Cover: *"Waterplace Park,"* pen-and-ink draging by Anthony Demings. The artist is artist-owner of the Brooklyn Coffee and Tea House in Providence, where his paintings are on display.

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COMMENTARIES

Boston Migraines

In the recent past, I had written several columns with the theme of humility. Stan Aronson, MD, who was editor of this journal before me, and my own personal editor and advisor, thinks I overdo it. He told me the story of the sexton, a lower level religious figure at the temple, who listened intently to the rabbi's High Holy day sermon on his essential unworthiness. After a while the sexton, overcome with religious spirit, wailed and tore his clothes, denouncing himself as a useless, despicable worm. The rabbi, with great disdain, turns to the cantor and remarked, "Look who thinks he's worthless."

Humility, however, is a worthy topic for anyone to explore, perhaps more so for doctors who command a unique relationship with their patients. At the entrance to my medical school, carved in stone, is the phrase from Maimonides: "For of the most high come healing." My neurology residency (same place) was, in my judgment, the best training program in the country; at least everyone at the program thought so. We acknowledged, reluctantly, that a handful of programs might be as good, but none better. Queen's Square, in London, we thought, possibly was better. Our chair, a world famous neurologist, of course, was humble. It wasn't until my final year of residency that I realized when he turned to me at a case conference to tell me he was stumped and wanted my opinion, that he actually was teaching and that he really didn't want my opinion. He knew that if he didn't know, neither did I, but he cared that I should think independently. If I ventured an opinion he loved it, especially if it was different but defensible. In addition to teaching neurology he taught logic and honesty. Because he was famous he would see the cases that no one could diagnose. Patients might see him before or after they went to other prestigious centers. It was not unusual for him to end a conference by saying he didn't know. He was honest and honesty forces humility.

When I moved to Rhode Island twenty years ago there was a handful of neurologists here and no separate department of clinical neurology at Brown University. There were no subspecialists and no local authorities of national stature. So, of course, patients went to Boston for second opinions. The first thing I learned in this process was that virtually all episodic neurological events that were not seizures or TIAs were called migraines by the Boston consultants. Patients with bizarre spells but no headache had "acephalgic migraine" even if they'd never had a "cephalgic" migraine, even if they were at an unlikely age. I was surprised by this because these would not have been classified as migraines in New York. In New York the spells would have remained mysterious and undiagnosed, so I began to think of these spells as "Boston migraines."

An experience of a different kind then occurred. I followed a forty-year-old woman with a complex neurobehavioral picture. She carried a diagnosis of lupus erythematosis although her very extensive records showed that her lab tests were normal and that rheumatologists were never able (in the extant documents) to confirm that diagnosis. She had allegedly suffered a stroke with persistent right-sided weakness but one doctor's note clearly described seeing the patient, when she thought she was unobserved, stand up from her wheelchair and use both hands without difficulty to steal a cigarette from a pack he had left out. She also seemed to have "non-epileptic" (formerly called pseudo) seizures. She went to see a famous expert in Boston for whom I carefully summarized a large body of material and clearly stated the questions that were most pertinent to the staff at the chronic care hospital in which she resided. Imagine my surprise when her lengthy and erudite evaluation came back noting that this patient fit into a pattern he had been the first to recognize of left-handed people with premature graying having increased risks of immune disorders such as lupus. Not a word on her negative lupus studies. No mention of her weakness not being physiologic. No suggestions on diagnosing epileptic versus non-epileptic seizures. From my vantage point she didn't have lupus, was not prematurely gray and I was unsure that she ever was born left-handed rather than acquired left handedness after becoming weak on the right.

That sat poorly with me and when a second complicated patient saw the same neurologist and he again ignored the true history in favor of one more in keeping with a new ground-breaking syndrome, I stopped even thinking about this eminent neurologist's theories. Facts are facts and fiction does not support a theory.

This was one rotten apple. Not all neurologists in Boston are charlatans. Very few are, I think, now that I know many of them, but many of them share a common problem in being unable to say, "I don't know." Now it should be noted that patients like to have a diagnosis. There is a certain reassurance that comes from knowing the name of your illness. It can be addressed as another thing outside the body. "I have Parkinson's disease." "I have Alzheimer's disease." "I have Gershmann-Straussler Syndrome." Something to hang a hat on; something to look up on the Internet.

I learned over many years that patients who cannot be diagnosed in Rhode Island are routinely diagnosed in Boston. The diagnosis may not be correct, but the patient gets a name, an identifier and feels relieved. I call

these, "Boston diagnoses." When I'm stumped, which happens too often, I always tell the patient and when I think the patient can benefit from another opinion I tell him so, suggesting whom to see. Mostly I'm fairly confident that if I don't know the answer no one else will either and then I don't suggest another opinion. When a referring doctor tells me the patient is getting another opinion in Boston I generally tell the doctor, "He'll get one too. It may not be right but he'll get a diagnosis."

Humility and honesty go hand in hand.

– Joseph H. Friedman, MD

The Physician Who Ruled France

Of the many charismatic leaders who governed France during the tumultuous decades of the 20th Century, none was more eloquent, more dynamic, more influential, more revered than a physician, born in Mouilleron-en-Pareds, named Georges Clemenceau.

Clemenceau was born in 1841 when France was still undecided as to whether it preferred a monarchy, an authoritarian consular dictatorship or a republic rooted in a constitutent assembly. His middle-class family was firmly, outspokenly republican in their political views; and young Clemenceau learned from early childhood to defend his opinions against those advocating hereditary rule or military governance.

Clemenceau chose medicine for his future calling. He attended the medical school in Nantes, and received his degree and license to practice medicine and surgery. But he was increasingly influenced by the writings of the British philosopher, John Stuart Mill; and rather than establish a rural practice in western France, he elected instead to translate into French Mill's essays on political governance and human rights. He finally abandoned a medical career and sailed for the United States, arriving in New York City in 1866 to confront a nation still recovering from a grievous Civil War, a fractious South undergoing a painful Reconstruction and the contentious presidency of Andrew Johnson.

Clemenceau then embarked upon a tour of the northeastern United States, writing periodic columns on the social and cultural environment of post-war America for the Paris paper, *Temps*. To augment his income as a journalist, he taught French at a girl's school in Stamford, Connecticut. He met and married an American woman; and in 1869, after three years in New England, Clemenceau returned to Paris. In his autobiography, written in 1929, the year of his death, he stated that his stay in America were the happiest years of his life.

The Paris of 1870-71 was in turmoil both from the residue of the Franco-Prussian War and the ensuing revolution bloodying the streets of the French capitol. Clemenceau, the physician and journalist, now undertook a third career. He ran for and was elected as mayor of the 18th arrondissement [Montmartre]; and in the following year he was elected to the National Assembly as representative of the Department of the Seine. France was now to experience the biting sarcasm, the mordant wit, the aggressive speeches of the erstwhile physician from western France. Within months he was engaged in fierce controversies, recriminations and even a duel. His combative behavior made many enemies, some of whom charged him with unpatriotic behavior. He was brought to trial, convicted and sentenced to a fortnight in jail.

During the next decade Clemenceau was in and out of a succession of elected posts, simultaneously writing polemic tracts - his enemies called them vile diatribes - analyzing a variety of social and economic issues. Much of his immense energies were now invested in fighting the anti-republican factions of France. In furtherance of his radical opinions, Clemenceau also founded and wrote for a number of periodicals, the most enduring of which was the famous *l'Aurore*.

By 1887 a politically redeemed Clemenceau returned to his elected position in the General Assembly, representing the far left political parties. He played an increasingly pivotal role in making and breaking successive governing ministries.

In 1893, with the writers Emile Zola and Anatole France, Clemenceau involved himself in the futile defense of a French army officer, Captain Alfred Dreyfus, convicted of treason and exiled to a tropical prison camp. By 1900 new evidence of Dreyfus's innocence emerged and Clemenceau assumed the leadership in demanding, and obtaining, a pardon for Dreyfus. Clemenceau also directed the drive leading to legislation separating church and state in France.

An increasingly conservative Clemenceau was elected to the Senate in 1903 and was finally selected for the premiership in 1906 through 1909. The beginning of World War I saw Clemenceau in the ranks of the opposition party, declaring that the military effort was inadequate and faint-hearted. By 1917 following military reverses by the French armies defending the Marne region, Clemenceau was again made premier of France. And for the next year he rallied the people of France to exert themselves more. He combined the separate British, French and American armies into one unified command under Marshal Foch. A series of victories in the trench warfare of northern France finally led the Germans to request an armistice. "War," declared Clemenceau, "is a series of catastrophes that finally results in a victory." A relentless Clemenceau demanded of the Germans total disarmament, transfer of the territories of Alsace-Lorraine and reparation. And a treaty, named Versailles, was finally agreed upon by Clemenceau, Lloyd-George and Wilson.

Two years after the cessation of the war, Clemenceau

resigned his premiership and devoted the next few years travelling in the United States, speaking in behalf of the Versailles Treaty and the League of Nations. He retired to Paris in 1927 to write his memoirs, called "In the Evening of my Thoughts." "A man's life," he wrote, " is interesting primarily when he has failed. For it is a sign that he has tried to surpass himself."

Physicians are said to avoid open political activity. Rarely do they run for office, and more rarely are they elected. In this past century there has been only a scattering of physicians in the U.S. Congress. Which is not so say that physicians never get their hands muddied by political conflict. There was, for example, Sun Yat Sen, a graduate of the medical school in Hong Kong, who assumed the presidency of Nationalist China in 1912. And then there was Che Guevara, a graduate of the medical school in Buenos Aires, who foreswore his clinical obligations to join Castro's Cuban revoluation.

Georges Clemenceau, an intensely ambitious, fiercely aggressive, unforgiving statesman, was called the Tiger of France [despite a family name that translates as merciful, mild and forebearing.] He was an outstanding journalist, twice a premier of France, co-author of the Treaty of Versailles and a patriot. A complex man, he was a major force determining the destiny of 20th Century France. He was a physician, too; but if his medical training exerted any visible role upon years of vigorous public life, it has steadfastly remained hidden to historians.

– Stanley M. Aronson, MD, MPH

The Joys of Bernard Berensoning: A Note on Journal Covers

This cover features "Waterplace Park" by Anthony Demings - the latest in five years of artist-covers.

More than 50 artists have said "yes," when I've asked: "Could we feature your work on the cover of *Medicine & Health/Rhode Island*?"

From the start, the exchange has been patently, painfully uneven: we give the artists no compensation, do not even reimburse expenses. We give ten copies of this journal, which is of minimal interest to anybody but a physician. Nevertheless, almost every artist I've approached has graciously acceded.

If the artist gets very little, journal readers get a monthly treat.

Sometimes readers see a picture that evokes the issue's theme. Serendipitously I will have found that perfect image. In Zane Sherman Jr's Providence studio, stacked among the large city-scapes, was a small sketch of a man in a wheelchair - fitting for Spinal Cord Injuries (3/00). Regina Partridge's golden apples proclaimed Nutrition (11/00). For sports topics, we used Joan Boghossian's empty sneakers (6/20), Brian Larkin's divers (2/00), George Simmons's bicyclists (9/02). Steven Boksenbaum's '59 Ford, on display at the Wickford Art Festival, seemed custom-made for Driving (12/99). Denise Baxter's figure captured the anguished uncertainty of a woman who may have a sexually transmitted disease (10/97). Paul Olson's quilters hinted at the disability of arthritis (9/99). Trinity's graphic artist Michael Guest let us use a discarded sketch of "Romeo" embracing "Juliet" for Adolescent Medicine (11/99). For Family Medicine (0/00), we made a collage of Jennifer Iwasyk's portraits. The list goes on and on.

Occasionally serendipity has needed a boost, and artists have come to the fore—again, without compensation. Cartoonist Robert Walker had done a delightful map I spotted in a magazine: would he adapt it for the graduation issue? Amazingly (maybe not amazingly in the world of artists, but amazingly in my world), he said sure, drew students (his original featured ducks) plodding their way toward residency (8/98). For obesity, I asked to see Allen Avery's sketches (he had illustrated a flyer for an off-Trinity production, and I detectived out his name and address). A sketch of people dining would do. But, he queried, why couldn't he do a cover just for the issue? Why not? So he photographed a junk food feast (11/97). I hoped that children's illustrator JW Alley might have something on hand for assisted reproduction: instead, he sketched a tree with babies-as-buds (12/97). Bloodborne pathogens was a challenge: a CME issue going to all physicians should have a theme cover. Ellen Watt drew a quartet of latex-clad fingers (7/00). Chris Pierson designed the modern take on Michelangelo for Plastic Surgery (4/01); John Teehan did the barred cage for Penal Medicine (12/00); Jason Hamel photographed the Van Wickle gates for Medical Education (8/00); Gretchen Halpert, who teaches scientific illustration, painstakingly drew a poppy for Substance Abuse (3/99). Nobody mentioned billable hours.

Most covers have featured Rhode Island. Not surprisingly, water has predominated: boats moored (Richard Harrington, 6/98), fishermen on the Barrington River (Kathleen Weber, 6/01); fish (Joan McLellan, 4/00), a beach house (Gretchen Dow Simpson, 6/99). Readers have seen landmarks: Hanging Rock (Elizabeth Goddard, 3/01), Block Island Moviehouse (Garry Bliss, 9/02), Prospect Park (Richard Benjamin, 8/01). The 7/97 cover (Munir Muhammed) showed renovation around the Westin.

