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Social Conflict-Management Training for In-patients with Complex Anxiety Disorders

S.A. Cohen, H. Böhme
Department of Psychiatry and Psychotherapy, University Hospital Essen, Barkhovenallee 171, 45239 Essen (Director Prof. Dr. M. Gastpar)

Background: In-patient psychotherapeutic treatment is offered for patients with therapy resistent, refractory or multimorbid anxiety disorders; a common feature is lack of social competence. Economic and social reasons are limiting the duration of hospital stay, so the primary goal is to enable and motivate patients to undergo further ambulatory treatment with techniques granting rapid success and a broad awareness of social problems and skills.
Methods: In an ongoing study 20 hospital patients, suffering from social anxiety coincident with affective, somatoform and personality disorders, some with secondary substance abuse, participated in social conflict-management training in addition to their regular psychiatric-psychotherapeutic treatment. Cognitive-behavioral techniques were amplified by elements of Moreno's psychodrama. After patient information and behavioral analysis, patients worked through psycho-educational lessons and defined socially challenging personal situations, one from their recent environment and one from their early experiences. Training consisted of weekly group sessions with role-playing and weekly homework sessions. The Liebowitz Social Anxiety Scale (LSAS) was administered weekly to gauge impact and change.
Results: Significant effects took place by the 5th session of training and remained stable in patients able to participate in 1 - 2 additional sessions. LSAS-scores were reduced by more than 1 SD, in both anxiety and avoidance sub-scores. Training was well accepted by patients, and experiences could be integrated flexibly into their regular behaviorally and analytically oriented therapies.
Conclusion: This improvement in patients with severe and complex anxiety disorders is comparable to published outcomes of phenelzine-, paroxetine- and cognitive-behavioral treatments of social anxiety and was achieved in a shorter time period. The results will have to be validated in further research with a larger number of patients and improved control-conditions.

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CREM gene promoter polymorphisms - Association with panic disorder

Domschke K, Kuhlenbäumer G, Schirmacher A, Garritsen HS, Franke P, Fritze J, Nöthen MM, Sibrowski W, Maier W, Beckmann H, Propping P, Ringelstein EB, Arolt V, Stögbauer F, Deckert J
Departments of Psychiatry, Universities of Muenster, Würzburg, Bonn and Frankfurt and Department of Neurology and Institute of Transfusion Medicine, University of Münster, Germany

A novel approach to define candidate genes for association studies in panic disorder is the behavioral characterization of knockout mice. Cyclo-AMP responsive element

modulator (CREM)-knockout mice exhibit significantly lower anxiety behaviour than wildtyp mice (Maldonado R et al., 1999). In addition CREM has been shown to play a pivotal role in the hypothalamo-pituitary-adrenal axis which is disturbed in panic disorder (Abelson et al. 1996). In the present study, we therefore searched for genetic variants in the human CREM gene in patients with panic disorder and their possible association with the disorder.
We performed a systematic mutation screening of the complete coding region as well as the regulatory regions of CREM and its alternative product ICER by means of SSCA in a sample of 40 patients with panic disorder (DSM-III-R). Polymorphisms were identified by direct sequencing. Allele frequencies were determined in the extended sample of 88 patients and matched healthy controls using RFLP and fragment analysis.
So far six polymorphisms have been detected: four single nucleotide substitutions in CREM promotors 1 and 4, one trinucleotide repeat polymorphism in ICER promotor 2 (8 – 11 ATT repeats) and a single aminoacid substitution in CREM exon glut II. A significant excess of the 8-repeat allele of the ATT trinucleotide repeat polymorphism in panic disorder was observed (p=0.02). This corresponded to an even more significant underrepresentation of the 9-repeat allele (p=0.01). To evaluate the relevance of these findings, replications in independent samples and a functional characterisation of the trinucleotide repeat polymorphism are necessary.

