

# Sound and Fury: Understanding Post-Traumatic Stress Disorder



**Thursday, April 10, 2014**

**6:00 – 7:30 p.m.**

The Joseph B. Martin Conference Center  
Harvard Medical School  
77 Avenue Louis Pasteur  
Boston, MA 02115



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MEDICAL SCHOOL

## Sound and Fury: Understanding PTSD

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### Moderator:



#### **Col (ret.) John C. Bradley, MD**

Chief of Psychiatry and Deputy Director, Mental Health Service,  
VA Boston Healthcare System,  
Clinical Professor of Psychiatry,  
Uniformed Services University School of Medicine  
Lecturer on Psychiatry, Harvard Medical School  
War veteran, Operation Iraqi Freedom

### Speakers:



#### **Roger K. Pitman, MD**

Professor of Psychiatry  
Massachusetts General Hospital



#### **Michael E. Charness, MD**

Chief of Staff, VA Boston Health Care  
Faculty Associate Dean, Professor of Neurology,  
Harvard Medical School  
Assistant Dean, Professor of Neurology,  
Boston University School of Medicine



#### **Paula K. Rauch, MD**

Founding Director, Marjorie E. Korff PACT Program Cancer Center,  
Massachusetts General Hospital  
Chief, Child Psychiatry Consultation Service to Pediatrics,  
Massachusetts General Hospital  
Director, Family Support, Red Sox Foundation-MGH Home Base  
Program  
Associate Professor of Psychiatry, Harvard Medical School

## About the Speakers

### **John C. Bradley, MD**

Dr. John Bradley serves as the Chief of Psychiatry and Deputy Director for Mental Health for the VA Boston Healthcare System. Dr. Bradley joined the VA in 2011, after retiring from active duty in the United States Army where he served as Chair of the Department of Psychiatry at Walter Reed National Military Medical Center, and Clinical Professor of Psychiatry and Vice Chair at Uniformed Services University.

A native of Massachusetts, Dr. Bradley earned his B.S. in Zoology at the University of Rhode Island, and his M.D. from the Uniformed Services University. He completed his residency in Psychiatry at Letterman Army Medical Center in San Francisco where he served as Chief Resident. His military career included operational assignments for peacekeeping, stability and support operations, and combat operations in Operation Iraqi Freedom with the 528th Medical Detachment in support of 101st Airborne and 3rd Infantry Divisions. He is board certified in Psychiatry.

Dr. Bradley has been honored to serve on a number of national committees and advisory councils. He is co-chair of the VA/DoD Clinical Practice Guideline for the Assessment Management of Patients at Risk for Suicidal Behavior. He served as the clinical subcommittee chair and Army representative for the DoD Task Force for the Prevention of Suicide by Members of the Armed Forces, and co-investigator for the Army Study To Assess Risk and Resilience in Servicemembers (ArmySTARRS). Dr. Bradley has provided expert testimony to the US Congress and served on numerous scientific advisory committees. He has published extensively on the treatment of combat trauma and PTSD, the management of Suicide, and on the American Revolution.

Dr. Bradley's awards and decorations include the Legion of Merit, the Bronze Star, the Iraq Campaign Medal, the Order of Military Medical Merit and numerous teaching awards to include the Lieutenant General Claire Chennault Award for outstanding teaching faculty, and the National Capital Consortium Teacher of the Year Award which was renamed the "John Bradley Award" in 2011 in his honor.

## **Roger K. Pitman, MD**

Dr. Pitman is a psychiatrist at Massachusetts General Hospital and Professor of Psychiatry at Harvard Medical School, Boston, MA. He served as a psychiatrist in the U.S. Navy during the Vietnam War and went on to complete a 30-year career in the Department of Veterans Affairs prior to moving to MGH. He is board certified in Psychiatry and Forensic Psychiatry. He is a Distinguished Life Fellow of the American Psychiatric Association and a Fellow of the American College of Neuropsychopharmacology. He is a recipient of the International Society for Traumatic Stress Studies' Award for Outstanding Scientific Achievement in the field of PTSD and its Lifetime Achievement Award. Dr. Pitman's research into the psychobiology of post-traumatic stress disorder (PTSD) spans more than 30 years. He has more than 150 peer-reviewed publications on PTSD and more than 200 overall publications in the general psychiatric and clinical psychological literature.

## **Michael E. Charness, MD**

Dr. Charness earned his B.Sc. in Psychology from McGill University (1972) and his M.D. from the Johns Hopkins University School of Medicine (1976). He served as resident in Internal Medicine at the Johns Hopkins Hospital (1976-78). He was resident and Chief Resident in Neurology (1978-81) and postdoctoral fellow in Neuroscience (1981-83) at the University of California, San Francisco. He is board certified in Internal Medicine and Neurology. Dr. Charness was Assistant Professor of Neurology at UCSF before coming to the Brockton/West Roxbury VA in 1989. In 2003 he was appointed Chief of Staff at the VA Boston Healthcare System, where he is responsible for clinical, education, and research programs. Dr. Charness serves on VA's national committee for the transformation of VA healthcare and is VA's representative to the Chief Medical Officer Group Steering Committee of the Association of American Medical Colleges. Dr. Charness is Professor of Neurology and Faculty Associate Dean at Harvard Medical School and Professor of Neurology and Assistant Dean at Boston University School of Medicine.

Dr. Charness's laboratory has enjoyed 30 years of continuous support from NIH and the VA to study the molecular and cellular mechanisms of alcohol toxicity in the nervous system and the development of drugs that block alcohol neurotoxicity. He also serves as scientific director of the NIAAA Collaborative Initiative on Fetal Alcohol Spectrum Disorders, an international effort to improve the diagnosis, prevention, and treatment of this condition. He was a member and Chair of the Alcohol-Toxicology 3 Study Section, NIH (1997-2000) and Chair of the Medical Advisory Council, Alcoholic Beverage Medical Research Foundation. He served on the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effects (2000-2003) and the National Advisory Council for NIAAA (2005-2010).

Dr. Charness is Director of the Performing Arts Clinic at Brigham and Women's Hospital, where he treats musicians with performance-related nerve entrapments, soft tissue injuries, and focal dystonia. He is a preceptor for Partners Neurology residents, both at the

Performing Arts Clinic, and at a weekly VA general neurology clinic. He teaches the neurological examination annually at the VA in the Harvard Patient-Doctor II course.

### **Paula K. Rauch, MD**

Dr. Paula Rauch is the Founding Director for the Marjorie E. Korff PACT (Parenting At a Challenging Time) Program at the Massachusetts General Hospital's Cancer Center. Dr. Rauch also serves as Director of the Family Support component of the Red Sox Foundation-Massachusetts General Hospital Home Base Program for veterans of Iraq and Afghanistan and their families. She recently stepped down as Chief of the Child Psychiatry Consultation Service to Pediatrics at the Massachusetts General Hospital to devote more time to PACT and Home Base. She is an Associate Professor of Psychiatry at Harvard Medical School.

Dr. Rauch has been honored with numerous awards throughout her distinguished career, including the Schwartz Center Compassionate Caregiver of the Year Award in 2003, the Partner's in Excellence Award for Leadership and Innovation, The Medical Economics Award for Doctors Who Go the Extra Mile and both teaching and mentoring awards from the Massachusetts General Hospital and Harvard Medical School. She is also the 2011 American Academy of Child and Adolescent Psychiatry Simon Wile Leadership in Consultation Award recipient.

