Cover: "Flower", a Chinese brush painting by Sanford Schatz. Dr. Schatz is a radiologist with Rhode Island Medical Imaging, based at The Miriam Hospital. His work is displayed at Studio Hop in Providence, Holland's Gallery in Warwick, and Wellspring Books in Newport.

Medicine & Health
RHODE ISLAND

VOLUME 86, NO. 2 FEBRUARY, 2003

PUBLICATION OF THE RHODE ISLAND MEDICAL SOCIETY

COMMENTARIES

COLUMNS

Medicine and Health\Rhode Island *(USPS 464-820), a monthly publication, is owned and published by the Rhode Island Medical Society, 235 Promenade St., Suite 500, Providence, RI 02908, Phone: 401-331-3207. Single copies $5.00, individual subscriptions $50.00 per year, and $100 per year for institutional subscriptions. Published articles represent opinions of the authors and do not necessarily reflect the official policy of the Rhode Island Medical Society, unless clearly specified. Advertisements do not imply sponsorship or endorsement by the Rhode Island Medical Society. Periodicals postage paid at Providence, Rhode Island. ISSN 1086-5462. POSTMASTER: Send address changes to Medicine and Health\Rhode Island, 235 Promenade St., Suite 500, Providence, RI 02908. Classified Information: RI Medical Journal Marketing Department, 175 Mathewson Street, Providence, RI 02903, phone: (401) 331-4637, fax: (401) 331-3594*

COMMENTARIES

First Do No Harm—Unless…

The identification of the syndrome "**normal pressure hydrocephalus**" (**NPH**) has caused more problems than it has solved. A disorder that was rampant (primarily in Boston) in the late 70s and early 80s has, luckily, gotten a lot rarer. The most reliable estimate for incidence is about one per million per year, somewhat rarer than **Creutzfeld-Jacob disease** (**CJD**). Since CJD is a lot easier to recognize, and has less overlap with common disorders, it isn't much mentioned during hospital rounds when yet another 85 year-old demented, gait-impaired patient is discussed.

A syndrome consisting of ventricular expansion, gait impairment, reduced mental function and bladder hyperactivity became a recognized entity once people began to survive disorders such as bacterial meningitis and subarachnoid hemorrhage. Presumably the arachnoid granulations, where CSF is absorbed, were "glopped up" by proteinacious debris from blood products or inflammatory material, leading to increased pressure, then ventriculomegaly then normal pressure again. The neurological status of these patients, after shunting, returned to normal.

In the era just before CT was invented, Hakim and Adams identified a group of patients who looked as though they had this syndrome but with no primary disorder to cause it. Their CSF pressures were normal. Their pneumonoencephalograms showed dilated ventricles and they improved after shunting. The triad of symptoms, dementia, gait disorder and bladder incontinence became the cardinal features, but, unfortunately this triad is seen in Alzheimer's disease, vascular dementia and dementia with Lewy bodies, that is, over 90% of dementing illnesses in the United States. Ventricular enlargement is common as well in the elderly and more so in the elderly demented.

Since there is no way to diagnose NPH without shunting, one can never know in advance whether the shunt will work. The literature suggests that dementia (what most doctors believe is the prime feature of this disorder) is a poor prognostic sign: that is, if the patient is more than slightly demented, he is unlikely to improve. This means, in plain English, that if the patient is demented, he is unlikely to respond to shunting, meaning that he doesn't have NPH. This makes sense, since one study on shunting obtained brain biopsies demonstrating that 78% had Alzheimer's disease. NPH has no defined pathology. Thus the triad of NPH is really a dyad and since the bladder doesn't get much better either, it is really a gait disorder, but such is the hold of a potential cure for a dementing illness that shunting demented patients has refused to die.

When I was a resident, my chief facetiously suggested that if shunting cured NPH for no apparent reason (after all, the pressure was "normal"), why not shunt everyone who was demented? And so, twenty years later the first report of shunting Alzheimer patients has been published. Armed with a rationale that holds some water, the surgeon can now offer a research treatment for a progressive, incurable and devastating disease and such a trial is in progress.

Unfortunately shunting isn't benign. Subdural hematomas, strokes and seizures are not uncommon side effects. In addition, the placebo effects are enormous. In his introduction to the first report on a placebo (sham surgery)-controlled trial of embryonic brain cells transplants for Parkinson's disease, the speaker noted, "The most impressive outcome of this trial was the magnitude and duration of the placebo response." Even sham spinal taps have produced dramatic improvements in NPH-suspected cases.

I do not know enough to give an informed opinion on shunting Alzheimer's patients. I do know that shunting is done too much on Alzheimer patients misdiagnosed as NPH; and the results, for the neurosurgeon, are often impressive, because follow-up stops by three months when most patients revert to their pre-shunt state.

I am concerned by this multi-center trial on Alzheimer's disease. I worry that this is snake oil with a bite. I do not know that I would refer a patient. The study would need to be carefully monitored, patients carefully followed for at least two years, costs would need to be borne by a funding agency and not the patient; sham surgery would be required; a strong safety monitoring committee would need to be empowered, with close supervision by independent neurologists and neurosurgeons. Then I might.

This is only one study of risky therapy for an incurable disease. The issue is an important one. In most neurological studies, the risks are quite small but not always. Destruction of bone marrow followed by bone marrow transplantation to treat the immune disorder of multiple sclerosis is another example of a heroic therapy. The ethical issue always boils down to whether the rationale justifies the riskiness. The decision generally rests with experts, who themselves may be split in their decision. However, doctors who refer patients for potentially harmful studies need to be familiar with the studies, even when the disease is otherwise incurable. "First do no harm," even if…

– Joseph H. Friedman, MD

If I Be Injured, Who Will Help?

Does society regard the historic sanctity of the patient-physician relationship as forever assured? Or might the collective needs of society sometimes transcend the historic covenants which allow patients to receive medical aid regardless of their status while also permitting them to speak their minds, disclose their innermost secrets to their physicians, secure in the knowledge that such disclosures will remain confined to the physician?

The Hippocratic Oath enjoins the physician to adhere to the imperative that entrusted confidences shall be regarded as holy secrets. Nowhere does the Oath suggest that there are extenuating circumstances allowing this promise to be breached. And in the opinion of many state boards of medical discipline, the breaking of this segment of the Oath is regarded as legitimate grounds for disciplinary action up to and including suspension of the license to practice medicine. While society and courts of law also recognize the historic trail and the merit of such a sacred promise, the Oath carries no explicit statutory protection. Society seems to say that the cloak of secrecy provided by the Hippocratic Oath [and its successor oaths] is a tradition to be applauded; unless, of course, specific protection of the citizenry requires that the promise be overridden.

The various medical oaths stipulate that the physician, if requested, shall also provide appropriate medical care regardless of the political or religious orientation of the patient. This too seems to fulfill what the medical profession and society in general expect of its licensed physicians. And yet there have been instances where the rendering of needed emergency medical care ran contrary to the wishes of society.

An injured male arrived by horseback to the Bryanstown, Maryland, home of the local physician. It was 4 AM on the predawn morning of April 15, 1865. The physician answered the knock upon his door, brought the man in and determined that a bone in his right leg had been fractured. The fracture was reduced, the injured leg properly bandaged, splinted and the patient given food and drink. Following a short rest upon the physician's couch, the man paid the requested fee and before noon was on his way to an unknown destination.

Within days the physician was arrested, brought before a nine-man military tribunal convened on May 10, 1865, by President Andrew Johnson; and in the company of other prisoners he was convicted of conspiracy to murder the President. The physician, Samuel Alexander Mudd, was sentenced to life imprisonment at Fort Jefferson, a remote, fever-infested military camp in the Dry Tortugas off the Florida keys.

Given this meager body of information, it would appear that this gentle country doctor, awakened from his well-earned slumbers, did no more than treat a distressed stranger in accordance with the precepts of his profession and was then unwittingly entangled in "the vengeful hysteria following the wartime assassination of a President."

The Hunter Commission, delegated the responsibility of determining the guilt or innocence of those involved in the assassination of Abraham Lincoln, saw otherwise. They were confronted with a young physician who had been born in 1833 on a plantation in southern Maryland, who openly advocated slavery and sympathized with the Confederate cause; was known to have shot a male slave who showed disrespect; had profited materially from slave labor; had been educated at Georgetown College and then the University of Maryland School of Medicine, receiving his medical degree in 1856; and then returned to his role as plantation owner while simultaneously practicing medicine. Further, there was strong evidence that he sympathized with the South and aided secret couriers in their transit across the battlefields of Virginia.

The Commission also produced evidence that Mudd had met secretly with John Wilkes Booth, Lincoln's assassin, on November 13, 1864, and again on December 23, where they were joined by two other men later shown to have been implicated in a broad conspiracy. A plot to kidnap Lincoln on March 17, 1865, initiated by Booth and his colleagues, was aborted when the President canceled his scheduled visit to a military hospital.

Lincoln loved the theater and on April 14, 1865 [just five days after Lee had surrendered to Grant at the Appomattox Court House] the President attended a performance of "Our American Cousin" at the Ford Theater in Washington. Booth shot Lincoln, leaped onto the stage [thus fracturing one of his leg bones], uttered something about death to tyrants and limped off, aided by David Herold.

The following morning at 7:22 AM Secretary of State William Seward was stabbed by another accomplice, Lewis Paine. The plot to assassinate the Vice President, however, failed.

At his trial, Mudd claimed that he did not recognize Booth despite compelling evidence that they had met on at least two prior occasions and that Booth had sent provisions to Mudd's home days before the successful assassination of the President.

In the summer of 1867, yellow fever erupted in Fort Jefferson; and prisoner Mudd assumed medical duties when the prison physician died of the fever. Because of his exemplary behavior, Mudd was given an unconditional pardon by President Johnson on February 8, 1869, after four years of imprisonment. He returned to his Maryland plantation and resumed his practice of medicine while also serving in the state legislature. On New Year's Day, 1883, Mudd developed pneumonia and pleurisy and died at age 49.

For over a century Mudd's many zealous descendants have fought vigorously to get his conviction overturned. [His Presidential pardon in 1869 did not include a reversal of his sentence.] Presidents Eisenhower, Carter and Reagan had all recommended that the conviction be nullified but the Army has remained intransigent, declaring that the documentation proving Mudd's complicity was irrefutable.

Let us assume that Mudd had indeed entered into a conspiracy with Booth, Surratt and others; and let us assume further that he was fully aware of the identity of his late-night patient. The question then arises: Was Mudd committing a crime when he rendered complete medical care to a person in need of such care?

– Stanley M. Aronson, MD, MPH

Sacred Space or Market Place: The Kern Case and The Meaning of Medicine as a Profession

Michael Fine, MD

In 1996, David Kern, MD, Assistant Professor of Medicine at Brown, Director of the Division of Occupational Medicine at Brown and the Occupational Health Clinic at Memorial Hospital of Rhode Island, submitted an abstract to the American Thoracic Society describing "flock workers' lung," a new occupational lung disease affecting workers in the nylon flocking industry.[1,2] That report provoked controversy, not because of the quality of the methodology, but because of alleged conflicts between Dr. Kern, the local factory that was the site of the research, Kern's employer, Memorial Hospital of Rhode Island, and Brown Medical School, where Kern held an academic appointment. [1,2]

The hospital alleged that Kern had violated confidentiality agreements signed when he undertook his research. Brown reflected on the implications of confidentiality agreements, and the economic and legal risks physicians take when they sign such agreements, risks which may be shared by the medical schools that appoint those researchers. Kern asserted that the hospital may have been pressured by the factory, and that Brown had failed its academic mission by refusing to defend his academic freedom.

At the end of the controversy, Memorial Hospital did not renew Dr. Kern's contract and closed its Occupational Health Clinic, ostensibly because it had been losing money for years. A new non-profit academic occupational and environmental health center, the Occupational and Environmental Health Center of Rhode Island, opened in 2000 to continue Kern's work, spearheaded by a coalition formed by the labor movement, the medical community, and the Rhode Island Affiliate of the American Lung Association.

The Kern case illuminates contradictions in the social theory of medicine. The social theory of medicine views medicine as a profession from the perspective of the profession's role in the economic, legal and cultural life of the society as a whole, a role which is rarely explicated and poorly understood.[1] From the vantage of social theory, the Kern Case pitted one party, which believed medicine was an owned industrial process, containing proprietary information protected by the laws which govern commerce, against another party, which believed medicine represented a covenant between a physician and the public, vesting physicians with unusual responsibilities and privileges that transcend those commerce-laws

In this paper, I will examine the social role of medicine, arguing that medicine as a profession is defined by the tension between its altruistic character, which uses oaths and covenants to insure that medicine is practiced for patients' benefit, and its marketplace role, where medicine is practiced wholly for the benefit of its practitioners. I will consider the role of advocacy in the context of the derived meaning of medicine as a profession, and then reflect on the Kern case in the context of the meaning of medicine as a profession.

Social Contracts and Professionalism

Medicine exists in a complex, constantly changing society, with different actors and interests, roles and relationships. Medical Professionalism is a matrix that includes the participants and their perspectives, interests, relationships, personal covenants, and contracts. The social contract between society, individuals, and the profession as a class is the glue that holds the matrix together.

Physicians, as a class, accept certain unusual responsibilities. In exchange, physicians, as a class, are awarded certain unusual privileges. Individual physicians are judged by the extent to which they discharge the accepted responsibilities, and evaluate their participation in the social contract by the extent to which they receive the promised privileges.[2,3,4,5]

The social contracts that govern medicine as a profession are complex, and, like most social contracts, are rarely explicit. A few parts of the social contract have made their way to paper as law or regulation — the laws governing licensure, the laws governing commerce, the regulations of specialty societies, the staff-admitting regulations of hospitals — but most of society's expectations of medicine are not written: expectations about professional behavior, about diagnostic accuracy and therapeutic effectiveness, about the intensity of disinterested advocacy, about the care of the poor, and about the role of physicians in identifying and advocating for social interventions that reduce the social cost of disease and injury.

Of course, social contracts like these are fluid, changing as society changes, with expectations that are renegotiated in the public arena by a contest of wills and the tussle of competing interests.

Some Definitions of Professionalism

In general, professions employ a body of knowledge or skill that they use for the good of others. The exercise of that knowledge or skill has inherent dangers to the community, because the quality of the service cannot be independently verified at the time it is performed, and is therefore not subject to the usual process by which the community evaluates a service or commodity—the process of comparison intrinsic to the marketplace. When a consumer buys a car, s/he can quickly evaluate it ("caveat emptor"); the same does not hold for the patient undergoing a triple bypass.

