CLONAZEPAM

A MEDICAL DICTIONARY, BIBLIOGRAPHY,
AND ANNOTATED RESEARCH GUIDE TO
INTERNET REFERENCES

JAMES N. PARKER, M.D.
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FORWARD

In March 2001, the National Institutes of Health issued the following warning: "The number of Web sites offering health-related resources grows every day. Many sites provide valuable information, while others may have information that is unreliable or misleading."[1] Furthermore, because of the rapid increase in Internet-based information, many hours can be wasted searching, selecting, and printing. Since only the smallest fraction of information dealing with clonazepam is indexed in search engines, such as **www.google.com** or others, a non-systematic approach to Internet research can be not only time consuming, but also incomplete. This book was created for medical professionals, students, and members of the general public who want to know as much as possible about clonazepam, using the most advanced research tools available and spending the least amount of time doing so.

In addition to offering a structured and comprehensive bibliography, the pages that follow will tell you where and how to find reliable information covering virtually all topics related to clonazepam, from the essentials to the most advanced areas of research. Public, academic, government, and peer-reviewed research studies are emphasized. Various abstracts are reproduced to give you some of the latest official information available to date on clonazepam. Abundant guidance is given on how to obtain free-of-charge primary research results via the Internet. **While this book focuses on the field of medicine, when some sources provide access to non-medical information relating to clonazepam, these are noted in the text.**

E-book and electronic versions of this book are fully interactive with each of the Internet sites mentioned (clicking on a hyperlink automatically opens your browser to the site indicated). If you are using the hard copy version of this book, you can access a cited Web site by typing the provided Web address directly into your Internet browser. You may find it useful to refer to synonyms or related terms when accessing these Internet databases. **NOTE:** At the time of publication, the Web addresses were functional. However, some links may fail due to URL address changes, which is a common occurrence on the Internet.

For readers unfamiliar with the Internet, detailed instructions are offered on how to access electronic resources. For readers unfamiliar with medical terminology, a comprehensive glossary is provided. For readers without access to Internet resources, a directory of medical libraries, that have or can locate references cited here, is given. We hope these resources will prove useful to the widest possible audience seeking information on clonazepam.

The Editors

[1] From the NIH, National Cancer Institute (NCI): **http://www.cancer.gov/cancerinfo/ten-things-to-know**.

CHAPTER 1. STUDIES ON CLONAZEPAM

Overview

In this chapter, we will show you how to locate peer-reviewed references and studies on clonazepam.

The Combined Health Information Database

The Combined Health Information Database summarizes studies across numerous federal agencies. To limit your investigation to research studies and clonazepam, you will need to use the advanced search options. First, go to **http://chid.nih.gov/index.html**. From there, select the "Detailed Search" option (or go directly to that page with the following hyperlink: **http://chid.nih.gov/detail/detail.html**). The trick in extracting studies is found in the drop boxes at the bottom of the search page where "You may refine your search by." Select the dates and language you prefer, and the format option "Journal Article." At the top of the search form, select the number of records you would like to see (we recommend 100) and check the box to display "whole records." We recommend that you type "clonazepam" (or synonyms) into the "For these words:" box. Consider using the option "anywhere in record" to make your search as broad as possible. If you want to limit the search to only a particular field, such as the title of the journal, then select this option in the "Search in these fields" drop box. The following is what you can expect from this type of search:

- **To Hurt or Not to Hurt**

  Source: Diabetes Forecast. 48(6): 30, 33-33. June 1995.

  Contact: Available from American Diabetes Association. 1701 North Beauregard Street, Alexandria, VA 22311. (800) 232-3472. Website: www.diabetes.org.

  Summary: In this article, the author answers some common questions about diabetes-related nerve disease in the feet and legs. Topics covered include the causes of neuropathy, including high blood glucose, smoking, alcohol, high blood pressure and cholesterol levels, and autoimmunity; avoiding neuropathy; the chances of developing neuropathy; diagnostic tests used to confirm diabetic neuropathy; the symptoms of neuropathy; and treatment options for neuropathy, including better control of blood glucose, acetominophen, use of transcutaneous electrical nerve stimulation (TENS),

capsaicin cream, and prescription drugs such as antidepressants, phenothiazines, carbamazepine, and **clonazepam.**

- **A Possible Therapeutic Solution for Stomatodynia (Burning Mouth Syndrome)**

  Source: Journal of Orofacial Pain. 12(4): 272-278. Fall 1998.