Upon graduating magna cum laude from Amherst College in 1977, Dr. Rauch was awarded the John Woodruff Simpson Fellowship for the Study of Medicine, prior to earning her medical degree from The University of Cincinnati College of Medicine. Dr. Rauch completed her medical internship at Yale University, her Psychiatry residency at the Massachusetts General Hospital and fellowship in Child Psychiatry at Cambridge Hospital and the Massachusetts General Hospital.

Dr. Rauch currently serves on the Amherst College Board of Trustees. She is married to Dr. Aubrey Dickman with whom she has three young adult children. Dr. Rauch is the co-author of "Raising an Emotionally Healthy Child when a Parent is Sick."

# Battling PTSD

National consortium works to improve diagnosis, treatment

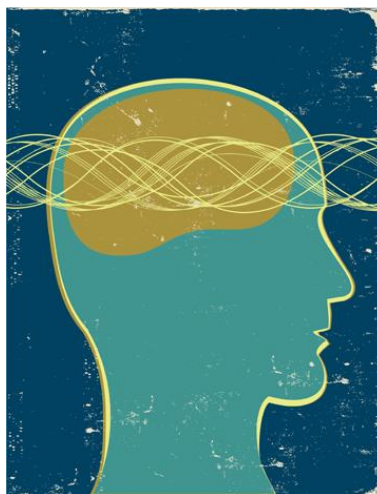
By JEREMY SINGER, DRAPER LABORATORY

August 14, 2012

<http://hms.harvard.edu/news/battling-ptsd-8-14-12>

Post-traumatic stress disorder (PTSD) has been diagnosed in more than 200,000 U.S. military veterans returning from combat in Iraq and Afghanistan. The disorder is also commonly found in civilians who have been involved in an accident or an assault, or have suffered the unexpected loss of a loved one. Approximately 8% of the U.S. population will suffer from PTSD at some point in their lives. The disorder can lead to panic attacks, substance abuse, depression, suicide and a host of other serious medical complications, including, most notably, cardiovascular disorders.

Estimates suggest that half of the patients suffering from PTSD are undiagnosed, and treatment for those who are diagnosed is only partially effective. But a new consortium of nationally recognized PTSD experts aims to improve diagnostic tools and treatment outcomes. The consortium, convened by Draper Laboratory in Cambridge, Massachusetts, will include researchers and clinicians from Harvard Medical School and its affiliated hospitals, including [Massachusetts General Hospital](#), [Brigham and Women's Hospital](#) and the [VA Boston Healthcare System](#).



Identifying objective biomarkers for PTSD will make diagnosis more reliable.

The current state of the art in PTSD diagnosis is based on clinical interviews, forcing clinicians to rely on patients' subjective reports of their own symptoms. Although the clinical history is a

good start, PTSD diagnoses would benefit if reliable biomarkers of the condition were available, as is the case in many other areas of medicine.

The team plans to develop more objective and personalized diagnostic and treatment protocols by using sophisticated algorithms to integrate data from a spectrum of biomarkers, including neuroimaging, psychophysiology, chemical assays and gene expression. The resulting tools will complement today's primarily subjective means of evaluation and treatment selection.

"Although some biological characteristics that point to a PTSD diagnosis have already been identified, more comprehensive study is critical to examine the integrated roles of multiple potential biological factors of the condition," according to HMS professor of psychiatry Roger Pitman. "This will help clinicians develop personalized treatment plans to improve outcomes, rather than relying on 'one-size-fits-all' approaches."

Reducing inconclusive diagnoses and avoiding ineffective treatments, in turn, will help significantly reduce costs, both for patients and society, said Pitman, who is also director of the [PTSD and Psychophysiology Laboratory](#) at Mass General.

The technology platform underlying the proposed solutions to PTSD diagnosis and treatment is based on systems that Draper has developed to synthesize complex data from multiple sources. One version currently helps run the International Space Station.

"We have the most advanced data-fusion technology in critical decision making available to apply to PTSD diagnosis and personalized treatment care," said Len Polizzotto, Draper's vice president in charge of the program.

Bringing together a national team of leading PTSD experts from a variety of disciplines and institutions offers several advantages over pursuing the problem from within a single organization, including the ability to look at a diverse spectrum of factors from neuroimaging to gene expression, and the capacity to conduct human and animal studies in parallel, thus accelerating knowledge and development of solutions, consortium researchers said.

"No one of us could do this alone, but collaboratively, we will be able to create a solution to one of the most expensive healthcare problems our nation is facing, both in financial and human cost," said David Diamond, professor of psychology at the University of South Florida.

Adapted from a [Draper Laboratory](#) news release.

# Healing Hidden Wounds of War

HMS joining forces to better serve returning veterans

By JAKE MILLER

November 8, 2012

Nearly 30 percent of the hundreds of thousands of troops who have served in the wars in Afghanistan and Iraq suffer from some kind of post-deployment illness, according to recent studies. Their chronic disorders, including post-traumatic stress disorder (PTSD), traumatic brain injury, and other invisible wounds of war, also affect the returning veteran's families. This national health crisis will likely have a profound effect on our communities for many years, according to Michael Charness, HMS professor of neurology and chief of staff of the VA Boston Healthcare System.

Harvard Medical School has joined the Association of American Medical Colleges, along with more than 100 other medical schools, in a national initiative entitled Joining Forces. Formed under the leadership of first lady Michelle Obama and Vice President Joe Biden's wife, Jill, the goal of the program is to improve care for veterans and their families, and to better understand and treat post-deployment disorders by enhancing medical education, clinical treatment techniques and research. VA Boston will be collaborating in the initiative with HMS and with Boston University School of Medicine.

"Veterans have risked their lives to serve their country and many have suffered terribly as a consequence of their service. We owe it to them to provide the best care that they can have," said Charness, who is chair of the HMS committee that is coordinating and developing Harvard's part of the initiative.

## High rates of PTSD

Post-deployment disorders are more common in veterans of contemporary wars for several reasons, Charness said. Unlike previous wars, the conflicts in Iraq and Afghanistan have never been divided into dangerous front lines and safe rear zones.

Instead, troops live in near-constant fear for their lives.



**U.S. Navy photo by Lt. j.g. Matthew Stroup**

In addition, many servicemen and women have repeatedly witnessed deadly explosions and seen their colleagues killed, even in their own bases. The all-volunteer U.S. armed forces have also required



troops to make longer and oft-repeated deployments.

Also, because the returning veterans are largely within a population that has been confined to bases and deployed overseas, their presence in local communities at home has not been felt as much as it would be if they were integrated in the population at large. As a result, the problems that veterans are experiencing are not sufficiently understood or appreciated by the public, said Charness.

While many veterans interact directly with VA healthcare workers, Charness said it is critical for all medical students and health care professionals to be aware of the issues that veterans may face to ensure that they are able to guide the veterans they treat toward appropriate treatment.

Living with veterans who are suffering from disorders such as PTSD may also be particularly challenging for family members, who may need treatment and services of their own, Charness said. Since many of the post-combat disorders are chronic, this is likely to remain a concern throughout the working careers of current medical students, he said.

