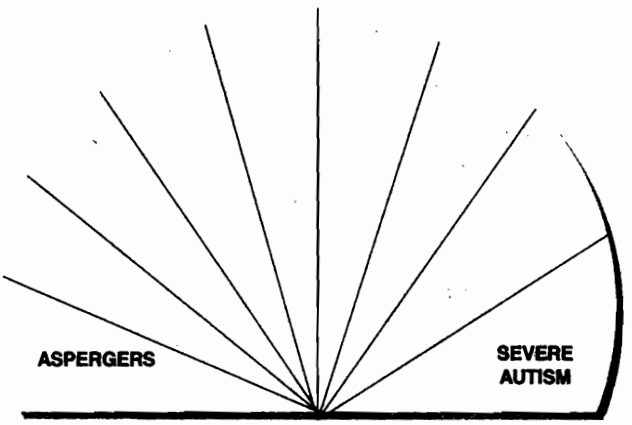
Autistic-Spectrum Quotient (AQ)[42]:

It comprises 50 questions, divided into 5 subscales of 10 questions each. The 5 subscales are: Social Skill, Attention Switching, and Attention to Details, Communication and Imagination. On the whole, the items are not couched in language of disability, but rather ask the respondent to agree or disagree with statements about personal preferences and habits. Each of the items scores 1 point if the respondent re-



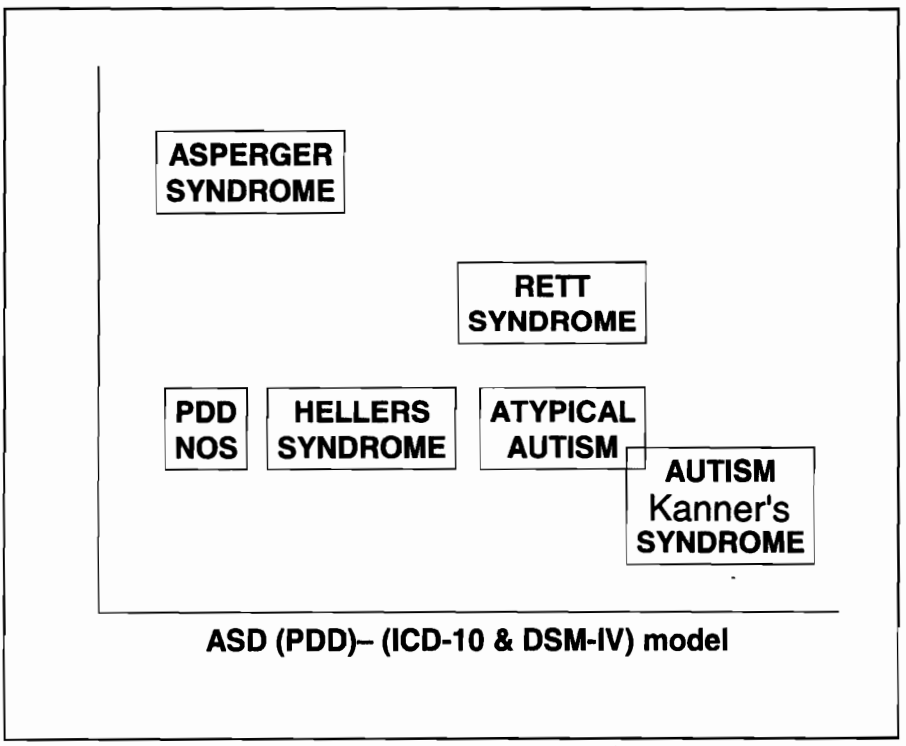
ASD – new model

ASD new model: A spectrum from normality to severe autism with Asperger Syndrome as a bridge.



ASD – Wing's model

ASD old model of L Wing: A spectrum between mild and sever forms of autism.



ASD (PDD) model as in ICD-10 and DSM-IV: A cluster of separate syndromes.

