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COMMENTARIES

CONTRIBUTIONS

SPECIAL ISSUE: PSYCHIATRY
Guest Editor: **Robert J. Westlake, MD**

COLUMNS

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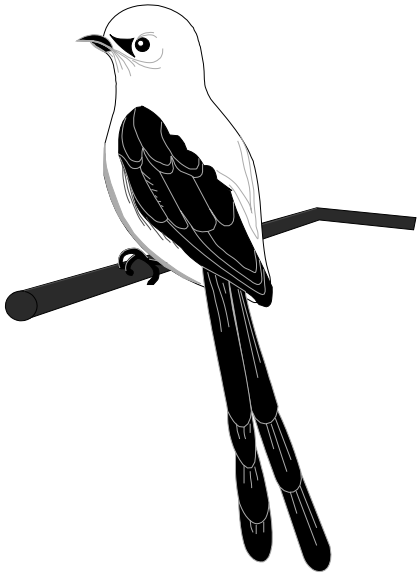
Commentaries

Infected Teeth and Blind Eyes



In his review for the *New England Journal of the Medicine*, the psychiatrist Robert Michels described the book, *Madhouse*, by Andrew Scull, as a compelling set of "lessons on how not to structure a clinical research program." While this is certainly true, I took home a different message. One much more troubling because it speaks to us now.

The "madhouse" refers to the Trenton State Psychiatric Hospital, and the book is about Henry Cotton, MD, psychiatrist-acolyte of Adolf Meyer, former long -standing chair of the psychiatry department of Johns Hopkins Medical School, and one of the leaders of American psychiatry. After completing medical school, Cotton studied with Meyer when he was working in Worcester. When Meyer moved to Hopkins, Cotton did not move with him, but was later brought in, somewhat under Meyer's wing, to run a state psychiatric hospital in New Jersey in 1909. His mission was to help establish a scientific basis for the practice of psychiatry. In the early days of the twentieth century there were no treatments for the insane, and psychiatry was disparaged as a field of mere care -taking. In Europe there was a move behind Freud to establish a psychological basis for mental illness, whereas those Americans not under the influence of psychoanalysis were motivated to seek organic causes, building on the scientific breakthrough that general paresis of the insane was caused by syphilis.

Cotton latched on to the theory of focal infection as the basis for virtually all psychotic illnesses. He denigrated the notion that heredity played any influence at all, or even that environment was important. The sole cause of psychosis was focal, and to the "untrained," occult, infection.

As a neurologist, I hesitate to go overboard in disparaging theories that are 100 years old, especially when they are so close to theories we invoke today, yet this theory, as I will explain, is indicative of something other than poor logic. We neurologists use the catch phrase, "toxic/metabolic," for a lot of our consults. We find a patient to be delirious, to have worsened aphasia, worsened hemiparesis, worsened Parkinson's disease, worsened multiple sclerosis, but without any new focal abnormalities. We conclude that the problem is either metabolic, from deranged electrolytes, liver failure, hypoxia, or some other similar cause, or we say it's toxic, due to occult infection, non-occult infection, drug or medication mishap, etc. We don't know the connection between pneumonia and worsened Parkinson's disease, and, so far as I'm aware it's never been studied, yet I'd bet my life that this connection exists, and I'd venture to say every neurologist would agree. We even have a "critical care neuropathy" and a "critical care myopathy (a little better defined)," that is, an actual "diagnosis" (i.e., name) for a condition that seems to occur in very sick people in the intensive care units, for reasons unknown. So, even in the twenty first century, we invoke an "evil humor" theory not much different than that of Hippocrates or Galen, or even Henry Cotton. I have no problem with this.

Cotton, however, took it a step further. Nowadays we use the "toxic/metabolic" diagnosis as an interpretation of the current condition, counsel restraint, and make sure the focus is on the underlying medical condition which we think is causing the bad humors. Cotton believed that occult infections were almost omnipresent. They primarily affected the teeth, even when x-rays revealed no abnormalities. They also affected the abdomen, especially the colon. In a reflection of an unstated but obvious primitive belief that feces were the source of illness, he used colectomies or extremely aggressive enemas with gallons of fluid to destroy the source of infection. His institution aggressively courted public attention and actually garnered a large clientele of private patients. What was so disturbing however, is not that he embraced this theory, but rather that he clung to it until his death, fighting against people who doubted him, backed up continually by his former mentor, Meyer. Assessors had reviewed his work and concluded that his mortality rate, over 30% initially, then down to something over 20% in physically healthy young people, and his reduced rate of "cure," as measured by hospital discharge, compared to the usual passive observation, was the opposite of what he claimed in public. This is the crime. Cotton was clearly megalomaniacal, unwilling to believe his own eyes, even to the point of having his teenage sons' teeth removed entirely for fear that a change in their behavior signaled the onset of a mental illness, then subjecting one to an abdominal operation when his behavior again started to raise concern. (They both committed suicide). Here was a man of conviction. Unfortunately that conviction wasn't dispassionate logic.

When Cotton was challenged on his high mortality rate for colectomies, he was puzzled until he experienced a dramatic insight. The cause for the deaths was, of course, peritonitis. He was operating in the pre-antibiotic era. One day he operated on two young, healthy psychotic patients. One survived and the other died. He noted that the one who survived was edentulous, having failed that first surgical treatment for psychosis, but the second had only had a few teeth removed. The problem was that the remaining teeth were the source of the pathogen for the peritonitis. From then on all abdominal surgery was preceded by the extraction of all teeth.

Yet despite these blatant crimes, Henry Cotton had only minor limitations placed on him. He lost his position as superintendent and instead became director of clinical research, where he continued to perform his surgeries on often unwilling people. Hysterectomies, cholecystectomies, right-sided colectomies, left-sided colectomies, and massive numbers of tonsillectomies were all treatments for behavioral disorders, psychotic or otherwise, even when family members tried to intervene. But usually this took place only after the teeth had been extracted.

We all know that many medical crimes have taken place in the United States. Psychiatry will forever be tainted by the thousands of lobotomies performed, although public health programs like the Tuskegee syphilis project were even more heinous. The question in the Cotton story is: Where were the authorities? Why did Meyer not step in? When Cotton died unexpectedly, while still a fierce advocate of his focal infection theory, Meyer lionized him, as did the psychiatry journals. Why did the state authorities in New Jersey not intervene? How could a medical staff believe for twenty years what their own eyes told them was wrong, even while they maimed and tortured the most helpless among us? Was it that the insane counted less? Recall that this was a time when eugenics was a popular topic throughout the western world, only ending when Hitler gave it a bad name. Cotton actually argued against a eugenics solution to psychosis since he knew the etiology, and it wasn't inherited. It was bad teeth.

I get disturbed whenever I learn about things like this. We haven't changed much in the past hundred years. We will always have unethical doctors among us. These stories make me wonder what are the things we do today that our grandchildren will judge as equally egregious.

– Joseph H. Friedman, MD

May Sheep Safely Graze?

March 12, 2004: An otherwise healthy 51 year-old woman, living in a rural, upstate New York community, visited her physician because of an isolated reddish area at the tip of her right middle finger, with a small blister in its center. The lesion, which started about ten days before, had gradually enlarged, reaching about an inch in diameter. She was treated with an antibiotic and warm water soaks without obvious improvement. Her physician then brought her to the hospital where the lesion was excised. By April 1 it had spontaneously healed with no further complications or residua.

Within months, three further cases of self-limiting lesions of the fingers or palms were reported to the **US Public Health Service (USPHS).** All shared the following characteristics: All healed spontaneously, whether treated or not, within four weeks. The victims of this mild pustule had all physically contacted sheep within four to six days of the emergence of the lesion which, usually, was then superimposed upon some minor injury to the skin. The USPHS isolated a virus, from all four cases, with the characteristics of a common, but rarely fatal, disease of sheep called *orf.*

Orf is a widespread disorder of sheep, encountered globally wherever sheep or goats are raised. The virus has now also appeared in feral reindeer and other wild ruminants. Whether in sheep, goats or reindeer, the lesion typically appears on or near the mouth. Among shepherds it is called scabby-mouth and amongst veterinarians, *ecthyma contagiosum* or orf [the word, orf, is derived from the word *huerrf,* of Scandinavian origin, meaning scab or blister.] Orf, in its early clinical presentation, may mimick more serious diseases such as tularemia [a disease generally following contact with infected rabbits] or anthrax [representing a transfer from domesticated sheep.]

Orf, readily transferred from infected sheep to humans, is common with shepherds, sheep-shearers and slaughterhouse employees; but there are no instances, yet, of human-to-human transfer. Both human males and females appear to be equally susceptible, although, curiously, no human cases of cutaneous orf have been reported in humans of African origin.

Two medical concerns have recently arisen concerning orf infection. The first is the realization that while orf infection is widely viewed as a circumscribed disease of negligible clinical importance, there is fear that it might become disseminated in immunocompromised persons [such as those with HIV infection or those with cancer receiving intensive chemotherapy or radiation therapy, each known to depress immune response.] In one case report of an AIDS patient, his orf lesions became widespread and life-threatening. Physicians also advise that their patients with extensive skin disease, such as children with eczema, be kept from having any physical contact with sheep or goats lest they contract orf with the strong likelihood that it will become generalized.

A second medical concern is less defined and more speculative. It is based upon the recognition that many of the great historic contagions—smallpox, measles, tuberculosis, influenza and others—had initially commenced as zoonoses, that is, contagions primarily of animals. Smallpox may have originated, many millennia ago, as a pox-disease of cattle; measles may have begun sometime in the very distant past as distemper of dogs or rinderpest of cattle; tuberculosis may have first been a disease primarily of domesticated cows and only then of humans milking or otherwise handling them; and influenza may have transferred to humans only after the continued human intimacy with ducks, geese and swine as domesticated farm-based creatures.

The domestication of swine, cattle, sheep, horses and dogs—and avian species such as ducks and geese—first took place in the Eurasian landmass perhaps 8,000 or more years ago; and the continuing coexistence of humans with these barnyard creatures facilitated the transfer of pathogens from one species to another. It required a series of critical mutations in these viruses to allow them to adapt to another species. By now large numbers, perhaps hundreds, of animal viruses have bridged the species-barrier and have achieved an effective adaptation to, and a congenial home in, human territory.

The metamorphosis of an animal virus to one acclimating itself to the human body seems like an apocalyptic happening restricted to prehistoric days; yet such viral transfers are part of our current history. Consider that a hitherto unknown retrovirus, a pathogen of feral monkeys of western Africa, found its way to infect humans in the 20th Century. The ensuing systemic infection of humans was slow but inexorable in its clinical evolution and the carriers of this new disease, yet nameless, then spread to the Western Hemisphere, particularly the Caribbean, during the next three decades.

The first inkling of a previously unknown human disease surfaced in reports of a unique systemic affliction amongst young homosexual males in San Francisco and New York. The disease was unique in that it seemed to be complicated by a number of superimposed infections; infections which, in the past, had been found afflicting people with advanced immunosuppressive disease. A few years of intensive research isolated a retrovirus which suppressed the body's immune reaction to such a degree that secondary infections, called opportunistic, overwhelmed the body. The disease was called **Acquired Immune Deficiency Syndrome** [**AIDS**] and its pathogen, **Human Immunodeficiency Virus** [**HIV**]. Its known victims now number in the tens of millions.

Can the orf virus infection, now little more than a momentary annoyance, ever transfigure itself into a disseminated, lethal affliction, spreading from person to person by physical contact? Probably not. But the burden of clinical epidemiologists, especially those government scientists concerned with future calamities, is to seek out the ramifications of unlikely scenarios, to provide strategies in the event that their nightmares are transformed into reality, and then lead lives of gnawing anxiety while the rest of us go about the business of carefree living.

– Stanley M. Aronson, MD

From Asylum to Neurobiology and Behavioral Genetics: Butler Hospital Today

Patricia R. Recupero, JD, MD

INPATIENT PSYCHIATRIC CARE HAS UNDERGONE A TRANSFORMATION from the asylums of the 1800s to the acute, intensive care of today's psychiatric hospitals.[1] As Rhode Island's first hospital, Butler Hospital has fostered major changes in the evolution of behavioral healthcare. Dr. Isaac Ray, Butler's first superintendent, was a pioneer in the humane treatment of mental illness and recognizing the mind/body connection in psychiatric disorders.

For over a century, Butler was primarily a long-term facility. With the introduction of effective pharmacotherapies in the 1950s, Butler developed an acute-care paradigm for psychiatric treatment. Butler's **average length of stay (LOS)** in the late '70s and '80s decreased to two weeks; the national average was one to two months. Today the average LOS for adults is one week, reflecting a slight increase in recent years.

Three major trends face psychiatric hospitals today: managed care and the shift from public to private care, obstacles to access, and stigma. This paper highlights major acute-care functions performed by inpatient services and discusses the role of the psychiatric hospital of the future.

TRENDS IN INPATIENT PSYCHIATRY

From 1990 to 2000, the Centers for Medicare and Medicaid Services reported a significant decline in inpatient psychiatric beds per capita.[2] State and county beds decreased by 44%; private psychiatric hospital beds and general hospital psychiatric unit beds decreased 43 and 32% respectively. The number of psychiatric hospitals in the United States also declined. State mental hospitals were reduced by 35%, private psychiatric hospitals by more than 50%, and the number of general hospital psychiatric units by 18%.

Simultaneously, occupancy rates in acute-care psychiatric settings increased. Public funds and state mental health grants declined; the state hospitals' focus shifted from acute care to forensics and long-term care. **Seriously and Persistently Mentally Ill (SPMI)** patients, traditionally served in the public sector, now had to rely on private psychiatric and general acute-care hospitals. In some communities, SPMI individuals were forced to turn to the emergency department for help.

A **National Association of Psychiatric Health Systems (NAPHS)** survey reported that 45.2% of patients admitted to member hospitals were covered by Medicare or Medicaid, the major source of support for SPMI patients.[3] The greatest shift occurred in patients covered by state funds. From 2000-2002 the percentage of state-supported patients in NAPHS hospitals increased 124% from 2.9 to 6.5%.

Concurrent with this shift from public to private care, demand for mental health services increased. During the last two years, one in four adults (59 million people) received mental health treatment.[4] An estimated 48 million were treated with a prescription medication.

Mental illnesses are among five conditions accounting for roughly 31% of the change in healthcare spending between 1987- 2000.[5] For mental disorders, a rise in treatment prevalence, not rising treatment costs per case or population growth, accounted for most of the spending growth. Although the prevalence of mental disorders in the United States remained stable during these years, treatment rates nearly doubled from 4,373 to 8,575 cases per 100,000.

Yet more than one-third (24 million people) who needed care did not receive it. Over a 12-month period, 60% of mentally ill persons received no treatment. Those who did seek treatment often faced a decade or more of delays from the onset of symptoms.[6] In Rhode Island, the SHAPE Foundation reports that substantial unmet need may still exist and that stigma may prevent individuals from seeking care.[7]

INTEGRATED CARE DELIVERY

Nationally, as inpatient care shifted from long-term to acute care, stabilizing and transitioning patients to a less restrictive level of care has become the goal. The nationwide average LOS dropped 61% from 30.5 days in 1987 to 11.7 in 1995. This average has since dropped to one week.[8]

A psychiatric hospital has five essential functions:

- Keeping a patient safe (medically and from harming self)
- Keeping others safe from the patient (aggression, destructive behavior)
- Improving the likelihood of implementing a rigorous treatment strategy (e.g., detoxification, refeeding) and preventing rehospitalization
- Intensive monitoring and diagnostic assessment to help overcome a clinical outpatient impasse, improving hospital readmission patterns
- Providing education and respite to caregivers and outpatient treatment providers.[1]

These functions comprise an integrated care delivery system that must include access to good outpatient treatment, with a focus on preventing crises and avoiding hospitalization. As inpatient care focuses on stabilization with the goal of transitioning to less restrictive therapy, outpatient care is usually essential to integrated treatment.

