Editors’ Introduction
The following reminiscence by Lucy Balian Rorke-Adams is the eighth autobiography in a series published in the Journal of Neuropathology and Experimental Neurology. These have been solicited from senior members of the neuropathology community who have been noted leaders and contributors to neuroscience and to the American Association of Neuropathologists (AANP) and have a historical perspective of the importance of neuropathology in diagnosis, education, and research. It is hoped that this series will entertain, enlighten, and present members of the AANP with a better sense of the legacy that we have inherited, as well as reintroduce our respected neuroscientists as humans having interesting lives filled with joys and sorrows and allow them to present their lives in their own words.

MNH, RAS

GROWING UP
My career as a pathologist began at age 4 when, after a heavy rain, I would gather angle worms that had crawled from cracks in the sidewalk and “dissect” them to see what lay beneath the surface (Fig. 1). This curiosity about things seen and unseen was the engine that powered my career, although there were a few brief detours before I was challenged by the largely undefined field of pediatric neuropathology. As the youngest in a family of 5 girls, I benefited from the guidance of my sisters, along with parents who provided a secure, nurturing home environment and constant encouragement to achieve in whatever challenges presented themselves (Fig. 2).

From my earliest years, my insatiable curiosity was stimulated by remarkable teachers at all levels and continued from grade school to the last quarter of medical school. I became a bookaholic at an early age—the books opening vistas of an exciting world beyond the restricted life in the country setting of my home.

In my teens, I had dreams of a career in opera. Ours was a household filled with music and I became captivated with opera when I was 6 years old, through Saturday afternoon broadcasts of the Met. An audition with one of the glamorous Met stars, Gladys Swarthout, who was considering taking on a protégé, came to naught because she became ill. I was devastated. Upon reflection, I decided that my destiny did not include a career in the opera world, although operas have remained a lifelong passion.

CHOOSING MEDICINE
About this time I came upon a novel, The Magnificent Obsession, by Lloyd Douglas; it is the story of a feckless playboy who indirectly caused the death of a famous neurosurgeon. This led to a dramatic decision to train himself to fill the role of the man whose life he had inadvertently destroyed. The powerful effect of this book fired my decision to select a career in medicine.

As I was also fascinated by human behavior, I entered the University of Minnesota in 1947 as a premed student with a major in psychology and a minor in anthropology. My goal was to become a psychiatrist; hence, I went onto graduate school and obtained a Masters degree in Psychology before entering medical school in 1953. During the first 2 years in medical school, I had a private practice as a clinical psychologist, but during the last 2 clerkship years, I did independent research on the etiology of schizophrenia. Alas, an inside view of how psychiatry was practiced was disillusioning and directed me away from that specialty (Fig. 3).

My next detour led me to consider a career in neurosurgery, although I received no encouragement from Dr. William Peyton, Chief of Neurosurgery at the University (1). Following graduation from medical school in 1957, I began a highly coveted rotating internship at The Philadelphia General Hospital (PGH). As one of 108 interns serving the needs of Philadelphia’s indigent population at this 1,800-bed hospital, I experienced the most remarkable year of my life.

Although accustomed to functioning on only a few hours of sleep during my 10 years at the University (as I had to work for tuition and books), I was severely sleep-deprived during that 12-month period. It was not really meant to be so difficult, but reality intruded by dropout of several interns for a variety of reasons: one became psychotic during the first month, another died during the flu epidemic in September 1957, a third developed tuberculosis, another had a brain abscess caused by Staphylococcus abscessus on his neck during the obstetrics rotation, to which we were both assigned. So, naturally, he was not allowed on the delivery floor, and we who were healthy had 18-hour shifts for a month.

There were problems with residents as well. My first month was on neurosurgery, and only a few days into the internship, the resident developed acute appendicitis, leaving me alone on the service. The psychiatry resident the second month decided to enjoy a long holiday, so abandoned me after the first week. I was devastated. Upon reflection, I decided that my destiny did not include a career in the opera world, although operas have remained a lifelong passion.

There were problems with residents as well. My first month was on neurosurgery, and only a few days into the internship, the resident developed acute appendicitis, leaving me alone on the service. The psychiatry resident the second month decided to enjoy a long holiday, so abandoned me after she had reviewed charts of all patients for whom I would be responsible. Had I not had experience as a clinical psychologist, I could not have coped.