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Spontaneous electrodermal activity and thought content: Arousal, worry and GAD

Gerlach, A. L., Upatel, T. & Rist, F.
University of Münster & Christoph Dornier Foundation

Introduction: According to DSM-IV the defining feature of Generalized Anxiety Disorder (GAD) is constant worrying which is phenomenologically related to current concerns. Heightened physiological arousal is also a hallmark of GAD. In the ICD-10 hyperarousal is characterized as the defining feature of GAD. Nikula et. al. (1991,1993) demonstrated that non-specific electrodermal skin fluctuations (NSF) are associated with current concerns in students. We planned to evaluate the link between this physiological marker and worrying in GAD.
Method: We recruited 32 GAD patients and 31 controls. To induce worrying, we asked the participants to watch two different sets of video clips. One half watched clips which were related to worry topics, the other half watched clips related to neutral topics. Then participants were asked to report whatever came to their mind whenever they were prompted by a beep. Half of twenty prompts were triggered by NSF's measured online the other half was triggered randomly.
Results: We found no differences in heart rate and skin conductance levels between groups. NSF's were associated with more anxious thoughts and a reduced feeling of control in the GAD group. Controls reported less pleasant thoughts when prompted by NSF's as compared to random prompts.

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Intensity of anxiety after acute vestibular disorder as a critical life event

Godemann F, Linden M, Heipp E, Dörr P
Department of Psychiatry, Free University of Berlin

Background: Critical life events induce intense emotions. The most common emotion is anxiety. It remains open which factors determine intensity and course of anxiety after a critical life event. To elucidate such factors we studied patients during the six weeks after an acute vestibular disorder.
Methods: 69 patients were interviewed for anxiety at three time points during the six weeks after an acute vestibular event. Subsequently, the influence of cognitive, personality and disorder variables on intensity and course of anxiety were evaluated.
Results: Acute vestibular disorder is a critical life event because it is accompanied by intense anxiety. Catastrophisizing and dysfunctional cognitions ($p<0.01$) partly explain the intensity of anxiety directly after the loss of vestibular function. In most patients anxiety decreases in parallel with the remission of vertigo. A relevant subgroup (n=10, 14.5%) remains anxious or even experiences an increase in anxiety. Cognitive parameters, personality dimensions and coping styles do not explain the differential course of the experience of anxiety.
Conclusions: The acute vestibular disorder is successfully coped with by most patients. Dysfunctional fears contribute to the intensity of the acutely experienced anxiety due to this critical life event. It still remains open though why part of the patients do not recover from their acute anxiety.

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Anxieties of patients with an automatic implantable cardioverter defibrillator (AICD) - starting results in frame of an examination via longitudinal section

A. Grzbiela, W. Dengler, V. Kuhlkamp, P. Pauli, G. Wiedemann

University Tübingen, Dept. of Psychiatry and Psychotherapy, Tübingen, Germany
University Würzburg, Psychology I, Würzburg, Germany

Purpose of the investigation is to ascertain how anxiety-values of patients compare to average anxiety-values.
For that reason, we examinated patients immediately before the implantation. As investigation-instruments questionaires (ACQ, BSQ, MI, FQ, STAI-STAI-Trait, BDI, Hama, BAI, ASI), an interview, expecially developed for this purpose and SKID have been used. Results are still pending.
Possible results might be:
1. The anxiety-value of a defi-patient is low and shows no, respectively only minimal difference to the average value.
2. Anxiety-values are significantly higher than the average.
3. The patient shows anxieties, dominantly based on the cardiac disease or anxiety disorder.

The interview represents the first step of the longitudinal examination. After T1 (3 Months after the implantation) and T2 (1 year after the implantation) we are going to find out, whether anxieties increase and which factors are to be made responsible for this increase.

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C-type natriuretic peptide augments the anxiogenic action of cholecystokinin tetrapeptide in man

M. Kellner, A. Yassouridis, Y Hua, M. Wendrich, H. Jahn, K. Wiedemann
University Hospital Hamburg-Eppendorf, Dept. of Psychiatry and Psychotherapy, Hamburg, Germany