### **Enhancing medical education**

Since HMS signed on to Joining Forces, the committee has been working with faculty members to find ways to integrate related materials into the HMS curriculum. The second year neuroanatomy class, for example, will now include a case study of a military amputee.

Terry Keane, associate chief of staff for research and development for VA Boston Healthcare System and director of the Behavioral Science Division of the National Center for Posttraumatic Stress Disorder, will lead the lecture, emphasizing the interplay between PTSD and substance abuse problems.

Col. John Bradley, lecturer on psychiatry at VA Boston Healthcare and former chief of psychiatry at Walter Reed, is developing an advanced clinical elective in Post-Deployment Psychiatry. In this course, students will be trained to assess and diagnose veterans' post-deployment mental illnesses. They will also learn about psychosocial and psychopharmacological treatment approaches for these conditions.

### **Focused research**

"Not everyone who is deployed has these problems; our investigators are trying to understand the factors associated with risk and resilience," Charness said.

Researchers are also trying to develop new treatments for these disorders.

The committee has identified more than 100 potential research opportunities for students participating in the Scholars in Medicine program, including clinical, translational and basic science and epidemiological studies.

HMS Edward Wigglesworth Professor of Dermatology, Emeritus, [John Parish](#), is also leading an initiative through [CIMIT](#), with assistance from Terry Keane and others. The goal is to promote interdisciplinary, collaborative research—bringing together engineers with basic, translational and clinical scientists—that might lead to new technologies and improvements in the treatment of post-deployment disorders.

### **Integrated resources**

In addition to the many research and clinical resources available through the VA Boston, which provides a comprehensive and integrated model of neurobehavioral and mental health treatment that may serve as a national model for the Joining Forces initiative, the HMS community has developed and collaborated on many programs that combine research and clinical care in integrated programs.

Among them, the [Home Base program at Mass General](#), which combines clinical care and support services to Iraq and Afghanistan service members, veterans and their families throughout New England. Its aim is to help those affected by deployment- or combat-related stress or traumatic brain injury (TBI) and it offers clinical and community education about the “invisible wounds of war,” and the challenges faced by military families. It is also conducting research to improve the treatment and understanding of PTSD and TBI.

“HMS is proud to be contributing to this crucial effort at this critical time,” said Nancy Tarbell, HMS C.C. Wang Professor of Radiation Oncology at Mass General and dean for academic and clinical affairs. “As the wars in Iraq and Afghanistan wind down and disappear from the news, the invisible wounds of those wars continue to need healing.”

## Sound and Fury

Understanding post-traumatic stress disorder  
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# Heart attack can trigger PTSD

POSTED JUNE 25, 2012, 2:06 PM

**Holly Strawbridge, Harvard Health Publications**

A heart attack is a life-changing event. For some people, surviving a heart attack brings renewed appreciation for life. For others, the event is so traumatic that worrying about having a second heart attack consumes their lives.

[By the latest account](#), 1 in 8 heart-attack survivors experiences a reaction that might be called post-traumatic stress disorder (PTSD). Although PTSD is usually associated with extreme trauma such as war, rape, or a natural disaster, heart-attack survivors can experience the same key symptoms: flashbacks that occur as nightmares or intrusive thoughts. As a result, the survivor actively tries to avoid being reminded of the event and becomes hypervigilant worrying that it will happen again.

It's a high price to pay for having your life spared.

"Everyone knows that a heart attack is a serious medical condition. What might not be as obvious is the psychological trauma that can result from having a major heart attack—especially the fear that it might occur again and cannot be predicted. In a small proportion of patients, this fear itself can be disabling," says Dr. Deepak Bhatt, chief of cardiology at the VA Boston Medical Center and a professor at Harvard Medical School.

The 1 in 8 number comes from researchers in New York and Boston, who searched all observational studies relating PTSD to heart attack published since 1948. In the 24 that met their inclusion criteria, the mean age of those who experienced this extreme reaction varied from 53 to 67. The researchers found that the diagnosis of PTSD was significantly more common in younger heart-attack victims, without regard to gender. In the three studies that reported clinical outcomes, heart-attack survivors with PTSD had double the risk of dying or experiencing a second heart attack as those without PTSD. The work was published online in [PLOS One](#).

And although heart attack severity was not recorded in most of the studies that were analyzed, Dr. Bhatt suspects that PTSD might be more likely to occur when a person dies from the event and is resuscitated.

As treatments for heart attack continue to improve, 1.4 million people a year are now surviving the event long enough to be discharged home. If the study is correct, 168,000 of them will be diagnosed with PTSD. It's a grim reminder that as we get better at fixing the body, we must recognize the need to treat the mind.

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“The connections between the heart and mind are powerful, and we are only starting to understand these linkages,” says Dr. Bhatt.

### ***Recognizing and treating PTSD***

The symptoms of PTSD can arise suddenly, or come on gradually. There are three main types of symptom:

- Re-experiencing the traumatic event
  - intrusive memories of the event
  - flashbacks
  - nightmares
  - intense reactions to reminders of the event, such as pounding heart, rapid breathing, muscle tension
- Avoiding reminders of the traumatic event
  - trying to avoid activities, places, thoughts, or feelings that are reminders of the event
  - loss of interest in activities and life in general
  - feeling emotionally numb
- Increased anxiety and emotional arousal
  - trouble falling asleep or staying asleep
  - irritability or anger
  - difficulty concentrating
  - being easily startled

If you recognize one or more of these symptoms in yourself or someone else after a heart attack or other traumatic event, don't write it off as something that will pass. Identifying PTSD early is an important step to coping with it. The sooner treatment is started, the more likely it will be successful.

Treatment generally includes a type of talk therapy known as cognitive behavioral therapy. It may also include an antidepressant such as fluoxetine (Prozac) or sertraline (Zoloft). An experimental treatment known as [eye movement desensitization and reprocessing \(EMDR\)](#) may also help.

#### **To learn more...**

This information was prepared by the editors of the Harvard Health Publications division of Harvard Medical School. It is excerpted from our Harvard Health Blog, available at [hvr.d.me/ujWYp](http://hvr.d.me/ujWYp).

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# Healing from emotional trauma after the Marathon bombing

POSTED APRIL 19, 2013, 10:52 AM

Michael Craig Miller, M.D., Senior Editor, Mental Health Publishing, Harvard Health Publications



The bombs that exploded near the finish line of the Boston Marathon killed three people, physically injured nearly 200 others, and traumatized thousands more. Recovery and healing are beginning for the families of those who died, for the injured and their families, and for others touched by this tragedy. For some, healing will be swift. For others it will be measured in small steps over months, and possibly years.

The Marathon explosions will leave a legacy of emotional scars along with the physical ones, even among those who weren't anywhere near the blasts. Those close to the explosions saw things humans aren't meant to see—grievously injured children, shattered bodies, severed limbs. Others were traumatized from afar, sick with worry about loved ones running in the Marathon or cheering on runners near the finish line. For some, the explosions reignited the terror caused by the September 11<sup>th</sup> attacks.

The very nature of the Boston Marathon may also contribute to the emotional reverberations of the attack. The Marathon has traditionally been a day of joy and camaraderie. We celebrate the elite runners, and then cheer on thousands of ordinary folks trying to do something extraordinary. The bombing turned that into horror and anguish.