The professions struggle to convince the community that their practice is performed free of self-interest. The community struggles to convince itself that professionals practice without self interest, so it can use the valuable specialized knowledge or skill provided by professionals without fearing that what purports to be disinterest isn't a fiction - that the physician does not share the same unbridled self-interest of the used car dealer.

Consider the Oxford English Dictionary definition of Profession (III.6): "The occupation one professes to be skilled in and follow. A. A vocation in which a professed knowledge of some department of learning or science is used in its application to the affairs of others or in the practice of an art founded upon it. Applied specifically to the three learned professions of divinity, law and medicine."[6] Paul Starr also recognized the role of specialized knowledge and its application to the affairs of others, but included the need of professions to portray themselves as free of self-interest. "A profession ... is an occupation that regulates itself through systematic, required training and collegial discipline; that has a base in technical, specialized knowledge; and that has a service rather than a profit orientation, enshrined in its code of ethics."[7]

Expectations about professional behavior derive from professionals' desire to convince others that they come from a social class which allows them to attain the requisite education and training, and from the professions' need to appear devoid of self interest. These expectations, which include expectations about dress and speech, extend to personal integrity.

Codes of ethics, oaths and covenants percolate through the professions, as they struggle to emphasize the dis-interested character of their practice. It is these codes, oaths and covenants, and the attempt to establish a space devoid of self-interest that gives rise to the notion of a Sacred Space, which will be discussed in detail below.

Medicine in the Marketplace

Even though the specialized knowledge and skill of medicine makes its quality difficult to evaluate by the usual comparative judgments of the marketplace, and even though professionals struggle to appear devoid of self-interest to maintain their legitimacy, physicians do function in the marketplace, as they develop a product and merchandise that product, which is their skill, knowledge, and expertise. Indeed, in a consumer society, the rules of the marketplace dominate social intercourse, and the professions must live by those rules, even when the rules conflict with professional ethics.

Economists view the professions differently from the way the professions see themselves, focusing on the professions' monopoly of expertise, and their to stand behind the cloak of disinterested advocacy while performing like self-interested capitalists. "Skeptical economists view professions as state-protected cartels whose primary purpose is to raise barriers to entry through education and certification requirements, to restrict competition from outsiders, and ultimately extract monopoly rents by combining to fix prices and the terms of service to clients."[8]

The history of professionalism in the United States documents the legislative and judicial acceptance of the economists' view—a view occasionally justified by the actions of physicians. At the turn of the century, state legislatures began banning the fee schedules set by state medical societies to prevent fee-gouging, for fear that these fees were a thinly disguised method of setting minimum prices, and could discourage contract, or lodge, practice, a forerunner of group health purchasing cooperatives. In 1937, the officers of the American Medical Association and many local medical associations were indicted for conspiracy in restraint of trade because of their attempt to destroy the Group Health Association, a non-profit health cooperative organized by employees of the Federal Home Loan Bank.[9] In the 1960s and 1970s, a number of legislative actions and judicial decisions permitted the hitherto banned advertising by professionals, under the theory that prohibition of advertising stifled competition, and the professions were businesses competing in the marketplace to sell services, not institutions whose primary role was disinterested advocacy or altruism. In 1975, in *Goldfarb v. Virginia State Bar*, a young attorney contested the right of the Virginia Bar Association to set fees for title searches. The Court ruled that the professions were subject to the Sherman Antitrust Act.[10] In 1976 the courts ended an experiment in health services delivery at Hunterdon County Medical Center, in Flemington, New Jersey. Hunterdon County Medical Center was a population-based health services delivery system that restricted the economic and clinical activity of specialist physicians while supporting the delivery of primary care to a population of 75,000 people. The Hunterdon experiment, which floundered under the weight of antitrust actions (as well as internal financial and administrative challenges), established once and for all that competition would take precedent over the public health in the eyes of the courts, at least as far as the delivery of health care services was concerned.[11]

Most legal precedent in the United States has judged physicians to be economic competitors who must not collude or conspire to fix prices. As such, there is little legal support for physicians contravening the rules of the marketplace to act in the interests of the public health. While both case law and legislation support contravening the rights of individuals when the public health is at stake, there is no such legal justification for overturning the law of the state governing contracts or antitrust statues if the public health is at risk. While there is precedent for protecting the confidentiality of the patient-physician relationship, for example, that confidentiality is not protected from discovery in a civil or criminal action (and so is less protected than attorney client privilege), and there is no precedent for invalidating confidentiality agreements if an individual physicians judges that there is risk to the public health if such agreements are respected. While there is precedent for the override of individual rights to privacy or freedom from confinement in cases of danger to self or others from infectious diseases or behavioral disorders, there is

no precedent for overriding the antitrust statutes to allow the integration of individual practices into a single health care system, and better serve the public health, and there is no precedent for overriding property rights, when a business or industry threatens the public health with its products (in the case of television producers, and tobacco, alcohol, and handgun manufacturers), or undermines the public health by its prices (which may be the case of pharmaceuticals).

Although, as George Soros has noted, the marketplace rules may not be the best way for society to make decisions about the public interest, or to make ethical determinations for resource distribution, those rules govern how health care is delivered by physicians. [12] Although disinterested altruism may be central to professionalism, professionalism has no special claim to exclusion from marketplace rules on the basis of such altruism, at least presently.

On the one hand, disinterested altruism is necessary to establish the integrity of medicine as a profession. On the other hand, the law of the marketplace does not recognized disinterested altruism. What is a doctor to do?

The Sacred Space of Medicine

For many years, physicians were paid by their patients, and both patients and physicians relied on the appearance of disinterested advocacy to justify that payment. Patients needed to believe that physicians were devoid of self-interest in order to attach value to physician services, and physicians needed to appear devoid of self-interest so that patients would patronize them, and so that they could afford to practice.

Early in the history of the professions, the deity was often invoked as the guarantor of relationships between people when no other governing principal could be found. To take an oath was to invite the deity to guarantee what could not be independently verified. A professional's oath promised the public that the physician would practice in the absence of self-interest.

More recently, the state has become the guarantor of relationships between people, and the direct economic tie between patients and physicians has been lost, as patients rarely pay physicians themselves, and rely on intermediaries — the state, and HMO, or a hospital - to guarantee that the physicians' advice is devoid of self-interest.

But we still use oaths and covenants. Although they are a carryover from the time when religious authority was important to guarantee or supervise the relationship between people, the presence of oaths and covenants still serves to remind patients and physicians alike that medicine is to be practiced in a way that is not colored by self-interest. In addition, oaths and covenants evoke the sense of "sacred" supervision of professional obligations, the sense that these obligations are supervised from "on high."

…the Kern Case pitted one party, which believed medicine was an owned industrial process, containing proprietary information protected by the laws which govern commerce, against another party, which believed medicine represented a covenant between a physician and the public…

If oaths and covenants evoke the *sense* of the sacred, then the disinterested advocacy in matters of life and death helps reinforce the *perception* of the sacred, which exists as an undertow in the practice of medicine. That is, the professional relationship is set apart from the usual relationships of the marketplace, which are self-interested as a matter of course. The existence of disinterested advocacy suggests a sacred quality to the patient-physician relationship, as selfless advocacy evokes concepts in many religious theologies. [13] In addition, the medical profession appears to have power over life and death, a power once ascribed only to the deity, a power that brings a sacred tone to the patient-physician relationship. Finally, because physicians access knowledge and skill obtained by the collective action of many people over time, physicians appear to be using a power greater than the power of a single individual, power which reinforces the presence of the sacred in the practice of medicine as a profession.

Of course, portraying medicine as a sacred calling in the early years of the twenty-first century is almost laughable, because common experience with the medical profession is anything but sacred. Materialism and consumer capitalism have changed values, so physicians, along with the rest of society, focus more on income, less on relationship. Physicians' income depends on their production of procedures, which are purchased independently of relationships with patients. The focus on specialty medicine has limited relationships to that between a physician and a body-part —an organ system, gender, age bracket or just a picture or piece of tissue. With nformation about the preservation of health widely available, there is less dependence on physicians' knowledge and skill. Society has projected a regulatory role into the patient-physican relationship, asserting a duty to supervise this once-private relationship. [14] And physicians have allowed intrusions into the patient physician relationship, which exploit the existence of that relationship for personal gain. Though these relationships, which occur with pharmaceutical or health insurance companies, may not always present a *frank* conflict of interest, the intrusion of *some* self-interest into the sacred space of the patient-physician relationship devalues its sacred nature.

Still, there are elements of physicians' behavior that keep the sense of the sacred alive in the hearts and minds of patients and physicians alike. Many physicians practice with integrity, and put patient care before other considerations, keeping self-interest at arms

length. Many share in the care of the poor, the kind of disinterested altruism that makes professionalism real in the public eye, and justifies the exclusion from the rules of the marketplace that professionalism suggests (but the law does not allow). And many physicians advocate for interventions that reduce the social cost of disease and injury, using their expertise to advocate for the common good.

The Special Case of Advocacy

The responsibility of the individual physician for the health of her/his patient is well defined. The responsibility of the individual physician for the public health is less well defined. The responsibility of physicians as a class for the public health is not well defined at all, and intersects with responsibilities of other bodies and institutions (the state, hospitals, medical schools, other organizations, professions and professional organizations).

Individual physician and physician organization advocacy of social interventions that reduce the social cost of disease and injury is not a clearly articulated part of the job description. But disinterested advocacy of social interventions that may reduce the social cost of disease and injury is advocacy for the common good. Advocacy of the common good may well be its own reward, as that which strengthens all also strengthens one, and there is inherent satisfaction in creating social justice, but such advocacy is also in the interest of the profession, as it builds and strengthens the public perception of a Sacred Space of Medicine as a Profession.

The Kern Case, Redux

A wise observer described the Kern Case thus: "At best, this is a story of incorrect assumptions and mutual misunderstandings by well-intentioned persons on all sides..."[15] That analysis suggests that the incorrect assumptions exist at the level of the social theory of medicine, and are inherent in the social contract that gives medical professionalism its legitimacy. That is, the social meaning of medicine as a profession has at least one fundamental contradiction, the contradiction between the sacred space of medicine, the space in which physicians are constrained, by the ethics of their profession, to act from disinterested advocacy; and the rules of the marketplace, which force physicians to protect and advocate for their own self-interests, and which are the governing law in a consumer capitalist society.

This contradiction played itself out in the following way. Dr. Kern, acting from the perspective of disinterested advocacy, proceeded as if disinterested advocacy was the governing principle of society itself. The other actors, understanding, from the perspective of the State, that medicine can be an owned industrial process, protected what they saw as their property, actions legitimized by case-law and legal precedent.

But all participants lost sight of the opportunity inherent in this conflict. It was necessary to explicate the conflict in order to understand it; thus there is reason to be disappointed that the academic institution involved did not seize the opportunity for explication, so that the participants, its students and faculty, and the public could have learned from the process. Instead, lives were disrupted, and reputations sallied.

Once explicated, there remains an opportunity to resolve the theoretical part of the conflict by attending to overarching principles that unite the interests of the parties to the conflict, and in the process sketch the outlines of a social theory of medicine that serves society as a whole.

There is, in fact, social utility to the disinterested advocacy that the profession of medicine sees as critical to its ethical practice. There is also real concern that the cloak of professionalism can be exploited in the self-interest of physicians, other professionals, and professional organizations.

A social theory of medicine, then, must account for both the sacred space of medicine, providing social space for practice with disinterested advocacy, and account for society's need to assure itself that what claims to be disinterested advocacy is in fact, disinterested. The health care delivery experiments of the last forty years (HMOs, PPO, IPAs, PSOs et al.) test the hypothesis that the marketplace and the laws protecting commerce are rational and effective checks and balances to the claim of disinterested advocacy. It is not clear how effective the marketplace is in that role, but an elegant alternative to the marketplace for that role has not yet been clearly articulated. My own sense is that the elegant alternative to the marketplace as an effective check and balance for disinterested advocacy gone astray is local government itself, which could enter the marketplace and purchase needed services directly, assuring the absence of self-interest by controlling the volume and extent of services purchased, and is still fundamentally democratic in its function. But the ability of government to be either rational or effective is closely questioned by most, and there is certainly no societal consensus on the ability of government to function in this role.

In any case, the problem posed by the Kern case to the people of Rhode Island [the loss of an independent academic medical occupational and environmental health consulting service] was solved by old-fashioned community organizing. Key participants — the labor unions, which are still powerful in Rhode Island, the medical community, and a disease advocacy group, the Rhode Island Affiliate of the American Lung Association - united to muster support for a new occupational and environmental health consulting service, one with multiple streams of funding so that it could not be easily silenced. That service, the Occupational and Environmental Health Center of Rhode Island, opened in late 2000.

In this way, a conflict caused by the ambiguity and contradiction in the meaning of medicine as a profession was resolved by building a coalition between the medical profession and those parts of society that believed its interests are best served by preserving the independence and integrity of medicine as a profession.

But the inability to resolve this conflict any other way exposes social uncertainty about whether there is a common good, about who is to determine what comprises that common good; about whether advocating for the common good should be a shield against

the demands of the market, and whether marketplace values or public health values are to be the dominant principles determining the behavior of physicians.

Conclusion

The meaning of professionalism is ambiguous, beset by contradiction. At its best, medicine as a profession denotes a sacred space, where physicians trade self-interest for patient and public health advocacy. But many factors - consumer capitalism, mass culture, materialism, legal process, specialization and self-interest - have eroded that space. The law under which medicine operates is the law of the State protecting commerce, in place to protect society from those physicians who would hide behind the cloak of disinterested advocacy while they exploit their monopoly on knowledge and skill to serve themselves.

The Kern case suggests that a redefinition of the social contract of medicine is possible, but hints that such a redefinition requires an exercise in community organizing, so that all interested parties create a new way to both protect disinterested advocacy, and guarantee that the claim to disinterested advocacy is not unfairly exploited.

Acknowledgement

This paper was written with the support of the Open Society Institute/ Medicine as a Profession Fellowship. The author is solely responsible for its content. I am indebted to the fellowship board and the other OSI/MAP fellows for their commitment, ideas, help and encouragement. I am indebted as well to George Nee, Secretary-Treasurer of the Rhode Island AFL-CIO, Margaret Kane, Executive Director of the Rhode Island Affiliate of the American Lung Association, Jim Celenza, Executive Director of the Rhode Island Committee on Occupational Health and Safety, and other members of the board of the Occupational and Environmental Health Center of Rhode Island, for their help as this paper was being written and the Center was being created. In my view, we are all indebted to David Kern, MD, who put his career on the line in defense of the integrity of medicine as a profession.