The B-type natriuretic peptide receptor agonist C-type natriuretic peptide (CNP) exerts an anxiogenic effect in rodents [1]. We investigated the action of CNP upon experimentally provoked panic attacks in humans using cholecystokinin tetrapeptide (CCK-4). – Twenty healthy male volunteers were pretreated with an intravenous infusion of 300 µg CNP or placebo from 11:40 to 11:10 in a double-blind, randomized and balanced design. At 11:00 all subjects were given 25 µg CCK-as an iv bolus. Provoked panic, anxiety and acute dissociative symptoms were measured by different psychometric scales before and after CCK-4. – CNP pretreatment significantly increased visual analogue scale ratings for „anxiety“ and Acute Dissociation Inventory scores (univariate F-tests $p<0,05$), while no effect upon panic symptoms was observed. – Our preliminary data support a role of B-type natriuretic peptide receptors in anxiety modulation in normal man. The anxiogenic effects of CNP observed are in contrast to antipanic effects of atrial natriretic peptide (ANP), another member of the family of natriuretic peptides that predominantly occupies the A-type natriuretic peptide receptor subtype [2]. The pharmacotherapeutic potential of A-type agonists and B-type antagonists needs further research.

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Maternal Psychosocial Distress after Birth of a Very-Low-Birth-Weight Infant

Kersting A MD, Reutemann M PhD, Schütt K MD, Wesselmann U MD, Hörnig-Franz I MD, Louwen F MD, Arolt V MD
University Münster, Department of Psychiatry and Psychotherapie

The birth of an infant with very low birth weight is a critical life event for which parents are usually not prepared. The life threatening immaturity and its consequences for the child's development are traumatic stressors with which mothers have to cope. The aim of this study was to measure the traumatic symptoms of mothers who gave birth to a very-low-birth-weight infant.
In a prospective longitudinal study 21 mothers whose infants were born before the 32nd gestational week and/or had a birth weight of less than 1500g were recruited. The control group comprised 39 mothers who had a normal, uncomplicated delivery. The traumatic experience, depression and anxiety were assessed 2-3 days, 14 days, 6 months and 14 months postpartum.
Preliminary results 2-3 and 14 days after birth show that mothers of preterm babies have to cope with higher levels of traumatic distress, depression and anxiety than mothers of mature babies. The results 6 and 14 months postpartum will reveal whether the traumatic experiences lead to posttraumatic stress disorder according to DSM-IV in some mothers.

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Clinical relevance of pain syndromes in posttraumatic stress disorder

Kohl F, Freiburg

The accepted main criteria of international classification systems such as flash backs, vegetative arousal and avoidance behavior typically dominate the symptomatology and the therapy of posttraumatic stress disorder. However, in our experience – and in agreement with parts of the literature - pain syndromes may be a relevant feature of the clinical symptomatology. Individual characteristics of pain and concomitant clinical symptoms may be diverse allowing for differential diagnostic classification as part of comorbid somatic disorder or as somatoform pain disorder.
We will present a clinical case which in our opinion will demonstrate, however, that the pain syndrome can also be interpreted as an intrinsic component of the posttraumatic stress disorder. Characteristics and course of the symptomatology provide plausible evidence that the pain syndrome in this case was an integral part of the posttraumatic stress disorder and that there was no need to assume a second independent disorder.
On the basis of the presented case and the relevant literature the role of pain syndromes in posttraumatic stress disorder will be discussed. We suggest to look more intensively for pain syndromes as facultative symptoms (or common comorbid disorder) of posttraumatic stress disorder and to take account of them early in the diagnostic and therapeutic process.

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Elektrophysiological findings in panic disorder

Silvia Kostorz [a], Christine Amrhein [a], Georg Wiedemann [a], Paul Pauli [b], Wilhelm Dengler [a]
[a] University Hospital of Psychiatry and Psychotherapy, University of Tuebingen,
Osianderstr. 24, 72076 Tübingen, Germany
[b] Institute of Psychology, Biological and Clinical Psychology, University of Wuerzburg, Germany

Our previous study revealed a frontal brain asymmetry (FBA) with a stronger right frontal activity in panic patients as compared with control subjects. This FBA seems to be associated with negative emotions and is an indicator of the activity of an avoidance/withdrawal system.
This study was designed to replicate these effects and further examine whether the FBA is caused by the arousal or the valence of the situation.
17 panic patients (diagnosed according to DSM-IV) and 16 healthy control participants matched for sex, age and education were examined. Spontaneous EEG was recorded during exposure to several picture stimuli and during rest. Alpha activity at frontal (F7-F8, F3-F4, T3-T4) and parietal (P3-P4) electrodes was analysed. The emotional state was assessed with questionnaires.
Preliminary results revealed a significant Group x Hemisphere x Region x Condition interaction which was caused by a Hemisphere x Region x Condition interaction in panic patients. Only panic patients showed a FBA while exposed to a panic provoking picture. The found FBA in panic patients indicates a pathological processing of panic relevant stimuli which may be a characteristic of this disorder.

