

THE YEAR BOOK  
*of*  
MEDICINE  
1973

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# THE YEAR BOOK *of* MEDICINE 1973

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There are twenty YEAR BOOKS in various fields of medicine and one in dentistry. Publication of these annual volumes has been continuous since 1900. The YEAR BOOKS make available in detailed abstract form the working essence of the cream of recent international medicoscientific literature. Selection of the material is made by distinguished editors who critically review each year more than 500,000 articles published in the world's foremost journals.

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# INFECTIONS

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DAVID E. ROGERS, M.D.



## INTRODUCTION

M. Glenn Koenig, M.D., died on November 13, 1972. He was 41 years old. During the 17 years of our association he was initially my student, soon my dear friend, my closest working associate and, in later years; my teacher. I miss him enormously.

Glenn packed a remarkable career into his short lifetime.

He was an imaginative and skilled scientist. His contributions to our understanding of the pathogenic mechanisms of staphylococci, the therapy of bacterial endocarditis, the diagnosis of botulism, the treatment of disseminated fungal infections, earned him high honors, including early election to the American Society for Clinical Investigation and the Association of American Physicians and full professorship at Vanderbilt.

He was a splendid clinician. These words are nowadays sometimes applied for trivial skills. Not in Glenn's case. He was a meticulous historian. His conduct of a physical examination was a joy to behold. His tenacity in getting to the root of a complex problem of illness was a lesson for all who worked with him. His wise and empathic continuing care of his patients was a model for colleagues and students. He cared and it showed.

He was a great learner-teacher. His lectures, seminars, ward rounds and research reports were fascinating, informative and fun. Glenn was both a scholar and an inveterate showman. He could be an incredible clown when the situation was appropriate, but he always did his homework, and his presentations were lucid, well organized and thoughtful. He was an early recipient of the "Golden Shovel" award given by the Vanderbilt students for the best teacher in the institution—and he prized it muchly.

Perhaps what makes him most memorable was his humaneness. Glenn was a fully formed man with many of the qualities we each hope to have as *Homo sapiens*. He always did his best at whatever he undertook and expected others to do the same. But this he coupled with tolerance and understanding of the foibles of most of mankind. Glenn did not suffer fools kindly, but he had deep sensitivity to the needs of those who were insecure or frightened or less gifted than he. He believed deeply in the worth of every human being, hated hypocrisy, prejudice, posturing or dishonesty in any form, and he made his opinions known. He was always himself and one always knew where Glenn stood. This was not always comfortable, but this remarkably consistent openness made it easier for others to try to be likewise. Glenn grew bigger people by example and they recognized it.

I could say much more about this fine man—his obsession with fishing, his never-ending battles with crab grass and bare

spots in his lawn, his story-telling and mimic session when he got wound up, or the relationship with his equally remarkable wife, who shared in these qualities and similarly shares them with their children and with others. However, by now Glenn would be grumping to get down to work on this volume and we shall.

In midyear Dr. Zell A. McGee and Dr. William Schaffner, two close colleagues of Glenn's and mine in the George Hunter Laboratory, took over much of Glenn's labors on selecting material for the YEAR BOOK. We have just spent 2 intense days together in which they have given me the 1973 cram course and arranged our offerings. We have talked much and thought more about Glenn and dedicate our efforts to him with a salute to his wife, Connie, and his two young hockey players, Mark and Scott.

DAVID E. ROGERS

## PART I

# INFECTIONS

### INFECTIONS AND THE HOSPITAL

► In pre-Listerian days, the hospital was dreaded—and for good reason. Although plagues from without were the major killers of the day, infections from within commonly accompanied admissions for childbirth, trauma or surgery. Control of the environment, sanitation, vaccination and, to a lesser degree, antimicrobials have led to the effective control of many of the acute microbial diseases of man, but not their elimination. Despite our microbiologic sophistication, our hospitals—by virtue of the kinds of patients admitted, the manipulations we put them through and the human traffic flow—remain risky places to be. We have emphasized this each year in a chapter entitled “Infections of Medical Progress.” That some infections are due to our tendency to forget simple epidemiologic precepts of former times is illustrated in the 2 following articles. The potential hazards of some of our new technologies are well illustrated in the remaining articles in this chapter.—Ed.