  Contact: Available from Quintessence Publishing Company, Inc. 551 Kimberly Drive, Carol Stream, IL 60188. (800) 621-0387 or (630) 682-3223. Fax (630) 682-3288. E-mail: quintpub@aol.com.

  Summary: Stomatodynia (burning mouth syndrome or BMS) is a difficult disease for both patients and clinicians. When facing true stomatodynia, i.e. idiopathic burning mouth (where the cause cannot be determined), patients are offered poorly effective treatment. This article reports on a study of the results of local application of **clonazepam** (0.5 or 1 mg) two or three times daily in 25 subjects who suffered from idiopathic stomatodynia. At the first evaluation, 4 weeks after the beginning of treatment, a visual analog scale (VAS) that represented the intensity of pain decreased significantly. At the second evaluation, 3 to 29 months after the first consultation, the VAS scores dropped significantly further. Analysis of the individual results showed that 10 patients were totally cured and needed no further treatment, 6 patients had no benefit at all, and the remaining 9 patients had some improvement but were not considered to be cured since they did not wish to stop the treatment. Blood level tests that were performed 1 and 3 hours after the topical application revealed the presence of small amounts of the drug. The authors propose a hypothesis that **clonazepam** acts locally to disrupt the neuropathologic mechanism that underlies stomatodynia. The risk factors that are recognized for this condition could decrease the density or ligand affinity of peripheral benzodiazepine receptors. This, in turn, could cause spontaneous pain from the tissues concerned. 1 figure. 34 references. (AA-M).

- **How to Help Patients with Restless Legs Syndrome**

  Source: Postgraduate Medicine. 105(3): 59-61, 65-66, 73-74. March 1999.

  Contact: Available from McGraw-Hill, Inc. 1221 Avenue of the Americas, New York, NY 10020. (612) 832-7869.

  Summary: This article describes the diagnosis and treatment of restless legs syndrome (RLS), a condition characterized by four basic elements: a desire to move the limbs, often associated with paresthesia or dysesthesia; symptoms exacerbated by rest and relieved by activity; motor restlessness; and nocturnal worsening of symptoms. The authors note that, once correctly diagnosed, RLS can usually be effectively treated symptomatically and, in some secondary cases, it can even be cured. The authors focus on clinical features that enable timely identification of the condition and on current management strategies. Secondary causes can include iron deficiency and peripheral neuropathy. The most effective drugs for treating RLS are dopaminergic agents, **clonazepam,** opioids, gabapentin, and clonidine. Those patients with uremia (one possible cause of RLS) may have relief from the syndrome after kidney transplantation or after correction of anemia with erythropoietin. 1 table. 28 references. (AA-M).

- **Diagnosis and Management of Sensory Neuropathies in HIV Infection**

  Source: AIDS Clinical Care; Vol. 6, No. 2, Feb. 1994.

  Contact: Johns Hopkins University, Department of Neurology, Baltimore, MD, 21205.

Summary: This article focuses on the two most common HIV-associated neuropathies: predominantly sensory neuropathy (PSN) or distal symmetric polyneuropathy (DSPN) and medication-induced toxic neuropathies (TN). The epidemiology, clinical features, history, physical findings, diagnosis and treatment are discussed. No large-scale, controlled-treatment trials have been conducted thus far, and therefore symptomatic treatment with pharmacologic agents is largely empiric. Choice of medication is based on the severity of the patient's symptoms and side-effect profile. When pain or other dysesthetic symptoms begin to limit functional ability, tricyclic antidepressants may be useful. Where tricyclics are not effective, second-line choices include mexiletine, carbamazepine, phenytoin, baclofen, and **clonazepam.** For patients with more severe neuropathic pain which inhibits walking, narcotic analgesics may be necessary. The authors present an algorithm for the management of sensory neuropathy as well as guidelines for narcotics use in patients with a history of substance abuse.

- **New Developments in Treatment for Spasmodic Dysphonia**

  Source: Advance for Speech-Language Pathologists and Audiologists. 7(8): 13, 18. February 24, 1997.

  Contact: Available from Merion Publications, Inc. 650 Park Avenue, Box 61556, King of Prussia, PA 19406-0956. (800) 355-1088 or (610) 265-7812.