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Investigation of a NET gene variant and cardiovascular autonomic function in patients with panic disorder

Sand P, Flachenecker P, Jacob C, Stöber G
Dept. of Psychiatry, University of Würzburg, Füchsleinstr. 15, 97080 Würzburg

Background: Several lines of evidence suggest that norepinephrine (NE) is implicated in the etiology and symptomatology of panic diosrder (PD). At the cellular level, functional noradrenergic neurotransmission is closely related to synaptic reuptake of NE as mediated by the NE transporter (NET).
Methods: We investigated a NET variant (G1287A) with respect to autonomic function in 14 PD patients (12 female, 2 male) by means of a PCR-based RFLP. Cardiovascular autonomic dysfunction was assessed by standard tests of parasympathetic and sympathetic function.
Results and Discussion: The variant allele accounted for 17.8% of total alleles. In patients homozygous for the variant allele (n=2), measures of parasympathetic function were reduced as determined by deep breathing and a subscore on a composite autonomic rating. Sympathetic activity was increased as measured by blood pressure responses to active change of posture and sustained handgrip; however, these differences failed to reach statistical significance. No difference was seen for any autonomic parameter with respect to additional agoraphobia. Genotype and allele distributions were comparable to frequencies found in previous reports in Caucasian control populations. Our results do not disprove the impact of the NET variant on the complex physiology of autonomic function and warrant further investigations in larger samples.

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Anxiety disorders in patients with chromosomal aberrations

Schlesiger C, Nyhuis P, Panhorst H, Michael N, Erfurth A, Deckert J
Department of Psychiatry, University of Münster

In the search for the molecular basis of mental disorders phenotypic characterization of patients with chromosomal aberrations has been widely applied for defining potential

candidate loci and genes in schizophrenic and affective disorders.
Recently, a genomic duplication on human chromosome 15q24-26 has been suggested as a susceptibility factor for panic and phobic disorders associated with joint hypermobility syndrome and mitral valve prolaps (Gratacos et al., Cell 106:367-378, 2001). In fact, increased anxiety and anxiety disorders have been reported for a series of chromosomal aberrations. They include Turner syndrome and fragile x syndrome on chromosome Xq, Prader-Willi-syndrome on chromosome 15q11 and velo-cardio-facial syndrome on chromosome 22q11. The phenotypic expression of anxiety varies from social phobia in individuals with premutation status in fragile x syndrome over panic disorder in the 15q24-26 duplication to separation anxiety in velo-cardio-facial syndrome. Some of these anxiety syndromes have been shown to occur independently from intellectual impairment and thus appear not to be secondary to it.
In a replication study on a possible association of panic disorder with joint hypermobility syndrome we have systematically investigated patients and gender- and age-matched controls for joint hypermobility syndrome according to the criteria of Beighton. Preliminary results (n=10) do not support the hypothesis of an increased rate of joint hypermobility syndrome in our German sample. A deletion of the short arm of chromosome 18 (18p) was observed in a woman who was admitted to our department with phobias and depression as well as intellectual impairment. This observation is consistent with linkage findings on chromosome 18p in bipolar affective disorder and harm avoidance. Searching for the gene on chromosome 22q11.2, which causes increased separation anxiety in velo-cardio-facial-syndrome, we studied the functional valine/methionine polymorphism of the COMT gene. In accordance with observations of Hamilton et al. (2000) we observed an association in women with panic disorder. However, this association was due to an excess of the functionally more active valine COMT variant and not the functionally less active methionine COMT variant.
Genes contributing to the pathogenesis of anxiety disorders in most instances do so to a rather small degree. Linkage studies have so far failed to provide unambiguous results. Observations of increased anxiety and anxiety syndromes in patients with chromosomal aberrations may thus help to define candidate genes for molecular studies.