In recent years, Butler has created a number of specialty treatment programs for adults, including intensive inpatient treatment; an addictions program; specialty services for mood and psychotic disorders; and a treatment center for seniors with Alzheimer's disease and related behavioral problems. Butler also offers programs for adolescents, children, and children with developmental problems needing intensive care.

The hospital's Partial Day Hospital Program combines intensive hospital services with routine activities at home in five primary programs: a **cognitive behavioral therapy (CBT)** program, an alcohol and drug program, a women's program

that provides **dialectical behavior therapy (DBT),** an eating disorders program, and a shorter, less intensive intervention to help with basic cognitive functioning and activities of daily living.

Butler Hospital provides acute care for persons of all ages suffering from behavioral disorders of all kinds; patients include developmentally disabled children, adolescents, adults and geriatric patients.

Serving the greater community

Butler has developed two group homes and a learning center for adolescents who are unable to return to their families. These are managed by the **North American Family Institute (NAFI);** Butler's associate medical director for child and adolescent services serves as medical director for the NAFI program.

Butler reserves several inpatient beds for every community mental health center in Rhode Island. This ensures that the area's uninsured and underprivileged have access to psychiatric professionals and necessary acute care when experiencing a mental health crisis. Working with its Care New England partner, Kent Hospital, Butler offers inpatient services to patients covered by Medicaid.

Serving those with substance use disorders

Butler's Alcohol and Drug Treatment Services' multidisciplinary approach helps people conquer their addictions. A 2002 study reported that five million adults 18 years and older with a **serious mental illness (SMI)** also used illicit drugs ("dual diagnosis").[9] For the past three decades, Butler has provided comprehensive services to dual-diagnosis patients in both inpatient and partial day hospital settings.

Commitment to training

Butler Hospital serves as the administrative center for Brown Medical School's general psychiatric residency and geriatric psychiatry fellowship; the hospital is a major teaching site for the psychology internship, the medical school's core clerkship in psychiatry, and the Brown neuropsychology consortium interns and fellows. Over 100 Brown faculty are based at Butler. Butler has relationships with other area universities and colleges for programs in pharmacy, nursing, occupational therapy, and social work.

Creating new knowledge

Over the past two decades, Butler Hospital and Brown Medical School's Department of Psychiatry have attracted some of the best clinicians and researchers in the country. In 1990, external funding for research at Butler was $500,000. In 2005, it was almost $10 million.

A new generation of psychiatric researchers, coupled with improved imaging and advanced computer technology, has led to new treatments. The gamma knife, a radio-surgical device capable of directing gamma rays with pinpoint accuracy at specific areas of the brain, was found in research conducted at Butler to help patients with severe, previously untreatable forms of **obsessive compulsive disorder (OCD). Deep Brain Stimulation (DBS),** approved by the **Food and Drug Administration (FDA)** for treating Parkinson's disease, is under study at Butler to see whether it can help people who have not responded to traditional treatments for OCD, and people with unresponsive forms of severe depression. Butler played a role in the development of the **vagus nerve stimulator (VNS)**, approved in late 2005 by the FDA. In the area of addictions, the hospital is looking at the impact of exercise and various therapeutic techniques on recovery.

Basic science and translational research

Dr. Isaac Ray questioned why diseases of the body should be viewed differently from diseases of the brain.[10] Today, we are on the threshold of understanding the biological relationships between brain, behavior and emotion. Many Butler physicians are participating in translational research — a two-way interchange between theoretical research and direct patient care. Examples of translational work include the development of neurosurgical treatments for intractable forms of OCD and depression, transdisciplinary studies of addiction, and the development of models to understand how early-life stress can trigger vulnerability to depression in later life.

The hospital is working to establish an integrative research program in basic, pre-clinical and clinical neuroscience. The goal is to develop integrative translational studies in human genetics and neuroimaging. The program, based at Butler Hospital, will take advantage of brain science and genetic research programs already active within Brown Medical School. Studies will examine the role of defined neural circuits and molecular mechanisms in psychiatric disorders, expanding scientific knowledge of basic neuropharmacology and genetics in mental health and fostering the integration of disease-oriented research in neuropsychiatry.

Supported by its Board and in partnership with Brown, Butler Hospital is strengthening its clinical research program, extending its research agenda to functional brain imaging and molecular research. Butler faculty in molecular genomics will utilize techniques of gene mapping, linking disequilibrium, or mutational analysis to identify the presence and role of candidate genes in certain neuropsychiatric diseases. Interacting with campus-based molecular biology programs, the Center for Genomics will study neuropsychiatric disease models. The team will explore molecular neurobiology and genetics, cellular models and molecular mechanisms of neurological and neuropsychiatric drugs, and animal (transgenic mouse and rodent) models of neuropsychiatric disorders.

Conclusion

Just as deinstitutionalization did not end the need for acute care, these new treatments will not make psychiatric hospitals obsolete. The importance of individualized therapy, support groups, and patient and family education cannot be overstated. A kind word, an offering of support, time spent teaching patients how to cope with their problems, and compassionate care are critical to treatment success. Devices, procedures, and medications may reduce symptoms to allow patients to benefit from therapy. Only trained, caring professionals can help patients and families navigate the often difficult road to recovery.

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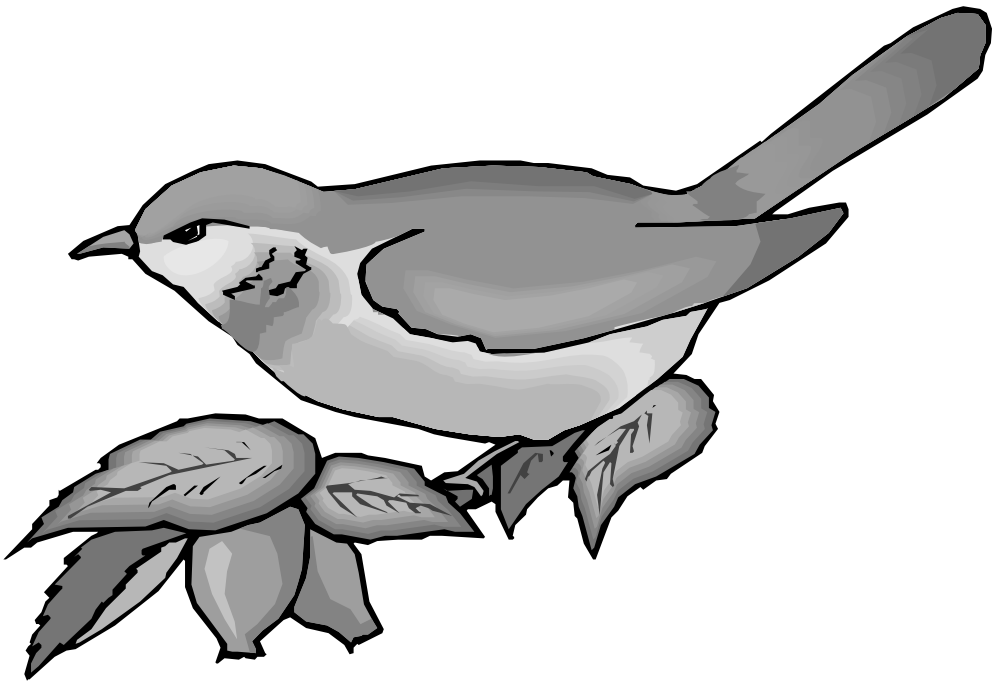

Obsessive-Compulsive Disorder: Recognition Across Medical Settings, and Treatments from Behavior Therapy to Neurosurgery

Benjamin D. Greenberg, MD, PhD, Anthony Pinto, PhD, Maria Mancebo, PhD, Jane Eisen, MD, Steven A. Rasmussen, MD

Obsessive Compulsive Disorder (OCD) is characterized by persistent, intrusive thoughts (obsessions), and repetitive intentional behaviors (compulsions). These symptoms persist despite individuals' attempts to eliminate them and are accompanied by marked and often overwhelming anxiety. Typical obsessions are unrealistic concerns with cleanliness, order, and harm avoidance. In extreme cases, symptoms occupy every waking moment. Compulsions are typically excessive hand washing, counting, checking and rituals that can disrupt all routine activities. Many patients function despite OCD, but in many others the illness leads to profound impairment. OCD ranked as the 10th leading cause of disability among all medical and psychiatric illnesses in industrialized countries.[1]

Treatment can reduce symptoms and improve quality of life.[2] The illness often goes unrecognized and untreated in medical practice. While the one-year US population prevalence of OCD was 2.4%,[3] in a large HMO only 0.095% of patients were treated for OCD.[4] While awareness of OCD in the general population is increasing, gradually lessening stigma, its symptoms can be intensely embarrassing and patients may not volunteer them. Functional impairment due to OCD was greater than that in diabetes and hypertension.[5] Moreover, undetected OCD can worsen outcomes of other illnesses. The diagnosis of OCD is clinical. It rests on the identification of obsessive thoughts and compulsive behaviors (including covert mental rituals) and the resulting distress, time consumed, and interference with function (including how much normal work and social activities are avoided for fear they might trigger OCD symptoms).[6]

Family/genetic factors

While not formally part of diagnostic criteria, a family history of OCD may increase confidence in an uncertain diagnosis. The risk of OCD was 12% in first-degree relatives of OCD probands vs. 3% in relatives of psychiatrically-healthy controls. The risk was highest (18%) in people whose siblings developed OCD before they were 18 years old.[9] We are collaborating in the first large-scale family-genetic study of OCD funded by the **National institutes of Health** (**NIH**) to learn more about 1) core features of OCD (e.g., symptom subtypes) which are most familial and so are most likely to be genetically influenced, and 2) which chromosomal regions and candidate genes are associated with OCD.

Brain circuitry and OCD

Hypotheses of OCD pathophysiology focus on **cortico-striato-pallido-thalamic** (**CSPT**) circuitry.[10,11] Although a primary pathological process underlying core OCD symptoms has not been definitively identified, functional imaging studies have established that metabolism or perfusion in CSPT circuits 1) is abnormally elevated in symptomatic patients; 2) increases during OC symptom provocation; and 3) decreases in response to successful medication or behavioral treatment.[12] The neuroimaging findings support a cohesive neuroanatomical model of OCD. In addition, activity within the orbitofrontal component of this circuit predicts response to subsequent medication or behavior therapy. However, brain imaging is not yet clinically useful for individual patients.

Recognizing OCD

Systematic screening for OCD is usually necessary for diagnosis. Our research (Pinto et al., submitted) finds five general groups of symptoms. The most common obsessions are fears of contamination, pathologic doubt ("What if something terrible happens"), unwanted aggressive, religious, or sexual thoughts, somatic illness concerns, and/or the need for symmetry and precision. The Obsessive-Compulsive Foundation has a useful online screener:[7] http://www.ocfoundation.org/ocf1070a.htm. Sample questions are: *"Do you often have disturbing thoughts or images that you can't put out of your mind?"* (e.g., worries about contamination (dirt, germs, or chemicals), getting a serious illness, over-concern that things must be perfectly placed or arranged, or religious, sexual or aggressive thoughts unacceptable to the patient. The most common compulsive rituals are checking, cleaning, counting compulsions, ordering or arranging things, and hoarding/collecting.: *"Have you felt driven to do certain things over and over again?"* (e.g., excessive or ritualized washing, cleaning, or grooming, checking light switches, stoves, locks, or checking the body for signs of illness, counting or arranging things, doing things over until they "feel right," collecting useless objects or inspecting the garbage before it is thrown out.). Compulsive hoarding and collecting are a particular source of shame. Hoarding can pose significant health risks. Patients may be especially reluctant to reveal hoarding, and often seek treatment only under pressure from family or public health authorities.

Most OCD patients have additional neuropsychiatric diagnoses, commonly depression.[8] So screening for OCD may be most fruitful in patients with symptoms of mood or non-OCD anxiety disorders (e.g,, panic, phobias, social anxiety), or eating disorders, tics, or dystonia.

OCD Across Medical Settings

People may endure OCD for a long time before seeking treatment. In our ongoing, large-scale longitudinal study, we found that patients were first treated for OCD in their early 30s on average, even though their average OCD onset was in their late teens (Pinto et al., submitted). Before seeing a psychiatrist, many people with OCD will see other physicians with one or more of several presenting complaints: nonspecific anxiety or depression; asking for medical tests to relieve unfounded obsessional fears of illness; or to seek treatment for medical sequelae of OCD. Conversely, people with OCD, afraid they will be diagnosed with a serious illness, may avoid medical settings.

OCD in Practice Settings

Dermatology

Ritualized washing or skin-picking in OCD can damage skin: [3] 20% of consecutive referrals to a dermatology clinic met OCD criteria on a screening questionnaire.[14] Only one patient had been diagnosed previously.

Internal Medicine/Primary Care.

People with OCD may complain, not of obsessions or compulsions, but of anxiety or depression. They may describe the obsessions and compulsions driving their anxiety and depression when asked about them directly. Since two thirds of patients with OCD have a lifetime history of major depression,[15] it is good practice to ask about obsessions or compulsions in patients with depression. In addition, almost 10% of medical clinic outpatients with hypochondriasis had lifetime OCD.[16]

Obstetrics/Gynecology

OCD may begin, worsen, or even improve during pregnancy.[17] Postpartum, almost a third of women with OCD may worsen.[18] Postpartum onset of OCD is usually sudden. It is not uncommon for new parents to have fleeting distressing thoughts (e.g., fear that infection or other harm will come to newborns) or to take extra precautions. But a parent with existing or new-onset OCD (sometimes it is the new father who becomes affected[19]) will experience marked distress and start excessive cleaning, ritualistic behaviors, or avoidance to cope with distress that is for most parents manageable. These behaviors can quickly become time-consuming and impair functioning. Women who develop postpartum depression are at risk for also developing obsessions and compulsions, frequently aggressive obsessions about hurting their baby.[20] Hormonal factors appear to influence OCD severity: symptoms worsened premenstrually in 40-60% of women with the disorder.[15] Similarly, biological factors as well as psychosocial factors may contribute to the well-described relationship between pregnancy and OCD. Gestational changes in estrogen and progesterone might alter serotonergic mechanisms[21] which are implicated in OCD severity.[22] A role for oxytocin, important in childbirth and lactation, has been hypothesized in OCD.[23]

Neurology

Because association between tic disorders and OCD is well known, neurologists may be the first to diagnose OCD in patients with tics. An association between OCD and certain forms of dystonia has also begun to be appreciated.[24] More rarely, OCD symptoms appear or dramatically worsen after brain lesions or injury (eg, tumors, basal ganglia ischemia,[25] or closed head trauma). OCD secondary to neurologic illness may be late onset (after age 40), have atypical content, or be associated with new neurological symptoms.[26]

Pediatrics

OCD very commonly develops in childhood, so pediatricians may be the first physicians to evaluate affected individuals. Usually there is a gradual prepubertal onset of obsessions and compulsions, not infrequently associated with tic disorders and attention deficit difficulties. Treatment is similar to that of adults with OCD. Behavioral approaches are the treatment of choice.[27] Cautious use of serotonin transporter inhibitors remains the cornerstone of medication treatment for pediatric OCD.[28] The recent controversy over the use of this class of medications in children has highlighted the need for close patient monitoring, especially early in treatment. There has also been consistent interest in a subgroup of children characterized by an abrupt OCD onset and an episodic course associated with **Group A B-hemolytic Streptococcal (GABHS)** infections. Some children are hypothesized to have OCD caused by cross-reactions of anti-GABHS antibodies with brain antigens, as is postulated for Sydenham's chorea, the neurological variant of rheumatic fever. Illness in this group is known as **pediatric autoimmune**

Helpful OCD information resources for practitioners, patients and families

Obsessive-Compulsive Foundation (OCF) phone: 203:315-2190; www.ocfoundation.org

National Institute of Mental Health OCD Facts - http://www.nimh.nih.gov/publicat/ocdfacts.cfm

Anxiety Disorders Association of America (ADAA) phone: 301.231.9350; www.adaa.org

National Alliance for the Mentally Ill (NAMI). Phone: 1-800-950-NAMI [6264] www.nami.org

National Depressive and Manic Depressive Association (NDMDA) phone: 800.826.3632; www.ndmda.org

National Mental Health Association (NMHA) phone: 800.969.6642; www.nmha.org

Internet Mental Health OCD Synopsis (and links) http://www.mentalhealth.com/dis/p20-an05.html

U.S. Surgeon General's Report on Anxiety Disorders: http://www.surgeongeneral.gov/library/mentalhealth/chapter4/sec2.html

A handout entitled "A Guide for Patients and Families" is available in the Expert Consensus Guidelines for Obsessive-Compulsive Disorder - http://www.psychguides.com

Books:

Programs for self-paced behavior therapy are found in a number of books, including:

Stop Obsessing by Edna Foa

Getting Control by Lee Baer

The OCD Workbook by Bruce M. Hyman and Cherry Pedrick

A more cognitive approach is described in:

Brain Lock by Jeffrey Schwartz

neuropsychiatric disorders associated with streptococcal infections (PANDAS).[29] In this group acute and prophylactic antibiotic use might be effective.[30]

Treatment

The psychotherapy and medication management of OCD are specific. Behavioral techniques are effective for OCD; the supportive or insight-oriented therapies are not.