Kurita and colleagues[4] administered the Japanese version of the AQ (AQ-J) to 25 normally intelligent high-functioning pervasive developmental disorder (HPDD) patients (mean age, 24.2 years; 24 male, one female) and 215 controls (mean age, 30.4 years; 86 male, 129 female) randomly selected from the general population. The AQ-J had satisfactory internal consistency reliability (Cronbach's $\alpha > 0.70$ in the two groups), test retest reliability, and discriminate validity [i.e. the AQ-J score was significantly higher in the HPDD (mean, 29.6) than controls (mean, 22.2)]. At a cut-off of 26, the AQ-J had satisfactory sensitivity, specificity, and negative predictive value, but it had low positive predictive value (0.24) possibly due to the facts that the 25 mild HPDD patients scored lower and the controls scored higher on the AQ-J than British counterparts on the AQ. The AQ-J-21 (consisting of 21 items significantly associated with HPDD diagnosis) and the AQ-J-10 (consisting of 10 of the 21 items with an effect size >0.17) had higher, although not satisfactory, positive predictive values of 0.35 and 0.46 at cut-offs of 12 and 7, respectively, than the AQ-J. The AQ-J and two short forms are useful not to predict but to rule out mild HPDD, the most difficult part of HPDD to be distinguished from non-PDD conditions, in persons scoring under the cut-offs and to consider professionals' examination of HPDD in persons scoring over them, because their negative predictive values were satisfactory.

(AS) or high-functioning autism (HFA); randomly selected controls; students in Cambridge University; and winners of the UK Mathematics Olympiad. The study proved that the AQ is a valuable instrument for rapidly quantifying where any given individual is situated on the continuum from autism to normality.

Bishop and colleagues [53] used the Autism Spectrum Quotient (AQ), to assess features of the broad phenotype in adults, with parents of people with autism, to see whether they would be more likely to obtain extreme scores than a control group. The AQ was administered to parents of 69 people with an autism spectrum disorder and parents of 52 controls. On two of the five subscales of the AQ, social skills and communication, parents of people with autism obtained higher scores than control parents. The other three scales, attention to detail, attention switching, and imagination, did not differentiate groups. The correlation between social skills and communication scales was .663. Bishop and colleagues [53] concluded that the AQ scales can be combined to give an index of broad phenotype. The AQ appears to be sensitive to the broad phenotype, provided attention is restricted to the social skills and communication scales.

Wakabayashi and colleagues [64] report on a Japanese version of the AQ instrument (AQ-J). Three groups of Japanese subjects were assessed. Group 1 (n = 57 consisted of adults with Asperger Syndrome (AS) or high-functioning autism (HFA). The other two groups were control groups. Subjects of Group 2 (n = 194) were normal adults and those of Group 3 (n = 1 050) were students selected from five Universities in Tokyo and Chiba. The adults with AS/HFA had a mean AQ score of 37.9 (SD = 5.31), which was significantly higher than the two control groups (Group 2: X = 18.5, SD = 6.21, and Group 3: X = 20.7, SD = 6.38). While eighty-eight percent of the adults with AS/HFA scored more than 33 points, only 3% of subjects in the two control groups indicated those points. Among the controls, males scored slightly but significantly higher than females. The reliability of the AQ in both test-retest and inter-rater measures were significantly high.

Kurita and colleagues[65] administered the Japanese version of the AQ (AQ-J) to 25 normally intelli-

autism in a child are likely to be genetically mediated [53].

Of particular interest in the debate over the nature and range of expression of the underlying genetic liability for autism have been studies suggesting that there is a higher than expected rate of some psychiatric disorders in the relatives of autistic individuals. Some family studies, employing comparison groups and direct psychiatric assessment, have shown high rates of some psychiatric disorders in relatives ascertained through a single autistic probands [54-59].

Widening the concept of Autistic Spectrum to include normality:

Some researchers emphasised the view, which still under debate, that the spectrum including Autism and AS can be seen differently in a wider perspective to be a part of a bigger spectrum of social-communication disability. In this bigger spectrum, Asperger Syndrome lies as a "bridge" between Autism and Normality [22,34,60,61]. The continuum view shifts thinking away from categorical diagnosis and towards a quantitative approach[62]. This approach allows to identify "autistic traits" in normally functioning individual e.g. relatives of autistic patients.