In retrospect, those were primitive times; as for the 2-week period that we were assigned to the laboratory, we were the night laboratory technicians for the entire hospital! The second half of that month, I was on the anesthesia service...
because the State of Pennsylvania required all licensed physicians to be experienced in administering anesthetic agents. That meant, of course, that stretching out on a bed was only dreamed of, as one sat for hours in the operating room handling nighttime emergencies. It is with some amusement that I listen to complaints from today’s house staff regarding frequency of night calls, work load, and others. We all survived the tremendous demands, and in the process, I do not think any patients suffered because we were sleep-deprived.

The major casualty of this state of affairs, however, was reconsideration on my part of pursuing a career in surgery. It was a given at that time that surgeons routinely got little sleep and I realized that, although I had not made any grievous errors, a life with only minimal sleep was not attractive.

In view that decisions regarding residency training had to be made months ahead of time, I opted not to seek a position in a surgical program, but instead temporize with a year in pathology.

Given my precocious start in dissecting angleworms at age 4, and a wonderfully inspiring professor of pathology in medical school, Dr. James Dawson (of Dawson’s inclusion body encephalitis fame), I applied for a residency position in Pathology at PGH. It was a fabulous program, headed by Dr. William Ehrich, distinguished for his expertise in diseases of the kidney and the reticuloendothelial system. There were about 1,200 autopsies each year; hence, exposure to a wide array of diseases was without parallel.
ON BECOMING A PEDIATRIC PATHOLOGIST

As I have related elsewhere, a directive from Dr. Ehrich on my first day of residency was pivotal to my transformation into a pediatric neuropathologist. On that day, Dr. Ehrich walked into the room where the residents were waiting, looked around, and then came to where I was sitting and said, "You're the only girl (!) here, and since pediatrics is the province of ladies, you have to do all the pediatric autopsies."

Well, we learn by doing under guidance of our mentors, so during the 3 years of anatomic pathology residency, more than half of the 500 plus autopsies that I performed were infants/children, and my interaction with the pediatricians stimulated in-depth study of diseases of infancy and childhood.

NEUROPATHOLOGY AS A SUBSPECIALTY

It was at the suggestion of my husband, Bob Rorke, whom I married in 1960, that I pursued a career in neuropathology, as that would combine my longtime interest in mind/brain and diseases of children. A National Institutes of Health Fellowship allowed me the opportunity to remain at PGH to study under Helena E. Riggs, one of America's pioneer and premier neuropathologists.

How fortunate I was to have learned from and worked with this brilliant, accomplished woman for the last 7 years of her life. Dr. Riggs had entered the University of Pennsylvania medical school in the 1920s, when few women chose such a difficult profession. She was one of 4 daughters, and only her father encouraged and supported her decision. She originally chose to specialize in neurology and practiced with another neurologist to whom she was engaged. Following her fiancé’s tragic death from a brain tumor, Dr. Robert Winkleman invited her to consider a position as a neuropathologist at PGH, very much a fledging endeavor. Dr. Winkleman was also a neurologist, but it was customary in the 1930s for neurologists and neurosurgeons to do their neuropathology because most general pathologists abdicated responsibility for the diagnosis of CNS diseases—it was just too difficult to deal with the complex neuroanatomy, both gross and microscopic.

Upon completion of the Fellowship in 1962, I was invited to join the PGH staff as chief of pediatric pathology and assistant to Dr. Riggs. My 7-year association with her was priceless. She generously shared her knowledge of clinical neurology and CNS pathology and refined my writing skills as well. A manuscript summarizing CNS lesions in congenital rubella syndrome underwent 8 revisions before she allowed me to submit it!

Literature documenting CNS development and disorders of infancy and childhood was relatively sparse in the 1960s; the richest sources were in German. Whereas
embryological features were well known, there was a giant gap between this developmental phase and infancy. In particular, aside from the study of isolated portions of the nervous system based on a single specimen, little was known about myelination development (2–5). Although pediatricians generally reassured parents of prematurely born infants that they would “catch up,” there was no documentation as to how and when in postnatal life this would occur. That question could not be answered without knowledge of the normal development of myelin.