  Summary: This article, from a professional newsletter for speech-language pathologists and audiologists, describes new developments in the treatment of spasmodic dysphonia (SD). Injecting botulinum type-A toxin (Botox) directly into the vocal cords is the most widely accepted approach for treating SD. This article describes the recent change in the dosage of Botox needed to decrease the symptoms of the disorder, which impairs or impedes the ability to speak clearly and with a normal voice. The toxin chemically denervates the vocal fold, weakening the muscle but allowing it to move more easily. Larger amounts of Botox resulted in more side effects such as breathiness or difficulty in swallowing liquids. The article describes this change in Botox treatment, then outlines other treatments for SD, including voice therapy and other medications (notably clonazepam). The author notes the importance of exercise and relaxation as part of a program to increase the patient's ability to breathe and phonate properly. The article concludes with the contact information for the researcher interviewed.

Federally Funded Research on Clonazepam

The U.S. Government supports a variety of research studies relating to clonazepam. These studies are tracked by the Office of Extramural Research at the National Institutes of Health.[2] CRISP (Computerized Retrieval of Information on Scientific Projects) is a searchable database of federally funded biomedical research projects conducted at universities, hospitals, and other institutions.

Search the CRISP Web site at **http://crisp.cit.nih.gov/crisp/crisp_query.generate_screen**. You will have the option to perform targeted searches by various criteria, including geography, date, and topics related to clonazepam.

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[2] Healthcare projects are funded by the National Institutes of Health (NIH), Substance Abuse and Mental Health Services (SAMHSA), Health Resources and Services Administration (HRSA), Food and Drug Administration (FDA), Centers for Disease Control and Prevention (CDCP), Agency for Healthcare Research and Quality (AHRQ), and Office of Assistant Secretary of Health (OASH).

For most of the studies, the agencies reporting into CRISP provide summaries or abstracts. As opposed to clinical trial research using patients, many federally funded studies use animals or simulated models to explore clonazepam. The following is typical of the type of information found when searching the CRISP database for clonazepam:

- **Project Title: ANXIOLYTIC DRUGS EFFECTS ON HIV1 NEUROPATHOGENESIS**

  Principal Investigator & Institution: Lokensgard, James R.; Assistant Professor; Minneapolis Medical Research Fdn, Inc. 600 Hfa Bldg Minneapolis, Mn 55404

  Timing: Fiscal Year 2001; Project Start 30-SEP-1997; Project End 30-APR-2002

  Summary: (Adapted from the Applicant's Abstract): Although the precise mechanisms whereby HIV-1 infection induces neurodegeneration have yet to be determined, a large body of evidence has incriminated glial cells and the production of proinflammatory mediators. For this reason, ideal therapeutic agents for the treatment of AIDS dementia would possess anti-inflammatory as well as anti-viral properties. Benzodiazepines, such as diazepam (Valium), are extensively prescribed drugs for anxiety disorders which readily cross the blood-brain barrier and have demonstrated immunomodulatory properties as well as antiviral activity in HIV-1-infected cell lines. In this application, the central hypothesis to be tested is that anxiolytic drugs attenuate HIV-1 neuropathogenesis through both inhibition of viral expression and suppression of brain cell-produced immune mediators. To characterize their inhibitory effects on HIV-1 expression in brain cells, human glial and mixed glial/neuronal cell cultures, as well as chronically infected promonocytes (U1 cells), will be infected with HIV-1 and maintained in the presence or absence of anxiolytic drugs. Expression of HIV-1 p24 Ag in culture supernatants will be quantified by ELISA. To test the hypothesis that the antiviral properties of anxiolytic drugs are mediated through an inhibition of cellular transcription factor activation, nuclear extracts from HIV-1-infected human glial cells as well as U1 cells, incubated in the presence or absence of anxiolytic drugs, will be probed for nuclear factor kappa B (NF-kB) activation. To link the effects of anxiolytic drug-induced inhibition of NK-kB with direct inhibition of HIV-1, transient transfection assays using HIV-1 promoter-reporter gene constructs, which contain either normal or mutated NF-kB consensus sequences, will be performed. To test the hypothesis that anxiolytic drugs attenuate HIV-1 neuropathogenesis by inhibiting the production of immune mediators, glial and mixed glial/neuronal cell cultures will be infected with HIV-1 and examined for the production of proinflammatory cytokines and beta-chemokines. The results of the proposed studies aims to contribute to a further understanding of HIV-1 neuropathogenesis and will hopefully have therapeutic implications regarding suppressing viral replication and neurodegeneration in HIV-1-infected patients.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: CELLULAR MECHANISMS IN EPILEPTOGENESIS**