Baron-Cohen and colleagues [62] developed a new instrument called: the Autism-Spectrum Quotient (AQ).The AQ is a self administered instruments for measuring the degree to which an adult with normal intelligence has the traits associated with the autistic spectrum Individuals. Baron-Cohen and colleagues [62] tried by developing this scale to identifying the degree to which any individual adult of normal IQ may have 'autistic traits', or what has been called 'the broader phenotype' [63]. This would be useful for both scientific reasons (e.g., establishing who is "affected" and who is not, or the degree of "caseness" of an individual, in scientific comparisons), and potentially for applied reasons (e.g., screening for possibly "affected" individuals to assist in making referrals for a full diagnostic assessment). For both of these reasons, the Autism-Spectrum Quotient was developed. The instrument's name was chosen because of the assumption, mentioned above, that there is an autism spectrum [34].

Baron-Cohen and colleagues [62] assessed four groups of subjects: adults with Asperger syndrome

The Broader Autism Phenotype (BAP):

The Broader Autism Phenotype (BAP) is a sub-clinical set of personality and other features that is thought to index familiality and/or genetic liability to autism [46]. These subtle abnormalities include social and communication impairments, as well as repetitive and stereotyped behaviours [47-49]. The precise boundaries and components of this lesser variant of autism are nevertheless still debated [50]. Social abnormalities have been found to include: lack of affection, social dysfunction, impaired friendships, impaired social play, odd behaviour and impaired conversation [50, 51]. Developmental language abnormalities, particularly language delay, language pragmatics [52] and language based cognitive deficits [48], also appear to be increased in siblings of probands with PDDs. Obsessional and repetitive behaviours in a clear-cut form seem to occur in only a small minority of relatives of autistic probands, and usually in association with either social or communication difficulties[47]. These also include: circumscribed interests, rigidity, obsessions/compulsions and repetitive interests and activities [50].

In the past the notion of a genetic basis to autism was dismissed because the disorder did not appear to run in families. In a genetic disorder, it is usually expected to find evidence of the same condition in parents and their children, and in affected cases and their siblings, yet in autism it is unusual to find families where more than one individual is affected. Indeed, people with autism are unlikely to have children, because their social impairments make it difficult to establish close relationships with others. Nevertheless, over the years there have been repeated suggestions that parents of children with autism may themselves show unusual traits reminiscent of autism, such as difficulties with social interaction and communication. In the past, there was a tendency to assume that abnormalities in parents were either direct causes of a child's autistic behaviour (e.g., the notion of the 'refrigerator parent' who is unable to engage emotionally with a child), or were reactions to the stress of rearing a child with a serious developmental disorder. However, over the last decade of the twentieth century new evidence has emerged to indicate that associations between unusual characteristics in a parent and

PDD, including AS, disintegrative disorder, and Rett syndrome. PDD-NOS were retained and could also be referred to as atypical autism [6].

However, with greater understanding and greater precision in the diagnosis of autism in the 1980s and early 1990s, there was a growing realization that many children who had autistic symptoms failed to meet criteria for that diagnosis. There was also dissatisfaction with the term "PDD-NOS," particularly among parents, who did not appreciate being told that their child had a "non-specific disorder". They were in fact looking for something specific to explain their child's developmental difficulties and to give them a concrete plan for intervention [40].

An alternative to classifying is to adopt a dimensional model. It is possible to view autism (or PDD) as a categorical distinction, in the broad sense, from other emotional and behavioural disorders, with the various types of PDD differing along a single underlying dimension of severity. This is, in fact, the theoretical model underlying the notion of autistic spectrum disorders. In this context, severity is usually measured using some cognitive measure, such as IQ or verbal ability, and the distinctions between PDD subtypes is largely arbitrary. For example, lower-functioning individuals with autism tend to have lower IQ and more severe autistic symptoms, whereas those at the other end of the spectrum (AS) or high-functioning autism) tend to have fewer autistic symptoms and to be brighter.