**Tuberculosis Outbreak in a General Hospital: Evidence for Airborne Spread of Infection** is presented by N. Joel Ehrenkranz and J. Leilani Kicklighter.<sup>1</sup> At Jackson Memorial Hospital, Miami, Florida, a 1,200-bed municipal general hospital with 3,786 employees, 7 cases of active tuberculosis occurred in employees in 1962–68. A time-and-place clustering was noted. Of 5 infected employees who worked mainly in one hospital wing, 4 became ill in 1967–68. Up to 200 employees work in this building at any given time. Patients with active tuberculosis may be admitted to either of two units in this section.

In 1969, a patient with unrecognized active tuberculosis was ventilated in the emergency room and then spent 57 hours in one of the units. Vigorous nasotracheal suctioning was carried out, and spontaneous extubation and leakage around the endotracheal tube occurred. The patient lived for 127 hours after hospital admission and died of tuberculous bronchopneumonia and pulmonary edema.

There were 24 employee converters from tuberculin-negative to tuberculin-positive skin tests among direct and indirect contacts with the patient, including 21 of 60 persons working on the study unit. Ten had little or no direct contact with the patient and probably were infected by the spread of *Mycobacterium tuberculosis* through an unbalanced air-conditioning system. Conversion occurred in 13 of 26 employees with close contact with the patient. Three of 8 workers with possible active tuberculosis, found in 1969–70, had worked in the study unit, and 2 had converted after exposure to the patient.

Air from patients' rooms in the study unit was not well filtered and was recycled to employee areas. Distant infection of susceptible personnel through ventilation systems should be preventable by using bacterial filters and, perhaps, by the correct use of ultraviolet irradiation of air. Corridors should not be used to exhaust or supply air for patients' rooms. The chain of tuberculous infection can be maintained in hospitals by spread between patients and employees. Routine skin-testing

(1) *Ann. Int. Med.* 77:377–382, September, 1972.

programs are the simplest means of identifying infected hospital employees.

► [Tuberculosis has long been recognized as an occupational hazard for those who work in hospitals. Although the risk has been sharply reduced by the dramatic fall in the number of people with active tuberculous disease, the marked *increase* in tuberculin-negative (and thus susceptible) adults makes for impressive epidemics when live tubercle bacilli are introduced into a crowded environment.

In the late 1950s and early 1960s, Riley and his co-workers showed clearly and conclusively that airborne transmission of tuberculosis could occur (Am. J. Hyg. 70:185, 1959; Am. Rev. Resp. Dis. 85:511, 1962). His studies appear reaffirmed in the Jackson Memorial outbreak. It would appear that, in 57 hours, 1 patient with unrecognized pulmonary tuberculosis infected 21 persons. This was accomplished via the medium of one of the glories of modern technology—air conditioning! In a summer in the mid-1960s in Tennessee, my colleagues and I treated a young woman with cryptococcal meningitis. In attempting to determine the source, we found she sat regularly in front of her kitchen air conditioner. On examination it was discovered that the aging unit pulled air in over a bird's nest, and bird droppings lodged in the outer air intake. We believed (but did not prove) that she had acquired her infection in the same manner from this cooling aerosol. — Ed.]

**Spread of Pertussis by Hospital Staff.** Many physicians do not recognize pertussis as a disease that adults easily may acquire. Thomas L. Kurt, Anne S. Yeager, Sandra Guenette and Stuart Dunlop<sup>2</sup> report two outbreaks of pertussis that occurred among hospital staff members at the University of Colorado Medical Center in 1969. The first outbreak was not recognized for several weeks. The organism was introduced into the hospital by a child aged 2 months and spread to the house officer who cared for the child. The physician infected 2 outpatient children and 2 adults.