With funding from the National Institutes of Health, Dr. Riggs and I studied myelination in the infant brain, from the stage of viability (around 500 g body weight) to term. During that same period, Yakovlev and Lecours published their detailed studies of myelination of the infant brain (6).

Our work was in the final stages of preparation for publication when Dr. Riggs died in October 1968. An atlas illustrating myelin development in the fetus was published the following year (7). Aside from daily mentoring, Dr. Riggs introduced me to the larger community of neuropathology at American Association of Neuropathologists (AANP) meetings, which in the 1960s traditionally took place in Atlantic City, NJ, in conjunction with the American Neurological Association. She forced me to participate in discussion of papers, even though I was a neophyte and would never have dared speak up if left to my own inclinations.

Perhaps her closest colleague was Orville T. Bailey, another giant in the field. They created the Diagnostic Slide Session in the late 1950s, a well-established feature of our annual meetings to this day. Both Dr. Riggs and Dr. Bailey were reserved and dignified in their social contacts, and Dr. Riggs was so self-effacing that when she was nominated to become the first woman president of the AANP, she declined.

It was a privilege to be included in Dr. Bailey’s circle because we had a common interest not only in CNS diseases of infants/children (to which knowledge base he had made considerable contributions) but also in our love of opera and literature. Dr. Bailey was a member of the Chicago Literary Society, an exclusive group devoted to maintaining high standards of literacy. His magnificent presentations to that Society remain prizes in my library.

**BRANCHING OUT**

Shortly after joining the staff at PGH, I was asked to participate in endeavors beyond the confines of PGH. This resulted in involvement in many fascinating projects and greatly broadened my world. Eventually, I became known as “the girl who couldn’t say no” (in matters limited to science!).

**Vaccines**

The first major foray came from an invitation from Wyeth Laboratories to do neuroviroence studies connected with manufacture of Sabin’s oral polio vaccine, which were required by the Division of Biologic Standards for safety testing. A major component involved injection of vaccine from a given lot into the thalamus and spinal cord of 18 monkeys. After 21 days, these monkeys were killed, and it was my responsibility to examine the tissue for evidence of any excessive inflammation caused by the vaccine or contamination by B virus, a viral agent tolerated in monkeys but fatal for humans.

This work expanded into involvement with the development of rubella vaccine in collaboration with Dr. Stanley Plotkin, Smith Kline-French Laboratories and Institut Merieux in Paris, and adenovirus and influenza vaccines produced by Wyeth.

My experience in the world of vaccines led to an invitation to serve as an expert for the Division of Vaccine Compensation program established by an Act of Congress in 1988, to deal with parental allegations that vaccines had caused serious disease or death in their children. This program was established when pharmaceutical manufacturers threatened to discontinue vaccine production because court judgments involving millions of dollars in frivolous suits were devastating.

In those cases where serious CNS injury was claimed, and for which tissue was available for study (biopsy or autopsy), I was asked to review the clinical and pathological features and to render an objective opinion relative to what role, if any, the vaccination(s) played. Findings in 37 cases established no credible evidence of a cause-effect relationship between permanent CNS injury/death and vaccination (8).

**The Children’s Hospital of Philadelphia and the Wistar Institute**

In 1965, Dr. Charles Kennedy, Chief of Neurology of The Children’s Hospital of Philadelphia (CHOP), invited me to rectify the haphazard state of neuropathological diagnosis that prevailed there—sometimes being done by the neurosurgeon or an itinerant neuropathologist. It was about this time that children with subacute sclerosing panencephalitis (SSPE) were challenging the neurologists and brain biopsies were being done. Dr. Michael Katz, a pediatrician-virologist at The Wistar Institute of Philadelphia, requested a portion of the tissue to attempt animal transmission and perhaps to culture the responsible agent. The experimental animal chosen was the ferret (about which I knew nothing), which was chosen because ferrets were susceptible to rubella, although at that stage, the causative agent of SSPE was not known.

The evolution of this work was fascinating. Sometime after the homogenized brain was injected intracerebrally into the ferrets, the animal handlers noted a change of “personality.” Basic ferret behavior is aggressive, and animal handlers routinely wore thick gloves when working with the creatures. Well, the animals became placid and offered no resistance to being handled.