The quest-for-art began pragmatically. We had waded deeply enough into the thicket of legalese to see that we could no longer use anything published anywhere, without permission—even a 15th century painting, printed in a 20th century book, is protected. And artists, as well as their heirs, often have "use" rights to their work, so that somebody lucky enough to own a Picasso probably couldn't let us feature it. The easiest, and cheapest recourse, was to stick the Table of Contents on the cover. But after years of wonderful covers, that sounded grim.

Dr. Aronson found the solution. Claudia Cockerill had done a sketch of Stanley Simon for an article. Would she let us use one of her other works on a cover? Claudia's "Market Square" graced the June 1997 issue. (Her "Learning Tree" graced the 1/99 festschrift for Dr. Aronson).

If Claudia agreed, would other artists?

I started a pilgrimage through studios and galleries and shows. When I have lost my mind (the dementia issue convinced me the loss is forthcoming), I will remember the delight of seeing Priscilla Cane's works spread out in her studio (her "Fisherman Filleting His Catch" is slotted for summer 2003), or of happening upon the perfect injury control cover at the Warren Art Festival, or discovering Michael Bryce's fanciful cherubs (his "Valentine" was the 2/01 cardiology cover). And I will marvel at this exhilerating world of kind people, who introduced me to colleagues and friends, who opened their portfolios to me. (We have more than occasionally finished our conversations over coffee.)

Bernard Berensoning has been a joy. Indeed, Dr. Friedman, the editor-in-chief, and his Memorial Hospital colleagues have gotten into this mode, when I lay out photographs for everybody to rank. The conversations with PrintSource about background and tone (their staff, plus the Heidelberg press, make the colors vibrant) take on the intensity of a Soho gallery, as everybody weighs in.

So, befitting the month of Thanksgiving, thank you to all the artists (I've mentioned only some) who said yes to this disturbingly uneven exchange.

– Joan Retsinas, PhD

Joan Retsinas, PhD, is Managing Editor of Medicine & Health/Rhode Island.

ACP-ASIM Abstracts: Introduction

Fred J. Schiffman, MD, FACP

The Annual Spring Meeting of the Rhode Island Chapter of the American College of Physicians-American Society of Internal Medicine was held at the Radisson Airport Hotel on May 2 and 3, 2002. As usual, the academic work of medical residents was at the core of the meeting.

The Selection Committee was once again overwhelmed by the large number of submitted abstracts from which we chose eight oral presentations and approximately ninety poster presentations, all written by residents, fellows and medical students.

As usual, all of the teaching hospitals in the State participated. Roger Williams Medical Center, Memorial Hospital of Rhode Island, The Miriam Hospital, the Providence VA Medical Center and Rhode Island Hospital were sources of abstracts.

Once again, the titles of the house officers' works were interesting and varied. The eight abstracts presented in oral form are included below; and they ranged from epidemiologic topics to the molecular basis of metabolic disorders with many pathophysiologic principles demonstrated and many subspecialties of internal medicine were highlighted.

The approximately ninety posters that were exhibited were similarly varied and interesting. Titles included the following: "A Case of Mistaken Identity: Prominent Eustachian Valve Masquerading as a Right Atrial Mass," "Smoldering TTP" and "A Case of Relapsing and Cholestatic Hepatitis A."

Dr. Rowan Zetterman, MD, MACP, Vice-Chairman of Medicine, Nebraska Medical Center, Professor of Medicine, University of Nebraska, our ACP/ASIM College Representative, was extremely impressed with the scholarly work performed by our residents and fellows, especially given the time demands and stresses of house staff training.

From a personal and scientific point of view the meeting was a satisfying one and was augmented by panel discussions on bioterrorism and chemical warfare and controversies involved in MRI and CT total body screening.

Our residents are already hard at work preparing for the 2003 meeting, which we look forward to with great interest.

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Missed Opportunities for HIV Screening: Patients Diagnosed with HIV in Rhode Island Hospital over a Two-Year Period

Curt G. Beckwith, MD, and Michelle A. Lally, MD, MSc

THE MIRIAM HOSPITAL & RHODE ISLNAD HOSPITAL/BROWN MEDICAL SCHOOL

Highly active antiretroviral therapy (**HAART**) has dramatically decreased the incidence of opportunistic infections in the HIV population. Despite this, significant numbers of persons at risk are still not being tested for HIV until they present to a hospital with a severe infection. We identified persons diagnosed with HIV on admission to Rhode Island Hospital from 9/1/99 - 8/31/01 through a medical records search. A chart review was performed to identify missed opportunities for HIV screening prior to this hospitalization. [The study and chart review were approved by the RIH/Miriam Hospital IRB with the caveat that patients would not be contacted based on the medical records search.] Missed opportunities included any of the following: a chronic medical condition or medication that required ongoing care of a physician or other healthcare provider, previous history of drug rehabilitation, STD clinic visit, hospitalization or ER visit within a two-year time period prior to their hospitalization. Other data examined included reporting of risk factors, presence of opportunistic infections, and initial CD4 count.

We identified 26 patients diagnosed with HIV during this two-year period. Thirteen (50%) were at least forty years old. In terms of missed opportunities for screening, 16 (62%) had some type of chronic medical condition and 12 (46%) were on prescription medications. Furthermore, 18 (69%) had been hospitalized or had visited an emergency room or walk-in clinic within two years prior to their admission. In terms of HIV risk factors, only 7 (27%) reported active or former drug use and only 3 (12%) men reported having sex with other men. Only one patient had a history of drug treatment, no patients reported a history of either STDs or attending an STD clinic. Twenty-four patients had initial CD4 count data available and 20 (83%) met the CDC definition for AIDS at the time of their diagnosis with a CD4 count $\leq$ 200. Sixteen patients (67%) had advanced AIDS with a CD4 count below 50. Thirteen (50%) had opportunistic infections diagnosed during their hospitalization. Given only ten (38%) patients identified typical "at risk" berhaviors such as IVDA and risky sexual behavior, clinical suspicion for HIV was frequently based on the presence of opportunistic infections as well as emigrating from a country where HIV is endemic.

This study demonstrates that significant numbers of patients still present to our hospital with undiagnosed HIV and often AIDS. The majority had some opportunity for screening prior to this hospitalization and half of the patients were at least forty years old. Given their advanced age and stage of HIV infection, many patients had likely been HIV positive for years. This study demonstrates that our current screening practices are inadequate. Not only do we need to be aggressive in eliciting risk factors from our patients, but we also need to consider immigrants from countries where HIV is highly prevalent to be at high risk.

Two Cases of Bradycardia Induced by Presumed Ciguatera Toxin

Maen Hussein, MD, Emily Harrison, MD, Anthony Murat, MD, Ahmad Al-Mubaslat, MD, Hussam Hamdalla, MD, Ahmad Al-Raqqad, MD, John Miskovsky, MD

INTERNAL MEDICINE PROGRAM, MEMORIAL HOSPITAL OF RHODE ISLAND

An 80 year-old husband and his 70 year-old wife presented to the emergency department with nausea, vomiting and diarrhea several hours after eating barracuda. They were treated for gastroenteritis and discharged from the emergency department. Two weeks later, the wife re-presented with persistent diarrhea, dysarthria, weakness and dizziness. On examination she was orthostatic with a low pulse (30 b/min); she also had signs of left-sided weakness with left facial droop. Her blood work was significant for elevated cardiac enzymes (CK of 130 with MB of 10.9%); her EKG showed sinus bradycardia. The patient was admitted to the coronary care unit to rule out ischemia and to monitor her heart rate. She was treated with intravenous fluid hydration, atropine to treat her bradycardia, and dopamine to elevate her blood pressure. The dopamine and the atropine were discontinued three days later after her symptoms improved. Her husband presented 4-5 hours after her presentation complaining of pruritus and generalized weakness. He was found to be bradycardic but without hypotension or neurological symptoms. His heart rate was 45 bpm and his EKG showed first-degree heart block. He was treated with famotidine for his itching but did not need atropine for his low heart rate. He was monitored in the coronary care unit for two days, then transferred to the telemetry floor and discharged the next day.

Ciguatera fish poisoning is most commonly seen in the spring and summer and results from ingestion of a neurotoxin called ciguatoxin, which is carried by coral reef fish, including barracuda. It has a short incubation period, approximately 2 to 6 hours. Symptoms are mainly gastrointestinal - nausea, vomiting, watery diarrhea, and crampy abdominal pain - which tend to appear earlier; and neuralgic - weakness, ataxia, vertigo, and more specifically for this toxin dysesthesias, paresthesias and pruritus. Patients may suffer autonomic dysfunction leading to hypotension and bradycardia. [There was no diagnostic test done in these two patients to confirm the diagnosis. It was based on the history and the clinical picture. Upon reviewing the literature, most published cases of Ciguatera fish poisoning were also diagnosed based on the clinical history and no tests were done to confirm the diagnoses in many of the published cases. Only one test (ciguacheck) is commercially available to detect the presence of the toxin.] Treatment is symptomatic: intravenous fuid hydration to replace losses caused by vomiting and diarrhea, antihistamines for pruritis, and atropine for autonomic dysfunction. Manitol and amitriptyline have roles in relieving neuromuscular symptoms, but the mechanism is unknown.

Cerebrospinal Fluid (CSF)- Administered Soluble Human Serum Albumin (HSA) Induces Interleukin (IL)-4 and IL-10 in the Cervical Lymph Nodes (CLNs) and Spleen in Balb/c Mice

Joel T. Park, MD (Assoc), Paul M. Knopf, PhD, Christine J. Harling-Berg, PhD

Rhode Island Hospital/Hasbro Chidlren's Hospital, Brown Medical School

The immune privileged status of the brain, empirically defined by prolonged survival of tissue transplants, is not, as originally proposed, a passive process resulting from anatomical isolation due to the absence of conventional lymphatics and to the presence of brain-barrier membranes. It is now believed that the privileged state is maintained by active immune regulation, and this state does not *exclude* immune responsiveness to antigen introduced into the brain. In our prior work, we have shown antigen administration into CSF induces a robust serum IgG1 response in the absence of **delayed-type hypersensitivity** (**DTH**) priming. In contrast, the subcutaneous (s.c.) route elicits a significantly smaller humoral response and primes for DTH.

Using a mouse model with normal blood-brain barrier permeability, the cytokine response was measured in the draining CLNs and spleen following antigen administration. HSA in saline was microinfused into CSF or microinjected s.c. After 7 days each mouse received a challenge immunization of s.c.-administered HSA in complete Freund's adjuvant. At 10 days post-challenge, CLNs and spleen were harvested and cultured with and without *in vitro* HSA stimulation. The ratio of a cytokine concentration with antigen and without antigen stimulation in culture gives the antigen-specific **stimulation index** (**SI**). The SI of **T helper 2** (**TH2**) cytokines (IL-4 or IL-10) were compared to the **T helper 1** (**TH1**) cytokine SI (interferon y). The ratio of the TH2 SI to the TH1 SI gives the **T Helper Index** (**THI**). The value of the THI provides a relative bias within the TH1/TH2 paradigm.

In CLNs, mice initially given HSA into CSF induce a significantly greater antigen-specific IL-10 THI vs. s.c.-treated mice. Similarly with splenocytes, the IL-4 and IL-10 THI is significantly greater in mice microinfused into CSF. This data support the induction of a TH2 immune response to antigen effluxing from the brain. These studies have significant ramification for understanding multiple sclerosis remission and immune-mediated neuronal dysfunction in PANDA Syndrome and Sydenham's chorea. In addition, this new perspective on CNS immune regulation may help to advance the development of a potential Alzheimer's vaccine.

Hepatic Failure Involving Alpha-1 Antitrypsin Deficiency MZ PI Type

Romel C. Cunanan, MD, Michele L. Ledoux, DO, Giovannia DePetris, MD, Alan Epstein, MD

Roger Williams Medical Center/Boston University School of Medicine

A 77 year-old man with a history of a monoclonal gammopathy of unknown significance and prostate cancer status post anti-androgen therapy, LH-RH agonist therapy, and brachytherapy was evaluated in January 2002 for recurrent ascites. Two months prior to admission, as part of an initial evaluation for possible new ascites, an echocardiogram revealed biventricular heart failure with significant tricuspid and mitral regurgitation. Abdominal CT scan showed no liver lesions or ascites. The patient presented in early January 2002 for increasing abdominal girth. There was no pulmonary disease or hepatitis B or C, however there was a history of chronic hepatitis since 1996 when anti-androgen therapy, thought to be causing mild drug-induced hepatitis, was discontinued. There was no history of alcohol use, although the patient washed his hands with isopropyl alcohol daily. The patient also lived 3.5 miles downhill from a toxic landfill. Physical exam revealed jugular venous distension, an abdominal fluid wave, and dullness to percussion in dependent areas of the abdomen. Abdominal CT scan now demonstrated ascites and a "cirrhotic-appearing liver." The findings were felt to be secondary to congestive heart failure, and the patient was treated medically with diuretics. The patient returned in mid-January 2002 with refractory ascites. Laboratory studies showed a prothrombin time of 15.9s, serum albumin of 1.8g/l, total protein of 7.3 g/l, alkaline phosphatase of 422u/l, AST of 196u/l, ALT of 73u/l, and total bilirubin of 5.1 mg/dl (direct 3.3mg/dl, indirect 1.8mg/dl). Ascitic fluid analysis supported a transudative process (fluid chemistry: total protein=1.1g/dl, total allbumin <1,0g/dl, SAAG>1.1). Iron studies showed a serum iron of 122 mcg/dl, total iron-binding capacity of 138mcg/dl, ferritin of 1363.4ng/ml, and transferin saturation of 88.4%. Repeat screening for hepatitis B and C was negative. ANA, p-ANCA, anti-mitochondrial antibody, and anti-smooth muscle antibody tests were negative. Alpha-fetoprotein level was 2.3ng/ml. During transjugular liver biopsy, corrected sinusoidal pressure was 10mmHG consistent with portal hypertension. Hemotoxylin-Eosin stains showed evidence of fibrosis and widespread hepatocellular necrosis, but no evidence of fatty change or passive congestion. Prussian blue stains were negative for iron overload. However, Periodic acid-Schiff stains revealed multiple areas of cytoplasmic eosinophilic globules characteristic of **alpha-1 antitrypsin deficiency** (**AATD**). The alpha-1 antitrypsin level was 89mg/dl, and the AAT Pi type was MZ heterozygote. While liver cirrhosis from ASAT Pi ZZ homozygote type is well documented in younger cases, this case illustrates the rare occurence of cirrhosis involving AAT PiMZ heterozygote type at later onset. Without careful biopsy, the case would have been most likely labeled as a case of cryptogenic cirrhosis.