Behavior therapy, or "exposure and response prevention," is a systematic program of deliberate exposure to OCD symptom-provoking situations under expert therapist guidance. Well-tested in clinical studies, It is the treatment of choice for most patients. Patients are coached to resist the compulsive urges that arise in response to symptom triggers, and to tolerate the temporary (though possibly very marked) resulting increased anxiety. With practice, anxiety and compulsive urges provoked by successive exposures gradually diminish. Meta-analyses of randomized controlled trials demonstrate that behavior therapy is effective for OCD.[31] Behavior therapy may reduce OCD symptoms in patients who remain symptomatic despite **serotonin reuptake inhibitor (SRI)** monotherapy.[32] Unfortunately, finding an experienced behavior therapist can be difficult (one referral resource is the Association for Advancement of Behavior Therapy: http://www.aabt.org/). About half of OCD patients may refuse to begin or fail to complete a behavior therapy trial: they cannot tolerate the intense anxiety induced by exposure to symptom triggers. Newer cognitive-behavioral approaches to OCD, emphasizing changing dysfunctional thinking as well as behavior, remain early in development and clinical testing, but appear promising for patients who have more purely obsessional symptoms or for those who are reluctant to engage in exposure therapy.[33] Some patients can engage in behavior therapy successfully after medication treatment. This is similar to our experience that a subgroup of patients with disabling OCD resistant to all proven treatments may benefit from behavior therapy after, but not before, neurosurgery for the illness.

Supported by several meta-analyses of randomized controlled trials,[4] **serotonin reuptake inhibitors (SRIs)** (including fluvoxamine, fluoxetine, sertraline, paroxetine, citalopram, and the non-selective tricyclic SRI, clomipramine) are more effective for OCD than other classes of antidepressants. Escitalopram, the *s*-enantiomer of citalopram, appears effective anecdotally. Higher SSRI doses may be required for maximal reduction of OCD symptoms compared to those of other psychiatric disorders. Venlafaxine, a serotonin and norepinephrine transporter inhibitor which preferentially affects serotonin uptake at lower doses, also appears effective in OCD.[35] Clomipramine, the first medication used successfully for OCD, might be more effective than the newer selective SRIs,[36] but is not used as a first-line treatment because of its comparatively greater side effects. Therapeutic benefit of SRI treatment usually begins within three months after an adequate dose is reached, and may evolve over one year or more.[2] Overall, the effectiveness of SRI monotherapy for OCD ranges from complete abolition of symptoms to no effect. About 40 to 70% of patients experience substantial benefit. In patients who prove resistant to SRI monotherapy, controlled and open data support the use of selected combination medication strategies.[37] These should generally be guided by a specialist. OCD is generally chronic. Stopping SRI treatment leads to relapse, delayed by weeks to months, in the majority of patients.[38] However, relapse rates are notably lower for patients who successfully complete a course of behavior therapy. The hoarding/collecting subtype of OCD is particularly difficult to treat, either with medications[39] or behavior therapy.[40]

Intractable or "Malignant" OCD and Neurosurgery

Although most patients with OCD benefit from conventional treatments, some have severe, chronic illness that resists treatment. The mid-20th century experience with prefrontal lobotomy remains an enduring caution in any use of psychiatric neurosurgery. Current stereotactic methods, developed empirically, use more focal and precise targets, with much lower morbidity. An increasingly specific neurobiological rationale for psychiatric neurosurgery has emerged from neuroimaging research (see above). We recently reviewed the efficacy and safety of lesion procedures for OCD[41] and the non-destructive technique of **deep brain stimulation (DBS)**[42] as a promising new treatment. We are leading a multidisciplinary team, including neurosurgeons Georg Noren and Gerhard Friehs, to study the safety and efficacy of two approaches: gamma knife anterior capsulotomy, a lesion procedure, and the newer nondestructive technique of DBS. Potential candidates come from the subgroup of severely affected OCD patients who fail to benefit after adequate trials of all proven behavioral and medication treatments. Results of studies using either procedure, which target the same fronto-basal brain circuits, are grounds for cautious optimism. The **Food and Drug Administration (FDA)** is considering an application from Medtronic, Inc., the manufacturer of the DBS devices approved for movement disorders, for humanitarian use of this therapy in otherwise-intractable OCD. Further development of neurosurgical approaches for OCD and other neuropsychiatric illnesses, including intractable depression, will require a major commitment across disciplines, including psychiatry, neurosurgery, neurology, neuropsychology, bioengineering, and bioethics.

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Improving the Diagnosis and Treatment of Alzheimer's Disease

Stephen Salloway, MD, MS

The field of Alzheimer's research has progressed since the first case report of Auguste D in the Allgemeine Zeitschrift fur Psychiatrie in 1907.[1] As we approach the 100th anniversary of the discovery of Alzheimer's disease, it is worthwhile to reflect on the advances in diagnosis and treatment, and the work that remains to be done.

Dr. Alzheimer carefully described the symptoms of his patients with chronic mental disorders, following them throughout the course of their illness, and performing post-mortem brain examinations, whenever possible, to learn the etiology of their mental disturbance. His training as a psychiatrist included formal training in neurology, neuropathology, and histology.

Alzheimer's first patient, Auguste D, became jealous of her husband at the age of 51. Alzheimer described her "rapidly worsening memory weakness" and trouble finding her way around her house. At times she feared she would be murdered, and she yelled loudly. When she came to Dr. Alzheimer's hospital, she was disoriented to time and place and had poor memory, trouble producing the correct word and difficulty understanding what was said to her. She died 4.5 years after the onset of symptoms.[2]

At post-mortem examination Alzheimer described her brain as "evenly atrophic with atherosclerosis in the large cerebral vessels." Bielschofsky stain, still used today to detect amyloid plaques, showed peculiar numbers of neurofibrils with neurons replaced by tangled bundles of fibrils and "deposits of pathological metabolic products inside neurons." Alzheimer felt that this was a peculiar disease that did not fit any entity known in his day. He speculated that there were many psychic diseases that would later be revealed to have histological explanations.

Why is there an explosion in the number of cases of Alzheimer's disease?

Alzheimer reported only a handful of cases of the dementia that was later to bear his name because **Alzheimer's disease** (AD) was uncommon when life expectancy was much shorter. The incidence of AD doubles every five years after age 65, reaching a level of 30-50% in people 85 and older. Age is far and away the biggest risk factor for Alzheimer's disease. People 75 and older are the fastest growing segment of the US population and we are facing a world wide explosion of Alzheimer cases as the population ages.[3]

...Alzheimer's disease was uncommon when life expectancy was much shorter.

Alzheimer diagnosed his first patient in the moderate stage of dementia. Are we now diagnosing AD earlier?

The general consensus today is that the amyloid deposits, described by Dr. Alzheimer, begin to accumulate many years before the onset of symptoms. Auguste D had probably been experiencing neuronal degeneration for a number of years before she was seen by Dr. Alzheimer.

Are we making the diagnosis any earlier today? The answer is yes and no. Half of AD cases are diagnosed in the moderate stage, with symptoms similar to Alzheimer's first patient. How can we diagnose AD earlier when the symptoms are mild? Modern medicine is based on the foundation of early diagnosis and treatment of chronic disease to prevent or delay the onset of morbidity or disability. Degenerative disorders such as AD should be handled in the same way as other serious chronic disorders in the elderly. The most important advance we can make is to debunk the myth that cognitive impairment is normal in the elderly. Serious cognitive impairment and dementia, though common in the elderly, is not a normal part of aging.

What can we do to improve the diagnosis of Alzheimer's disease?

Knowing the cognitive status is important to the care and well being of older patients. Patients with AD experience an early phase before the full blown dementia syndrome called **mild cognitive impairment** (**MCI**). The amnestic form of MCI, is characterized by loss of short-term memory with only mild impairment in activities of daily living. Patients with amnestic MCI have subjective and objective impairment in short-term memory. Behavioral symptoms such as depression, irritability, anxiety, and loss of motivation often accompany or precede the memory loss and may be the first signs of AD. Clinicians can be trained to recognize the symptoms of amnestic MCI and early dementia. Brief screening for cognitive impairment should be routinely conducted for people 70 and over in the primary care setting during annual physical examinations or at any age when cognitive symptoms are present. The screen should consist of simple questions about cognition, function and behavior posed to the patient and someone who knows the patient well.[4]. Use of written or computer-based questionnaires in the office may increase the efficiency of administration. A simple word recall task and clock drawing test could be conducted during the office visit to directly assess the patient's cognitive status to help determine which patients require further evaluation.

Are there biomarkers that can reliably predict the development of AD?

An antecedent biomarker of disease is a characteristic that is objectively measured and evaluated that indicates a particular risk or likelihood that a clinically detectable disease will occur in the future. For example, high levels of **low density lipoprotein**, (**LDL**), lead to build up of cholesterol in blood vessels and atherosclerosis. Is there an LDL for Alzheimer's disease? The answer is not yet. However,

changes in brain imaging, alterations in **cerebrospinal fluid (CSF)** metabolites, and genetic markers offer the best hope of future biomarkers for AD.

The decrease in whole brain and hippocampal volume occurring throughout the course of Alzheimer's disease can be quantitatively measured on MRI and used to predict the likelihood of progression from normal aging and MCI to dementia[5]. Mild decreases in glucose metabolism in the temporal and parietal cortices on **positron emission tomography (PET)** scanning may be the first sign of AD in people at risk for developing dementia. Amyloid imaging, using a tracer in PET imaging that binds to amyloid in plaques, is the most exciting area of dementia imaging research. This technique should prove useful in detecting AD early and can be used as an outcome measure in treatment trials aimed at lowering brain amyloid.[6] The results of the ongoing NIH-sponsored multi-year Alzheimer's Disease Neuroimaging Initiative should yield valuable information for the development of imaging biomarkers in dementia.

In the early phases of AD levels of CSF ABeta42, a toxic amyloid metabolite, go down and levels of CSF tau go up.[7] Recently CSF phosphotau was shown to be a reliable marker of conversion to dementia in patients with MCI.[8] Utilizing CSF biomarkers in the diagnosis of AD will require overcoming psychological barriers to performing more frequent lumbar punctures in patients with cognitive impairment. Colleagues in Europe have demonstrated a high level of patient acceptance of the routine performance of lumbar punctures in the assessment of memory disorders.

Genetic testing is gaining prominence in the diagnosis of AD. AD can be definitively diagnosed in family members with specific autosomal dominant mutations by genetic testing. The **Apolipoprotein Epsilon genotype (ApoE4),** can denote the relative risk for developing AD, but the presence or absence of one or more ApoE4 alleles does not definitively confirm or refute the current or future diagnosis of AD. The presence of one or more ApoE4 alleles increases the rate of conversion from MCI to AD and has been associated with a longer delay to conversion to dementia in MCI subjects receiving a cholinesterase inhibitor.[9] The ApoE4 test, though commercially available, is still a research tool and is not currently recommended for use in clinical practice.

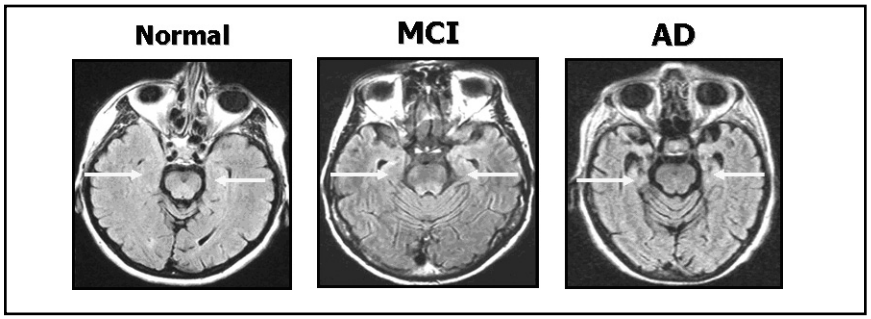

Figure 1. Change in hippocampal volume form normal aging through AD.

What can be done to promote healthy brain aging?

As the population ages the focus of primary care will be to promote healthy aging and especially healthy brain aging. There is growing evidence that maintaining an active lifestyle filled with mental stimulation, social engagement, and regular physical activity may delay the onset of dementia. Controlling risk factors for cardiovascular disease, such as hypertension, diabetes mellitus, smoking, hypercholesterolemia, and obesity, may have a brain protective effect as well. Specific nutritional and endocrine factors, yet to be determined, will also likely play a role in promoting healthy brain aging.

What treatments are available to treat AD?

The primary treatment for patients with memory disorders is helping the patient and family establish a structured daily routine that supports the individual's highest level of independent functioning. The daily routine will need to be modified over time as the patient's symptoms and functional ability change. Four **cholinesterase inhibitors (CHEIs)** have been approved for the treatment of mild-moderate AD. These medications tend to stabilize cognitive and functional ability during the first year of treatment compared to declines in these measures without treatment. After the first year, patients on CHEIs decline below baseline but more gradually than predicted from the natural history of AD. Some patients show a decrease in behavioral symptoms and a number of studies have shown a delay in time to nursing home care in patients on CHEIs.[10] An NMDA antagonist, memantine, has been approved for the treatment of moderate-severe AD. This glutamate blocker slowed decline in activities of daily living and cognition, when prescribed alone or in combination with the cholinesterase inhibitor donepezil. The recommendation is to start treatment with a cholinesterase inhibitor early in the course of AD and add memantine to the CHEI when the patient advances to the moderate stage.

Do any of these treatments work for MCI?

