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Scientific Presentations

Drumroll Please

alumni president's greeting

Dear Fellow Alumni,

I have attended every Alumni Reunion weekend for many years. As soon as one is over, the date for the next year is saved in my calendar. They are always enjoyable, but none more than the immediate past reunion (May 2016) when I was inaugurated as President of our 136 year old Alumni Association. During my acceptance speech, I promised to be a good steward of the organization.

Now I need help from all of you. My profession as a physician was a calling that without Downstate, I wouldn't have been able to fulfill. I want to call on all of my fellow Alumni, to make it possible for medical students who attend Downstate to say what I heard a graduating student say during his induction in AOA earlier this spring. He said, "I couldn't have asked for a better four year experience, than I have had at SUNY Downstate. " The College of Medicine Alumni Association helped make that possible. Your membership and donations to the Alumni Association makes all that we do possible. The scholarships for research, the stipends for Global Health in Developing Countries, and the Brooklyn Free Clinic-run by students with attending preceptors are just a few activities that help students develop to their fullest potential. We also support the graduating students with a gift. As they leave, we hope they will return and help the next generation.

During my speech to SUNY Downstate College of Medicine Class of 2016 at Carnegie Hall, I asked all the graduates to become active immediately in the Alumni Association. I requested that during their residencies if they couldn't afford anything else, skip four lattes per month and send the Association that small donation. I told them "we want you back." Participation is good for each of us, and it's good for the Alumni Association.

I will close by asking everyone who reads this article or our monthly newsletter if you are not a member, please become one. Ask every alumnus or alumna that you know to do the same. We can do more for the students, more for the Association, more for the community, and more for the world if we work together.

Monika Sweeny, MD '75

President, Alumni Association

editor's greeting

One of the best things about a reunion is the cheerful buzz of doctors talking to one another. Greeting one another after 10 or 25 or even 50 years is a joyful time.

This year was our 137th reunion and the scientific session, the award ceremony, and the evening dinner dance were all exciting and filled with hugs and handshakes and pride. Our alumni again presented some remarkable research and the awardees have had amazing careers and some of their work has been groundbreaking in their fields.

The alumni association has been true to its mission for all these years. It was established that the following three things were paramount in importance:

1. Scholarship for medical students
2. Continuing educational programs
3. Annual reunions

It is so important that we continue to fund these vital functions.

Each year we request dues support to maintain the office and its administrative duties. We also have several fund appeals. I believe we must support the missions. Please get involved. We all received an invaluable education at Downstate. Please give back by paying dues and giving donations. The future doctors at Downstate depend on our help.

Thank you,

Constance Shames, MD '63

Editor, *Alumni Today*

Carlos N. Pato, MD, PhD

It is a pleasure to be here. I was just commenting that I started in June of last year, to visit frequently before my start, July 1, so I've been here for nearly a year. For some, that is very new, and I still have much to learn, but I have to say, it's been an eventful year, in many, many ways.

First of all, I want to welcome you all. It's delightful that you're here. We're in this period of celebration that happens around graduation which, really, we do too little of. I'm going to beg your indulgence to give me a minute to introduce myself, as most of you don't know me. I emigrated from Portugal to New York, and actually grew up here. I was on Staten Island, so it wasn't really Brooklyn, but I came through Brooklyn on my way to Stuyvesant when I wasn't on the Ferry.

New York is where my grandparents, who actually lived here, my mother was American, were based. And the public education that, in New York, has always been extraordinary – I've had tremendous history of going to premiere places, but nothing compared with my high school education here – is one of the things that I find exciting about what we do.

I was lucky enough to marry a person I met in college who is now my partner in research – for the past nearly 40 years. And we have been privileged to do research with populations. We do population geonomics. We're both psychiatrists. Our children are sad about it— no, they're not. (Jokingly.) They're doing very well, actually.

The bottom line is, we've had the privilege to do some exciting things. Being Portuguese, our initial work was in the Portuguese islands, where we studied whole populations of patients with schizophrenia, bipolar disorder, and all of their families. And then in the last 10 or 15 years, we expanded that to a very diverse cohort of patient partners we work with on an ongoing basis, and that group now is nearly 40,000 people who suffer from psychiatric disorders, and half of which who, along with their families, have no history of major psychiatric disorders. They all suffer everything else, of course; many have cancer, many have diabetes. They are people, and what we study are human genomics.

In coming here, my wife is actually directing a new institute for genomic health. Why health? Because the idea is, our genome carries tremendous patterns that relate to risk, not necessarily program you for illness, but relate to risk of illness, and equally to patterns that program you for resilience. So you could have full risk, but never have a particular illness because you also contain the elements that protect you. So understanding both is a critical issue. Without continuing too much more about myself, let me talk a little about Downstate.

I think you all know better than I that Downstate has had a rich history of people who have made tremendous contributions in the scientific field, but also in clinical leadership, and health system leadership. I'm always amazed at what the graduates from Downstate have done. How many are in academics, but also, how many are in key leadership roles in terms of clinical delivery. It's really an extraordinary setting.

Tonight I'm going to be coming to the dinner and I'm going to be sitting with one of the most important mentors in my life, she and her husband, they're graduates from 1970. She and her husband both came from Downstate. I met her at Mass General where I was doing my residency, and she was, for both my wife and I, one of our most important guides in terms of understanding what we could do in our specialty, and how to do it.

That, to me, speaks very personally. Because when we see what Downstate has gone through, which is a series of challenges over the past five years, the most amazing thing is the

incredible quality of our students. They are phenomenal. They are doing incredibly well. They continue to graduate and pretty much go anywhere they want, and that is a true statement of quality, in terms of an institution.

I will tell you that our research is a program that is struggling. I tend to be tremendously transparent, so I can make it sound better than that, but it is struggling. Why? Because we are in an era where it has been challenging, in terms of funding. But, more importantly, we're in an era of team science. We're in an era of strategic science. So, we need to define what Downstate and our community needs, and what role we can play on a national and international level.

And, in this era, we have access to a tremendously important population. A population that is underserved in terms of research that helps us understand how to care for them. Our population in Brooklyn is tremendously diverse. It is a population that, in the past, because of racial and ethnic differences, has not been participants in research. There's also been stigma to research which has, in the past, raised ethical concerns.

And what that leaves us with is a population that suffers a number of disparities, in terms of their care. But at least one of them, a very important aspect, from my point of view, is how much we understand about the appropriate treatments, and the nature of illness in those populations. So, I think we can play an incredibly important role in that – a role where we will be leaders in the national partnerships, the national networks of team science, because we provide a unique opportunity to partner in a way that will be directly applicable to the health outcomes in Brooklyn.

I will leave time for questions, or as much time as possible, but let me talk a little about what's happening in clinical care because we know our missions are education, research, but really tremendously importantly, the clinical health system that we are based in – that is our foundation, where our students and our researchers get to partner with our patients. And in healthcare right now, what we're seeing is a unique situation. We're seeing a very major transition in the way health care is delivered. And I will be, again, brutally frank. Right now, if we look at our major academic systems in New York, they are creating massive networks that are designed to provide a scale that allows for a value-health proposition. Now, what does that mean?

What we're talking about is that healthcare finance is shifting from paying for an activity, paying for a procedure, to something that will reflect a demand for demonstrable quality, where outcomes become incredibly important, and where the system deals with managing risk. Where health is what you're looking for, instead of the ability to treat illness. For that, you need scale.

So, all of these systems, Mt. Sinai, NYU, New York Presbyterian, Northwell, are expanding to create that scale, but they do it from a business-proposition point of view. In Brooklyn, what that means is, there's a desirable part of our population. They're more affluent. They most likely prefer to go to premiere settings in Manhattan or elsewhere. And at the same time, they present a risk pattern that is less problematic. They tend to have had ongoing health care and have been more participatory in their health care.

What happens to the remaining population, who we serve, who is our community, is now, in some ways faced with greater disparities. And the hospitals and the health systems where we are partnered, struggle to meet those needs. So, to me, the challenges are extraordinarily interesting. It may be overwhelming. I'm hoping not, and obviously I was aware of it and came with that knowledge. I think we have an incredibly important role to play with a population that is both very deserving, and underserved. And so I think this is an extraordinary institution.

"SH-T Happens" Fecal Transplantations

Noel Kleppel, MD '56 *earned a BA from Washington Square College, New York University in 1952 with a major in Psychology, and minor in Chemistry, Pre-Medical, with honors. He earned his medical degree from SUNY Downstate in 1956, and interned with Methodist Hospital and Long Island College Hospital in Brooklyn. Dr. Kleppel completed his residency in surgery at Long Island College Hospital, and the Veterans Administration Hospital in Brooklyn.*

He served in the United States Air Force Medical Corps, and was commissioned as First Lieutenant in 1957. He went on to several (continuing) surgery appointments and chairmanships at Caledonian Hospital, the Veterans Administration Hospital, Brooklyn-Cumberland Medical Center, Kings County Hospital, State University Hospital of Brooklyn, and the Brooklyn Eye and Ear Hospital, all located in Brooklyn, New York. Dr. Kleppel also undertook teaching appointments at SUNY Downstate and Touro College in New York City.

In October 1959, he published Fecal Feedings as Therapy in Staphylococcal Enterocolitis in the NY State Journal of Medicine, one of his most lauded findings.

My former colleague, the late George Degenshein, a surgeon and medical historian, and the former Director of Surgery at Maimonides Hospital, once told me that "in order to understand anything in medicine, you have to know its history for 300 years." I don't go back quite that far, but today I will give you all a bit of SUNY historical perspective as a lead in to my talk this morning.

Following World War II, with the return of many veterans and the availability of the GI Bill of Rights, it became apparent that not only would there be a need for many more physicians, but there was a four-year backlog of returning veterans whose education would not be hampered by lack of funds. The state of New York did not have its own medical school, and there was pressure in the legislature to create a new one. The Long Island College of Medicine was in financial danger of closing, but the speaker of the assembly, Irwin Steingut, who had been a patient at the Long Island College Hospital, got the idea of having the state pump money into an already existing medical school and saving it, thus providing an immediate answer to the state's need. He persuaded Governor Dewey and the legislature that this was the ideal solution and in 1949, SUNY Downstate admitted its first freshman class, which eventually became the initial SUNY graduating class in 1953. Each subsequent year another SUNY class was admitted, and in 1952, with the admission of my class, all four were SUNY classes.

Sixty-two years ago, in the spring of 1954, I was standing across the street from Kings County Hospital beside a sign which had been torn down and which read, "Flatbush tennis courts." Also there that day were Dwight D. Eisenhower, the President of the United States, Thomas E. Dewey, the governor of the State of New York, Vincent Impelliteri, the mayor of the City of New York, our Emeritus Dean, Dr. Jean Curran, Dean Howard Potter and other assorted celebrities. I had been

chosen by SUNY to be an usher at the groundbreaking for the Basic Sciences Building where many of you began your medical education, and which was then a vacant lot. A shovel was passed from one celebrity to another as they dug a hole which began construction of the SUNY Downstate Medical Center Basic Sciences Building on Clarkson Avenue here in Brooklyn.

> "I was a sophomore at the medical school when the groundbreaking was held, and a member of the fourth class to graduate from the SUNY Downstate Medical Center."

I was a sophomore at the medical school when the groundbreaking was held, and a member of the fourth class to graduate from the SUNY Downstate Medical Center. My class was the last class to graduate from the Henry Street campus downtown, adjacent to the Long Island College Hospital. I never imagined in 1954 that today, along with my classmates, some present here today, that today I would be marking our 60th graduation from medical school. As many of you will agree, it was not a walk in the park. It never was meant to be one. But since 1952, I have spent my entire professional career connected to this institution, except for a period as a US Air Force Strategic Air Command surgeon during the Vietnam era. So much for a bit of SUNY's history. But now, let's get down to business with some medical history involving some Downstaters.

Can anyone tell me who Jean Redman Oliver was? Dr. Oliver, who was the chairman of our Department of Pathology for 25 years, did the first microdissections of the kidney, and was the first in the world to describe the anatomy and function of the nephron.

Does the name Alexander S. Weiner ring a bell for any of you? Dr. Weiner, a LICM graduate himself, spent his entire career at the Brooklyn Jewish Hospital of LICM and Downstate, and there discovered both ABO typing and the Rh factor. He is responsible for saving countless lives with safer blood transfusions and the transfusion used for Rh babies.

How about Robert F. Furchgott? Dr. Furchgott, chairman of our Physiology Department, received the Nobel Prize for describing nitrous oxide as the signaling molecule in the vascular system. In addition to its functions in cardiovascular physiology, it is the basis for the development of drugs such as Viagra.

In my 64 years associated with this institution, I have had interaction not only with these three, but many others I consider great and memorable, and with the light of such momentous contributions such as theirs, I don't know whether it's something anyone usually is proud of, but if you don't already know – I'm famous for shit.

Some of you may already be familiar with one of the current treatments being used for Clostridium difficile enterocolitis, when total bacterial resistance to all available antibiotics is encountered. Gastroenterologists have begun to treat this disease, when they have no effective antibiotics available with what is now being called a fecal transplant. In this situation, physicians collect normal human feces specimens, and a quantity of processed material is placed into a gelatin capsule,

interestingly known as a "crapsule," which is then given to patients as treatment for this otherwise devastating and often fatal condition, where the Clostridium organism has proven to be otherwise resistant to all available antibiotics. And it works.

I will tell you how it all began, right here in Brooklyn, at the Long Island College Hospital of the SUNY Downstate system. About 60 years ago patients were being ravaged by a similar disease known as Staphylococcus enterocolitis. This almost always fatal disease was being seen in post-operative patients in the 1950s, early in the antibiotic era, at just about the beginning of my medical career.

When I began my postgraduate training, we had only two or three antibiotics available to treat our patients. They were penicillin and streptomycin, sometimes given together in a medication known as Combiotic, and a so-called "broad spectrum antibiotic" – the first of the tetracyclines. It was common practice at that time for surgeons to prep our GI surgery patients preoperatively, with a course of tetracycline in homes of eliminating potentially harmful bacteria and minimizing post-operative infections.

To a degree, this was beneficial. The normal bacterial inhabitants of the human small intestine and colon, such as Strepococcus faecalis, E. coli, and many other bacteria, were for the most part susceptible to the tetracycline. There was, however, another bacterial species, a rather bad actor, Straphylococcus aureus, many strains of which were NOT sensitive to the tetracycline and which were also resistant to the combination of penicillin and streptomycin in the Combiotic. Left unhindered and unchallenged in the GI tract, the resistant Staphylococci overgrew in the colon and in a number of our post-ops, within 24 to 36 hours, we were faced with a desperately ill patient dying of Staphylococcus enterocolitis and nothing effective to use for the treatment. The symptoms included a watery diarrhea with a characteristic odor, overwhelming sepsis, GI bleeding, sloughing of the colonic mucosa, severe dehydration, and then in most cases, the rapid death of the patient.

With no other antibiotics available at the time to combat the resistant Staphylococcus organism, we were helpless. We experienced a succession of almost a dozen post-operative patients who developed the disease and rapidly proceeded to die from the bleeding, the slough of the intestinal wall, the sepsis and dehydration. We soon became aware of this problem being reported in post-operative patients throughout the United States. In the fall of 1958, one of our own patients, who happened to have been an individual who was the "gun" of Murder Incorporated, developed the disease following a cholecystectomy at LICH, and within 48 hours had gone to his final reward. Ironically, in less than 48 hours, microscopic Staphylococcus organisms accomplished what his enemies and law enforcement had been unable to do for years.

Shortly thereafter, following a colon resection for carcinoma on another patient, Robert Kelleher, who had been given the usual pre-operative tetracycline prep, we noted the symptoms and signs of Staphylococcus enterocolitis, including watery diarrhea with flecks of sloughed colonic mucosa, hypovolemic shock, GI hemorrhaging and dehydration. We were at our wits end as to what to do to try to save this man.

That evening, following our dinner, I was sitting in the surgical office with my roommate, colleague and friend Dr. Louis Cutolo, our chief surgical resident and a 1953 SUNY Downstate graduate, reviewing and discussing the case and our frustrations, not knowing what we could do. Suddenly, a thought came to me and I said, "Lou, I have an idea." But I also suddenly realized that my bizarre thought was just that, bizarre, and said, "No, never mind, Lou. You'll say I'm crazy." "What's your idea?" Lou asked.

I tried to change the subject, but he insisted on my telling him what my idea was. I said, "Lou, why aren't there millions of zebras in the jungle?" "Why aren't there?" he asked.

> "How about if we just use normal human bacteria that normal human beings can provide and put normal human feces back into the patient?"