References

1. Kern DG, Crausman RS, Durand KT, Nayer A, Kuhn C 3d. Flock worker's lung: Chronic interstitial lung disease in the nylon flocking industry. *Ann Intern Med* 1998;129:261-72.
2. Chronic interstitial lung disease in nylon flocking industry workers-Rhode Island, 1992-1996. *MMWR* 1997;38:897-901.
3. Schuchman M. Secrecy in science: the flock worker's lung investigation. *Ann Intern Med* 1998;129:341-4.
4. Davidoff F. New disease, old story [Editorial]. *Ann Intern Med* 1998;129:327-8.
5. Lemert C. Social theory – its uses and pleasures. In Lemert C. *Social Theory – The Multicultural and Classic Readings* (Boulder, Colorado: Westview Press, 1999), 1-20
6. Ozar D. Professional and professional ethics. In Reich W. *Encyclopedia of Bioethics.* Simon & Schuster McMillan, New York, 1995: 2103-12.
7. Stevens R. Old, new, and déjà vu. Briefing Book of the Medicine As A Profession/ Soros Advocacy Fellows Conference, November 2000.
8. Gordon R. Professionals and Professionalism: An Overview. Briefing Book of the Medicine As A Profession/ Soros Advocacy Fellows Conference, November 2000.
9. Daniel N. Professional values and their institutional context. Briefing Book of the Medicine As A Profession/ Soros Advocacy Fellows Conference, November 2000.
10. *The Oxford English Dictionary*. New York: Oxford University Press, 1971: 1427.
11. Starr P. *The Social Transformation of American Medicine*. New York: Basic Books, 1984:15.
12. Gordon R. Professionals and Professionalism: An Overview. Briefing Book of the Medicine As A Profession/ Soros Advocacy Fellows Conference, November 2000.
13. Starr, *The Social Transformation of American Medicine*, 299-310.
14. *Goldfarb v. Virginia State Bar.* 1975. 421 U.S.773.
15. Wescott L. Hunterdon: The rise and fall of a medical Camelot. *NEJM* 1979;300: 952-9.
16. Soros G. *Open Society. [Reforming Global Capitalism.]* New York: Public Affairs, 2000.
17. Eliade M. *The Scared and the Profane: the Nature of Religion.* New York: Harcourt Brace and Company, 1959: 20-65.
18. Rothman D. *Strangers at the Bedside.* New York, Basic Books, 1991.
19. Davidoff F. New disease, old story [Editorial]. *Ann Intern Med* 1998;129:327-8.

Michael Fine, MD, is Senior Managing Partner for Strategic Planning, Hillside Avenue Family and Community Medicine, and Physician-in Chief, Department of Family and Community Medicine, Rhode Island Hospital.

Correspondence:
Michael Fine, MD
Hillside Avenue Family and Community Medicine
349 Gleaner Chapel Road
North Scituate, Rhode Island 02857
phone: (401) 934 3545
fax: (401) 934 0671
e-mail: m1fine@aol.com

Lifestyle Differences Between Surgical Attendings and Residents at Rhode Island Hospital

Sutchin R. Patel, Victor Zaydfudim, Shirine M. Nassery, Nasser I. Khan, Dennis K. Jorgensen, Michael G. Garvey, Nina Mirchandani, Alex Derevianko MD, Anthony J. Moulton MD

Recently many papers have been written regarding the attitudes and concerns of surgical residents.[1,2,3] The number of graduating medical students choosing to match in general surgery experienced a slight decline in 2002, and there is concern for the future direction of surgical education.[4, 5, 6] While it is important to understand why surgical residents are dissatisfied, it is equally important to compare their satisfaction levels against that of their attendings. In our study we surveyed surgical residents and attendings at the Rhode Island Hospital in order to distinguish the differences between the various aspects of the surgical lifestyle in these two populations.

Methods

A 33-item questionnaire was administered to attending physicians and residents at Rhode Island Hospital during the Thirty-first Annual J. Murray Beardsley Surgeon-in-Chief Surgical Grand Rounds on May 30, 2001. The questionnaire was limited to resident and attending surgeons within the Department of Surgery (Rhode Island Hospital). Seventy questionnaires were distributed; 41 total questionnaires (59%) were collected, with 18 responses from attending physicians and 23 from residents.

The questionnaire was limited to a single page with concise, objective-type questions. Answer choices were limited to yes/no, modified Likert scale from 1 [very dissatisfied] to 9 [very satisfied], or multiple-choice selections. The size of the questionnaire restricted the quantity and depth of inquiry, but the questions allowed for the compilation of data on multiple aspects of a surgeon's life. These categories included: General Profile, Work/Sleep/Call Hours, Satisfaction with Aspects of the Surgical Lifestyle and Health/Stress. The small sample size of our study restricted the analysis of our data and limited the conclusions we could draw to only the surgeons from Rhode Island Hospital.

Results

I. Demographics of Survey Respondents

Twenty-three residents and 18 attending physicians completed the survey. Among the residents, there was an even distribution of males and females (Table 1). The male-to-female ratio among attendings was not as evenly distributed (13 males, 5 female attendings). The average ages of male and female residents were similar (29.8 years for males and 28.8 years for females), though there was a significant difference in the average age between male and female attendings (60.6 years for males and 40.8 years old for females). Thirty percent of the residents compared to 72% of the attendings were married.

II. Work, Call and Sleep

On average, residents slept approximately 10 hours less per week than attendings (Table 2); interns reported the least amount of sleep (33.1 hours of sleep per week on average). The gap between the average work hours per week for residents compared to attendings was significant (107.8 versus 59.7 hours per week respectively). Again, interns were working the greatest number of hours (115 per week). As such, attending physicians had more hours to spend with their family or friends (38% of attendings spent 21 hours or more per week with their family/friends compared to 9% of residents). While, on average, residents were on call approximately the same number of days per week as attendings (3.0 and 2.7 days per week respectively) the amount of sleep that residents obtained while they were on call was significantly less than

Table 1. Demographics of Questionnaire Respondents

	Surgery Residents	Surgery Attendings
Total	23	18
Males	11	13
Females	12	5
Average Age	29.3	55.1

Table 2. Work, Call and Sleep

	Surgery Residents	Surgery Attendings
Total Hours of Work per Week	107.8 hours/week	59.7 hours/week
Hours of Work per Week for PGY-1 (Interns)	115.0 hours/week (8)	
Hours of Work per Week for PGY-2 to PGY-5	104.0 hours/week (15)	
Total Hours of Sleep per Week	35.6 hours/week	45.8 hours/week
Hours of Sleep per Week for PGY-1 (Interns)	33.1 hours/week (8)	
Hours of Sleep per Week for PGY-2 to PGY-5	36.8 hours/week (15)	
Days on Call per Week	3.0 days/week	2.7 days/week
Average Hours of Sleep on Call	2.4 hours	5.6 hours
Has Lack of Sleep Ever Affected Your Performance in the Operating Room?	65 % say Yes (15)	6 % say Yes (1)

Numerical values above are listed as averages. Number of questionnaire responses are in ( ).

Table 3. Satisfaction Within the Surgical Field

Satisfaction with:	Surgery Residents	Surgery Attendings
Overall surgical lifestyle	4.14 (2.43)	6.67 (2.05)
Occupation as a surgeon	5.86 (2.34)	7.39 (2.06)
Salary	2.86 (1.69)	3.65 (2.23)
Responsibility given for patient care	5.87 (2.00)	8.00 (1.79)
Treatment by superiors	5.00 (2.15)	6.28 (2.35)

Scores reported are as raw means (and standard deviations), on a 9-point modified Likert scale, with 1 = Very Dissatisfied, 5 = Neither Satisfied nor Dissatisfied and 9 = Very Satisfied.

attendings (2.4 and 5.6 hours respectively). Moreover, 65% of residents compared to 6% of attendings claimed that lack of sleep had affected their performance in the operating room on at least one occasion.

When asked to choose what was most important in their life right now: their Surgical Career, Family/Friends, Health or Salary; 52% of residents and 63% of attendings said that their family and friends were the most important. Twenty-six percent of residents and 11% of attendings ranked their surgical career most important. Twenty-two percent of residents and 19% of attendings ranked their health as most important. Only 6% of attendings and none of the residents listed "salary" as the most important aspect of their life.

III. Satisfaction within the Surgical Field

Surgical attendings reported greater satisfaction with their jobs as surgeons and their surgical lifestyle compared to residents (Table 3), with no difference in the level of satisfaction between male and female physicians (data not shown). Both attendings and residents were somewhat dissatisfied with their salaries. Attendings were very satisfied with the responsibility they had for patient care (8.00 (SD=1.79)); residents were less satisfied (5.87 (SD=2.00)). Both attendings and residents were satisfied with treatment by their superiors, with no significant differences between male and female surgeons.

IV. Effects of the Surgical Lifestyle on Stress and Health

Both groups reported a moderate amount of stress from their surgical career (residents 6.78 (SD=1.41); attendings: 6.83 (SD=2.09). The differences in work schedule had a greater impact on the health of a resident than the health of an attending. The majority of attending physicians and residents did not experience substantial weight changes over the past year: 57% of residents and 56% of attendings gained or lost 5 pounds or less. Regardless of their weight change, 74% of residents and 18% of attendings attributed their change in weight over the past year to their schedule as a surgeon.

…65% of residents compared to 6% of attendings claimed that lack of sleep had affected their performance in the operating room on at least one occasion.

Discussion

Surgery is marked by long hours and demanding call schedules. Our study confirms that surgical residents suffer from some sleep deprivation, with little improvement through the course of their postgraduate medical training and that residents felt that sleep deprivation had affected their performance in the operating room. Though this finding does not support a definitive correlation between sleep deprivation and surgical outcome, it does suggest that work hours and conditions must be evaluated in the context of postgraduate surgical education.[7,8,9,10]

The residents in our study ranked their satisfaction with their overall surgical lifestyle significantly lower than that of attendings. When acknowledging that surgical residents work more hours per week, sleep less, and earn less money, it is not surprising that they are less satisfied.[1,2] Other plausible contributors to resident satisfaction include contact with attendings and lack of responsibility for patient care. In our study, surgical residents, while admitting to less sleep and longer work hours than attendings, did not report dissatisfaction with the treatment by attending physicians or the level of responsibility for patients. Some of the differences in the overall satisfaction between attendings and residents may be related to their age and the different stage each population is at in their professional career and family life. A common source of dissatisfaction for residents and attendings was their salary. It is difficult to compare a resident's salary to that of an attending surgeon because residents are still in training. Furthermore, a physician's salary must take into account the length of training, the number of hours worked, the type of work performed and the cost of malpractice insurance.[11,12] We did not look at any of these factors and thus cannot comment as to the causes of surgeons' dissatisfaction with their salary.

Surgical attendings and residents consider themselves to be under the same level of stress, yet the quality and source of this stress seems different. Residents work long hours, sleep less and suffer stress related to the pressures of successfully completing their training.[13,14,15] Attendings, on the other hand, likely suffer stress more directly related to professional duties.[2] Patient care and the conduct of residents is ultimately the responsibility of the attending, thus it is not surprising to find that attendings consider themselves to be under a similar level of stress as residents.

Our study shows that surgical residents were less satisfied with their overall lifestyle than their attendings. On average, residents worked twice as many hours, reported higher levels of job-related stress and enjoyed less time

with family and friends. The overall dissatisfaction with the surgical lifestyle of residents, however, does not seem to lie in their occupation as a surgeon, their responsibility for patients or in their treatment by superiors. With the exception of the overall surgical lifestyle, residents and attendings in general were satisfied and dissatisfied with the same aspects of the field of surgery, albeit to a different degree.

Special thanks to our Affinity Group Fellows, Amit Joshi, Joshua Landes and Mark Zanfrillo for their help and guidance, and to Suzanne Smeaton for her assistance in collecting our surgical questionnaires.

References

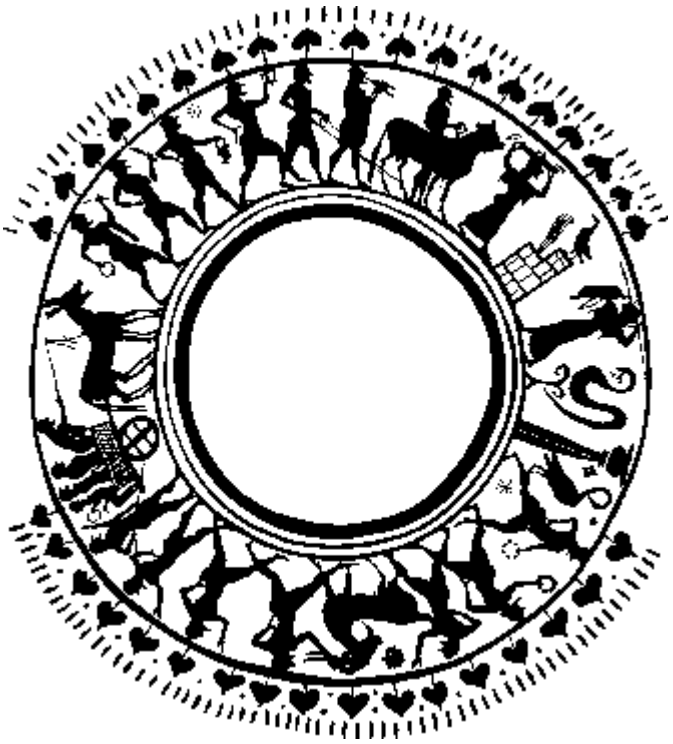
1. Daughtery SR, Baldwin DC, Rowly B. Learning, satisfaction and mistreatment during medical internship: a national survey of working conditions. *JAMA* 1998; 279:1194-9.
2. Gabram SG, Hoenig J, Schroeder JW, et al. What are the primary concerns of recently graduated surgeons and how do they differ from those of the residency training years? *Arch Surg* 2001;136:1109-14.
3. Gabram SG, Allen LW, Deckers PJ. Surgical residents in the 1990s: issues and concerns for men and women. *Arch Surg* 1995;130:24-8.
4. Gawande AA. Creating the educated surgeon in the 21st century. *Am J Surg* 2001;181:551-6.
5. Bland K, Isaacs G. Contemporary trends in student selection of medical specialties: the potential impact on general surgery. *Arch Surg* 2002;137:259-67.
6. Spitz L, Kiely EM, Peirro A, et al. Decline in surgical training. *Lancet* 2002;359:83.
7. Barden CB, Specht MC, McCarter MD, et al. Effects of limited work hours on surgical training. *J Am Coll Surg* 2002;195:531-8.
8. Veasey S, Rosen R, Barzansky B, et al. Sleep loss and fatigue in residency training: a reappraisal. *JAMA* 2002; 288:1116-24.
9. Lamberg L. Long hours, little sleep: bad medicine for physicians-in-training? *JAMA* 2002; 287:303-6.
10. Sawyer RG, Tribble CG, Newberg DS, et al. Intern call schedules and their relationship to sleep, operating room participation, stress and satisfaction. *Surgery* 1999;126:337-42.
11. Sturm R. The impact of practice setting and financial incentives on career satisfaction and perceived practice limitations among surgeons. *Am J Surg* 2002;183:222-5.
12. Woodworth PA, Frederic CC, Helmer SD. Debt and other influences on career choices among surgical and primary care residents in a community-based hospital system. *Am J Surg* 2000; 180:570-6.
13. Bunch WH, Dvonch VM, Storr CL, et al. The stresses of surgical residency. *J Surg Res* 1992; 53:268-71.
14. Aufses AH, Slater GI, Hollier LH. The nature and fate of categorical surgical residents who "drop out." *Am J Surg* 1998; 175:236-239
15. Levy RE. Sources of stress for residents and recommendations for programs to assist them. *Acad Med* 2001;76:142-50.