Some people who were at the scene of the explosions will undoubtedly develop post-traumatic stress disorder (PTSD). But PTSD is not the only response to frightening events. In fact, most people exposed to a trauma do not develop this condition. They may develop an anxiety disorder, for example, or become depressed. Most people do have some emotional response, but the majority develops no illness at all.

PTSD can be triggered by any traumatic experience that involves a significant threat—or reality—of death, serious injury, or damage to physical integrity. Or by an event, like this one, that inspires intense fear, helplessness, or horror. A person may experience the event directly, witness it, or be confronted with it in some other way.

For anyone touched by the Boston Marathon bombing, it's useful to know a little about PTSD. Whether it is PTSD or not, the sooner symptoms are confronted, the easier it is to overcome them.

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### ***PTSD defined***

PTSD generally causes three kinds of symptoms:

**Hyperarousal.** Individuals with PTSD become irritable, easily startled, and constantly on guard. They sleep poorly and have difficulty concentrating.

**Re-experiencing or intrusion.** The traumatic event involuntarily pops up in the mind as vivid memories, nightmares, or flashbacks. A person with PTSD may feel or act as though the traumatic event is happening again. Any object, situation, or feeling that reminds the person of the trauma can cause intense distress.

**Avoidance and emotional numbing.** Individuals with PTSD try to avoid feelings, thoughts, persons, places, and situations that evoke memories of the trauma. They lose interest in their usual activities. They feel estranged from other people and even from their own feelings.

A mental health professional should be able to review symptoms to help make a judgment whether or not PTSD is the central problem. The diagnosis is, in many cases, less important than focusing on symptoms that either undermine a sense of well-being or are obstacles to getting on with life.

### ***Coping with PTSD***

Treating PTSD can be challenging. Part of the process involves confronting the painful memory, which most people would prefer to avoid. But pushing away the memory may only make things worse. It can emerge when you are under stress or let down your guard. The mental and emotional energy spent avoiding the memory can harm relationships and the ability to function.

No consensus exists about how best to treat PTSD. Various forms of talk therapy can help, and medications are sometimes used.

**Cognitive behavioral therapy.** This entails carefully and gradually “exposing” yourself, usually with the help of a therapist, to thoughts, feelings, and situations that remind you of the trauma. The purpose of the exposure is to support a person functioning better. It is generally not a good idea to simply reinforce memories, because that can reinforce the trauma. Instead, cognitive behavioral therapy for PTSD involves identifying upsetting thoughts about the traumatic event, especially those that are distorted or irrational, and replacing them with calmer or more realistic thoughts.

**Family therapy.** The effects of PTSD often spill over to family members. Family therapy can help in several ways: it can let your loved ones understand what you are going through, it can improve communication, and it can work on relationship problems caused by, or worsened by, PTSD.

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**Medication.** Antidepressants such as fluoxetine (Prozac) or sertraline (Zoloft) can help with some of the symptoms of depression or anxiety if they are present. Sometimes other types of anti-anxiety medications are offered. It's important to keep in mind that while medications can help you feel less depressed or worried, they don't do anything to relieve the underlying cause of PTSD—your memories.

## ***Healing***

An [interfaith service held yesterday](#) at the Cathedral of the Holy Cross in Boston aimed to help the city and those victimized by the bombings begin to heal. Speakers included Boston Mayor Thomas Menino, Massachusetts Governor Deval Patrick, President Barack Obama, and clergy from many Boston congregations.

Coming together, and talking about what we've experienced, is one way to begin the healing process. In the words of Boston's Cardinal Sean O'Malley, we need to be "united in the resolve not to be overcome by evil, but to combat evil with good, working together to build an ever more just, free and secure society for generations to come."

### **To learn more...**

This information was prepared by the editors of the Harvard Health Publications division of Harvard Medical School. It is excerpted from our Harvard Health Blog, available at [hvrld.me/ujWYp](http://hvrld.me/ujWYp).

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# On Veterans Day, don't let the "invisible wounds" of PTSD remain hidden

POSTED NOVEMBER 11, 2013, 11:11 AM

Patrick J. Skerrett, Executive Editor, *Harvard Health*



Millions of American men and women have served in the Armed Forces, protecting and defending our nation. Although many have died, most returned home to "pick up their lives." That isn't always easy. For some veterans, the trauma of war changes the brain in ways that can cause long-term problems.

War-related mental health problems have been with us for centuries. [They probably afflicted Achilles](#), the Greek warrior at the center of Homer's *Iliad*. During the Civil War, such problems were called "nostalgia" or "soldier's heart." In World War I, the term was "shell shock." "Combat neurosis" and "battle fatigue" were the preferred descriptions during World War II and the Korean War. By the late 1970s, the condition had evolved into post-traumatic stress disorder (PTSD).

According to the [American Psychiatric Association](#), more than 300,000 veterans of the wars in Iraq and Afghanistan have been diagnosed with PTSD. Countless others probably suffer from this condition but have never sought help for it. Even sadder, in 2012 more military deaths were caused by suicide than by combat.

Many veterans don't seek help because they feel there's a stigma attached to these invisible wounds. That's a shame, because help is available. "Seeking help for a mental health issue is a sign of strength, not weakness," says former U.S. Rep. Patrick Kennedy, [in a video](#) encouraging the American family to "embrace our veterans so they stop suffering in silence."

If you know a veteran, thank him or her for having served our nation. And if you think he or she is having trouble, bolster your courage and ask. Beginning the conversation may open the door to healing.

### To learn more...

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# Post-traumatic stress disorder (PTSD)

In the weeks after returning home from combat in Iraq, a 26-year-old soldier is in a state of anxiety. He can't sleep at night, and during the day, he is preoccupied with horrific images. Even after he has been home for a few months, he has trouble falling asleep and is often awakened by nightmares. At home, he withdraws from his wife and children. He often feels sad and apprehensive. He goes to his primary care physician because he just doesn't feel well. The doctor tells him that he has symptoms of post-traumatic stress disorder (PTSD) and refers him to a psychiatrist.

## ***Symptoms of PTSD***

PTSD symptoms occur after a person experiences or witnesses a traumatic or extreme stressor, such as a life-threatening event, a natural disaster, or an assault. Symptoms fall into three categories: reliving the event, avoidance, and hyperarousal. A person may relive the event through flashbacks, dreams, or intrusive thoughts. Avoidance often comes in the form of withdrawing from people or certain situations, or having difficulty remembering important aspects of the trauma. Common symptoms of hyperarousal include having trouble sleeping, being unusually vigilant, and startling easily. The symptoms must last more than a month to be considered signs of PTSD. (Symptoms that fade within a month of a traumatic event are signs of a related condition, acute stress disorder.) PTSD has three forms:

- acute, in which symptoms last one to three months after the trauma
- chronic, in which the symptoms last three months or more
- delayed onset, in which at least six months pass between the traumatic event and the start of symptoms.

Experts differ in their views on the nature and severity of events that produce PTSD. Some argue that the event responsible must be extreme, such as being raped, being involved in combat, or witnessing a murder, while others say that a more ordinary frightening event — such as a car accident, an illness, or a problem at work or in a personal relationship — may also cause PTSD if it induces intense fear, helplessness, and horror.