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Heart rate variability in experimentally induced panic attacks

A. Siegmund[1], R. Heese[2], F. Strian[2], A. Ströhle[2]
[1]Justus-Liebig-University, Gießen; [2]Max Planck Institute of Psychiatry, Munich

To further study the pathophysiology of panic disorder and possible new treatment approaches, panic attacks can be experimentally induced. The aim of this study was to discern the differential effects of two different panicogenic challenges on autonomic function in 23 patients with panic disorder and 13 healthy control subjects. In a double-blind randomized design each subject received either placebo, sodium lactate or 25 µg cholecystokinin tetrapeptide (CCK-4) intravenously on separate days. Heart rate variability was assessed using an analogous ECG and subsequent power spectral analysis of RR-intervals. 17/18 of the patients and 2/3 of the controls developed a panic attack following the administration of CCK-4/sodium lactate. In patients and controls the heart rate increased in both active treatments conditions. In both groups a shift from mid frequency power (PMID) to high frequency power (PHIGH) occurred after sodium lactate, indicating vagal predominance. Following CCK-4 only the patients showed an increase of the PLOW (low frequency power): PHIGH ratio, representing increased sympathic activity. The differential effects of the two panicgenic substances point to differential mechanisms in the development and regression of panic attacks. Whether the PMID to PHIGH shift during lactate induced panic attacks represents counterregulatory mechanisms and whether they may be used in the treatment of panic disorder is studied.

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$GABA_A$ receptor modulatory neuroactive steroids in panic disorder

A. Ströhle, E. Romeo[1], A. Pasini, F. Holsboer, R. Rupprecht
Max Planck Institute of Psychiatry, Munich, Germany
[1]University Tor Vergata, IRCCS Santa Lucia, Rome, Italy

Certain metabolites of progesterone such as 3α,5α-tetrahydroprogesterone (3α,5α-THP, allopregnanolone) and 3α,5β-tetrahydroprogesterone (3α,5β-THP, pregnanolone) are potent positive allosteric modulaturs of $GABA_A$ receptors. Altough animal studies suggest anxiolytic properties of these endogenous modulators of central nervous excitability no data exist whether they are also involved in anxiety disorders. We quantified the concentrations of 3α,5β-THP, 3β,5β-THP and the functional antogonistic isomer 3β,5α-tetrahydroprogesterone (3β,5α-THP) and their precursors of patients with panic disorder and healthy control subjects during experimentally induced panic attacks and during paroxetine treatment using a highly sensitive gaschromatography/mass spectrometry analysis.

Unexpectedly patients with panic disorder had significantly increased concentrations of the positive allosteric modulators 3α,5β-THP and 3β,5β-THP together with significantly decreased concentrations of 3β,5β-THP, a functional antagonist for $GABA_A$ agonistic steroids, which might results in an increased GABA-ergic tone. Paroxetine treatment did not affect neuroactive steroid concentrations which were highly stable for 24 weeks. In contrast, sodium lactate- and CCK-4 induced panic attacks of patients were accompanied by pronounced decreases in the concentrations of 3α,5β-THP and 3β,5β-THP and a concomitant increase in the concentrations of the functional antagonist isomer 3β,5β-THP which might result in a drastically decreased GABA-ergic tone. No changes in neuroactive steroid concentrations could either be observed following placebo in patients panic disorder or following placebo, lactate or CCK-4 in control subjects. The association between changes of plasma neuroactive steroid concentrations with panic attacks and the well documented pharmacological properties of these compounds as $GABA_A$ receptor modulators suggest a causal relationship and may provide a lead for new drugs for the treatment of anxiety disorders such as panic disorder.

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Impact of GABAergic treatment on CCK-4 induced anxiety

P. Zwanzger, D. Eser, T. Baghai, F. Padberg, C. Schuele, H.-J. Möller and R. Rupprecht,
Department of Psychiatry, Ludwig-Maximilian University of Munich, Germany