Sutchin R. Patel, Victor Zaydfudim, Shirine M. Nassery, Nasser I. Khan, Dennis K. Jorgensen, Michael G. Garvey, Nina Mirchandani are Brown Medical School students who conducted this study as part of the Affinity Group Program.

Alex Derevianko, MD, is a general surgeon from the Ukraine.

Anthony Moulton, MD, is Clinical Professor of Surgery (Cardiothoracic Surgery), Brown Medical School, and Chief of Thoracic Surgery at the Providence VA Medical Center.

Correspondence:
Sutchin Patel
Brown University
Box G-8115
Providence, RI 02912
phone: (401) 421-2508
e-mail: Sutchin_Patel@Brown.edu



The Identification and Management of Hereditary Breast and Ovarian Cancer

Jennifer L. Scalia, MS, and Robert D. Legare, MD

The last few years' advances in the field of hereditary breast cancer genetics have altered clinical practice. It is generally accepted that the process of carcinogenesis is caused by the serial acquisition of multiple genetic mutations within one or more cells, ultimately leading to the malignant phenotype. The cause of this initial genetic error differentiates hereditary cancers from sporadic cancers. Only 5-10% of cancers are primarily caused by genetic mutations inherited in the germline. However, due to the high penetrance and autosomal dominant nature of inherited genetic mutations, hereditary cancer families have been identified, leading to the rapid localization and cloning of several predisposing cancer genes.

Although only 5-10% of breast and ovarian cancers are hereditary,[1] this accounts for approximately 20,350 breast cancers and 2,330 ovarian cancers diagnosed in the United States. Hereditary mutations influence cancer risk but are not one hundred percent penetrant. The subsequent environmental and/or genetic factors influencing the cell's progressive malignant development are largely unknown. Many patients are believed to be at increased risk for cancer. Physicians and healthcare specialists often refer these patients for risk assessment and recommendations for preventive risk-reducing strategies.

Cancer Genetic Counseling

Cancer genetic counselors often take on the primary role in relaying new medical information to patients, assessing cancer risk, and offering options for cancer genetic testing and medical management strategies. Because of rapid advances in cancer genetics, the cancer genetic counselor has become essential to a comprehensive multidisciplinary cancer program.[2]

Breast and Ovarian Cancer Syndrome (BOCS)

BOCS is largely accounted for by deleterious BRCA1 or BRCA2 gene mutations. Those genes normally function as tumor suppressors; however, when a deleterious BRCA germline mutation is present, the risk of cancer increases, causing the BOCS. Approximately 16% of mutations in families with hereditary breast cancer are not found by standard BRCA1 and BRCA2 DNA sequencing and are believed to be caused by either an undiscovered gene or mutations within BRCA1 and BRCA2 that are missed by the current testing methods.[3] Additionally, less common hereditary breast cancer syndromes can be recognized by associated clinical characteristics and confirmed with molecular testing; e.g., Cowden syndrome, Bloom syndrome, Peutz-Jeghers syndrome, Werner syndrome, Xeroderma Pigmentosum, Ataxia-Telangiectasia and Li-Fraumeni syndrome. Similarly, there are other syndromes associated with hereditary epithelial ovarian cancer such as **Hereditary Nonpolyposis Colon Cancer (HNPCC)**.

Guidelines from the **American Society of Clinical Oncology (ASCO)** recommend that three conditions be met before offering genetic testing; 1) a likelihood >10% of a positive test; 2) genetic test results that can be adequately interpreted and 3) results that will influence medical management.[4] Because many more families have undergone BRCA gene testing, more accurate carrier assessments can now be constructed; and the ASCO is revising their guidelines, slated for publication in 2005.

BRCA1 and BRCA2 Cancer-Predisposing Mutation Probability

Overall, studies indicate that a BRCA1 or BRCA2 cancer-predisposing gene mutation is more likely present if family history includes: breast cancer diagnosed before age 50 years, bilateral breast cancer, ovarian cancer, Ashkenazi Jewish ancestry, or the occurrence of both breast cancer and ovarian cancer in the same woman.[5-9] [The "family history" depends on the size of the family, the age of cancer diagnosis and type of primary cancer — any unusal pattern, young onset cancers, multiple primaries, syndromic cancer constellation in any family members would be of concern, including uncles and cousins.] Because each study evaluated BRCA carrier risk from a selected population, it is often best to determine an individual's/family's carrier probability according to the data set that is clinically most similar or applicable.

A software model (BRCAPRO) calculating the probability for the presence of a cancer-predisposing BRCA1 or BRCA2 mutation has been developed. This calculation is based on observations in referral populations in which the majority of women tested were affected with breast or ovarian cancer.[8,10] BRCAPRO adjusts risk according to bayesian theorem, however, it may over or under estimate carrier risk depending on the familial characteristics.

Due to vast familial cancer variability, it is recommended that hereditary cancer risk assessment be performed by experienced clinicians to assure the most accurate assessment, in accordance with the most applicable data set(s).

Cancer Risks Associated with a BRCA Deleterious Mutation

The majority of **hereditary breast and ovarian cancer (HBOC)** families have been linked to gene mutations affecting the normal function of the BRCA1 or BRCA 2 protein. Women carrying a deleterious BRCA gene mutation have a 56% to 85% cumulative lifetime risk of developing breast cancer up to age 70,[11,12]

and a 27%[13] to 44%[14] cumulative lifetime risk of ovarian cancer. Additionally, male BRCA heterozygotes carry an approximate 6% male breast cancer risk by age 70, and a 3 to 4 fold increase relative risk of prostate cancer by the age of 80.[15] Although low, the risk of pancreatic cancers in men and women carrying a BRCA2 mutation was estimated to be approximately 2 to 3% by age 80.[15] There remains suggestive (but not substantiated) evidence of additional cancer risks influenced by an absent BRCA protein, such as colorectal cancer and melanoma.

Management of Risk in Carriers of BRCA Gene Mutations

Management of individuals found positive for hereditary breast and ovarian cancer susceptibility syndrome includes discussion regarding cancer screening protocols, options for chemoprevention, as well as prophylactic surgery. Although a number of interventions have been postulated to reduce the morbidity and mortality from breast cancer in women confirmed to carry a BRCA1 or BRCA2 cancer-predisposing mutation,[16] data are evolving to substantiate these claims. Nevertheless, several strategies have been prospectively studied and proven to detect early staged cancers in BRCA heterozygotes;[17] e.g., cancer screening, prophylactic mastectomy and/or oophorectomy, and chemoprevention.

Cancer Screening

Recommendations for cancer screening of individuals with a BRCA1 or BRCA2 cancer-predisposing mutation have been made by a task force convened by the **Cancer Genetics Studies Consortium (CGSC)**, an NIH-sponsored consortium of researchers assessing the ethical, legal, and social implications of genetic testing for cancer risk.[16] The CGSC recommendations were based on presumed benefit and may change as new evidence becomes available; therefore, patients must be counseled regarding the limited knowledge about strategies to reduce risk. Furthermore, patient preference should be taken into account for follow-up decisions. Recommendations similar to those of the CGSC are practiced in 16 European family cancer centers.[18]

Breast Cancer Screening

The three-pronged breast cancer screening regimen is based on data from families with cancer-predisposing BRCA1 or BRCA2 mutations, addressing the elevated breast cancer risk beginning in a woman's late 20s or early 30s.[16] Individuals predisposed to an inherited breast cancer risk are recommended to consider:

* Monthly breast self-examination starting at age 18 to 21.
* Annual or semi-annual clinical breast examination beginning at age 25-35 years
* Annual mammography beginning at age 25-35 years

There is evidence that BRCA1 and BRCA2 gene products may be necessary to assist with DNA damage repair caused by radiation.[19] Despite concern regarding the repeated low doses of radiation exposure in BRCA positive women, the **Human Genome Research Institute's (NHGRI)** task force states that this hypothetical risk would likely be outweighed by mammography's benefit on early cancer detection.[20]

Other breast imaging modalities are being studied in germline BRCA carriers. Early studies report that breast MRI may detect cancers in mutation carriers that were occult on both mammogram and clinical breast exam and, thus, a potentially promising screening devise for hereditary breast cancer families.[21]

Men with BRCA mutations may also be at increased risk for breast cancer, and evaluation of any breast mass or change is advisable; however, there is insufficient data to recommend a formal surveillance program at this time.[16]

Ovarian Cancer Screening

The ovarian cancer screening measures have limited sensitivity and specificity and have not been shown to reduce ovarian cancer mortality. Nevertheless, the CSGC[16] and NHGRI task force recommends for women with a BRCA1 or BRCA2 cancer predisposing mutation the following:

* Annual or semi-annual pelvic examination beginning at age 25-35 years
* Annual or semi-annual transvaginal ultrasound examination with color Doppler beginning at age 25-35 years
* Annual or semi-annual serum CA-125 concentration beginning at age 25-35 years. Serum screening can be associated with a high false positive rate, especially in premenopausal women, and is often abandoned by physicians even in germline carrier screening.

The Memorial Sloan Kettering Cancer Center reports the first prospective evidence demonstrating that the above surveillance strategy employed in BRCA positive women may result in the diagnosis of early staged ovarian tumors.[17]

Prophylactic Surgery

Prophylactic Mastectomy

In a study[23] of 6039 women found to carry a BRCA gene mutation and/or with a family history of breast cancer who underwent prophylactic mastectomy, Hartmann et al estimated a 90- 94% reduction in breast cancer risk and an 81-94% reduction in breast cancer-related deaths. Additional prospective data on 251 BRCA positive individuals followed at Memorial Sloan-Kettering Cancer Center demonstrated the detection of two occult intraductal breast cancers within the 29 individuals choosing risk-reducing mastectomy.[17]

Although only a small percentage of women from high-risk families choose to undergo prophylactic bilateral mastectomy, those who do generally feel content with their decision. In a follow-up study of high-risk women who pursued preventive surgery, approximately 74% reported a reduced emotional concern regarding breast cancer development and seemed to naturally sustain other psychological and social functioning.[25]

Prophylactic Oophorectomy

Data presented at the American Society of Human Genetics 50th Annual Meeting (2001) indicates that prophylactic **bilateral salpingo-oophorectomy (BSO)** reduces the risk of ovarian cancer by 95% in women with BRCA deleteri-

ous mutations. The risk of primary peritoneal carcinomatosis does not appear to be affected by salpingo-ophorectomy. The National Cancer Institute found that women from families at high risk for ovarian cancer had an equal rate of primary peritoneal cancer after oophorectomy compared to the rate of primary peritoneal cancer in women who had not had the procedure.[26] Additional studies are necessary to investigate whether hysterectomy or any other strategy would further reduce the risk of primary peritoneal cancer post salpingo-ophorectomy.

Rebbeck et al[27] also demonstrated that prophylactic oophorectomy reduces the risk of breast cancer by approximately 50% in BRCA carriers. Additionally, of 21 of 36 BRCA gene positive women diagnosed with breast cancer who underwent BSO either before or within 6 months of their cancer diagnosis, only 1 of 21 relapsed versus the 7 of 21 of women who retained their ovaries.[28] Therefore, experts now suggest that BRCA heterozygotes consider risk-reducing prophylactic salpingo-oophorectomy to reduce the risk of both ovarian and breast cancers

As preventive oophorectomy become more common secondary to the identification of an inheritable mutation, specific recommendations are emerging related to the surgical procedure, as well as regarding pathologic examination of the tissue removed. Unexpected gynecologic neoplasms were discovered in five high-risk breast/ovarian cancer patients (4 of the 5 patients had a documented deleterious BRCA mutation) who underwent prophylactic salpingo-oophorectomy with hysterectomy. Therefore, more rigorous tissue examination, as well as specified surgical interventions should be considered for the detection of early neoplastic changes when BRCA carriers choose preventive BSO as a risk-reducing strategy.[29]

After comprehensive cancer genetic counseling, the majority of women are pleased with their decision to pursue surgical interventions for ovarian cancer prevention. Although approximately 93% of high-risk women who underwent prophylactic oophorectomy expressed no regret about their decision, 50% preferred more information about the risk and benefits of **hormone replacement therapy (HRT)** prior to decisions about surgery.[30] Thus, although counseling appears to addresses the direct surgical issues, more attention should be directed to the outcome implications.

…prophylactic bilateral salpingo-oophorectomy reduces the risk of ovarian cancer by 95% in women with BRCA deleterious mutations.

Chemoprevention

Breast Cancer Risk Reduction

The **national surgical adjuvant breast and bowel project (NSABP P-1)** prevention trial assessed the treatment of tamoxifen (a partial estrogen antagonist) in women identified by the Gail model to have an increased breast cancer risk. This study reported a 49% reduction in breast cancer in the five-year group treated with tamoxifen. It was concluded that tamoxifen prophylaxis was most beneficial in women with an elevated risk of breast cancer who were under age 50, because premenopausal women did not seem to be at increased risk for venous thrombosis or uterine cancer when compared to their post- menopausal counterparts. However, tamoxifen reduced the incidence of breast cancers that were estrogen receptor-positive, but not estrogen receptor-negative. Since breast cancers occurring in women with BRCA1 mutations are more likely to be estrogen receptor-negative,[28] it is difficult to estimate the benefit of tamoxifen prophylaxis without testing the effect in women with BRCA1 or BRCA2 cancer-predisposing mutations.