  Principal Investigator & Institution: Prince, David A.; Professor and Chairman; Neurology & Neurological Scis; Stanford University Stanford, Ca 94305

  Timing: Fiscal Year 2001; Project Start 01-JAN-1978; Project End 31-JUL-2006

  Summary: Experiments examine modulation of thalamic neuronal and network activities by the inhibitory neurotransmitter gamma- aminobutyric acid (GABA), and by neuropeptide Y (NPY) and vasoactive intestinal polypeptide (VIP). One series of experiments focuses on molecular differences in GABA type A receptors in neurons of mouse nucleus reticularis (nRt) and the ventrobasal relay nucleus (VB), and the

functional and pharmacological consequences of receptor subunit heterogeneity in these structures, whose reciprocal connectivity underlies thalamic rhythm generation. The hypothesis that alpha3 subunit- containing receptors in nRt cells confer sensitivity to the benzodiazepine anticonvulsant **clonazepam** will be tested in mutated mice. The role of beta3-containing receptors in generating prolonged inhibitory postsynaptic currents (IPSCs) that have an anti-rhythmogenic action in nRt will be examined. A second group of experiments explores actions of NPY and VIP on neuronal and circuit activities in mice and rats. The hypothesis that NPY is released during intense intrathalamic oscillatory activity, and in turn has anti-oscillatory effects, will be tested. The possibility that chronic dosing of the anticonvulsant valproic acid in vivo enhances expression of NPY in nRt cells, resulting in increased release, will be explored. VIP effects on membrane properties and synaptic currents in nRt and VB neurons, and on thalamic circuit oscillations will be studied. Techniques will include whole cell patch clamp recordings of IPSCs and voltage-dependent membrane currents; application of pharmacological agents; single cell RT-PCR from neurons of in vitro thalamic slices; in situ hybridization; the use of mice mutated for various alpha and beta GABAA receptor subunits; and measurements of peptides released from thalamic slices. The long-term goals are to understand the control of thalamic neuronal and circuit activities and potential abnormalities that may underlie pathophysiological states such as absence epilepsy.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: CENTRAL MODULATION OF LOCOMOTOR RHYTHMS**

  Principal Investigator & Institution: Garcia-Rill, Edgar E.; Director; Anatomy; University of Arkansas Med Scis Ltl Rock 4301 W Markham St Little Rock, Ar 72205

  Timing: Fiscal Year 2001; Project Start 01-MAR-1984; Project End 30-NOV-2001

  Summary: (Adapted from applicant's abstract): Cholinergic mesopontine cell groups, in concert with catecholaminergic (and serotonergic) neurons, participate in the descending control of locomotion and of postural muscle tone (e.g., startle response and atonia of REM sleep). As part of the Reticular Activating System (RAS), ascending projections of these cells modulate changes in state (e.g., sleep-wake cycles) and the response to afferent input (e.g., sensory gating). That is, they are in a crucial position to modulate fight vs flight responses. The applicants' work has implicated these neurons in psychiatric (e.g., schizophrenia, anxiety disorder), neurological (e.g., Parkinson's Disease) and sleep (e.g., narcolepsy, REM behavior disorder) disturbances, all of which have sleep-wake cycle and motor dysregulation in common. Studies during the previous grant period identified the presence of a novel mechanism whereby mesopontine cholinergic neurons may induce changes in state in descending target neurons. The proposed studies will investigate the characteristics and pharmacological control of this mechanism with a view towards determining the manner in which postural and locomotion systems are switched on and off. In addition, the applicants have developed a preparation in the behaving animal allowing the non-invasive recording of a waveform which is a measure of the ascending output of the RAS. Preliminary data suggest that localized injections of neuroactive agents into the mesopontine region can modulate this vertex-recorded waveform. The proposed studies will investigate the characteristics and pharmacological control of this waveform with a view towards determining the manner in which arousal and sensory gating systems are controlled. This work is of critical importance in the understanding of, and design of therapeutic strategies for, a number of psychiatric, neurological and sleep-wake cycle disorders.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: MOLECULAR MECHANISMS OF NEURONAL EXCITABILITY & EPILEPSY**

  Principal Investigator & Institution: Delorenzo, Robert J.; Professor and Chairman; Neurology; Virginia Commonwealth University Richmond, Va 232980568