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### **Symptoms of post-traumatic stress disorder**

- Recurring flashbacks, nightmares, or intrusive thoughts about a traumatic event
- Withdrawal from family and close friends
- Avoidance of reminders of the event
- Numbing of emotions
- Survivor guilt
- Difficulty sleeping
- Being easily startled

### ***How common is it?***

As many as 7% to 12% of people will have PTSD at some point in their lives. Among people who experience traumatic events, its incidence varies according to the event. The highest rates are found among survivors of rape, military combat or captivity, and ethnically or politically motivated imprisonment or genocide. More than one million Vietnam veterans — roughly 20% to 30% — were diagnosed with PTSD, a percentage similar to that found among survivors of Hurricane Katrina in New Orleans. And 300,000 Iraq and Afghanistan vets — that's almost 20% — experienced symptoms of PTSD or major depression, according to a 2008 RAND Corporation study.

### ***Who's at risk?***

Among people who experience a traumatic event, the risk for PTSD is especially high among those with a family history of depression (see "Do you have post-traumatic stress disorder?" below).

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### Do you have post-traumatic stress disorder?

Many people who've survived a life-threatening or extraordinarily stressful event experience aftershocks. But in many cases, these symptoms aren't sufficiently intense, pervasive, or long-lasting to constitute post-traumatic stress disorder (PTSD).

If you answer yes to four or more of the questions below, you may have PTSD. Although it's often difficult for people with PTSD to discuss their experiences, it's worthwhile to see a psychiatrist or psychotherapist because treatment can offer tremendous relief. Even if your symptoms don't meet the criteria for PTSD, you may not have escaped a traumatic event unscathed. If you have troubling symptoms related to the event, you may benefit from making an appointment with a mental health professional.

- Have you witnessed or experienced a traumatic, life-threatening event in the past several months?
- Did this experience make you feel intensely afraid, horrified, or helpless?
- Do you have trouble getting the event out of your mind? Do you keep thinking about it during the day, dreaming about it, having flashbacks, or experiencing intense psychological distress when you're reminded of it?
- Do you go out of your way to avoid activities, people, or thoughts that remind you of the event?
- Do you have more trouble falling asleep or concentrating than you did before the event?
- Do you startle more easily and feel more irritable or angry than you did before the event?
- Have your symptoms lasted for more than a month?
- Is your distress making it hard for you to work or function normally?

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### ***Causes of PTSD***

Because not everyone who lives through a traumatic event develops PTSD, biological factors probably increase the risk. The disorder tends to affect certain families more than others, so there may be a genetic predisposition. Someone who has had depression or who has a first-degree relative (such as a parent, sister, brother, or child) who's had depression is more likely to develop PTSD. For example, a study of 81 rape survivors found that those with family members with depression were more likely to develop PTSD than those whose family members did not have depression. One theory is that people with PTSD have a particularly strong response to stress, and that excess emotional stress reduces the size of the hippocampus, the part of the brain that oversees memories. Preliminary research has shown that the hippocampus is smaller than normal in people with PTSD, as well as some people with depression. This might be why people with PTSD often have memory problems and depression. More research is needed, though, to tease out genetic or biological factors that influence PTSD.

### ***Effective treatments for PTSD***

Cognitive behavioral therapy (CBT), exposure therapy, and antidepressants such as selective serotonin reuptake inhibitors (SSRIs) are usually helpful. Tranquilizing medications such as benzodiazepines are not recommended, unless you have severe anxiety, in which case short-term use may be helpful. Some studies have found that benzodiazepines are not effective for PTSD. People with PTSD are also at greater risk for substance abuse (to get relief from the intense anxiety), so drugs like these should be used with caution.

One technique that has not proved effective is a practice known as critical incident stress debriefing, which originated in the military and has been used on firefighters, police officers, and emergency medical technicians, as well as immediate survivors of traumatic experiences. In this approach, participants meet within days of a traumatic experience for a single session lasting for several hours, recounting the event and discussing their thoughts and feelings about it with a counselor. Controlled trials have found that debriefing is not helpful and may even make natural recovery from stress reactions more difficult. Possibly people who are told to probe their feelings at such an early stage feel overwhelmed and misinterpret later symptoms as more serious than they are. It's now generally agreed that no one should be pressured or ordered to participate in critical incident stress debriefing.

### **Cognitive behavioral therapy (CBT)**

CBT, the leading form of therapy for many anxiety disorders, aims to correct ingrained patterns of negative thoughts and behaviors. As the name suggests, it has two parts. Cognitive therapy helps people change patterns of thinking that prevent them from overcoming their fears. Behavioral therapy works to change their reactions in situations that trigger anxiety or stress. The goal of CBT is to break the chain of negative thoughts and a person's reaction to them.

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Because negative thoughts and behaviors tend to come to the fore when people are under stress, the first step in CBT is to help you recognize when you're stressed. It's important to have an inner "thermostat" that can tell you just how stressed you really are and how to dial it down.

Behavioral therapists say there are three components to a stress reaction. These are commonly called the ABCs: affect, behavior, and cognition. *Affect* is how you feel; it refers to your emotional response to a particular situation. *Behavior* is what you do; for example, it can include tensing your jaw, tapping your foot, pacing, or overeating. *Cognition* refers to the thoughts you have when you are stressed; for example, thinking, "I'm going to miss my work deadline and get fired."

Research has shown that CBT is effective for post-traumatic stress disorder. CBT can be done individually or in a group. If the anxiety is the result of a traumatic event that affected more than one person, group therapy may be most effective. CBT usually takes place weekly for several weeks or months; once your condition stabilizes, you may see the doctor once or twice a month or only if symptoms start to worsen.

Researchers have begun looking at the possibilities of combining CBT with what might be thought of as an unusual medication for PTSD. In several small studies, people being treated for anxiety disorders were given a single dose of the antibiotic D-cycloserine immediately before a CBT session. The drug, normally used in much higher doses to treat tuberculosis, is thought to interact with brain receptors involved in the fear response. Based on initial experiments, the scientists theorized that the drug could potentially accelerate the mental processes involved in "un-learning" fears. This treatment is still being studied and isn't used in doctors' offices yet.

## Exposure therapy

Sometimes exposure therapy is used alone. Under the guidance of a therapist, a person imagines or actually confronts his or her fears. Studies show that exposure therapy yields significant, long-lasting results: 60% to 80% of people improve, and the benefits last two to six years.

During exposure therapy for post-traumatic stress disorder, people are asked to talk or write about the trauma that they experienced. At first, the process can induce tremendous fear and even terror, but as people learn that they can "relive" the experience without being harmed, they are less affected by it, and their anxiety gradually diminishes.

## Selective serotonin reuptake inhibitors (SSRIs)

The serotonin system is active in many regions of the brain, affecting anxiety, mood, arousal, impulses, and aggression. SSRIs work by slowing the reuptake of serotonin, which means they prevent the neurons that release this neurotransmitter from reabsorbing it quickly. This prolongs the time that the serotonin

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can work at receptor sites in the brain. SSRIs also appear to change the number and sensitivity of receptors and may indirectly influence other neurotransmitters that play a role in anxiety, including norepinephrine and dopamine.

SSRIs are prescribed for generalized anxiety disorder, obsessive-compulsive disorder, social phobia, panic disorder, and post-traumatic stress disorder. There are several kinds of SSRIs; each works a slightly different way.