There are no approved treatments for MCI. However, a 36 month placebo-controlled trial in amnestic MCI showed some delay in conversion to dementia with the CHEI donepezil and a 24 week placebo-controlled trial of donepezil in amnestic MCI showed benefits for the drug in secondary measures of cognition and in patient's self-rating of memory improvement.[9,11] Preliminary reports of placebo-controlled trials with galantamine and rivastigmine in MCI did not show delay in the conversion to dementia with study medication but did show some evidence of larger brain volumes in patients receiving the CHEIs.

What new treatments are on the horizon for AD?

The five medications approved for the treatment of AD alter neurotransmitter functioning and are thought to provide symptomatic benefit without exerting a major effect on the disease course. New treatments are urgently needed that can modify the disease process and slow the progression of AD. Lowering brain amyloid, by decreasing deposition, inhibiting fibrilinogenesis or enhancing clearance is currently a major target for slowing disease progression. The following are some of the amyloid-lowering compounds in clinical development that are being tested at Butler Hospital and other memory centers in Rhode Island.

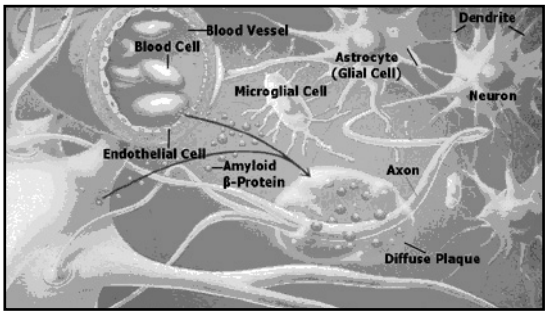

Figure 2. Lowering amyloid to slow progression of AD.

One important amyloid-lowering strategy involves modulating the enzymes, gamma and beta secretase, that cleave amyloid from the longer **amyloid precursor protein (APP).** Flurizan is an example of an allosteric modulator of gamma secretase, that shifts cleavage of APP away from ABeta42, producing shorter less toxic fragments. [12] This R-enantiomer of flurbiprofen lacks cyclo-oxygenase I and II inhibition and is in phase III trials for the treatment of mild AD.

Much attention has focused on immune-modulating agents that can bind to and enhance removal of brain amyloid. Hopes were high for an "amyloid vaccine" following a report showing a decrease in amyloid pathology in transgenic mice after vaccination with aggregated ABeta42.[13] A phase II trial of this compound, AN1792, had to be stopped after 18 of 298 subjects developed aseptic meningoencephalitis. Follow-up data revealed clearance of brain amyloid from subjects with high titers to AN1792.[14] Despite the initial disappointment, new active vaccine formulations continue in clinical development. Passive vaccine strategies, where a monoclonal antibody to ABeta42 is administered intravenously, are also in clinical trials. There is recent evidence that gamma globulin infusions may decrease brain amyloid and gamma globulin is being tested in mild-moderate AD.

Amyloid fibrils cluster together as clumps that form the core of amyloid plaques. Another amyloid lowering strategy involves the use of Alzhemed, a **glycosaminoglycan (GAG)** mimetic agent. GAG binds to ABeta, facilitating polymerization into amyloid plaques.-GAG-mimetics compete for GAG binding sites, blocking fibril formation and reducing soluble Abeta.[15] Large-scale phase III trials are underway with Alzhemed for mild-moderate AD.

Alois Alzheimer's description of the first case of AD and his discovery of the brain proteins that are the pathological hallmarks of AD have guided AD research for almost a century. We still often diagnose dementia late in the disease course when there is significant disability; but systematic screening of cognitive impairment in older people in the primary care setting, including input from an informant, will lead to earlier recognition of AD. New brain imaging techniques, measurement of blood and CSF metabolites, and genetic markers will be increasingly available to aid diagnosis. Medications are available to ease the symptoms of AD, and the search is on for disease-modifying treatments that can slow the progression and eventually delay the onset of AD. The new treatments focus on decreasing brain amyloid.

The Senior Specialty Service at Butler Hospital provides comprehensive evaluation and treatment for older people with dementia, depression, and other psychiatric disorders. There are several centers dedicated to patients with dementia within the Brown system, including ours at Butler Hospital. For more information about clinical trials for patients with Alzheimer's disease, contact the Memory and Aging Program at 401-455-640 (www.memorydisorder.org,) or the Rhode Island Alzheimer's Association at 401-421-0008 (www.alz-ri.org).

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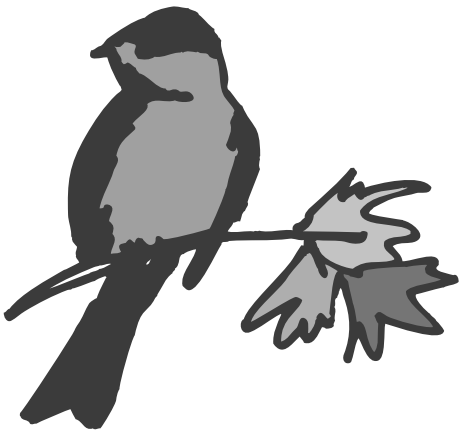

Cigarette Smokers Who Have Difficulties Quitting:
The Role of Negative Mood

Ana M. Abrantes, PhD, Kathleen M. Palm, PhD, David R. Strong, PhD, and Richard A. Brown, PhD

WHILE THERE HAVE BEEN DECREASES in the rates of smoking among adults over the last decade, the **Centers for Disease Control and Prevention** (**CDC**) report that 25.2% of males and 20% of females, totaling 45.8 million adult Americans, smoke daily.[1] Cancer, cardiovascular diseases, respiratory diseases, adverse reproductive effects, cataracts, hip fractures, low bone density, and peptic ulcer disease are all consequences of smoking. Further, the diminished health status associated with smoking often contributes to absenteeism from work and increased use of health care services. In sum, smoking is responsible for more than 440,000 deaths each year and an economic burden of $157 billion.[2]

In this paper we briefly review existing interventions for smokers. We also describe the role of mood in hindering smokers' efforts to stop; and we describe novel interventions for smokers with depressive mood.

Smoking Interventions

Most smokers (70%) express a desire to quit. The challenge has been identifying the most effective points of intervention and matching treatments to individual patients. Conducting even a brief assessment of smoking status, smoking history, level of nicotine dependence, and readiness to change could inform physicians' decisions to recommend one treatment versus another. Prochaska and DiClemente[3] proposed that different interventions are more effective based on a smoker's stage of readiness to quit (e.g., precontemplation, contemplation, preparation, action, and maintenance). For example, if a smoker is in precontemplation, a brief motivational intervention to get the smoker to consider quitting may be a more appropriate strategy than a more intensive approach.

Available treatments for smokers include self-help manuals, brief interventions, intensive clinical interventions, and pharmacotherapy.[4] While self-help manuals can be widely disseminated, only approximately 5% of patients using them report successful long-term cessation. Thus, they are not recommended as stand-alone treatments.

Primary care providers are in a unique position to impact a large number of smokers. While many smokers do not actively seek out smoking cessation treatments, 70% of smokers see a physician each year.[5] Studies suggest that smoking cessation interventions conducted through primary care clinics are more successful when multiple health care providers are involved and when 4 or more person-to-person sessions are scheduled.[3] While brief interventions are well-suited for the primary care setting and help smokers who are initially ambivalent about quitting, more intensive interventions should be made available whenever possible.[6] Cognitive-behavioral approaches, including problem solving/skills training, aversive smoking strategies, cue exposure, and nicotine fading, have resulted in higher cessation rates than brief interventions. Finally, pharmacotherapy can aid smoking cessation efforts. Common pharmacologic interventions include buproprion, nicotine gum, patch, lozenges, and nasal spray. Pharmacotherapy, in conjunction with behavioral counseling, has been associated with higher cessation rates (17-30%) at 6 months when compared to placebo (10-17%).

Despite the varied approaches to smoking cessation, long-term abstinence rates remain low. Seventy to eight-five percent of smokers who attend treatment programs relapse within one year.[6] Rather than expecting any single treatment to be a panacea for all smokers, researchers have identified subgroups of smokers at high-risk for relapse and developed targeted treatments. One risk factor is negative mood.

Negative Mood as Risk Factor for Relapse

Recent evidence suggests a strong link between negative mood and smoking behavior.[7] However, the exact nature of the linkage is unclear. Some evidence suggests a causal relationship between depression and nicotine dependence, while other research points to other common factor(s), such as neuroticism or general vulnerability to psychopathology, which predisposes toward both disorders. The relationship between negative mood and smoking has typically been examined with respect to history of **Major Depressive Disorder** (**MDD**) and depressive symptomatology. Below is a description of these relationships along with a review of treatment developments for this high-risk subgroup of smokers.

Major Depressive Disorder (MDD)

MDD is the psychiatric disorder most frequently associated with cigarette smoking in adults.[8] A history of MDD is not only more common among smokers than non-smokers, but is related to severity of nicotine withdrawal symptoms and poorer long-term cessation outcomes. Researchers have found that between 31% and 46% of smokers entering a treatment program report a lifetime history of major depression. In a catchment area survey[9] positive depression history was more common among smokers vs. nonsmokers (6.6% vs. 2.9%). These findings were not the result of co-occurrence of major depression with other psychiatric disorders but were specific to a major depression diagnosis.

Among smokers who report unsuccessful quit attempts, severity of nicotine withdrawal symptoms is associated with a history of MDD. In a prospective study,[10] intensity of withdrawal symptoms was elevated in smokers with past MDD, compared to those without a past history of depression. Others have found that increases in ratings of sadness following a quit attempt predicted subsequent failure.[11] Further, a positive depression history has been found to be associated with greater frequency of regular smoking and decreased likelihood of quitting smoking. Thus, history of MDD is associated with

greater smoking prevalence, greater nicotine dependence among smokers, increased nicotine withdrawal among smokers who try to quit, and inability to quit smoking.

Depressive Symptoms

Evidence from community and treatment-seeking samples suggests that current level of depressive symptoms may be a more reliable predictor of smoking cessation failure than history of MDD. For example, pre-cessation negative moods and increases in negative moods after a quit attempt have been associated with early lapse to smoking among smokers in cessation treatment. Further, current depressive symptoms and negative affect have been consistently associated with poorer smoking outcomes.

Cross-sectional analyses from the **National Health and Nutrition Examination Survey (NHANES)** and the NHANES Follow-up Study[12] showed that, as depressive symptoms increased, the prevalence of smokers increased whereas the quit ratio decreased. Results from treatment-seeking samples concur. For example, adult smokers who have been unable to quit smoking after a 4-week, smoking cessation treatment program tended to have higher baseline depression scores than did those who quit successfully. Other researchers have reported that 34% of smokers participating in smoking cessation treatment demonstrated significant levels of baseline depressive symptoms and that these high depressive symptom smokers were less likely to be abstinent at 3-month follow-up than were low depressive symptom smokers.

Interventions for Smokers with Negative Mood Disturbances

Cessation treatments that provide strategies for managing depressive symptoms and negative mood have been developed and examined.[13,14]

Cognitive-Behavioral Approaches for Mood Management

In the early 1990s, smoking cessation interventions were developed for smokers with a past history of MDD. These interventions consisted of incorporating **cognitive-behavioral skills for dealing with depression (CBT-D)** along with standard smoking cessation treatment. CBT-D included the following components, presented as alternatives to smoking that could combat feelings of depression and fill the void following the loss of smoking as a reinforcing activity: daily mood rating, increasing pleasant activities, increasing positive-decreasing negative thoughts, identifying and challenging depressed and distorted negative thoughts, social skills-assertiveness training, and maintaining gains.

Contrary to prediction, CBT-D did not produce significantly higher abstinence rates compared to standard treatment for smoking among samples of past MDD smokers. However, smokers with recurrent past MDD (i.e., two or more past MDD episodes) who received CBT-D had significantly higher abstinence rates than those receiving standard treatment.[13] Therefore, smokers with recurrent depression may benefit more from mood management treatments for smoking cessation than smokers with only one or no major depressive episode history.

Pharmacotherapy

While studies have examined the efficacy of nortriptyline and fluoxetine for smoking cessation, much of the focus has been on bupropion hydrochloride. In a recent study, bupropion, in comparison to placebo, resulted in better smoking outcomes among smokers receiving CBT-D and/or standard treatment. The federal **Food and Drug Administration (FDA)** has approved its use for the treatment of cigarette smoking. The impact of cognitive-behavioral mood management treatments and bupropion have primarily been studied independently. It is possible that the combination of these approaches could synergistically influence both mood and smoking outcomes among depressed smokers.

New Directions

Telephone Counseling

Telephone counseling is becoming a front line treatment in public health efforts to decrease the morbidity and mortality associated with smoking. Telephone counseling is cost-effective, efficacious, efficiently reaches large numbers of smokers, and can target at-risk subpopulations. A majority of smokers, moreover, favor telephone counseling over face-to-face programs. A next step is to create an integrated treatment that combines proven methods from telephone counseling with interventions that have shown promise with smokers with recurrent MDD but that are too clinically intensive to attain widespread usage. Although smokers with a history of recurrent MDD are likely to make up a significant proportion of smokers seeking telephone counseling for smoking cessation and are likely to have worse outcomes, no specialized telephone counseling intervention is available for these smokers with MDD. An integrated telephone counseling treatment that combines proven methods from telephone counseling with effective mood management skills training may improve outcomes among smokers with recurrent MDD.

Rhode Islands Tobacco Control Program offers 1-800-TRY-TO-STOP and has multilingual services. More information is available at http://www.trytostop.org

Treatment for Early Lapsers

A significant percentage of individuals attempting smoking cessation lapse within days, and very few of these achieve abstinence from smoking. In our own work,[15] we investigated the association between negative affect and early smoking lapse. Relative to delayed relapsers, immediate relapsers had higher baseline levels of depressive symptoms, a greater tendency to react to stress with negative affect and greater urge to smoke following 12-hr nicotine deprivation. In addition, we assessed individuals' distress tolerance, as indexed by their persistence on psychological and physical challenge tasks. The physical challenge tasks consisted of inhalations of **carbon dioxide (CO_2)** enriched air and of a timed, breath-holding procedure, while the psychological challenge consisted of the completion of a stressful version of the **Paced Auditory Serial Addition Task (PASAT)**, a mental arithmetic challenge task. To examine task persistence, participants were given the option of terminating each challenge task prior to its scheduled end point. Immediate relapsers were more likely to terminate the challenge tasks and had a shorter duration of breath-holding than delayed relapsers. Importantly, these differences

in responding could not be attributed to the level of nicotine dependence, number of years smoked, number of serious quit attempts, and other theoretically-relevant characteristics.

Distress tolerance is a reaction to states of affective discomfort. Acute nicotine withdrawal produces uncomfortable interoceptive symptoms. Such distress could be considered an inherent emotional consequence of smoking. A low threshold for tolerating such unavoidable types of distress could plausibly be associated with increased smoking behavior. Specifically, an inability or reduced ability to tolerate such distress may interfere with efforts to establish non-smoking behavior change. From this perspective,[16] it is not simply affective distress that influences smoking outcomes, but one's response to affective distress and related withdrawal.

Convergent evidence suggests that distress tolerance may play a key role in the success of smoking cessation efforts. We are developing a specialized protocol for the treatment of nicotine dependence in early smoking lapsers. This protocol utilizes behavioral exposure to nicotine withdrawal and training in Acceptance and Commitment Therapy skills to facilitate tolerance of distress and persistence in the face of discomfort.

