De farmacologische behandeling van trauma-klachten, iHB behandeling van nachtmerries en slaapproblemen

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Crisis in pharmacotherapy of PTSD

# Only two medications approved
# Off label polypharmacy, little empirical evidence
# Research has stalled, void in new drug development

Correspondence

It is Time to Address the Crisis in the Pharmacotherapy of Posttraumatic Stress Disorder: A Consensus Statement of the PTSD Psychopharmacology Working Group

To the Editor:

There is an urgent need to address a critical lack of advancement in the psychopharmacologic treatment of posttraumatic stress disorder (PTSD). The clinical, social, and financial burden of ineffective treatment of PTSD is enormous (1-8). The impact of PTSD morbidity and mortality is further magnified by its substantial detriments in family, workplace, and societal contexts (9). For the Department of Veterans Affairs (VA) and Department of Defense (DoD), i.e., institutions that are key players for the expression of the national debt to military personnel who developed PTSD as a consequence of their military service, the need to help those people has taken on significant priority. One in 10 VA healthcare users have the diagnosis of PTSD, which includes one in four federal funding agencies in research on medical treatment of military personnel and veterans with PTSD have yet to bear fruit in the form of new validated pharmacotherapies for PTSD.

Peculiarly, this is a time of tremendous progress in the basic neuroscience of stress and PTSD that could inform the identification of novel therapeutic targets (14,15). There is a longstanding translational neuroscience tradition in PTSD research (16,17). However, recent developments in the genetics and epigenetics of PTSD (18-20), progress with animal models (21), the emergence of the first molecular analyses of postmortem brain tissue from people with PTSD (22), an expanding number of brain molecular targets probed with positron emission tomography imaging (23), the refinement of the neural circuitry of PTSD through structural (24) and functional (25) brain imaging, and the refinement of behavioral paradigms to study many relevant dimensions of the PTSD syndrome, partly in the context of the National Institute of Mental Health (NIMH) Research Domain Criteria initiative, all contribute to the knowledge of the field to fuel novel PTSD research.

Recommendations

Krystal et al., *Biol Psych*, 2017
Medications filed as prescriptions in 2004 -2013 after first diagnosis PTSD

<table>
<thead>
<tr>
<th>Medication</th>
<th>Fiscal Year</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2004</td>
</tr>
<tr>
<td>New PTSD Episodes</td>
<td>51,750</td>
</tr>
<tr>
<td>Mean Number of Psychotics</td>
<td>3.5 ± 2.5</td>
</tr>
<tr>
<td>All Antidepressants</td>
<td>85.1 (4,026)</td>
</tr>
<tr>
<td>Amtriptyline</td>
<td>5.7 (994)</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>12.4 (6292)</td>
</tr>
<tr>
<td>Nefazodone</td>
<td>1.3 (638)</td>
</tr>
<tr>
<td>Phenelzine</td>
<td>0.0 (0)</td>
</tr>
<tr>
<td>Trazodone</td>
<td>33.4 (17,296)</td>
</tr>
<tr>
<td>Any SSRI or SNRI</td>
<td>70.1 (36,206)</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>13.9 (7212)</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>10.3 (5631)</td>
</tr>
<tr>
<td>Sertraline</td>
<td>26.0 (13,410)</td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>9.2 (4770)</td>
</tr>
<tr>
<td>All Anticonvulsants</td>
<td>21.8 (11,267)</td>
</tr>
<tr>
<td>Gabapentin</td>
<td>11.1 (5736)</td>
</tr>
<tr>
<td>Topiramate</td>
<td>2.1 (1072)</td>
</tr>
<tr>
<td>Valporate</td>
<td>7.3 (3794)</td>
</tr>
<tr>
<td>Propoxon</td>
<td>6.1 (3171)</td>
</tr>
<tr>
<td>All Atypical Antipsychotics</td>
<td>29.7 (15,390)</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>4.5 (2347)</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>18.9 (9758)</td>
</tr>
<tr>
<td>Risperdone</td>
<td>9.9 (5126)</td>
</tr>
<tr>
<td>All Typical Antipsychotics</td>
<td>1.8 (948)</td>
</tr>
<tr>
<td>All Addiction Medicines*</td>
<td>7.8 (4027)</td>
</tr>
<tr>
<td>All Sedative Hypnotics</td>
<td>38.2 (19,776)</td>
</tr>
<tr>
<td>Zolpidem</td>
<td>4.6 (2404)</td>
</tr>
<tr>
<td>Any benzodiazepine</td>
<td>34.9 (18,066)</td>
</tr>
<tr>
<td>All Opioid*</td>
<td>35.4 (18,325)</td>
</tr>
<tr>
<td>All Stimulants</td>
<td>1.1 (932)</td>
</tr>
<tr>
<td>Lithium</td>
<td>1.8 (942)</td>
</tr>
<tr>
<td>Buspirone</td>
<td>5.1 (2669)</td>
</tr>
</tbody>
</table>

*Values are mean ± SD or % (n). Data from Shiner and Westgate (2). Cohort is described in detail elsewhere (3).

PTSD, posttraumatic stress disorder; SSRI, serotonin and norepinephrine reuptake inhibitors; SNSR, selective serotonin reuptake inhibitors.
*Includes acamprosate, buprenorphine, disulfiram, naloxone, nicotine replacement, and venlafaxine.

*Includes all opioids in this class code (excluding methadone from methadone clinic) plus tramadol.
Table 4. Phase II Investigator-Initiated Drug Clinical Trials for PTSD in the United States Since 2006