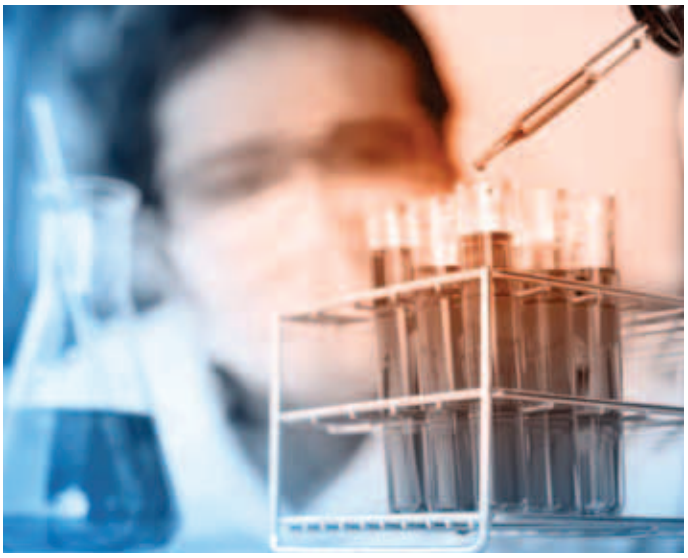
"Well," I said, "because there are millions of lions in the jungle and the lions kill lots of the zebras." And then I continued, "But how come there aren't millions of lions in the jungle?" And I answered my own question by saying, "Because there are lots of elephants and the elephants stamp out loads of lions." I said to Lou, "If you think about it, all life is a competition for the available environment. So, why don't we just make these resistant Staphylococcus organisms have to fight for their own existence by putting back other normal bacteria to compete with them for the environment?"

"Of course," I said, "we can't just put plain bacteria into the patient because we have no idea what concentrations to use and which combinations of bacteria to provide. If we guess wrong, we might kill the patient ourselves." "So, what do you suggest?" Lou asked. I said, "How about if we just use normal human bacteria that normal human beings can provide and put normal human feces back into the patient? We could feed it to the patient through a long Cantor tube. The distal end would be passed beyond the pylorus to prevent regurgitation, and maybe even we could give some back by enema." Lou responded immediately, "YOU ARE RIGHT. YOU ARE CRAZY." And I shot back, "Got a better idea?" He didn't have one and reluctantly, though I was out-ranked by my chief resident, he agreed to try.

We realized we had a problem. Where do you get normal human fecal specimens in a hospital which would be safe to use? Fortunately, this was 1958 and before IRBs. We decided that we could not use sick patients as donors, as we might be criticized, and we couldn't use doctors or nurses, since they were around sick patients all day. Cancer patients or those with infections were out—so where do you find patients in a hospital who are totally bedridden but not sick? I realized that we had a bunch of healthy orthopedic patients on another floor who were bedridden in traction. They seemed to be ideal donors.

We contracted the nurses on the orthopedic floor and instructed them on how to put sterile bedpans under these patients and how to transfer the specimens to sterile containers and to send them down to the surgical floor. There, we mixed the donor specimens with normal saline, creating a suspension. Using a bulb syringe, we injected it through a previously inserted Cantor tube, with its leading end beyond the pylorus. We also gave enemas of it mixed with yogurt and Lactobacillus acidophilus cultures, which we knew to be harmless, thrown in for good measure. We employed this regimen several times a day.

Within 12 hours, the diarrhea and sloughing had ceased



> "Our interest in resistant staphylococcus infections subsequently led us into a series of further research projects involving the development of other new antibiotics."

abruptly and the patient perked up. With appropriate hydration and electrolyte correction, in a day or two, the clinical Staph enterocolitis was gone and the patient was clearly improved. He recuperated from the disease and the surgery. Subsequent stool cultures showed normal intestinal flora. When he died about three months later of myocardial disease, at postmortem examination, we found an entirely normal gastrointestinal tract with normal bacterial flora.

During the course of his treatment, and the subsequent successful use on several other patients, we took a lot of ribbing. Somebody said, "You know Mr. Smith had a bowel movement today, but it was Mr. Jones's." One of our colleagues opined, "The only reason patients are getting better is that they didn't want to have to keep taking the medicine." We even heard comments that, "I told you the food at this hospital tasted like—." Well, you know what. A few cynics suggested we try to develop an intravenous preparation of our product, but our treatment worked. Not only on Mr. Kelleher, but also successfully on several subsequent patients. In 1959, we published the first paper in the world's literature outlining, "Fecal Feedings as Therapy." This became the accepted treatment for the disease until newer and more effective anti-staphylococcus agents were developed. Its recent rediscovery and use in the treatment of resistant Clostridium Difficile enterocolitis now being encountered is being reported today in our contemporary journals.

Our interest in resistant staphylococcus infections subsequently led us into a series of further research projects involving the development of other new antibiotics and more specifically research in treatment of other resistant Staphylococcus aureus diseases.

Lederle Laboratories in Indiana periodically provided us with samples of their new experimental agents called Vancomycin and Ristocetin, of which you have certainly heard, as well as several others. The two I have named have become useful in effective treatment of difficult Staph aureus infections and are employed frequently today. Both were developed using the rapid isolation and identification technique we developed and used in a Staphylococcus Bank we created back in the 50s and 60s.

One of the major problems with treatment of Staphylococcus enterocolitis is that its rapid onset and progression often proves fatal within 36 hours. We realized that rapid isolation and identification was essential in suspected cases and, Lou and I, working with Jeanne Holker, our hospital bacteriologist, developed such a method to accomplish this. We found that Staphylococcus aureus was the only common bacteria that survived in a 7.5% saline solution which killed all the other bacteria in a diverse mixture. We then subsequently developed a Staph aureus growth enhancer, Phenyl-ethyl alcohol agar medium, which would grow the bug lickety-split in three to four hours. Combining the two phenomena, we had a method for rapid isolation and then identification, as well as the antibiotic sensitivity results for an unknown mixed specimen, at least insofar as being able to rapidly respond to

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suspected resistant Staph enterocolitis. Newer methods and newer antibiotic agents have immeasurably improved our abilities today, but remember, I am comparing them to about 50 years ago.

First, we would shake up a mixed infection specimen with 7.5% saline, thus killing everything except the Staphylococcus. Then, we would pour the material on a Phenyl ethyl alcohol agar plate containing antibiotic sensitivity discs. In three to four hours, inspecting the growth around the discs, we could tell if we had a staff infection and what, if any, antibiotic it might be sensitive to, including some of the experimental products being developed. We did not have a large antibiotic armamentarium, but if we suspected Staphylococcus enterocolitis clinically and identified Staph growth on the agar, we could initiate logical and appropriate treatment rapidly using our method before the patient became desperately ill and succumbed to it.

Subsequently, we got the idea to develop a Staphylococcus bank. We circulated requests throughout the country and received, collected and categorized about 2000 Staphylococcus specimens along with their phage type pedigrees, and the clinical infection situations from which they originated. In those years, Staphylococcus organisms were identified and categorized by phage type profiles though today, with more sophisticated methodology, we identify various species using DNA profiles. With our 2000 pedigreed Staphylococcus specimens, we were able to evaluate in vitro any newly developed experimental agent and work with the pharmaceutical companies developing a series of anti-Staphylococcus antibiotics. Within a day or two of receiving a sample of the product under development, we were able to provide guidance to the pharmaceutical company as to whether it was an effective agent worth pursuing. At that time, we were even allowed to be paid for this work, and I confess that the Staphylococcus bacteria bought my wife's engagement ring.

So, I hope you can see how sometimes thinking out of the box, or being "crazy," if you prefer, can lead to advancements and useful methodologies in the treatment of human disease. And while today's gastroenterologists are treating Clostridium difficile enterocolitis with a somewhat more sophisticated method than we did, namely a "crapsule," our less sophisticated pioneering method did work at the time, and not only made us famous but proved, as the kids say today, "SHIT HAPPENS."

In closing, I'd like to add just a couple of thoughts. I have been very fortunate to have had a long and productive life. I have been involved in more fascinating and diverse things, besides the practice of medicine, than you can imagine. But in the 60 years since my graduation, the most rewarding thing I look back upon is sitting in front of me now. In 52 years on the Downstate faculty, my proudest accomplishment is the many of you, and the approximately 3,000 surgeons and 8,000 medical students I have had the honor of associating with, and helping to educate, and who have become the fine physicians you all are. Thank you.

Susan Krown, MD. '71 *is a medical oncologist involved in clinical investigations in HIV associated malignancies for over 30 years. Since November, 2010, she has served as the Vice-Chair for International Activities for the AIDS Malignancy Consortium (AMC), developing the AMC's international program in Africa, facilitating protocol development, logistics, training, and implementation, enhancing infrastructure, and building clinical trials capacity at these sites. Previously, she chaired the AMC's Kaposi's Sarcoma Working Group, charged with developing the AMC domestic and international scientific agenda for Kaposi's sarcoma (KS) prevention and treatment studies. She has also been the primary AMC liaison with the AIDS Clinical Trials Group (ACTG) in the development of two, large joint AMC-ACTG KS clinical trials being conducted in resource-limited settings in Africa and South America. Dr. Krown has published more than 200 peer reviewed articles and is the recipient of numerous awards for her research and humanitarian work.*

Challenges in Developing a Clinical Trials Network Treatment of AIDS-Associated Malignancies in Sub-Saharan Africa

Dr. Jenny Libien: Our next speaker is Dr. Susan Krown. She is a graduate of Barnard College, and received her MD degree summa cum laude from SUNY Downstate were she was elected to Alpha Omega Alpha. She completed her residency in internal medicine at Mt. Sinai, and then fellowship in clinical immunology and medical oncology at Memorial Sloan Kettering, where she has remained since, primarily working on HIV-associated malignancies, now as member emerita at Memorial Sloan Kettering.

She also rose through the academic ranks at Cornell University Medical College, becoming full professor of medicine in 1994. She has received a number of awards, including the Milstein Award from the International Society for Interferon and Cytokine Research, and the Women of Achievement Award from Barnard College.

Dr. Krown was also the SUNY Downstate keynote speaker at the 2008 White Coat Ceremony for entering medical students, a program that is sponsored by our Alumni Association. Dr. Krown is the author of over 200 publications, many of which are on Kaposi sarcoma. She has been the principal investigator on many funded clinical trials, sat on NIH study groups and WHO working groups. She has also reviewed manuscripts for journals, such as AIDS, Cancer, The New England Journal of Medicine and JAMA. We welcome Dr. Krown to her 45th reunion, and I very much look forward to hearing about her work with the AIDS Malignancy Consortium, for which she serves as Vice Chair of International Activity.

DR. KROWN: I spent a long time at Memorial Sloan Kettering, over 36-years, including my fellowship and time on attending staff. During that time, as part of my early studies of interferon in cancer, I serendipitously became involved with HIV-associated malignancy research. It was just the right time,

and the right drug, and the right patients, and I happened to see some very amazing responses of Kaposi sarcoma to interferon, and I got sort of hooked. I've been doing HIV malignancy research ever since. And then, in 2010, just as I was about to retire from my full-time job at MSKCC, I was asked to lead a new clinical trials initiative of the AIDS Malignancy Consortium in Africa, and that's what I'd like to talk to you about today.

So, the AIDS Malignancy Consortium, for those of you who don't know, is a multi-center cooperative group that was established in 1995 and was funded by the National Cancer Institute. I was one of the first chairs of the group, and we entered our first patient into clinical trials about a year later. We are one of the few clinical trials groups in the world that is dedicated to malignancies in HIV. And as part of our work, we try to establish the Standards of Care for treatment of AIDS-associated KS and AIDS-associated non-Hodgkin lymphoma. And during that time, when our activities were centered entirely in the United States, many of the people working in the AMC had ad-hoc collaborations with international investigators, and had established their own relationships with international investigators, both in South America and, most notably, in Africa.

As part of the recompetition of our grant in 2010, and with the prodding of the National Cancer Institute, our group expanded, and formalized our international collaborations, as part of a reorganization of our group and a recognition that a lot of stuff was happening in Africa. So, we added four sites in Africa in 2010, just as I was taking over as International vice Chair of the group, and then, late last year, in a round of competitive visits, we added three more sites in South Africa. The sites that we've recently added are in Mwanza, in Tanzania, which is on the shores of Lake Victoria, and very nice, but you have to take Malaria prophylaxis, Lilongwe in Malawi, and in Cape Town, South Africa. So that's where we're trying to build a research capacity and do research trials in HIV-associated cancers.

So briefly, the mission of our group is to investigate new treatments and prevention interventions for malignancies in people living with HIV, and to study the pathobiology of these tumors through clinical trials. And to this we added, in the US and internationally, to recognize the fact that we're now on a couple of continents. We're in three continents, because we just added a site in Australia, but they are not a low-resource area.

> "The mission of our group is to investigate new treatments and prevention interventions for malignancies in people living with HIV, and to study the pathobiology of these tumors through clinical trials."

Our goals are to improve the health of people infected with HIV by improving treatment and prevention of cancers, to evaluate new treatments and prevention approaches, and to contribute to the scientific understanding of the pathobiology of cancers in the setting of HIV. I think many of you are aware that cancer was one of the first heralds of the AIDS epidemic in the United States, and cancers were among one of the first AIDS-defining conditions, as I've already mentioned, Kaposi sarcoma, which is how I got into this business. That and (could not identify type of pneumonia from the recording) pneumonia were the first two AIDS-defining conditions

> “There are tremendously high rates of HPV infection in Africa, as well as a lack of a cervical cancer screening infrastructure.”

back in 1981. Soon after, non-Hodgkin lymphoma, and invasive cervical cancer were added to the list of AIDS-defining cancers.

But there are many other so called non-AIDS-defining cancers that occur in excess in HIV positive people. Many of them, and not all of them, are infection-related or associated with immune-suppression. But HIV itself may be associated independently with increased risk for these cancers, and it's not clear how. HIV burden is clearly highest in Sub-Saharan Africa, and probably accounts for 70% of the global HIV burden.

I also wanted to make the point that the risk and spectrum of HIV cancers may vary by geography. So For example, in Africa there are very high rates of KSHV, and KSHV is the herpes virus that is both associated with the development of both Kaposi sarcoma, and well as some other conditions including primary effusion lymphomas, and Castleman's disease which is more of a problem in Africa than people realize.

There are tremendously high rates of HPV infection in Africa, as well as a lack of a cervical cancer screening infrastructure. There are high rates of chronic Hepatitis B and C, which are associated with hepatocellular cancers, and high rates of an unusual neoplasm that's most seen in Africa which is mostly ocular squamous neoplasia which may be related to HPV or may be related to ultraviolet radiation, the studies are inconclusive, and still ongoing. And there may be roles of other infectious or genetic or environmental cofactors that have to be explored.

I just want to give you an idea on why it's so important. And this is data from cohorts in Europe or North America and people who initiated modern day anti-retro-viral therapy between 1996 and 2006. You may say, well, we've got all these great drugs for HIV, should we be worrying about this? Well, this is a retrospective study of close to 40,000 patients, enrolled in 13 HIV cohort studies, where they had a definitive cause of death in close to 1600 patients.

34% of the deaths were AIDS-related, including cancer. But a nearly equal number were linked to either AIDS-related or non-AIDS-related malignancies. So, 14% of the total was AIDS-related malignancies, and almost another 12% were non-AIDS-related malignancies, so you have 26% of the deaths in that group that were from, attributed to, cancers. Where a lot of people are making a lot of noise lately about the importance of HIV infection's role in cardiovascular disease, you can see that, not that I want to diminish the importance of research in cardiovascular disease and stroke, but the deaths attributable to those were far less than those attributed to cancer.

So, what does this mean in terms of the incidence particularly of AIDS-associated cancers? These are data, again, from a couple of very large cohort studies involving over 65,000 women, and these were presented just a few months ago at a conference in Boston. And this is looking at the hazard ratio for invasive cervical cancer five years after starting ART in HIV-positive women.Compared to Europe and North America, women in Latin America, and especially southern Africa, have a tremendously increased risk of invasive cervical cancer, even five years after starting effective antiretroviral therapy. You can see that the risk in southern Africa is more than 12 times higher than the risk in Europe and the United States.

This is not explained by differences in their T cell count, their age, or how many years they had been on ART. And in southern Africa, the rates did not decline with increasing time on ART, so this seems to be a particularly susceptible group. These rates may be linked to higher incidence and prevalence of HPV infection, but also probably to limited access to effective cervical cancer screening.

Another tumor, we'll call it KS instead of Kaposi, it makes life easier, and this just shows people in Uganda who were just starting on ART and who were otherwise similar in their characteristics, except one group had KS when they started on ART, and another group did not, so you can see here, that just by having KS, the cumulative mortality at four years was 30%, compared to only about 5% or 7% in people who didn't have KS.

I don't know how many of you as clinicians have seen patients with Kaposi, but here you're talking about 30,000 cases per year, which is a lot. And this just compares it to the US where the KS incidence is vanishingly small.– while the incidence of non-Hodgkin-related sarcoma is maybe similar to that in Africa, the mortality rate is considerably lower in the US.

