using the GHQ-12 and those scoring greater than 2 were then evaluated using the MINI. Following this those interviewed with the MINI were then evaluated by a psychiatrist within a 3-day period.

Results: Out of the total of 126 patients 78 scored greater than 2 on the GHQ-12. The mean age of these 78 patients was 47.8 (SD 16.4), 28.2% were male, 66.7% were married and 25.6% were employed. The diagnoses most frequently found on the MINI were major depression (50%) followed by generalised anxiety disorder (44.9%) and social phobia (17.9%). The most common diagnoses made by the psychiatrist were major depression (21.8%) followed by generalised anxiety disorder (16.7%) and dysthymia (16.7%). The sensitivity and the specificity of the most common diagnoses were major depression 94.1 and 62.2, generalised anxiety disorder 92.3 and 64.6, and social phobia 100 and 84.2 respectively. The positive predictive value and negative predictive for these disorders were as follows: major depression 41.0 and 97.4, generalised anxiety disorder 34.2 and 97.6, and social phobia 14.2 and 100 respectively.

Conclusion: The agreement between the MINI and the psychiatrist's diagnostic judgement may be considered as acceptable for the most prevalent disorders at the level of primary health care.

SEC55-3

THE MIND: AN UPDATE ON RATING SCALES FOR THE MINI COMPENDIUM

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During the 1970's the diagnostic criteria for mental disorders changed from an etiological principle (e.g. endogenous versus reactive depression) to a symptom-based principle. Thus, the Feighner criteria (Feighner et al 1972) introduced the screening diagnosis of mental disorders based on symptoms alone. A few years later the Research Diagnostic Criteria (Spitzer et al 1978) were released, which provided the foundation for the third edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-III, APA 1980). At the beginning of the 1990's the World Health Organization accepted the DSM-III principle when the tenth revision of the International Classification of Disease (ICD-10) was released (WHO 1993). However, both ICD-10 and DSM-IV (APA 1994) are still separated classification systems for mental disorders.

The MINI (International Neuropsychiatric Interview) developed by Sheehan and Lecrubier (1994) has both a DSM-IV and an ICD-10 version. The MINI was designed as a very brief structured interview for mental disorders to be used by clinicians after a brief training session. The MINI is mainly a tool for psychiatrists analogue to the PRIME-MD developed by Spitzer et al (1994) for general practitioners.

The objective of the MIND has been to offer quantitative assessments of mental disorders from DSM-IV or ICD-10 following as close as possible the MINI. The scales are all designed for clinicians (psychiatrists, psychologists, physicians as well as family doctors). The reference to DSM-IV or ICD-10 has been essential for the scale collection. It should be emphasized that this collection of rating scales is no attempt to replace MINI, DSM-IV nor ICD-10. The MIND is a collection of rating scales with a content validity equal to DSM-IV or ICD-10 but with a quantitative objective, e.g. to measure outcome of neuropsychiatric therapies.

SEC55-4

PRIME-MD: THE ICD-10 VERSION

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Aim of Study: To validate the Primary Care Evaluation of Mental Disorders (Prime-MD) diagnoses against ICD-10 diagnoses made by The Schedules for Clinical Assessment in Neuropsychiatry (SCAN) in patients commonly seen in primary care.

Methods: Current diagnoses were assessed by the Prime-MD and SCAN interviews in 36 women with somatoform disorders (fibromyalgia or functional dyspepsia) and 33 female random sample controls.

Results: Agreement and sensitivity showed great variability among the different diagnostic groups with highest degree of agreement and sensitivity for somatoform disorders and depressive disorder. Agreement and sensitivity was low for anxiety disorders (sensitivity = 0.36). Overall sensitivity for any psychiatric diagnosis was 0.41, specificity was 0.89. Specificity was high for all diagnostic categories and overall efficiency was good.

Comments: The validity of Prime-MD diagnoses in this population was good for depressive disorders, but not for anxiety disorders. This was mainly due to low sensitivity and might be related to the high prevalence of non fearful panic and somatized anxiety in this special population.

SEC55-5

PRIME-MD: FROM DSM-IV TO ICD-10

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It is well documented that primary care physicians constitute a low threshold service for patients suffering from mental disorders. For instance, panic disorder has a life time prevalence between 1 and 3% in the general population, but 7% of all patients attending general practitioners suffer from panic disorder, and some studies show that among frequent attendees of GPs the prevalence of panic disorder is over 20%. Yet knowledge and skills for recognising and treating mental disorders are not well developed in primary care and many patients suffering from depression, anxiety disorders and substance abuse remain undiagnosed - and therefore untreated. PRIME-MD (= Primary Care Evaluation of Mental Disorders) is a structured tool, tailored to assist the busy general practitioner in recognising the most common mental disorders among patients attending their surgery. After a short training period most GPs can use PRIME-MD reliably. It takes between 8 to 10 minutes to arrive at a diagnosis. The presently available DSM-IV version covers 5 diagnostic groups: major depression, anxiety disorders (panic disorder and generalised anxiety disorder), alcohol abuse, eating disorders and somatoform disorder. The system consists of a one page screen form, which the patient may fill in as a self-assessment device (but which can alternatively be used in the form of assisted self-rating). It takes 3 minutes on average to apply this screening sheet. In a second step, the GP carries out a short structured interview covering only those modules, which get a positive rating in the screening questionnaire. This interview takes around 5–8 minutes, depending on the number of suspected disorders. The application of PRIME-MD to 1000 GP patients in the US has shown that it is feasible to use the instrument in routine work and that it is valid when compared to diagnoses made by mental health

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professionals (Spitzer et al 1994). At present an ICD-10 version of PRIME-MD is being developed and tested.