My first encounter with microscopic features of the ferret brains was both positive and negative. I found a small amount of inflammation and thought I saw an inclusion body. In retrospect, this turned out to be wrong, but because Dr. Katz had such faith in my expertise (!), he decided to expand the studies. Eventually, we not only succeeded in transmitting the disease to ferrets, but with the help of Dr. William Maslund, a neurologist at the University of Pennsylvania, we found that EEG changes appeared shortly before clinical changes became manifest.

Following the success with SSPE, research attention was focused on multiple sclerosis, for which a viral etiology...
was a leading hypothesis at that time. This work at Wistar was done in collaboration with Dr. Hilary Koprowski, a distinguished scientist and virologist, who had developed a vaccine for both polio and rabies.

For these studies, we used chimpanzees housed in a primate colony in San Antonio, TX. This necessitated trips to the colony from time to time, and a series of adventures that, by themselves, would fill many pages.

Success in these investigations eluded us; so funding was withdrawn after 20 years, and my long association with Wistar came to an end. Although disappointed at our failure, I was relieved not to be on call 24/7 for 20 years to do postmortem examinations on patients with multiple sclerosis who donated their brain for the advancement of science.

Challenges of a Pediatric Neuropathologist

The Perinatal Brain

When I was forced to do all of the pediatric autopsies in the late 1950s, most of the babies were preemies who died consequent to what was called “respiratory distress syndrome” or “hyaline membrane disease.” Aside from and associated with the pulmonary problem, these babies commonly had a variety of CNS lesions, the pathogenesis and pathology of which were emerging consequent to the pioneering studies of Drs. Betty Banker and Jeanne-Claudie Larroche. To assist colleagues with limited access to such cases, I prepared a monograph describing the clinical and pathological features of hypoxic-ischemic CNS lesions in the developing brain; the material available for study was enormous (9).

Malformations

One of the major challenges of pediatric neuropathologists is diagnosis of malformations. Early in my career, I was frustrated by the lack of information regarding the pathogenesis of developmental abnormalities. Evaluation was limited to careful dissection, photography, and description of the findings, but the larger questions of “HOW?” and “WHY?” remained obscure. This frustration drove me to explore work of developmental biologists, and in the process, I stumbled on the intriguing world of Caenorhabditis elegans and Drosophila. Studies using mutant genes suggested parallels that could explain certain developmental malformations in humans, particularly migration disorders. Thus, almost 20 years ago, I boldly advanced the hypothesis that pathogenesis of this group of malformations was a consequence of disordered genetic control of neurogenesis (10). A rapidly growing body of data since then has supported this hypothesis.

It is fascinating to reflect on the extraordinary shift of pediatric neuropathology in the 1960s to contemporary times. Advances in obstetric care, genetic diagnosis, use of ultrasound, and fetal magnetic resonance imaging associated with legalization of abortion have essentially eliminated the detailed morphological study of complex CNS malformations in newborns. Current practice and methods used to terminate a pregnancy generally result in delivery of a fetus that is too autolyzed to study; hence, photographic archives along with tissue slides of material accumulated in the past have become increasingly precious.

Tumors

Medulloblastomas

A major passion has been the study of brain tumors in children with the ancillary benefits of close collegial association with neurosurgeons, neuroradiologists, oncologists, and geneticists. Advances in neuro-oncology for the past 50 years have been breathtaking, although much remains to be learned.

Among tumors that primarily affect children are those categorized as embryonal, the most common neoplasm in this group being medulloblastoma (MB). My increasing experience with the spectrum of histological features of this tumor and knowledge of the literature raised some perplexing issues. The most prolific and strongest voice in this arena was Lucien J. Rubinstein, one of the most famous neuropathologists of his time, and a colleague whose views were not lightly challenged. Prevailing opinion during the 1960s and 1970s was that MB was a uniquely cerebellar tumor, the cell of origin of which was the external granular cell (less commonly the internal granular cell). The problem with this opinion was basically 2-fold, that is, histologically similar tumors arose in other parts of the nervous system, and studies had demonstrated that, under normal circumstances, the external granular cell could only differentiate into neurons.

The first problem was solved by giving a different diagnosis to the noncerebellar tumors, for example, central neuroblastoma, pineoblastoma, among others, but the second remained because some of the MBs exhibited a glial component.