Lethal Acute-Onset Ammonemia in a 28-Year-Old Man

Elizabeth Tillman, MD, Sharon Flynn, MD Kelly McGarry, MD, Andrea Flory, MD

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Ammonemia is defined as a plasma ammonia (NH3) level greater than 60(mol/dL. Common etiologies include hepatic failure, portacaval shunt, and several medications. NH3 is produced during protein metabolism and by intestinal bacteria. Once in the bloodstream, it is broken down via two mechanisms: the urea cycle and conjugation with glutamate to form glutamine. If either or both of these pathways is dysfunctional and/or if these pathways are overwhelmed by massive NH3 production, ammonemia results.

A 28-year-old male presented with nausea, emesis, ataxia, and increasing confusion for two days. His past medical history was significant for cystic acne, depression, and severe poison ivy. One month prior to presentation, he was started on isotretinoin. One week prior to presentation he was started on paroxetine and prednisone. He took one dose of prescribed alprazolam the evening prior to presentation. The patient's social history was significant for employment at a pest control company, where he wore protective gear to prevent exposure to organophosphates. His family insisted he was not suicidal and had not overdosed. On initial exam, the patient was diaphoretic and markedly confused. The remainder of his exam was non-focal. Laboratory studies demonstrated a **creatinine phophokinase** (**CPK**) of 460U/L and NH3 of 165umol/dL. Liver enzymes were normal, except a mildly raised alanine transaminase. Toxicology screen was positive only for benzodiazepines, consistent with his medication history. Several hours after admission, the patient became unresponsive, exhibited pronounced anisocoria, and developed rigor-like movements and up-going plantar reflexes bilaterally. Computed tomography scan failed to reveal brain hemorrhage, herniation, or mass; electroencephalogram revealed no ictal activity; and lumbar puncture was negative for meningitis. Repeat laboratory tests revealed both increased CPK at 601U/L and NH3 at 270umol/dL. The next day, the patient's tremor generalized to include his entire body and his NH3 continued to rise, now 419umol/dL. He was curarized to prevent further muscle breakdown and was started on arginine and sodium benzoate for a presumed urea cycle disorder. Yet later that evening, his NH3 rose to 531umol/dL and he required emergent dialysis. Despite all efforts, the patient was deceased by morning. Results of a complete amino acid panel became available after his death. His glutamine level was grossly elevated and multiple urea substrates were aberrantly low, providing additional evidence of a urea cycle disorder. Post-mortum work-up demonstrated a partial ornithine transcarbamylase deficiency.

Creatine Dietary Supplement Induced Atrial Fibrillation

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We report a case of a young body-builder taking creatine to highlight the potential association with paroxysmal atrial fibrillation when taken with alcoholic beverages.

The patient is a 34 year-old white, athletic man in his usual state of health who presented via EMS to the emergency department complaining of six-month history of palpitations, associated with shortnesss of breath, feeling anxious and a ten-pound weight loss. Symptoms occured spontaneously, lasting briefly for a few minutes and were relieved according to him by "throwing cold water on his face." He complained of recent episodes being prolonged and occurring without alcohol consumption. The patient denied drug abuse but admitted to "alcohol binges" over weekends for approximately the last ten years. He initiated a self-prescribed regimen of creatine (glutamine and protine) dietary supplement approximately six months prior. The visit to the emergency department had been prompted by palpitations lasting an hour associated with post-prandial mild substernal chest pain, five out of ten in intensity, non-radiating and no association with nausea, vomiting, diaphoresis, loss of consciousness, fever, chills, cough, abdominal pain, diarrhea, nor viral symptoms, not relieved by the use of cold water on his face. His exam revealed a well-built man with normal vital signs, except for an irregularly irregular pulse. The EKG was significant for Rapid A. Fib. with a ventricular response of 130 and no ischemic changes. Lab data devealed Ca, Mg and Phosphate within normal, undetectable alcohol level; negative urine toxicology and a CK of 301 IU/L and normal CKMB%. In the emergency department an attempt was made to chemically convert to normal sinus rhythm with diHiazem V and anticoagulation without success. A trans-esophageal echocardiogram was performed and revealed no intramural thrombus, an ejection fraction of 65-70% with normal LA and LV dimensions. During the hospital stay we excluded myocardial infarction by enzymes and EKG changes. Within 72 hours of abstinence for creatine and Etoh he converted to and remains in normal sinus rhythm over the past four months. Upon discharge he was instructed to take aspirin [81mg], and abstain from caffeine, alcohol and creatine supplements.

There have been no prior reports of combined effects of alcohol and creatine-induced A fib. as described here. One report in *Drugs and Therapeutics Bulletin* describes creatine supplement adverse effects as A. Fib, RF, myopathy, fatigue, migraine and anxiety.

HIV/AIDS in Eldoret, Kenya

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In 1999 the **World Health Organization** (**WHO**) and UNAIDS estimated the national prevalence of adults (ages 15-49) living with HIV/AIDS in Kenya to be 13.5%. These estimates were extracted from HIV surveillance data available only for certain populations (pregnant women, sex workers, and **sexually transmitted disease** (**STD**) clinics) in mainly urban areas (Nairobi and Mombasa). This abstract describes information collected on HIV/AIDS from an epidemiological survey at **Moi University Faculty of Health Sciences** (**MUFHS**), Eldoret, Kenya.

During 3 weeks in October 2001, we conducted a prospective chart review of all admissions to one male and one female ward on the medical service at the district referral hospital at MUFHS. Located in Kenya's 5th largest city in the Western Highlands, MUFHS is one of two medical schools in the country. The associated hospital serves an encachement of 13 million. Data collected included: patient demographics, presenting signs and symptoms, admitting and discharge diagnoses, temperature, hemoglobin, and ELISA results. A compendium of diagnostic algorithms created using the best available published criteria for the developing world was utilized to assign diagnosis. HIV/AIDs was defined in accordance with WHO criteria.

Date were collected on 145 patients (76 women, 69 men), with a mean age of 37.1 (n=138). Of these, 22 (13.2%) met WHO criteria for AIDS. Many of the patients diagnosed with AIDS (63.6%) were also diagnosed with one or more of the following possible opportunistic infections: pulmonary **tuberculosis** (**TB**), TB meningitis, extrapulmonary TB, acute respiratory infection, and infectious diarrhea. There were a total of 50 ELISA tests ordered of which 40 were completed. Of all ELISA tests performed, 23 (57.5%) were positive. 4 patients had a positive ELISA but did not meet WHO criteria for AIDS. 4 patients who met WHO criteria for AIDS had no ELISA result available. The average length of stay for patients without AIDS was 4.4 days; the average length of stay for a patient with AIDS was 7.7 days. Of 708 total patient days, 169 (23.8%) were due to HIV/AIDS.

HIV/AIDS contributes significant morbidity in hospitalized patients in Eldoret, Kenya. A prevalence of 18.0% (15.2% by WHO criteria; 2.8% ELISA positive but not meeting WHO criteria) likely represents an underestimate in this population, given that less than 27% of hospitalized patients underwent HIV testing at all. Although antiretroviral therapy is presently unavailable to the majority of Kenyans (a fact which some utilize to argue against testing at all), applications of low-cost measures such as co-trimoxazole prophylaxis and TB treatment and prevention in HIV/AIDS has been demonstrated to extend lives, making early diagnosis a worthwhile endeavor.

Interferon-Induced Acute Insterstitial Pneumonia

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A 43 year-old woman with a seven-year history of hepatitis C and 30-pack-year tobacco use presented with progressive shortness of breath, nonproductive cough, wheezing and dyspnea on exertion for three weeks. The patient had started therapy with interferon and ribavirin for hepatitis C in December 2001 (about three weeks prior to symptom onset).

Physical examination was significant for an inspiratory squawk, expiratory wheezing diffusely, and decreased breath sounds diffusely. Oxygen saturation was 95% at rest and 93% with ambulation. This prompted an initial differential diagnosis which included bronchitis, chronic obstructive pulmonary disease exacerbation, atypical infection and interferon-induced reaction. The patient was started on a trial of azithromycin and fluticasone plus salmeterol. Four days later, patient exhibited worsening shortness of breath with a resting oxygen saturation of 83% and ambulating oxygen saturation of 81%.

Liver biopsy in October 2001 showed chronic hepatitis with mild portal inflammation - consistent with activity Grade I/IV and periportal fibrosis consistent with Stage II/IV. Hepatitis C genotype was positive, type 1a. Hepatitis B core antibody was positive, but hepatitis B surface antigen and antibody were negative. HIV tests were negative. Results of bronchoscopy were negative for legionella, pneumocystis carinii, acid fast bacilli, and cytomegalovirus. Chest radiograph showed increased interstitial markings. Serial chest radiographs over the following week revealed worsening interstitial lung disease, especially in the upper lobes. Computed tomography of the chest revealed scattered cystic changes throughout the lung. Mild diffuse bronchiectasis was seen in the lower lobes and in the lingula, with no discrete areas of honeycombing. A groundglass pattern was seen scattered throughout the lung fields. Lung wedge biopsies revealed organizing bronchopneumonia with obliterative bronchitis, chronic inflammation, acute interstitial pneumonitis and fibrosis of uncertain etiology, and an advanced degree of consolidation with complete obliteration of air space. The spared alveoli showed varying degrees of alveolar lining cell hyperplasia with focal squamous metaplasia.

These studies and the clinical course indicated an acute interstitial pneumonitis exacerbated by interferon and ribavirin therapy. There are rare case reports of this entity in the literature. The patient was treated with corticosteroids and pulse cyclophosphamide with improvement, but may need lung transplantation.

Medical Complications of Anabolic Steroids

Alexander A. Feller, Eleftherios Mylonakis, MD, Josiah D. Rich, MD, MPH

In the United States alone one million people are estimated to be current or past users of anabolic steroids.[1,2] Many anabolic-androgenic steroids users inject steroids intramuscularly, and up to 25% of adolescent anabolic-androgenic steroids users share needles, drastically increasing their risk of infection due to contaminated needles and unsafe injection practice.[3]

Initially, anabolic steroid use was limited to competitive bodybuilders and weightlifters. Now, its use extends to recreational athletes, body-conscious youth, and intercollegiate competitors. Among older high school adolescents use of anabolic steroids ranges from 4%-12% for males and 0.5%-2.9% for females.[1,2]. The prevalence of steroid use is 14% in college athletes and 30 to 75% in professional athletes or bodybuilders.[1]

Anabolic steroids in the United States are deemed Schedule III Uniform Controlled Substances, making their non-prescribed use illegal. In the 1980s, the government embarked on a policy of strict steroid control. Regulations mandating a decrease in the production of anabolic steroids resulted in a scarcity of steroids available to the recreational user. An unregulated black market emerged replete with drugs occasionally marred by infectious agents and mislabeled dosages. Laws controlling purchase and possession of needles and syringes may also increase contamination due to needle sharing. Major League Baseball has been grappling with how to implement a cogent steroid policy given recent allegations by current and past players of rampant steroid abuse.

Strength Effects

There has been much speculation in the medical community and in the public at large concerning the actual effects of anabolic steroids on strength and athletic performance. By the 1956 Olympics in Melbourne, Australia, reports of their androgenic effects led many competitors in the strength events to experiment with anabolic steroids. Anabolic steroids were banned from use in competition for the first time for the 1976 Olympics in Montreal, Canada. This International Olympic Committee policy has faced criticism from some scientists who claim that there exists no conclusive evidence that anabolic steroid use aids athletic performance. The effects on muscle size and strength are well documented, however. In one study, men were randomly assigned to the following four groups: testosterone and exercise, testosterone only, exercise only, and no exercise. Those men given supraphysiologic doses of testosterone showed significantly greater increases than those assigned to placebo in muscle size of arms, change in triceps area and legs, strength in bench press and squatting exercises.[4] Those men assigned to the testosterone and exercise group demonstrated the greatest increases in fat-free mass, muscle size, and strength.