Conclusions

After years of declining smoking prevalence, smoking rates have begun to stabilize, suggesting that current smokers have been unable to quit successfully due to risk factors or characteristics, such as depressive symptoms, that make it particularly difficult to quit. Interventions that have proven useful for smokers in the past may have limited effectiveness for this high-risk, residual group of smokers. As a result, we believe that significant contributions can be made to the field of smoking cessation through the development of specialized smoking cessation treatments for smokers at risk for failure due to negative mood.

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What Physicians Need To Know About Body Dysmorphic Disorder

Katharine A. Phillips, MD

PHYSICIANS—ESPECIALLY PSYCHIATRISTS, dermatologists, plastic surgeons, primary care physicians, and pediatricians—need to understand **body dysmorphic disorder (BDD)**. Even though BDD is a mental disorder, a majority of patients with this illness seek surgery or nonpsychiatric medical treatment (e.g., dermatologic treatment) for their symptoms.[1-3] Patients tend to be dissatisfied with these treatments.[1-3] Some sue, or are even violent towards, the treating physician.[4] As a noted dermatologist stated: "I know of no more difficult patients to treat than those with body dysmorphic disorder."[4]

Individuals with BDD are preoccupied with an imagined or slight defect in their physical appearance (for example, "scarred" skin or a "large" nose).[3] They believe that they look abnormal, whereas the appearance defects they perceive are slight or nonexistent. The appearance preoccupations go beyond normal appearance concerns: they cause clinically significant distress or impairment in functioning. While such concerns may sound trivial, on the Medical Outcomes Short Form (SF-36), individuals with BDD have poorer quality of life than patients with depression, type II diabetes, or a recent myocardial infarction.[5,6] From 78% to 81% have experienced suicidal thinking; 22%-28% have attempted suicide.[7-9] A retrospective study of dermatology patients known to have committed suicide over 20 years found that most of the patients who suicided had acne or BDD.[10] In the only prospective study of the course of BDD, the rate of completed suicide was higher than has been reported for any other mental illness.[11]

The Body Dysmorphic Disorder and Body Image Program at Butler Hospital (www.BodyImageProgram.com or www.butler.org/body.cfm?id=123) is a leading research center for BDD.

A PATIENT WITH BODY DYSMORPHIC DISORDER

Ms. A, a 27-year-old single white female, presented with a chief complaint of "I see a lot of skin doctors." She had consulted with dozens of dermatologists, to no avail. Convinced that she had severe acne, scars, and "veins" on her face, she frequently checked mirrors, spent hours a day applying makeup, and picked at her skin. She stated that because she so incessantly sought reassurance from dermatologists, "most of the dermatologists in Boston are probably seeing therapists because of me."

Ms. A. had dropped out of college, was unemployed, and was housebound. She had attempted suicide and had been psychiatrically hospitalized. Treatment with numerous antibiotics and isotretinoin had not diminished her concerns. However, treatment with psychotropic medication (fluoxetine [Prozac]) significantly improved her BDD symptoms. Her preoccupation, distress, and suicidality diminished, and her functioning improved.

CLINICAL FEATURES OF BODY DYSMORPHIC DISORDER

BDD occurs in all age groups. It most often begins during early adolescence.[9,12] The gender ratio appears to be in the range of 1:1 to 3:2 (female:male).[9,12] Individuals with BDD may describe themselves as ugly, unattractive, "not right," deformed, or abnormal. Some describe themselves as "hideous" or looking like a "freak" or "monster." Patients most often focus on the skin (e.g., acne, scarring, skin color), hair (e.g., hair thinning or excessive facial or body hair), or nose (e.g., size or shape).[3,7,9,12] However, they can focus on any body area. The preoccupations are usually difficult to resist or control and occur for an average of 3 to 8 hours a day.[3]

Nearly all patients perform compulsive, repetitive behaviors which aim to check, hide, or fix the perceived defects.[3,9,12] These behaviors are time consuming and are difficult to resist or control. They include checking mirrors and other reflecting surfaces, comparing with other people, excessive grooming, touching the body areas, seeking reassurance about the perceived flaws, changing clothes, and compulsively buying clothes or makeup. Camouflaging the perceived defects –with clothing, makeup, a hat, hair, hand, or body posture – is common. About one quarter of patients tan to cover perceived acne scarring, facial marks, or "pale" skin.[13] One third to half of patients pick their skin[9,12,14] to try to improve the skin's appearance (e.g., "smooth out" or remove blemishes). However, the picking, particularly with implements like pins, needles, razor blades, or knives, can damage the skin. Thus, some patients with BDD who pick their skin are an exception to the rule that people with BDD look normal. Skin picking is occasionally life-threatening—for example, when major blood vessels are ruptured.[14]

Level of functioning is typically very poor.[3,5,6] A high proportion of patients are unemployed, unable to stay in school, socially isolated, or even housebound.

BODY DYSMORPHIC DISORDER IS RELATIVELY COMMON

The reported prevalence in community and nonclinical student samples ranges from 0.7% to 13%.[3] A US study[15] found that 12% of 268 patients seeking dermatologic treatment screened positive for BDD. A study from Turkey found that 9% of acne patients had BDD.[16] The prevalence of BDD in cosmetic surgery settings has ranged from 6%-15%.[3] A study of 122 general psychiatric inpatients found that 13% had BDD, which was more common than schizophrenia, obsessive compulsive disorder, post-traumatic stress disorder, and eating disorders.[17] In that study, 81% of patients with BDD said that BDD was their major or biggest problem.

SURGERY AND NONPSYCHIATRIC MEDICAL TREATMENT: INEFFECTIVE TREATMENTS FOR BDD

A majority of patients with BDD pursue surgical, dermatologic, dental, or other medical treatment for their perceived defects—some ardently.[1,2] In fact, patients with BDD have been dubbed "polysurgery addicts."[18] Dermatologic treatment appears most frequently sought and received (most often, topical acne agents), followed by surgery (most often, rhinoplasty).[1,2] BDD patients appear to respond poorly to these treatments. In one study of 250 subjects with BDD, only 7% of 484 treatments (retrospectively assessed) led to overall improvement in BDD.[1] In another study (n=200), only 4% of all procedures improved overall

BDD symptoms.[2] In a study of 50 subjects with BDD, 81% reported being dissatisfied with past medical consultation or surgery.[7] In the only prospective study of the course of BDD, receipt of surgery or nonpsychiatric medical treatment for BDD concerns was not associated with a higher probability of remission from BDD.[19]

In a 2001 survey of plastic surgeons,[20] respondents reported that BDD patients tended to have poor surgical outcomes; 40% of respondents reported that a BDD patient had threatened them legally and/or physically. Some patients sue the surgeon or dermatologist despite an objectively acceptable outcome. Occasional patients murder the physician.[3]

Psychiatric Treatments Are Often Effective for BDD

Serotonin-reuptake inhibitor (SRIs, or SSRIs) medications and **cognitive-behavioral therapy (CBT)** are often effective for BDD. These treatments may be provided together or individually. It is not known whether SSRIs or CBT are more efficacious for BDD. However, in the author's experience, an SSRI is always indicated for suicidal patients.

SSRIs are considered the medication of choice for BDD. Their efficacy is supported by controlled studies, open-label studies, and clinical series.[3,21-23] SSRIs often diminish the appearance preoccupations, associated distress, and BDD behaviors. Insight, functioning, and associated symptoms such as depression usually improve. The best-studied SSRIs for BDD are fluoxetine (Prozac), fluvoxamine (Luvox), clomipramine (Anafranil), escitalopram (Lexapro), and citalopram (Celexa).[3,21] However, paroxetine (Paxil) and sertraline (Zoloft) also appear efficacious.[3,21] Reported BDD response rates range from 63% to 83%.[3,21] It appears that a substantial proportion of patients who fail an initial SSRI trial will respond to a subsequent SSRI.[3,21] Other psychotropic medications, including other antidepressants (with the possible exception of venlafaxine), appear less effective than SSRIs, although data are limited.[3,21]

Successful treatment often requires SSRI doses that are higher than those typically used for depression.[3,21] Some patients benefit from doses that exceed the maximum recommended dose (this approach is not advised for clomipramine, however). Most patients with BDD, however, appear to receive relatively low SSRI doses, which appears associated with a poorer treatment response than higher doses.[24]

The average time to SSRI response has varied from 4-5 weeks to 9 weeks.[3,21] However, many patients will not respond until the 10th or 12th week of SSRI treatment, even with rapid dose titration. If SSRI response is inadequate after 12-16 weeks of treatment, and the highest dose recommended by the manufacturer or tolerated by the patient has been tried for 2-3 weeks, another medication should be tried.

CBT is the psychotherapy of choice for BDD.[25,26] CBT focuses on changing problematic thoughts and behaviors. CBT for BDD usually consists of 1) cognitive restructuring, which focuses on identifying inaccurate beliefs and cognitive errors and developing more accurate and helpful new beliefs; 2) behavioral experiments, in which patients empirically test inaccurate and dysfunctional beliefs; 3) ritual (response) prevention, which teaches patients how to resist repetitive behaviors such as mirror checking and excessive grooming; and 4) exposure, which helps patients enter into feared and avoided situations without ritualizing. Mindfulness and mirror retraining may be helpful. The optimal session frequency and treatment duration are unclear. In the author's experience, most patients require weekly or more frequent sessions for at least 5-6 months, plus regular homework. Maintenance/booster sessions should be considered to reduce the risk of relapse.

Although psychotherapy research is very limited, it appears that general counseling, supportive psychotherapy, and insight-oriented psychotherapy alone are usually not helpful for core BDD symptoms.[3]

The Body Dysmorphic Disorder and Body Image Program at Butler Hospital offers medication studies and a CBT study for adults with BDD, as well as a medication study for children and adolescents.

How to Recognize and Diagnose BDD

Clinicians under-recognize and underdiagnose BDD.[3,17] In mental health settings, patients are often ashamed of their symptoms and reluctant to reveal them, because they worry they will be considered vain or not taken seriously. In addition, many patients do not want to draw attention to the perceived flaws. Instead, patients may reveal only their depression, anxiety, or substance use. However, patients generally want mental health clinicians to be aware of their BDD symptoms.[17]

BDD can be diagnosed using straightforward questions. (Table 1[3]) A useful screening question is: "Are you very worried about your appearance in any way? *OR* Are you unhappy with how you look?" If the patient replies affirmatively, you can ask more about the patient's concerns, determining whether the concerns are preoccupying and causing emotional distress or impeding functioning. Problems in functioning may consist of difficulty concentrating, being late for work or school, missing work or school, decreased productivity, avoiding dating, marital discord, problems with intimacy, and avoiding social interactions.

Most patients believe that they have an accurate view of their physical flaws. It

Table 1: Questions to Ask Patients To Diagnose BDD*

1) Are you very worried about your appearance in any way? *OR* Are you unhappy with how you look? *If yes:* what is your concern?
2) Does this concern preoccupy you? That is, do you think about it a lot and wish you could worry about it less? If you add up all the time you spend each day thinking about your appearance, how much time would you estimate you spend?
3) What effect has this preoccupation with your appearance had on your life? For example, has it.....
   - Significantly interfered with your social life, dating/marriage, school work, job, other activities, or other aspects of your life?
   - Caused you a lot of distress?
   - Affected your family or friends?

* BDD is diagnosed in patients who are 1) concerned about a minimal or nonexistent appearance flaw, 2) preoccupied with the "flaw" (for example, think about it for a total of at least an hour a day), and 3) experience clinically significant distress or impairment in functioning as a result of their concern.

usually is not effective to try to talk them out of their concern. It is also very important not to ridicule the patient or trivialize their concerns, because this can be deeply wounding and even trigger suicidal thinking. Instead, it is best to ask the questions in a supportive way, keeping in mind that it can take courage for patients to reveal their appearance concerns, even to clinicians.

You can also look for clues to the presence of BDD; e.g., excessive mirror checking, unusual camouflaging (e.g., covering one's face with a hat or hair), or seeking reassurance about one's looks.[3] Other clues include depression, anxiety, social avoidance, being housebound, and referential thinking (i.e., believing that other people take special notice of the person in a negative way because of how they look).

How to Recognize BDD in a Medical or Surgical Setting

Diagnosing BDD in surgical and medical settings can be complex, as many patients seek surgery or dermatologic treatment for minimal flaws. The optimal approach to diagnosing BDD in such settings has not been well studied. However, in the author's experience, the questions in Table 1 can be applied in these settings. Although patients who seek cosmetic surgery have appearance concerns, most patients are not preoccupied with these concerns, and most do not experience clinically significant distress or impaired functioning as a result of their concerns.

A self-report screening measure, the Body Dysmorphic Disorder Questionnaire-Dermatology Version, developed for use in a dermatology setting, contains questions similar to those used in psychiatric settings.[27] Surgeons and dermatologists may find it helpful to ask additional questions; e.g., 1) what are the patient's expectations for the cosmetic procedure, and 2) has the patient had cosmetic procedures in the past, and, if so, what was the outcome and were they satisfied? Patients who expect the procedure to significantly improve their life (e.g., they will start to date or get a job) and those who were dissatisfied with past procedures despite an objectively acceptable outcome may have BDD and are probably poorer candidates for cosmetic procedures.

How to Encourage Patients to Accept Psychiatric Treatment

Most BDD patients are convinced or fairly certain that they look abnormal.[3,12] Very few, prior to treatment, realize that the perceived defects are slight or nonexistent. In addition, many patients do not realize that their appearance concerns are due to a psychiatric disorder. This can make it difficult to persuade patients to accept psychiatric care.

An important first step is to take patients' concerns seriously. Ask them the questions in Table 1 to assess whether they have BDD. Do not dismiss their concerns or simply reassure them that they look normal. Most patients do not believe reassurance and may interpret it as trivializing their concerns. On the other hand, do not agree that something is wrong with their appearance: this may even trigger suicidal thinking. It can be helpful to note that you view their appearance differently than they do and that you think they may have a body image disorder known as BDD.

If you think a patient has BDD, you can explain why the diagnosis may apply to them. You can also convey that BDD is a relatively common and treatable body image disorder in which people view their appearance differently than other people do, for reasons that are not well understood. You can emphasize that people with BDD are preoccupied with their appearance and as a result experience significant distress and difficulty functioning. Most patients can agree that this fits with their experience.

Research has not been done on how to dissuade patients from obtaining surgery, dermatologic treatment, dental treatment, and other nonpsychiatric medical treatments. Nonetheless, you can explain that these treatments do not appear to help BDD, and you cannot recommend such treatment because you worry they will not be satisfied with the outcome and may dislike their appearance even more. If the patient will not agree to forgo such treatment, encourage them to at least delay it and first try psychiatric treatment, which is much more likely to help.

Explain that effective treatments are available for BDD, and strongly encourage the patient to try them. You can also recommend reading about these treatments (e.g., www.BodyImageProgram.com).

Some patients resist the diagnosis of BDD and psychiatric treatment, insisting that they truly are ugly. With these patients it is best to avoid arguments over how they actually look and to focus on the potential for treatment to diminish their excessive preoccupation, suffering, and impaired functioning.

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Understanding Families: From Criticalness to Resilience

Alison M. Heru, MD

Family research has shifted from the study of criticalness [the tendency to criticize] to the study of family strengths and resilience. This shift facilitates the introduction of health-promoting family-oriented treatments into clinical practice. This article reviews the changes in paradigm through several medical disciplines and concludes with recommendations for the implementation of resilience research findings.