To assess the effect of tamoxifen in BRCA carriers, complete BRCA sequencing analysis was performed on 288 of the 315 women who developed invasive breast carcinoma.[32] However, only 19 (6.6%) were found to be heterozygous for BRCA mutations. Due to the small sample and wide confidence intervals, conclusive data could not be drawn; but encouraging results from Narod et al.[33] demonstrated an estimated 75% reduction for a contralateral breast cancer in BRCA1 and BRCA2 cancers.

Nevertheless, because tamoxifen treatment can have significant adverse consequences (a higher rate of endometrial cancer and thromboembolic episodes, including pulmonary embolism), patients should be counseled accordingly.

Ovarian Cancer Risk Reduction

One case control study found a significant decreased risk of ovarian cancer in women with BRCA1 or BRCA2 cancer-predisposing mutations who took oral contraceptives for more than three years.[34] These data remain consistent with general population studies which indicate a reduced risk of somatic ovarian carcinoma with oral contraceptive use; but the study is debated, primarily because it did not assess other outcomes, such as the effect of oral contraceptives on breast cancer risk.

Our understanding of inherited and acquired genetic mutations that eventually give rise to the malignant phenotype is evolving. Still in its infancy, the discipline of cancer genetics will evolve more rapidly over the next 10 years with the newer techniques, such as DNA chip analysis and micro array processing, which will yield a better understanding of the genotype-phenotype relationship.

References

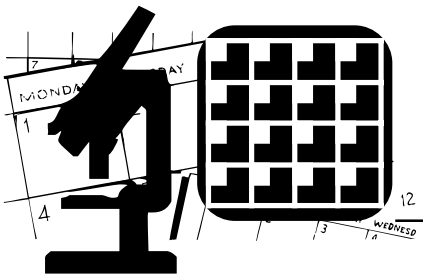
1. Ries LAG, Eisner MP, Kosary CL, et al. SEER Cancer Statistic Review, 1973-1998. National Cancer Institute, Bethesda, MD, 2001.
2. Meiser B, Halliday JL. *Soc Sci Med* 2002;54:1463-70.
3. Legare RD. *Med&Health/RI* 1999;82:172-5.
4. Statement of the American Society of Clinical Oncology: genetic testing for cancer susceptiblity. *J Clin Oncol* 1996;14:1730-6.
5. Couch FJ, DeShano ML, Blackwood MA, et al. *NEJM* 1997;336:1409-15
6. Shattuck-Eidens D, Oliphant A, McClure M, et al. *JAMA* 1997;278:1242-50.
7. Chang-Claude J, Dong J, Schmidt S, et al. *J Med Genet* 1998;35:116-21.
8. Parmigiani G, Berry DA, Aguilar O. *Am J Hum Genet* 1998;62:145-58.

9. Frank TS, Deffenbaugh AM, Reid JE, et al. *J Clin Oncol* 2002;20: 1480-90.
10. Berry DA, Parmigiani G, Sanchez J, et al. *J Natl Cancer Inst* 1997;89:227-38.
11. Ford D, Easton DF, Bishop DT, et al. *Lancet* 1994;343:692-5.
12. Strewing JP, Hartge P, Wacholder S, et al. *NEJM* 1997;336:1401-8.
13. Whittemore AS, Gong G, Itnyre J. *Am J Human Genetics* 1997;60:496-504.
14. Easton DF, Ford D, Bishop DT. *Am J Human Genetics* 1995;56:265-71.
15. The Breast Cancer Linkage Consortium: Cancer Risks in BRCA2. *J Natl Cancer Inst* 1999;61:1310-16.
16. Burke W, Daly M, Garber J, et al. *JAMA* 1997;277:997-1003.
17. Scheuer L, Kauff N, Robson M, et al. *J Clin Oncol* 2002;20:1164-6.
18. Vasen HF, Haites NE, Evans DG, et al. *Eur J Cancer* 1998;34:1922-6
19. Fridenson B. *Med Gen Med* 2000;9:E9.
20. Pierce LJ, Strawderman M, Narod SA, et al. *J Clin Oncol* 2000;18:3360-9.
21. Tilanus-Linthorst MM, Obdeijn IM, Bartels KC, et al. *Breast Cancer Res Treat* 2000;63:53-60.
22. Warren R. *Eur J Radiol* 2001;39:50-9.
23. Hartmann LC, Schaid DJ, Woods JE, et al. *NEJM* 1999;340:77-84.
24. Meijers-Heijboer H, van Geel B, van Putten WL, et al. *NEJM* 2001;345:159-64.
25. Frost MH, Schaid DJ, Sellers TA, et al. *JAMA* 2000;284:319-24.
26. Struewing JP, Brody LC, Erdos MR, et al. *Am J Hum Genet* 199;57:1-8.
27. Rebbeck TR, Levin AM, Eisen A, et al. *J Natl Cancer Inst* 1999;91:1475-9.
28. Moller P, Borg A, Evans DG, et al. *Int J Cancer* 2002;101:555-559.
29. Agoff SN, Mendelin JE, Grieco VS, et al. *Am J Surg Pathol* 2002;26:171-8.
30. Swisher EM, Babb S, Whelan A, et al. *J Reprod Med* 2001;46:87-94.
31. Noller KL. *JAMA* 2002;288:368-9.
32. King MC, Wieand S, Hale K, et al. *JAMA* 2001;286:2251-6.
33. Narod SA, Brunet JS, Ghadirian P, et al. *Lancet* 2000;356:1876-81.
34. Narod SA, Risch H, Moslehi R, et al. *NEJM* 1998;339:424-8.
35. Grabrick DM, Hartmann LC, Cerhan JR, et al. *JAMA* 2000;284:1791-8.

Jennifer L. Scalia, MS, is Clinical Program Coordinator, Cancer Risk Assessment and Prevention Program Women & Infants' Hospital.

Robert D. Legare, MD, is a medical oncologist at Women & Infants' Hospital; Medical Director, Cancer Risk Assessment and Prevention, Program; and Assistant Professor of Obstetrics & Gynecology, Brown Medical School.

CORRESPONDENCE:
Jennifer L. Scalia, MS
Women & Infants' Hospital
101 Dudley St.
Providence, RI 02905
phone: (401) 453-7540
fax: (401) 453-7785



Severe Hyponatremia Caused By an Instrasellar Carotid Artery Aneurysm

Susanna I. Lee, MD, PhD, Shyoko Honiden, MD, Elaine B. Fain, MD, Dominick Tammaro, MD, Fred J. Schiffman, MD

A 65-year-old woman presented with fatigue, anorexia, and persistent nausea and vomiting for seven days. She had a history of hypertension and hypercholesterolemia, but had otherwise been in her usual state of health. Examination revealed a blood pressure of 150/110mmHg supine and 145/80mmHg erect. The skin and mucous membranes were dry without hyperpigmentation. The neurological exam including visual fields and acuity testing were normal. The patient had a serum sodium of 114 mM and urine sodium of 105 mM. Other serum chemistries including potassium, chloride, bicarbonate, urea nitrogen, creatinine, and glucose were normal. Serum AM cortisol was 4.0 mcg/dL (normal 4.0-18.0) and rose to 20.9 mcg/dL (normal>11.0) after **adrenocorticotropin (ACTH)** administration. The patient's plasma ACTH level was 5pg/ml (normal 9-52). Thyroid function tests revealed a total T4 of 4.3 mcg/dL (normal 4.0-11.0), resin T3 uptake of 24% (normal 25-36) and **thyroid stimulating hormone (TSH)** level of 0.72mU/L (normal 0.38-6.13) Prolactin and growth hormone levels were 33ng/ml (normal 2-20) and <1.0ng/ml (normal 1.0-5.0) respectively.

The patient was rehydrated to euvolemia with intravenous normal saline. However, her serum sodium continued to decline despite fluid restriction. (Figure 1) Three days after admission, the patient was hypotensive with a systolic pressure of 65mmHg and a serum sodium of 106mM. She was resuscitated with hypertonic saline and started on intravenous hydrocortisone. A head CT with contrast revealed a 3cm. enhancing mass eroding into the clinoid processes and the sellar floor. (Figure 2A) Magnetic resonance imaging (MRI) revealed a single lobed aneurysm filling the sella turcica and obliterating the sphenoid sinus. (Figure 2B) No mass effect was seen on the optic chiasm. **Magnetic resonance angiography (MRA)** demonstrated slow flow in the aneurysm arising from the posterior cavernous portion of the left **internal carotid artery (ICA)**. (Figure 2C) The patient had the aneurysm embolized by the placement of 17 Guliemi Detachable Coils through an endovascular catheter. Subsequent angiography revealed greater than 90% occlusion of the aneurysm and a patent left ICA. The patient has been maintained for several months on prednisone without any endrocrine or neurologic complaints. Her serum sodium has normalized.

DISCUSSION

The Presentation: Endocrine/Neurologic Deficits

Parasellar intracranial aneurysms as a cause of endocrinopathy and neurological deficit are a well-described but infrequent phenomenon. Since Bramwell's 1887 description,[1] numerous cases have been reported.[2,3,4,5] Cushing, for example, noted that an intracranial aneurysm "by its compression effect can cause outspoken hypopituitarism."

By far, visual change is the most frequently encountered symptom. In White and Ballantine's review of 35 patients, 80% presented with visual changes - of which 33% were bitemporal hemianopsia. Anterior pituitary deficiency was clinically noted in a third of the patients. A more recent review by Fernandez[8] demonstrated a female preponderance (2.7:1) as well as a pituitary-gonadal axis involvement in 67.5%, pituitary-adrenal in 48.6%, pituitary-thyroid in 40.5%. This differential involvement is comparable to those described in other types of hypopituitarism.

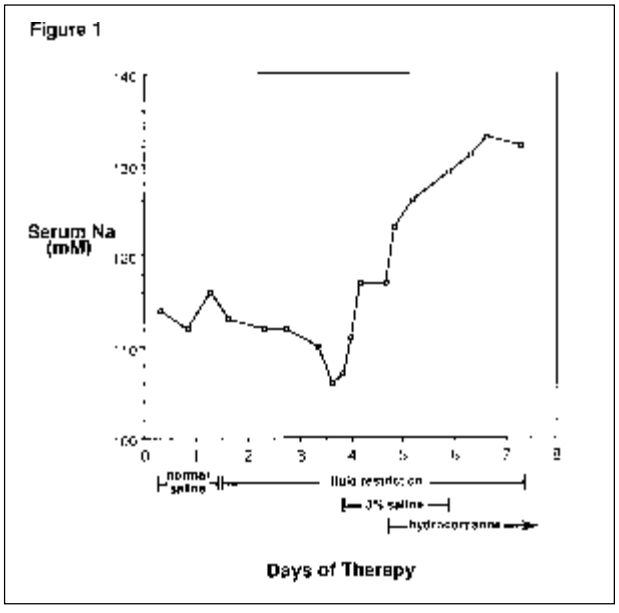


Figure 1. Serum sodium levels during hospitalization. the duration of fluid restriction (800cc/day) and hydrocortisone therapy (100 mg every 8 hours) are shown below. The administration of normal saline and 500cc of 3% saline is also indicated.

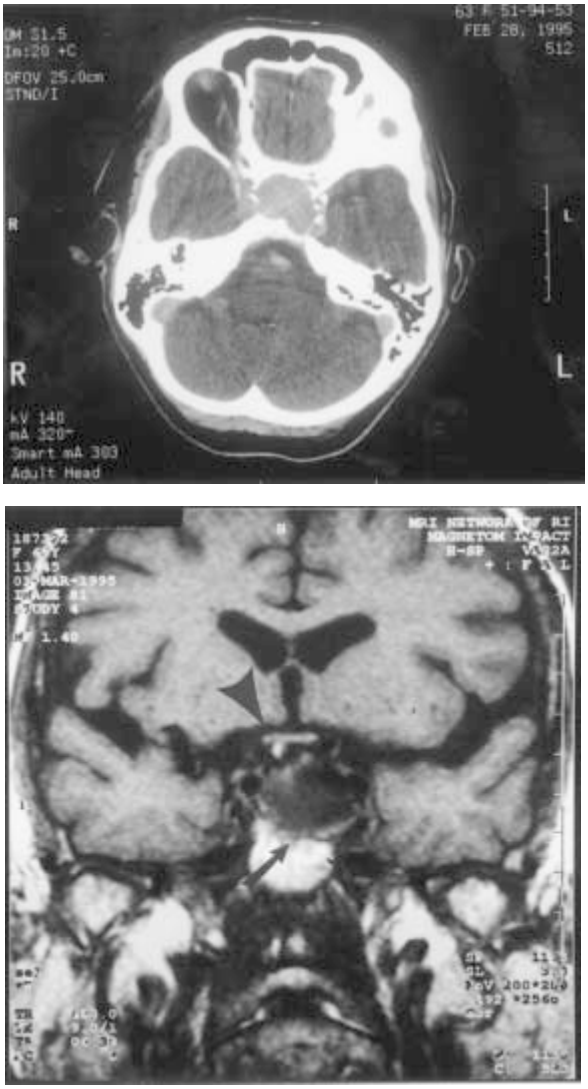


Figure 2. Images of the instrasellar carotid artery aneurysm.

(A) CT scan at the leval of the sella shows an enhancing mass within the sella.

(B) Coronal section of a brain MRI demonstrates the intra- and suprasellar aneurysm (arrow). The optic chiasm (arrowhead) shows no mass effect.

(C) 2D time of flight MRA demonstrates the slow flow within the aneurysm which originates from the left internal carotid artery (arrow).

Our case is unique in that the patient presented with evidence of hypopituitarism without any visual change or other neurological signs related to mass effect or hemorrhage. Review of the literature shows that most patients do have subtle signs of neurological involvement.[9] Nukta[10] described a 69 year-old man who presented with non-specific symptoms of weakness, weight loss, nausea and vomiting. The patient had no evidence of visual defects, but did have a broad-based gait and a positive Babinski sign. Work-up revealed a global pituitary deficiency secondary to an aneurysm of the anterior communicating artery. Similarly, Cartlidge's[11] 50 year-old female patient was somnolent with a positive Babinski sign and brisk deep tendon reflexes, though without visual changes. Perhaps the closest approximation to our patient is that presented by Michils,[12] who described a 73 year-old woman who presented with severe hyponatremia and seizures without focal neurologic findings. Past medical history, however, did reveal transient diploplia secondary to palsy in the right extraocular muscles.

Pathophysiology: The Mechanism of Pituitary Dysfunction

Various pathophysiologic mechanisms have been proposed to explain hypopituitarism. In 1956, Gallagher[13] described a 54year-old patient who presented with panhypopituitarism and cited pituitary atrophy from compression as a factor. Van't Hoff[14] proposed ischemia in the hypothalmic nuclei as a competing theory.