(1) Spitzer, R.L., Williams, J.B.W., Kroenke, K. et al.: Utility of a new procedure for diagnosing mental disorders in primary care, JAMA 272 (1994) 1749–1756

S56. The treatment of panic disorder: rationale and empirical findings

Chairs: R Rosenberg (DK), JA den Boer (NL)

S56-1
EPIDEMIOLOGY AND COURSE OF PANIC DISORDER

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Approximately one in sixty to one in thirty persons in the general population suffers from panic disorder at one or the other time of their life. At least, this is what the two large epidemiological studies, the ECA (1.6% lifetime prevalence) and the NCS (3.6% lifetime prevalence) carried out in the USA over the last fifteen years, suggest. In countries as diverse as Korea and Italy, Germany and Puerto Rico, or Canada and Lebanon, the rates are in the same order. Female rates are usually at least twice as high as male rates. However, lifetime prevalence is a clinically not very useful concept, since it does not tell us anything about the severity, let alone about the chronicity of a disorder. Clinically panic disorder is regarded as a chronic and even progressing condition, starting with spontaneous panic attacks which are followed by situational attacks and phobic avoidance, leading, finally, to agoraphobia and a number of comorbid conditions, such as depression and substance abuse. But how frequent is this pattern? Is panic disorder really a chronic and progressing condition? And how many patients go on to develop agoraphobia? In the light of new research some of these questions can be answered in a differentiated way. First, the 12 months and one month prevalence rates of the ECA and the NCS study reveal that a large proportion of patients must have experienced a remission of panic disorder, since 12 months prevalence rates are around two thirds, and one month prevalence rates only around one third of the lifetime prevalence rates. Second, while in clinical populations up to 80% of all panic disorder patients suffer from agoraphobia, this proportion is much smaller in epidemiological studies, where it is less than one third. Finally, while, based on ECA data, around one in ten persons experiences at least one panic attack over their lifetime and 1.6% (i.e. one in six of these persons) develops panic disorder, among those 8.6% who *do not* meet criteria for panic disorder, around one in seven develops phobic avoidance, which is usually a disabling condition. While these epidemiological data already hint at the diversity of patterns of course, prospective follow-up study demonstrate that around one third of those who have developed full panic disorder remit, 50% show a waxing and waning course, and 20% experience a severe unremitting chronic course. Duration of illness and phobic avoidance seem to be the most relevant predictors for developing a chronic course. Clinically, it is therefore important to identify those patients who will have a severe and chronic course, at an early stage in order to concentrate therapeutic resources more vigorously on them.

S56-2
COMPONENTS IN 5-HT NEUROTRANSMISSION IN DEPRESSION AND ANXIETY

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We have suggested that anticipatory anxiety involves increased 5-HT release onto 5-HT_2 receptors. This also reduces the intensity and the probability of a panic attack. We have attempted to demonstrate these effects in studies using 5% CO_2 challenge in patients with panic disorder and normal volunteers.

We have previously reported that tryptophan depletion increases ratings of anxiety in patients with panic disorder when they breathe CO_2. Now, we report that increasing 5-HT_2 function using fenfluramine causes significant increases in anticipatory anxiety but on some measures attenuates the severity of anxiety during CO_2 challenge.

Recently we have investigated whether allelic variations in 5-HT-related genes predisposed to anxiety diagnoses in 100 samples from stressed women in the community. There are no clear cut associations with variants in the 5-HT transporter or in 5-HT_{2c} or 2a receptors. However, there are some intriguing hints. It is clear that 5-HT has different functions in different components of anxiety.

S56-3
NEUROBIOLOGY OF PANIC DISORDER

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Many different provocative agents, including metachlorphenypiperazine (mCPP), CO_2, lactate, yohimbine and cholecystokinin (CCK) have been used as panicogenic agents. Most of these challenge agents lack specificity which limits their use in identifying neurotransmitter systems or receptor dysfunctions involved in panic disorder (PD). CCK clearly has advantages over other provocative agents because it induces panic attacks dose-dependently and reliably in PD. A disadvantage, however, is that it lacks diagnostic specificity as it also induces panic attacks in social phobia and obsessive compulsive disorder. In a series of studies we have shown that CCK_B receptor antagonists like L-365,260 are able to block CCK_4 induced panic attacks, mitigates lactate induced anxiety, but is without anxiolytic effects in patients suffering from anxiety disorders.

After successful treatment with the SSRI fluvoxamine, CCK_4 was unable to induce panic attacks, which suggests a relationship between CCK and 5-HT neuronal systems.

A large number of studies has shown that several SSRI's are effective in reducing panic attacks and agoraphobia in PD. A number of patients, however, appears to be resistant to treatment. In depression, preliminary data have shown that the β-adrenoceptor/5-HT_A agonist pindolol reduced the latency and potentiated the response of several SSRI's. In view of the fact that there may be a shared biological diathesis between depression and PD we conducted a study in which we treated patients with PD with either fluvoxamine or a combination of fluvoxamine plus pindolol. In this pilot study no evidence was found for an accelerating or anxiolytic response of pindolol augmentation in PD. Possible reasons for this lack of efficacy will be discussed.

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