Criticalness in Psychiatry

In psychiatry, the study of the family environment began when George Brown and colleagues at the Social Research Unit in London developed the construct of **expressed emotion (EE).**[1] This construct consists of three components: criticalness, emotional over involvement and hostile comments. EE is a predictor of relapse in many illnesses,[2] including schizophrenia,[3] depressive disorders,[4] acute mania[5] and alcoholism.[6] High EE relatives, believing that the abnormal behavior of patients is under their control,[7] hold patients responsible for their actions, rather than blaming the illness.[8] This stance may lead to a critical attitude, with attempts to coerce the patients. Although initially used with psychiatric patients and their families, EE and the role of criticalness is studied in diabetes, asthma, epilepsy, rheumatoid arthritis, obesity and recovery from heart surgery.[9]

Family psychoeducation, based on the premise that families need support in their care of the patient, reduces EE. Family psychoeducation reduces relapse rates in schizophrenia, bipolar disorder and other illnesses.[10] Family psychoeducation provides emotional support, illness education, help with finding resources during crises and help with problem-solving skills. In schizophrenia, family psychoeducation reduces relapse rates by 50%.[11] Psychoeducation is also used to treat childhood conduct disorder,[12,13] obsessive compulsive disorder in children and adolescents,[14] bipolar disorder in children[15] and eating disorders.[16,17]

General Medicine

Family factors in chronic medical illness have a powerful influence on health, equal to traditional medical risk factors.[18] Marital relationships affect both mortality and morbidity rates. Emotional support is the most important support provided by families; negative, critical or hostile family relationships have a stronger influence on health than positive or supportive relationships.

Family psychoeducational interventions are successful in general medicine. The Joslin Diabetes Center in Boston developed a low-cost intervention to reduce family conflict in the management of diabetes in adolescents.[19] One hundred and five children and adolescents, aged 8 to 17 years, with insulin dependent diabetes for at least 6 years, were randomly assigned to a family-focused teamwork intervention or to standard multidisciplinary diabetes care and followed for 1 year. Both groups had the same number of visits and received the same educational materials. The family-focused intervention group emphasized family team work; at each visit one of four modules was implemented: 1) communicating about diabetes, 2) educational material review pertaining to the disease, 3) encouraging family discussion regarding elevated blood sugars and 4) facilitating the use of a log book to problem-solve out-of range-blood sugar values. The family-focused intervention prevented the expected deterioration in diabetes in adolescence.

The visit to the general practitioner can include family involvement and family education. In family practice, studies show that meeting with the family improves the patient's compliance with the treatment plan,[20,21] that involving families strengthens the alliance between the physician and the patient without lengthening the visit,[22] and having family members present at the interview sets the stage for future problem-solving involving the family.[23]

Within the general hospital, family involvement is also shown to benefit patients. For example, when parents participate in the post-anesthesia care of their child, the children cry less and require less medication. [24] These interventions require no specific expertise in working with families. Psychoedu-cational interventions are aimed at increasing family members' knowledge of the illness, improving coping skills, thus reducing criticalness.

Resilience

Family Strengths

Families can offer emotional support. Having emotional support is associated with a better outcome among elderly patients hospitalized for acute myocardial infarction.[25] In this prospective, community-based study of 194 patients, lack of emotional support was significantly associated with 6-month mortality even after controlling for severity of myocardial infarction, comorbidity, smoking, hypertension, and sociodemographic status.

Family strengths can offset family difficulties. For example, childhood and adolescent parenting quality is a predictor for competency in children and adolescents ($p<.001$). Academic achievement, conduct and peer social competence were evaluated in 205 children who were followed for 10 years. Parenting quality was protective, even in the context of severe adversity. Structure, rules, closeness, warmth, high expectations for the child's achievement and prosocial behavior are the parenting qualities associated with higher levels of competency.[26]

Families can be taught to improve their functioning. For example, parents and their children who required intensive medical care were provided with support through the **Creating Opportunities for Parent Empowerment (COPE)** program. A randomized, controlled trial was conducted with 163 mothers and their 2- to 7-year-old children. Mothers in the experimental COPE group received a 3-phase educational-behavioral intervention program; control mothers received a structurally equivalent control program. The COPE program focused on increasing parents' knowledge and understanding of the range of behaviors and emotions that young children typically display during and after hospitalization and encouraged parent participation in their children's emotional and physical care. One year after discharge, a significantly higher percentage of control group children (25.9%) exhibited clinically significant behavioral symptoms, compared with COPE children (2.3%).[27]

However, families are complex. A family's response to adversity can reveal internal resources that help the family emerge strengthened when faced with a stressor, such as illness. Therefore researchers have turned to the study of family processes to

understand what goes on within families that enables them to cope well with adversity.

Family Resilience

Family resilience is the interplay of multiple risk and protective factors that occurs over time and involves individual, family and other sociocultural influences.[28] Initially, resilience research followed individuals over the course of their lifetimes. For example, 700 children of plantation workers living in poverty in Hawaii were followed into adulthood.[29] By age 18, two-thirds of the children had done poorly, but one third were competent, caring, and confident young adults. Through midlife, all but two of these competent adults lived successful lives. All the competent adults had a significant relationship with family members, partners, coaches or teachers. These significant 'family' relationships were thought to act as mediating protective factors that positively influenced the trajectory of these children's lives. In sum, family factors were found to influence individual resilience.

Family resilience implies that a family has an internal organization or has reorganized to protect against stress. Many families report that through weathering a crisis together their relationships are enriched and more loving than they might otherwise have been.[30] In one study of families coping with a relative who had mental illness, 87.7% of families reporting experiencing family resilience and 99.2% of family members reported experiencing personal resilience.[31] One family member stated, "When a family experiences something like this, it makes for very compassionate people—people of substance. My brother created a bond among us that we will not allow to be broken."

Family resilience describes dynamic processes that foster positive adaptation to adversity. What is known about resilient families? High functioning families have strong affiliative value,[32] and approach adversity as a shared challenge. Cohesion or connectedness provides mutual support and collaboration among members when a family faces a crisis.[33,32] Clear, direct communication facilitates effective functioning, which in turn facilitates problem-solving, an essential skill in times of adversity.[34,35] The key family processes thought to contribute to family resilience occur in three main domains: family belief systems, organizational patterns and communication / problem solving.[36]

The Institute of Medicine and the National Academy of Sciences outlined the family processes that influence chronic medical illness.[37] The family protective processes linked to improved outcomes are family closeness, mutuality, connectedness, caregiver coping skills, mutually supportive family relationships, clear family organization and direct communication about the illness and its management. Family factors and processes that are linked to poorer outcomes are intrafamilial conflict, criticism and blame, perfectionism and rigidity, delayed family developmental tasks, lack of an extra-familial support system and psychological trauma related to diagnosis and treatment.

Family resilience is not immediately apparent; therefore, a clinician should assess a family for the presence of strengths, Many caregivers attending the memory disorder clinic at Butler Hospital reported a sense of reward in caring for their relatives, even in the presence of deteriorating patient functioning.[38] The caregivers reported; "feeling needed and responsible", "feeling good inside", "doing for someone, what you want for yourself", "knowing I've done my best", "being able to help", "to brighten her days", "I know he is being cared for the way he is used to" and "I feel that she is loved and not alone."

Conclusion

Family research now focuses on strengths and resilience. Focusing on criticalness left families feeling blamed and reluctant to engage with the health care system. By focusing on strengths and resilience, the medical profession can involve the family in assessment, include family members in the treatment team, and develop educational programs to help patients and family members cope with chronic disease. Evidence shows that successful family interventions are primarily educational and applicable to all health care settings. Nationally, just as clinicians promote diet, exercise and healthy life habits for their preventative effects on health, so too clinicians can promote good family functioning.

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Biomarkers in the Human Stress System:
Do they Signal Risk for Depression?

Audrey R. Tyrka, MD, PhD, Linda L. Carpenter, MD, John P. Carvalho, and Lawrence H. Price, MD

While the specific causes of major depression are unknown, family, twin, and adoption studies show that depression, like most other psychiatric disorders, involves both heritable and environmental factors. Animal and human studies indicate that exposure to early life stress (for example, childhood maltreatment or prolonged separation from a parent) leads to an abnormal neuroendocrine response to stress. In humans, this abnormal stress response may predispose individuals to the development of depression later in life. Recent developments in the field of molecular genetics have enabled investigators to begin to identify candidate genes that may be implicated in sensitivity of the biological stress system and risk for the development of major depression. Researchers have identified specific gene-environment interactions that predispose to depression. This work holds promise for the development of targeted interventions aimed at prevention and treatment of this disorder.

Depression and Dysregulation of the HPA Axis

Corticotropin-releasing hormone (**CRH**) and the **hypothalamus-pituitary-adrenal** (**HPA**) axis play a major role in coordinating the biological stress response. Dysregulation of this system frequently occurs in individuals suffering from major depression. Depressed patients have elevations of basal serum cortisol levels and CRH in **cerebrospinal fluid** (**CSF**). Post-mortem studies have revealed elevated levels of CSF CRH and a decreased density of CRH receptors in the frontal cortex of suicide victims.[1-3] A number of neuroendocrine challenge tests have probed functioning at various levels of the HPA axis. Depressed patients have non-suppression of cortisol in the **dexamethasone suppression test** (**DST**), a blunted **adrenocorticotropic hormone** (**ACTH**) but normal cortisol response to CRH, and an exaggerated ACTH and cortisol response to CRH after pre-treatment with dexamethasone (Dex/CRH test). This pattern of HPA dysfunction, often referred to as "hyperactivity," is likely due to hyper-secretion of CRH and possibly impaired corticosteroid receptor signaling.[4-7]

Is HPA Hyperactivity an Endophenotypic Marker for Depression?

There is evidence that treatment of depression is accompanied by a gradual reduction in neuroendocrine abnormalities,[4,6] suggesting that HPA dysfunction may be state-related. However, a significant proportion of depressed patients have continued HPA dysfunction during remission, and the persistence of these abnormalities predicts future recurrence of depressive episodes.[8-10] Secondly, individuals who are not depressed but are at high risk for depression due to a personal or family history have exaggerated cortisol levels that are intermediate between those of depressed patients and low-risk controls.[11] Thus, in addition to a state-related effect, there is some evidence that HPA axis hyperactivity may represent a trait marker, or endophenotype, for depression.

Moreover, evidence suggests that excessive activation of the HPA axis may be causally implicated in the development of major depression. Preclinical studies have found that stress and glucocorticoid exposure result in decrements in hippocampus-mediated cognitive function and hippocampal cell loss.[3] In humans, acute adminstration or long-term exposure to corticosteroids results in attention and memory deficits[12] and may be associated with depressive and psychotic symptomatology.[13] Depressive episodes are associated with similar difficulties with attention, and verbal and declarative memory.[12]

Determinants of HPA Axis Regulation: Heritability

So if HPA axis hyperactivity is a risk factor for depression, what influences the activity of this stress axis? As with major depression itself, there are both biological and environmental determinants of HPA axis function. Animal studies using genetically engineered mouse models or rat strains bred for HPA axis hyperactivity or hyporeactivity have elucidated a number of genetic mechanisms impacting HPA axis function.[14,15] Moreover, rat lines bred for anxiety-related behaviors suggest a link between high inborn emotionality and HPA system stress responsivity.[16] Similarly, studies of non-human primates have demonstrated genetic influences on both basal cortisol levels and individual differences in vulnerability to emotional distress.[17]

HPA axis function has also been shown to be heritable in humans. Twin studies have shown a genetic impact on features of the 24-hour cortisol profile.[18,19] Several genes, including variants of the glucocorticoid receptor and the angiotensin converting enzyme gene, influence sensitivity to HPA hormones.[20,21]

Determinants of HPA Dysfunction: Early-Life Adversity

Genes are not the only influences that shape an individual's neuroendocrine response to stress; environmental factors are also critical determinants of HPA axis reactivity. Stressors, such as emotional deprivation and abuse, can have profound and long-lasting effects on the activity of this neuroendocrine system.

Animal Studies

Exposure to stressors during critical periods of early brain development can trigger lasting CRH and HPA changes in laboratory animals. In rodents, the separation of pups from their mothers results in an irregular pattern of HPA activity. Animals that experience early maternal deprivation show baseline and stress-induced hyperactivity of CRH and the HPA axis in adulthood. These effects are associated with behavioral indices of distress reminiscent of some of the cardinal features of major depression in humans, including psychomotor agitation,

hypervigilance or anxiety, and diminished sleep, appetite, and sexual function. Moreover, there is some evidence that the neuroendocrine effects are reversed when animals are given antidepressants.[3,7]

An experimental stress model has extended this finding to non-human primates. In this paradigm, primate mothers have intermittent and variable difficulty in obtaining food (while their infants consistently have food available to them).[22] This paradigm leads to alterations in normal maternal-infant interaction, in which mothers become anxious, inconsistently attentive, and sometimes neglectful toward their young. As adults, the offspring of these mothers exhibit behavioral signs seen in humans with anxiety and depression and have chronically elevated CSF CRH concentrations.[7]

Human Studies

Early adverse experiences, such as childhood physical and sexual abuse, are well-documented risk factors for the development of depression and **posttraumatic stress disorder (PTSD)**. It is also common for negative life events to precede the onset of affective episodes in both children and adults. The presence of such stressors predicts depression severity and the probability of relapse.[7,23]

How then do the major stressful events one experiences relate to the way in which the stress response system operates?

Much of the literature on HPA axis function in humans has focused on differences between depressed and non-depressed subjects. More recently, investigators have started to explore how HPA axis functioning is affected by childhood adversity. Our group examined the relationship between perceived childhood stress and levels of CSF CRH in depressed and healthy adults. We found perceived early-life stress, but not depression, to be a significant positive predictor of CSF CRH.[24] Two investigations have shown associations between loss of a parent in childhood and HPA axis hyperactivity in adulthood.[25,26] Other studies have documented increased cortisol and/or ACTH responses in individuals with a history of childhood abuse who have undergone a neuroendocrine challenge test (administration of CRH or the Dex/CRH test) or a psychosocial stress test.[7,23]

We found cortisol hyporeactivity in a group of healthy adults with a history of childhood maltreatment;[27] this is consistent with some previous work in children and adults. Current research in this area is directed at elucidating the determinants of the pattern of the HPA axis abnormality. Likely factors include genetic vulnerabilities as well as the nature and developmental timing of the stress exposure.

Diathesis-stress models of psychiatric disorders posit that stressors have an especially deleterious effect only in individuals who are biologically vulnerable for a particular disorder.

In addition to objective characteristics of a stressor, an individual's experience of "stress" is influenced by subjective qualities inherent to the individual, such as temperament or personality. Neuroticism, which can be broadly characterized as the tendency to experience negative affect, has been strongly associated with the development of major depression and was recently found to be highly correlated with increased cortisol concentrations in the Dex/CRH test.[28] Similarly, we found that personality traits associated with inhibition and neuroticism are associated with increased cortisol response to the Dex/CRH test as well as a psychosocial stress test.[29]

Risk for Depression: Gene-Environment Interactions

Although the association of childhood and adult stressors with major depression is robust, not everyone who experiences a significant loss or has a history of childhood maltreatment develops major depression. Diathesis-stress models of psychiatric disorders posit that stressors have an especially deleterious effect only in individuals who are biologically vulnerable for a particular disorder. Thus, both the biological and environmental risk factors may be necessary for the disorder to develop. A recent landmark study by Caspi and colleagues demonstrated just such an interaction. In a large prospective epidemiologic study, these investigators examined interactions of childhood or adult stress with a functional polymorphism in the serotonin transporter gene (5-HTTLPR). Subjects with at least one copy of the short allele of the serotonin transporter gene were more likely to become depressed in the wake of childhood or adult stressful events than those with one or two long alleles.[30] This interaction has now been replicated several times. Most recently, Kaufman and colleagues replicated this finding in children, and in addition, found that another gene, **brain-derived neurotrophic factor (BDNF)**, mediated this effect in a 3-way interaction. Finally, this interaction of genotype and maltreatment in producing risk for depression was ameliorated in children who had high levels of social support.[31]

Summary and Directions for Research

The clinical and preclinical findings reviewed show that HPA axis dysregulation frequently occurs during an episode of depression and may also represent an endophenotype of depression. Such neuroendocrine dysfunction may result from effects of early-life stress, perhaps particularly in individuals who are genetically at-risk for depression and neurobiological or psychological sensitivity to stress. Specific genes that interact with early-life and adult stress and may lead to depression have been identified, and a recent study showed that social supports may prevent this effect. Further research is necessary to elucidate the role of HPA axis function in gene-environmental interactions. Genes that confer neuroendocrine or psychological sensitivity to stress may be involved in the pathogenesis of major depression.

