

Recollections of 40 Years of Hospitalization of Children

Presidential Address to the American Pediatric Society, May 24, 1972

WARREN E. WHEELER^[1]

Department of Pediatrics, College of Medicine, University of Kentucky, Lexington, Kentucky, USA

When I first faced the realization that the honor of being President of this Society carried with it the awesome responsibility of a Presidential Address, one of my first thoughts went back to the days when I was a young resident, and old John Lovett Morse gave the house staff a preview of a paper he'd written entitled, "Recollections of 45 Years of Infant Feeding." This was a wonderful bit of nostalgia and it gave me for the first time a concept of the progress that had been made in infant feeding, and why. It was my recollection that this was Doctor Morse's Presidential Address before this Society, but I was wrong; as a matter of fact, he was never even President of the Society. So you see, my memory is not so good and if you care to challenge me on some of the recollections I am about to make, you are welcome to do so.

My first contact with children in hospitals came in 1932, at the Boston Children's Hospital, and I look back now at the tremendous changes that have taken place in the 40 years since that time. In those days precarious infants and prematures died early, and the infants ward was considerably smaller in census than the older children's wards. The duration of hospitalization of all infants and children was considerably longer than it is today, and the city afforded far fewer beds than there are today. These 40 years have seen a tremendous rise in scientific medicine including what I would consider the two most important developments relating to hospitalization which are the use of antibiotics, and the development of safe anesthesia for children. There has been a gradual shift away from the hospitalization of medical patients with the prolonged stay of those with rheumatic fever and nephritis, toward the use of intrepid surgery and the vogue for salvaging the unsalvageable. Today money raising publicity goes to the great feats, such as open heart

surgery, the kidney machine, and transplantation of organs; and the public has come to expect medical miracles each new week. Not all the progress has been favorable because these years have seen, except for intensive care nursing, the passing of the skilled loving care of bedside nurses. In those days, nurses even kept children occupied and happy; now we hire specially trained play ladies to do this. I have no idea of what the *per diem* cost of hospitalization was in those days at the Children's Hospital, but when I went to Columbus, in 1945, the *per diem* cost at the Children's Hospital there was \$6.43. At the end of the 1st year, I was called into conference by the superintendent to see what might be done about our financial crisis since the cost had risen to \$6.87 per day. Today *per diem* costs in the neighborhood of \$100 or \$125 are commonplace.

Such is progress, and with these developments I had little to do. I want instead to talk with you today about some of the things that I have participated in, namely, making hospitalization for children safe; and although I am afraid time will not permit, I would have liked to talk to you about making hospitalization for children smooth. A great deal of the hospitalization in my early days was concerned with infectious disease. Most large cities had fever hospitals such as the Haynes Memorial and the South Dept. of the City Hospital in Boston, the Willard Parker in New York, the Sydenham in Baltimore, and the Herman Kieffer in Detroit. The reason for these hospitals was to prevent the spread of infectious disease in ordinary children's hospitals. At the Children's we took care of children with syphilis, and gonorrhoea, and polio on the open wards with what we called cubical isolation. On the older children's wards I remember four isolation rooms close by the nurses' desk where we nursed dysen-

tery and typhoid patients. As I recall these had screen doors because there was, of course, no air conditioning, and although the main ward windows were screened, we couldn't guarantee the absence of flies. Our separate isolation ward was only open from December to May. Our main reliance against cross-infection was "technique" which was taught to us and enforced by hard-nosed nurses who didn't hesitate to chew us out if they caught us retrieving articles from the floor or leaning on the crib side. Actually about our only weapons against the development of infection once a child was exposed were horse serum against scarlet fever and the meningococcus, convalescent serum against chickenpox and mumps, and Charlie McKhann's new placental extract which miraculously prevented children from developing measles.

I suppose my first experience with nosocomial epidemic disease was when I was a house officer on the infants' ward and we began to receive baby after newborn baby from the hospital in Lynn where diarrheal disease in the nursery had become quite common. Even then it was curious that no one had the fortitude to stop deliveries in that hospital since the babies we received all died within a day or two after transfer. After we'd lost a lot of infants, the Lynn people finally began to refer us babies when they had their first liquid stool, and we began the most vigorous parenteral fluid treatment we could think of even though the babies looked in very good condition. Now I can look back and see why my attending pediatrician referred to these babies as having cholera infantum and can realize why safe conduct through the illness depended upon what we considered prodigious quantities of glucose and salt solution. I still remember the peculiar odor of the stools of those babies, something I learned later to associate with diarrhea due to *Escherichia coli* 0 111 B₄. Later on in the 1930's and the early 1940's, epidemic diarrhea of the newborn became a scourge in obstetrical hospitals all over the country except in the town of Chicago. Epidemics broke out there, too, but a very unpopular but perceptive health officer by the name of Bundeson decreed there should be no epidemic diarrhea of the newborn in Chicago. Though no one else was able to accomplish this with isolation "technique," he did. When two babies in a nursery had loose stools, he simply closed the delivery room and prohibited the introduction of new susceptibles to the nursery.