In fact, the utility or "validity" of classifying individuals as high- and low-functioning has quite a bit of evidence. Many studies have reported that lower-functioning individuals have more symptoms than those who are higher-functioning[41,42]. They also differ on etiological parameters [43], on natural history and outcome [44], and on response to treatment. A massive study undertaken by Rapin and others [45] of a large number of preschool children clearly demonstrates the existence of two "taxa," or subgroups, of children with autism: higher-functioning and lower-functioning. Ironically, the clinical distinction that has the most evidence of validity that is, high versus low functioning is not captured by either of the two official classification systems (DSM or ICD).

term PDD became included in the official classification system of the American Psychiatric Association (APA) in 1980. In 1988, Wing [34] has argued that rather than thinking rigidly in terms of a discrete syndrome of autism, we should be aware that there is a continuum of autistic disorders. She regards social impairment as the core symptom of such disorder. Children with this social impairment are characterised by a triad of deficits in social development. In each of these domains, a wide range of severity of impairment is recognised. Wing would regard a child as falling on the autistic continuum provided they showed this triad of social impairment, irrespective of other symptoms. However, she noted that impairments in other areas do tend to co-occur with the social triad, in particular repetitive and stereotyped activities, poor motor coordination and abnormal responses to sensory stimuli.

The World Health Organisation (WHO) and the American Psychiatric Association (APA) have defined Autism Spectrum Disorders (ASD) to mean the group of Pervasive Developmental Disorders (PDD) characterised by qualitative abnormalities in reciprocal social interactions and in patterns of communication, and by a restricted, stereotyped, repetitive repertoire of interests and activities. These qualitative abnormalities are a pervasive feature of the individual's functioning in all situations, although they may vary in degree [5,6]. Atypical autism in ICD-10 and DSM-IV has been included under the category of: 'Pervasive developmental disorder not otherwise specified' (PDD-NOS). PDD-NOS refer to what is a residual diagnostic category. ICD-10 provides the possibility for various forms of special coding-for example, failure to meet the onset criteria for autism, failure to meet developmental/behavioural criteria, failure to meet both, and so forth. Essentially this diagnostic concept refers to a 'sub-threshold' condition that has similarities to autism and the other explicitly defined PDDs but which does not meet criteria [23]. At the same time, several cross-sectional studies were being published showing symptom patterns in Asperger syndrome, atypical autism, and disintegrative disorder different from those seen in autism[35-38]. The DSM-IV task force completed a review of this literature[39], and the new DSM-IV again specified several other types of

world for many years, his description had points of difference, as well as similarity, to Kanner's report. For example, verbal abilities tended to be an area of strength, concerns typically did not arise until later in the preschool period, and there was a tendency for the condition to run in families-particularly in fathers. Lorna Wing's report [22] of Asperger's work and publication of a series of cases brought wider attention to the diagnostic concept. The validity of this condition, particularly apart from higher functioning autism, remains the topic of much debate [23]. A major complication has been the marked differences in the ways in which the concept has been used and its potential overlaps with other diagnostic concepts (e.g. schizoid personality [24], semantic-pragmatic disorder [25], and right hemisphere learning problems[26]. As a result, the literature on this condition is difficult to interpret, although several areas of potential differences from autism have been identified (e.g. neuropsychological profiles [27] and family history [28].

Heller [29] reported children who had a period of several years of normal development prior to a marked regression with loss of skills in multiple areas and minimal recovery. He initially termed the condition dementia infantilis; subsequently it has been referred to as Heller's syndrome, disintegrative psychosis, or childhood disintegrative disorder[30]. Once it develops the condition is indistinguishable from autism [31], but it is accorded separate diagnostic status since it appears distinctive in terms of onset and course [23]. In 1966, Andreas Rett described an unusual syndrome in girls. In addition some features suggestive of autism were present[32]. His findings were replicated and extended by Hagberg et al [33].