<table>
<thead>
<tr>
<th>Study Name</th>
<th>Intervention</th>
<th>Status</th>
<th>Funding Agency</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharmacotherapeutic Clinical Trial of N-acetylserotonin for PTSD</td>
<td>5HT17 (serotonin) vs. placebo</td>
<td>Completed (1/2009)</td>
<td>Department of Defense</td>
<td>Negative*</td>
</tr>
<tr>
<td>Repetitive Treatment for Military Service Related Chronic PTSD (CSP-507)</td>
<td>Repetition vs. placebo</td>
<td>Completed (1/2011)</td>
<td>VA Office of Research &amp; Development, Janssen provided drug</td>
<td>Negative (36)</td>
</tr>
<tr>
<td>Repetitive for Symptoms of Anosial in PTSD</td>
<td>Repetitive vs. placebo</td>
<td>Completed (2/2014)</td>
<td>University of Colorado, Neurogenetics Pharmaceuticals (collaborator)</td>
<td>No published results</td>
</tr>
<tr>
<td>Gabaotone in Posttraumatic Stress Disorder</td>
<td>Gabaotone vs. placebo</td>
<td>Completed (3/2014)</td>
<td>Department of Defense, Marinus provided drug</td>
<td>Pending results not published yet</td>
</tr>
<tr>
<td>N-acetylserotonin for PTSD in OIF/OEF Veterans</td>
<td>N-acetylserotonin vs. placebo</td>
<td>Completed (9/2014)</td>
<td>Department of Defense</td>
<td>Negative*</td>
</tr>
<tr>
<td>Evaluation of GS551679 in Women With PTSD</td>
<td>GS551679 vs. placebo</td>
<td>Completed (9/2014)</td>
<td>VA Office of Research &amp; Development, National Institute of Mental Health</td>
<td>Negative</td>
</tr>
<tr>
<td>GABA Regulators for Testing Comorbid PTSD and Substance Use Disorders</td>
<td>GABA vs. placebo</td>
<td>Completed (9/2014)</td>
<td>Medical University of South Carolina, Department of Defense, Institute for Translational Neuroscience</td>
<td>Participants treated with N-acetylserotonin compared with placebo showed significant improvements in PTSD symptoms (63)</td>
</tr>
<tr>
<td>Trial of Milteplone in Combat Veterans With PTSD</td>
<td>Milteplone vs. placebo</td>
<td>Recruiting</td>
<td>James J Peters VA Medical Center (Bronx, NY)</td>
<td>Ongoing</td>
</tr>
<tr>
<td>A Randomized Clinical Trial of Milteplone in PTSD</td>
<td>Milteplone vs. placebo</td>
<td>Recruiting</td>
<td>Bronx VA Medical Center, San Diego VA Medical Center, Durham VA Medical Center</td>
<td>Ongoing</td>
</tr>
<tr>
<td>Novel Therapies in PTSD: A Randomized Clinical Trial of Milteplone</td>
<td>Milteplone vs. placebo</td>
<td>Recruiting</td>
<td>VA Office of Research &amp; Development</td>
<td>Ongoing</td>
</tr>
<tr>
<td>Repeated Dose Intravenous Ketamine for PTSD</td>
<td>Ketamine vs. midazolam (active comparator)</td>
<td>Recruiting</td>
<td>Johns Hopkins School of Medicine at Mt. Sinai</td>
<td>Ongoing</td>
</tr>
<tr>
<td>CAP-Ketamine for Antidepressant Resistant PTSD</td>
<td>Ketamine vs. placebo</td>
<td>Recruiting</td>
<td>VA Office of Research &amp; Development</td>
<td>Ongoing</td>
</tr>
<tr>
<td>Zonisamide in Addition to E-CPT-C for Veterans With PTSD and Comorbid Alcoholic Dependence</td>
<td>Zonisamide vs. placebo E-CPT-C</td>
<td>Recruiting</td>
<td>Department of Defense</td>
<td>Ongoing</td>
</tr>
</tbody>
</table>

CPT, Cognitive Processing Therapy; E-CPT-C, Enhanced Cognitive Processing Therapy-C; OIF, Operation Enduring Freedom; OEF, Operation Iraqi Freedom; PTSD, posttraumatic stress disorder; VA, Veterans Affairs.

*L. Davis, M.D., personal communication, Feb 17, 2017.
### Phase III Investigator-Initiated Drug Clinical Trials for PTSD in the United States Since 2006

<table>
<thead>
<tr>
<th>Study Name</th>
<th>Intervention</th>
<th>Status</th>
<th>Funding Agency</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levetiracetam in PTSD</td>
<td>Levetiracetam vs. placebo</td>
<td>Completed (3/2008)</td>
<td>Duke University, UCB Pharma</td>
<td>No published results</td>
</tr>
<tr>
<td>CSP 563: Prazosin and Combat Trauma PTSD</td>
<td>Prazosin vs. placebo</td>
<td>Completed (5/2013)</td>
<td>VA Office of Research &amp; Development</td>
<td>Negative *</td>
</tr>
<tr>
<td>Prazosin for Treatment of Patients With Alcohol Dependence and PTSD</td>
<td>Prazosin vs. placebo</td>
<td>Completed (10/2014)</td>
<td>Department of Defense and VA VISN 1</td>
<td>Negative (59)</td>
</tr>
<tr>
<td>Prazosin for Nightmares and Sleep Disturbance</td>
<td>Prazosin vs. placebo</td>
<td>Completed (2/16/2006)</td>
<td>VA Office of Research and Development and NIMH</td>
<td>Positive (40)</td>
</tr>
<tr>
<td>Prazosin for Combat Trauma PTSD</td>
<td>Prazosin vs. placebo</td>
<td>Completed (8/29/2012)</td>
<td>VA Office of Research (VISN 20 MIRECC)</td>
<td>Positive (41)</td>
</tr>
</tbody>
</table>

CSP, Cooperative Studies Program; MIRECC, Mental Illness Research, Education and Clinical Center; Institute of Mental Health; PTSD, posttraumatic stress disorder; VA, Veterans Affairs; VISN, Veterans Integrated Service Network.

M. Raskind, M.D., personal communication, Feb 1, 2017.

*Prazosin and doxazosin for PTSD are underutilized and underused.*
Review studies pharmacotherapy in PTSD

Pharmacotherapy for post-traumatic stress disorder: systematic review and meta-analysis

Matthew Hoskins, Jennifer Pearce, Andrew Bethell, Lija Sykes, Cornelia Sarloul, Weelee A. Toi, Mark van Ommeren, Joep de Jong, Soraya Seedat, Hanhui Chen and Jonathan R. Esken

Group (N = 27)
Top therapeutic Targets for PTSD from Expert Group

NMDA Receptor Antagonists 78
Cannabinoid Receptor Modulators 70
Glucocorticoid Receptor Agonists 58
Non-SRI Antidepressants 50
Opioid Receptor Agonists 25
Alpha-1 Adrenergic Receptor Antagonists 21
5HT2-D2 Receptor Antagonist (Other Than Risperidone) 20
Riluzole 18
Alpha-2 Adrenergic Receptor Agonists 18
NPY Receptor Modulators 10
Glucocorticoid Low-Activity Partial Agonists And/Or Antagonist 10
Orexin Receptor Antagonists 9
NMDA Receptor Coagonists 9
Anticonvulsants 8
D2 Receptor Agonists 8

D2, dopamine type 2; NMDA, N-methyl-D-aspartate; NPY, neuropeptide Y; PTSD, posttraumatic stress disorder; SRI, serotonin reuptake inhibitor; 5-HT2, 5-hydroxytryptamine-2.
Recommendations

# The urgent need to find effective pharmacologic treatments for PTSD should be considered a national mental health priority.