So you can see the relative weights and the difference in mortality between all of those diseases between the African sites and the US. I've spent the first part of this telling you that there's a lot of cancer there, and it's bad, and people die. And so, what can we do to make it better? And what are the challenges and barriers that we're facing? I've divided that into four parts: diagnosis, treatment, certain societal elements, and things that are related to clinical trials instead of general care; and I'm going to briefly deal with those.

So, diagnosis is a big challenge. In Africa, late presentation is very common, and that's a function of lack of screening programs. There are many other things happening to people – people's health in Africa that may mimic certain cancers, and knowledge deficits among practitioners and the general public about how to recognize cancer. Or issues relating to the fact that many times, and this is particularly true for Kaposi sarcoma, that the diagnosis is made clinically without a pathological diagnosis. And, I'll show you why, if you don't already know, why that's a problem.

"There is also a tremendous scarcity of not only pathologists, but also pathology laboratories that can adequately process tissue"

There is also a tremendous scarcity of not only pathologists, but also pathology laboratories that can adequately process tissue, and often there are problems with poorly fixed tissue, tissues sitting around for weeks before it ever gets fixed, and that's a big problem. And finally there's a lot of understaging of disease, because they have limited resources to detect visual disease, so you're lucky to get a chest X-ray and maybe an ultrasound, but access to CTs and MRIs, and forget PET scans and even bronchoscopies and even GI endoscopies, is quite unusual. I think that the bad statistics that you see about treatment of things like cervical cancer, for example, relates to the fact that they're understaging patients because they're doing it without modern methods of staging. There are some attempts being made to increase recognition.

"When you have people presenting with late-stage disease, their survival rate is far inferior than those who present with earlier stage disease."

There is a poster from my friend Margaret Borok from the University of Zimbabwe, which they've produced in both English and Shona, which is the local language, that they've posted in clinics, just to make people aware of what KS lesions look like so the patient can point it out to their caregivers, and also the healthcare professionals can be more aware of what the disease looks like, particularly in early lesions. This is sometimes challenging in dark-skinned individuals where the lesions may look like hypertrophic scarring, or something people don't really notice.

This is important because when you have people presenting with late-stage disease, their survival rate is far inferior than those who present with earlier stage disease. And this is a study that was done by my friend Fred Okuku at the Uganda Cancer Institute showing that the extent of KS was the single-most predictive clinical factor associated with survival. There's also the whole issue of, when you're making a clinical diagnosis and all you have is a chest X-ray, what do you do with an X-ray . In Africa, the first thing you think of is tuberculosis. And these are actually patients I've had in New York. They're from my own practice. But there's a paper that recently came out from Malawi showing that TB typically delays a cancer diagnosis and these patients that they identified were diagnosed clinically, treated empirically, and only when they didn't respond did someone think they should biopsy something, like a neck mass, and 23 were diagnosed with lymphoma. Not all of these patients were HIV positive, but some of them were known to be.

There's also this over-diagnose of KS in Africa because people are looking at clinical appearance of skin lesions without pathologic confirmation. And in an ongoing study, about 10 % of patients who were thought to have KS had another skin condition on biopsy. In another study, 19% of over 700 lesions of people where there was a clinical suspicion of KS had another diagnosis when they were examined pathologically.. It's important because if you're going to jump in and give chemotherapy based on your clinical impression, and they really have syphilis, or tuberculosis, you should be doing something else. So, that's a problem.

This is from a recent paper, just looking at the tremendous lack of pathologists and also oncologists in Africa. I won't go into detail on this blow-up, but we're showing the number of pathologists to million people in Africa. It's about 4 ½ per million population. In the United States, it's 57 per million population. Some countries have no pathologists, and it's a problem. See, Jenny? I told you I was going to talk about why pathologists are important.

Okay, so, from a therapy standpoint. There are even more

> “In Botswana, which is a fairly rich country, they actually give free care to everyone, and there are tremendous delays to access to cancer care, even with free care.”

challenges. I'm not going to read off everything here, except there are problems in getting chemotherapy, in being sure if it's any good, because they sometimes get it from some pretty strange places. There are very few arrangements for staff and patient safety. I think on one of my first trips to Africa, I found a nurse was drawing up Adriamycin in a syringe in an open room and then sort of pushing the air out of the syringe. And fortunately that has changed at that site. Now they have a hood, and take precautions. But this is not good for patients, this is not good for nurses, this is not good for anyone.

Tremendous radiotherapy equipment shortages, and I'll show you that in a moment, very few trained surgical oncologists. There are surgeons, but to do cancer operations, it's tough, and very little in the way of supportive care that allows you to manage adverse events, to deal with pain and all the baggage that goes with cancer treatment. And this is just a slide that shows you where there is radiation therapy in Africa. And a lot of these – I can tell you that the one in Uganda is there, but it's non-functional because cobalt sources died and they're trying to get another one showing a tremendous imbalance between where there is a growing need for radiotherapy machines and where radiotherapy machines have been added.

There are also tremendous societal and cultural challenges, including payment for care. A lot of patients – I remember a friend who worked in Kenya for a number of years said that sometimes people would have to go home and sell a couple of goats before they could come back and afford to be treated. And there is a lot of poor coordination in the whole healthcare system.

In Botswana, which is a fairly rich country, they actually give free care to everyone, and there are tremendous delays to access to cancer care, even with free care. This shows tremendous loss to follow-up rates for patients with Kaposi sarcoma.

I did want to tell you more about our accomplishments, but we seemed to have run out of time; so I'll stop here, except to say, that we are doing clinical trials in KS in advanced cervical cancer, and in lymphoma. We've also set up a pathology training and certification process for Kaposi sarcoma and lymphoma and have been working hard on developing a clinical trials infrastructure and training so that people know how to do their job. So, I think I've shown you that cancer is an important contributor to morbidity and mortality, that it's a very big problem in Sub-Saharan Africa, and there are many barriers to successful management, but I think we're making progress.

Mark Stewart, MD. '91 *is a graduate from the College of Medicine at SUNY Downstate. He currently serves as Professor of Physiology & Pharmacology, and Neurology; Dean, School of Graduate Studies; Vice Dean for Research. His extensive involvement in both the summer research program and in supporting our research award committee has been invaluable. He has trained 16 Postdoctoral candidates, 9 predoctoral graduate students, 2 visiting scientists, 13 other predoctoral students, 1 masters student, and 17 undergraduate and high school students as a researcher. He teaches in both the College of Medicine and in the School of Graduate Studies. He has published more than 60 research papers, 11 proceedings papers, 11 books or book chapters, 96 research abstracts, and is currently best known for his research into Epilepsy.*

> "A sudden death in epilepsy is defined by a sudden death that is not traumatic."

An Explanation for Sudden Death in Epilepsy

Dr. Jenny Libien: Our next speaker is Dr. Mark Stewart, who is an MD, PhD graduate of SUNY Downstate's class of 1991, so it's his 25th reunion. He is Dean of the School of Graduate Studies at SUNY Downstate, Vice Dean for Research in the SUNY Downstate College of Medicine, and professor of Physiology, and Pharmacology, and Neurology.

He was awarded the Downstate Chapter of AOA Alumni Membership in 2014, and has over 60 publications in journals such as Epilepsia, Cardiology, Journal of Neurophysiology, Hippocampus and the Journal of Comparative Neurology. He has been grant-funded by the NIH and has developed two SUNY Networks of Excellence in brain and health. And he's also the Brooklyn psychology director of the New York Academy of Sciences afterschool mentoring program, which brings young SUNY scientists to middle schools to improve the math and science literacy of Brooklyn children.

He has been a mentor to many students and residents at SUNY Downstate, and today he's going to speak to us on his research on the causes and consequences of seizures. We welcome him as he celebrates his 25th reunion year, and his talk will be on an Explanation for Sudden Death in Epilepsy.

Dr. Stewart: Thank you this is quite an honor to be asked to present here, to this group of alumni. In following with Dr. Kleppel's first talk, I'll recall Chandler Brooks, who was the founding Dean of the School of Graduate Studies, the first dean. I'm the fourth, and my two degrees of separation from Brooks is, still an active mentor for me.

What I'm going to talk about today is sort of the punchline of a bunch of experiments we've been doing for more than eight years now. We've been exploring autonomic cardiovascular, and respiratory consequences of epileptic seizures in a rat model, the goal being, the most severe consequence, death, that is termed "sudden death in epilepsy." there are two published definitions for sudden – that a sudden death in epilepsy is defined by a sudden death that is not traumatic.

It's not drowning. If you had a seizure and crashed your car, that's not "sudden death in epilepsy." In fact, a weakness in the entire exploration of sudden death is that the diagnoses are labeled such when there is nothing on post mortem investigation to point to as the cause of death.

When there is, the death gets labelled by that cause, and pulled out of the sudden death category. So, what we're left to look at is a mixture of things many people haven't been quite sure how to approach. The general thinking is that a seizure is essential to trigger some set of events that ends in death within a short period of time. The natural mechanism for linking an abnormal electrical brain activity pattern to the heart or respiration is obviously the autonomic nervous system, and it's clear from various kinds of data, that respiratory derangements are as potentially important as cardiovascular; but that's where we sat for a long time. What I'm going to propose is actually more or less a definitive description of potential outcomes with some prioritization.. The hippocampus is notorious for learning and memory, but the same features that confer the ability to learn on a cortical structure put it at risk for electrical activity that's abnormal, namely, seizure activity. We studied this normal activity pattern in animals that were anesthetized with a drug called urethane, and so it took a little bit of time, but we came to the idea that we might try exploring seizure activity in animals that were anesthetized with urethane.

The result was, we were able to get beautiful seizure activity using a chemical convulsant, kainic acid, which is a glutamate receptor agonist. The remarkable feature of urethane is that that animals don't show motor convulsions, but they do show the motor activity in hippocampus, it just does not spread to neocortical areas to activate motor cortex. It does spread through hypothalmus and brain stem.

So we have an anesthetized animal that is not convulsing, showing limbic cortical seizure activity just like temporal lobe epileptic patients with the full set of hypothalamo-medullary cardiovascular, respiratory changes associated with seizure activity. So this allows us to instrument the animals in a variety of ways, and make recordings during seizure activity of a large

> "We were under the impression there was no way a seizure could kill you."

number of parameters, many of which I'll show you here to get to the full set of consequences. This is basically everything that can happen to you as a result of a seizure. We'll spend our time talking about the bottom, the punchline. But the first points that I'll make like this is that what's clear, through our work and others' work, is that when a seizure occurs, it very strongly activates regions of hypothalamus and medulla to increase the activity of both sympathetic and parasympathetic divisions of the autonomic nervous system. Each seizure will increase the activity in both divisions. What you see in the outcomes, in terms of a brady- or tachyarrythmia, depends on the relative activity and the patterns and amounts of activities in those two subdivisions.

In most cases, what happens is the seizure activates those two divisions, you have some cardiac derangement that's evident. In many cases, it's a mild tachycardia, the seizure ends, the stimulus for the autonomic derangement goes away. . In that case, the cardiac output drops to essentially zero, the brain blood flow decreases, the seizure stops because it's not supported by brain blood flow, the stimulus for the autonomic derangement goes away, and again, you come back to baseline.

So, for the first three or four years, we were looking at things. We were under the impression there was no way a

seizure could kill you. Something else had to happen. And one of the examples people have thought about, as a result of your convulsion, perhaps you go face-down in the pillow of your bed, and because of the derangements in muscle movements, postictal depression, where you're not moving very well after the seizure, you basically suffocate. That was one option, and I'll explain some of those things in a bit.

But I want to talk about two possible ways to get to death. What we started talking about was perhaps the seizure can produce a tachyarrythmia, a ventricular tachycardia that would even devolve into ventricular fibrillation at that point, even if the seizure stopped, because of the cardiac output collapsing. You're going to expire unless there's resuscitation. And the last step, we'll come to.

But first, this bit on whether a seizure might elicit ventricular fibrillation. We spent a good deal of time looking at ways to manipulate the physiology in order to get here, and the answer is that you can get there, in a manner of speaking, with a set of conditions that are hard to configure exactly the right way. The preconditions are a near absence of parasympathetic tone. The vagus is a very protective nerve. And so what we were doing, in many cases, was actually completely eliminating vagal input into the heart by cutting the nerve. You need a sympathetic activation, and we were using isoproterenol and the doses of isoproterenol needed to be above the doses you would use to get to a maximal heart rate, or actually probably crossing over the beta receptor activation, and likely producing ischemic events in the heart itself.

The cases where we were able to initiate ventricular fibrillation had an oxygen saturation change that was just right. Too fast and too deep, and you produce non-sinus bradycardia, not deep enough you develop a slight sinus bradycardia, "just wrong" gives you the conversion to ventricular fibrillation.

Normal sinus rhythm spontaneously converts to a short run of v tach and then v fib under a right set of conditions that are properly configured. The interesting thing here, , is us restoring vagal activity with a high-frequency vagus stimulus. When we do, we actually convert the v fib back to a normal sinus rhythm. That's actually the basis for a device that we have that solves all of the problems of the current ICD device in terms of lead failure and things like that. So this device is just developing. In fact, I just heard yesterday that we got a small grant to move this out of the Valley of Death, so we can potentially get this to market.

The closest we could actually get to conditions we'd see in a seizure triggering ventricular fibrillation is this example of vagus nerve over-activity from stimulation. I've suppressed the shock effects here, that we needed to do in part for the device. So, the run of v fib starts at the top. And in the second sweep, there's a flurry of beats of ventricular tachycardia, If we imagine the vagus stimulus had stopped somewhere in the middle of this run of v tach, I would expect this might

Epileptic hearts are enlarged, but in fact the dilation is an eccentric type of hypertrophy, which means the heart is enlarged, but the walls are not thickened. So, our thinking is that the actual path links for the electrical conduction of the heart are longer, and makes it more resistant to entering type A arrhythmias.

The real action is here, where we've been looking for a way in which we could produce a period of much more severe hypoxia as a way of getting us to death, so we started a collaboration with otolaryngologists, developed an laryngoscope for the rat, a more sophisticated version made by a machinist in our department, , with the arytenoid cartilages at the bottom of this cross-shaped structure and vocal folds at the top.

With this type of imaging, we can plot the movement of the tips of the arytenoid cartilages, essentially track the opening and the closing of each half of the airway. . So, we have a sensitive measure of tracking what's happening in the airway during a seizure.

So, these are the first real descriptions of what's happening to the airway during a seizure, and we can do this again because the animal is not convulsing. . ,, the first is the respiratory rate is higher. The second is that, even though the rate is higher, these are not synchronized, if you actually look at the arytenoid cartilages on video, the airway is mostly open.

That's not going to be a cause of death. So, we started to make recordings of recurrent laryngeal nerve, and to look a bit deeper, an animal that was recorded with a window cut in its trachea. During that time, we're recording from recurrent laryngeal nerve, , so a seizure is progressing and stops. So, what you're seeing in the recurrent laryngeal nerve is natural bursts firing with each breath. But an increase in activity, including the recruitment of new, firing new units gets to be quite strong at this point, and then calms back down before the seizure is over, suggesting very intense laryngeal activity. When you actually look at the airway during these episodes, the airway is completely closed from laryngospasm.

So, we have a seizure-induced storm, essentially, on the recurrent laryngeal nerve that produces a complete airway closure, obstructive apnea that', a significant change in the EKG, ST elevation in the EKG. Very quickly, we get hypoxic

"Epileptic hearts are enlarged, but in fact the dilation is an eccentric type of hypertrophy, which means the heart is enlarged, but the walls are not thickened."

changes in the EKG.

In these animals, there are also episodes of central apnea, meaning, there's no evidence of the animal attempting to breathe.

In contrast, there is no change at all in the central apnea cases. Interestingly, in the seizure-induced central apnea episodes, the airway is open, so it's held actively in an open state. We actually think this is very similar to what you would observe in the case of just someone holding their breath, as opposed to somebody where you're covering their face and they can't breathe; two very different experiences.

And so the critical aspect in terms of physiology deteriorating is the laryngospasm. As a way of exploring the airway obstruction with a bit more precision, we simulate the airway obstruction from laryngospasm by just closing essentially, a failure to pump by the time the animal arrests.

And, with this model, we can very precisely tell the point of respiratory arrest. So, in terms of sequence of events, we can identify, very accurately, the timing of the onset of a com-

> "So, the combination of what started with the seizure and continues with the airway obstruction looked to us like a very good series of events that would lead you from seizure to death."

plete occlusion to the point of respiratory arrest.