  Timing: Fiscal Year 2001; Project Start 01-JUL-1985; Project End 31-JUL-2005

  Summary: The long term goal of this laboratory is to understand the pathophysiology of limbic epilepsy (LE) at the molecular level. External influences can produce permanent plasticity changes in normal neuronal tissue that cause spontaneous recurrent epileptiform discharges (SREDs) in hippocampal neuronal networks. This research effort has discovered that the induction of epileptogenesis in several models of LE caused a long term increase in the expression of Ca2+-regulated transcription factors (TFs) that were associated with decreased expression in the gamma-amino-butyric acid (GABA)A receptor (GABAAR) alpha subunit gene expression. These findings may have considerable significance in understanding the molecular mechanisms that cause LE and will be the focus of this research proposal. The Central Hypothesis to be tested in this research effort is that epileptogenesis in three models of LE produces an altered "epileptic" neuronal phenotype characterized by long lasting alterations in the regulation of [Ca2+]i levels that induce persistent changes in the expression o specific TFs that in turn regulate the expression of several somatic genes, including the expression of specific subunit isoforms of the GABAA receptor that ultimately play a role in producing alterations in neuronal excitability and the development and maintenance of SREDs. A corollary to this hypothesis is that NMDA receptor activation during epileptogenesis elevates [Ca2+]i levels (induction), which in turn causes persistent decreases in CaMKII activity that in turn changes the ability of "epileptic" neuron to release and uptake Ca2+i from intracellular sources, resulting in long lasting increased [Ca2+]i levels in both the cytoplasm and nucleus (maintenance) maintaining some of the long term plasticity changes associated with epileptogenesis. This research project will combine the multi-disciplinary approaches of molecular genetics, biochemistry, and electrophysiology to study 3 models of LE and to accomplish the following Specific Aims: Aim 1: Determine whether NMDA receptor activated increased [Ca2+]i during epileptogenesis causes the long lasting changes in the development of SREDs and the decreased genetic expression and function of GABAAR; Aim 2: Determine whether long lasting changes in the expression of specific NMDA/Ca2+-regulated TFs occur during the induction and maintenance of SREDs; Aim 3: Evaluate whether the NMDA/Ca2+-induced changes in TF expression that occur in epileptogenesis cause the long term changes in GABAAR gene expression and function and the development of SREDs; Aim 4: Determine if [Ca2+]i homeostatic mechanisms are altered in a NMDA/Ca2+ manner during epileptogenesis and contribute to the maintenance of SREDs, decreased GABAAR gene expression and function and increased TF expression; Aim 5: Evaluate the molecular mechanisms causing long-term alterations in [Ca2+]i homeostasis and determine their role in regulating TF expression and GABAAR gene expression and function. Results from this study may elucidate some of the molecular mechanisms that regulate the persistent reductions of GABAAR gene expression and function and will provide an insight into the pathophysiology of LE and may offer new treatment strategies and opportunities to prevent this debilitating condition. Results from this study may elucidate some of the molecular mechanisms that regulate the persistent reductions of GABAAR gene expression and function and will provide an insight into the pathophysiology of LE and may offer new treatment strategies and opportunities to prevent this debilitating condition.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: TREATMENT REFRACTORY PANIC DISORDER**

  Principal Investigator & Institution: Simon, Naomi M.; Massachusetts General Hospital 55 Fruit St Boston, Ma 02114

  Timing: Fiscal Year 2001; Project Start 15-FEB-2000; Project End 31-JAN-2005

  Summary: This is an application for a Mentored Patient-Oriented Research Career Development Award with a focus on developing expertise in the study of treatment refractory panic disorder. The candidate proposes to build upon her expertise studying novel therapeutics for panic disorder, and obtain training to assess "next-step" psychopharmacologic and cognitive-behavioral therapy approaches for patients who remain symptomatic despite initial intervention. Panic disorder with or without agoraphobia is a common anxiety disorder, and when broader measures assessing remission including panic attacks, anticipatory anxiety, agoraphobic avoidance, and functional and quality of life measures are used, it is clear that many patients remain symptomatic and significantly impaired despite initial treatment. However, there is minimal data to guide clinicians in their approach to these patients, and the proposed study is designed as an initial step in addressing this issue in a systematic manner. Research Plan: The primary study is a three phase, twenty-four week clinical trial in which patients who remain symptomatic at the end of one phase enter the next. Phase I is a six-week open sertraline treatment trial to prospectively determine treatment refractoriness. Phase II is a six-week double-blind three arm randomized trial of sertraline at continued dose, sertraline at elevated dose, and sertraline plus **clonazepam.** Phase III is a twelve-week randomized single-blind trial of the addition of cognitive-behavioral therapy versus "medication optimization" with sertraline and **clonazepam.** Environment: The proposed study will be based at the Massachusetts General Hospital and will complement a program of training and supervised research under the mentorship of Dr. Mark Pollack, with consultation from experts. Career Development Plan: Training will emphasize skills necessary for designing and carrying out studies to evaluate treatment interventions for patients with panic disorder who remain symptomatic despite initial intervention, and will include work at the Harvard School of Public Health on research methodology and statistics, and supervision with consultants regarding training in outcome assessment, cognitive-behavioral therapy training, and strategies to study the transmission of findings regarding panic treatment to primary care and community settings that will lay the foundation for future independent investigation by the candidate in this area.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