Verbalis, however, wrote the most detailed and recent analysis in 1982.[15] Three disease processes were hypothesized: functional pituitary adenomas, nonfunctional pituitary adenomas, and nonpituitary mass lesions. Nonfunctional pituitary adenomas and nonpituitary masses have a common pathway-mass effect. The mass effect leads to damage via compression or ischemia.

By impinging on the hypothalamus or pituitary stalk, a parasellar mass can cause secondary hormone hypersecretion or hormone deficiencies due to interruption of releasing factors arriving at the anterior pituitary. Hypersecretion is often manifest as hyperprolactinemia. In either case, compression can leave viable pituitary tissue such that reversal of the pituitary dysfunction maybe achieved after appropriate therapy. Indeed,

complete recovery is well-documented in the literature.[16,17,18,19] For example, Kahn's 42 year-old female patient with left temporal hemianopsia and galactorrhea returned to her usual health after surgery. Thorough endocrine evaluations, including dynamic testing with releasing factor stimulation, are therefore crucial to document viability prior to treatment. Some findings suggestive of reversality are: hyperprolactinemia, positive but blunted responses to releasing factors, and short duration of symptoms. Reassessment 6-8 weeks post-operatively is recommended before instituting indefinite hormone replacement therapy. Lastly, mass effect may take the form of ischemia and necrosis of pituitary tissue. This phenomenon is typified by a primary hormone deficiency and non-response to dynamic testing with releasing factors.

Pathophysiology: The Mechanism of Hyponatremia

Although classically described, symptomatic hyponatremia as an initial indicator of hypopituitarism is unusual. The resulting hyponatremia is corrected by administration of glucocorticoids, but not mineralocorticoids.[20] This is explained by the fact that aldosterone secretion is relatively independent of pituitary control via ACTH. At first glance, it seems as if hyponatremia is caused by excess retention of water through an inappropriate ADH effect, rather than a salt-wasting syndrome as in a mineralocorticoid defect.[21] This construct, however, is flawed as evidenced by the serum sodium trend in our patient. Sodium values declined despite fluid restriction. Sodium imbalance in the setting of hypopituitarism is corrected by the administration of saline.[20] ADH plays a supporting role at best in a mixed sodium wasting/water retention picture.

How then does hyponatremia occur? Decreased activity level of renin leading to increase in urinary sodium plays a key role. Hyporeninism responsive to cortisol administration has been reported in patients with hyponatremia secondary to hypopituitarism.[22] An indirect regulatory or permissive effect of cortisol on plasma renin activity has been postulated. This hypothesis is supported by the fact that patients with Cushing's syndrome display increased plasma renin activity.

Diagnosis: The Association Between Aneurysms and Adenomas

Historically, confusion between pituitary tumors and aneurysms has been common. Raymond in 1978 estimated that between 1.4-5% of aneurysms simulated pituitary tumors.[23] Numerous reports describe an aneurysm initially diagnosed as a pituitary tumor and only later properly identified by carotid angiography, or at the time of autopsy in some unfortunate cases.[24,25,26,27]

In addition to the similarities in plain film, CT and clinical presentations there are other factors that contribute to the confusion. There is a well-documented association between intracranial aneurysms and pituitary adenomas, such that the chances of the two simultaneously existing is far greater than that explained by coincidence.[28,29,30] Pia studied the occurrence of brain tumors in general with aneurysms and speculated local circulatory changes as one mechanism in which tumors facilitate aneurysmal dilatation.[31] Anticipating possible coexistence is prudent for a neurosurgeon, as evidenced by Tsuchida's misfortune of rupturing an anterior communicating artery aneurysm during transsphenoidal removal of a pituitary adenoma.[32] Wakai estimated the frequency of association between an intracranial aneurysm and pituitary adenoma to be 7.4%,[33] while Jakubowski, in his review of 150 pituitary tumors, approximated the incidence to be 6.7%.[34]

Confirming the Diagnosis: Imaging

In the pre-CT era, calcification in the posterior fossa on lateral plain film with enlargement of superior orbital fissures was used as suggestive evidence of intracranial aneurysms. The enlarged fissure was estimated to occur in 75% of patients with aneurysms but only in 5% of pituitary tumors.

The gold standard for diagnosis continues to be angiography, but less invasive techniques are playing an increasingly large role. Aneurysms on CT appear as hyperdense lesions that enhance with contrast. Numerous reports exist, however, wherein contrast enhanced CT scans failed to identify giant aneurysms.[35,36,37]

MRI has become a first-line diagnostic tool, because it helps to characterize location, size, residual lumen size and flow. It does not require contrast administration as in angiographic studies.[8] On precontrast T1 weighted images, aneurysms have a similar density as cerebrospinal fluid. On "spin" echo imaging, it shows up as a distinctive ìflow voidî with a black appearance. This occurs because rapidly flowing areas have no signal. Partially or totally thrombosed aneurysms therefore can have areas of high intensity. Finally, MR/CT angiography has added to the armamentarium of the modern neuroradiologist. It further helps in assessing the vessel of origin, contiguity of the aneurysm with adjacent vessels, lumen size, and additional aneurysms.[38,39]

Management Principles

Direct surgical clipping or endovascular coiling is the preferred treatment of intracranial aneurysms.[40] Both techniques eliminate the aneurysm from normal circulation and prevent further dilatation or hemorrhage.

In weighing the risks and benefits of treating a patient with an unruptured aneurysm, accompanying symptoms, size and accessibility to direct surgical clipping must be considered. Though the natural history of asymptomatic, unruptured, untreated aneurysms is not well known, bleed rates have been estimated at 3-4% per year.[41] There is widespread agreement that larger size accelerates this rate further.[42] Surgical management should therefore be offered to patients with giant aneurysms regardless of symptoms especially if direct clipping is possible.[19] In fact, in his editorial comment, Ojemann recommended surgical treatment for all aneurysms greater than 7mm in size.[43]

Surgery is also recommended for symptomatic aneurysms because symptoms are thought to be a marker of rapid enlargement. Improvement in neurological function is immediately recognizable. Potentially avoiding the long-term effects of hypopituitarism on life expectancy is another advantage. In ret-

rospective analysis of 172 patients between 1967-94 with partial or complete hypopituitarism (excluding Cushing's and acromegaly), Bates found an increase in all cause mortality compared to an age and sex-matched control population.[44] The ratio of observed to expected deaths was 1.73, while that restricted to females was even higher at 2.29.

Conclusion

We report a case of a giant ICA aneurysm which presented as profound hyponatremia. Hyponatremia was likely caused by insufficient ACTH levels resulting from pituitary insufficiency, which lead to a state of hyporeninism. The unique aspect of this case is that the patient had no neurological complaints related to mass effect or hemorrhage. The absence of typical neurologic symptoms should therefore not dissuade the clinician from considering intracranial aneurysms as a cause of pituitary dysfunction. Given the grave mortality ruptured aneurysms and the morbidity of persistent endocrine deficits, thorough evaluation (including dynamic hormone testing, MRI and angiography) and treatment is recommended.

References

1. Bramwell B. *Edinb Med J* 1887;32:911-22.
2. Wallace S, Jaffe ME. *Radiol* 1967;88:90-3.
3. Uozumi T, Shima T, Moris. *No Shinkei Geka* 1979;7:25-31.
4. Shantaram VV, Clift GV. *JAMA* 1974; 229:1473.
5. Dussault J, Plamomndon C, Volpe R. *Can Med Assoc J* 1969:101:51-6.
6. Cushing H. *The Pituitary Body and its Disorders: Clinical States Produced by Disorders of the Hypophysis Cerebri.* Philadelphia. JB Lippincott, 1912.
7. White JC. Ballantine HT, Jr. *J Neurosurg* 1961;18:34-50.
8. Fernandex-Real JM, Fernandex-Castaner M, Billabona C, et al. *Clin Investigation* 1994; 72:302-6.
9. Ooi TC, Russell NA. *Can J Neurol Sci* 1986;13:70-1.
10. Nukta EM, Taylor HC. *Can Med Assoc J* 1987;137:413-5.
11. Cartlidge NE, Shaw DA. *J Neurosurg* 1972;36:640-3.
12. Michils A, Baleriaux D, Mockel J. *Postgrad Med J* 1991;67:285-8.
13. Gallagher PG, Dorsey JF, Stefanini M, Lonney JM. *Neurol* 1956;6:829-37.
14. Van't Hoff W, Hornabrook RW, Marks V. *Brit Med J* 1961;2:1190-4.
15. Verbalis JG, Nelson PB, Robinson AG. *Neurosurg* 1982;10:604-11.
16. Giustina A; Scalvini T, Cerudelli B, et al. *Minerva Endocrinol* 1989;14:255-8.
17. Kahn SR, Leblanc R, Sadikot AF, Fantus IG. *Can J Neurol Sci* 1997;24:64-6.
18. Kita Y, Kawato M, Nakabayashi H, et al. *Nippon Naika Gakkai Zasshi* 1986;75:1756-63.
19. Heroes RC, Nelson PB, Ojemann RG, et al. *Neurosurg* 1983;12:153-63.
20. Bethune JE, Nelson DH. *NEJM* 1965;272:771-6.
21. Davis BB, Bloom ME, Field JB, Mintz DH. *Metabolism* 1969;18:821-32.
22. Major P, Kuchel O, Boucher R, et al. *J Clin Endocrinol Metab* 1978;46:15-9.
23. Raymond LA, Tew J. *J Neurol Neurosurg Psychiatry* 1978;41:83-7.
24. Arlot S, Lalau JD, Galibert P, Quichaud J. *Rev Med Interne* 1985;6:505-9.
25. Barotini F, Ammannati F, Gagliardi R, et al. *Ital J Neurol Sci* 1994;15:369-72.
26. Chien WY, Want PW, Huang HS, Huang MJ. *Chang Keng I Hsueh Tsa Chih* 1989;12:161-6.
27. Kayath MJ, Lengyel AM, Nogueira, et al. *J Endocrinol Invest* 1991; 11:975-9.
28. Gokalp HZ, Avman N, Ozkal E,Gokben B. *Acta Neurochirurgica* 1980;53:267-73.
29. Wada M, Takahashi K, Hasegawa T, et al. *Neuro Surg* 1982;10:215-20.
30. Plangger CA, Twerdy K, Mohsenipour I, Grunert V. *Nervenarzi* 1987;58:279-86.
31. Pia HW, Obrador S, Martin JG. *Acta Neurochir* 1972;27:189-204.
32. Tsuchida T; Tanaka R, Yokoyama M, Sato H. *Surg Neuro* 1983;20:67-70.
33. Wakai S, Fukushima T, Furihata T, Sano K. *Surg Neurol* 1979;12:503-7.
34. Jakubowski J, Kendall B. *J Neurol Neurosurg Psychiatry* 1978;41:972-9.
35. MacPherson P, Anderson DE. *Neuroradiol* 1981;21:177.
36. Lumenta CB, Lins E, Bock WJ. *Neurochirurgia (Stuttg)* 1984;27:117-9.
37. Mindel JS, Sachdev VP, Kline LB, Sivak MA. *Surg Neurol* 1983;19:163-7.
38. Fitzpatrick M, Tartaglino LM, Hollander MD, et al. *Radiol Clinics N Amer* 1999;37:101-21.
39. Demaerel P, Marchal G, Casteels I, et al. *Neuroradiol* 1990;32:322-4.
40. Chang SD, Steinberg GK. *Vasc Med* 1998;3:315-26.
41. Jane JA, Winn HR, Richardson AE. *Clin Nerosurg* 1974;24:176-84.
42. Hamburger C, Schonberger J, Lange M. *Neurosurg Rev* 1992;15:97-103.
43. Bates AS, Van't Hoff W, Jones PJ, Clayton RN. *J Clin Endocrinol Metab* 1996;81:1169-72.

Susanna I. Lee, MD, PhD, is Instructor in Radiology, Harvard Medical School, and Assistant Radiologist, Massachusetts General Hospital.

Shyoko Honiden, MD, is is a medical resident, Mt. Sinai Hospital, New York City.

Elaine B. Fain, MD, is in private practice.

Dominick Tammaro, MD, is Assistant Professor of Medicine, Brown Medical School.

Fred J. Schiffman, MD, is Professor of Medicine, Brown Medical School.

Correspondence:
Fred J. Schiffman, M.D.
The Miriam Hospital
164 Summit Avenue
Providence, RI 02906
phone: (401) 793-4035
fax: (401) 793-4022
e-mail:fred_schiffman@brown.edu

RIQP paste-in

Health by Numbers

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

Edited by Jay S. Buechner, PhD

Tobacco-Related Cancer Incidence in Rhode Island and the United States

Leanne C. Chiaverini, Jay S. Buechner, PhD, and John P. Fulton, PhD

Previous analyses[1,2] on three decades of cancer mortality in **Rhode Island** (**RI**) and the **United States** (**US**) found that age-adjusted mortality rates for tobacco-related cancers fell slightly for RI males and rose for RI females in the 1990s compared to the 1970s and 1980s. In addition, the percent elevation (RI rate relative to US rate) in tobacco-related cancer mortality among males decreased from the 1970s to the 1980s but did not change from the 1980s to the 1990s. The percent elevation among females increased in each decade from the 1970s to 1990s, but was lower than the elevation among males. In the 1990s, tobacco-related cancer mortality rates in RI were 10% higher among males and 6% higher among females when compared to the US.

A closer analysis of tobacco-related cancer trends in the most recent decade has produced noteworthy findings. This report presents data on the patterns of tobacco-related cancer mortality and incidence since the establishment of the Rhode Island Cancer Registry in 1987, through 1999, with comparison to US data. Data on historical patterns of cigarette smoking for both areas are also presented to examine the correlation with this important risk factor.

Methods

Following previous studies,[1,2] cancers of the following anatomic sites were considered to be "tobacco-related": lung-bronchus, urinary bladder, kidney and renal pelvis, oral cavity and pharynx, esophagus, pancreas, larynx, and trachea, mediastinum and other respiratory organs.

RI and US cancer mortality rates for 1987-1999 were obtained from the Centers for Disease Control and Prevention's WONDER system.[3] RI cancer incidence rates for 1987-1999 were obtained from the RI Cancer Registry and US cancer incidence rates for 1987-1999 were obtained from the National Cancer Institute, SEER Cancer Statistics Review.[4] All rates were computed for males and females, including all races, and were directly standardized for age, using the 2000 population of the US as the standard population. For trend analysis, annual rates are presented as three-year moving averages and expressed as "average annual deaths [mortality] or cases [incidence] per 100,000 population per year." The percent elevation of tobacco-related cancer rates in RI was calculated relative to the corresponding US rates.