Objectives: There is increasing evidence that dysregulation of the GABAergic system plays an important role in the pathophysiology of panic disorder. Selective enhancement of GABAergic neurotransmission has been shown to improve anxiety in experimental animals and in patients with panic disorder [1;2]. To evaluate the impact of selective GABA enhancement on anxiety in humans we investigated anxiolytic effects of vigabatrin and tiagabine on cholecystokinin-tetrapeptide (CCK-4) induced panic [3].
Methods: In study I ten healthy volunteers received vigabatrin 2 g daily for one week. In study II 15 healthy volunteers received tiagabine 15 mg daily for one week. A CCK-4 challenge was performed before and after treatment. Panic was assessed using the API- and PSS-score. Blood samples were taken for determination of ACTH and cortisol plasma levels.
Results: Subjects reported a marked reduction of CCK-4 induced panic symptoms and anxiety after both vigabatrin and tiagabine administration. API and PSS–scores showed a significant reduction after one week of treatment. Moreover, we observed a marked and significant blunting of CCK-4 induced HPA stimulation after vigabatrin treatment, whereas HPA stimulation patterns after tiagabine treatment were not affected.
Conclusion: Our data show a marked improvement of CCK-4-induced panic-symptoms following GABAergic treatment in healthy volunteers and suggest that GABAergic drugs might be useful in ameliorating panic symptoms also in patients with PD.

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Update on Panic Disorder

Symposium

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Pharmakotherapie der Panikstörung

Borwin Bandelow
Psychiatrische Klinik der Universität Göttingen, von-Siebold-Str. 5, D-37075 Göttingen

Für die Behandlung der Panikstörung kommen in erster Linie Antidepressiva wie die selektiven Serotoninwiederaufnahmehemmer (SSRI) und trizyklischen Antidepressiva (TZA) zum Einsatz. Die Wirkung ist für SSRI wie Citalopram, Fluvoxamin, Fluoxetin, Paroxetin und Sertralin sowie für TZA wie Imipramin und Clomipramin belegt. In Deutschland sind Citalopram, Paroxetin und Clomipramin zugelassen. Bei Therapieresistenz können Benzodiazepine wie z.B. Alprazolam verwendet werden, wenn sich in der Anamnese keine Hinweise auf eine Abhängigkeitsentwicklung (Hochdosisabhängigkeit) finden. Auch zur Überbrückung der Zeit bis zum Wirkungseintritt der Antidepressiva können kurzfristig Benzodiazepine gegeben werden.
Wenn andere Mittel versagen, kann unter fachärztlicher Kontrolle ein Versuch mit dem irreversiblen MAO-Hemmer Tranylcypromin unternommen werden. Allerdings existiert nur für den in Deutschland nicht erhältlichen MAOH Phenelzin ein Wirksamkeitsnachweis.
Medikamentöse Maßnahmen sollten immer durch eine Psychotherapie unterstützt werden. Unter den nichtmedikamentösen Verfahren hat sich vor allem bei Patienten mit Agoraphobie die Expositionstherapie bewährt. Panikattacken, die „aus heiterem Himmel", d.h. nicht in den typischen Agoraphobie-Situationen, auftreten, können mit Hilfe der kognitiven Verhaltenstherapie behandelt werden.

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Psychodynamische Therapie der Panikstörung

PD Bassler, Klinik für Psychosomatische Medizin der Universität Mainz

In dem Vortrag werden zunächst die wesentlichen pathogenetischen Annahmen zur Panikstörung aus Sicht der psychodynamischen Psychotherapie skizziert. Drei sich wechselseitig ergänzende Konzepte spielen hier eine Rolle: 1. das Konfliktmodell (Angst als Folge unbewusster intrapsychischer Konflikte), 2. das Bindungsmodell (unsichere Bindungserfahrungen in der Kindheit und Jugend begünstigen Angststörung im Erwachsenenalter), 3. Strukturmodell (unzureichende Entwicklung von Ichstrukturellen Ressourcen zur Angstbewältigung – wie z.B. bei der Borderline-Persönlichkeitsstörung). Abgeleitet von diesen Modellvorstellungen werden einige psychodynamische Prinzipien bei der Behandlung der Panikstörung (mit und ohne Exposition) vorgestellt. Ein ambulantes psychodynamisches Behandlungskonzept, das bis etwa 50 Therapiestunden umfasst, wird eingehend erörtert und ergänzend hierzu empirische Ergebnisse diskutiert. Dabei wird deutlich, dass die Einbindung von angstkonfrontierenden Maßnahmen für moderne psychodynamische Behandlungskonzepte unverzichtbar ist, allerdings wesentlich ergänzt um die Aufklärung bzw, Bearbeitung der ursprünglich angstauslösenden unbewussten Konflikte und ihrer korrespondierenden Phantasien.