# There is a need to increase the number of early phase clinical trials through novel collaborations among government, industry, and academia.

# There is a need to develop new trial designs and/or methodologies specifically in the area of PTSD psychopharmacology trials.

# Foundational studies are required to inform the optimal prescription of commonly prescribed medications for the treatment of PTSD.

# The development of a psychopharmacology clinical trials workforce and infrastructure for PTSD would advance the goal of increasing clinical trials in this area.

# Studies exploring the pathophysiology of PTSD will be critical to inform the rational development of novel pharmacologic interventions.

# There is a need to continue to invest in initiatives in translational neuroscience to enhance the expansion of the pipeline of new PTSD pharmacotherapeutics.

Krystal et al., BP, 2017
Brief Report

Reduction of Nightmares and Other PTSD Symptoms in Combat Veterans by Prazosin: A Placebo-Controlled Study

Murray A. Raskind, M.D.
Elaine R. Peskind, M.D.
Evan D. Kanter, M.D.
Eric C. Petrie, M.D.
Allen Radiant, M.D.
Charles E. Thompson, M.D.
Dorcus J. Dobie, M.D.
David Hoff, PA-C
Rebekah J. Rein, J.D.
Kristy Straits-Tröster, Ph.D.
Ronald G. Thomas, Ph.D.
Miles M. McFall, Ph.D.

Objective: Prazosin is a centrally active α1 adrenergic antagonist. The authors' goal was to evaluate prazosin efficacy for nightmares, sleep disturbance, and overall posttraumatic stress disorder (PTSD) in combat veterans.

Method: Ten Vietnam combat veterans with chronic PTSD and severe trauma-related nightmares each received prazosin and placebo in a 20-week double-blind crossover protocol.

Results: Prazosin (mean dose=10 mg/day at bedtime, SD=5.5) was superior to placebo for the three primary outcome measures: scores on the 1) recurrent distressing dreams item of the Clinician-Administered PTSD Scale and 2) change in overall PTSD severity and functional status according to the Clinical Global Impression of Change. Total score and symptom cluster scores for reexperiencing hyperarousal on the Clinician-Administered PTSD Scale significantly more improved in the prazosin condition, and prazosin was well tolerated.

Conclusions: These data support the efficacy of prazosin for nightmares, sleep disturbance, and other PTSD symptoms.


The [α1]-Adrenergic Antagonist Prazosin Improves Sleep and Nightmares in Civilian Trauma Posttraumatic Stress Disorder

Taylor, Fitcher, Raskind, Murray


10mg dd for nightmares
Use of Prazosin for Pediatric PTSD-Associated Nightmares and Sleep Disturbances: A Retrospective Chart Review

Emma R. Kandel, Gail Dang, Angela P. Posner, Steven J. Vairaktaris

ABSTRACT

Introduction: Youth exposed to trauma have an increased risk for developing posttraumatic stress disorder (PTSD) and associated sleep disturbances and nightmares. The alpha-1 receptor antagonist prazosin has been used successfully in the treatment of posttraumatic stress disorder and nightmares in adults.

Hypothesis: In this study we evaluate the use of prazosin for pediatric PTSD-associated nightmares and sleep disturbances in a retrospective chart review.

Methods: We reviewed the electronic medical record of all pediatric patients seen by a tertiary pediatric psychology service from 2015 to 2018 who received prazosin for treatment of nightmares or sleep disturbances. Data was collected on demographics, diagnosis, treatment course, response, and adverse effects.

Results: Seventeen patients received prazosin for treatment of nightmares or sleep disturbances. The median age was 11 years (range 4-18) and 13 patients were male. The median duration of treatment was 8 weeks (range 4-26). The most common adverse effect was headaches (6%). Prazosin treatment was associated with improved sleep and nightmares over time (pre-treatment 7.3 ± 0.9, post-treatment 3.1 ± 2.4; p < 0.001).

Conclusion: Prazosin was well-tolerated and associated with improvements in nightmares and sleep in youth with PTSD. Adverse events were consistent with the known side-effect profile of prazosin and included dizziness and nausea.

Table 2 Posttraumatic stress disorder symptoms and vital signs at baseline and last time point, and change-over time

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline, mean (SD)</th>
<th>Last time point, mean (SD)</th>
<th>Time estimate (95% CI)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep score (range 0-8)</td>
<td>7.32 (0.94)</td>
<td>3.09 (2.40)</td>
<td>0.939 (-1.219, -0.659)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>UCLA score</td>
<td>51.65 (10.42)</td>
<td>35.08 (14.52)</td>
<td>-4.5 (-5.821, -3.179)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Intrusive</td>
<td>3.65 (3.52)</td>
<td>8.53 (4.44)</td>
<td>1.241 (-1.601, -0.791)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Avoidance</td>
<td>6.03 (1.95)</td>
<td>4.53 (1.95)</td>
<td>0.35 (-0.565, -0.134)</td>
<td>0.0019*</td>
</tr>
<tr>
<td>Negative mood</td>
<td>16.71 (4.61)</td>
<td>12.75 (5.27)</td>
<td>1.217 (-1.682, -0.751)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Arousal</td>
<td>15.26 (3.33)</td>
<td>9.78 (4.72)</td>
<td>1.62 (-2.062, -1.178)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Weight</td>
<td>54.51 (17.16)</td>
<td>55.80 (17.32)</td>
<td>0.331 (0.15, 0.512)</td>
<td>0.0005*</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>104.64 (8.88)</td>
<td>106.83 (11.81)</td>
<td>0.103 (-0.901, 1.106)</td>
<td>0.84</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>61.29 (8.15)</td>
<td>62.63 (8.30)</td>
<td>0.565 (-0.269, 1.4)</td>
<td>0.18</td>
</tr>
<tr>
<td>Heart rate</td>
<td>77.58 (13.03)</td>
<td>81.83 (10.70)</td>
<td>1.241 (-0.417, 2.551)</td>
<td>0.07</td>
</tr>
</tbody>
</table>

* Significant difference between baseline and last time point at p ≤ 0.05

CI, Confidence interval
DOXAZOSIN FOR PTSD

The safety and scientific validity of this study is the responsibility of the study sponsor and investigators. Listing a study does not mean it has been evaluated by the U.S. Federal Government. View the U.S. Federal Government announcement for details.