Although they are usually well tolerated, SSRIs can have troublesome effects in some people. Reactions to SSRIs vary between different drugs and different people. What induces side effects in one person may not cause any problems in another. Therefore, it may take some trial and error to determine which medication is right for you.

## **Benzodiazepines**

These tranquilizers induce mental and physical relaxation. They enter the brain quickly and bind to receptors for the neurotransmitter GABA, which reduces brain activity. When they bind to GABA receptors, benzodiazepines enhance GABA's calming effects (see Figure 1).

These medications are often used for generalized anxiety disorder, panic disorder, and specific phobia. They're often paired with a longer-acting drug such as an SSRI, and the dose is gradually reduced once the SSRI starts working. They can also be prescribed alone, as needed, to treat specific phobias. Someone with a fear of flying, for example, may take a benzodiazepine before getting on a plane to control anxiety immediately before and during the flight.

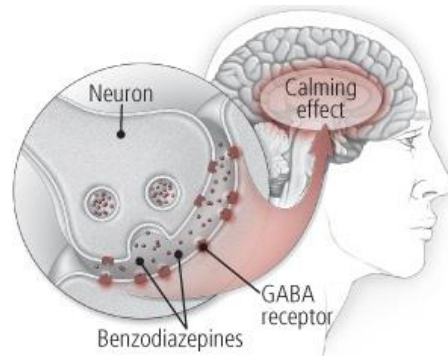
Doctors try to limit the use of benzodiazepines because they can cause tolerance—that is, a need for greater amounts of the drug to produce the same effects. This means people taking benzodiazepines for an extended period should be monitored carefully for signs of tolerance. Benzodiazepines can also cause drowsiness and cognitive impairment. Older adults and people with a history of substance abuse should avoid them whenever possible because they may be particularly sensitive to their side effects.

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### Figure 1: How benzodiazepines work



Benzodiazepines encourage calm by entering the brain quickly and binding to receptors for the neurotransmitter gamma-aminobutyric acid (GABA). GABA reduces brain activity. When benzodiazepines bind to GABA receptors, they enhance GABA's calming effects.

#### To learn more...

This information was prepared by the editors of the Harvard Health Publications division of Harvard Medical School. It is excerpted from our Special Health Report *Coping with Anxiety and Phobias*, available at [hvr.d.me/uUndZ](http://hvr.d.me/uUndZ).

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# What causes anxiety?

Does anxiety arise from a traumatic event or overwhelming stress, as many people assume? Not necessarily. While experiences such as the death of a parent during childhood or another early trauma can play a significant role, such events aren't always at the root of the problem. Not everyone who has lived through a tragedy or terrifying occurrence develops an anxiety disorder, and not everyone who develops an anxiety disorder has endured such an ordeal.

Increasingly, researchers are attempting to zero in on factors that make some people "stress-hardy" and make others "stress-intolerant" and more susceptible to anxiety disorders.

Some scientists compare stress resilience to a green twig that bends but doesn't break when you twist it. This trait seems to be the product of biological, environmental, and emotional factors. Some of the factors that are at play include genetic makeup, having an adaptive coping style, and having or developing a "realistically optimistic" outlook in which you acknowledge life's negatives but don't dwell on them. Having had a nurturing, supportive adult involved in your life during childhood seems to be protective. Bonds forged with others continue to play a key role. Research reveals that a high level of social support even in adulthood cushions you from the effects of stress.

## ***Genetic underpinnings***

People with a parent or sibling who has had an anxiety disorder are at greater risk of developing such a disorder themselves. Certain genetic variations may cause changes in levels of chemicals in the brain and perhaps affect nerve cell connections, nerve cell growth, and neural circuitry in ways that can predispose an individual to anxiety.

Throughout life, different genes turn on and off. In the best-case scenario, genes make the right proteins at the right time. But if the genes get it wrong, they can alter your biology in a way that results in your mood becoming unstable. This biological tendency toward anxiety may be latent for years until an exceptionally stressful event triggers its expression. A person's genetic vulnerability is often intensified by anxiety-provoking behaviors learned in the family and stressful childhood experiences.

Still, much is unknown, and the genetic factors are hardly straightforward. Because anxiety and other mood disorders are thought to arise from genetic variations working in concert with life events and environmental factors, identifying the combinations that lead to anxiety, as well as depression, is quite challenging.

Researchers studying families with a history of anxiety disorders have scrutinized their genetic makeup in hopes of finding common features. Several candidates have been identified. Some are variants of genes, while others are regions on chromosomes that seem similar. For example, researchers found that



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a duplication on a region of chromosome 15 is especially common in families with high rates of panic disorder and phobia, according to one study published in *Cell*. Potential genetic markers for panic disorder have also been found on chromosomes 1 and 11, and a possible marker for agoraphobia was found on chromosome 3. In addition, a 2008 study published in *The Journal of the American Medical Association* linked four of the eight variations of the FKBP5 gene with the more severe symptoms of post-traumatic stress disorder in adults who had suffered abuse in childhood.

But none of these genetic traits appears uniformly in people with anxiety disorders. Therefore it's unlikely that there's any single "anxiety gene." Many genes probably work together to influence the stress response, leaving us more or less likely to become anxious.

Proteins produced by genes, and peptides — amino acid chains that build those proteins — affect anxiety levels. A protein called stathmin, for example, is necessary to form fear memories. Animal research suggests knocking out stathmin reduces protective responses to danger and makes mice act less fearful. Too little of the peptide GRP (gastrin-releasing peptide) may prompt greater, more lingering memories of fear, according to another study on mice.

Researchers hope that pinpointing the genes and natural chemical compounds involved in anxiety and other mood disorders in humans, and gaining a better understanding of their functions, will pave the way for treatments that are more individualized and more successful. Patients would receive the best medication for their type of anxiety. But for now this isn't possible. Nor are any genetic tests available to determine whether an individual is at higher risk for anxiety. Because the study of genes related to anxiety is in its infancy, the particular genes involved and the ways in which their variations induce anxiety have yet to be uncovered. And even promising links between genes and anxiety may not pan out as further research is done.

## ***The brain***

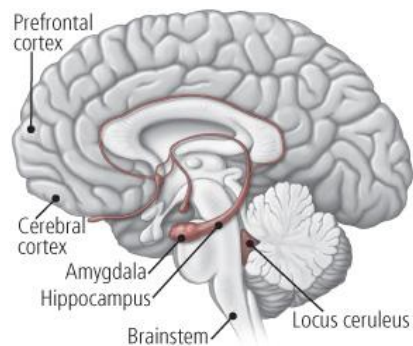
For decades, scientists have believed that anxiety is related to abnormalities in brain chemistry. They base this conviction on the effects of drugs that reduce anxiety by increasing the availability of certain neurotransmitters in the brain. The first anti-anxiety drugs were benzodiazepines, which raise levels of the neurotransmitter gamma-aminobutyric acid (GABA). Later, drugs that increase serotonin levels and affect norepinephrine and other neurotransmitters associated with mood also proved effective. But these findings have raised even more questions. For example, what brain structures are involved? What changes in the brain induce anxiety? And what role do neurotransmitters play?