In 1973, Hart and Earle (11) proposed calling noncerebellar tumors that looked like MB, “primitive neuroectodermal tumor.” Despite their suggestion, there was continuing introduction of diagnostic terms for tumors that bore a resemblance to MB but displayed variable histological features. Some of these arose in the cerebellum but could be found in other sites as well. Consequently, our clinical colleagues had increasing difficulty understanding this jumble of unfamiliar diagnostic terms.

I attempted to provide a dispassionate review of the dilemma and used the forum of my Presidential Address to our Association in 1982. It was my hope that others would take an interest in the issue and frame objective studies to investigate the fundamental biology and oncology of these embryonal tumors. Although I expected some opposition from Dr. Rubinstein, I was unprepared for the bitterness of his attack. In his opinion, the Presidential Address was an inappropriate forum to advance new concepts. Rather, he told me, one should review one’s past accomplishments, that is, provide a summary of the career up to that point. His ire was perhaps accentuated by his opinion, which he had previously shared following the announcement that I was to become President of the Association, that I was too young for that honor.

He was also annoyed that the Address would be published in our Journal without benefit of peer review, as was the tradition for the Presidential Address at that time. Actually,
John Moossy, who was editor of the Journal did make a few modifications, but the Address was published almost exactly as it was given (12). As it turned out, Dr. Rubinstein’s strong opposition to my suggestion that we use Hart and Earle’s term, “primitive neuroectodermal tumor” for all CNS tumors that basically looked like MB regardless of their location in the CNS focused attention on the issue and stimulated investigations that might have taken longer to unfold. Of interest, Dr. Laurence Becker, a neuropathologist at The Hospital for Sick Children in Toronto, independently proposed the use of the diagnostic term, “PNET” in an article published in the same year as my Address (13).

Thirty-one years after my address ignited this firestorm, it is gratifying to note that much has been learned about embryonal tumors. The fact is that research in the intervening years has shown that the problem was considerably more complex than the conceptual approach that I had proposed at the time. It is with great satisfaction that I view the research efforts by many investigators who have established the cytogenetic and biological signatures of embryonal tumors including four subtypes of MB. Indeed, it may now be appropriate to discard the diagnostic term PNET, as suggested by Raffel and Rutka (14), for it has served its purpose.

**Atypical Teratoid/Rhabdoid Tumors**

By the mid-1980s, I had seen a large number of pediatric brain tumors and was sensitive to a wide range of histological features within a given diagnostic group. It was about that time that I received a cerebellar tumor from an 11-month-old baby, which contained a remarkable combination of features, all of which I had hitherto not seen in a single tumor. These consisted of fields of typical MB plus other regions resembling sarcoma and adenocarcinoma. Most intriguing were sheets of rhabdoid cells, identified for me by my colleague, Dr. Jane Chatten. Kidney tumors composed of such cells were familiar to pediatric pathologists but were not generally in the ken of neuropathologists. The tumor could not be classified as a teratoma as none of the germ cell tumor markers were expressed.

Exploration of the literature, consideration of many tumors sent for consultation, discussions with the oncologists, and results of cytogenetic studies of embryonal tumors led to the realization that CNS tumors with these diverse biological, histological, and genetic features represent a unique entity. The diagnostic term that I thought most appropriate was “atypical teratoid/rhabdoid tumor” (15).

Although this is now an established diagnosis for a clinically and cytogenetically unique, primarily infantile tumor, my presentation of 32 cases at a satellite brain tumor symposium in Ottawa (Canada) in 1994 was greeted with considerable derision by at least 1 outspoken member of the audience.

**The Forensic World**

In the meantime, PGH was closed by mayoral edict in 1977. I was then asked to take on a half-time position at the Office of the Medical Examiner, for which, up to that time, I had done many consultations. Simultaneously, I shifted from a part-time to a full-time position at The Children’s, so now my official workday was 12-plus hours. In retrospect, I am not certain when I slept, as intercalated in all of this, I did the neuropathology (including brain cutting and lectures) for 2 large hospitals in the vicinity, plus the research at the Wistar Institute. Cecilia Bartoli, the marvelous Italian diva, once remarked that she thought she was “born to sing.” Well, I guess I was “born to work”!