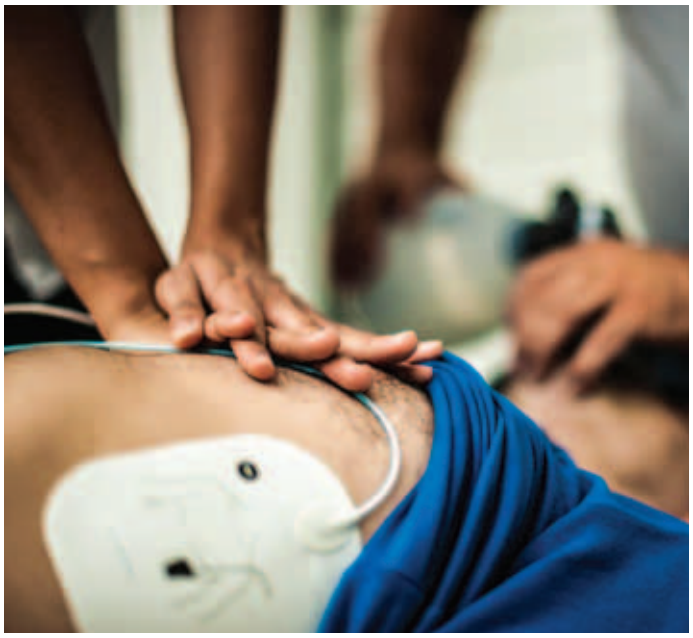
This is a summary of some of those examples where, from the point of occlusion, which is the second point where the heart rate is dropping, the respiratory rate is slowing down, the effort to inspire increases, and goes to zero when that effort stops. So, there are a lot of variables that we have a good understanding of. What's interesting about the autonomic response to the airway occlusion is that it's in the same direction as the autonomic changes induced by the seizure activity in the first place.

So, the combination of what started with the seizure and continues with the airway obstruction looked to us like a very good series of events that would lead you from seizure to death. Right now, we have a paper in review that's in its sixth month after only two revisions by us, and the main problem is that this report, which is a paper published in Lancet Neurology, is describing the results of something called the MORTEMUS study . For them, terminal apnea is occurring sometime after the seizure ends. The seizure ends for them, in their recordings. So, when we talk about seizure-induced obstructive apnea, what we get from the reviewers is that apnea doesn't occur until after the seizure is over.

Their evidence that apnea is occurring after the seizure is over comes from records that they've obtained from the epilepsy monitoring unit, which is a simple EEG, and usually one or two EKG leads, essentially a rhythm strip. When you look at those recordings, there are these artifacts in the records that are clear indications of some type of respiratory effort. So, in their study, whenever they have these, they're concluding that the individual is still breathing until they stop seeing them, at which point they label it terminal apnea.

So, the best evidence of artifacts, in our opinion, is occurring when the animal, or human, is actually obstructed. This animal was ventilated, hyperventilated, and we stopped the ventilator. So, we suppressed its natural drive to breathe, stopped the ventilator, the animal starts trying to breathe, but since the system is closed, that effort increases, and you see the same type of artifacts, here.

The neat thing is, we have a second bio marker, here. The first bio marker is the presence of these artifacts, which are beautifully correlated in their size to the amplitude of the attempt to breathe. The second bio the RR interval is plotted as a function of time, and what you see is a tiny variation of the RR interval, and then the variant blows up. This is all during the occlusion. If you look at the relation of the RR interval with the peak effort to inspire, the normal pattern of RR interval to



> “When you get to that point of respiratory arrest, the game is over unless somebody resuscitates you.”

lengthening the effort to inspire is shown in the top, and we get two short intervals only during these intense attempts to breathe, not during the first part of the obstruction, and even not during a missed breath, late in the obstruction.

So, that's our summary, and the key is reaching that point of respiratory arrest. When you get to that point of respiratory arrest, the game is over unless somebody resuscitates you. So, I've talked about our two bio markers, and the alternative mechanism for SUDEP mainly being ventricular fibrillation. Interestingly, in the fibrillation example, we wrote a review on this, there were three cases published, and we were told by the editor that there's a fourth case and, actually, when we looked at that fourth case, it was a person who came in and they got whacked with atropine, which is step number one for us, got wacked with epinephrine, which is step number two, and had his own issues with hypoxea. He's a living example of our pharmacological experiment.

The nice thing now is that we have an animal model that allows us to, rather aggressively, test manipulations that can be used to intervene. If somebody starts one of these episodes, of obstructive apnea, to potentially prevent them.

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Jeffrey Farkas, MD '91 (in absentia) • Carl J. D'Orsi, MD '66

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SUPPLEMENTAL ISSUE
DRUMROLL PLEASE

Monica Sweeney, MD '75

Global Engagement at SUNY Downstate

Dr. Sweeney, President of Alumni Association and Vice Dean of Global Engagement at SUNY Downstate School of Public Health

In September 2015, I was appointed to the faculty of SUNY Downstate School of Public Health. My late husband once said that, "I had done everything at SUNY Downstate except be a patient." Well that's not quite the case, and it was well before the School of Public Health was founded. I was appointed as the Vice Dean of Global Engagement at SUNY Downstate School of Public Health. My role is to provide leadership for the School of Public Health's numerous globally engaged teaching, service and research activities both locally and internationally. Additionally I am a Clinical Professor and the Chair of the Department of Health Policy and Management.

In May 2016, I was inaugurated as the President of SUNY Downstate College of Medicine Alumni Association. The timing of my inauguration as the President of the Alumni Association and my joining the faculty of the SUNY Downstate School of Public Health was fortuitous. One of the activities supported by the College of Medicine Alumni Association is that fourth year Medical Students can apply to take part in the overseas Global Health Elective to under resourced countries. Being involved with both the School of Public Health and the Alumni Association has allowed me not only to be on the forefront addressing health disparities, but also influence the next generation of healthcare providers.

As an advisor and mentor to MD/MPH students enrolled in various programs in the School of Public Health, I am able to help guide them to reach their professional goals. This of course includes helping Medical Students develop and increase their understanding of their role as healthcare providers, as part of a team here and around the world. I work to ensure that the enthusiasm, the sensitivity, and desire to make the world better which most Medical Students have upon entering Medical School, continues to be nurtured throughout their education. Students are often anxious to be sure their education will serve them well, and I do my best to help ease their apprehension.

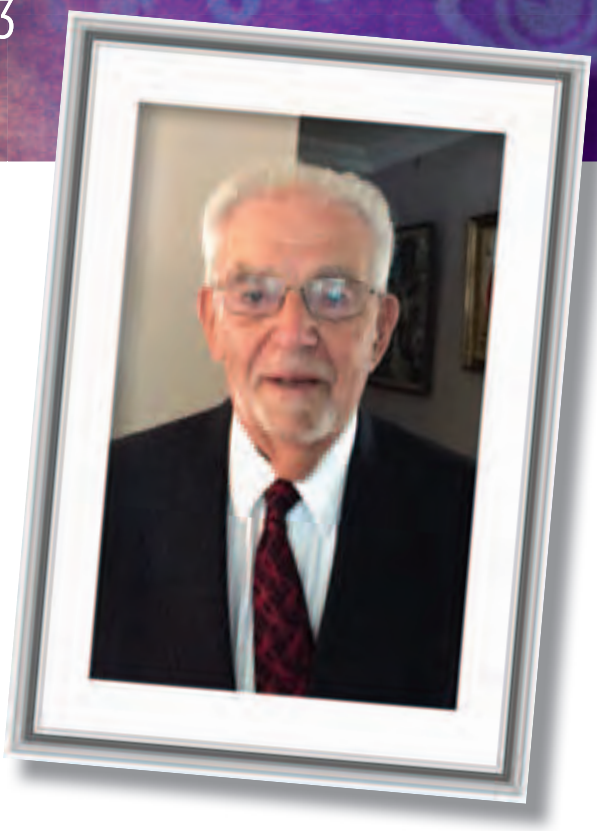
All of my previous professional experience has provided me with a comprehensive and sensitive understanding of existing population health challenges on a local, national and international level. I continue to work regularly with Medical Students who want to go to sites overseas that have not yet been approved. The activities involved in the Global Health Elective are extremely rigorous, since safety is our number one concern. So working with the Medical Students is rewarding when we are successful in establishing a new site or increasing the numbers of students at established sites, or when students return enriched and excited by their experience.

I am reminded of a particular student who took the Global Health Elective and what he shared with me during his debriefing. He said that it was the best, most important and most enjoyable course that he had taken in his four years of Medical School. He shared that he had heard about and given lip service to cultural competency for years. It was not until he was in a small village, and observed how the locals handled and cared for the disable population that the true meaning of being culturally sensitive really sank in. He went on to say, that at first he thought the local way of dealing with the disabled was all wrong. However after a while, he understood what and why they approached and handled the disabled population the way they did. By the time he left, he said their approach was superior to the United States for similar conditions. He was transformed by the experience. His mastery of Science was strong before he left. His sensitivity to the human condition was improved upon his return.

After all these decades in Medicine, I continue to maintain the same enthusiasm and passion for my calling as a physician, as an educator, and as a humanitarian, which I try to impart to my students.

Portrait of a Donor – Marvin Kochman, '53

DOCTOR, TEACHER, ATHLETE, WORLD TRAVELER



By Constance Shames, MD '63

Dr. Kochman was born in Brooklyn and has spent his entire medical career practicing in Brooklyn. He notes that he was always interested in how things work and was especially interested in the human body. He chose ophthalmology as his specialty because it allowed him to emphasize on the entire body and neurology as well. He became a surgeon as he has always had excellent finger dexterity.

After graduation from Downstate he studied with Dr. Richard Troutman and then spent time in the military (Captain U.S. Air Force). When entering private practice he was able to open the first licensed ambulatory surgical facility in New York (Brooklyn) and the second one in New York State.

While in medical school he became friendly with Dr. Randy Bloomfield whose mother started a group to help students at Downstate. Dr. Kochman's parents also became involved and after graduation Dr. Kochman attended an Alumni Association Board of Managers and this began his early interest in advocacy for scholarships for medical students.

In a career which spans over 60 years Dr. Kochman he remains as Senior Partner in the Brooklyn Eye Surgery Center, Fellow in the American Academy of Ophthalmology and Fellow in the American College of Surgeons. He is also a member of numerous professional societies and affiliations. He has served as an instructor in the Department of Ophthalmology at Downstate and as guest faculty at Manhattan Eye and Ear. He has a number of publications in New York State Journal of Medicine, as well as others.

Since his retirement in 2000,his major interest at the present is to support the school and the Alumni Association. He initiated the Kochman Family Fund for scholarships for medical students at Downstate and was the driving force to start the 1953 class fund. He always thinks of Downstate and medical school as the place that allowed him to achieve his dreams. "I owe everything to Downstate" he says frequently. He has served as President of the organization as well as Chair of the Board of Trustees and remains a devoted member who continues to advocate for students and alumni.

Dr. Kochman has traveled all over the world to such places as the Artic and the Antartic. He has been all over Europe, China, Israel, Jordan, Turkey New Guinea and more. He loves athletics and has been an avid skier, hiker, tennis enthusiast, and he loves to read history books.

Dr. Kochman has distinguished himself as a major force and a voice for progress for our students and our association,

David H. Abramson, MD, FACS
Chief, Ophthalmic Oncology Service
Memorial Sloan Kettering Cancer Center
Professor of Ophthalmology,
Weill Cornell Medical School

SUNY Downstate – Ophthalmology Lecture

Douglas Lazzaro, MD, Chairman of Opthalmology, SUNY Downstate: Before I introduce the speaker, I want to bring up the Dean of the College of Medicine, Dr. Carlos Pato, to say a few words to the audience.

DEAN PATO: As many of you heard during Match Day, I'm a strong proponent of celebration, and excellence is something that is not recognized often enough. So, I want to congratulate you, as well as your residents and your faculty who have assisted you in achieving this. So, congratulations.

Douglas Lazzaro, MD '90 Notes: What is Alpha Omega Apha? It's a professional medical organization, actually the only one in the United States, that recognizes and advocates for excellence and scholarship and the highest ideals in the profession of medicine.

AOA was started in 1902, the first chapter in the United States, and currently 133 chapters exist for US medical schools. Downstate was Chapter 49, started in 1948. There are numerous categories one can be elected into, into AOA, as a student, as a Junior AOA, and a Senior AOA, as a house staff member, based on the size of the institution. Here at Downstate, we can have up to six house staff. Up to two alumni can be elected, up to two faculty members. There's one honorary faculty elected every year, and then there's a charter member of a new chapter.

> "AOA was started in 1902, the first chapter in the United States, and currently 133 chapters exist for US medical schools."

What's the mission statement of AOA? It's dedicated that, in the profession of medicine, we will improve care by recognizing high educational achievement. You all have done that. We honor gifted teaching, by the way the faculty, in particular, the honorary faculty member is selected by the AOA students. It encouragements the development of leaders in academia and the community, supports the ideals of humanism, and promotes service to others. Even though healthcare is changing radically year by year, these are all things that you can take in your careers for the next thirty and forty years.

You can go onto the AOA website that tells you all the categories of elected positions. There are 57 Nobel laureates. Downstate had one in 1998, Dr. Robert Furchgott, for his work with nitric oxide. He was the AOA faculty member in 1967. I had the honor of having him as my pharmacology teacher. After tonight, going back to the 1940s, we will have had more than 2,200 members. By the way, you all have been nominated, but to complete the process, you will have to connect with the AOA national society, send in your dues, to be an official member of AOA.

Now, some words on our speaker today. Dr. David Abramson is a tenured professor of pediatrics and ophthalmology at the Weill Cornell Medical School, currently running the largest ocular oncology service in the United States, and

the largest retinoblastoma service in the world. He actually is the founder of the ocular oncology service at Memorial Sloan Kettering after serving many years, doing similar service at Cornell. He's a graduate of Albert Einstein Medical School, resident of ophthalmology at New York-Presbyterian. He's published over 400 books, chapters and original articles, and over 340 peer-reviewed articles. He's the editor of the American Academy of Ophthalmology's instruction book on ophthalmic oncology, and he's delivered more than 400 lectures, worldwide.

AOA Mission Statement

In the profession of medicine, we will improve care by recognizing high educational achievement.

He's received awards from all over the United States and beyond, including the Swiss Ophthalmological Society, the Association for Research in Vision, the Hellen Keller Society, a lot of teaching awards, really, really, a great teacher, and I think you'll enjoy his lecture. He's received the honor award, and the senior honor award, and the lifetime honor award from the American College of Ophthalmology.

He's publishing constantly, and improving the field. When I was a resident in ophthalmology, most of the eyes that had retinoblastoma were enucleated. Currently, under Dr. Abramson's leadership, only 5% of these eyes currently get enucleated. He and his team are single-handedly changing the way this devastating disease is managed. So, without further ado, Dr. David Abramson, it's an honor to have you give the AOA lecture.

Dr. Abramson: Thank you, my friend, Doug. Congratulations, to all of you, it truly is a huge honor, and something you will carry with you, the rest of your lives. I salute you. I'm thinking about my comments, and I think I should be giving you wisdom, since I graduated from medical school almost 50 years ago.

And then I was thinking, well, I don't have that much wisdom, so it would be a very, very short talk. And you know, when I finished medical school, I didn't know how much about medicine I understood. When I finished my internship, I thought I had some level of competency. When I finished my residency, I thought, I'm pretty close to the top. When I finished my fellowship, I was convinced I knew everything there was to know.

So, what I'd like to do is take you through the changes that have occurred since I finished my fellowship to now. Many of these, I've done, but so have many in the field. To give you a clue about how far you can go in your career, from what you think is the pinnacle of knowledge, to realize that it's just a pimple on the surface of knowledge.

I have no financial interests at all, but I think at this point I have to declare that I do have a relationship with Downstate. It's not that both my parents grew up in Brooklyn. It's not that I and two brothers and father went to Brooklyn Tech, but my older brother, Allen, recently stepped down as the chair of EMT tech at Long Island North Shore-Jewish, is a graduate of the medical school here.

I am going to talk about the off-label use of drugs. You know, it's remarkable, that we have no chemotherapy drugs that are FDA-approved for children. Everything you do is off-label.

Retinoblastoma is the most common primary cancer to affect the eyes of children worldwide. It's a relatively rare cancer, and it's actually not the most common cancer to affect the eyes of children. Retinoblastoma is on your left, and on your right is the most common cancer to affect the eye of children, and that is leukemia. So, depending on your institution, it is between the fourth and seventh most common pediatric cancer. There might be a slight increase in survival, but it is a cancer that affects young children. And perhaps about 10% of all children in the first year of life who develop cancer

> "Retinoblastoma is the most common primary cancer to affect the eyes of children worldwide."

have retinoblastoma. Now, the incidence of retinoblastoma is exactly the same, worldwide, independent of who you are, where you live, what your environment is, but the incidence of melanomas of the eye, which occur in adults, is very much related to ethnicity. As a result of that, in the United States, we have about four times as many melanomas as retinoblastomas, because we have so many people of European origin.

But throughout the rest of the world, retinoblastoma is actually the most common form of cancer that occurs in the eye of children or adults. There are about 300 cases a year in the United States, making it rare. Almost all the children in the US present with the disease in the eye, which is important, which you will see in a moment. Boys and girls are the same, right and left eye are the same. In 12% of the time, there's a family history, someone else has retinoblastoma. This is a clue that almost 100 years ago there was a gene involved. It can present unilaterally or bilaterally. When it presents, it presents like this so called leukocorially, white pupillary reflex.