Another comprehensive review of the subject determined that anabolic steroid use leads to a greater strength gain among well-trained athletes compared with placebos with a median strength gain of 5%.[5] No conclusions regarding the efficacy of steroids in enhancing athletic performance or on the generalizability of low-steroid dose studies to megadose studies were made. Megadose steroid use in conjunction with heavy exercise may lead to both structural and mechanical changes of the tendon. There are five reported cases of tendon ruptures associated with anabolic steroid-users. One case involved a 23 year-old bodybuilder who suffered a bilateral distal biceps tendon avulsion after ingesting megadoses of anabolic steroids for six years.[6]

Much of the research on the medical effects of anabolic-androgenic steroids has focused on males. Though significantly less likely than males to use anabolic steroids, females face some additional risk of steroid-induced masculinization, including acne, hirsutism, voice deepening, clitoral hypertrophy, and male-pattern baldness.

Cardiovascular Effects

The concern with body image and strength among young anabolic steroid-users does not appear to translate into a healthier lifestyle. Adolescent users of both sexes engage in more frequent cocaine, marijuana, cigarette, and alcohol use.[7] The difficulty in controlling for these adverse behaviors is an impediment that every analysis of anabolic steroids must confront.

Beyond behavioral correlates, the exact role of anabolic steroids in cardiac pathology is complicated and not entirely clear. The current experimental data suggest that use is linked to several cardiovascular risk factors including hyperinsulinism and decreased glucose tolerance, a variation in lipoprotein fraction, elevated triglycerides, increases in various clotting factors, initiation of platelet hyperaggregability, and alterations of the myocardium.[1] More specifically, anabolic steroid use has been demonstrated, in many studies, to lead to extreme reductions in **high-density lipoprotein** (**HDL**) subfractions and elevations in **low-density lipoprotein** (**LDL**) levels.[8] In terms of the relationship between coagulation and steroid use, animal studies have helped to delineate the link between increased thrombosis and elevated testosterone levels.[9] Such studies have verified that elevated doses of steroids impede cardiac pumping and circulatory regulation in untrained animals.

The cardiovascular implications of anabolic steroid use have garnered some scientific interest given the dozen case reports of sudden death among users. All the reports of morbid circulatory events associated with self-administered anabolic steroid use occurred among bodybuilders and power lifters who stacked multiple steroid packages, often in doses 10 to 100 times greater than the typical therapeutic dosages. Such extraordinarily high doses are quite common among bodybuilders. One representative case, for example, involved the instantaneous cardiac death

of a 20 year-old bodybuilder with no past or family history of cardiac disease.[10] The man had recently finished a three-month cycle of concurrent administration of several anabolic steroids. Autopsy revealed a heart that weighed 515g (250g normal).

Potential or current users should be cautioned that adverse circulatory effects exist whose severity may vary with type, dose, and time. With the exception of the alteration of the myocardium, all of these adverse circulatory effects are entirely reversible within several months after last steroid use.

Liver Functions, Lipid Profiles, and Blood Pressure

Among the more disputed effects of anabolic steroids are the impact on liver function, lipid profiles, and blood pressure. The literature contains instances where anabolic steroid use was associated with ultimately fatal liver dysfunction.[11] Conversely, several investigations have postulated that anabolic steroids have little impact on liver function. The distinction appears to be between intramuscular and oral administration of steroids. Intramuscular injection of anabolic steroids does not appear to be significantly associated with fluctuations in liver functioning. Orally administered steroids, however, especially 17-alpha-alkylated androgens, were associated with elevated liver enzyme levels in serum. These elevations corrected themselves upon discontinuation of the anabolic steroids.

Orally administered steroids have in some cases led to extreme liver dysfunction, including hepatocellular carcinoma.[12] In cases where tumor formation did not occur, hyperplasia and nodule formation was often present. Oral steroid use has also been associated with pelosis hepatis, a disease characterized by blood filled lesions within the liver[13] and subcapsular hematoma.[14]

For the clinician, a typical presentation of liver dysfunction among anabolic steroid-users is acute epigastric pain in the right upper quadrant. Within hours, the pain normally becomes more severe, and often radiates to the back. A **computerized tomography** (**CT**) scan can be used to evaluate the condition. Blood and fluid transfusions may be needed to stabilize the patients. A liver biopsy can be obtained to confirm the condition, but the procedure may result in hemorrhage of the weakened liver and should be viewed as risky. The long-term prognosis varies. After discontinuation of anabolic steroids, the condition, whether a tumor, nodule formation, or hepatic pelosis, may worsen, persist, lessen, or even disappear on its own.

Infertility commonly precipitates seeking medical care.

Infertility

Anabolic steroid-injectors frequently seek medical attention for infertility. Anabolic steroids are a derivative of testosterone, and the massive quantities injected by many users have an inhibitory effect on the hypothalamic-pituitary axis via negative feedback. The resulting condition of hypogonadotropic hypogonadism leads to azoospermia and often infertility.

Infertility commonly precipitates seeking medical care. Patients typically present with various sub-normal semen parameters and, at times, with testicular atrophy.[15] The normal concentration of semen is greater than 60 x 106 sperm per ml. Reports of anabolic steroid-users with sperm concentrations as low as zero exist.[15]

The initial treatment option is to immediately discontinue use of anabolic steroids. Depending on the combination of anabolic steroids used and their respective dosages, discontinuation may alleviate the problem. Though steroid-induced infertility is generally thought to be reversible, case reports suggest it may not always be. If discontinuation fails to promote healthy sperm production in sufficient quantities with normal endocrine balance after six months, administration of testosterone, human chorionic gonadotropin, and human menopausal gonadotropin can produce a dramatic return in sperm production and fertility rates.

Infectious Complications

Infections may represent the most severe adverse health consequence of anabolic steroids. Potential infections stem from : (1) contaminated needles, (2) shared injection equipment, and (3) non-sterile injection practice.

Intravenous drug users have higher rates of injection-related infections than anabolic steroid users. This disparity may be from the three orders of magnitude greater volume of blood transferred during intravenous compared to intramuscular injection.[16] The paucity of hepatitis B, hepatitis C, and **human immunodeficiency virus** (**HIV**) cases attributable to anabolic steroid use probably in part stems from the intramuscular versus intravenous injection and the smaller number of anabolic steroid users.

Though not approaching endemic proportions, the literature contains case reports of infections attributable to anabolic steroid-injection including HIV, hepatitis B, hepatitis C, and abscesses caused by atypical mycobacteria as well as Staphylococcus spp. and Pseudomonas spp. There are three separate reports of HIV infection among male heterosexual bodybuilders who each engaged in needle sharing associated with anabolic steroid-injection.[16] One of the bodybuilders acquired hepatitis B as well. We have reported a case of hepatitis C transmission via anabolic steroid-injection.[17] In addition, there have been eight case reports of abscesses and one report of Candida albicans endophthalmitis caused by anabolic steroid-injection.[18]

Infectious complications alone make needle sharing a dangerous habit. A physiologic and psychological addiction to steroids may also result from long-term use with withdrawal symptoms including depression, flu-like symptoms, and decreased libido.[19] Though not as addictive as heroin, for instance, many anabolic steroid users refuse to cease injecting. It is incumbent upon health care providers to apprise these individuals of the infectious disease risk of anabolic steroid use and educate them regarding the use of proper sterile injection technique. Though the risk of transmitting HIV and other communicable diseases via shared needles is slim, it is not nonexistent. All athletes with a history of prior needle sharing should receive HIV, hepatitis B, and hepatitis C testing. The Centers for Disease Control and Preven-

tion recommends hepatitis B vaccination for all individuals injecting drugs.

Immunology

There appear to be some negative consequences of heavy anabolic steroid use on immune function. Evidence exists that excessive anabolic steroid use may hinder immune responsiveness by triggering a reduction in serum immunoglobulins.[10] Supraphysiological doses of steroids also increase natural killer activity that could lead to the beneficial heightening of natural immune activity or the damaging potential of autoimmune tissue damage.[20]

Psychological Complications

Anabolic steroids can lead to psychological abnormalities including uncontrolled aggression, alterations in libido and mood, and psychotic symptoms.[19] Major mood disturbances associated with anabolic steroids may represent an important public health problem for athletes using steroids and sometimes for the victims of their irritability and aggression. Pope and Katz demonstrated in a cohort study that anabolic steroid exposure was significantly associated with increased major mood disorders. Up to 23% of steroid users had an affective disorder, with 10% having hypomanic episodes and 5% having manic episodes associated with steroid exposure.[21] Depressive symptoms have been described during the withdrawal episode and some cases of suicide, homicide, and violence toward women have been observed in subjects who were under the influence of anabolic steroids.[22,23] Finally, anabolic steroids may serve as "gateway" drugs, progressing to opioid dependence.[24]

Safe Injection and Harm Reduction Strategies

Though some anabolic steroids can be taken orally, a significant proportion are injected intramuscularly. Many of the aforementioned infection risks can be partially or fully mitigated by the use of safe injection techniques. Physicians and trainers should make every effort to discourage the use of anabolic steroids. If these efforts fail, harm-reduction strategies may provide an alternative.

Although controversial, harm minimization, as applied to anabolic steroid use, has gained proponents in other countries in which strategies have varied from a needle exchange scheme to a medical management program. Special needle exchange programs for steroid users have been implemented and proven effective in parts of Europe. However, to date, similar needle exchange programs have not been widely accepted in the United States. In a physician-managed harm reduction program for steroid users in Australia, Millar has reported success in that smaller doses are used, adverse effects are fewer, and more than half of the users ceased use within one year.[25] A pilot RI syringe prescription program provides a local model of an effective initiative to minimize harm among injection drug users.[26] Given the lack of effectiveness with less controversial approaches, harm reduction policies and programs may offer an opportunity for greater success with current steroid users.

Conclusions

Anabolic steroid use carries with it a host of medical, psychological, and social consequences. Medical practitioners treating potential or current steroid users should be aware that resistance to stopping steroid use is often considerable. At times, with regard to infection control, for instance, counseling steroid users about safer clean injection practice may represent the best strategy in the face of intractable steroid use. All steroid users should be warned about the variety of medical perils associated with anabolic steroids. Physicians treating athletes, especially in strength sports, should be vigilant for abnormal presentations that may stem from steroid use.

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Management of Hepatitis C in Rhode Island:
Opportunities for Improvement Within and Beyond the Department of Corrections

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Current estimates indicate that at least 4 million Americans are infected with hepatitis C, making it the most common chronic blood-borne infection in the United States and the most common indication for liver transplantation in adults.[1,2,3] The virus is most effectively transmitted through repeated or large direct percutaneous exposure to blood. Eighty-five percent of individuals acutely infected with the virus will develop chronic hepatitis C with persistence of **hepatitis C virus (HCV)** RNA in the blood. Approximately 15-20% of patients with chronic infection will ultimately progress to cirrhosis and 1-5% will die from liver cancer, resulting in 8,000-10,000 deaths annually in the United States.[3,4] The annual cost of acute and chronic hepatitis C care in 1999 was over 600 million dollars and costs continue to grow.[5]

In Rhode Island, active or past injection drug users comprise the vast majority of HCV-infected individuals. Data suggest that nearly 90% of injection drug users have been infected with the virus.[6] Approximately 60% of federal prisoners and 25% of state prisoners are incarcerated due to drug-related offenses, and a much higher percentage have a history of drug use.[7] A blinded sero-prevalance study in Rhode Island's prison population using discarded blood from mandatory intake screening for HIV showed that HCV serology was positive in 25.8% of tested individuals.[8] The prison system has been increasingly recognized as an excellent opportunity to screen and treat a group of individuals at risk for HCV in a controlled environment. The pervasive opinion that recent or active drug use is an absolute contraindication to interferon-based therapy has been a major barrier to the effective management of HCV.[3] However, a recent **National Institutes of Health (NIH)** consensus panel on hepatitis C emphasized that injection drug users, including individuals on methadone and those still actively using illicit drugs, may be effectively treated for this disease in certain settings.[9]

Hepatitis C Screening and Evaluation

Hepatitis C is potentially curable. When pegylated-interferon and ribavirin therapy are used in combination, the virus can be eradicated serologically (disappearance of HCV RNA in the blood) in over half of individuals.

Numerous challenges to effective treatment exist. First, funding for screening programs in Rhode Island is limited. Nationally, it is estimated that only 25% of HCV-infected individuals have been diagnosed. In comparison, because of the wide availability of HIV screening, up to 75% of HIV+ individuals are aware of their diagnosis.[10] The cost of HCV screening can range from $50 to $200 per patient. Consequently, some at-risk patients decide to donate blood or plasma at local donations centers, which screen all blood products for HCV and are obligated to inform patients if they are positive. Even those patients who have been screened for the virus at community clinics may not receive further evaluation, such as HCV genotyping, liver biopsy and other tests required for treatment initiation.

The **Rhode Island Department of Corrections (RIDOC)** does not offer universal screening for hepatitis C; therefore the majority of Rhode Island inmates have never been tested for the virus. Inmates on certain psychotropic medications receive **liver function tests (LFTs)** regularly; those with abnormal results are offered HCV testing. A documented history of injection drug use is also sufficient reason to screen for HCV. Inmates complaining of abdominal pain and fatigue are typically screened as well. However, HCV infection is typically a silent disease with a latent period that may last decades before symptoms appear. Additionally, abnormal LFTs are not consistently present in chronic hepatitis C. Many otherwise healthy inmates, particularly those with brief sentences, will not be tested for the virus. Often the opportunity to diagnose a potentially fatal disease is missed.