Im Schlussteil des Vortrags werden noch einige Überlegungen von Grawe (aus: Grawe, Psychologische Therapie, Hogrefe 1998) zu einem erweiterten ätiologischen und therapeutischen Verständnis von Angststörungen (am Beispiel der Agoraphobie) vorgestellt.

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Entstehungsbedingungen der Panikstörung

Jürgen Deckert
Klinik und Poliklinik für Psychiatrie und Psychotherapie, Universitätsklinikum Münster, Albert-Schweitzer-Straße 11, D-48149 Münster

Panikstörung ist eine Angsterkrankung, die charakterisiert ist durch situationsunabhängige Panikattacken, Erwartungsangst und zu einem hohen Prozentsatz auch durch Vermeidungsverhalten. Ihre Lebenszeitprävalenz liegt bei ungefähr 1-2%. Frauen sind im Verhältnis 2:1 im Vergleich zu Männern überwiegend betroffen. Der Erkrankungsbeginn liegt in der Regel im frühen Erwachsenenalter. Der Verlauf ist unterschiedlich und variiert von kompletter Remission bis zu chronischen Verläufen. Im höheren Alter wird das Krankheitsbild praktisch nicht beobachtet. Als Vorläufer im Kindes- und Jugendalter gelten gehemmtes Verhalten und Trennungsangst, eine Entsprechung im höheren Alter sind möglicherweise hypochondrische Befürchtungen. Es besteht eine erhebliche Komorbidität zu Phobien, Depressionen und Alkoholabhängigkeit.
Zwillingsuntersuchungen sprechen für einen erheblichen Beitrag genetischer Faktoren. Dieser wird auf ungefähr 40% geschätzt. Eigentlich familiäre Formen sind dabei selten, die Angaben aus Familienuntersuchungen schwanken zwischen weniger als 10 % und bis zu 25 %. Möglicherweise eine höhere Familiarität weist eine Subgruppe von Patienten mit einer Auslösung der Panikattacken durch CO_2 aus. Kopplungsuntersuchungen haben allerdings bisher keinen eindeutig replizierten Befund erbracht. Dies wird so interpretiert, dass der Beitrag einzelner Gene eher klein anzusetzen ist. Erste replizierte Befunde haben Assoziationsuntersuchungen zu Genen erbracht, über deren Proteine Medikamente wirken oder Attacken ausgelöst werden können. Hierzu gehören Gene der katecholaminergen Neurotransmission wie MAO-A und COMT, aber auch der Adenosin A_{2A} Rezeptor und Cholezystokinin. Die Risikoerhöhung um einem Faktor 2-4 ist dabei jeweils klein - ApoE bei Alzheimer erhöht im Vergleich dazu das Risiko um einen Faktor 10. Diese Befunde sind so nicht für eine prädiktive Diagnostik geeignet, legen aber möglicherweise die Basis für die Entwicklung neuer Psychopharmaka. Auffällige Befunde in bildgebenden Untersuchungen weisen auf eine mögliche Rolle unspezifischer Hirnschädigungen als Risikofaktor hin.
Zwillingsuntersuchungen belegen aber auch die wesentlichen Rolle individueller traumatischer Lebensereignisse, deren Beitrag an der Entstehung auf bis zu 60 % geschätzt wird. Solche sind wahrscheinlich Verlust eines Elternteils, Trennung der Eltern, aber auch Gewalt einschließlich sexueller Gewalt. Tierexperimente belegen die dauerhafte Auswirkung solcher Trennungserlebnisse auf endokrine Parameter der Hypothalamus-Hypophysen-Nebennieren-Achse, aber auch elektrophysiologische Parameter im Hippocampus. Multiple, kleinere Traumata einschließlich der Panikattacken selbst wirken sich möglicherweise nach dem Modell des Kindling aus, das ursprünglich zur Erklärung der Rolle von Traumata bei der Entstehung von Depressionen entwickelt wurde. Diese Veränderungen können im Tierversuch durch Quenching umgekehrt werden. Im Gegensatz dazu liefern die Zwillingsuntersuchungen wenig Hinweise auf eine Rolle des Erziehungsverhaltens der Eltern, das in Kohortenstudien von einem Teil der Patienten als eher kontrollierend und überprotektiv erinnert wird.
Verlaufsprägende Faktoren können Persönlichkeitsfaktoren wie Neurotizität, aber auch die Komorbidität mit anderen psychischen Erkrankungen und das Verhalten von Bezugspersonen sein. Mögliche Erklärungen des Geschlechtsunterschiedes neben soziokulturellen Faktoren sind genetische Faktoren wie sie sich zum Beispiel bei knockout-Mäusen beobachten lassen.
Zusammenfassend geht man heute von einem Zusammenwirken komplexer Entstehungsbedingungen aus. Verschiedene Aspekte des Krankheitsbildes haben wahrscheinlich unterschiedliche Ursachen, die ihrerseits wiederum nicht krankheitsspezifisch sein müssen. Faktoren, die zur Entstehung beitragen, unterscheiden sich möglicherweise von denen, die den Verlauf prägen.
Für die Therapie der Panikstörung ergibt sich daraus die Forderung eines integrativ und individuell ausgerichteten Ansatzes.