It is the cancer success story of the 20th century. We have gone in 100 years, which is not when I finished medical school, to now, from 95% of children dying in the US to more than 95% of children surviving. It is, in all of pediatric oncology, the cancer with the highest cure rate. It was not that when I finished my fellowship. Worldwide, at the present time, it's still a devastating disease because 50% of the children who get this worldwide die. In the United States, survival in all centers is over 95%. Doug mentioned that the center I run over at Memorial is the largest by far in the world, and the oldest. And our present survival rate is actually over 99%. And, remarkably, 90% of these children have 20/20 vision in at least one eye.

In fact, the most common cause of death of children with retinoblastoma is not retinoblastoma, it is their subsequent, so called "second" malignancy related to their gene and environment. If you would ask, "Why is that 50% of the children in the world die?" Your immediate response would be, "Well, they don't have the resources or facilities you have at Memorial Sloan Kettering." But the reason is very simple. Children who are brought in, worldwide, are sometimes brought in like this, with very advanced disease, but are more commonly brought in and told, to cure the cancer, all you have to do is remove the eye. It's an operation I've done, and Doug has done, but around the world it's an operation that is unacceptable in most societies. So the child is seen, correctly diagnosed, goes home and is never treated. They die because they're not treated. The treatment, which is available, and curative, they do not accept.

This is not a simple medical problem. This is a social, this is a cultural problem. In the entire continent of Africa, there is not one country with a survival rate that is even as high as 50%.

Now retinoblastoma has some very curious genetics behind it, and I suspect you've had this in more than one course. It is the first tumor suppressor gene that was ever cloned in 1986, and it curiously develops in two different forms: one, we'll call it "genetic," and the other, we'll call it "non-genetic." We'll get back to that in a moment.

The retinoblastoma is a gene on chromosome 13, with

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200,000 base pairs and 27 exons. It is a gene all of us have functioning, all the time. It's a cell cycle inhibitor so it is a gene, if you will, that is built into human cells so that those cells don't have uncontrolled division and develop cancer. It is the loss of this normal gene that gives you the cancer. So, on the simple level, it's an autosomal dominant gene but it's a tumor suppressor on the molecular level.

So let's briefly go through the two forms. In the genetic form, the first mutation happens either in the sperm or the egg, so that there is a hit, there's a defect in chromosome 13 in every cell of the body. Now, that defect has to occur in the sperm or the egg. It would be most interesting to see the future generation tell me, how many of you think that occurs in the sperm? Okay, how many of you think it's most commonly in the egg? (Not everyone votes.) Is there something else in Brooklyn I don't know about?

So, it's actually more than 95% of the time found in the sperm; which is true about many genetic diseases, so this is when I point to the men in the room and say, "Your biological clocks are ticking." So, the first mutation occurs, and if there's a second mutation, you lose the protein, you lose the cell-cycle control, and you develop retinoblastoma. But there are other consequences of this. Your gametes, the child, now have one haplotype that's affected. So, you've got a 50% chance of passing this on to your children. It's an autosomal dominant.

Because you have one of the most important chromosome gene defects known to give cancer, you are at risk for cancers, through life; so called "second" cancers. And, because every cell in the body is affected, it is likely you will get multiple tumors, including multiple in the eyes, and retinoblastoma is, in fact, in both eyes, 30% of the time.

The second form, nothing wrong with the sperm or the egg, you don't pass it on, you don't have second cancers, and just one cell in the retina of one eye, when it gets that second hit, will develop retinoblastoma and, characteristically, one tumor in one eye.

So, as I mention, I thought I would, for fun, go through ten major shifts we have undergone in the treatment of retinoblastoma management since I finished my fellowship. The first is, the introduction of PGD. How many of you even know what PGD is? Okay. I'll tell you about PGD. We were the first to do it for retinoblastoma, but not the first to do it. PGD stands for preimplantation genetic diagnosis, so it is an assisted reproductive technique. In one sense it is very simple. It is in vitro fertilization but, if you wait three days – in three days, you're an eight-cell embryo. We go ahead and remove one cell. Now, you're a seven-cell embryo. Seven-cell embryos develop quite normally.

With nested PCR techniques, you can say whether this embryo has the retinoblastoma gene or not. It takes about an hour and a half in the lab. You then implant the embryo or embryos that have mom's genes and dad's genes, you haven't added a gene, you haven't taken away the gene, you've preselected the embryo that doesn't have the gene defect.

This is what we did for the first case that was done for reti-

> "Retinoblastoma is one of the very few solid cancers that can be cured with radiation alone."

noblastoma, done by us in 2003. I decided to go through the alphabet to find at least one example for every letter of PGD that's been done for genetic diseases. It's now been done for large numbers of genetic diseases. The first family that was done in the world was done for my patients. This is a child that, many years ago, had bilateral retinoblastoma, grew up, had normal vision in one eye, got married years later, had a child who had bilateral retinoblastoma. There was a 50/50 chance. That child went on and developed a second cancer, a brain tumor, pineal tumor. They liked mom's genes, they liked dad's genes, they were not so crazy about the retinoblastoma gene. So we did PGD, and this is the child at birth, perfectly normal eyes, and actually, ultimately, in this family, three of these four children were born with PGD, all looking like each other, because they're brothers and sisters, three of them don't have the retinoblastoma gene, the one does. She's gone on and gotten a third cancer.

The introduction to PGD is major. When I finished my fellowship, there was not one survival of metastatic retinoblastoma in the world. Not one. Not even a case report. One-hundred percent of those children died. We introduced some protocols that are now used worldwide, and in all centers adopting these protocols, more than 75% of these children survive. They still die, we don't salvage all of them, but I've seen, from the end of my fellowship, go from zero to 75%. That's a big step. Similarly, these children are at risk for what is called trilateral retinoblastoma, the so called "third eye," the pineal gland. They go on to develop cancers in the pineal gland, which often kill you.

When I finished my fellowship, we just described trilateral retinoblastoma, it wasn't even known. There were no survivors of this disease at all. Now, worldwide, with protocols we introduced, about a third of these children are surviving. Again, that may not sound good, but when you're coming from zero percent and you're a family, that is a step up.

Now, during my residency, when we saw a child with retinoblastoma, we did X-rays routinely to look for calcification because retinoblastoma calcifies in the eye. When CT scans were introduced in the early 70s, we switched to CT. It was obviously more elegant. But then we've come to recognize that CT scans in children are something you really, really want to avoid. This is the alert sent out by the National Institute of Health in 2003 on the web, pointing out that CT scans, especially in children, contribute to the development of subsequent cancers. Figures published by the NIH, those that are interested in this, we can talk and argue, point out that 2.2% of all deaths in the United States are from diagnostic radiation. This year in the United States, there will be more X-ray procedures on humans, than there are humans in the United States.

And rarely do the people who have X-ray procedures, CT scans or whatever, have it only once. So, abandoning CT is something we've done. We no longer do CTs. During my fellowship, and residency, I learned about radiation for retinoblastoma. Retinoblastoma is one of the very few solid cancers that can be cured with radiation alone. That's a powerful statement. An entire generation of children worldwide were treated with external beam radiation, they were cured, their

> "The only cancer pediatricians are required to screen for is retinoblastoma, even though it's not the most common cancer."

eyes were saved, their vision was saved and they lived.

I wrote an entire book on radiation and retinoblastoma and, as you mentioned, I'm tenured in radiation oncology, and we haven't used it in ten years. And the reason why we haven't used it in ten years is we realized that, long term, the children with this RB1 defect are exquisitely sensitive to the damaging effects of radiation. And so the exact treatment that allowed them to live, to save their eyes and vision, shortened their lives because of the cancers we induced.

So, when I finished my fellowship, I was an expert on radiation, but knew that, over the years, we'd develop better and better radiation techniques. I had no idea that I'd be the one responsible for it being abandoned in the world, but I am.

When radiation was abandoned, everyone decided, well, if radiation is bad, then chemotherapy must be good, right? It certainly had shorter follow-up, and everything with shorter follow-up looks better. So, systemic chemotherapy for retinoblastoma was introduced. You know, unfortunately, systemic chemotherapy rarely causes a cure for a solid cancer. And, in fact, it doesn't cure retinoblastoma, but it does cause it to shrink, and if it gets small enough, you can laser it and cure it. But there are significant problems with the systemic chemotherapy in children. There are deaths reported from the chemotherapy in China. Ten percent of children given systemic chemotherapy die from the chemotherapy alone. Obviously, they have transfusions, fever and nutropenia, permanent hearing loss from Carboplatin, and they too develop second cancers, so-called "secondary AML," mostly induced by the topoisomerase inhibitors.

As Doug mentioned, when he was a resident, retinoblastoma was treated by the removal by one or both eyes. It's a very good treatment for the cancer, it's not such a great treatment for the eye, and certainly isn't a good treatment for vision. And so you'll hear, very shortly, about the technique I introduced so that we don't have to do as many enucleations. And, in fact, we are now treating eyes with very advanced disease that, years ago, I and everyone else would have removed. Not only that, but using the same treatments, we have enabled us from removing, just ten years ago, 95 % of the eyes in unilateral blastoma, to only about five percent.

Now, if you want to go into something where you can make a difference, please figure out how to screen for cancer, because the previous generation has done a lousy job. The most common cause of death in the United States is rapidly becoming cancer, as you know, as our disease management as gotten better and better, or its outcomes have gotten bet-

> “We’ve been interested in this for the second cancers because the children from conception have a gene that is ticking, waiting to give them multiple cancers.”

ter and better. And we have increasing numbers of people living, and increasing numbers with cancer. You know it’s coming, but the screening has been very disappointing. In pediatrics, the only cancer pediatricians are required to screen for is retinoblastoma, even though it’s not the most common cancer. So, we don’t pick up cancer any earlier today than we did when I was a medical student. We’re better at treating it, but we’re not picking it up any earlier.

We’ve been interested in this for the second cancers because the children from conception have a gene that is ticking, waiting to give them multiple cancers. And we’ve instituted a screening program, and published on this, and our data would suggest that the screening program that we have not only picks up on it earlier, which is simple, but actually prolongs life and doesn’t just have, so called, lead time bias. I think that’s an important development.

Now, when we give systemic chemotherapy, it always causes a response, as it does in most cancers, and almost never cures the cancer. And why is that? That bothered me for years. Think about phase 1 trials. What you do in phase 1 trials is determine, basically, what kills 50% of animals, humans, whatever you’re studying. Then you take that dose and decrease it, because you really can’t have a treatment that kills 50% of your patients can you? Or can you? And you go down in the dose. So you use a dose that is toxic, but a toxic that we can handle, that doesn’t kill your patient, or doesn’t often kill your patient. That makes no sense. That guarantees that you’ll fail. Why would you ever think that a cancer cell is more susceptible to chemotherapy? The way they got to be a cancer cell is that they’re devious in a variety of mechanisms and can survive, not dying. So you’re doomed by that.

But wait. If you give a dose that kills the patient, you’ll kill the cancer. All right. There’s a little problem with that. I understand that. But the point is, that’s how high you have to go in the dose to kill a cancer. Maybe even higher. You say, “Thank you very much, you taught me how to cure cancer – kill my patients.” Very good. So, I wondered one day if we could deliver a dose so high that that concentration would kill the cancer, but the exposure to the patient would be so low, it wouldn’t make the patient sick. Could we put a catheter in the groin of babies, pass it up through the abdomen, bypassing the heart, go through the abdominal aorta, thoracic aorta, into the internal carotid artery, on that side … And then, could I go – and now I’m injecting to see exactly where we’re going, and could I pass a catheter into the ophthalmic artery? The ophthalmic artery is 900 microns. It’s pretty thin. It’s like Doug’s cornea that he operates on. It’s actually the size of angel hair pasta when it’s dry. I have no financial relationship with angel hair pasta.

If I put that catheter directly into that blood vessel remotely, I could deliver an extremely high concentration in an extremely small volume. So here we are going up in the internal carotid artery I’m going to go up to the first major branch of the internal carotid artery, which is the ophthalmic artery. It’s not the first branch of the internal carotid artery. No one had

> "What we did was develop a technique where you go above, and it's a flow-directed catheter so when I get to the ophthalmic artery, it's going to pop in."

been able to do this, ever. Obviously, I wasn't the first person to think of this. They couldn't do this because the ophthalmic artery does kind of an abrupt, right-hand turn. Kind of like some of the streets here in Brooklyn. You're driving and the next thing, you want to go that way (he gestures sharply to the right).

So, what we did was develop a technique where you go above, and it's a flow-directed catheter so when I get to the ophthalmic artery, it's going to pop in. Pop. And now I inject dye, and I'm in the ophthalmic artery. Yea, you can do that. When you do that, you can show the ophthalmic artery. And here, you're actually seeing the eye and the branches. And as we do that, this is the kind of angiogram you see of the eye with all of the blood vessels that you memorized for exams.

Now, I have what I call a "parentheses," here. I've had the opportunity to write a chapter for the newest Gray's Anatomy. When you do that, you don't have a lot of original work, but you do add some things. And this is the drawing from Gray's Anatomy. This anatomy, we've now done 1,600 angiograms we have never seen in a human. Nonetheless, it's the way you'll pass the exams and get to the boards, but we've yet to see a human that has this. But it's not that simple, because, the ophthalmic artery, though it is very small, has laminar flow, and the ophthalmic artery itself has branches that go to the muscle, the lid and the eye. So, if you inject directly into the ophthalmic artery, it will go down the center of the ophthalmic artery, and not get into the eyeball.

So what we realized we had to do is create eddy currents, so we actually physically push once a minute for 30 minutes with a push that creates these currents and now they'll get to the sidewall and into the eye. The largest branch of the ophthalmic artery is not to the eye. You'd think it is, wouldn't you? The largest branch of the ophthalmic artery is actually – doesn't anyone know this? Now, it's been awhile since you had anatomy, right? The largest branch of the ophthalmic artery actually goes to the supratrochlear artery, which supplies the upper inner part of the orbit onto the lid. Here it is. And that's why, in these children, sometimes they'll have this hyperemic area because we injected it in the eye. Can you see some of the lashes are lost? Because the supratrochlear artery is supplying that, and it's all transient. At least it's telling you you're in the right place.

So in May 2006, after my IRB was approved, we did our first patient, and this was our first patient. Scheduled for an

> “More than 45 countries in the world have come to us, and we’ve trained them on how to do it. Interestingly, more than half of them are in developing nations.”

enucleation, I think you can appreciate that this eye is largely filled with cancer, total retinal detachment, the vessels that you see are on the surface of the retina, which is detached behind the lens. There’s a little bit where there’s no cancer. This is an eye that is appropriate to take out.

We did it. We had no animal experiments before to tell us what dose, we chose the drugs and waited three weeks. And three weeks later, the cancer was almost completely gone. We realized, boy, we are on to something. Since then, there are more than 200 publications on this technique, most of them ours. More than 45 countries in the world have come to us, and we’ve trained them on how to do it. Interestingly, more than half of them are in developing nations. Remember I said the main cause of death is children not being treated. But even in countries where you’d be shocked to think that medical care couldn’t do this, they can do this, and there, the children can come once or twice for this treatment, and don’t have to have blood transfusions, they don’t have complications, so it’s actually being adopted in developing nations.

By 2014, which is eight years after I did the first, the survey, in the world, showed that this treatment that I introduced is the main treatment for children with unilateral retinoblastoma. We’ve learned it can be done worldwide, we learned that it can save eyes that were previously enucleated. We’ve learned that for very advanced eyes, we can save most of the eyes. This is what you asked me earlier, Doug. This is our experience, published lately, showing our experience between 2006 and 2009, then, 2010 and 2014 for very advanced eyes. And doing the same technique, but simply getting smarter.

We’ve moved the curve up so that 95% of these eyes are saved. Some of these results are so good, they’re hard to believe. So here’s an eye largely filled with tumor, an ultrasound showing the vitreous completely filled with tumor. One treatment, three weeks later, the tumor is completely calcified, flattened down, calcific here, just about gone. One treatment, one drug.

So, these very advanced eyes, before and after, are nothing less than dramatic. With total retinal detachment, the retina settles down. Now, I mentioned earlier that the systemic chemotherapy causes tumors to shrink, but doesn’t cure them. What do you do in cancer when you do a treatment and you have regrowth?

Unfortunately, that’s common in cancer, and rarely do you have a patient survive that. The reason is, you’ve done the most potent, appropriate treatment in the first-line treatment. Second-line treatment is second line because it doesn’t work very well. But actually, we’ve been able to salvage about three quarters of the eyes that have failed conventional treatment, offering them a treatment that never existed.