Hepatitis C Treatment

An additional challenge to treatment access is the expense of interferon and ribavirin. The one-year course of primary therapy required to treat the most common HCV genotype costs approximately $10,000-20,000. In comparison, however, the expense of coronary artery bypass is more than that of interferon-ribavirin therapy, and provides a lower gain in life expectancy.[11] It is estimated that medical care for a patient with compensated cirrhosis may cost $50,000 during the patient's lifetime. Patients with decompensated cirrhosis, requiring liver transplant, can generate costs well in excess of $100,000. The incidence of severe liver disease can be markedly reduced by early intervention with interferon-based therapies.

Interferon and ribavirin-based therapy itself commonly causes major side effects. Hematologic problems such as anemia may necessitate even more expensive pharmacologic interventions such as erythropoietin. Treatment expense can be a major barrier even for individuals who have insurance. With a 20% co-payment for HCV therapy, costs can approach $400 per month. Patients receiving interferon-based therapy must also receive close medical follow-up and have

access to complete psychiatric care, because treatment side effects such as depression, irritability and aggressiveness are common.

Hepatitis C management and treatment require the collaboration of multiple medical providers. Opportunities for uninsured patients in Rhode Island to receive this coordinated care are severely limited. For the small percentage of uninsured individuals infected with HCV who have completed pre-treatment evaluation, Schering-Plough Pharmaceuticals, the primary manufacturer of interferon and ribavirin, offers "Commitment to Care." The program provides 80 million dollars worth of free medication each year to those HCV patients nationwide who would otherwise be unable to afford therapy. It does not cover clinic visits, laboratory testing, and liver biopsies. Organized efforts would be useful to inform providers and relevant community agencies about this program.

Within RIDOC, the cost of evaluation and therapy that would result from the initiation of a universal screening program for HCV would overwhelm the department's already limited pharmacy budget. Currently, the total expense of hepatitis C therapy at RIDOC matches the total expense of HIV medications. Only psychiatric medications make up a greater proportion of the pharmacy budget. This is particularly noteworthy in light of the fact that only 15-20 individuals receive HCV treatment at any given time, out of the estimated 4000 HCV-positive male inmates who pass through the Rhode Island correctional system each year.

In Rhode Island and many other states, most inmates who are evaluated for HCV treatment must have at least 15 months left in their sentence, sufficient to complete the hepatitis C therapeutic regimen, to be considered for therapy. Individuals with viral subtypes that are particularly responsive to treatment (HCV genotypes 2 and 3) may be treated during shorter sentence periods. The rationale for this overall strategy is three-fold. First, the psychiatric side-effects of therapy could be a major liability for individuals who are going through the already-difficult "reentry" period after leaving prison. Additionally, at least 80% of prescribed interferon doses must be administered in order to maximize the likelihood of

Table 1. Goals of Rhode Island HCV Taskforce

1. **Improve Access to HCV Education, Screening and Diagnostic Evaluation**
   - Establish free HCV screening at local health clinics and drug treatment centers.
   - Develop programs enabling uninsured patients with HCV to receive pre-treatment clinical workups, in order to enhance access to Schering-Plough's Commitment to Care Program.

2. **Expand RI Internet Resources on HCV for patients and providers***
   Appropriate topics should include:
   - information regarding HCV prevention, diagnosis, treatment, insurance coverage, and local resources
   - a framework for HCV evaluation intended for general healthcare providers in order to expedite appropriate referral to specialty care

3. **Increase Focus on HCV treatment within the Correctional System**
   - Target treatment resources towards a population in which the disease is highly prevalent and provide therapy in a controlled setting to patients with patterns of high-risk behavior.
   - Adopt the model of current bridge programs available for HIV+ inmates: improve medical followup after release from prison by connecting providers in the community to inmates prior to release. This would require collaboration between the Dept. of Health, RIDOC and local health clinics.

4. **Assess Feasibility of Providing Interferon Therapy at Community Medical Clinics**
   - Within the Rhode Island prison setting, physicians trained primarily in internal medicine manage interferon therapy with excellent results.[8] With adequate training and support, more community providers could be capable of providing interferon therapy.
   - Hire physician's assistants (PAs) trained in coordinating HCV management to rotate between several community health clinics. The PAs could also be involved in an HCV bridge program for released inmates.
   - Encourage the use of the computer-based hepatitis C Tracking Program** which helps organize lab values, treatment dates, and other aspects of HCV management.

5. **Consider methods of improving adherence to interferon therapy**
   - The once-per-week interferon regimen lends itself readily to the possibility of directly-observed therapy, which has been used effectively for patients infected with TB and HIV in Rhode Island and elsewhere.
   - Weekly interferon administration would also provide an excellent foundation for the close medical followup necessary during hepatitis C treatment.

* A model for such a website can be accessed at www.hepccalifornia.org, a site developed in response to California's recent taskforce on hepatitis C. The "Strategic Plan" developed by California's hepatitis C taskforce is available at www.dhs.ca.gov/ps/dcdc/html/publicat.htm

** Tracking software for HCV management (HepTrak) is available for free download from the Maine Bureau of Health website: http://www.state.me.us/dhs/boh/ddc/Downloads.htm

virologic clearance of HCV from the serum. Access to interferon (or healthcare of any sort) is difficult to ensure immediately after prison release. Finally, these criteria limit the number of individuals eligible for treatment, thus reducing the overall expense of providing interferon and ribavirin to the largely transient RIDOC population.

HCV-infected inmates with expected sentences of less than 15 months, including inmates in the women's prison, are interviewed, counseled, and given information from Rhode Island Department of Health representatives. A social worker will also visit inmates prior to discharge and provide them with a listing of resources for HCV management in the community. Only a minority of released inmates ultimately seek healthcare following release; those who do are presented with the multiple barriers to HCV treatment described previously. A strategy to create a "safety net" to increase the opportunities for medical therapy at this point would be an important future management goal.

Effectiveness of Treatment in the Prison Setting

Correctional facilities offer an opportunity to make a major impact on HCV in the general population. An estimated one-third of Americans infected with hepatitis C will pass through the correctional system at some point during a year.[12] Given the prevalence of HCV in this population and the overall effectiveness of treatment in halting the progression of this potentially life-threatening disease, the opportunity to detect and treat HCV should not be missed.

Following release, many inmates return to the unstable environment which contributed to their criminal activities. Although active drug use should not be considered a contraindication to HCV therapy, it can complicate management. Treating patients during incarceration, a period of forced sobriety, is clinically sensible. In a recent study, over 85% of Rhode Island inmates who were willing and clinically eligible to undergo HCV treatment

Correctional facilities offer an opportunity to make a major impact on HCV in the general population.

completed the full year-long regimen.[8] In comparison, research focused on a metropolitan clinic population found that less than 40% of willing and clinically eligible patients were even started on interferon therapy.[13] In this outpatient setting, large numbers of patients were lost due to psychiatric problems, active drug dependence, and nonadherence to evaluation and education (e.g. not attending required appointments or lab visits). Not only are the problems of adherence and illicit drug use minimized in the prison setting, but the assurance of careful psychiatric management during treatment allows interferon to be administered effectively even in patients with psychiatric histories.[8] Because of close collaboration with the psychiatry team, none of the inmates in the RIDOC study had treatment discontinued for psychiatric reasons. These data support the efficacy of directly-observed therapy in an incarcerated population to enhance compliance and increase the likelihood of sustained virologic response.

Conclusion: Addressing Hepatitis C in Rhode Island

The magnitude of hepatitis C as a public health problem justifies the organization of a working group or taskforce to coordinate HCV prevention, screening, and treatment efforts in the state. The organization of a hepatitis C taskforce is feasible in Rhode Island and will help increase collaboration between the many academic and community experts within the state. The Department of Health is in the initial stages of assembling such a taskforce, which would include Brown Medical School faculty and other physicians, community members, RIDOC representatives, and individuals from the pharmaceutical and health insurance industries. The state registry for hepatitis C would be a valuable tool with which to make informed public health decisions and to monitor the effectiveness of various interventions. Community physicians are not uniformly aware that reporting of hepatitis C in Rhode Island is mandatory, so further efforts must be initiated to standardize information collection. Potential goals of the proposed Rhode Island Hepatitis C taskforce are outlined in Table 1.

Given that injection drug users are at great risk for contracting HCV, incarceration may represent for some persons their only potential encounter with the health care system. The prison environment is uniquely suited for high-yield HCV screening and education, initiation of HCV management and/or treatment in a controlled setting.

Rhode Island is already a leader in HIV care and syringe access programs. HCV/HIV coinfected patients have a relative wealth of resources available for medical care, but funding and programs for HCV-monoinfected patients have not kept pace. Identifying physicians who will accept the responsibility of managing hepatitis C, particularly in uninsured patients, is a continuing challenge. The Department of Health has begun efforts to improve the resources available to HCV-infected individuals in Rhode Island and there are clearly multiple areas in which improvements can be made.

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Controversies Regarding Low Blood Lead Level Harm

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Lead is the common name for the element Plumbum, a metal valued for its high density, malleability and corrosion resistance. No essential biochemical role for lead has been identified but evidence of its toxicity is abundant. Syndromes affecting workers in ore-related industries were described in the early 1900s. In 1932 a cluster of childhood lead poisonings was traced to inhalation of fumes from the burning of discarded lead battery cases. About 1940 it became clear that children were presenting with encepholopathy and high lead levels without any occupational exposures. Investigation revealed that the ingestion of chips of deteriorated house paint and soil contaminated by leaded fuel exhaust products were the likely domestic exposures. About this time the technology for reliably measuring **blood lead levels (BLLs)** became available and clinical correlation followed. The mean BLL of normal children with no suspicion for lead related problems was 30ug/dl.[16] The BLLs of the encephalopathic children exceeded 100, some exceeding 500.[19] A toxic profile for lead was established: (1) Very high BLLs (>100 ug/dl) cause encephalopthy, nephropathy or neuropathy and may be fatal, (2) Intermediate BLLs (45-100) may cause nephropathy, neuropathy, and a sideroblastic anemia, (3) moderately elevated BLLs (25-45) may interfere reversibly with heme synthesis, (4) low BLLs (<25) cause no harm.

A broad national effort was launched to protect the public from lead-related harm. The paint industry voluntarily withdrew leaded interior paints in 1955 and progressively reduced the lead content of exterior paints until all leaded paints were banned outright in 1978. A 1973 **Environmental Protection Agency (EPA)**-sponsored federal law required a phase out of leaded gasoline, the dominant domestic fuel since the 1920s. By 1975 new passenger cars and light trucks were required to burn unleaded fuels only. A regulatory goal was the reduction of lead in any gasoline to one tenth of a gram per gallon by 1986. In 1995, with leaded fuels accounting for less than 1% of domestic consumption, a total ban was enforced.

These measures effectively reduced BLLs and associated harm. By 1991, mean BLLs throughout the screened population had fallen to < 3 and the toxicities originally targeted were virtually eliminated. There was a new concern however.

About 1979, the toxicity paradigm was challenged by the hypothesis that even low lead exposure (BLL<25) can harm the very young. Infants and toddlers were considered a special risk group because they mouth and suck reflexively, because they hyperabsorb lead and because their nervous systems are not yet fully developed. Needleman et. al.,[1] in a widely cited paper, correlated lead levels in shed milk teeth with IQ scores and with various behaviors in first and second graders. The low lead group (BLL=25) slightly outperformed the high lead group (BLL=35) in most spheres. Multiple subsequent studies sought to link **deficits in IQ (DIQ)** or **Attention Deficit Hyperactivity Disorder (ADHD)** with low range BLLs in the very young.[2,3,4,5,6,7,8,9] In response, the **Centers for Disease Control and Prevention (CDC)** lowered their influential "level of concern" BLL from 30 to 25 in 1985 and from 25 to 10 in 1991. Mandatory screening of the very young was legislated and an extensive national BLL database was accumulated.

The premise of low BLL harm was not universally accepted. Skeptics questioned the statistical validity of the small effect, in a debate which continues to this day. Activists hold that low BLLs are a silent epidemic damaging the very young, that the threshold should be lowered further and that more aggressive preventative measures should be legislated. Skeptics remain unconvinced of the existence and/or significance of such harm and predict further sufficient declines in BLLs under existing policy.

This debate is being reprised in Rhode Island Superior Court. The state has filed a lawsuit holding the paint industry liable for the removal of lead paint from any RI building which children may access and for the costs of identifying and treating affected persons. The suit contends that lead paint causes ongoing harm to RI children and that the industry, aware of this potential toxicity, nonetheless conspired to market the product. If the suit is found meritorious and adopted by other states, the cost to defendant industries has been estimated at billions of dollars since a majority of structures built before 1970 contain lead paint.

The contention that the industry marketed paint knowing that *low* BLLs were harmful may be problematic. Major medical textbooks identified the toxic BLL threshold for children to be 60 as recently as 1982.[10] The hypothesis of low level harm did not appear until after the paint ban in 1978. The CDC's cautionary BLL>10 "level of concern" was not promulgated until 1991. The demonstration of harm in RI children may also be difficult to document. Unlike encephalopathy, neuropathy, or anemia, the harm attributed to low BLLs cannot be demonstrated in an individual child. It cannot be biopsied or imaged. Demonstration of the population effect requires formal testing of exposed and less exposed cohorts while controlling for variables which may affect intelligence or behavior. It is this abundance of covariables which makes the small low BLL harm controversial. Whether there is lead-associated DIQ or ADHD; if so, whether the association is causative; if so, whether paint among the many domestic lead sources is culprit; if so, whether it is poorly maintained paint that threatens; if so, whether maintenance is the responsibility of the manufacturer; if so, whether the proposed abatement will be effective, are links in the state's chain of logic which will be tested.