Sponsor:
Baye College of Medicine

Information provided by (Responsible Party):
Thomas H. Hadden, Baye College of Medicine

Study Details
Tabular View No Results Posted

Study Summary:
The aim of this study is to determine the effects of treatment with doxazosin 8 mg and combined with virtual reality (VR) for a Trauma Stress Disorder.

The effects of treatment with doxazosin 8 mg and combined with virtual reality (VR) exposure therapy will be assessed in a placebo-controlled study. The study will enroll 30 participants. The investigators will use a within-group design in which all participants will receive both placebo and doxazosin (N=15) in the order counterbalanced across participants. A second group of participants (N=15) will receive placebo and doxazosin (N=15) in the order counterbalanced across participants.

Condition on Disease

Phase 1

Phase 2

Outcome Measures

1. The primary outcome measures are the Subjective Units of Distress Scale (SUDS) and PTSD Checklist (PCL) anxiety during VR exposure (T1 T2 F0 and F10 day)
Treatment of sleep disturbances in refugees suffering from post-traumatic stress disorder

Hinuga Sandahl, Erik Vindbjerg and Jessica Carlson

Competence Centre for Transcultural Psychiatry, Mental Health Services in the Capital Region of Denmark, Denmark

Abstract

Sleep disturbances are often referred to as Disorders (PTSD). Although PTSD is prevalent in trauma-affected refugees are scarce. This systematic review of the literature on trauma-affected refugees and a study of the role of structure. Study 1, the literature review, is focused on the role of structure. The identified studies had small sample sizes. It was not possible from the literature review to determine treatment of sleep disturbances. In Study 2, the transcultural psychiatry of PTSD and included in this study, the Transcultural Psychiatry, Denmark, compared to (HTQ) before and after treatment. It was tested with a Rasch model 99% reported nightmares. The Rasch analysis displayed fit 1.16 for nightmares, indicating sufficient discrimination. Important parts of the HTQ region. The HTQ region of disturbances are a prominent part of the PTSD research. In this study, sleep disturbances in trauma-affected refugees

Keywords

refugee, post-traumatic stress disorder, PTSD, sleep

Table 1. Overview of the literature on treatment of sleep disturbances in refugees suffering from PTSD

<table>
<thead>
<tr>
<th>Author</th>
<th>Intervention</th>
<th>Design</th>
<th>Population</th>
<th>Sleep result</th>
<th>Nightmare result</th>
<th>PTSD result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kresna et al.</td>
<td>TCA + clomipramine</td>
<td>Prospective, open-ended study</td>
<td>9 patients</td>
<td>Improved sleep in 6 patients (YESS)</td>
<td>Nightmares increased in 7 patients (YESS)</td>
<td>Improvement of PTSD symptoms in 8 patients (YESS) (adapted from 2017)</td>
</tr>
<tr>
<td>Kresna et al.</td>
<td>Clobazam</td>
<td>Observational pilot study</td>
<td>4 patients</td>
<td>Decreased length of sleep (PSQI)</td>
<td>Subjective increase in sleep</td>
<td>Decreased in sleep</td>
</tr>
<tr>
<td>Siermons et al.</td>
<td>Placebo</td>
<td>Retrospective chart review</td>
<td>30 patients</td>
<td>Decreased frequency of nightmares</td>
<td>Nightmares decreased significantly (CAPS)</td>
<td>Decreased in intensity</td>
</tr>
<tr>
<td>Siermons et al.</td>
<td>Placebo</td>
<td>Case report</td>
<td>2 patients</td>
<td>Improved sleep</td>
<td>Nightmares frequency decreased</td>
<td>Decreased in intensity</td>
</tr>
<tr>
<td>Jespersen et al.</td>
<td>Relaxation</td>
<td>Group pilot experiment</td>
<td>15 patients</td>
<td>Significant improvement in quality (PSQI)</td>
<td>No significant change of trauma symptoms (PTSD)</td>
<td></td>
</tr>
</tbody>
</table>
The efficacy of nabnilone, a synthetic cannabinoid, in the treatment of PTSD-associated nightmares: A preliminary randomized, double-blind, placebo-controlled cross-over design study

Rakesh Jetty, Alexandra Heber, George Fraser, Denis Boisvert

* Canadian Forces Health Services Group Headquarters, Ottawa, Canada
* Operational Trauma and Stress Support Centre, Canadian Forces Health Services Centre, Ottawa

Received 19 August 2018; revised 26 October 2018; accepted 3 November 2018

Summary
Objective: To investigate the efficacy of nabnilone capsules in decreasing the intensity of nightmares experienced by military personnel with PTSD. Methods: This was an open-label, randomized, double-blind, parallel group, placebo-controlled, cross-over design study. In this study, military personnel using PTSD were randomly assigned to receive nabnilone capsules or placebo for a period of two weeks. The primary endpoint was the change in nightmare frequency as measured by the Clinical Persistent and Distressing Dream Questionnaire (CPDDQ) from baseline to 6 weeks. Results: A total of 20 subjects were enrolled in the study. Of these, 15 completed the treatment period. The mean change in nightmare frequency from baseline to 6 weeks for the nabnilone group was -3.6 ± 2.1 (p = 0.03) and for the placebo group was -1.6 ± 2.1 (p = 0.05). Conclusions: Nabnilone significantly reduced the frequency of nightmares in military personnel with PTSD. Further studies are needed to confirm these findings.

Table 1: Change from baseline for both periods.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Nabnilone</th>
<th>Placebo</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPDDQ</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>0.03</td>
</tr>
<tr>
<td></td>
<td>n = 10</td>
<td>n = 9</td>
<td></td>
</tr>
<tr>
<td>CNDBQ</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>n = 10</td>
<td>n = 9</td>
<td></td>
</tr>
<tr>
<td>NDDQ</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>n = 10</td>
<td>n = 9</td>
<td></td>
</tr>
</tbody>
</table>

* Wilcoxon Rank-Sum test.
* Clinical Administered PTSD Scale (CAPS-IV), Recurring and Disturbing Dream Items, Frequency + Intensity.
* Clinical Global Impressions of Change.
* General Well Being Questionnaire.
Marijuana and other cannabinoids as a treatment for posttraumatic stress disorder: A literature review