Data on the rate of current cigarette smoking among RI males and females for 1975-2001 were obtained from the RI Health Interview Survey,[5] a periodic telephone survey of approximately 2,600 households including 6,500 individuals per iteration. (Data for 2001 are preliminary data subject to change.) Comparable smoking rates for the US were assembled from published data from the National Health Interview Survey.[6] For both sources, a "current smoker" is defined as a person who has smoked at least 100 cigarettes and who now smokes.

Results

The distribution of anatomic sites contributing to tobacco-related cancers differs for cancer mortality and incidence. In 1999, cancer of the lung and bronchus was responsible for the largest proportion of tobacco-related cancer mortality (67%) and incidence (50%) in RI. (Figure 1) Cancers of the pancreas and urinary bladder were, respectively, the second and third leading sites among tobacco-related cancer deaths in RI, while the second and third largest sites for tobacco-related cancer incidence were cancers of the urinary bladder and of the kidney and renal pelvis.

Consistent with earlier findings,[1,2] RI age-adjusted tobacco-related cancer mortality from 1987-89 to 1997-99 fell slightly for males (-5%) and rose for females (+22%). Relative to the US, the percent elevation of RI tobacco-related cancer mortality decreased in the late 1980s then increased in the 1990s. (Figure 2) During the most recent decade (between 1989-91 and 1997-99), the percent elevation rose from +4% to +12% among males and from -2% to +12% among females.

The age-adjusted tobacco-related cancer incidence rate among RI males was virtually unchanged from 1987-89 to 1997-99 (+1%) while their US counterparts saw a decrease of 10%.

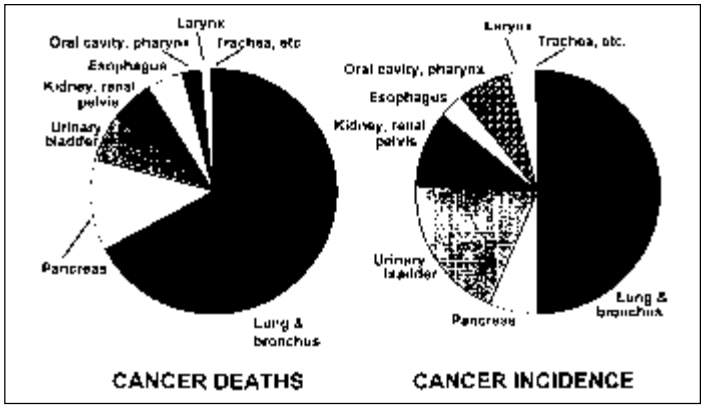


Figure 1. Cancer mortality and incidence by anatomic site, Rhode Island, 1999.

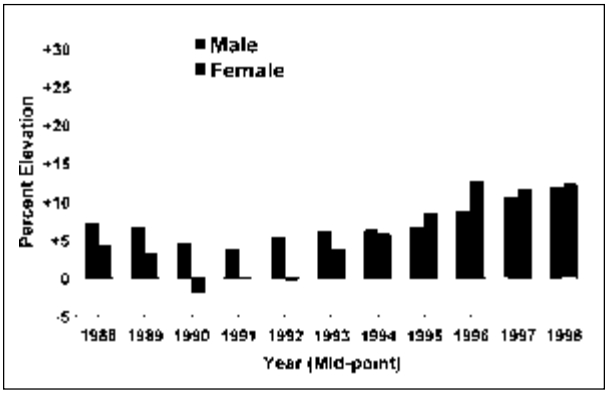


Figure 2. Elevation of Rhode Island mortality per 100,000 population (age-adjusted) relative to US mortality, tobacco-related cancers, by sex, 1988 – 1998 (three-year moving average)

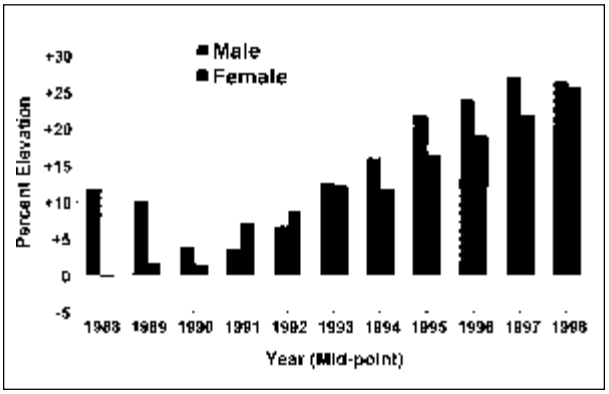


Figure 3. Elevation of Rhode Island incidence per 100,000 population (age-adjusted) relative to US incidence, tobacco-related cancers, by sex, 1988 – 1998 (three-year moving average)

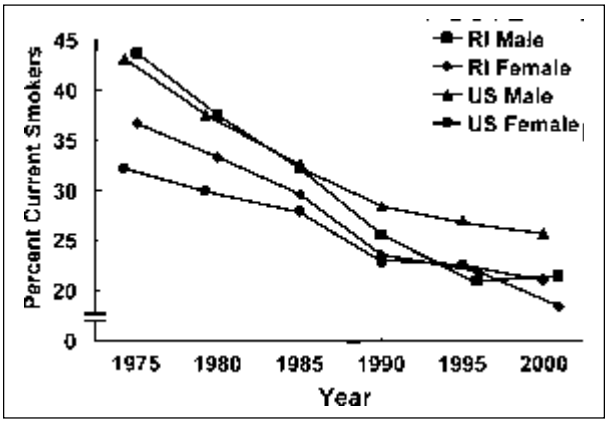


Figure 4. Percent current cigarette smokers, RI and US, 1974 – 2001.

Over the same period, the RI rate among females increased by 35%, while the rate among US females increased by 7%. During the most recent decade, RI male rates relative to the US rose from an elevation of +4% in 1989-91 to +26% in 1997-99, and the percent elevation among females rose from +2% in 1989-91 to +26% in 1997-99. (Figure 3) Among both males and females, the RI vs. US elevation of cancer incidence in 1997-99 was more than double the percent elevation for cancer mortality.

The percent of current smokers in both RI and the US has steadily declined since at least 1975. (Figure 4) From 1975 through 1985, RI males had smoking rates similar to those for US males; since 1990, their rates have fallen below US rates. RI females have smoked more than US females historically, but the large differential observed in 1975 (36.6% in RI vs. 32.1% in the US) had disappeared by 1995-6 (21.9% in RI vs. 22.6% in the US).

Discussion

Tobacco-related cancer mortality and incidence in RI relative to the US has become increasingly elevated over the past decade. In RI relative to the US, mortality attributed to tobacco-related cancers is elevated by 12% for both males and females as of 1997-99, while incidence is elevated by 26% for both males and female. This has occurred despite significant improvement in smoking rates in Rhode Island, both absolutely and when compared to the US.

The incidence of tobacco-related cancers in Rhode Island needs to be investigated further, both because the incidence data do not correlate with historic smoking patterns in the state and because they are not fully echoed in mortality rates. Some areas proposed for study are — (1) Changes in the population of the state: Are some of the observed patterns due to changes in the race and ethnicity composition of the population or to differential out-migration of non-smokers over time? (2) Accuracy of the data on smoking rates: Has the smoking data for the state been collected comparably over the period for which such data are presented, given declining participation rates in telephone surveys and other effects? (3) Detailed smoking patterns: The prevalence of former smokers and the amount smoked by current and former smokers may explain part of the observed patterns of incidence and mortality. (4) Non-tobacco risk factors: All of the cancer sites included in the definition of tobacco-related cancers are also related to other risk factors, such as occupational and environmental exposures, which impact urban dwellers. Are the incidence rates responding to changes in these other risk factors? It is likely that such further investigations will produce a number of factors that have contributed to the observed increase in cancer incidence over the past decade.

Leanne Chiaverini is Research Associate, Division of Disease Prevention and Control, Rhode Island Department of Health.

Jay S. Buechner, PhD, is Chief, Office of Health Statistics, and Assistant Professor of Community Health, Brown Medical School.

John P. Fulton, PhD, is Associate Director, Division of Disease Prevention and Control, Rhode Island Department of Health, and Clinical Associate Professor of Community Health, Brown Medical School.

References

1. Fulton JP, Buechner JS, Baker RS, et al. Cancer in Rhode Island, an old urban state. *RI Med J* 1988;71:139-46.
2. Chiaverini L, Fulton JP. Reassessing Cancer Mortality in Rhode Island, an Old Urban State. *Med & Health / RI* 2002;85:256-7.
3. Centers for Disease Control and Prevention, National Center for Health Statistics. CDC WONDER's Compressed Mortality Files. http://wonder.cdc.gov/
4. National Cancer Institute. SEER Cancer Statistics Review 1973-1999. Bethesda, MD: National Cancer Institute, 2002.
5. Rhode Island Health Interview Survey, Office of Health Statistics, RI Department of Health.
6. National Center for Health Statistics. Health, United States, 2002 with Chartbook on Trends in the Health of Americans. Hyattsville, Maryland: 2002. (See also previous editions.)

PUBLIC HEALTH BRIEFING

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

Edited by John P. Fulton, PhD

Progress in the Control of Cancer of the Cervix in Rhode Island, 1987-2000

Leanne Chiaverini, John P. Fulton, PhD, Dorothy M. Darcy, CTR

[Fourth in a Series]

Profile

In Rhode Island, about 800 women have been diagnosed with cervical cancer (837 in 1998), about 50 women are newly diagnosed with cervical cancer each year (46 in 2000), and about 10 succumb to the disease annually (11 in 1999). In Rhode Island, cervical cancer accounted for less than one percent of all newly diagnosed cancers in 2000, and only 0.4% of all cancer deaths in 1999. However, given the effectiveness of regular screening with the Pap test, any case of cervical cancer and any death from this disease must be seen as a public health failure.

Control Strategy

Several risk factors for cancer of the uterine cervix have been identified.[1] However, the most clinically significant strategy for the reduction of cervical cancer is use of the Pap test (Pap smear), a noninvasive, inexpensive, simple screening procedure that allows physicians to find and treat precancerous dyplasias and localized tumors. The effectiveness of screening with the Pap test for the reduction of cervical cancer mortality has been demonstrated by several studies.[2] Although reports of high false-negative and false-positive rates have caused the accuracy of the Pap test to be questioned, the rescreening of smears and the development of computer-based automated technology have reduced the proportion of false results.[3] Aggressive use of the Pap test remains a key control strategy accompanied by multidisciplinary, state-of-the-art treatment, if necessary. The Rhode Island Cancer Control Plan,[4] published September, 1998, recommends:

Cervical Cancer Screening

* For women in high risk groups — women with multiple sex partners, sexually promiscuous partners, early age at first intercourse, and/or a history of a sexually transmitted disease (including human papilloma virus) — Pap smears should be performed annually.
* For women who are HIV positive, Pap smears should be performed at least annually.
* For asymptomatic women with a cervix and no risk factors, regular Pap smears should be performed if a woman is or has been sexually active. There is no upper age limit for the performance of regular Pap smears.
* If a history of past and/or present sexual activity cannot be accurately determined and a woman is 18 years of age or over, routine Pap screening should be initiated.
* Women who have had a hysterectomy cannot be presumed to be without cervical tissue and the decision to screen them with Pap smears should be determined on a case by case basis.

Basic Treatment Infrastructure

* Promote and support the adoption of American College of Surgeons (ACOS) approved cancer programs in all acute care hospitals in Rhode Island.
* Assure accurate tumor staging with American Joint Committee on Cancer (AJCC) staging methodology.

2010 Targets

Healthy People 2010, the most recent set of health objectives for the United States, [2] suggests the following targets for the control of cervical cancer:

Screening

By 2010, increase the proportion of women aged 18 years and older who have ever received a Pap test to 97% (baseline = 92% in 1998), and increase the proportion of women aged 18 years and older who have received a Pap test within the preceding 3 years to 90% (baseline = 79% in 1998).

Mortality

By 2010, reduce the cervical cancer death rate to 2.0 deaths per 100,000 females (age-adjusted to the year 2000 standard population of the United States; baseline = 3.0 deaths per 100,000 females in 1998).

Trends

(Please refer to Table 1.)

Screening

The proportion of Rhode Island women of all races, aged 18 years and older, who had received a pap test within the preceding 3 years increased from 80% in 1992 to 89% in 2000. Among all the states, the median proportion of

Table 1. Progress in the control of cervical cancer.

- % women who have had a Pap smear within the past three years
- Average annual age-adjusted cervical cancer incidence rates by summary stage of disease at diagnosis among women of all races
- % cases in RI ACOS-approved treatment programs, of cases with AJCC staging, and of localized cases with recommended treatment
- Average annual cervical cancer mortality rates among women of all races

Place	*Measure*	Source	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
RI	% Screened *	[a]				80.0	83.9	NA	82.9	82.9	87.4	85.2	86.0	88.5
U.S.	% Screened					84.1	84.7	85.4	84.5	84.6	84.6	84.8	85.5	86.8
RI	Incidence - Local **	[b]	4.8	5.4	5.7	6.0	6.3	6.4	6.2	5.7	5.5	5.1		
RI	Incidence - Regional	[b]	2.7	3.0	2.5	2.8	3.0	2.9	2.9	2.7	2.6	2.4		
RI	Incidence - Distant	[b]	1.1	1.0	1.0	1.1	1.2	1.4	1.5	1.5	1.5	1.4		
RI	Incidence - Unknown Stage	[b]	1.4	1.5	1.4	1.2	1.1	1.1	0.9	0.8	0.9	0.8		
RI	Incidence - All Invasive ***	[b]	10.0	10.8	11.0	11.2	11.6	11.8	11.4	10.7	10.4	9.7		
U.S.	Incidence - All Invasive	[c]	10.4	10.4	10.2	10.0	9.6	9.5	9.4	9.3	9.0	NA		
RI	% Cases in RI ACOS Tx Pgms	[b]	32	28	27	23	29	26	20	39	73	82	90	96
RI	% Cases with AJCC Staging	[b]	72	77	81	73	84	97	90	93	94	87	92	87
RI	Mortality	[d]	2.6	2.8	3.0	3.0	2.8	3.3	3.2	2.9	2.9	NA		
U.S.	Mortality	[d]	3.6	3.6	3.5	3.5	3.4	3.4	3.3	3.2	3.1	NA		

* Percentage of women who have had a Pap smear within the past three years

** Incidence and mortality rates are based on five years' data (e.g., 1989 = 1987-1991; 1998 = 1997-2002), age adjusted to the 2000 U.S. standard population, expressed as cases per 100,000.