In the seventies of the last century, Lorna Wing completed her epidemiologic work on autism in Camberwell and delineated more carefully a subgroup of children with autism and autistic-like conditions [4]. Lorna Wing demonstrated a link, not between autism and schizophrenia, but rather, between autism and mental retardation. Wing also clearly formulated the notion of a triad of impairments-in socialization, in social communication, and in social play. This triad, then, translated into the concept of "pervasive developmental disorders" (PDDs) characterized by a similar (but somewhat different) triad. The

a true increase in incidence, but most of the increase can be accounted for by changes in case-finding methods and diagnostic criteria, and by differences in sample sizes, and the age range and intellectual ability of the populations studied [13]. The increase in numbers identified has led to a corresponding increase in demand for services. This is thought to be an effect of the broadness of the concept of autism from classical autism as a rare specific syndrome to become Autistic Spectrum[14].

The term 'autistic spectrum disorder' (ASD) provides a clearer representation of the continuity between autism and related disorders within the spectrum [4, 15] thus acknowledging the importance of the varied manifestation of these core deficits and the need to plan for the assessment, diagnosis and provision of intervention and support services for the much larger number of individuals and their families than might have been previously considered.

Development of the Autistic Spectrum Disorder (ASD) concept:

Autism was first recognized by Leo Kanner [16] Although children with autism had undoubtedly previously been observed [17], it was Kanner's particular genius to so precisely describe the condition. At the same time certain of his initial impressions were incorrect, for example his use of the term autism introduced an (originally) unintended confusion with schizophrenia and assumed that the children had normal intellectual potential. Subsequently, it became clear that autism and schizophrenia were distinct and that attribution of low IQ scores to 'poor testability' was incorrect [18]. Kanner also mentioned that parents were very well educated and successful; this led to a notion, common during the 1950s, that autism might somehow result from deviant patterns of care. A large body of evidence shows that this is most certainly not the case [19]. It is clear that families of children with autism come from all social classes and circumstances [20].

In the year following Kanner's description, a medical student, Hans Asperger [21], described four boys with marked social problems, unusual perseverative interests, and motor clumsiness. Although unaware of Kanner's work, Asperger used the term 'autistic psychopathy' to describe this condition. Unknown to the English-speaking

The autistic traits have been positively correlated to psychological distress as expressed by the score of GHQ.

Conclusion: The conclusion of the study suggests that the AQ is a potentially useful tool to assess autistic traits in highly functional individuals in Egyptian subjects. Tracing such traits in such individuals can be of significant clinical importance and would be a significant addition to clinical assessment methods which would enrich our knowledge of human behaviour.

Introduction

The criteria of autism have evolved over the almost 60 years since Kanner and Asperger first introduced the term autism for childhood disorders of social interaction. In response to research findings, there has been a progressive widening of diagnostic criteria [1]. Kanner and Eisenberg [2] identified as the two key features of autism; social aloofness and insistence on sameness, and to these Rutter [3] added impairment in language development. Wing and Gould [4] introduced the notion of an autism spectrum, covering a range of ability levels and severities, but characterised by qualitative impairments in social, communicative and imaginative development. It is this 'triad' of impairments that is captured in current international classification systems of the World Health Organisation's, "International Classification of Diseases", 10th edition (ICD-10) [5], and the American Psychiatric Association's "Diagnostic and Statistical Manual," 4th edition (DSM-IV) [6]. These reflect agreement in the field that Autistic Spectrum Disorders (ASDs) are characterised by early emerging (before 3 years old), qualitative (i.e. abnormal and not merely delayed development) impairments in social interaction, communication (and imagination); with restricted and repetitive interests and activities [7].

In recent reports, the prevalence of autism has varied widely from 5 to 67 cases per 10 000 children, an increase compared with the 3.5–4.5 cases per 10 000 children reported in the 1970s [8,9]. This high prevalence is consistent with a number of recent reviews that agree that the ASD affect approximately 60 per 10,000 under 8 years, of whom 10-30 per 10,000 children have narrowly defined autism [9-12]. The increase in prevalence might indicate

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Autistic Traits in Individuals with Normal Intellectual Level and Associated Psychological Distress:

A Pilot Study in an Arabic Culture

Omaima A Daoud, Mick Loughren , K Mansour, Abdulshafi Khashaba

السمات التوحدية لدى الأفراد ذوي المستوى العقلي العادى والإضطراب النفسي المرتبط بها:

دراسة أولية في ثقافة عربية

أميمة داود، ميك لوفرن، خالد منصور، عبد الشافي خشبه

Abstract

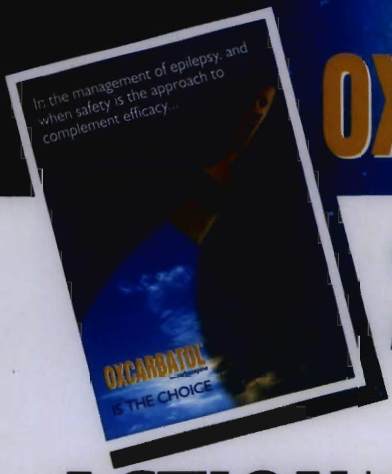
Introduction: The advances in the concept of Autism are among the most significant developments in the recent history of psychiatry. Psychiatric literature in the Arab world needs to put more emphasis and to encourage the local research in this area. This pilot study is adopting the wider concept of the Autistic Spectrum Disorders (ASD).

Aims: This study aims to introduce a new questionnaire i.e. The Autistic-Spectrum Quotient Questionnaire (AQ) to the psychiatric literature in the Arab World. The (AQ) questionnaire has been translated to Arabic and used on Egyptian participants. The questionnaire could not be formally validated as this is beyond the scope of this pilot study. The study has also been designed to find out if highly functioning Egyptian students could have some "autistic traits" and if these traits are of any clinical significance.

Method: 202 students from different colleges at Zagazig University in Egypt have fully completed the Autistic-Spectrum Quotient Questionnaire (AQ). The students have also completed the Arabic version of the General Health Questionnaire (GHQ-30) as a measure of any associated psychological distresses.

Results: The average Autistic-Spectrum Quotient (AQ) total score is 22.72 (SD 4.44) which followed normal distribution. The General health Questionnaire (GHQ) average total score is 34.43 (SD 13.09). A positive correlation has been found between the AQ total scores and GHQ total scores (Pearson's correlation one tailed test: P = 0.036). Results suggest that autistic traits as expressed by the score of AQ do present in a differed range in Egyptian students not dissimilar from the British and the Japanese studies.

Contents



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References:

1) Epilepsia, 1995; 36 supp. 2. 2) Epilepsy - Res. 1996 Nov; 25 (3): 299-319. 3) Clin - Pharmacokinet. 1996 Oct; 31 (4): 309-24. 4) Pharmacol Res. 1995 Mar - Apr; 31 (3/4): 155-62.

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Fluvoxamine: Safety Profile in Extensive Post- Marketing Surveillance

R. Buchberger
W. Wagner

The safety profile of the selective serotonin reuptake inhibitor, fluvoxamine, has been assessed in clinical and post-marketing studies. Post-marketing surveillance provides the opportunity to assess a drug's safety in every day clinical conditions in a much greater patient population than in clinical trials and therefore serves as a useful tool to detect signals for adverse effects with an incidence of less than 1:10,000. The safety profile of fluvoxamine was evaluated based on data from 17 years of global post-marketing surveillance in an estimated 28 million patients exposed to fluvoxamine. A total of 6,658 adverse drug reaction reports received from world-wide sources were reviewed and analysed. Post-marketing surveillance data co firmed the favourable safety profile already observed in clinic and post-marketing studies. A remarkably low level of suicida ity, switch to mania, and sexual dysfunction was found. Serot nin Syndrome appeared to be a very rare complication of fluvo amine treatment. No signals for drug interactions unknown far were identified. Withdrawal symptoms were observed in e eryday clinical conditions, which were generally mild and r solved spontaneously. However, no cases suggestive for drug d pendence have been reported. In conclusion, the data presente underlined that fluvoxamine offers a safe and well-tolerated o tion in the treatment of depression and obsessive-compulsive disord

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Page 2: Abstract in English (max: 250 words). It should follow a structured format (objectives, method, results and conclusion). It should include key words (max. 5).

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1. Zeigler FJ, Imboden, JB, Meyer E. Contemporary conversion reactions: a clinical study. Am. J. Psychiatry 1960; 116:901-10.
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