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### Figure 2: The brain and anxiety



Several regions of the brain influence anxiety. The amygdala is associated with emotions and coordinates the body's response to fear. The cerebral cortex evaluates data about a perceived threat and forms judgments about it, shaping the response to fear. The hippocampus processes emotions and long-term memories. The locus ceruleus helps determine which stimuli deserve attention. The prefrontal cortex is involved in complex reasoning, decision-making, and social behavior, and it seems to play a role in subduing the fear response.

### Regions that influence anxiety

Brain imaging technologies have begun to answer some of these questions. Positron emission tomography (PET), single-photon emission computed tomography (SPECT), functional magnetic resonance imaging (fMRI), and other tools have allowed scientists to observe brain activity even while a panic attack is occurring. This technology has led to a better understanding of which brain regions regulate mood and how other functions may be affected by anxiety. Here's a summary of what researchers have discovered.

**Amygdala.** The amygdala, a small structure deep in the brain (see Figure 2), coordinates the body's fear response. Part of the limbic system, a complex group of structures associated with emotions, it helps mount a swift response to perceived and complex stimuli. The amygdala also plays a role in learning not to be afraid in a situation that previously sparked fear.

In the face of danger, two brain circuits become active. One circuit feeds sensory information about the danger — the sight and smell of a fire, for example — to the cerebral cortex (the rippled outer layer of gray matter jacketing both brain hemispheres), the thinking part of the brain. The cerebral cortex evaluates this information and makes a rational judgment about it. For example, that judgment may determine that the fire is small, but tell you to get out of the house anyway and call the fire department.

The other circuit relays the sensory information to the amygdala, which sends impulses to the autonomic nervous system. This system triggers the "fight-or-flight" response even before the cerebral cortex has made sense of the information. Once activated, it increases heart rate, routes blood to muscles, releases stress hormones and glucose into the bloodstream, and spurs other responses to help you respond quickly to the danger.

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The amygdala stores memories of frightening events and other emotional experiences. In people with anxiety disorders, the amygdala may be so sensitive that it overreacts in situations that aren't threatening. Research on animals suggests that different parts of the amygdala are activated for different anxiety disorders.

**Hippocampus.** Another brain structure in the limbic system, the hippocampus, has a central role in processing emotions and long-term memories. Research has found that the hippocampus is smaller than normal in some women who were abused as children, an experience that increases the risk for post-traumatic stress disorder and other anxiety disorders. Research shows that the hippocampus is also smaller in some depressed people. Stress, which plays a role in both anxiety and depression, may be a key factor here, since there is some evidence that stress may suppress the production of new neurons (nerve cells) in the hippocampus.

**Locus ceruleus.** The locus ceruleus is an area of the brainstem that helps determine which brain stimuli are worth paying attention to. In experiments with animals, when the locus ceruleus was electronically stimulated, the animals displayed anxiety-like symptoms. Some researchers speculate the same response may occur in humans.

**Prefrontal cortex.** The prefrontal cortex is involved in making decisions, solving problems, and exercising judgment. It also appears to have a hand in storing memories of extinguishing fears — as might occur during treatment for an anxiety disorder — and turning down the fear response, according to the National Institute of Mental Health. One area of the prefrontal cortex, for example, helps control the stress response by suppressing the amygdala. Another area — the ventromedial prefrontal cortex — helps support long-lasting extinction of fearful memories. Research suggests the ability to do this may be affected by the size of the ventromedial prefrontal cortex.

## Brain cell communication

Understanding the intricate workings of neurons and neurotransmitters—so-called neuroendocrine responses—can help identify the sources of anxiety disorders. In time, this research may lead to new treatments.

**How the system works.** If you trained a high-powered microscope on a slice of brain tissue, you might see a loosely braided network of neurons, or nerve cells, that send and receive messages. While every cell in the body has the capacity to send and receive signals, neurons are specially designed for this function. Each neuron has a cell body containing the structures that any cell needs to thrive. Stretching out from the cell body are short, branchlike fibers called dendrites and one longer, more substantial fiber called the axon.

A combination of electrical and chemical signals allows communication within and between neurons (see Figure 3). When a neuron becomes activated, it passes an electrical signal called an action potential down the axon to its end (known as the axon terminal), where neurotransmitters are stored. This signal triggers the release of certain neurotransmitters into the space between the axon terminal and the

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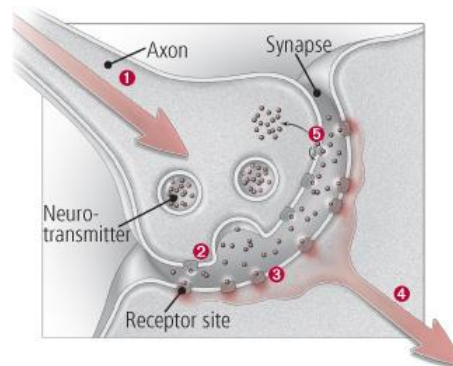
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dendrite of a neighboring neuron. That space is called a synapse. As the concentration of a neurotransmitter rises in the synapse, the neurotransmitter molecules begin to bind with receptors embedded in the membranes of the two neurons.

The release of a neurotransmitter from one neuron can activate or inhibit a second neuron. If the signal is activating, or excitatory, the message continues passing farther along that particular neural pathway. If it's inhibitory, the signal will be suppressed. The neurotransmitter also affects the neuron that released it. Once the first neuron has released a certain amount of the chemical, a feedback mechanism (controlled by that neuron's receptors) instructs the neuron to stop pumping out the neurotransmitter and start bringing it back into the cell. This process is called reabsorption or reuptake. Enzymes break down the remaining neurotransmitter molecules in the synapse.

Messaging systems in the brain usually function well enough to keep senses, learning, movements, and moods perking along. But in some people with anxiety or other mood disorders, any of the complex systems that handle these functions may go awry. That is, the processes inside cells, the interactions between neurotransmitters and receptors, or the communication between brain regions may become overactive or underresponsive.

**Figure 3: How neurons communicate**



1. An electrical signal travels down the axon.
2. Chemical neurotransmitter molecules are released into the synapse.
3. The neurotransmitter molecules bind to receptor sites on the releasing neuron and the second neuron.
4. The signal is picked up by the second neuron and is either passed along or halted.
5. The signal is also picked up by the first neuron, causing reuptake, the process by which the cell that released the neurotransmitter takes back some of the remaining molecules.

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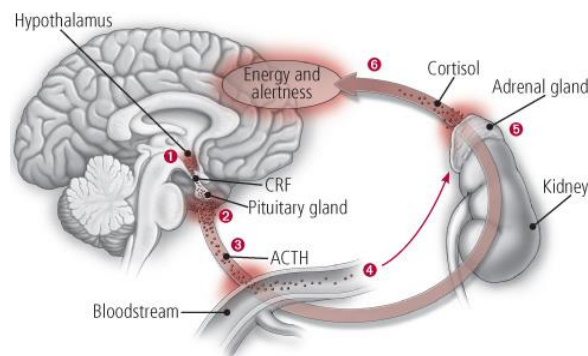
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### Hormones and the HPA axis

While neurotransmitters help transmit signals along nerve pathways, other chemicals, called hormones, carry messages to organs or groups of cells throughout the body. Imbalances of certain hormones increase the risk for anxiety and may induce anxiety symptoms.