Clinical studies on low BLL harm vary widely in methodology and selection criteria. There are no large multicenter studies of the type usually employed to resolve controversial issues. The Johns Hopkins University, site of

much original research on lead toxicity, has been the target of multiple lawsuits for conducting a IRB-approved lead abatement study with a control arm.[11] The mother of a control child, as an example, has sued the University claiming that a BLL=32 allowed a learning disability to proceed untreated in her daughter. The control children in that study have been portrayed as "canaries in the mine". Nonetheless, many respected scientists from academic faculties and science advisory groups have expressed doubts about the existence or significance of low BLL harm.[2,3,4,12a,12b,13,14] In this brief article I will summarize some of the controversy regarding low BLL harm.

1) Lead-Associated DIQ

The clinical harm attributed to low BLLs is controversial, in part, because it is very small. While many peer-reviewed studies identify a lead-associated DIQ, others do not. A 1992 CDC review of 35 studies was unable to calculate a meaningful DIQ concluding that "...definitive conclusions regarding the effect of low-level body burdens of lead on IQ could not be determined from the longitudinal data...evidence however suggests an adverse relationship of lead on the intelligence of children."[9] A representative 1994 meta-analysis of the major DIQ studies[20] ascribed a decrement of 1-2 IQ points to a doubling of the BLL from 10 to 20. Since most "lead-poisoned" RI children have BLLs<15, the population risk would be an IQ point. Whether IQ can be measured precisely and reproducibly in the infant/toddler group; whether small differences are statistically or clinically significant; whether covariables were sufficiently controlled to make any small DIQ assignable to lead; whether the DIQ, if real, persists, are but a few of the questions raised in the literature.

Many factors affect IQ. Iron deficiency, not uncommon in the poor young, has been demonstrated to cause a DIQ of 4-7 points;[21,22] vitamin and mineral deficiencies, a DIQ of 3 points;[23] prematurity and/or low birth weight (which afffect 9% of RI births), a DIQ of 6-10;[25,26,27] flouridation of municipal water, a 5 point DIQ;[28,29] maternal smoking, a DIQ of 5; [30,31] lack of breast feeding, a DIQ of 4-6;[32,33] PCB exposure, a DIQ of 6 points;[34] maternal hypothyroidism a DIQ of 4-6;[24] etc. Elevated BLLs are more likely in poor neighborhoods where family, economic and nutritional deficiencies are themselves more likely. Home evaluations were declined by the responsible adult in a third of the "significantly poisoned" RI cases. Prenatal health and care, parental substance abuse, parental IQ, parental education, race, school quality, play quality, medical conditions, toxins and myriad other factors may affect IQ in the very young.

Causation is complex indeed. DIQ, if it exists, is multifactorial. Demonstration that lead is the principal cause of a small DIQ would require a very large study with control of covariables and extensive, reliable histories. No such database exists. Some DIQ studies contained fewer than 30 subjects. The largest involved 800 subjects. By comparison, over 9,000 acute **myocardial infarction** (**MI**) patients were required to identify a controversial 1% advantage of TPA over Streptokinase[35] in trials where causation was not an issue and the endpoint (death) was neither subtle nor subject to measurement error.

The significance of a small DIQ is arguable. The average IQ score is 100 with a standard deviation of 15. A 1-2 point variation is very small. Subjects do not record identical scores on retesting or on different IQ tests. Intertest and intratestee variations exceed the DIQ attributed to lead. A DIQ of 1 point may be compared to a height decrement of 1% from average (0.7"), a variation of 1% from ideal body weight (1.54 pounds), a diastolic BP rise of 0.8mm Hg, or a blood sugar fluctuation of 1 mg/dl. It is difficult to identify any health parameter in which a 1% change would be consequential to an individual. Many activists express concern, however, that a small shift of the population IQ curve would have significant national educational, financial and workplace effects. Historically this concern does not seem merited.

The absence of demonstrable intellectual harm in prior generations of lead-exposed children poses a challenge to the low BLL harm hypothesis. America, through the 1950s, was an inadvertent experiment in human lead tolerance. The domestic load was rising with escalating production of leaded paints and fuels. There was no medical concern, no mandated screening of the young, no early detection, no treatment and no abatement. Tens of millions of American children were raised with BLLs at least ten times greater than present levels.[15,16,17,18,19] Since toxicities are magnified as the dose excalates, children born between 1930 and 1960 should be the most affected group. Yet, decades later, those generations, now adults, have not been noted to be cognitively or behaviorally impaired.

Despite concern about the myriad factors including lead which may adversely affect cognitive skills, test scores of Amercian children on standardized IQ tests are rising steadily. Raw childhood IQ scores in industrialized countries throughout the world have risen about 3.5 IQ points per decade since 1900 when the tests were first widely applied.[36,37,38] This unexplained "Flynn Effect", named for the researcher who first noted the phenomenon, has required periodic downward recalibration of scoring algorithms and modification of theories of intelligence. This steady rise in IQ shows no correlation with population lead levels. IQ scores rose before and during the high lead era of 1930-1960. IQ scores rose at the same rate after 1970 as lead levels declined. The IQ gains occured in all industrialized countries some of which still permit leaded fuels. The preserved IQ performance of the millions of American children born and raised in the 1930-1960 era, when lead levels were ten times greater than today's, however, would suggest that any present low BLL harm willl not be reflected in population IQ scores.

2) Lead-associated ADHD

A lead-related causation for behavioral variants such as ADHD is even more controversial. Whether ADHD, characterized by hyperactivity, distractibility and impulsivity, is actual pathology or the tail of a distribution of normal behaviors is argued.[39] Some experts caution against the "medicalization" of behavioral characteristics, fearing the

appearance of "courage-deficit" or "truth-deficit" disorders.[39] Others postulate a genetic **basis claiming** that a variant dopamine receptor links alcoholism, drug addiction, tobacco addiction, compulsive gambling, ADHD and Tourette's Syndrome. Recent **positron emission tomography (PET)** Scan studies suggesting altered glucose metabolism in the "executive" areas of the prefrontal lobes[40] in ADHD have not been confirmed. That the reported prevalence of ADHD varies widely, as high as 16% in some studies, suggests a diagnostic inconsistency.[41] A recent Mayo Clinic study, for example, reported prevalences ranging from 7% to 16% in Minnesota youth depending on the strictness of application of DSM-IV criteria.[42] In England and some European countries, prevelances of 2% are representative. Most children referred for ADHD do not have elevated BLLs. One clinic reported a single BLL>10 among more than 100 children referred for diagnosis,[43] a rate below that of the general population. There are no published data on the prevalence of ADHD in RI children with BLLs>10. Multiple social, genetic and toxic factors have been alleged to be causative for ADHD, including PCBs, second hand smoke, parental substance abuse, dietary deficiency of fatty acids, parental attention, number of parents in the home, marital status of the parents, educational level of the parents, presence of the diagnosis in first-order relatives and birth order. In the decades prior to 1970, when lead levels were high, ADHD was an uncommon diagnosis. Since then the prevalence of ADHD has risen sharply as lead levels have fallen.

3) RI Statistics.

Last year, the **Rhode Island Department of Health (RIDOH)** reported 2887 cases of childhood lead poisoning. RIDOH defines as "lead poisoned" any child with a BLL>10. In neighboring Massachusetts the DOH defines lead poisoning by the more stringent criterion of a BLL>25 and reported fewer than 400 cases of lead poisoning last year. The Massachusetts definition, applied in Rhode Island, would identify fewer than 200 new poisonings each year. New York State defines lead poisoning as a BLL>20 and recorded 601 cases in its most recent (1999) report.

Such arbitrary definitions overcount, however, since to be "poisoned" means to have been "harmed by a substance". Given that demonstrable harm is uncommon at BLLs<45, the number of RI children literally poisoned may be fewer than 10 per year. For example, in 1994 of 8200 lead-poisoned RI children only 18 had BLLs>45.[49] A similar distribution among the present 2887 poisonings would find 6 RI children with BLLs>45.

The absence of demonstrable intellectual harm in prior generations of lead-exposed children poses a challenge to the low BLL harm hypothesis.

In their seminal 1942 article,[16] Kaplan and McDonald specifically cautioned: "Lead absorption is not synonymous with lead poisoning...A high blood lead level is not, of itself, diagnostic of poisoning but must be correlated with other findings, both clinical and laboratory..." OSHA policy conforms to this usage. OSHA identifies a screening BLL> 40 as the signal to evaluate adult workers for harm. If early reversible harm (altered heme synthesis) is detected, or if the level rises to 50, the worker is removed from the exposure. Levels of concern are deliberately set below the toxic threshold to allow for intervention before significant harm.

RIDOH acknowledges this stratification, de facto, by maintaining a second category, called "significantly poisoned" . Even this category, however, is defined by an arbitrary BLL (>15 twice or >20 once). In 2001, 237 RI children newly qualified as significantly poisoned without specific documentation of harm. Although elevated BLLs may occur in any locale in RI, 92% of the BLL>20 cases originated in Providence, Pawtucket, Central Falls, West Warwick, and Woonsocket. Providence alone accounted for two thirds of such cases. Medical treatment was recommended only for those few with BLLs>45.

The DOH's position is appropriate. Their charge is to protect the public, especially the young, from harm, even hypothetical harm. Their definition assumes that every RI child with a BLL>10 sustains significant cognitive harm, an unrealistic "worst case scenario". It is a cautionary policy intended to do the greatest good. The harm is neither measured nor documented; it is assumed to exist. This policy, however, creates a scientific catch-22. Children with BLLs>10 are assumed (defined) to be harmed until proven otherwise. Such proof, however, is unlikely to emerge because the harm cannot be measured in individuals and prospective controlled studies are legally perilous, requiring that control children remain "poisoned". Publication of the raw BLL data correlated with reported harm data would provide a useful database for clinicians, researchers, parents and advocacy groups. How many of the 2887 children with BLLs>10 were harmed? What type of harm? How is the harm documented? At what BLL?

4) Abatement

It is widely accepted that paint and fuel deposits are the principal sources for high BLLs.There are multiple domestic lead sources other than paint, including vinyl blinds, cosmetics, ceramic cups and bowls, gasoline precipitates in soil, municipal water supplies with lead piping, domestic lead piping, soldered cans, herbal remedies, candles, chocolate and other foods which may contribute to low BLLs. No study partitions low BLLs among these sources. Even if it were possible to identify and eradicate all domestic lead exposures, low BLLs would decline slowly as bone lead leaches into the plasma and is cleared by the kidneys, a process with a time constant of years.

Perhaps for this reason, the effectiveness of abatement protocols is disappointing. Abatement does not reduce BLLs to or near zero. An initial rise in BLLs occurs, likely related to generation of lead-containing debris. Long term results are not impressive. In a 1994 UMass study,[44] BLLs four years after paint abatement fell only 20% in the high

BLL (>25) group and actually rose 15% in the lower BLL (<20) group. Soil contamination, however, was not addressed. A 1997 Boston University study[45] involving paint and soil abatement confirmed that post-abatement BLLs were higher and concluded that combined remediation "…is not an effective secondary prevention strategy among children with mildly elevated BLLs." A CDC-sponsored abatement study[46] showed that 5 year BLLs in the "high" BLL (>35) group declined more (24% v.12%) than those of unremediated children but the lower BLL group (25-35) showed no advantage over controls.[46] Since 99% of RI's "lead-poisoned" children have BLLs <35, an abatement program is unlikely to lower their BLLs more rapidly or more effectively than present policy and may temporarily raise them.

Accordingly, many recommend a "sleeping dog" policy (as applied to residential asbestos or river bed PCBs) arguing that disrupting well maintained lead-painted surfaces is counterproductive. "Lead safe" housing, in which lead painted sites are identified, maintained and/or encapsulated, may be as effective as "lead free" housing.

Conclusion and summary.

The campaign to reduce lead related harm has been remarkably effective. BLLs have fallen to values considered "impossible"[47] when the paint and gas bans were enacted only decades ago. The originally targeted morbidities have been eliminated. The decline in lead levels among school children, considered a "public health success story of almost unprecedented magnitude" by the CDC's Director of the Bureau of Health Statistics[48] in 1998, has continued. In the latest 1999 NHANES data[49] the national mean BLL of the 1-5 year old group was 2. The CDC's goal to eliminate childhood lead poisoning by 2010 may be achievable. Whether low BLLs cause significant morbidity remains a complex topic on which reasonable scientists differ. Certainly they do not cause severe deficiencies in individual children. Public health agencies have adopted an appropriate and effective cautionary policy. The inherent "softness" of the cognitive and behavioral diagnoses, their multifactorial causation and present impediments to large controlled studies will extend the debate and may even preclude a definitive conclusion. The BLLs of RI children in 2002, however, are at historic lows, dramatically lower than those of their parents and grandparents, which generations, in retrospect, did not demonstrate measureable intellectual or behavioral harm. Since the risk of toxic harm is proportional to exposure, today's RI children are at least an order of magnitude less at risk. RI law mandates annual screening BLLs for the very young. RIDOH programs identify and control exposures in the two to three hundred new annual cases with BLLs > 20, over 90% of which occur in a handful of RI cities and towns. Such statewide vigilence, while mean BLLs continue to fall, would seem to amply protect RI children from the putative risk of low BLL harm.