Same for subretinal seeds. Vitreous seed populates the vitreous and grows like crazy, but it has no blood supply, and has been very difficult to defeat because they don’t respond to anything. Here’s an eye before and after treatment with intra-arterial chemotherapy, it’s extremely effective for this. Now, I was taught, in my residency, so were you Doug, that if you

> "Two-thirds of the money you spend in treating childhood cancer is not treating the childhood cancer, it's treating the complications caused by treating the cancer."

have a retinal detachment that's there for more than – some people say hours, some people say a day or two – that even if you get it back, surgically, you never regain vision. And that's definitely true, unfortunately, in adults. And that's why retinal detachment is a semi-emergent procedure for ophthalmic surgeons. So, when we had children like this, with total retinal detachment, we said to the families, we'll save the eye, but this retina has been detached for weeks, maybe months, we really have no hope for vision. The family said, "Well, at least, if you can save the eye," but 30% of these eyes, this is the ERG tracing, have regained vision. Eyes with total detached retinas, with total blindness, the exact indication for removing a human eye. Hopeless eye. But that was hopeless when I was in your seat. Not hopeless. Now I'm here (indicating podium).

Money. Deans care about money, don't they? Well, one way or another. It's nice to have new treatments but, you know, some of our new treatments for cancer are thousands of times more expensive than the old treatments, and people question whether that increased money is truly worth it. So I decided to look into this. Two-thirds of the money you spend in treating childhood cancer is not treating the childhood cancer, it's treating the complications caused by treating the cancer. It's a huge amount of money. These children don't have ports. These children don't develop fever/neutropenia, or need transfusions. We compared the costs at Memorial Sloan Kettering of the two treatments we've had, and then the same study was repeated in Argentina, and in Chile, and this treatment, which is more expensive, per day, is half the price of the standard treatment.

Well, are we compromising lives by trying to save these eyes? That's a very good question. So, just about three months ago, we got together with the three largest centers in the world, Argentina, Philadelphia and Switzerland, to look at our collective results, and out of 634 cases that had been treated up to that point, there was one death. So, clearly, we're not increasing the chance of patients dying.

Well, it obviously avoids the side effects of chemotherapy and radiation, but it does something very interesting. Years ago, I was intrigued and published extensively on the observation that, with time, in children with retinoblastoma, new tumors develop – remember they're genetically primed, every cell in the body is affected – and, with time, even if you cure tumors in the back of the eye, within a few months, they'll develop tumors in the mid-part of the eye and, a few months later, in the periphery of the eye. And that's something we've all dealt with, and published extensively.

And it's common, if at birth, or in the first months of life, you have retinoblastoma in an eye, 96% of the time you'll

> "We realized that the molecular events that occur are such that all these retinoblastomas form between 26 and 28 weeks intrauterine."

develop a new tumor. It varies with age. By six months it's 50%, and overall, in the world it's 25-50%, and at Memorial, previously published, it's about 53%. So commonly, these children develop new tumors, and when people ask me, since I'm the one who wrote about this, I always said, well, the retina develops from the optic nerve out, cell division, Thymidine, however you want to do it, ends more at the posterior pole than it does in the periphery, so it's those dividing cells at the end which ultimately get the second hit and develop cancer. I said that. I wrote it many times. I got some awards for it. Nobody ever questioned it. Of course, I was wrong.

When we began to do the intra-arterial one day, I realized, we're not seeing any of the new tumors. This is something I treated every week of my life for thirty years. I took me about a year to realize, I wasn't seeing them. Why wasn't I seeing them? What was wrong with my explanation?

So, in 2015 we published a very nice paper in Nature on the cell of origin for retinoblastoma to be a cone precursor. Then we realized that there is no cell division in the human retina after birth. In fact, cell division during the third trimester. And we realized that the molecular events that occur are such that all these retinoblastomas form between 26 and 28 weeks intrauterine. On a molecular level, they form. Now, the children at birth may not have any visible tumors in the eye, but they're there. And what the intra-arterial is doing is treating these tiny tumors that are not visible ophthalmoscopically, but are visible, if you will, on a molecular level. So, it's not preventing cancer, it's treating tumors that were there all along. It's an interesting observation.

Now, we've broken two golden rules of cancer. When you go to break the golden rules of cancer, I strongly suggest you get tenured before you do it. The first is, don't use a single agent, drug, for chemotherapy. Why? Because you develop resistance. No doubt about that. In the conventional doses that you give intravenously, you cause tumors to shrink. But if you only use one drug, it will come back with a vengeance. You will regret it. So, when I started treating these children with single agent … a drug first used in the 1950s, so it's not a new drug, people said, Dummy, don't you know that single agent leads to resistance? Yea, but intravenous chemotherapy is like a gentle rainstorm. You get wet, it's a little messy, some side effects. But the intra-arterial chemotherapy is like a tsunami. If you give a dose that is 100 to 1,000 times what kills humans, you kill all the cells. You don't have to worry about resistance. There are no cells to give resistance. So, this is the first major time that single agent chemotherapy for solid can-

cers has worked. It has worked before, and, historically, the first person to ever do this at the NIH, for choriocarcenoma, when she first did it, and had survival, was fired from the NIH.

The second, I don't understand, but we've broken the rule. If you treat a patient with cancer, with some modality, and the cancer then regrows, you say, "It didn't work well enough," right? Therefore, the next time you treat shouldn't be that same treatment. It makes no sense. When we talk about resistance, we talk about all kinds of molecular mechanisms. And so we have a golden rule, if it didn't work the first time, don't do it. But of course, we're not going to do it a second time, because that treatment's not that good.

So, we have children, we have a reoccurrence rate that's about 10%, and with those children, we went back and treated them the same way, with the exact same drug, same dose, and 90% of those eyes have been saved. We published on that. It's now been done in Philadelphia, done and published from Switzerland, the other large centers. I don't really understand why this works, but we now have years of experience with this. It's quite dramatic. There's something wrong with our understanding. Fortunately, we didn't pay attention to what we thought we knew. Overall, we've done it more than 1,600 times.

And finally, the other advance that was inconceivable to me, at the end of my fellowship, was the idea of injecting chemotherapy directly into the eye. This is something people have tried over the years. The concern is we know that if you do operations on children with retinoblastoma and enter the eye, a cataract operation, glaucoma operation, a retinal detachment operation, in all of these, it's been reported, that through that tiny opening, the cancer cells come out, the cancer grows and the children dies.

So, it is a large no-no, to do this. But we're doing it. Frances Munier from Switzerland introduced modifications of the technique, and we then modified his technique. We do an electro-retinogram, there's a softening of the eye, so when the needle goes in, the likelihood of fluid coming out is less. Under sterile conditions, it's a very small needle, a 33-gauge

"So, we have children, we have a reoccurrence rate that's about 10%, and with those children, we went back and treated them the same way, with the exact same drug, same dose, and 90% of those eyes have been saved."

needle, right into the eye, and before removing it, that is, the needle, we freeze where the needle is, so there is no hole in the eye because it's just ice. And this is the kind of result you can get from vitreous seeds, this cloud. Vitreous seed, gone. These balls of vitreous seed, going away and, ultimately, gone. These recurrent seas of vitreous seeds, disappearing.

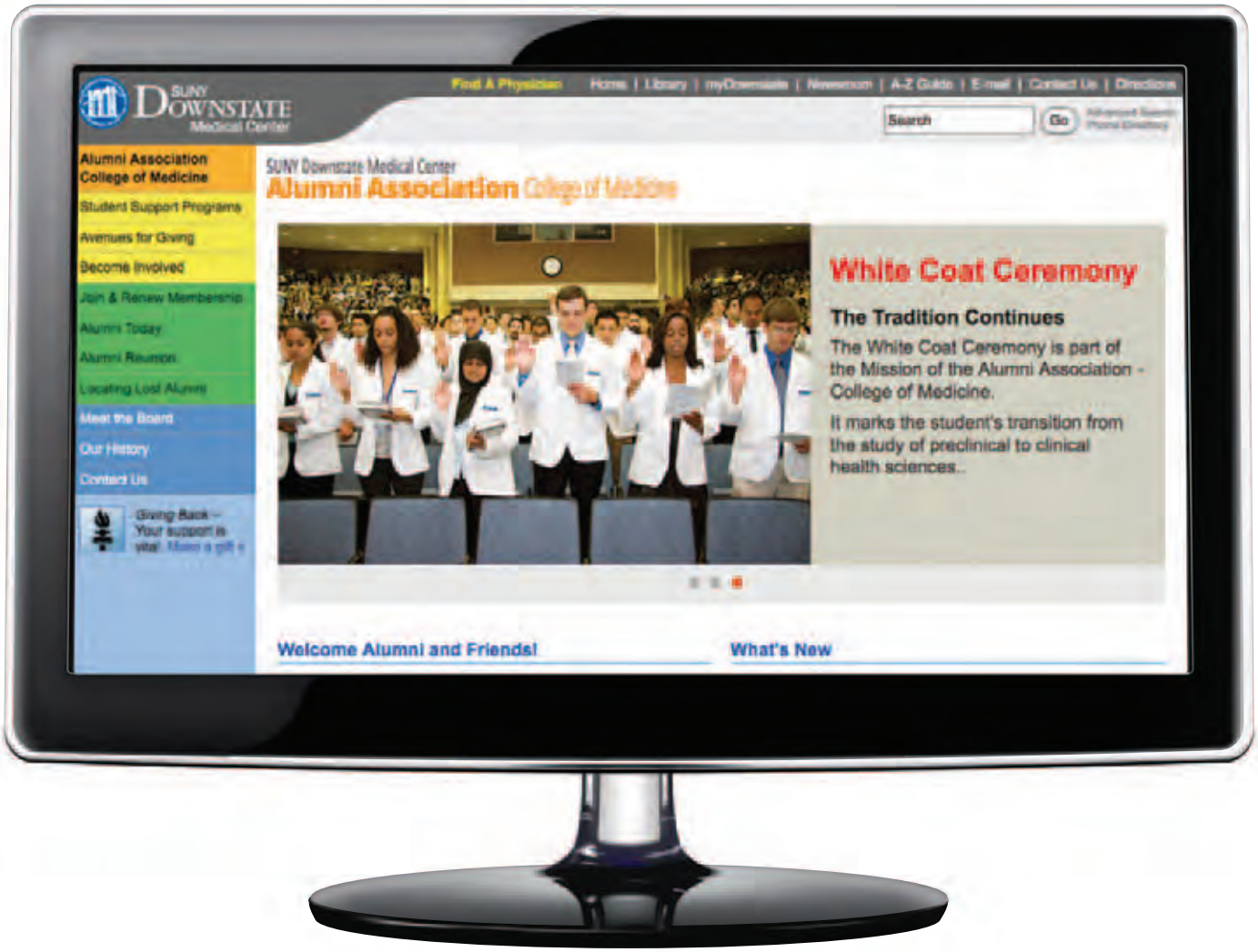
Originally Frances thought you needed weekly injections, between eight and 17. We now do them monthly, and the average child is only getting two or three. They get .072 CCs. We're getting down to minimalism.

It has some toxicity. We have published on the safety of this. We have washed all of our needles and looked at them to see if there were cells, and published a series of 200 consecutive needle samples and there were none. We washed the surface of the eye, collected the effluvient, there were no cells in 200 consecutive series also. So these are ten shifts, some of them absolutely something I was told I should never do, when I was in your position. All of which have helped humans. I look at you and I think, my goodness, what you're going to do in the future. I hope to be there for part of it.

WHAT'S NEW?

Visit the
Updated Web Site
of the
Alumni Association-College of Medicine, Downstate

- Join us for the Alumni Reunion festivities **May 19 - May 21, 2017**
- See the Alumni Reunion Weekend schedule
- Read the latest Alumni Today magazine
- Learn about the activities and programs we sponsor for our medical students and alumni
- Support our activities- pay your dues or make a gift on-line
- Update your contact information and help locate alumni
- See who serves on our Boards
- Provide us with your feedback



Thank you

Thank you cards received from students who graduated from the College of Medicine in May 2016

Dear Dr. Nicoll:

I would like to thank you and the SUNY Downstate Alumni Fund for your support and your generosity. On the journey to becoming a physician, the path is sometimes unclear and full of obstacles. When you finally figure out what it is that you want to do, the "how " is another mountain to climb. It can be overwhelming; however, the one thing I have learned from Downstate is that if there is a will there is a way. The support is available if you seek it out.

As an institution we are fortunate to have alumni who are willing to give back and I am inspired to follow in your footsteps upon graduation.

As a student, I simply feel blessed. The full year fellowship is an invaluable resource without which I would not be able to pursue my passion and career goal.

Your thoughtfulness is truly appreciated.

Again, thank you.
Kawah H. Laurent

Dear Dr. Constance Shames,

I am a third year medical student at SUNY Downstate. I am writing to personally thank you for your generous scholarship. I truly appreciate your support as I pursue my goal of becoming a competent and compassionate physician. I am inspired by your gift and it is my plan to one day help other students achieve their goals just as you have helped me. Thank you.

Sincerely,
Elizaveta Minko

To the Alumni Association:

Thank you so much. A simple kindness makes an ordinary day extraordinary. I just wanted to thank the Alumni Association for assisting me with the costs of medical school, and in the process of helping me achieve a life long goal. Words cannot express how truly thankful I am.

Kindest regards,
Christian Alexander Medina
COM class of 2016

Class Notes 2016

1950s

Horace Herbsman, MD '53 is "enjoying retirement with his wife, four children and ten grandchildren."

Robert L. Willenkin, MD '55 and his wife, Roberta, recently celebrated their 50th wedding anniversary.

Lewis B. Ward-Baker, MD '56 writes, "Looking back fondly on 30 years of child psychiatry, 20 years of retirement, family, making music and travel. Looking forward to what tomorrow will bring!"

Harry Weinstein, MD '56 retired from Westmed Medical Group Jan. 1, 2016.

Sidney Jerome Winawer, MD '56 was awarded a 2015 American Cancer Society Medal of Honor for Cancer Control. He was among three other individuals and one organization recognized at the Society's Medal of Honor ceremony and celebration dinner in Washington, DC. The Medal of Honor is awarded to those who have had a significant impact in the fight to end cancer through basic research, clinical research, cancer control or philanthropy.

Dr. Winawer received the award in recognition of his lifetime contributions and dedication to advance screening and prevention of colorectal cancer. His work has expanded the colorectal cancer knowledge base, documenting the impact of colonoscopy and polyp removal in reducing colorectal cancer incidence and mortality, according to the American Cancer Society.

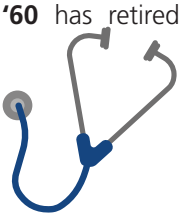
Daniel W. Schwartz, MD '57 retired from the KCHC 21 years ago, and now lives in Aventura, Fla.

Stratos Kantounis, MD '58 writes that he is "still assisting in the operating room, and teaching med students and surgical residents."

1960s

Herbert Pardes, MD '60 is Executive Vice Chairman of the Board of Trustees for New York Presbyterian Hospital.

Sandra A. Gilmore, MD '60 is retired, living in Westchester County, NY, and "enjoying it immensely," she writes. "Regards and good wishes to all my classmates."



Paul Schwartz, MD '60 has retired from active medical practice, but volunteers at Americas Free Clinics.

Robert M. Weiss, MD '60 received the Keyes Medal from the American Association of Genitourinary Surgeons, an association of the leading academic urologists from the United States, Canada and around the world. The Association is dedicated to the study of diseases of the genitourinary system. The Keyes Medal is presented to an individual for their "outstanding contributions in the advancement of Urology." It is recognized as the greatest individual citation in the specialty, and is awarded sparingly.

Stuart Bednoff, MD '61 writes, "I am happy to report that after a successful career lasting 47 years in private practice in OB/GYN, I have chosen to retire. My wife of 55 years and I hope to spend time with our family, and also plan to travel."

David A. Papernik, MD '62 received a Faculty Service Award commemorating 35 years, through 2014, from the NYU School of Medicine, where he is a clinical professor of psychiatry.

Michael Blumenfield, MD '64 conducts a part-time psychiatry practice in Woodland Hills, California, near Los Angeles. He writes, "Susan and I are fortunate to be living near our three kids and grandchildren." Dr. Blumenfield also blogs at MBlumenfieldMD.com, PsychiatryTalk.com, FilmRap.net and BookRap.net.

Michael J. Goldberg, MD '64 was named the first scholar-in-residence at the Schwartz Center for Compassionate

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Healthcare in Boston. The small not-for-profit is dedicated to ensuring that every caregiver-patient interaction is compassionate.

Diane G. Tanenbaum, MD '64 writes, "I'm still enjoying practicing medical dermatology on Manhattan's Upper East Side, and supervising dermatology residents one morning a week at New York University's Langone Dept. of Dermatology."

Andrew R. Schwartz, MD '65 and his wife, Lenore, are "doing well, still working as senior care providers."

Robert L. Stamper, MD '65 is still working at the University of California, San Francisco, where he is the Fortisure

Distinguished Professor of Clinical Ophthalmology, and involved in clinical care teaching and research projects in India, Ethiopia and Thailand.