These hormones circulate in a pathway called the hypothalamic-pituitary-adrenal (HPA) axis, which influences mood. The hypothalamus is a part of the brain located above your brainstem, the pituitary gland sits below your brain, and the adrenal glands are located atop your kidneys. Together these bodies govern a multitude of hormonal activities in the body and may play a role in anxiety disorders. The autonomic nervous system, which triggers the fight-or-flight response and directs functions throughout the body, is responsible for the function of the HPA axis (see Figure 4).

**Figure 4: Understanding the HPA axis**



When you're faced with a threat, the HPA axis allows you to respond quickly. However, in some people with anxiety disorders, this system remains in overdrive.

1. The hypothalamus secretes the hormone corticotropin-releasing factor (CRF), which rouses the body.
2. CRF travels to the pituitary gland.
3. The pituitary gland secretes adrenocorticotropic hormone (ACTH).
4. ACTH circulates in the bloodstream, traveling to the adrenal gland.
5. The adrenal gland releases cortisol, another hormone.
6. Cortisol stimulates many reactions in your body, including a rush of energy and alertness.

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The hypothalamus secretes corticotropin-releasing factor (CRF), a hormone vital to rousing your body when a physical or emotional threat looms. This hormone follows a pathway to your pituitary gland, where it stimulates the secretion of adrenocorticotropic hormone (ACTH), which pulses into your bloodstream. When ACTH reaches your adrenal glands, it triggers the release of cortisol, a steroid hormone. The rise in cortisol prompts a cascade of reactions in your body, including a rush of energy and alertness. This enables you to respond quickly to a threat. Normally, a feedback loop allows the body to disable these defenses when the threat passes. But in some cases, the floodgates never close properly, and cortisol levels rise too often or simply stay high.

Research suggests that having the HPA axis in persistent overdrive may lay the groundwork for depression as well as anxiety. Evidence points to excess CRF as the main culprit. Some studies have found that people with anxiety disorders have increased levels of CRF in the cerebrospinal fluid, a clear liquid surrounding the brain and spinal cord. Research sponsored by the National Institute of Mental Health revealed that individuals with post-traumatic stress disorder have above-average levels of CRF. One study showed higher-than-normal levels of pituitary and adrenal stress hormones, such as cortisol and ACTH, in the bloodstreams of women who had been physically or mentally abused as children. The levels were especially high in women who were experiencing symptoms of anxiety and depression at the time of the study.

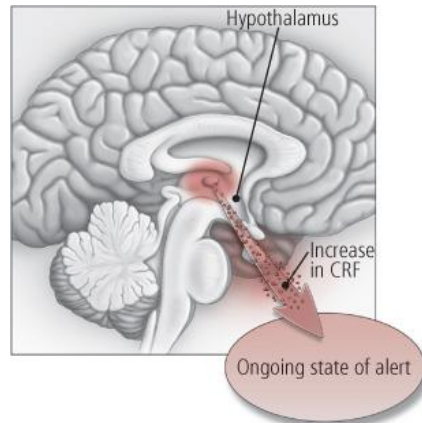
This research suggests a biological explanation for why early stress or trauma increases the risk of developing an anxiety disorder in adulthood. Early trauma may cause a lasting increase in CRF and other stress hormones, and the pumped-up levels of these hormones may keep the HPA axis and the autonomic system in a state of alert (see Figure 5). These findings also point to a possible treatment: drugs that block CRF receptors may help relieve or even prevent anxiety disorders related to early stress. Some drugs of this type are currently being investigated. One early-stage randomized trial recruiting women with post-traumatic stress disorder, for example, is designed to see if symptoms diminish in a group taking a drug that blocks CRF (a CRF antagonist) compared with a group taking a placebo.

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### Figure 5: Early emotional trauma may alter hormone levels



Some research has found that people with anxiety disorders have increased corticotropin-releasing factor (CRF) levels. Scientists believe that an early emotional trauma can cause a lasting increase in CRF, which may keep the body in a heightened state of alert.

### Lingering effects

As mentioned earlier, a traumatic event can be a trigger for acute stress disorder, post-traumatic stress disorder, and specific phobia. Either of the two stress disorders usually begins within days of a terrifying experience. While a phobia may not develop immediately after a traumatic event, it can often be traced back to one. For example, many adults who fear dogs were attacked by dogs as youngsters.

Growing evidence suggests highly stressful experiences, especially early in life, increase the risk for anxiety by impairing a person's ability to negotiate emotional bumps in the road later on. Such experiences include abuse or neglect, emotional deprivation, and the loss of or separation from one's mother. Studies show that rat pups separated from their mothers for just several minutes early in life have a much greater startle response than other pups when faced with stress several months later.

Traumas seem to alter the brain in a way that makes it more susceptible to anxiety. In addition to making the HPA axis hypersensitive (see "Hormones and the HPA axis"), they may also change the structure of the brain. The hippocampus, which works closely with the amygdala (the brain's "fear" center), is smaller in some people with post-traumatic stress disorder, as well as some who have endured extreme, prolonged stress (see "Hippocampus").

#### To learn more...

This information was prepared by the editors of the Harvard Health Publications division of Harvard Medical School. It is excerpted from our Special Health Report *Coping with Anxiety and Phobias*, available at [hvrld.me/uUndZ](http://hvrld.me/uUndZ).

## For More Information

\*If clicking on a link below does not take you to the website, please copy and paste the URL into your browser\*

### **PTSD: National Center for PTSD**

U.S. Department of Veteran Affairs

<http://www.ptsd.va.gov/public/index.asp>

### **PTSD: Post Traumatic Stress Disorder**

VA Boston Healthcare

<http://www.boston.va.gov/services/PTSD.asp>

### **PTSD Research Center**

Mass. General Hospital

[http://www.massgeneral.org/psychiatry/research/ptsd\\_studies.aspx](http://www.massgeneral.org/psychiatry/research/ptsd_studies.aspx)

### **National Center for PTSD**

About Face: Who I Am

<http://www.ptsd.va.gov/apps/AboutFace/>

### **Home Base Program**

<http://www.homebaseprogram.org/general-information.aspx>

### **Army suicides linked back to pre-deployment mental health woes**

CBS News

<http://www.cbsnews.com/news/army-suicides-linked-back-to-pre-deployment-mental-health-woes/>

### **The War Within: Treating PTSD**

CBS/60 Minutes

<http://www.cbsnews.com/videos/the-war-within-treating-ptsd/> (Video)

### **Not feeling strong after Marathon bombings?**

The Boston Globe

<http://www.bostonglobe.com/lifestyle/health-wellness/2013/05/12/those-traumatized-boston-marathon-bombings-may-just-starting-see-signs-experts-urge-anyone-affected-seek-help/OjcasbcFgXOYhcgXaiUH0M/story.html>

### **PTSD and the Aftermath of the Boston Marathon**

NECN/The Morning Show

[http://www.necn.com/04/17/13/PTSD-and-the-aftermath-of-the-Boston-Mar/landing\\_features.html?blockID=838043&feedID=8498](http://www.necn.com/04/17/13/PTSD-and-the-aftermath-of-the-Boston-Mar/landing_features.html?blockID=838043&feedID=8498)

### **Two Months After Bombings, PTSD Is A Concern**

Boston Magazine

<http://www.bostonmagazine.com/health/blog/2013/06/19/marathon-bombings-ptsd/>



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