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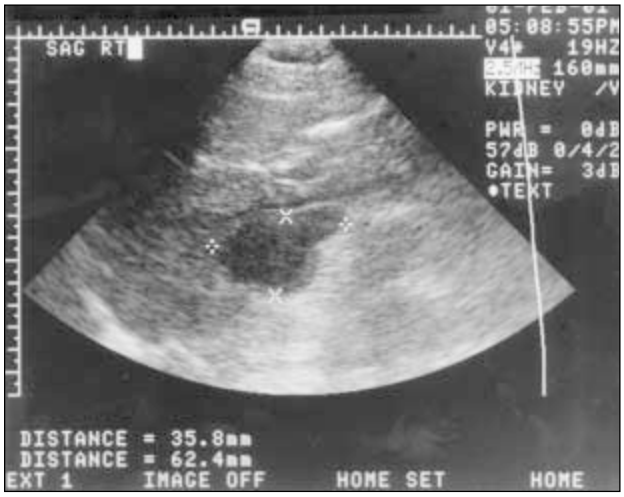

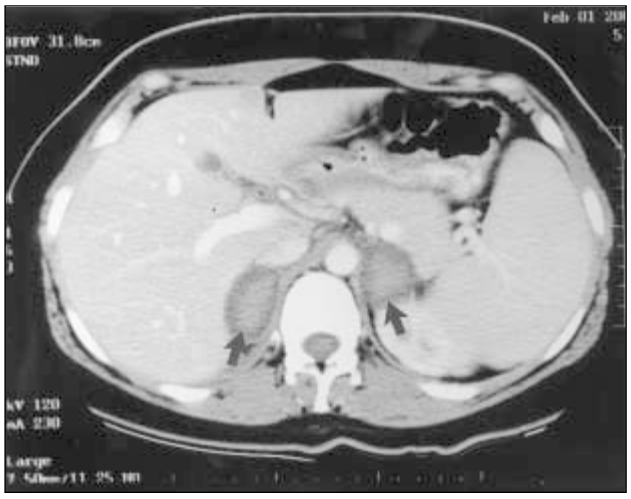

Spontaneous Adrenal Hemorrhage

A 33 year-old female presented to the emergency room two weeks post-partum complaining of fatigue, weakness, and vomiting for four days. Past medical history was significant for DVT and hypercoaguability secondary to Factor V Leyden deficiency and antiphospholipid antibodies. She was on maintained on coumadin. A right upper quadrant **ultrasound (US)**, performed for elevated liver function tests, revealed bilateral hypoechoic adrenal masses. (Figure 1) A **computed tomography (CT)** examination performed, with and without intravenous contrast, demonstrated enlarged, hyperdense, non-enhancing adrenal glands, (Figure 2) consistent with adrenal hemorrhage. Corticotropin stimulation testing confirmed adrenal insufficiency.

Spontaneous adrenal hemorrhage is an uncommon event which, if left untreated, can result in death from acute adrenal insufficiency. Although classically associated with meningococcemia sepsis in Waterhouse-Friderichson syndrome, there are many clinical settings that predispose a patient to adrenal hemorrhage. These include pregnancy, coagulopathy, underlying tumor, and other sources of physiologic stress, such as severe burns. The diagnosis of non-traumatic adrenal hemorrhage is often delayed secondary to vague clinical symptoms and lack of abnormalities on routine lab work. A high clinical index of suspicion is needed for early diagnosis.

– LEAH SCHAFER, MD, AND
JON VACCARO, MD

Leah Schafer, MD, is a third year radiology resident at the Rhode Island Hospital.
Jon Vaccaro, MD, is a staff radiologist at the Rhode Island Hospital.

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Using Behavior Change Staging to Change Patient Behavior in Chronic Disease

Raymond B. Maxim, MD

Successful outcomes in those with chronic conditions are dependent on patient involvement and participation in their own care. Patient participation in treatment is a series of behaviors that affect treatment success or failure. Those behaviors can change with timely and appropriate interventions. Individuals attempting a change in a behavior pass through identifiable stages on their progression to change.

Disease classifications and staging instruments are common and useful tools in the practice of medicine. The New York Heart Association Functional Classification and the TMN staging of cancers are examples of familiar staging methods used by primary care physicians and specialists alike to describe their patients' condition and guide their treatment. Physicians are comfortable with the concept of disease as a process. Physicians are, however, less comfortable with behavior as a process. A substantial body of evidence suggests that behavior change is a process. Like all processes, it is often amenable to change.

Prochska and DiClemente described five stages of change in their Transtheoretical Model of behavior change, each of which respond to different interventions in the progression to the next stage.[1-5] The Transtheoretical Model is a framework on which other interventions and techniques are hung. The five stages are precontemplation, contemplation, preparation, action, and maintenance. A patient attempting a behavior change passes through each of these stages on the way to change. Progress may stall at any stage or regress back to the precontemplation stage or any stage in between. The goals of a provider are to identify the current stage and provide the appropriate intervention. It is important for the physician and the patient to remember that regression is not failure. "Recycling" through stages is a natural part of the process of change.

Those not considering a behavior change in the next six months are in the **precontemplation stage**. This is the most challenging of stages. A patient in this stage is not thinking about changing behavior. The intervention for this stage is educational. To ask someone to change a behavior in this stage is unlikely to be effective. An "Action Message" will not be heard or acted upon by a patient in precontemplation. In patients with diseases requiring multiple behavior changes, it is important to remember that not all behaviors will be in the same stage. For example, a patient with diabetes may not be ready to begin a diet but may consider an exercise program. Address each behavior at the appropriate stage. Successful change in any behavior may provide a stimulus for further behavior changes.

The **contemplation stage** begins when the person is considering a change in behavior in the next six months. In this stage, the patient is actively weighing the pros and cons of changing his or her behavior. The decisional balance initially in precontemplation is strongly in the favor of continuing current behavior. As persons progress toward precontemplation the balance shifts to an equal balance between the pros and cons. Before entering the next stage, the pros to behavior change must outweigh the cons.

Efforts by providers should be directed at tipping the balance in favor of behavior change. An important factor for many patients at this stage is the matter of self-efficacy. The patient must reasonably believe that they can change their behavior. These are patients who are overwhelmed with the complexities of their disease. Simplification of the behaviors that need to be changed can be helpful. For example a patient who says, "I can't possibly begin to manage my diabetes myself" is better served by concentrating on a particular component of diabetes self-management. Isolating a single behavior that the patient feels most confident in changing, as other educational efforts continue at a less intensive level for other components, is more manageable for some. Family support has a positive effect at this stage as long as it is not artificial of the patient's efforts to change. Psychotherapy is helpful for some and may provide the beginnings of insight into the behavior change process.

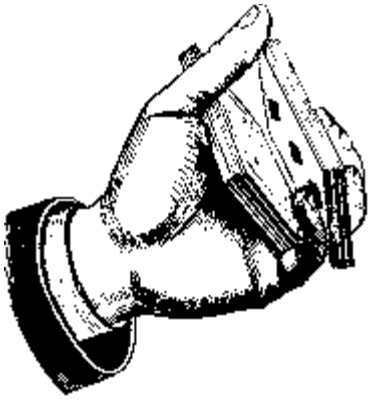
The next and perhaps the most interesting stage is the **preparation stage**. This stage by definition refers to someone planning a behavior change in the next thirty days. In preparation, the person has made the decision to initiate a behavior change. At this stage, persons are anxious to begin change but need direction to identify potential barriers to change. Enthusiasm is necessary when dealing with patients preparing to change. Anticipation of common pitfalls is part of the work of preparation. For example, those patients attempting to quit smoking need to recognize the environ-

mental triggers in order to prepare a strategy against temptation. It is difficult to develop an immediate response to a temptation as it arises. A plan for dealing with these situational temptations will increase the likelihood of success.

Once a person initiates active efforts to change behavior the **action stage** begins and continues for the next six months. In this stage, as well as the previous, enthusiasm is essential. Providers should continue attention into developing insight into the disease process or current detrimental behavior as well as the process of behavior change in order to improve the possibility of sustained change. Behavioral techniques such as substitution of behaviors or utilizing rewards are helpful for many during the action stage. An example of behavior substitution would be the substitution of the morning cup of coffee with tea or other beverage. Alone this behavior substitute will not have a large impact on the patient's likelihood of smoking cessation. However, in combination with other simple changes and behavioral techniques, the chance of success is greatly increased. Patient-designed rewards for obtaining short and long-term goals can stimulate success.

After behavior change has lasted for more than six months, the person has reached the **maintenance stage**. Similar to the action stage behavioral and cognitive techniques are helpful. Focusing on the reentry into situations previously avoided is done with care and planning to avoid relapse. Dealing with extreme stress is a frequent reason for relapse and contingency plans for these life events need to be considered. Helping patients find healthier ways to cope with stress such as exercise or meditation will provide them with a safety net when a major life event occurs. A well-developed support network can contribute to a successful weathering of a traumatic life event.

Assisting patients with behavior change whether if is smoking cessation or diabetes self-management can be rewarding. There are very few people as proud and thankful as someone who has mastered significant behavior change. To avoid frustration for patients and providers use only stage appropriate interventions. Keep in mind that behavior change is not a single event and that recycling is not failure.



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Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health
Edited by Jay S. Buechner, PhD

Disparities in Infant Mortality and Contributing Factors in Rhode Island

Samara I. Viner-Brown, MS, and Rachel Cain

Rhode Island's infant mortality rate (deaths among infants aged less than one per 1,000 live births) fluctuates from year to year due to the state's small population and relatively small number of infant deaths. The long term trend in this rate has been one of decline, with much less progress in the past fifteen years and none in the past five.[1] National data also indicate reductions in infant mortality over time.[2]

Within these trends, substantial disparities exist among different racial/ethnic groups at both the national and state levels. In Rhode Island, infant mortality rates among African Americans have consistently been 1.5 to 2 times greater than the White rate over many years.[3] This analysis presents data on factors that may contribute to this persistent disparity.

The Healthy People 2010 national objectives include reducing infant mortality to 4.5 infant deaths per 1,000 live births among all racial/ethnic groups; reducing low birth weight (< 2,500 grams or 5.5 pounds) to 5.0%; very low birth weight (<1,500 grams or 3.3 pounds) to 0.9%; prematurity (< 37 weeks) to 7.6%.[4] (Table 1) Rhode Island has assessed its progress towards achieving these objectives, and recent trends are not encouraging.

Methods

Birth certificate data were obtained for Rhode Island residents for the years 1997-2001. Rhode Island resident infant deaths were identified and linked to the birth record. Data include all deaths among Rhode Island resident infants aged less than 365 days. Infant mortality was calculated using race of the mother. However, when mother's race was unknown, infant's race on the death certificate was used. Data for 1999-2001 are provisional. Due to small numbers, three-year moving averages were calculated when comparing African American and White infant mortality rates.

Results

In Rhode Island, over the past three decades, infant mortality rates have declined from 19.7 (312 deaths) in 1970 to 6.6 (84 deaths) in 2001. (Figure 1) In 1994, Rhode Island experienced its lowest recorded infant mortality rate of 5.0 (68 deaths). Over the most recent five years, the infant mortality rate has varied between 5.7 and 7.1.

The large majority of infant deaths are born at low birth weight. During the early 1990s, Rhode Island saw a rise in the percentage of babies born at low birth weight. In 1991, babies born at low birth weights accounted for 5.9% of all births; by 1997 7.4% of babies were born at low birth weight, of which 1.5% were very low birth weight. Over the most

Table 1. Healthy People 2010: Maternal, Infant and Child Health Selected Health Objectives

Objective		U.S. 1998 Baseline	U.S. 2010 Target	Rhode Island 2001
[illegible]	Infant deaths (under one year) per 1,000 live births	7.2	4.5	6.6
[illegible]	Low birth weight (LBW)	7.6%	5.0%	7.5%
[illegible]	Very low birth weight (VLBW)	1.4%	0.9%	1.5%
[illegible]	Preterm births	11.6%	7.6%	9.1%

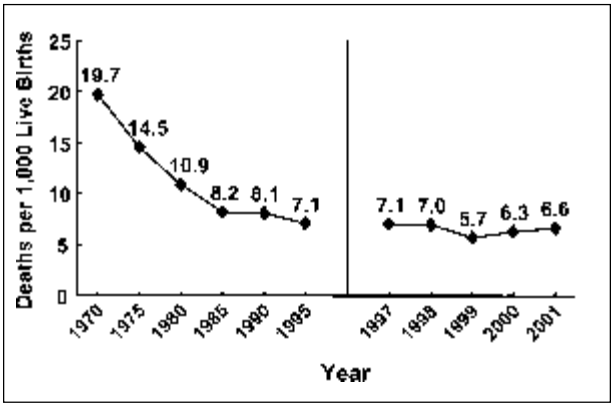

Figure 1: Resident Infant Mortality Rates, Rhode Island, 1970-1995 and 1997-2001

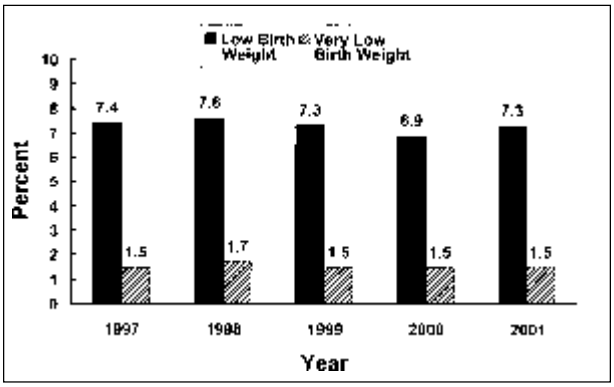

Figure 2: Percentage of Babies Born at Low Birth Weight, Rhode Island, 1997-2001

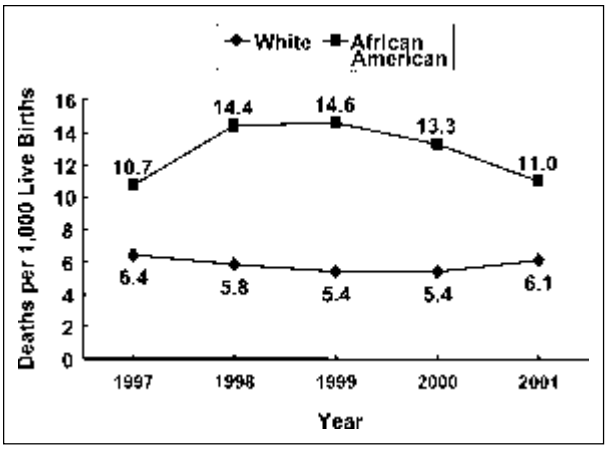

Figure 3: Resident Infant Mortality Rates by Selected Race, Three Year Moving Averages, Rhode Island, 1997-2001