In contrast to the relatively calm atmosphere of academia and hospital pathology, my association with the Medical Examiner of Philadelphia plunged me into the world of murder and mayhem. By nature of the field, the forensic pathologist is at a considerable disadvantage, as he/she is often presented with a body (or parts thereof) and little or no information explaining why it is there. In this arena, therefore, the pathologist must be both a physician and a detective—a sometimes-daunting challenge.

Whereas I dealt with cases of all kinds in my work with the Medical Examiner, all unexplained deaths of infants and children became my special focus. Although postmortem in some uncovered an obvious lethal disease, that is, meningitis, the majority were victims of abuse. Because many of them had been hospitalized at The Children’s, I had the advantage of knowing the clinical and radiological findings before I did the autopsy.

Over the many years of involvement with my clinical and neuroradiology colleagues, and my hands-on postmortem examinations, an understanding of the pathophysiology of abusive head trauma emerged. While pondering the CNS lesions and their biomechanical basis, it became apparent that the high frequency of cervical spinal injuries must be a consequence of whiplash forces; reports of spinal cord trauma in abused infants were spotty, however, most likely because the cord was not systematically examined or the lesion was destroyed because of an incorrect removal technique. In a publication in 2003, I outlined steps to be followed in a postmortem evaluation of infants suspected of abuse (16). I called attention to the importance of the removal of the brain and spinal cord in continuity to preserve the integrity of the tissue at the cervicomedullary junction, the region of greatest risk from whiplash forces. Our own studies documented spinal cord trauma in 70% of infants dying of abuse (17).

The forensic work led to the involvement of a number of bizarre cases and several that were high profile. Most notorious were the trial of Joel Steinberg, an attorney in New York City, who killed his adopted 6-year-old daughter Elizabeth in 1987, and the unsolved murder of Jon Benet Ramsey in 1996. In both, my work was behind the scenes assisting the prosecutors during and after the Steinberg trial; and in the Ramsey case, testimony before the Grand Jury involved a remarkable cloak and dagger journey to and from Boulder because the prosecutor did not wish to expose me to the media camped out at the Court House. Because of intermecine conflict between police and prosecution, there was no trial; hence, my involvement never became public.

Perhaps most fascinating was a several-week sojourn in London in June of 2005 for testimony in the hallowed chambers of the High Court of Justice of England and Wales before 3 bewigged Lord Chief Justices. Although complex,
the basic issue was the appeal of about 80 individuals who had been convicted of child abuse. The appeals followed repudiation of testimony by 3 neuropathologists (and others), namely Jennian Geddes, Waney Squiers, and Helen Whitwell, all of whom had testified in the original trials leading to the convictions. Repudiation was based on a study published by Geddes et al in 2003, claiming that subdural hematomas and retinal hemorrhages were consequent to hypoxia-ischemia, not trauma (18). Details of these memorable 3 weeks, both in and out of the courtroom, would fill many pages, and which therefore cannot be recounted here. However, 2 things stand out: 1) the difficulty of imparting to even intelligent jurists the medical complexities of abusive head injury in children and 2) the repudiation by Geddes under oath of her seriously flawed publication (19). This, sadly, has not prevented her disciples from publishing articles supposedly proving her assertion that hypoxia-ischemia causes subdural and retinal hemorrhages. Unfortunately, our system of jurisprudence gives authority for decisions regarding forensic medical issues to judges, lawyers, and jurors. Medical issues of homicidal child abuse must therefore be presented in a simplified manner to people who have no knowledge of the complex pathophysiology involved. Because the courts are basically adversarial, testimony of forensic pathologists, who are trained to be objective observers, is challenged by “experts” for hire who typically testify under oath that the lethal injuries found at autopsy are simply a consequence of an ordinary natural process, for example, venous thrombosis, hypoxia-ischemia, and others.

Years ago, Walt Kelly, creator of the comic strip, “Pogo,” had Pogo voice his sage observation, “we have met the enemy...and he is us....” It is sad to observe colleagues sell their integrity. The same fallacious arguments are used repeatedly, making an observation by Sarah Beckwith all too true: “... when something is shouted loudly enough, often enough, and to enough people, with no checking of the accuracy of what’s being shouted, a downright silly claim can come to sound like a long-suppressed truth” (20).