John M. Aversa, MD '67 continues to practice with Connecticut Orthopaedic Specialists. He is also an associate professor at Yale School of Medicine, and at the Frank H. Netter School of Medicine at Quinnipiac University. He and his wife, Ellen, have a son, John Jr., who is a colorectal surgeon at Yale and Griffin Hospital. Their daughter, Kristen Aversa, is an OBGYN in New Haven, and their son, David, is a triple-boarded psychiatrist at Yale and Netter School of Medicine. Their youngest daughter, Monica, works for her brother, David. "We have seven beautiful grandchildren," Ellen writes, "and one on the way."

Paul Silk, MD '67 works part-time as associate professor of radiology at Albany Medical Center. He frequently works from

home, but travels from Albany from his Lake George home to give resident conferences. He is also responsible for first-year resident orientation.

Lawrence J. Brandt, MD '68 writes that he is "still working full time and loving it" as Emeritus Chief of Gastroenterology at Montefiore Medical Center. He recently received his first NIH-sponsored grant (on fecal transplants for recurrent C-diff infection), is still happily married, and hopes "never to retire, but to play as much golf as possible."

Allen I. Goldberg, MD '68 recalls that

his "student travel in 1967 to France led to my professional development as a home care physician."

Robert D. Rudnicki, MD '68 retired from his rheumatology practice in October, 2015. Two of their eight grandchildren are in college, attending Georgia Tech and the University of Georgia.

Barry Green, MD '68 has now practiced diagnostic radiology for 45 years.

Edward Kersh, MD '69 retired from clinical practice, and is now medical director for telehealth with Sutter Care at Home part time.

1970s

Lawrence Cohen, MD '70 is a senior radiologist at Cedars Sinai medical Center in Los Angeles, and volunteer teacher at LAC-USC Medical Center.

Nancy (Smolen) Falk, MD '71 retired in December, 2015, after 40 years in a solo practice in internal medicine.

Neil J. Principe, MD '71 is retired and living in south Florida. He writes, however, "I do miss the five boroughs, and wish all graduates and students the best of what will be a truly rewarding career and profession."

Marilyn Joseph, MD '72 and Warren Regelmann, MD '72 are both retired, with two grandsons, ages 1 and 4. Their oldest son, an MD/PhD, is CEO of Quartzy.com, a web-based system for keeping lab inventories for clients. The couple's other son is an assistant professor of internal medicine at Frank Netter School of Medicine, practicing at St. Vincent's in Bridgeport, CT.

Class Notes 2016

Steven Brozinsky, MD '72 sings tenor in the San Diego Jewish Mens Choir.

Irwin Berkowitz, MD '72 after 40 years of pediatric experience, has published a book, "Instructions Not Included: A Pediatrician's Prescription for Raising the Best Kids on the Block." It's a common sense guide for child rearing and available on Amazon and Nook.

Gary Rosenberg, MD '73 was profiled in NJ Spotlight May 25 for his work with a new program that has successfully paired child psychiatrists with pediatricians.

Maria Arnett, MD '74's husband, Harvey, died in January, 2014, after a long battle with multiple myeloma. Dr. Arnett is still practicing ophthalmology in Manhattan, and enjoying her children and grandchildren, all living nearby.

Michael T. Goldstein, MD '74 was honored for his term as President of the New York County Medical Society, which finished at a June 7, 2016 ceremony. He is now a Trustee of the New York County Medical Society.

Howard Grill, MD '74 practices full time alongside David Grill, MD, '80. Howard Grill also has two grandchildren, Harper and Rowan, and his three daughters all work in health-related fields.

James H. Lewis, MD '75 is finishing his 38th year in academic practice in Washington DC at Georgetown University in gastroenterology and hepatology. He writes that he still enjoys all the "challenges of teaching, patient care and EHRs!"

Henry Lim, MD '75 was elected president of the American Academy of Dermatology, the world's largest dermatologic society. He is chairman of the Department of Dermatology at Henry Ford Hospital.

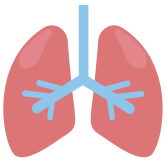
Richard A. Miller, MD '75 is CEO and Chairman of Corvus Pharmaceuticals, a biotech company in the Bay Area developing new treatments for cancer. Dr. Miller was the co-founder of IDEC (now Biogen), and Pharmacyclics, companies that developed Rituxan and Imbruvica. He is also an adjunct professor of oncology at Stanford Medical Center.

Gary B. Witman, MD '75 was hit by a wave in the back of the neck, making him a quadriplegic at the C3-C4 level in August 2010. He is currently evaluating patients for the usage of medicinal cannabinoid products, and is "thrilled to be able to practice on a full-time basis."

Millicent Comrie, MD '76 was named one of the 10 top Caribbean-born doctors by News Americas, Caribbean and Latin American News.

Elizabeth Legatt, MD '76 writes that she has practiced gynecology in the same community for 31 years, and loves it.

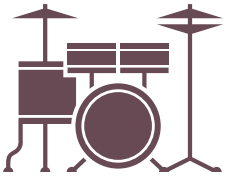
Alan Schecter, MD '76 is director of the Division of Pulmonary Medicine at St. Frances Hospital in Roslyn, NY.



Richard M. Zweig, MD '76 writes that he "continues to enjoy practicing medicine in Bristol, Connecticut," where he is Chief of Medicine and Director of Infectious Disease at Bristol Hospital.

Leonard Berkowitz, MD '77 and his wife, Joan, recently became grandparents to Sidney Max Casden, son of Jodi and Jason, and their daughter got engaged.

Kenneth A. Grossman, MD '77 was selected by New Jersey Monthly Magazine as one of the top doctors in the state for 10 consecutive years. He is also a drummer in a rock band composed of all doctors. The band plays for local charity events.



Thomas Quaresima, MD '77
John and Mark Quaresima both began family practices in July, 2016.

David S. Katz, MD '80's daughter, Abby, is board chair of Women for Economic and Leadership Development, a national organization promoting wom-

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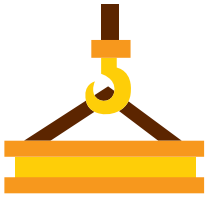
en in business. Dr. Katz is still enjoying his practice in occupational medicine as a medical director with US Healthworks medical group in Indianapolis.

Donna J. Barbot, MD '78 works for the Temple Health System as the Chair of Surgery at Temple Jeanes Hospital.

1980s

David Berger, MD '84 is Professor of Surgery at Baylor College of Medicine, and was named Senior Vice President and Chief Operating Officer of Baylor St Luke's Medical Center in the Texas Medical Center, Houston. He was also recently featured on FOX News Houston for a story on hospital construction.



Lawrence Halperin, MD '84 was named Chair of the American Academy of Orthopedic Surgeons (AAOS) Board of Councilors, and serves on the AAOS Board of Directors.

Kenneth Becker, MD '86 Dr. Becker is Director of Trauma/General Surgery at South Nassau Community Hospital in Oceanside, NY.

Raymond Resnick, MD '87 leads the cardiac catherization lab at Wayne Memorial Hospital in Honesdale, Pennsylvania.

Janet P. Piscitelli-Bosso, MD '87 was promoted to VP and Chief Laboratory Officer of Quest Diagnostics.

Lawrence Hakim, MD '88 was appointed Jan. 1, 2016 as the Cleveland Clinic Florida's Chief of Surgical Specialties, Urology, Gynecology, Head and Neck, Plastic and Hand Surgery and Department of Solid Organ Transplantation, which includes heart, liver and kidney transplants.

John Ratmeyer, MD '88 is in his 25th year of serving Native American children and their families through his work as a pediatrician at the Gallup Indian Medical Center in Northwest New Mexico. He is the Deputy Chief of Pediatrics, scheduling coordinator and medical consultant to the Child Protection Team.

Benjamin D. Freilich, MD '89 was appointed Senior Medical Affairs Advisor for Intellect Neurosciences, a research-based biotechnology company focused on the discovery, development and licensing of novel, disease-modifying therapeutics for the treatment and prevention of rare neurodegenerative orphan diseases with no approved therapies. Before entering private practice, Dr. Feilich was full-time academic faculty at SUNY Downstate, and then joined the academic faculty at The Mount Sinai School of Medicine, where he continues to hold the title of Assistant Clinical Professor of Ophthalmology.

1990s

Jonathan Gillen-Goldstein, MD '95 was named Interim Chairman of Obstetrics and Gynecology and Director of Maternal Fetal Medicine at Good Samaritan Hospital Medical Center in West Islip, and Director of Maternal Fetal Medicine at Catholic Health Services of Long Island. Together with his colleagues, David Bergman, MD, and Christopher Plummer, DO, Dr. Gillen-Goldstein and the Maternal Fetal Medicine Program at Good Samaritan provide specialized consultative care to women experiencing high risk pregnancies and those requiring prenatal testing to monitor routine pregnancies.

2000s

Samantha Lowe, MD '02, a pediatrician, joined the staff of White Plains Hospital in Armonk, NY. Prior to White Plains, she was an associate member of the Department of Pediatrics/Emergency Medicine at St. Barnabas Hospital, and an associate professor in pediatrics at Albert Einstein College of Medicine in the Bronx.

Abraham Loo, MD '06 is a hematopathologist at Monmouth Medical Center, New Jersey. Dr. Loo earned his Doctor of Medicine degree at SUNY Downstate College of Medicine, Brooklyn, where he was elected to the Alpha Omega Alpha Honor Medical Society.

Remembrances

Edited by Constance Shames, M.D. '63

Saul Rotter, MD '36

Dr. Saul Rotter of Palm Beach, one of the first physicians to open his waiting room to black patients in Palm Beach County in the 1940s, died Tuesday, May 12, 2015. He was 103.

Born in Brooklyn, N.Y., in 1912, Dr. Rotter graduated from Columbia College and Long Island College of Medicine. He practiced medicine in the Palm Beaches from 1941 until 2002, when he was 90.

A doctor of internal medicine, Dr. Rotter set up a practice in the back of a Lake Worth drugstore one month before the attack on Pearl Harbor. He opened his waiting room to black patients at a time when they typically were told to wait in the hallway. He also treated Rose and Joseph Kennedy in Palm Beach.

Sam Tally Simpson, MD '45

Dr. Simpson died September 15, 2015. He joined the US Army during World War II, and worked in the Army Medical Corps at the US Veteran's Hospital, Columbia, SC. After his discharge in 1946 with the rank of Captain, he practiced obstetrics and gynecology in Birmingham, Alabama. He earned a Master's Degree of Public Health from Tulane in New Orleans, and served as the health director of Manatee County, Florida. He later served as health director of medical service for Dade County.

He was a Fellow of The American Board of Abdominal Surgery, American Medical Society, and Manatee County Medical Society. Dr. Simpson retired from public health in 1982.

Herbert Leroy Abrams, MD '46

Dr. Abrams, a physician, scholar, author, and activist, died at home in Palo Alto, California, on January 20, 2016. He was 95. His multi-dimensional career embraced what he called the "four dimensions of bio-medicine," patient care, research, teaching, and advocacy. Dr. Abrams was the Founding Vice President of International Physicians for the Prevention of Nuclear War, the recipient of the 1985 Nobel Peace Prize. He served on the National Board of Directors of Physicians for Social Responsibility (PSR) for 20 years, and was a National Co-Chairman during the 1980's. PSR's message is that the medical community could not cope with the destruction caused by a massive nuclear exchange; the there was no cure, only prevention. Dr. Abrams's optimism led him to say, "I believe that you and I can make a difference, both as individuals and as part of a collective entity that shares the values and vision of a world of peace." (Image credit- Rod Searcey)

James F. Morrell, MD '46

Dr. Morell died Sept. 24, 2015. He was a longtime resident of Garden City, NY, and later moved to Blue Bell/Ambler.

Vincent DeLuca, MD '48

Dr. DeLuca died August 25, 2015. He served in the US Navy as a medical officer, and then settled in Woodbridge, CT, and joined the staff of Griffin Hospital. He served as the hospital's Director of Medical Education, and also started a gastroenterology fellowship program with Yale University School of Medicine.

He was a pioneer in gastroenterology, receiving a Distin-

guished Award from the CT Society of Internal Medicine in 1974.

Jay J. Gold, MD '50

Dr. Gold was an endocrinologist who devoted his career to research, and to a private gynecological endocrinology practice in Illinois.

Jerome Maisel, MD, '51

Dr. Maisel died September 25, 2015. He served in the Army Air Force as a weatherman in WWII, and lived out his devotion to humanitarian causes with spirit and zest for life. He was a compassionate pediatrician who practiced in Hewlett, NY for 50 years.

Augustus M. Tanaka, MD '51

Dr. Tanaka died December 14, 2015 in Ontario, Oregon, at 92. When the Japanese bombed Pearl Harbor in 1941, his

father was taken from their home in Portland, Oregon, and kept in a prison camp for four years. Gus and the rest of the family would be sent to detention camps at the Portland Stockyards and Minidoka, Idaho. In the fall of 1942, Dr. Arthur Scott of Reed College facilitated Gus's enrollment at Haverford College in Pennsylvania. In 1944, Gus was drafted into the U.S. Army. In 1945, he went to Japan where he taught the history of war and reading to American soldiers.

Gus returned and earned his BA from Haverford, and MD from SUNY in 1951. He married Teruko "Teddy" Wada in 1953, and returned to Ontario, Oregon, to open the Tanaka Clinic in 1959 with his father. He became Oregon Medical Association's first minority president in 1971-1972, and Outstanding Doctor by Oregon Foundation for Medical Excellence, among other honors.

Lauren Howard Lucke, MD '52

Dr. Lucke was born in Jefferson, Iowa, August 24, 1926, and died Dec. 15, 2015. He graduated from the University of Washington with a major in Zoology, and after serving in WWII, attended medical school at SUNY Downstate, paid for by the US Army. He had followed his father and grandfather into medicine, and met a nurse at SUNY Downstate, the late Jane Buskirk Lucke, who would eventually become his wife. Dr. Lucke practiced beside his father in a Seattle general practice before moving to his own practice in Sultan, Washington. He earned his Master's in Public Health, and spent the rest of his career as Director of Health for Grays Harbor and Pacific Counties in Washington.

Clifford Reichert, MD '52

Dr. Reichert, 91, died July 30, 2015, following a long ill-

ness. He was a retired ophthalmologist who had a private practice in Milford and Framingham, and was affiliated with the former Milford Hospital and Framingham Union Hospital, Mass. He served in the US Army Air Corps in WWII as a ball turret gunner in the European Theater. As a staff sergeant, he earned three Bronze Stars and the Air Medal with a Bronze Oak Leaf Cluster. He and his late wife of 55 years, Patricia (Lenox) Reichart, lived in Massachusetts, Florida and New Mexico.

Remembrances

Jacob Brody, MD '56

Jacob A. Brody, M.D., a world-traveled epidemiologist, public health researcher, professor and administrator, made his final journey home from his residence in Miami to safe haven in Chicago on April 15. He died on April 22 in the compassionate care of Horizon Hospice of Rush University Medical Center. There he was surrounded by his family, friends and colleagues from the School of Public Health at the University of Illinois at Chicago where he served as Dean from 1985-1992 and retired as Professor of Epidemiology in 2005.

Dr. Brody's career was varied and remarkably productive, beginning at the Centers for Disease Control's Epidemic Intelligence Service in 1957 where he embraced the opportunity to engage in medical research, travel the world, and learn languages during assignments in Bangladesh for small pox epidemics, tropical diseases in Panama and elsewhere in Mexico and South America, measles in Alaska, and a year spent in Russia as an exchange scientist.

Dr. Brody held a number of positions at the National Institutes of Health in Bethesda, Maryland in neurology, where he headed a research station on amyotrophic lateral sclerosis (ALS) on Guam, worked on effects of radiation of survivors of the atomic bomb in Hiroshima, Japan, and started epidemiology programs that had global reach in alcoholism research and in the field of aging, the research focus in the latter part of his career. He authored more than 250 scientific publications and in 2002 was recognized a being in the top 0.5% of the most frequently cited authors in the field. Honors included his service as the President of the American Epidemiology Society (1980), the Distinguished Service Medal of the U.S. Public Health Service (1981), and the Lilienfeld Award for outstanding contributions, leadership, and research in epidemiology Dr. Brody was born in Brooklyn, New York, the second son of Dr. Simon and Rosella Brody, and graduated from Poly Prep and Williams College where he excelled academically, but was perhaps most proud of his standing records in track. He remained a track and field enthusiast throughout his life, although scuba diving was his major athletic passion as an adult, along with photography, poetry, audio books on anything and everything, and an interest in foreign affairs. He graduated from SUNY Downstate College of Medicine.

He was an anchor and inspiration to many, included his devoted family. He is survived by his wife of 45 years, Ann Thomas Brody, son Thomas (Amy Huseth) Brody, Eva (Scott) Fujino, and five grandchildren.

Leonard Meltzer, MD '56

Leonard Meltzer graduated from New York University, class of '52, and the State University of New York, Downstate College of Medicine, class of '56.