It wasn't until the early 1950's in Columbus that I was able to study an outbreak of 0 111 diarrhea, to identify the causative organism with agglutinating serum supplied to me by Bill Ferguson of the Michigan Department of Health, and to try the effect of a new antibiotic, neomycin. We quickly found that neomycin would save lives,

and we lost no patients. So we thought if we could cure these patients with neomycin, then new cases would stop. But new cases kept breaking out and the epidemic on our ward came to a halt only after all susceptibles had been used up. By then it had become apparent that between the time diarrheal disease showed itself and we diagnosed it and used neomycin, the disease had already spread to other susceptibles. We reworked our thinking at that point and tried giving neomycin to all young babies with diarrheal disease as they entered the hospital, until we knew they did not have enteropathogenic *E. coli* in their stools. If one stopped feeding them, their stools stopped, and the chance for dissemination of enteropathogenic *E. coli* was tremendously diminished. By the time we fed them again their stools were almost sterile. By using this principle I was able to conduct a large diarrhea ward for the next 9 years with only three or four cross infections documented during that time, and with a reduction of hospital stay for diarrhea patients from 11.5 days before this procedure to about 6.3 days afterwards. The exception that proved the rule came in 1961 when a neomycin-resistant strain of 0 111 B₄ was imported to our wards from Chicago. Again we were unable to render babies noninfectious quickly and another epidemic broke out and continued until we hit upon colistin to do the job. My teacher, Doctor Blackfan, had taught me that if one increased a baby's feeding too rapidly in the postdiarrheal period, his nutritional status would "break" and he would have a relapse of his diarrhea. It turned out that if one prevented cross infection by other strains of enteropathogenic *E. coli* from his neighbors most babies could have their feedings increased rather rapidly without relapse, and they got well and went home in half the time. Here we saw working the principle that, for most bacterial disease, is our mainstay today. To prevent cross infection render the infectious patient noninfectious quickly, relying for only a brief period of time on the practice of "technique" to halt the spread of the organism.

A few years later the staphylococcus raised its ugly head. The nosocomial epidemics of the Golden Menace of the 1950's were bad enough on the adult wards of the hospitals, but they were especially insidious in nurseries. Although colonized by "hot" strains of staphylococci in the first few days of life, the impetigos, the fatal pneumonias, osteomyelitises, and septicemias didn't become apparent for a week or 2 after the baby went home. Then the baby often went to another facility for care. Often those who ran the nursery were the last to know the extent and seriousness of the outbreak. Again, this was a strain of a common organism which could be charac-

terized and identified, this time by phage typing. We were struck by the similarity to the situation with enteropathogenic *E. coli* and the thought struck us: was this another example of a particularly virulent member of a family of ubiquitous organisms? With Tom Schaffer we asked this question and were rewarded by finding that in nurseries all over the country the 80/81 strain of staphylococcus was a major "hot" strain. In another similarity to the O 111 *E. coli* it was obvious that the practice of isolation "technique" did not and could not contain this strain in nurseries and on hospital wards. Control eventually came from the prevention of colonization of patients and personnel, but it came by identifying and eliminating carriers, or by prevention of colonization through the use of a protective antibiotic such as erythromycin, and later with the intentional colonization of newborns by a dog-in-the-manger nonvirulent staphylococcus, the 502A strain. I think in those years we learned how to control hot strains of staphylococci, and I would like to think our efforts to spread the use of this knowledge led to the abatement of the widespread epidemic situations in the late 1950's. Unfortunately, I don't think we were that influential; rather, I think the epidemics ground to a halt because of the introduction of methicillin and the waning of the epidemic potential of the organism. Today's 80/81 staphylococci are not to be compared with the nascent 80/81's from hot lesions in 1954.

The last few years have seen a dramatic rise in the importance of gram-negative sepsis and a decline in staphylococcal outbreaks in hospitals. Public health officials have become complacent about the "staph" problem, and young doctors have never witnessed babies developing empyema and dying of staph pneumonia. Only 15 state health departments still perform phage typing in their laboratories, and several of these are having technical difficulties. So it is not surprising that no one protests when a branch of the Federal Government tells us to stop using hexachlorophene on our babies. Forgetful, but influential pediatricians have even stated that it never

helped anyway—just have nurses wash their hands! I think they're whistling in the dark. I have far more faith in the capacity of bugs to mutate than I have in the memory of the whistlers.

What made the epidemic 80/81 strain a hot strain?: its exquisite ability to invade and cause serious lesions, its ability to colonize the noses of patients and personnel in spite of competition with normal flora, and its ability to flourish despite the use of the popular antibiotics of the day. These properties are independent genetic traits subject to mutation. A truly hot strain has them all. It would amaze me if in the future some new strains don't develop which will smoulder without recognition for a while, passing from one human to another, juicing up their virulence as they do, until they add the property of methicillin resistance and explode.

History may be interesting and fascinating; it also ought to be instructive. I see no reason to deny that important hot strains of common bacteria will not rise again. Their emergence will be beyond our control, but we will be surprised and embarrassed if we don't keep constantly looking for them. I agree that today we don't have to do the extensive culturing that we did in the early 1960's. But we had better keep just as tight a surveillance on our end product—the baby—and make sure that we know that he stays well after he goes home. Let us hope we've learned enough in the past to nip future outbreaks in the bud. Prevention is still much better than cure.

It has been fun to be in pediatrics and to participate in the development of a few of our present concepts. These have been an exciting 40 years for me; I only hope you will be able to say the same when the time comes for you to turn and look back.

Notes

1. Requests for reprints should be addressed to: WARREN E. WHEELER, M.D., Department of Pediatrics, College of Medicine, University of Kentucky Medical Center, Lexington, Ky. 40506 (USA).
2. Received for publication June 26, 1972.