The Mood Disorders Research Program at Butler Hospital is conducting several studies that aim to further delineate the biological and environmental influences on stress sensitivity and risk for major depression. These include: 1) an

investigation of individuals with risk factors for HPA axis dysfunction and mood disorders, including a family history of depression and significant early-life stress; 2) a study of HPA axis hyperactivity as an identifiable endophenotype for major depression; 3) investigations of pharmacological interventions for healthy adults with this endophenotype who may be at-risk for major depression; and 4) studies of candidate genes involved in HPA axis function and psychiatric disorders, with particular focus on gene-environment interactions. The goal of these projects is to provide more precise information regarding the pathogenesis of depression and abnormal stress-reactivity that could guide treatment and prevention efforts for depression and other stress-related illnesses. In the long term, medications and psychotherapeutic interventions that target neuroendocrine function or otherwise mitigate stress responsivity could serve a role in the prevention of depressive illness in individuals with a family history of depression, stress-responsive genotypes, and those who have experienced early-life adversity.

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Health By Numbers
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Hospitalizations for Behavioral Illness in Rhode Island

Karen A. Williams, MPH, and Jay S. Buechner, PhD

The rates of utilization of hospital inpatient care for behavioral illness vary greatly with location within the United States, more so than is true for physical illness. The reasons for this variation are not fully understood, but among the hypotheses are differences in health coverage for behavioral health care, differences in the availability of providers for both inpatient and outpatient care, and differences in treatment-seeking patterns among those with behavioral health problems, as well as differences in underlying prevalence.

Although it is not possible to determine the relative contributions of these and other factors using hospital inpatient data alone, analysis of those data can be an informative first step in examining how the health care system responds to behavioral health issues. The authors have recently completed such an in-depth analysis [*Hospitalizations for Behavioral Illness, Rhode Island 2003* (in press)]; this report presents selected findings from that study.

METHODS

Acute-care hospitals in Rhode Island report patient-level data for every inpatient discharged, as required by licensure regulations. The data reported include up to eleven diagnoses made during the inpatient stay, coded to the **International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM)**.[1] This analysis includes discharges with a principal or additional diagnosis of mental disorders, defined as ICD-9-CM diagnosis codes 290-319, for comparison to national and regional rates only. All other data include discharges with a behavioral illness diagnosis defined according to the detailed diagnostic categories of the **Clinical Classifications Software**[2] **(CCS)** system, modified by the exclusion of two ICD-9-CM codes for tobacco use (305.1 and V15.82). To avoid double counting, discharges with both a principal and additional diagnosis of behavioral illness, there is no overlap between the two groups of discharges as defined for this analysis.

Rhode Island population-based rates were calculated using discharges of Rhode Island residents from Rhode Island and Massachusetts hospitals. Rates for the US and Northeast region were taken from national publications.[3] (Note: The rate for the Northeast in 2001 was calculated by the authors from the published data.)

RESULTS

In 2003, there were 12,726 inpatient discharges from non-Federal acute-care hospitals in Rhode Island with a behavioral illness principal diagnosis, representing 10.0% of all discharges (126,784 excluding hospital newborns) from these facilities. While accounting for 10% of patients, discharges with principal diagnoses of behavioral illness comprise 16.2% of the total days of care. There were an additional 23,844 discharges with diagnoses of behavioral illness secondary to a principal diagnosis of physical illness or injury. Together, the total of 36,570 discharges comprise 28.8% of all discharges.

Table 1.
Number of discharges, average length of stay and average charge per discharge for discharges with a behavioral illness principal diagnosis, by diagnostic category, Rhode Island, 2003

Diagnostic Category	Behavioral Illness Principal Diagnosis		
	Number of Discharges	Average Length of Stay (Days)	Average Charge per Discharge
Mental retardation	4	*	*
Alcohol-related mental disorders	1,294	4.3	$7,102
Substance-related mental disorders	1,530	4.8	$7,167
Senility and organic mental disorders	1,048	11.6	$19,164
Affective disorders	5,227	9.1	$13,313
Schizophrenia and related disorders	1,129	13.8	$21,645
Other psychosis	387	8.2	$12,860
Anxiety, somatoform, disassociative, and personality disorders	625	14.6	$19,368
Preadult disorders	91	11.8	$17,081
Other mental conditions	1,390	7.5	$9,636
Personal history of mental disorder, mental and behavioral problems, observation and screening for mental condition	1	*	*
Total	12,276	8.8	$13,073

*Data suppressed due to small number of observations.

Over half (56.7%) of discharges with a principal diagnosis of behavioral illness are treated at one of the six acute-care general hospitals offering behavioral health services. (Figure 1) The two psychiatric hospitals also provide a large proportion (39.7%) of the care to these patients. Only a small proportion (3.6%) of discharges with a principal diagnosis of behavioral illness are seen at acute-care general hospitals without behavioral health services. Among acute-care general hospitals with behavioral health services, the proportion of discharges with any behavioral illness diagnosis falls in the range 24%-46% compared to 8%-29% for hospitals without these services.

Over 2000-2002, the rate of discharges per 10,000 with a principal

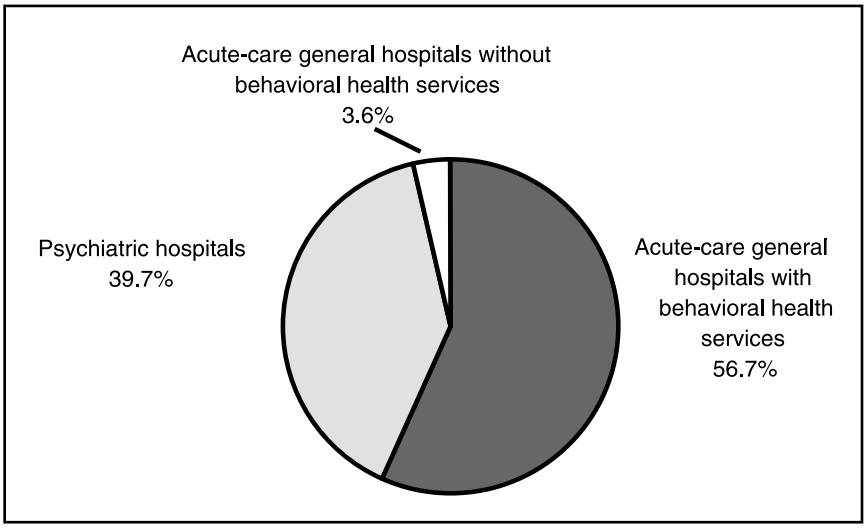

Figure 1. Hospital discharges with principal diagnosis of behavioral illness, by hospital type, Rhode Island, 2003

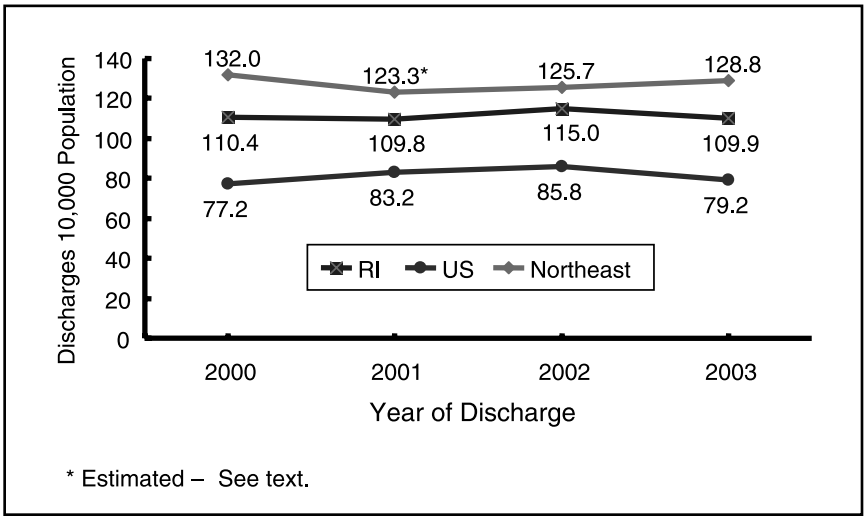

Figure 2. Discharges per 10,000 population for principal diagnosis of mental disorders, Rhode Island, Northeast and United States, 2000-2003

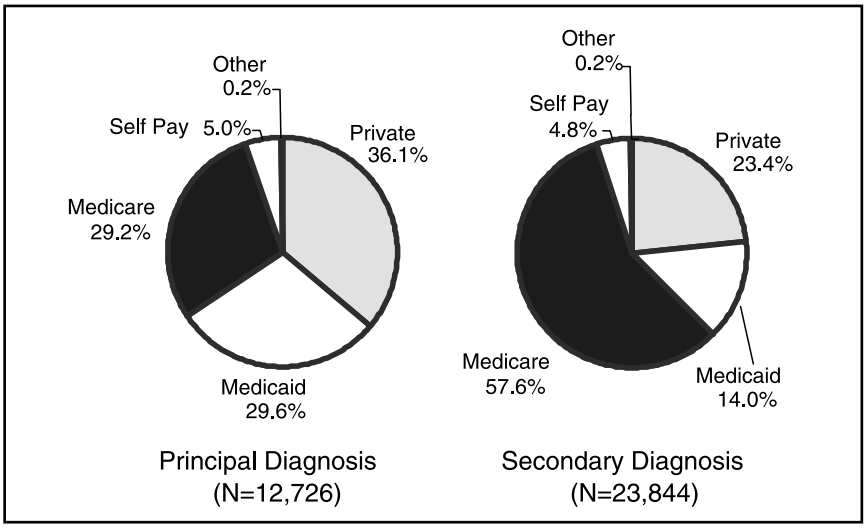

Figure 3. Hospital discharges with behavioral illness diagnosis, by expected source of payment and position of diagnosis, Rhode Island, 2003

diagnosis of mental disorders increased in both Rhode Island and the US.[3,4] (Figure 2) Rates for Rhode Island were substantially higher than for the US but lower than rates for the Northeast. In 2003, the Rhode Island rate, 109.9 per 10,000 population, was 38.8% higher than the national rate (79.2), but 14.7% lower than the rate for the region (128.8).

The most common expected source of payment for patients with a principal diagnosis of behavioral illness is private insurance, including Blue Cross, commercial plans and CHAMPUS, comprising 36.1% of these discharges. (Figure 3) Medicaid (including RIte Care) and Medicare also account for a large proportion of these discharges, 29.6% and 29.2%, respectively. Self-pay patients, presumably uninsured, comprise 5.0% of discharges with a principal diagnosis of behavioral illness, higher than the self-pay rate for all discharges (3.2%).

Examination of the expected source of payment for patients where the diagnosis of behavioral illness is only secondary to a principal diagnosis of physical illness or injury shows that Medicare accounts for more than half of these discharges (57.6%), followed by private insurance with 23.4%. (Figure 3) For comparison, among all discharges in 2003 Medicare accounts for 45.2%, Medicaid 15.6% and private insurance 35.4%.

Among patients with a principal diagnosis of behavioral illness, the average length of stay is higher than for all discharges (8.8 days vs. 5.4 days), and the average total charge per discharge is lower ($13,073 vs. $17,576). Within the group of patients with a behavioral health principal diagnosis, affective disorders is the most commonly reported diagnostic category, followed by substance-related mental disorders, other mental disorders and alcohol-related disorders. (Table 1) The longest lengths of stay and highest average charges occur in the diagnostic categories of anxiety, somatoform, disassociative, and personality disorders (14.6 days, $19,368) and schizophrenia and related disorders (13.8 days, $21,645). Alcohol- and substance-related mental disorders have the shortest average lengths of stay (4.3 days and 4.8 days, respectively) and lowest average charges ($7,102 and $7,167).

Discussion

At hospitals in Rhode Island, patients with behavioral illness diagnoses comprise 29% of total inpatient discharges and 36% of total inpatient days of care. The large majority of these patients, including nearly all those who have a behavioral illness diagnosis secondary to a principal diagnosis of physical illness or injury, are treated at acute-care general hospitals.

These findings identify an issue for further explication—the provision of behavioral health services in acute-care general inpatient settings. The sheer volume of cases and the frequent existence of serious co-morbidity together form a potential challenge for our state's healthcare system, particularly to ensure adequate behavioral healthcare providers, available services and continuity of care for these patients.

Another question is raised by the finding that the distribution of the expected source of payment for patients with a behavioral illness diagnosis differs from the distribution seen for all discharges. This result may reflect differing access to care across payers. This possibility also deserves further analysis, perhaps involving health plan data on behavioral health care in outpatient settings as well as inpatient care.

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Promoting Specialty Care for Lyme Disease: Lessons Learned

Carol Hall-Walker, MPA, Carla Lundquist, and Helen McCarthy PhD

LYME DISEASE (LD) is currently the most frequently reported vector-borne illness in the United States. Lyme Disease is endemic in Rhode Island, (Figure 1) and it is a significant cause of morbidity. From 1994 through 2003 the **Rhode Island Department of Health (HEALTH)** reported a total of 5,900 LD cases to the **Centers for Disease Control and Prevention (CDC).** In 2003, the last year for which data are available, Rhode Island ranked number one for the reported number of cases of LD per 100,000 population.[1]

In November 2004, a clinic specializing in the evaluation, diagnosis, and treatment of LD opened at **Rhode Island Hospital (RIH).** The LD Clinic ("the Clinic") accepts adult patients only, and they must be referred from other health care providers. (Pediatric LD patients are seen in another setting.)

In the spring of 2005, HEALTH and RIH collaborated to conduct an educational campaign to introduce and promote the services of the Clinic among rural primary care physicians. (LD is more common in rural settings than in urban settings.)[2]

The goals of the intervention were threefold: 1/ Inform primary care physicians serving rural communities about the Clinic and its services; 2/ Increase diagnosis and treatment of LD by encouraging referrals to the Clinic; 3/ Distribute easy-to-use patient education materials.

INTERVENTION DESIGN

Information about the Clinic was posted to HEALTH's web site and RIH's web site in November, 2004, just after the Clinic first opened.

In May 2005, anticipating increased LD activity in the summer months, a mailing was sent to 375 internists, family practitioners, pediatricians, and Ob/Gyns serving non-metropolitan areas of the State. The mailing included a letter describing the Clinic and an up-coming LD grand rounds, an 8.5" x 11" color poster on the Clinic, and patient referral cards.

The mailing was repeated in October 2005, following publication of an LD article in a statewide newspaper. The second mailing contained new information about a *pediatric* LD service at RIH. Posters and referral cards had been modified to include the telephone number of RIH's "Health Connection," a phone triage service. A newly developed flyer with "Frequently Asked Questions about LD" was also included in the mailing.