Teaching

By nature of the setting in which I work, it has been my privilege to teach many of the next generation of pathologists, neurologists, and neurosurgeons. Teaching is basically a personal sharing of a part of oneself with someone else and is most rewarding when the student not only absorbs the transmitted knowledge but also responds with ideas and facts that have sprouted from the seeds that were planted.

The students tended to fall into 3 groups: the majority who incorporated the material into a building block for their career, a fraction whose interests were elsewhere during the neuropath rotation, and a special group who, following the training, became lifelong friends. It is with pride that I have followed the stellar career of many of my trainees. They are too numerous to mention, except for Jeff Golden, who occupies a unique place in my heart. Jeff introduced himself to me after a lecture during his freshman year at the University of Pennsylvania Medical School in 1983 and told me that he wanted to become a neuropathologist. This was the start of a special student-teacher association that eventually culminated in his position as Pathologist-in-Chief at CHOP when he became my boss (Fig. 4)!

I was profoundly moved by statements of former students whose accolades led the Provost of the University of Pennsylvania to confer upon me his Award for Excellence in Teaching in 2003. Although this is an extraordinary honor, my greatest satisfaction derives from the knowledge that I succeeded in transmitting knowledge that I had acquired over many years to the next generation who have the responsibility and tools to advance the field in previously undreamed of directions. In this regard, my greatest legacy is the creation of the Lucy Balian Rorke-Adams Chair in Pediatric Neuropathology by CHOP in celebration of my 80th birthday. This will ensure advances in our knowledge of CNS disease in children for years to come.

Administration

When I chose medicine as a career, it never occurred to me that I would spend countless hours as an administrator. My professional career began at PGH in 1962 as an assistant to Dr. Riggs and Chief of Pediatric Pathology, with the additional responsibility for supervising the residents doing
When Dr. Riggs died in 1968, I became the sole neuropathologist, having to deal with about 1,000 brains each year and, consequently, was less active in the pediatric postmortems. Dr. Ehrich had died in 1967, and through a complex series of events, I reluctantly became Chairman of the Pathology Department at PGH in 1969. Now I had 2 jobs, chief of the department and neuropathologist, which I managed fairly well, along with my duties at The Children’s, research at Wistar, and vaccine work for Wyeth Labs.

Then in 1973, I became the first woman president of the medical staff at PGH and dealt with that without too much difficulty until the medical director at PGH had a heart attack and died suddenly. For the next 10 months, I had the added burden of his job, “24/7”!

My term as medical staff president lasted until 1975, and when the hospital closed 2 years later, I was thrilled to shed all of my administrative responsibilities. I promised myself that I would confine my activities to the full-time position at CHOP and half-time position with the Medical Examiner and undertake no further administrative responsibilities.

A few years later, I was invited to become the first woman medical staff president at CHOP and refused. Some pressure was applied and, in particular, I was told that the role of president was simply ornamental and only involved participation in charming social events. I subsequently relented but was horrified to learn that the Board had fired the Chief Executive Officer (CEO) shortly after I assumed office. This meant that the Chief Operating Officer and I were responsible for the day-to-day operation of the Hospital. At the same time, I was the sole neuropathologist at CHOP so I had to fulfill my professional duties as well; 18 months later a new CEO took over.

Life settled back into a pleasant routine until May 1995 when Dr. Camillus Witzleben, Pathologist-in-Chief, came into my office and, after quietly closing the door, informed me that he was retiring a year earlier than planned, and that although a search committee had already been selected, someone needed to take over the department until a permanent successor arrived. He and the CEO had ordained that I should be that person. Before I could respond, he hastened to assure me that I would probably be relieved of the responsibility within...
6 months; hence, the task should not be too onerous. As he was leaving, however, he voiced this cheery afterthought: “On the other hand, Lucy, it may be six years.” Well, he was remarkably prescient, as the 6-month period stretched into 5½ years!