Dr. Meltzer served as a Captain in the United States Air Force from 1957-1959 where he was stationed in the Far East Command.

Between 1957 and 1961, before and after his military service, Dr. Meltzer completed an internship and a residency at Beth Israel Hospital. In 1962 Dr. Meltzer completed a fellowship in cardiology at Mt. Sinai Hospital. For 32 years, Dr. Meltzer practiced internal medicine with a subspecialty in cardiology at the East Nassau Medical Center in Hicksville, New York.

Dr. Meltzer and his wife Sylvia retired to Holbrook, New York and Boynton Beach, Florida. In retirement, Dr. and Mrs. Meltzer enjoyed travel and were avid bridge players. Dr. Meltzer enjoyed spending time with his family and friends. While in retirement, Dr. Meltzer would regularly encounter former patients who would remember and thank him for the excellent patient care he had provided to them.

In January 2015, Dr. Meltzer passed away at home, sur-

rounded by his family, following a 15 year battle with prostate cancer. Dr. Meltzer is survived by two sisters, his wife of sixty years, two children and two grandchildren.

Gerald G. Cole, MD '56

Dr. Cole, of Del Mar, California, died July 22, 2015, after a brief illness.

Jack Eisert, MD '56

Dr. Eisert died May 11, 2015, at 84. He was a retired dermatologist and Clinical Professor of Dermatology at Columbia Medical School.

Maxwell J. Felton, MD '56

Dr. Felton of New York, NY, retired surgeon and ardent family man, died August 5, 2015.

Jerome Klein, MD '57

Norwalk Physician for Four Decades Jerome Joshua Klein, M.D., a gifted and caring physician to thousands of patients in the Fairfield County area during a 48-year career in medicine, died in hospice care on Sunday in Norwalk, surrounded by loved ones. His passage followed a long battle with cancer.

He was 84 years old. Dr. Klein joined the Norwalk Medical Group in 1965 as an Internist with specialties in Endocrinology, Diabetes and Metabolism—routinely making house calls to see patients. He held privileges at Norwalk Hospital, Bridgeport Hospital, St. Vincent's Medical Center, and Yale New Haven Hospital, among others. He cared for his patients wherever they were sent, at rehab centers and skilled nursing facilities, across Fairfield County. Working in both primary care and as a specialist, Klein soon became known as a diagnostician, galvanized by the hunt for the correct diagnosis to difficult-to-identify illnesses.

"His patients adored him," recalls son Jonathan. "As a boy, we'd be out somewhere and a patient would see dad and they'd stop to catch up. He always remembered their name and their care, even decades later. Many of them looked down at me and said 'You know, your father saved my life.'"

Jerome Klein was born in 1931 in Brooklyn, NY to parents Esther Wohl Klein and David Klein, an accountant. An only child, Klein attended the Little Red Schoolhouse in Manhattan, and later sister school Elizabeth Erwin High School. He attended Harvard, where he played clarinet in the Harvard Band. He received his B.A. cum laude in History from there in 1953.

Opting to study medicine, he attended State University of New York Health Science Center ("SUNY Downstate") in Brooklyn, receiving his M.D. in 1957. He completed an internship at Bellevue Hospital Center in New York City a year later. Klein then enlisted in the Air Force as a Captain, where he served for two years at Offutt Air Force Base in Bellevue, NE. Returning to New York City afterward, he completed residencies in Internal Medicine at Mount Sinai Medical Center, Bellevue Hospital Center, and the Montefiore Medical Center in the Bronx. In 1965, Dr. Klein joined the fledgling Norwalk Medical Group, where he practiced for the next 38 years.

He lived in Westport until 1983, when he moved to Weston. In 1987 he married Lynne Hummer Klein, his devoted and loving wife, and the love of his life. Dr. Klein retired from medicine in 2002 at age 72, moving with Lynne to Norwalk in 2011.

His interests were many, and included tennis, skiing, hik-

Remembrances

ing, cooking, gardening, playing the clarinet, hi-fi audio (with a passion for building loudspeakers), woodworking, home repair and art, in addition to history. He loved classical music and opera and was a lifelong patron of the arts. "Growing up, there was always classical music filling the house from loudspeakers he'd built," recalls son Dana.

Dr. Klein is survived by his wife Lynne Klein; his sons Dana Klein and his wife Sonya Klein of Darien, and Jonathan Klein and his wife Ellen Petri of Concord, MA; his step-daughters Karen Santora and her husband Jeff Santora of Ridgefield, and Cindy D'Acunto and her husband Thomas D'Acunto of Wilton. He is also survived by eight grandchildren: Rachel, Elana, Hannah and Harrison Klein, Eric Santora, Thomas and Christopher D'Acunto, and Christopher Porter. He is predeceased by his step-son Anthony Ciliberto.

Thomas Michael Cassidy, MD '57

Dr. Cassidy, passed away peacefully on April 20, 2015 at the age of 82, at Nazareth Home.

Born in Brooklyn, NY, Tom attended St. Michael's High School. In 1953, Tom graduated cum laude from St. John's University in Queens, NY. He received his M.D. in psychiatry in 1957 from the State University of New York/Downstate Medical Center in Brooklyn.

Tom's professional accomplishments in psychiatry spanned over 40 years. Tom was affiliated with U.S. Public Health Service for over 13 years and served in Paris, France for three years. He also served as the clinical director at Charterton Hospital in La Grange. Tom was the Medical Director at Lincoln Trail Hospital in Radcliff, KY until he retired in 1995.

Ron A. Sorvino, MD '58

Dr. Sorvino died Sept. 14, 2015 at 82. After completing his medical degree at SUNY Downstate, the Brooklyn native served in the United States Navy as a Lieutenant Commander from 1962-1964 where he was the Chief of Psychiatry for the Newport Naval Hospital. He was the Chief of Psychiatry at Overlook Hospital in Summit for years, and also operated a private practice at Overlook Hospital for more than 30 years.

Ann Wennhold, MD '58

Dr. Wennhold died July 10, 2015, at 83. A New York City native, she attended Seton Hill women's college, and completed her MD degree at SUNY when women only made up

10% of the student body. She moved to Salt Lake to pursue endocrine research and, over the next 20 years, published more than 100 studies. She later completed a residency in psychiatry while raising two children with her husband Phil. For the remainder of her career, she had joint appointments as Chief of Psychiatry at the VA Medical Center and Associate Dean at the University of Utah School of Medicine. An avid traveler and outdoorswomen, Dr. Wennhold conquered Base Camp III of Mt. Everest at age 68.

Gerald M. Greenberg, MD '59

Dr. Greenberg of Roslyn, NY, a former Brooklyn internist, died May 24, 2015.

William Erwin Paul, MD '60

Dr. Paul died on September 18, 2015. He was a major contributor to the development of modern immunology, and

helped to transform cytokine biology, the study of small proteins involved in cell signaling, from crude assessments of uncharacterized cellular "factors" into a science involving precise quantitative molecular analyses. He was director of the NIH Office of AIDS research from 1994 to 1997. Dr. Paul received many awards and honors over his long career and will be greatly missed.

Samuel Himelstein, MD '65

Dr. Himelstein died August 10, 2015 at 78. He was born in Springfield, Massachusetts, and graduated from Trinity College in Hartford, and SUNY Medical Center. He entered

private practice in Enfield & Rockville, Connecticut, and also served as Acting Chief of Ophthalmology at Johnson Memorial Hospital, Stafford Springs, and Chairman of the Dept. of Ophthalmology at Rockville General Hospital, Rockville. He returned to private practice in Boynton Beach, Florida, and Southern Pines, Greenville and Roanoke Rapids, North Carolina, until his retirement in 2009.

Sam was also a Travel Agent with his late wife Ruth's travel agency for 25 years. The couple traveled all over the world on a total 76 cruises.

Richard Luft, MD '68

Dr. Luft died April 9, 2016 at 72. He was a dedicated general surgeon who served Brooklyn through his decades-long career.

Roberta Flesh, MD '71

Dr. Flesh, an Albany resident, was the first female board-certified internist in the Capital District to open a private practice. In June 2009 she began her affiliation with the VA Medical Center, specializing in geriatric primary care. She died at age 67 on September 16, 2014.

Michael Ammazzalorso, MD '87

Dr. Ammazzalorso died May 27, 2015, at 54. He had served as Vice President of Medical Staff and Chief Medical Officer at Winthrop-University Hospital in Mineola, New York, and died after a long battle with pancreatic cancer. Dr. Ammazzalorso was a Master of the American College of Physicians, an honor granted to only a handful of physicians by the nation's largest medical specialty society.

Laurence Finberg, MD '92H

Dr. Finberg died January 22, 2016 at 92. He was a pediatrician and scientist, known for his contributions to the management of salt and water balance in children. He served

as Chair of Pediatrics at the State University of New York (SUNY) at Downstate in Brooklyn from 1982-1994, and was Dean of the School of Medicine at SUNY Downstate from 1988-1991.

A Chicago native, Dr. Finberg received an SB (1944) and MD degree (1946) from the University of Chicago. He served in the US Public Health Service before his residency at Baltimore City Hospitals and Johns Hopkins Medical School. In August 1950, during a major epidemic of poliomyelitis when he was Chief Resident in Pediatrics and the Admitting Officer for Contagious Diseases at Baltimore City, he boldly integrated medical services by admitting black adults to previously all-white wards. Dr. Finberg served on the faculty at Johns Hopkins

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from 1950-1963. He became Chair of Pediatrics at Montefiore Hospital and Professor of Pediatrics at Albert Einstein College of Medicine in 1963. He is the winner of multiple honors and awards including the Distinguished Service Award of the National Board of Medical Examiners, the President's Certificate for Outstanding Service of the Academy of Pediatrics, and the Nutrition Award of the American Academy of Pediatrics.

A prominent clinical investigator, Dr. Finberg was known for his many original papers and two books on fluid and electrolyte abnormalities in children. He was Chair of the Academy of Pediatrics Committees on Nutrition and Environmental Hazards, and an expert in the diagnosis and treatment of lead poisoning in children.

Andrew Parsa, MD '96

Andrew Thomas Parsa, a renowned neurosurgeon, innovative researcher, and dedicated mentor to scores of aspiring surgeons and scientists, died suddenly on April 13, 2015 at his home in Chicago. He was one of the most productive and admired neurosurgeons of his generation, a dedicated husband and father, and a proud and loyal son of New Canaan.

One might even go as far as to say that Andy embodied the raison d'etre of such a community: a privileged loam in which certain special seeds of marvelous human potential may germinate among the weeds to sink the deep roots required to anchor and sustain gargantuan, world-changing ambition.

He was born on Aug. 24, 1966 in Brooklyn, N.Y. to Micheline and Ismail Parsa, M.D. His mother was a nurse, his father a renal organ transplant and cancer immunology researcher and professor of Pathology at SUNY-Downstate Medical Center. They moved to New Canaan when Andy was four, to the house the Parsa family still owns on Smith Ridge Road. Andy attended Center School where he faced the existential terror of the knock-out pit with courage and aplomb. He mourned the loss of the murals in Ms. Stone's first grade classroom when the town council sages sacrificed that

historic building to the parking lot gods. In New Canaan High School, Andy excelled in academics and athletics, A.P. everything, captain of the indoor and outdoor track teams, as well as the soccer team his senior year. His mind's flame was kindled by Norman Ricker, Edward Ruszczyk and Warren Allen Smith, among others. He was widely known to be a gracious prom and homecoming date. His letterman jacket groaned with varsity pins.

Andy entered Yale College in 1984, a testament to the quality of New Canaan's school system and to the town itself, a community where a physician from pre-revolution Iran and a nurse from pre-hipster Brooklyn could settle and realize their dream of launching offspring into the Ivy League. At Yale, Andy earned a B.S. in Molecular Biophysics and Biochemistry, the most challenging of the science majors. He had been recruited as a soccer player and helped win the Ivy League Championship in his senior year.

Following a lifelong calling to be a physician, in 1988 he enrolled in Downstate Medical Center in Brooklyn, New York, the same institution where his father was a professor and a researcher. Sadly, Dr. Ismail Parsa died suddenly the summer before Andy started medical school, a devastating shock that might have stunted the professional trajectory of a less determined man. Truly, to love is to grieve, but Andy honored his father's memory by redoubling his already Herculean efforts. He earned an M.D., as well as a Ph.D. in Immunology and Cell Biology, finishing the program in 1996. While at Downstate, Andy was inducted into the Alpha Omega Alpha Medical Honor Society and also received an award for outstanding

achievement in Clinical Neurology, as well as first prize in the Alpha Omega Alpha Student Research Symposium. Following graduation, he was accepted into the neurosurgery residency training program at Columbia University's Neurological Institute. He completed an internship in General Surgery at Columbia before proceeding to his neurosurgical residency in 1997.

In 2002, Andy was lured away from his beloved East coast to become assistant professor of Neurological Surgery at the University of California San Francisco. He rapidly rose through the faculty ranks, becoming a tenured professor in 2011, as well as vice-chairman. He was the first recipient of the Reza and Georgianna Khatib Endowed Chair in Skull Base Tumor Surgery. His prolific clinical practice was focused on brain tumors, skull base tumors, and spinal cord tumors.

He established a successful laboratory in glioma immunology and immunotherapy where he maintained continuous NIH support. He was the recipient of the Elsberg Award through the New York Society of Neurosurgery, as well as the Preuss Resident Research Award from the American Associatceived the Young Clinical Investigator Award, the Mahaley Clinical Research Award, the American Brain Tumor Association Clinical Research Award, the Integra Foundation Award, and a Journal of Neuro-oncology Award through the AANS/CNS Joint Section on Tumors. In his prolific career, he published more than 300 peer-reviewed articles, review chapters, and monographs.

Among his major accomplishments was basic research creating vaccines using patient's innate glioblastoma tumor tissue. The subsequent innovative Phase II trial with autologous heat shock protein tumor vaccine was pioneering in the realm of personalized medicine. Early results of this experimental cancer vaccine showed promising potential for extending longevity and improving quality of life in newly diagnosed and recurrent malignant gliomas. These studies were still ongoing at the time of his passing. TL; DR: Andy discovered what might be a cure for the nastiest brain tumors, the kind of medical achievement that a certain committee in Oslo is known to reward with a prize. Alas, he will not be around to collect it.

Despite his ambitious schedule, he provided service and leadership in neurosurgery at a national level in many areas. He served with distinction on the Editorial Boards of Neurosurgery and the Journal of Neurosurgery.

His academic and professional accomplishments were impressive, but how he accomplished them was even more remarkable. He made everyone around him better, making him or her feel like a valuable member of the team.

Combining an energetic style, brilliant insights and passion for teaching, he transformed the cultures of the places where he worked, establishing lifelong and committed relationships with collaborators and colleagues.

His work ethic was driven by a fierce urgency and he approached every goal with the intensity and work ethic of someone who knew from hard-won experience that our days are finite.

Liam Walsh, MD '09

Dr. Walsh, 33, went missing in November 2015 while backcountry skiing in Alaska. Authorities found his body in early 2016. Dr. Walsh was a pain medicine specialist in Alaska Regional Hospital in Wasilla, and helped develop a successful clinic in Juneau.

Remembrances

IN MEMORIAM LIST FOR 2015-2016

Included below are all the reported deaths received by the Alumni Association in 2016

CLASS OF	NAME
’36	Saul Rotter, MD
’42	Fred Schilling, MD
’43	Franklin Cannizzaro, MD
’45	Sam Tally Simpson, MD
’46	Herbert Leroy Abrams, MD
’46	James F. Morell, MD
’47	William Kelly, MD
’47	Harold Menger, MD
’48	Vincent DeLuca, MD
’50	Jay J. Gold, MD
’51	Jerome Maisel, MD
’51	Morris Soled, MD
’51	Augustus Tanaka, MD
’52	Lauren Howard Lucke, MD
’52	Clifford Reichert, MD
’55	Isaac Sanders, MD
’56	Leonard Meltzer, MD
’56	Gerald G. Cole, MD
’56	Jack Eisert, MD
’56	Philip Alexander, MD
’56	Jacob Brody, MD
’56	Jack Eisert, MD
’56	Maxwell Felton, MD
’56	Barry Held, MD
’57	Thomas Cassidy, MD
’57	Jerome Klein, MD
’57	Sheldon Lang, MD
’58	A. Ron Sorvino, MD
’58	Ann Wennhold, MD
’59	Gerald Greenberg, MD
’60	William Paul, MD
’60	Joseph Zuch, MD
’65	Samuel Himelstein, MD
’65	Andrew Weiss, MD
’68	Richard Luft, MD
’71	Roberta Flesh, MD
’87	Michael Ammazzalorso, MD
’89	Jennifer Ng, MD
’92H	Laurence Finberg, MD
’96	Andrew Parsa, MD
’09	Liam Walsh, MD

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Name: Graduation Year:

Phone: (Home) (Office)

Fax: E-mail address:

Mailing Address:

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