Figure 1

Reported Cases of Lyme Disease – United States, 2004

1 dot placed randomly within county of residence for each reported case.

EVALUATION

To assess the success of the two mailings, the Health Connection logged calls to the Clinic, and staff of the Clinic assessed the appropriateness of the inquiries. Also, the 375 physicians who had been sent the mailings were sent a short mail-back evaluation.

RESULTS

Of the 375 surveys sent, 135 responses were returned. Eighty-eight percent of the respondents remembered the mailings, and 83% reported increased awareness as a result. About half of those who received the poster displayed it in a patient area. Almost a third of the respondents who diagnosed an LD patient after the first mailing made a referral to the Clinic. Additional materials targeting children, teens, and pregnant women were suggested.

Respondents were also asked how they prefer to be notified about new patient education materials from HEALTH, and what formats they found most useful for patient education. Most (76%) prefer to be notified through mailings, 15% prefer faxes, and 6% prefer on-line information. The vast majority (82%) prefers ready-to-use printed materials, 11% prefer on-line resources, and 7% prefer CD ROMs.

The Clinic noted an increase in telephone inquiries after the first mailing, but was unable to assess the relative contribution of the mailing to this phenomenon. Referrals from providers accounted for about two-thirds of the calls. Requests for second opinions or for information accounted for the remaining third.

DISCUSSION

Physicians practicing in rural areas were targeted to receive information on a new Clinic and LD information for patients. Most proved to be comfortable treating LD, and did not use the new referral service, but a third did. About half used LD posters in their offices.

In future, we may target physicians practicing in metropolitan areas where the lower incidence of LD may increase the desirability of a Clinic.

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Materials included in the mailing and the evaluation form can be viewed at: http://www.health.ri.gov/disease/communicable/lyme/rihclinic-resources.php

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The Case For Consistent Assignment In the Nursing Home Setting

David Farrell, MSW, Barbara Frank, MPA, Cathie Brady, MA, Marguerite McLaughlin, MA, Ann Gray

Nursing homes in Rhode Island and throughout the nation are increasingly assigning nurses and nursing assistants to care regularly for the same residents, a process called "consistent assignment." As a physician, you may have noticed an improvement in the level of detail and accuracy of information on your nursing home patients. If you are not aware of this scheduling process, ask the nursing home whether your patients receive care from the same nurses and nursing assistants on a consistent basis. If not, you may want to bring this article to the nursing administrator's attention. A group of 254 nursing homes recently completed a one-year pilot study led by Quality Partners of Rhode Island called **Improving Nursing Home Culture (INHC).** Many of the participants achieved significant improvement in quality and retention, identifying consistent assignment as an essential part of their success. The results of this pilot study confirm the findings of many in-depth research studies on the importance of consistent assignment.

In August of 2005, the national network of **Quality Improvement Organizations (QIOs)** began the continuation of the **Nursing Home Quality Initiative (NHQI).** QIOs have been at the forefront of the movement to promote wider adoption of proven, evidence-based quality improvement approaches in nursing homes since the launch of NHQI by the **Centers for Medicare & Medicare Services (CMS)** in 2002. Today, NHQI is widely recognized as a turning point for nursing home quality.

While nursing homes working with QIOs over the past three years have made progress on quality of care measures such as the reduction of pressure ulcers, nursing home staff turnover and high staff vacancy rates are significant problems. The American Health Care Association estimates that there are over 100,000 vacant full-time nursing positions (RNs, LPNs, CNAs) and an average turnover rate of more than 70% in our nation's nursing homes. Turnover leads to staff instability and vacant shifts, which result in rushed, de-personalized care.[1] Providers with severe staffing problems are unable to focus on quality improvement, until they can stabilize their staffing.

To address this concern, Quality Partners and the Colorado Foundation for Medical Care recently coordinated the INHC pilot study to explore strategies for improving the nursing home culture. Nursing homes worked with their local QIO to shift from institutionally driven care to person-directed care, and found that they needed to establish consistent assignments to establish meaningful relationships.

Consistent assignment (sometimes called primary or permanent assignment) is defined as the same caregivers (RNs, LPNs, CNAs) caring for the same residents (85% of their shifts) every time they are on duty. Experts estimate that 90% of nursing homes have policies that require staff to rotate their assignments. However, a thorough review of the literature strongly supports the practice of consistent assignment over rotating assignment.[1-13]

Based on these results, QIOs are now adopting this holistic approach with more than 2,500 volunteer nursing homes.

"*Every system is perfectly designed to achieve the results it gets*" is a key tenet of quality improvement. To alter outcomes, we need to examine the root causes of our current outcomes, and look at the systems that produce them. Quality Partners of Rhode Island began to address one root-cause of low staff morale and high rate of turnover by changing a longstanding workplace system, the practice of rotating staff assignment. Long-term nursing has inherent rewards for people attracted to the care of others. Yet management systems, such as rotating assignment, can interfere with, rather than support, the caring connection that draws people to this work. Consistent staff assignment builds on the intrinsic motivation of the staff—the opportunity to form and sustain close relationships with the residents. The system of ***rotating staff assignment*** severs relationships from forming, and inhibits the ability of staff to recognize nascent problems.

Relationships are at the heart of good work environments—relationships with co-workers, across departments, with supervisors, with the organization, and, most importantly in the case of long-term care, with residents and their families. The National Citizens Coalition for Nursing Home Reform has confirmed that residents and their families value the quality of the relationships they have with the frontline caregivers more highly than the quality of the medical care and the quality of the food. People work in long-term care and stay in the field because they care about their work, the people they care for, and the people they work with. They want to make a difference in people's lives.

There are many reasons why leaders in the long-term care industry believe that rotating staff assignment is effective. The most common benefits center around fairness, preventing staff burnout and the need for staff to be somewhat familiar with all of the residents. In other facilities, leaders discourage strong relationships between staff and residents to help shield staff members from experiencing grief when residents die. These reasons for rotating staff assignments are ***not*** supported by research. In fact, rotating assignment actually exacerbates low staff morale leading to staff burnout, call-outs, quitting and overall instability.[1]

Many research articles support the practice of consistent assignment over rotating assignment.[1-13] Bowers interviewed CNAs, who felt that relationships with residents undergirded

"good care giving."[2] Burgio compared two nursing homes with permanent assignments to two with rotating assignments. Residents in the former received higher ratings for personal appearance and hygiene; aides in the former reported higher job satisfaction.[3] Campbell found that one year after implementation of consistent staffing, the turnover rate was reduced by 29%.[4] Goldman found that primary nursing care assignment left residents feeling more comfortable, and staff more satisfied.[5]

No research-based articles take the opposite stance.

The following reasons support the adoption of consistent assignment:

- There are strong links between the quality of nursing home employee's work life, resident's quality of life and clinical outcomes of care.
- Frontline staff and residents flourish when facility policies support a consistent caring relationship.
- Relationships are the cornerstone of individualized, person-directed care.
- Residents who are cared for by the same staff members come to see the people who care for them as "family."
- Staff who care for the same residents form a relationship and get satisfaction from the bonds with the residents.
- Staff who care for the same people daily become familiar with their needs and desires in an entirely different way—and their work is easier because they need not spend extra time getting to know what the resident prefers.
- Relationships form over time—we do not form relationships with people we infrequently see.
- When staff routinely work together, they can problem-solve to re-organize daily living in their care area.
- When staff care for the same residents every day they are less likely to "call out." As one CNA recently said, "I don't call out now, because my residents would miss me."

In summary, consistent assignment is the prerequisite for person-directed care. The system of consistent assignment, backed by research-based evidence, is a first step toward a more stable workforce, improved clinical care and enhanced quality of life for the residents. The system of rotating staff assignment is obsolete.

See the change idea sheet on Consistent Assignment at http://www. riqualitypartners.org/nursing_homes/wfr_train_3.php

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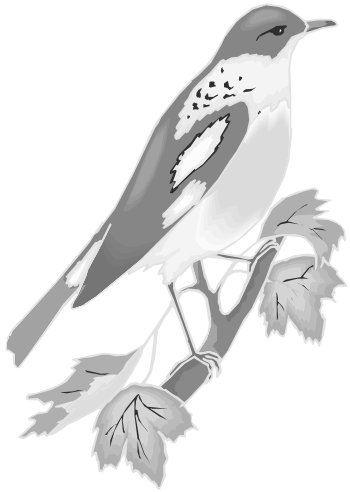

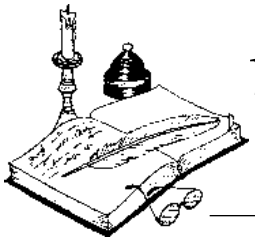

Physician's Lexicon

A Chestful of Words

HEART DISEASE, and particularly ischemic or occlusive cardiovascular disease, is the dominant medical disability of Western civilization. An expanding vocabulary of technical words is needed to define and describe the many clinical and pathophysiological featuress of these cardiac disorders.

Angina pectoris, for example, represents a compatible etymological marriage between angina, of Greek origin, and pectoris, of Latin derivation. *Angina,* in Greek, means to throttle or strangle [and is cognate with the English word, anger, but is not related to the Greek root, *angio-* meaning vessel as in words such as angiography or telangiectasia. Angina had also meant inflammatory disease of the throat, often called quinsy, and is associated with a sense of strangulation. *Pectoris*, in Latin means something pertaining to the chest. [A pectoral was an ornate chest plate worn by ancient priests.]

The word, occlude, is from the Latin, the prefix, *ob-* , meaning against; and the root *claudere,* meaning to shut off as in the clinical term claudication.]

Syncope is from the Latin, *syn-* , meaning together with; and *–cope*, meaning to cut off or sever, thus forming such English words as capon [a castrated rooster], comma [a hemisected punctuation mark, period] and kopek [a small Russian coin showing a tsar holding a lance.]

Palpitation is from the Latin, *palpitare*, meaning to tremble or move quickly. And thus the palpebra are the eyelids, so-called because of their capacity to flutter. The word, psalm, is also ultimately derived from *palpitare*, originally meaning a twitching of the musical instrument, the harp. Palpable comes from the Latin, *palpare*, meaning to touch softly. Paroxysm is from the Greek, *para-*, meaning beside or next to, and the root, *-oxysm* , from the Greek meaning to provoke or irritate. The irritant, oxalic acid, is a derived from the plant *Oxalis*.

Orthopnea is from the Greek prefix, *ortho-*, meaning straight, true or regular [as in words such as orthodontia, orthagonal and orthodox]; and *pnea* meaning to breathe, generating such English words as pneumatic and pneumonia.

Tachcardia/bradycardia are derived from Greek prefixes, *tachy-* meaning swift [as in tachygraph]; and *brady-* , meaning short or slow. The latter is cognate with the Latin, *brev-* .

Edema, formerly spelled, oedema, is from the Greek meaning swelling. Oedipus, the legendary wayward son of Laius and Jocarta of Thebes, was given his name, as an infant, because of the swelling of his feet.

– STANLEY M. ARONSON, MD

RHODE ISLAND DEPARTMENT OF HEALTH
DAVID GIFFORD, MD, MPH
DIRECTOR OF HEALTH

VITAL STATISTICS

EDITED BY ROBERTA A. CHEVOYA, STATE REGISTRAR

Rhode Island Monthly Vital Statistics Report Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	May 2005	12 Months Ending with May 2005		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	241	3,034	283.6	4,713.0
Malignant Neoplasms	184	2,422	226.4	6,265.0
Cerebrovascular Diseases	36	506	47.3	757.5
Injuries (Accidents/Suicide/Homicde)	39	430	40.2	6,674.0
COPD	38	524	49.0	455.0

Vital Events	Reporting Period		
	November 2005	12 Months Ending with November 2005	
	Number	Number	Rates
Live Births	1,063	13,501	12.6*
Deaths	838	10,173	9.5*
Infant Deaths	(7)	(96)	7.1#
Neonatal Deaths	(5)	(80)	5.9#
Marriages	432	7,510	7.0*
Divorces	277	3,239	3.0*
Induced Terminations	368	5,341	395.6#
Spontaneous Fetal Deaths	59	1,041	77.1#
Under 20 weeks gestation	(52)	(965)	71.5#
20+ weeks gestation	(7)	(76)	5.6#

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 1,069,725

(c) Years of Potential Life Lost (YPLL)

Note: *Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.*

* Rates per 1,000 estimated population
\# Rates per 1,000 live births

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Ninety Years Ago, May 1916

An Editorial, "Accuracy in Lab Work," recounted the following error. A Providence physician had sent smears from the inflamed eyes of a patient to the state Health Laboratory, which reported the smears positive for gonococcus. The "clinical manifestations" of the eyes did not jibe with the diagnosis, so the physician sent another smear to the lab, which made the same diagnosis. The physician pursued the finding. "Inquiry revealed that the report was based upon simple methylene blue staining reaction without being checked up by the differentiating test of decolorizing by Gram's method." The Editorial lamented not only the "inaccurate vital statistics," but the "mental anguish" for patients who believed, mistakenly, that they had a venereal disease.

Inspired by a speaker at the Hospital Club dinner, a second Editorial discussed "Graded Fees." Repeating the Ps of billing, "patients' plethoric pulse," the Editorial noted that because medicine is not a mercantile pursuit, graded fees might be acceptable.

George Blumer, MD, contributed "The Importance of Anaphylaxis in Clinical Medicine," stressing this "phase in the process of immunization."

F.E. Webb, MD, in "The Intraspinal Treatment of General Paresis and Tabes Dorsalis with Report of Cases," tabulated statistics. Of 20 cases of paresis treated by the intraspinal method, 10 went into remission and 2 improved. Of 6 cases of tabes, 4 improved. Injections ranged from 12 to 32.

V.L. Raia, MD, discussed "Primary Sarcoma of the Sclerocorneal Juncture Treated with Jequity [seeds of *Abrus Precatorius*]." The treatment was "either [injected] as fluid into parenchyma of the tumor or applied as gelatinous discs on its surface." The author said that this was the first report in the medical literature of a primary sarcoma "completely cured with this remedy." The patient was a 40 year-old tailor.

Fifty Years Ago, May 1956

Maurice L. Silver, in "Intracranial Surgery for Hemiplegia and Convulsions,"drew on the experience of Penfield at Montreal Neurological Institute and Walker at Johns Hopkins. Success "depends largely upon the surgeon's ability to localize the cerebral epileptogenic focus by electrical recording from the brain *during surgery*." A 14 year old girl and a 27 year old woman were treated successfully at the Miriam Hospital.

A.A. Savastano discussed "Coneplastic Surgery." He used residual muscular power in a stump to activate an artificial hand in a 49 year-old construction worker.

Warren Francis, MD, and Paul T. Welch, MD, discussed the 40% mortality rate for patients with peptic ulcers treated at Rhode Island Hospital since 1954, in "Massive Hemorrhage from Peptic Ulcer." The authors attributed the rate to the patient's age and other diseases. They stressed the need "to convince our medical confreres that the recurrent ulcer is a surgical problem and that, in these individuals, surgery alone can eliminate the devastating complications of hemorrhage, obstruction and perforation."

Twenty-Five Years, May 1981

This issue, devoted to the graduating medical school class, included 8 student essays; e.g.,: "On Medicine, Graduation and Patriotism, by Essie Nash, who commented on the time spent in a National Health Service Corps clinic in Pawtucket; and "To Code or Not to Code," by David B. Nash.

Horace F. Martin, MD, had delivered one of the "Medicine, Science and Humanism" talks at the Medical School commencement convocation in 1981. The Journal reprinted his talk. He urged: "Let us have caring physicians who are knowledgeable, current, effective and responsible."

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