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Posttraumatic stress disorder (PTSD) is common in the general population, yet there are limita-
tions to its effectiveness, tolerability, and accessibility of available first-line interventions. We
review the extant knowledge on the effects of marijuana and other cannabinoids on PTSD. Poten-
tial therapeutic effects of these agents may largely stem from actions on the endocannabinoid
system and we review major animal and human findings in this area. Preclinical and clinical stud-
ies generally support the biological plausibility for cannabinoids’ potential therapeutic effects, but
some inconsistencies remain. In outcomes depending on dose, dosing regimen, and individual
variation, Treatment outcome studies of wholeplant marijuana and related cannabinoids on PTSD are lim-
ited and not methodologically rigorous, precluding conclusions about their potential therapeu-
tic effects. Reported benefits for nightmares and sleep (particularly with synthetic cannabinoid
substances) are detectable, larger controlled trials to determine effectiveness and tolerability. Of con-
cern, marijuana use has been linked to adverse psychiatric outcomes, including comor-

<table>
<thead>
<tr>
<th>Source</th>
<th>Drug</th>
<th>Design</th>
<th>Sample</th>
<th>Length of Administration</th>
<th>PTSD Outcome</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brune et al. (2014)</td>
<td>Tetrahydrocannabinol (THC), dronabinol</td>
<td>Double blind, placebo-controlled, randomized, double-blind, parallel-group study</td>
<td>100 cannabis users</td>
<td>12 weeks</td>
<td>CAPS</td>
<td>Mean (95% CI) scores reduction of 7.5 (6.7–8.3)</td>
</tr>
<tr>
<td>Ritz et al. (2014)</td>
<td>THC</td>
<td>Uncontrolled pilot study</td>
<td>20 Israeli adults</td>
<td>3 weeks</td>
<td>CAPS</td>
<td>Mean (95% CI) scores reduction of 2.8 (1.5–4.1)</td>
</tr>
<tr>
<td>Fraser et al. (2009)</td>
<td>Tetrahydrocannabinol (THC)</td>
<td>Uncontrolled pilot study</td>
<td>47 Canadian civilian adults</td>
<td>12 months</td>
<td>CAPS</td>
<td>Mean (95% CI) scores reduction of 0.8 (0.2–1.4)</td>
</tr>
<tr>
<td>Cameron et al. (2014)</td>
<td>Tetrahydrocannabinol (THC), dronabinol</td>
<td>Chart review</td>
<td>104 Canadian military personnel</td>
<td>12 weeks</td>
<td>CAPS</td>
<td>Mean (95% CI) scores reduction of 0.2 (0.0–0.4)</td>
</tr>
<tr>
<td>Jelley et al. (2015)</td>
<td>Tetrahydrocannabinol (THC), dronabinol</td>
<td>Randomized, placebo-controlled, double-blind, parallel-group study</td>
<td>50 Canadian military personnel</td>
<td>7 weeks</td>
<td>CAPS</td>
<td>Mean (95% CI) scores reduction of 3.4 (2.6–4.2)</td>
</tr>
</tbody>
</table>

CAPS, Clinician Administered PTSD Scale (range 0–136); NA, not applicable; PCL, PTSD Checklist — Civilians version (range 17–85).
The Use of Lysergic Acid Diethylamide (LSD) in Psychotherapy

E. F. W. Baker, M.D., F.R.C.P.(C), Toronto

ABSTRACT

One hundred and fifty patients with neurotic or psychiatric disorders were treated with the hallucinogenic drug LSD. This drug, known commercially as D-LSD, was injected intramuscularly or subcutaneously in doses of 25 to 3000 micrograms. The dosages were adjusted according to the patients’ reaction. The results indicate that LSD produces therapeutic effects in the treatment of mental disorders. The drug is useful in the treatment of anxiety neurosis, schizophrenia, and depression. However, its use in other psychiatric disorders is still under investigation. The safety and efficacy of LSD in psychotherapy is discussed.

LSD may be used in psychotherapy for its psychological effects. It is used in the treatment of anxiety neurosis, schizophrenia, and depression. Its effects are often transient and may be combined with other therapeutic measures. The drug is believed to have a potential for use in the treatment of certain psychological disorders. However, its use is still under investigation, and further research is needed to determine its full potential.

LYSERGIC acid diethylamide (LSD) has been used in psychotherapy as an adjunct to existing treatments. Its use in the treatment of mental disorders is still under investigation. However, the drug has been shown to be effective in the treatment of anxiety neurosis, schizophrenia, and depression.

Significance

LSD, known as the prototypical "psychodevice," may be unique among psychotropic substances. By the 1950s, LSD was being used in psychotherapy and was considered a potential treatment for various mental disorders. The drug was later banned, but its use in psychotherapy continued.

Neural correlates of the LSD experience revealed by multimodal neuroimaging

The multi-modal imaging data have identified the occurrence of neurochemical fluctuations in response to LSD. These fluctuations include changes in cerebral blood flow, as well as changes in the concentration of neurotransmitters. The results suggest that LSD alters brain function and may have a role in the treatment of mental disorders.
Jan Bastiaans 1917-1997

J. Bastiaans, Psychiatrisch proefschrift van medisch studenten.

Jan Bastiaans (1917 - 1997) was een beroemde Nederlands psychiatre en neurolog. Hij was een vooraanstaand figuur in de medische wereld en is vooral bekend om zijn werk op het gebied van psychopathologie en neurologie. Bastiaans was een van de eerste Nederlanders die LSD onderzocht en publiceerde zijn eerste werk over het onderwerp in 1956. Hij was een pionier in het veld van de psychotherapie en heeft veel bijgedragen aan de ontwikkeling van de moderne psychiatrie.

Bastiaans was geboren op 20 januari 1917 in Amsterdam. Hij studeerde aan de Radboud Universiteit en behaalde zijn doctoraat in de medische wetenschappen. Na zijn afstuderen werd hij docent en vervolgens professor aan de Universiteit van Leiden. In zijn carrière was hij een veelgevraagd因子在dermedische wereld en schreef hij talloze artikelen en boeken over psychiatrie en neurologie.


Bastiaans' bijdragen aan de wereld van de wetenschap en de medische wereld zijn onvergetelijk en zijn werk blijft een bron van inspiratie voor onderzoeks- en praktijk-aanbevelingen in de领域 of psychiatrie.
The safety and efficacy of 3,4-methylenedioxymethamphetamine-assisted psychotherapy in subjects with chronic, treatment-resistant posttraumatic stress disorder: the first randomized controlled pilot study

Michael C Nutt1,2, Mark T Wagner2, Ann T Mith, Lisa Jeremy2 and Rick Doblin3

Abstract

The results indicate that psilocybin administered orally and methylenedioxymethamphetamine (MDMA) are both safe and effective in reducing PTSD symptoms. The preliminary data suggest that MDMA may be more effective in reducing PTSD symptoms than psilocybin. Further research is needed to confirm these findings and to explore the potential mechanisms of action of these compounds.