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Verhaltenstherapie bei Agoraphobien und Panikstörungen

W. Fiegenbaum, Universität Marburg
F. Rist, Universität Münster

Aufbauend auf psychophysiologischen, lerntheoretischen und kognitven Modellvorstellungen zur Erklärung von Agoraphobien und Panikstörungen hat die Verhaltenstherapie Veränderungsprinzipien entwickelt, die in den letzten 20 Jahren umfangreich empirisch überprüft und optimiert wurden.
Kernstück der Behandlung ist die Konfrontationstherapie, deren Wirkung auf Mechanismen der physiologischen Habituation, der kognitiven Umstrukturierung und der Dissonanzreduktion zurückzuführen ist. Im Vortrag werden die erprobten Bausteine der Konfrontationstherapie, wie sie in der Christoph-Dornier-Stiftung durchgeführt werden, dargestellt und erläutert:

- Diagnostik und Indikationsstellung
- Kognitive Vorbereitung auf eine Konfrontationstherapie: Gemeinsame Entwicklung eines plausiblen Erklärungsmodells, stringente Ableitung des Therapierationales und motivationale Vorbereitung auf die Expositionsübungen
- Planung, Gestaltung und Durchführung der Expositionsphase: Techniken der Angstintensivierung und der Kontrolle von Vermeidungs- und Fluchtverhalten, von Angst bzw. Angstunterdrückung während der Expositionsübungen; begleitende kognitive Integration neuer Erfahrungen
- Gestaltung der Transfer- und Stabilisierungsphase

Einzelne Wirkvariablen wie z.B. die Dauer der Konfrontation oder die Intensität der angstauslösenden Reize werden eingehend erläutert. Besonders eingegangen wird auf die kognitive Arbeit vor, während und nach den Expositionsübungen.
Zahlreiche Studien belegen, daß mit Expositionsverfahren im Rahmen kognitiv-behavioraler Therapieverfahren relativ schnell eine deutliche und anhaltende Reduktion der Angstsymptomatik erzielt werden kann. Dies gilt sowohl für das Auftreten von Panikattacken wie auch für die Reduktion agoraphobischen Vermeidungsverhaltens. Die langfristige Stabilität des Therapieerfolges konnte in einer Studie mit 416 unselegierten Patienten der Christoph-Dornier-Stiftung mit der primären Diagnose "Panikstörung und Agoraphobie" über Katamnesezeiträume von 6 Wochen, 1 Jahr und 3 Jahren belegt werden. Symptomverschiebung wurde in keiner Längsschnittstudie beobachtet.
Die Konfrontationsbehandlung gilt nicht nur als die Methode der Wahl bei Panikstörungen und Agoraphobien. Auch bei Zwangserkrankungen (in Kombination mit der Reaktionsverhinderung), bei spezifischen Phobien (häufig in Kombination mit „applied tension“), bei sozialen Phobien, generalisierten Angststörungen und postraumatischen Belastungsstörungen stellt die Konfrontationstherapie ein wesentliches Behandlungselement dar.

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