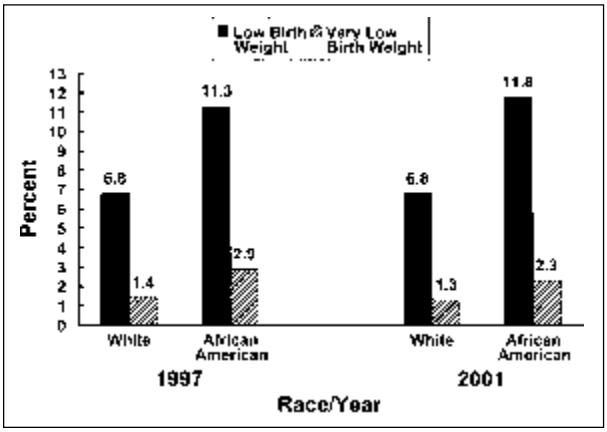

Figure 4: Low Birth Weight and Very Low Birth Weight by Selected Race, Rhode Island, 1997 and 2001

recent five years (1997-2001), both the low birth weight rate and the very low birth weight rate have been relatively stable. (Figure 2)

However, during the same five-year period, the proportion of infant deaths that were among low birth weight and premature infants rose. In 1997 the proportion of infant deaths among Rhode Island residents that were born at low birth weight was 64.8%; in 2001 it had increased to 77.4%. The proportion of infant deaths that were very low birth weight also increased, from 53.4% in 1997 to 70.2% in 2001. Similarly, in 1997, babies born prematurely accounted for 56.8% of infant deaths, rising to 78.6% in 2001.

Substantial disparities exist between African Americans and Whites in the rates of infant mortality, low birth weight, very low birth weight and prematurity. Historically in Rhode Island, the African American infant mortality rate has averaged twice the rate among Whites. During the five-year period, 1997-2001, the gap widened to the point in 1999 where the African American rate was nearly three times the White rate. (Figure 3) By 2001, the ratio of African American to White infant mortality had fallen back to 1.8.

Statewide rates for low and very low birth weight also differ substantially among African Americans and Whites. (Figure 4) In 2001, the low birth rate among African Americans (11.8%) was nearly twice the rate among Whites (6.8%). African Americans were also nearly twice as likely (2.3%) to deliver a very low birth weight infant as Whites (1.3%).

In addition, the rate of prematurity has been on the rise. In 1997, 6.1% of all babies were born prematurely compared with 9.1% in 2001, a 49% increase. Data for 2001 indicate that the prematurity rate among African Americans (12.8%) was nearly 1.5 times higher than the rate for Whites (8.7%). During the five-year period, 1997-2001, the proportion of African American babies born prematurely increased from 7.5% in 1997 to 12.8% in 2001. Among Whites, the prematurity rate increased more slowly, from 5.9% in 1997 to 8.7% in 2001.

Discussion

Rhode Island is not on course to meet the Health People 2010 infant mortality objective of 4.5. In order to do so, racial and ethnic disparities in birth outcomes must be eliminated. This can only be achieved if the risk factors for infant mortality (low birth weight and preterm deliveries), which have been increasing, are reduced.

Despite major investments in medical insurance and prenatal care for low-income women, pregnancy outcomes have not improved, and ethnic and economic gaps persist. We must study our recent pregnancy outcomes and plan much stronger emphasis on known challenges to women's health including nutrition, smoking, stress, and poverty, along with other elements of primary prevention. A more detailed look at recent time trends will be presented in a future issue.

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– A Physician's Lexicon –

The Words of Neurology

Neurology, more than most medical specialties, is endowed with a rich and etymologically intricate vocabulary. Consider a simple sentence such as: This 68 year-old male suffers from an aphasia, expressive agnosia, amusia, apraxia, acalculia and adiadokinesis. Each of these diagnostic terms represents a complex failing or lack of some vital function or activity; and each begins with the letter "a" denoting the way that classical Greek signifies a privative state, a negation or a deprivation of some quality.

Aphasia, the inability to speak, is derived from a Greek word *phasien* [meaning to speak] with the privative suffix, *a-* , denoting lack of. The root is cognate with another Greek word *phemien*, also meaning to speak. This is carried over to the Latin *faman*, meaning to report or discuss, which, in turn, has evolved into the English word, fame.

The word, **agnosia**, defines a neurological deficit characterized by the inability to understand receptive stimuli. It is derived from a Greek word meaning to know. Thomas Huxley, in 1864, coined the word, agnostic, to define those who declare that any understanding of a higher authority is unachievable, that they cannot know the unknowable.

Amusia [a form of phasia characterized by the selective inability to distinguish musical notes] is from the Greek word *mousa*, meaning music and again, the privative, *a-*. Amuse, on the other hand, is from the Latin, *muse*, meaning to ponder, to reflect upon. This in turn is unrelated to the Greek word, Muse, one of the nine daughters of Zeus and Mnemnone, and the guardians of the arts.

Apraxia is defined as the incapacity to execute purposeful, voluntary movements assuming the presence of an intact motor and sensory system. It is derived from the Greek workd *praxis*, meaning to do or to make. A number of English words have *praxis* as their etymological origin, including practice, practical, pragmatic, and chiropractic [literally, from the Greek, meaning to accomplish with the hand].

Acalculia is the loss of the ability to perform elementary arithmetical procedures. It is derived from the Latin, *calculus*, the capacity to reckon or to compute, which in turn is the diminutive of *calcis*, a small stone such as may have been used in primitive calculations. Words such as calcareous or urinary calculi demonstrate their "stony" relationship to *calcis*.

Adiadochokinesis represnts the inability to perform alternating motor movements. The Greek *diadoche* is defined as the succession [or the successor, as in the word Diadoche, those who succeeded Alexander the Great in military leadership]. And *kinesis* is from a Greek word meaning movement or quick motion [as in telekinesis or cinema].

– Stanley M. Aronson, MD, MPH

Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	November 2001	12 Months Ending with November 2001		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	235	3,046	290.6	4,479.0**
Malignant Neoplasms	206	2,394	228.4	7,388.0
Cerebrovascular Diseases	48	541	51.6	785.0
Injuries (Accident/Suicide/Homicide)	37	403	38.4	7,249.0**
COPD	48	509	48.6	507.5

Vital Events	Reporting Period		
	May 2002	12 Months Ending with May 2002	
	Number	Number	Rates
Live Births	1,062	13,085	12.5*
Deaths	802	10,140	9.7*
Infant Deaths	(6)	(102)	7.8#
Neonatal deaths	(2)	(85)	6.5#
Marriages	791	8,132	7.8*
Divorces	249	3,276	3.1*
Induced Terminations	504	5,599	427.9#
Spontaneous Fetal Deaths	61	1,085	82.9#
Under 20 weeks gestation	(56)	(1,004)	76.7#
20+ weeks gestation	(5)	(81)	5.8#

(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 1,048,319

(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
** Excludes one death of unknown age.
\# Rates per 1,000 live births

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Ninety Years Ago

[November, 1912]

An Editorial, "Mr. Roosevelt's Recent Peril," noted that the attempt on the ex-President's life ("fortunately averted by a roll of manuscript and a spectacle case which... deflected the course of the bullet") marked the fourth time assassins had attacked a President. This latest attempt "startled the entire world and especially impressed medical men." The Editor suggested that assassins, motivated by notoriety, should be shunned by the media. "Medical journal and medical men" should suppress the name of the assassin: "We can refer to him merely as 'Number Four' and leave him to ...merited oblivion."

In "Some Cardiac Disorders," Frank T. Fulton, MD, praised the "polygraph and string galvanometers." He described seven disorders: sinus arrhythmia, heart-block, premature contractions, paroxysmal tachycardia, auricular fibrillation, auricular flutter, alternation of the pulse.

D. L. Richardson, MD, reported on the "International Congress of Hygiene and Demography," held in Washington, DC, in September - the first time the 60 year-old Congress met in the United States. The Congress previously had met in 1907, in Berlin. President Taft, the Honorary President of this Congress, welcomed the 28 nations represented. (Germany had the largest delegation, 275). "One very interesting preliminary announcement was made by Dr. Roseneau of Harvard upon methods of transmission of epidemic poliomyelitis...Several speakers inclined to the belief that it is an insect-carried disease, because it dies out during the cold months and because it is largely a rural or semi-rural disease. Dr. Roseneau said that they had succeeded in infecting 6 monkeys by exposing them to stable flies, 'stomoxyscalatrans,' which had fed previously upon monkeys suffering from the disease."

The journal reviewed *An Essay on Hasheesh, Including Observations and Experiences*, by Victor Robinson, Pharmaceutical Chemist [and user], Columbia University, 50 cents. The reviewer commented: "...the author is...entertaining us with the interesting narrative of a voluntary excursion into the realm of cerebral stimulants, the ...results of which had perhaps not quite subsided even as portions of the account were written."

Fifty Years Ago

[October, 1952]

Jose M. Ramos, MD, in "Present Day Concept of Rheumatoid Arthritis and Allied Diseases," discussed the histophysiology of connective tissue, its relation to rheumatic disease, and clinical manifestations.

John Morgan McKenna, a Charles F. Chapin Research Fellow, 1951-52, and an Assistant in Bacteriology, Providence College, contributed "Grouping and Typing of the Streptococci with Specific Bacterial Viruses." In 1947 Mrs. Charles Chapin had established the fellowship for research in contagious diseases; this marked the third scientific investigation supported by the fellowship. The study was undertaken in the Laboratory Section, the Charles V. Chapin Hospital, and the Biology Department, Providence College.

Seebert Goldowsky, MD, in "Spontaneous Rupture of the Abdominal Aorta," reported on a study of 45 cases from the files of Rhode Island Hospital's Institute of Pathology, 1936-50. He found their commonest single trait: "pain of varying intensity."

An Editorial, "Careless Publicity," lamented the media attention to cancer and polio: "Even the doing of good can be overdone." The media's focus on cancer had made the public cancer-phobic, and media warnings on polio had sparked hysteria: "...consider the polio campaign: quantitatively the paralysis and death from polio are certainly not big compared to rheumatic heart disease, arthritis and automobile accidents. What a tremendous hysteria has been aroused ... out of proportion to the size of the problem..."

A second Editorial, "216 positives," praised the testing campaign, spearheaded by the diabetes committee of the Medical Society: of 11,347 tests during the 1951 detection campaign, 216 persons tested positive; 42 of them were juveniles.

Twenty Five Years Ago

[October, 1977]

In "Message from the Dean: The Veterans Administration Hospital, Providence, Rhode Island," Stanley M. Aronson, MD, cited 2 newly-announced events that showed the "pivotal role" of the VA in American medicine: 1) 2 of the 3 recent Nobel Prize (medicine) winners were full-time employees of VA Hospitals; and 2) October 14, the Providence hospital opened its new Ambulatory Care Pavilion. Rhode Island had 151,000 veterans; Southeast Massachusetts, 82,000 veterans. More than half of all Rhode Island males over age 20 were veterans. The first Veterans Administration hospital opened in 1949. In 1973 the Providence VA Hospital affiliated with Brown's Medical School.

Gerald A. Faich, MD, MPH, and William E. Martin contributed "Results of the Rhode Island Fight Flu Campaign." They cautioned: "A vaccination epidemic associated wth no disease is better than a disease epidemic associated with no vaccine." Rhode Island's program, launched in July 1976, marked the largest vaccination campaign in the state since the "End Polio Campaign" of 1960. Roughly half the target population (323,662 persons, from a target of 647,000) were vaccinated.

In "Participation of Providence Senior Citizens in the Swine Flu Inoculation Program," William R. Aho, PhD, Associate Professor, Department of Sociology and Social Welfare, Providence College, summarized results of a survey of 123 participants from 2 Providence senior centers. Over half had been inoculated, primarily at the senior centers. The reasons people gave for not being inoculated included: "...side effects, were advised no by their physician, or just didn't feel it was necessary."

Commenting on nosocomial infections, Carl W. Walter, MD, Clinical Professor Surgery Emeritus, Harvard Medical School, presented the 36th Annual Charles V. Chapin oration, "The Physician's Role in Cross Infection."