Keywords

PTSD, psilocybin, methylenedioxymethamphetamine, clinical trials

The Consequences

Current Efficacy on MDMA-Assisted Psychotherapy for Posttraumatic Stress Disorder

Could MDMA be useful in the treatment of post-traumatic stress disorder?

The use of MDMA in the treatment of PTSD has been controversial, with some researchers finding it to be effective while others have reported negative results. However, recent studies have shown promising results, suggesting that MDMA may be a potential treatment for PTSD.

Conclusion

Further research is needed to determine the safety and efficacy of MDMA as a treatment for PTSD. However, the preliminary data suggest that MDMA may be a promising treatment for this disorder.
<table>
<thead>
<tr>
<th>Guideline</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expert Consensus Guideline – 1999</td>
<td>First choice: SSRI's, venlafaxine, &amp; nefazodone</td>
</tr>
<tr>
<td></td>
<td>Second echelon: TCAs</td>
</tr>
<tr>
<td>Psychopharmacology Algorithm Project at Harvard South Shore Program – 1999</td>
<td>Early use of hypnotic agents for sleep; trazodone first choice, followed by SSRI for persistent PTSD symptoms</td>
</tr>
<tr>
<td>UK NICE – 2005</td>
<td>SSRI's bij PTSS revised → more modest effect demonstrated Psychotherapy as first line treatment</td>
</tr>
<tr>
<td>Canadian Clinical Practice Guideline – 2005</td>
<td>Eerste choice: one choice from fluoxetine, paroxetine, sertraline, &amp; venlafaxine XR</td>
</tr>
<tr>
<td></td>
<td>Second echelon: mirtazapine, fluvoxamine, phenelzine, moclobemide, plus adjuvant olanzapine of risperidone</td>
</tr>
<tr>
<td>International Psychopharmacology Algorithm Project – 2005</td>
<td>Once diagnosis of PTSD is made: SSRI first choice, followed venlafaxine &amp; mirtazapine</td>
</tr>
<tr>
<td>ISTSS- 2008</td>
<td>SSRI's recommended as first choice intervention, followed up with addition with atypical antipsychotics</td>
</tr>
<tr>
<td></td>
<td>Prazosine considered “promising&quot;</td>
</tr>
<tr>
<td>APA guideline - 2009</td>
<td>Concludes new studies suggest SSRI's are less effective than previously thought</td>
</tr>
<tr>
<td></td>
<td>Prazosine considered as promising option for sleep disturbance</td>
</tr>
<tr>
<td>VA/DoD Clinical Practice guideline for PTSD- 2010</td>
<td>Strongest recommendation SSRI's and SNRIs but give advantage to prazosine, mirtazapine, and adjuvant atypical antipsychotics</td>
</tr>
<tr>
<td></td>
<td>Prazosine for nightmares as adjuvant treatment when trazodone and other hypnotics are not sufficiently effective</td>
</tr>
</tbody>
</table>
Bij een ernstige co-morbid depressie is er een voorkeur om primair met medicatie te behandelen, ondanks de beperkte wetenschappelijke onderbouwing.
Wanneer de depressie voldoende is opgeklaard kan vervolgens CGT of EMDR worden toegevoegd.
MDR Trimbos 2009

Diagram:

- Geen herstel
  - Toepassing cognitieve preventie
    - Milt: hooi en p. positie
    - Milt: locatie, patiënt karakter
  - Cognitieve therapie
    - Milt
    - CGT (basis) bij versnel

- Evolutie antipsychoticum
  - Verlaatline of een TCA
    - TCA of serotonine
    - Antidepressivum
    - MAO-1
Zorgstandaard Psychotraumae- en stressorgerelateerde stoornissen
Algorithm for PTSD

1. Is the patient a survivor of a Traumatic Event?
   - Yes
     - Did the patient experience reexperiencing symptoms?
       - Yes
         - Go to step 2
       - No
         - Go to step 3
   - No
     - Go to step 4

2. Is the patient experiencing reexperiencing symptoms?
   - Yes
     - Go to step 5
   - No
     - Proceed to step 6

3. Is the patient experiencing avoidance or numbing symptoms?
   - Yes
     - Go to step 7
   - No
     - Proceed to step 8

4. Is the patient experiencing hyperarousal symptoms?
   - Yes
     - Go to step 9
   - No
     - Go to step 10

5. Is the patient experiencing increased symptoms severity or distress?
   - Yes
     - Go to step 11
   - No
     - Go to step 12

6. Is the patient experiencing significant impairment in functioning?
   - Yes
     - Go to step 13
   - No
     - Go to step 14

7. Is the patient experiencing persistent symptoms for more than 1 month?
   - Yes
     - Go to step 15
   - No
     - Go to step 16

8. Is the patient experiencing persistent symptoms for more than 1 year?
   - Yes
     - Go to step 17
   - No
     - Go to step 18

9. Is the patient experiencing persistent symptoms for more than 5 years?
   - Yes
     - Go to step 19
   - No
     - Go to step 20

10. Is the patient experiencing persistent symptoms for more than 10 years?
    - Yes
      - Go to step 21
    - No
      - Go to step 22

11. Is the patient experiencing persistent symptoms for more than 20 years?
    - Yes
      - Go to step 23
    - No
      - Go to step 24
Patient PTSS met comorbid cluster hoofdpijn.
Terug uit Afghanistan.
Behandeling 2 centra.
Cave polyfarmacie
‘For nearly every psychiatric disorder, it is common to distinguish between strategies applied to “firstline” treatments for unselected patients early in their course of illness and treatment approaches for more severe symptoms or symptoms that have not responded to first-line treatments, so-called treatment-resistant illness’.

Krystal et al., BP, 2017, p e53
The Need to Take a Staging Approach to the Biological Mechanisms of PTSD and its Treatment

Alexander Cowell McFerran 1, 2, E h a n 2, 3, M i r a n d a 4, Van Hoof 2, 3, G h i S. M a t h i i 5, 6, R a c h e l 6, V e m b e l 1, 2

Published online: 7 February 2018 © Springer Science+Business Media New York 2018

Abstract Despite the substantial body of neurobiological research, no specific target has been developed to treat PTSD and there are substantial limitations with the available interventions. We propose that advances are likely to depend on the development of better classification of the heterogeneity of PTSD using a staging approach of disease. A primary rationale for staging is to highlight the probability that distinct therapeutic approaches need to be utilised according to the disease progression of the disorder. Prospective studies, particularly in military populations, provide substantial evidence about the emerging biological alterations that provide the full-blown disorder. These need to be targeted with tailored interventions to prevent disease progression. Equally, the neurobiology of chronic untreated PTSD needs to be differentiated from the acute disorder which emerges across a spectrum of severity, and this range of presentations correspondingly needs to be addressed with differing therapeutic strategies. The staging approach also needs to take account of the range of somatic pathological values that are being identified as consequences of traumatic stress exposure.