The National Library of Medicine: PubMed

One of the quickest and most comprehensive ways to find academic studies in both English and other languages is to use PubMed, maintained by the National Library of Medicine.[3] The advantage of PubMed over previously mentioned sources is that it covers a greater number of domestic and foreign references. It is also free to use. If the publisher has a Web

[3] PubMed was developed by the National Center for Biotechnology Information (NCBI) at the National Library of Medicine (NLM) at the National Institutes of Health (NIH). The PubMed database was developed in conjunction with publishers of biomedical literature as a search tool for accessing literature citations and linking to full-text journal articles at Web sites of participating publishers. Publishers that participate in PubMed supply NLM with their citations electronically prior to or at the time of publication.

site that offers full text of its journals, PubMed will provide links to that site, as well as to sites offering other related data. User registration, a subscription fee, or some other type of fee may be required to access the full text of articles in some journals.

To generate your own bibliography of studies dealing with clonazepam, simply go to the PubMed Web site at **http://www.ncbi.nlm.nih.gov/pubmed**. Type "clonazepam" (or synonyms) into the search box, and click "Go." The following is the type of output you can expect from PubMed for clonazepam (hyperlinks lead to article summaries):

- **A case of anorexia nervosa associated with epileptic seizures showing favorable responses to sodium valproate and clonazepam.**
  Author(s): Tachibana N, Sugita Y, Teshima Y, Hishikawa Y.
  Source: Jpn J Psychiatry Neurol. 1989 March; 43(1): 77-84.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=2500550&dopt=Abstract

- **A case of Gilles de la Tourette's syndrome treated with clonazepam.**
  Author(s): Kaim B.
  Source: Brain Research Bulletin. 1983 August; 11(2): 213-4.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=6578866&dopt=Abstract

- **A case of mania secondary to hemodialysis: successful treatment with clonazepam.**
  Author(s): Jarecke CR, De Moya VF, Ware MR.
  Source: Journal of Clinical Psychopharmacology. 1990 August; 10(4): 298-9.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=2286703&dopt=Abstract

- **A case of temporal lobe epilepsy with improvement of clinical symptoms and single photon emission computed tomography findings after treatment with clonazepam.**
  Author(s): Ide M, Mizukami K, Suzuki T, Shiraishi H.
  Source: Psychiatry and Clinical Neurosciences. 2000 October; 54(5): 595-7.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11043812&dopt=Abstract

- **A comparison of the efficacy of clonazepam and cognitive-behavioral group therapy for the treatment of social phobia.**
  Author(s): Otto MW, Pollack MH, Gould RA, Worthington JJ 3rd, McArdle ET, Rosenbaum JF.
  Source: Journal of Anxiety Disorders. 2000 July-August; 14(4): 345-58.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11043885&dopt=Abstract

- **A double-blind comparison of clonazepam and placebo in the treatment of neuroleptic-induced akathisia.**
  Author(s): Pujalte D, Bottai T, Hue B, Alric R, Pouget R, Blayac JP, Petit P.
  Source: Clinical Neuropharmacology. 1994 June; 17(3): 236-42.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=9316669&dopt=Abstract

- **A double-blind randomized clinical trial of rapid tranquilization with I.M. clonazepam and I.M. haloperidol in agitated psychotic patients with manic symptoms.**
  Author(s): Chouinard G, Annable L, Turnier L, Holobow N, Szkrumelak N.
  Source: Canadian Journal of Psychiatry. Revue Canadienne De Psychiatrie. 1993 November; 38 Suppl 4: S114-21.
  http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=8306241&dopt=Abstract

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