*** Invasive includes the following stages of disease at diagnosis: local, regional, distant, and unknown

[a] Behavioral Risk Factor Surveillance System, Centers for Disease Control and Prevention

[b] Rhode Island Cancer Registry, Rhode Island Department of Health

[c] National Cancer Institute. *SEER Cancer Statistics Review 1973-1999*. Bethesda, MD. National Cancer Institute, 2002.

[d] CDC Wonder, Centers for Disease Control and Prevention

NA Data not available or not applicable

women of all races, aged 18 years and older, who had received a pap test within the preceding 3 years increased from 84% in 1992 to 87% in 2000.

Incidence

The age-adjusted incidence of invasive cervical cancer (2000 standard) among Rhode Island women of all races was 10.0 cases per 100,000 women in 1987-1991, peaked at 11.8 cases per 100,000 women in 1992-1996, then returned to 9.7 cases per 100,000 in 1996-2000 (based on five-year moving averages). In contrast, the age-adjusted incidence of invasive cervical cancer (2000 standard) among U.S. women of all races decreased from 10.4 cases per 100,000 women in 1987-1991 to 9.0 cases per 100,000 women in 1995-1999.

When age-adjusted incidence rates of invasive cervical cancer in Rhode Island are broken down by stage of disease at diagnosis, the incidence of local tumors peaked in the mid-1990s (from 4.8 cases per 100,000 women in 1987-1991 to 6.4 cases per 100,000 women in the mid-1990s to 5.1 cases per 100,000 women in 1996-2000). There was no significant change in the incidence of regional tumors until it declined slightly from 3.0 cases per 100,000 women in 1991-95 to 2.4 cases per 100,000 women in 1996-2000 (based on five-year moving averages). Age-adjusted incidence rates for both distant tumors and tumors of unknown stage hovered around 1 case per 100,000 women.

[Note: Adoption of the Bethesda System for classifying cervical cytology in the late 1980s made it impossible to distinguish in situ cervical cancer from high grade cervical dysplasias. Thus, cancer case reports for in situ tumors accepted after that time must be considered suspect. Recognition of this fact led to the termination of such reports by cancer registries around the country in 1996.]

Basic Treatment Infrastructure

From 1989 through 1996, the percentage of Rhode Island women newly diagnosed with cervical cancer who were treated under the auspices of in-state ACOS-approved hospital cancer programs averaged 28%. The addition of a program in 1997 and two more in 2000 brought the proportion of newly diagnosed cervical cancer cases treated under

ACOS-approved programs to 73% among Rhode Island women in 1997 and 96% in 2000.

Prior to a change in the Rules and Regulations of the Rhode Island Cancer Registry in 1992, only about 76% of the cervical cancer cases newly diagnosed among Rhode Island women were staged using the AJCC system, an important basis for choosing appropriate treatments. After the Rules change, the proportion of cases with AJCC staging increased to 84%, and has averaged 91% from 1993 through 2000.

Mortality

The age-adjusted mortality of invasive cervical cancer (2000 standard) among Rhode Island women of all races hovered around 3 cases per 100,000 women for the entire period of observation (based on five-year moving averages). The age-adjusted mortality of invasive cervical cancer (2000 standard) among U.S. women of all races experienced a small but steady decline from 3.6 cases per 100,000 women in 1987-1991 to 3.1 cases per 100,000 women in 1995-1999 (based on five-year moving averages).

Assessment

Gains have been made toward the achievement of basic treatment infrastructure goals as set forth in the second (1998) edition of the state's cancer control plan. The proportion of newly diagnosed cervical cancer cases treated under the auspices of in-state ACOS-approved hospital cancer programs increased from 32% to 96% during the period of observation. The proportion of cases staged with AJCC methodology increased from 72% in 1989 to 84% in 1993, and averaged 91% from 1993 through 2000.

In Rhode Island, increased use of the Pap smear in the 1990s was accompanied by a small peak in the incidence of invasive cervical cancer among women. The incidence of local and regional cervical tumors followed a similar pattern. There was no significant change in the incidence of distant cervical tumors and tumors of unknown stage, and little change occurred in cervical cancer mortality.

The Pap test is a known effective preventive for cervical cancer. Its aggressive promotion and use, followed by state-of-the-art therapy, if necessary, are important for the control of cervical cancer. However, given the rather flat trend in cervical cancer incidence and mortality in Rhode Island over the past decade, alternative strategies may be necessary to achieve the Healthy People 2010 goal. Screening programs, using social marketing strategies, should target low income and low education populations who are least likely to have been screened, and older women, who are often diagnosed at a later stage of disease and are more likely to die from the disease than younger women.[2]

References

1. National Institutes of Health, National Cancer Institute. *Cancer Rates and Risks*. 4th edition, 1996.
2. U.S. Department of Health and Human Services. *Healthy People 2010*. 2nd ed. With Understanding and Improving Health and Objectives for Improving Health. 2 vols. Washington, DC: U.S.Government Printing Office, November 2000.
3. Ku NN. Automated Papanicolau smear analysis as a screening tool for female lower genital tract malignancies. *Curr Opin Obstet Gynecol* 1999;11:41-3.
4. Rhode Island Department of Health. Cancer Control Rhode Island – Strategic Plan for 1998-2005. Providence, RI: Rhode Island Department of Health, September, 1998.

Leanne Chiaverini is Research Associate, Asthma Control Program, Division of Disease Prevention and Control, Rhode Island Department of Health.

John P. Fulton, PhD, is Associate Director of Health, Division of Disease Prevention and Control, Rhode Island Department of Health, and Clinical Associate Professor of Community Health, Brown Medical School.

Dorothy M. Darcy, CTR, is Director, Cancer Information System, Hospital Association of Rhode Island.

– A Physician's Lexicon –

This Way Madness Lies

Before the neuropathologists and neurochemists sought order within the diagnostic chaos of psychiatry, the nomenclature of 19th Century psychiatry abounded in vague and polysyllabic words.

Dementia, for example, was often subdivided by virtue of age of onset [e.g., dementia praecox, hebephrenic schizophrenia, senile dementia], by the dominant clinical sign [e.g., catatonic schizophrenia], by tangential reference to a syphilitic origin [dementia paralytica], or by reference to an environmental cause [toxic dementia].

Schizophrenia was a diagnostic word invented by Eugen Bleuler [1857-1939], a Swiss psychiatrist who needed a term to describe a form of mental disorder that he construed as a "splitting of the mind." The *schizo-* root is derived from a Greek word meaning split. It is the etymological source for such non-medical English words as schism [a cleft or a rent], schist [a layered crystalline rock], and schizogenesis [reproduction by fission]. The *phreno-* root usually refers to the brain [although sometimes to the diaphragm as in "the phrenic nerve."] Phrenology is now defined as a failed science attempting to find correlation between a human's cranial shape and his mental faculties. Phrenitis is an archaic term for encephalitis. A mad or delirious patient may be referred to as phrenetic, sometimes spelled frenetic. Frenetic, in turn, has evolved into the word, frantic. The root even crops up in Greek mythology. Neophron [literally, new brain] appears in some legends as a person with a childish spirit. And because he was transformed into a carnivorous bird, a genus of vultures is named after him.

Dementia praecox was an older term for schizophrenia of early clinical onset. The Latin word, *praecox*, means ripe or before its time and is the basis for the word, precocious.

Hebephrenia was a diagnostic term coined by the 19th century German psychiatrist, Ewald Hecker, to define schizophrenia of adolescent onset. Hebe was the Greek goddess of youth; and *Hebe-* is now a root defining pubescence or youth.

The catatonic form of schizophrenia is characterized by episodes of muscle rigidity accompanied by stupor. The word catatonia is comprised of two Greek roots: *cata-* variously meaning down or entirely or back. It is incorporated in such English words as catalytic, catapult and catastrophe. The *tono-* root defines tension or tone, as in words such as tonometer, tonality and tonic.

R.D.Laing once observed that schizophrenia is the name for a condition that most psychiatrists ascribe to patients they call schizophrenic. Certainly in the occasionally circular language of medicine there seems to be a relationship between the extent of etiological mystery and the complexity or meaninglessness of the employed diagnostic terms. Thus, if the cause and pathogenesis of a disease are unknown, there is a strong likelihood that its diagnostic name will be of Greco-Roman origin. And when the cause is clarified, the name is replaced by a far less romantic designation.

– 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			
	February 2002	12 Months Ending with February 2002		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	296	3,106	296.3	4,252.5
Malignant Neoplasms	191	2,414	230.3	7,493.0
Cerebrovascular Diseases	48	539	51.4	742.5
Injuries (Accident/Suicide/Homicide)	27	409	39.0	7,206.5**
COPD	58	507	48.4	470.0

Vital Events	Reporting Period		
	August 2002	12 Months Ending with August 2002	
	Number	Number	Rates
Live Births	1,143	13,564	12.9
Deaths	743	10,275	9.8
Infant Deaths	(6)	(112)	8.3
Neonatal deaths	(4)	(83)	6.1
Marriages	1,009	8,309	7.9*
Divorces	285	3,249	3.1
Induced Terminations	392	5,514	406.5#
Spontaneous Fetal Deaths	71	1,117	82.4
Under 20 weeks gestation	(65)	(1,042)	76.8
20+ weeks gestation	(6)	(75)	5.5

(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 # Rates per 1,000 live births
** Excludes two deaths of unknown age.

Quality *is* Worth More

With important purchases, the lowest price isn't always the best buy. Quality is more critical in the purchase of a home, car, medical equipment and that all-important purchase: medical malpractice insurance. With the future of your practice and your reputation on-the-line you can't afford second best.

Take a look at ProMutual Group:

- The largest provider of medical malpractice insurance in New England.
- Nearly 30 years experience.
- Insures more than 16,000 healthcare professionals.
- Financial strength that has earned us an A- (Excellent) rating from A.M. Best, the nation's leading independent insurance rating organization.
- Over $1.3 billion in total assets with $364 million in surplus.*
- Outstanding risk management service from the largest staff in New England.
- Experienced claim professionals close over 60% of claims without payment.

Isn't it time you got the best protection from one of the largest providers of medical malpractice insurance in New England? Call or visit our web site today.

www.promutualgroup.com

ProMutualGroup®

101 Arch Street, Boston, Massachusetts 02110
888-776-6888 www.promutualgroup.com
Connecticut • Maine • Massachusetts • New Hampshire • New Jersey • Rhode Island • Vermont

*As reported in our *2001 Annual Report.*

Not all products are available in all states.

ProMutual Group Agents:

Sidney Bailey
AON Risk Services of RI
Providence, RI
Phone: 401-331-7700

Hilda Barbosa
Starkweather & Shepley
East Providence, RI
Phone: 401-435-3600

John H. Lathrop
Lathrop Ins. Agency
Westerly, RI
Phone: 888-596-2530

Anthony Paolino, Jr.
Paolino Ins. Agency
Providence, RI
Phone: 401-421-2588

Joe Servant
Mastors & Servant
East Greenwich, RI
Phone: 800-335-5701

John Tickner
Babcock & Helliwell
Wakefield, RI
Phone: 401-782-1800

THE RHODE ISLAND MEDICAL JOURNAL

The Official Organ of the Rhode Island Medical Society
Issued Monthly under the direction of the Publications Committee

VOLUME 1
NUMBER 1

PROVIDENCE, R.I., JANUARY, 1917

PER YEAR $2.00
SINGLE COPY, 25 CENTS

Ninety Years Ago

[February, 1913]

In "Wrenched Knees and Slipped Semi-Lunar Cartilages," Frank E. Peckham, MD, argued against fixation ("either complete by means of plaster or splint, or partial by means of adhesive plastic strapping"). This conventional treatment provided immediate relief, but prolonged the functional recovery of the joint. Instead, he argued for baking and vibrating the knee, with a stockinet bandage applied between treatments for a few days only, or for a combination of modalities: "Morton wave current, followed by heavy sparking with a spark selector and vibrator … The current is delivered by placing a circular metal electrode about the sides of the knee, avoiding contact with the patella, this electrode to be connected with the positive pole of a static machine. The negative pole is grounded, patient being seated on an insulated table. The poles of the machine are separated slowly until a spark gap of 2 or 3 to 5 or 6 inches is obtained."

At the monthly December 1912 meeting of the Rhode Island Medical Society, with 70 members and 2 guests present, "Dr. Swartz read a communication from the Governor of Texas to the Governor of Rhode Island discouraging the immigration of consumptives to the South and Southwest, particularly "when they hope to seek employment upon their arrival."

Dr. John B. Murphy conducted the first of the St. Joseph's Hospital Clinics on November 14, speaking on bone surgery and "diseases affecting the osseous system."

Fifty Years Ago

[February, 1953]

Boston Univerity Professor of Surgery Reginald H. Smithwick, MD, contributed "The Physiological Effects of Operations for Duodenal Ulcer," (originally presented at the 5th Annual Interim Meeting, Rhode Island Medical Society). He concluded: "[It is] not essential that an operation result in histamine achlorhydria since jejunal ulcers will heal promptly after secondary procedures which do not produce achlorhydria to this stimulus."

Frederic J. Burns, MD, in "Problems of Organized Medicine," his Presidential Address to the 106th annual meeting of the Providence Medical Association, warned readers of Socialized Medicine ("aggressive, persistent, and unrelenting").

The Medical Society's Committee on Disaster reported support for certain Defense programs, national and state; but warned of "...the lack of proper equipment in most of our communities, and the absence of organized personnel for use in the event of a major catastrophe such as a fire, explosion or wreck on one of our transportation lines.....The hospitals of the state do not presently have available beds for immediate use in the event of a catastrophe."

Twenty Five Years Ago

[January, 1978]

In "A Message from the Dean: The Physician as Educator," Stanley M. Aronson, MD, reminded readers that the Hippocratic Oath called them to teach, yet standard curricula gave little attention to pedagogy, even to the techniques of instructing patients. "How often a few minutes of ...explanation can dispell ill-conceived mythology, how often candid and realistic descriptions can diminish a patient's anxieties and lessen the feelings of alienation between physician and patient."

Peter P. Reilly, MD, contributed "Depression and Its Treatment," arguing that "management of endogenous depression is faciliated by the judicious use of antidepressants." He also contributed "Efficacy of Lithium Carbonate in Alcoholism: Case Studies," citing the benefits of lithium "for chronic alcoholics who are endogenously depressed."

Demmie G. Mayfield, MD, and Robert G.M. Johnston, MD, MPH, in "Pharmacology Treatment in Drug Abuse," explained that the management of drug-abuse patients "requires an understanding of the pharmacology of the drugs."

Louis V. Sorrentino, MD, in "Group Psychotherapy in Rhode Island," explained the benefits of this supplement to individual therapy.