Keywords PTSD - Affective - Mood - Longitudinal course - Psychosis - Stress

Introduction In contrast to most significant levels of traumatic stress, PTSD is known to have a tight temporal relationship of its current presentation to the trauma.

Table 1 Proposed staging model for PTSD

<table>
<thead>
<tr>
<th>Stage</th>
<th>Presentation</th>
<th>Example of possible neurobiological changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Trauma-exposed asymptomatic but at risk</td>
<td>Down-regulation of glucocorticoid receptor sensitivity, increased amygdala reactivity, FKBP15 gene expression, increased cytokine activation, decreasim size of hippocampus, increased size of amygdala, increased size of hypothalamus</td>
</tr>
<tr>
<td>1a</td>
<td>Unill-defined symptoms of anxiety and distress</td>
<td>Decreased size of hippocampus, increased size of amygdala, increased size of hypothalamus</td>
</tr>
<tr>
<td>1b</td>
<td>Subsyndromal distress with some behavioral and functional decline</td>
<td>Decreased size of hippocampus, increased size of amygdala, increased size of hypothalamus</td>
</tr>
<tr>
<td>2</td>
<td>First episode of full-threshold symptoms with different trajectories</td>
<td>Decreased anterior cingulate and hippocampal volume, decreased size of hippocampus</td>
</tr>
<tr>
<td>3</td>
<td>Persistent symptoms which may fluctuate with ongoing requirement 1a: Successful remission of first episode 2b: Resurgence or relapse of PTSD and persistent impairment 3: Multiple relapses or worsening following incomplete response</td>
<td>Decreased size of hippocampus, increased size of amygdala, increased size of hypothalamus</td>
</tr>
<tr>
<td>4</td>
<td>Severe symptomatologic loss of increasing distress with substantial disability</td>
<td>High allostatic load, high levels of inflammation, medical comorbidities, enhanced sensitization of a range of neurobiological systems</td>
</tr>
</tbody>
</table>

This article is part of the Special Issue on "Diagnostic and Therapeutic Approaches to Post-Traumatic Stress Disorder (PTSD)". The final publication is available at Springer via https://doi.org/10.1007/s10283-018-0329-6.

1 Alexander Cowell McFerran alexander.mcferran@vub.ac.be

2 E h a n 3, M i r a n d a 4, Van Hoof 2, 3, G h i S. M a t h i i 5, 6, R a c h e l 6, V e m b e l 1, 2
Delayed onset 9.4%
Recovered 5.3%
Resilient 85.2%

Nieuw: Fasegericht Behandelen
Pathophysiology of PTSD

Trauma

Increased levels of catecholamines

Cortisol treatment?

‘Overconsolidation’ or greater pairing of memories and distress

Traumatic reminders would be distressing

Distress leads to further pairing with non-specific stimuli

More difficult to extinguish fear responses and achieve habituation or desensitization

Survivor is frequently activated and aroused by salient traumatic triggers

Other behavioral and biological consequences (such as avoidance behavior, hyperarousal and changes in the way one views or functions in the world)

Failure of extinction – behavioral avoicance, of reminders - stimulus generalisation

PTSD
Targeting fear memories: Golden hour opportunities

Compounds:
- Oxytocin (Koch et al., 2015)
- DCycloserin (de Kleine et al., 2015)
- Propanolol (Pitman, et al., 2015)
- Ketamine (Rasmussen, 2015)
- Corticosteroids (de Quervain et al., Mouthaan et al., 2015; Yehuda et al., 2015)
- Endocannabinoids, Nabilone, (Jetly et al., 2015)

Roadmap: New initiative out of ECNP Traumatic Stress Network

Vermetten, Zohar and Krugers, CPR, 2014
Integrating NIMH Research Domain Criteria (RDoC) into PTSD Research

Ulrike Schmidt and Eric Vermetten

Abstract: Three and a half decades of research on post-traumatic stress disorder (PTSD) have produced substantial knowledge on the underlying mechanisms of this severe and debilitating disease. However, treatment options remain limited, and new perspectives are necessary. The National Institute of Mental Health (NIMH) has recently adopted a new approach, the Research Domain Criteria (RDoC). This approach aims to bridge the gap between basic science and clinical research by focusing on identifying core domains of neural dysregulation and functional brain networks that are associated with psychiatric disorders. In this talk, we will discuss the potential of integrating the RDoC framework into PTSD research, highlighting the need for a more comprehensive understanding of the disease and the development of more effective treatment strategies.

Eric Vermetten, Leiden University Medical Center, NETHERLANDS, chair
Joseph Zohar, Chaim Sheba Medical Center, ISRAEL, co-chair,
Carmen Lior, Chaim Sheba Medical Center, ISRAEL
Harm Krugers, University Amsterdam, NETHERLANDS
Ingrid Philippens, Biological Primate Research Center, NETHERLANDS
Ulrike Schmidt, Max Planck Institute of Psychiatry, GERMANY,
Elisabeth Binder, Max Planck Institute of Psychiatry, GERMANY,
Victor Spoormaker, Max Planck Institute of Psychiatry, GERMANY
David Nutt, Imperial College London, UNITED KINGDOM,
Jonathan Bisson, Cariff University, UNITED KINGDOM,
Dominique de Quervain, Basel, SWITZERLAND
Ben Sessa, Brisol University, UNITED KINGDOM
1. Wat is de eerste keus behandeling van PTSS?
   A. Farmacotherapie met SSRI’s
   B. Farmacotherapie met anti-epileptica
   C. Farmacotherapie met atypische antipsychotica
   D. Psychotherapie

   D Psychotherapie is de eerste keus behandeling van PTSS (A-C onjuist, D juist). Daarnaast kan farmacotherapeutische therapie voor bepaalde patiënten goede verbetering van PTSS klachten laten zien, maar dit is dus geen eerste keus behandeling.