CONTRIBUTIONS TO MEDICAL HISTORY

Philadelphia General Hospital (PGH), where I spent the first 20 years following graduation from medical school, was the oldest hospital in America, originally founded as an almshouse in 1729. It was often referred to as “Old Blockley,” because the buildings were located on what had been the property of a displaced Englishman, George Warner, whose childhood home had been called “Blockley.” As such, it was steeped in the nation’s medical history, and many pioneers of American medicine contributed to its fame. One of the most famous, Sir William Osler, was actually a Canadian; while he was at the University of Pennsylvania from 1884 to 1889, he went to PGH next door and introduced the practice of ward rounds for medical students. He also performed autopsies in a small brick building on the grounds of PGH, called “The Dead House” (Fig. 5).

This building was still intact when I arrived at PGH in 1957 and contained Osler’s instruments, the autopsy table, and all of his handwritten postmortem reports. When the Mayor of Philadelphia closed PGH in 1977, I spearheaded a plan to transfer all of these treasures to The College of Physicians of Philadelphia, the oldest medical society in America, founded in 1787. The College building houses the Mütter Museum and a world famous medical history library.

My second contribution to The College of Physicians consisted of 46 slides of Einstein’s brain.

When Einstein died of a ruptured aortic aneurysm in Princeton Hospital in 1955, the postmortem was done by Dr. Thomas Harvey. Dr. Harvey had been an anatomical pathology resident at PGH and knew that Dr. Ehrich had a superb technician, Marta Keller, in his laboratory at the University of Pennsylvania. Mrs. Keller was highly skilled in the use of the sliding microtome and celloidin-embedding techniques.

Dr. Harvey removed Einstein’s brain (without permission) and asked Dr. Ehrich to have Mrs. Keller prepare the sections. She made 5 sets of slides, which Dr. Harvey then distributed as follows: Hartwig Kühlenbeck, neuroanatomist at Women’s Medical College (no longer in existence); Max Winternitz-Harry Zimmerman at Yale; Percival Bailey, neurosurgeon in Chicago; and a neuroanatomist in Los Angeles, whose identity is unknown to me. The fifth set he gave to Dr. Ehrich.

Following Dr. Ehrich’s death in 1967, his widow gave the slides to Dr. Allen Steinberg, who had been an intern and resident with me and had also remained at PGH as Dr. Ehrich’s assistant. One morning some years later, shortly after Mrs. Ehrich died, Dr. Steinberg appeared in the doorway to my office with a slide box in his hands and announced, “Lucy, since you’re a neuropathologist, and I know nothing about the brain, I’m giving you Einstein’s brain slides.”

The box contained 46 celloidin-embedded tissue slides; there were 23 serial sections of which one set had been stained by the Nissl technique and the other by Weigert. There was no section of cerebellum. The tissues were beautifully preserved, and I was impressed by the absence of vascular

**FIGURE 6.** (Left) With Bob Rorke, my husband of 42 years. (Right) With Boyce Adams, my second husband of only 2 years (photo taken by Dick Davis at the 2004 AANP meeting).
lesions, lack of gliosis, and the pristine nature of the neurons, which contained only minimal lipofuscin.

After remaining in my possession for about 35 years, I gifted them to the Mütter Museum at The College of Physicians, where they are now elegantly displayed. This exhibit has excited the curiosity of hundreds of visitors over the past 2 years and is an ironic turn of events because Einstein specified that following death, he should be cremated and his ashes scattered; he did not wish to have his tomb become a pilgrimage site. The ashes were thrown into the Delaware River, but the remnants of the illegally removed brain have now become the pilgrimage site. The whereabouts of the other 4 sets is currently unknown.

THE SUMMING UP

Upon reflection, I can think of no more exciting way to have lived my life. My nurturing family, teachers, and friends encouraged me to follow wherever my curiosity led me. My husband of 42 years, Bob Rorke, and my second husband, Boyce Adams (for a much shorter period) were number 1 in my fan club; and without their love and support, the career would never have flourished (Fig. 6). I accepted the challenges as they were presented and tried to do the best I could with each new endeavor. Somehow, I managed to accomplish everything I undertook each day and even had some hours to enjoy my family, friends, music, books, and worldwide travel. Even though I am well past the usual retirement age, I work full time and meet the daily challenges of this fascinating specialty with vigor. Who could ask for anything more wonderful?

REFERENCES

2. Bruce A. Illustration of Mid and Hind Brain. London: Young J Pentland, 1893