2. Mevrouw Stoel, 37 jaar, heeft een jeugd gehad met vanaf haar zesde jaar mishandeling door haar vader. Dit is nu nog steeds belastend voor haar. Ze komt bij u op het spreekuur nadat psychotherapie niet voldoende heeft gewerkt om haar van haar traumaklachten af te helpen. U bespreekt met mevrouw Stoel de mogelijkheden van een farmacotherapeutische behandeling. Ze stemt hiermee in. Ze heeft geen eerdere farmacotherapeutische behandelingen voor haar klachten ondergaan. U wilt haar graag conform de Nederlandse richtlijn voor de behandeling van PTSS behandelen met een geneesmiddel dat voor PTSS geregistreerd is. Welke twee middelen kunt u dan kiezen?
   A. Fluoxetine en citalopram
   B. Paroxetine en sertraline
   C. Venlafaxine en mirtazapine
   D. Topiramaat en lamotrigine

   B Paroxetine en sertraline zijn de enige twee geneesmiddelen die geregistreerd zijn in Nederland voor de behandeling van PTSS (A, C en D onjuist, B juist).

3. Op basis van de huidige onderzoeksgegevens is er geen plaats voor topiramaat bij de behandeling van PTSS. Deze stelling is:
   A. Juist
   B. Onjuist

   B Topiramaat is nog niet in alle richtlijnen opgenomen, maar heeft goede resultaten laten zien bij RCTs met verschillende doelgroepen. Op basis van deze studies mag topiramaat niet als eerste keus middel worden gekozen maar zou het goed ingezet kunnen worden bij patiënten die niet in aanmerking komen voor een SSRI of die geen effect laten zien op een SSRI. Topiramaat kan ook goed gebruikt worden als additie bij een SSRI of bij het terugdringen van alcoholabusus bij mensen met een geschiedenis van misbruik. Topiramaat heeft dus wel degelijk een plaats bij de behandeling maar PTSS (stelling onjuist, A onjuist, B juist). Een meer definitieve plaatsbepaling van topiramaat in de richtlijnen is pas goed mogelijk als er meer onderzoeksgegevens beschikbaar komen.
De heer Bus (36 jaar) groeit op in een onveilige gezins situatie waarin geweld en verslaving speelt. Op jonge leeftijd is hij er getuige van als vader in een ruzie moeder van de trap duwt. Ze belandt in het ziekenhuis, maar naar de buitenwereld zwijgt het gezin over wat er gebeurd is. Conflicten waarin geweld gebruikt blijven zijn hele jeugd spelen. Inmiddels heeft hij zelf kinderen en merkt hij onzekerheid in zijn vaderschap, bijvoorbeeld wanneer de kinderen zich moeilijk door hem laten troosten. De heer Bus heeft ook soms herbelevingen waarbij hij zijn moeder van de trap ziet vallen, wat angst oproept. De heer Bus vindt het vooral vervelend dat hij thuis steeds vaker geprikkeld en boos reageert. Nu de psychotherapie die hij afgelopen periode heeft ondergaan deze klachten niet heeft verminderd, wil hij graag met u in overleg over de volgende stap. Hij heeft op het internet gelezen dat pillen ook kunnen helpen. Welke groep geneesmiddelen zijn de eerste keus als farmacotherapeutische behandeling van PTSS?

A. SSRIs
B. SNRI's
C. TCA's
D. Anti-epileptica
E. Atypische antipsychotica

**Tussentoetsvraag** | **Juist antwoord** | **Feedback**
--- | --- | ---
DEEL I. OVERZICHT EN UPDATE

Stand van zaken van de farmacotherapie voor PTSS

Farmacotherapie

Dit artikel geeft een overzicht van de opgebouwde kennis en huidige stand van zaken met betrekking tot de farmacotherapie voor posttraumatische stressstoornis (PTSS) en de invloed van verschillende medicamenteuze behandelingen. Er wordt onder andere ingegaan op de behandeling van PTSS met antidepressiva, antiånxietymiddelen en benulstabilisers. Het artikel is geschreven door experts in de medicinalpsychologie, waardoor de informatie betrouwbaar en effectief is. De medische en psychologische voordelen van verschillende medicatieonderdelen worden besproken, waardoor het lezers een betere begrip krijgen van de effecten van elkaar.

Lerendioden

- Het doel van dit artikel is een overzicht te geven van de huidige wetenschappelijke kennis en praktische toepassing van farmacotherapie voor PTSS.
- Het artikel behandelt verschillende medicatieonderdelen en hun effecten op PTSS.
- De lezer verkrijgt een basisbegrip van de farmacotherapie voor PTSS en de invloed van medicatie op het condition van PTSS.

DEEL II. SPECIFIEKE FARMACOTHERAPIE EN NUJE NGEWONTEN

Stand van zaken van de farmacotherapie voor PTSS

Samenvatting

In deze update worden de aspecifieke farmacotherapie voor PTSS besproken, met nadruk op de effecten van antidepressiva, antiånxietymiddelen en benulstabilisers. Er wordt ingegaan op de invloed van medicatieonderdelen op PTSS en hoe deze effecten kunnen worden gemeten. Dit artikel wordt geschreven door experts in de medicinalpsychologie, waardoor de informatie betrouwbaar en effectief is. De medische en psychologische voordelen van verschillende medicatieonderdelen worden besproken, waardoor het lezers een betere begrip krijgen van de effecten van elkaar.

Leerdoelen

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- Het artikel behandelt verschillende medicatieonderdelen en hun effecten op PTSS. De lezer verkrijgt een basisbegrip van de farmacotherapie voor PTSS en de invloed van medicatie op het condition van PTSS.

Hoofdartikel

Sleep and Combat-Related Post Traumatic Stress Disorder

Giclymen-Ann Gemaah, Thomas C Meylan Editors

Inleiding

De aandacht voor PTSS is in de afgelopen jaren toegenomen, aangezien de toegenomen erkenning van de impact van PTSS op de gezondheid en de leefkwaliteit van individuen.

Methode

Deze studie bestond uit een casuïstische analyse van patiënten met PTSS die medicamenteuze behandeling ondergingen. De resultaten werden geëvalueerd met behulp van de Posttraumatic Stress Disorder Scale (PTSS). Het onderzoek is geïnitieerd door de auteur en is bewerkt door de redactie van deze publicatie.
Samenvattend

Crisis in psychofarmacologie?
Vintage drugs
Nieuwe ideeën over psychotherapie –
  vroeg, dan wel ondersteunend/katalyserend voor psychotherapie
Veranderende vormen van psychotherapie
Medicatie is ondersteunend
Symptom-based approach, slaap, RDOC
Slaap: Prazosine, doxazosine, medicinale cannabis
Slaapregistratie: horloge, objectivering slaap