In the second outbreak 3 cases occurred in persons attending a small graduate class. The source children had been seen at home by a nurse who transmitted pertussis to a ward nurse taking the graduate course. When the outbreak was recognized, all staff in contact with any pediatric patients were kept under surveillance; staff members with respiratory symptoms had nasopharyngeal smears taken for fluorescent antibody study and were sent home. Eleven adults had presumptive diagnoses made from positive smears. Nonimmunized infants received pertussis immune globulin on admission and were immunized. The wards were closed to elective admissions for 2 weeks. Two adults were seriously incapacitated. Sore throat occurred transiently in 5 of the 11 adult patients.

A total of 39 Bordet-Gengou cultures and 96 nasopharyngeal fluorescent antibody smears were obtained from 78 pediatric personnel during the outbreak. Two adults had positive cultures for pertussis. Five of 12 persons with positive fluorescent antibody smears did not show a rise in pertussis agglutination titer or acquire a clinical syndrome. All persons with positive smears received antibiotics, usually ampicillin. Three retained a positive fluorescent antibody test 5 days later. Serologic findings are given in the table. Pertussis agglutination titers of 1:20 or higher were considered evidence of protection or recent exposure. No rise in adenovirus complement-fixation titers was found in 11 persons suspected of having pertussis. No viral isolations were made in 7 cases.

This outbreak had economic consequences for the hospital. Fortunately other secondary cases did not occur in small children or newborn

(2) J.A.M.A. 221:264–267, July 17, 1972.

PERTUSSIS AGGLUTINATION TITERS IN STUDY GROUPS

Study Group	No. Studied	Pertussis Agglutination Titers	
		No. With Titers ≥1:20	Percent With Titers ≥1:20
Pediatric house staff 1969-1970, 10/69	33	23	69
New pediatric interns 1970-1971, 7/70	28	17	61
Inpatient nurses			
Older children	19	4	21
infants	26	12	46
Premature	22	4	18
Intensive care	12	4	30
Outpatient staff	26	2	7
New nurses, after 10/69	26	6	23
First year medical students, 7/70	114	31	27
Adults, no patient contact	47	10	21

infants. Pertussis is much more common in the hospital environment than is generally appreciated.

► [More common than appreciated, but it really should not be. The relative rarity of many of the serious infections of childhood has tended to make all of us less obsessive about comprehensive childhood immunizations. Witness the frequent epidemics of measles, the sporadic outbreaks of diphtheria (1972 YEAR BOOK, p. 71) and the occasional cases of neonatal tetanus in the South. The authors suggest that a vaccine "safe for adults" should be developed, but prevention of pertussis in children seems a more logical and well-tested approach. — Ed.]

**Sepsis Caused by Contaminated Intravenous Fluids: Epidemiologic, Clinical and Laboratory Investigation of an Outbreak in One Hospital.** A nationwide epidemic of hospital-acquired sepsis in 1970-71 has been traced to contaminated bottle closures of intravenous fluids produced by one manufacturer. Stephen K. Felts, William Schaffner, M. Ann Melly and M. Glenn Koenig<sup>3</sup> report a study of an outbreak of this sepsis at the Vanderbilt University Hospital. All 36 patients who had *Enterobacter cloacae*, *erwinia* or *Pseudomonas stutzeri* isolated from their blood from July, 1970, through December, 1971, were studied, and in 31, contaminated intravenous fluids appeared responsible. Daily change of all fluid administration lines curtailed the outbreak.

In the 31 patients, serious underlying disease predominated. All but 4 patients were febrile, with hectic fever patterns. Duration of fever averaged 4.9 days. Phlebitis occurred at the infusion site in 36% of the septic patients. Transient hypotension occurred in 23%. Fourteen patients were receiving an antimicrobial to which the isolate was sensitive *in vitro*. The clinical picture of persistent gram-negative sepsis was common. The average increase in hospitalization for 13 patients was 12.8 days. All epidemic strains were sensitive to gentamicin and kanamycin. The proportion of contaminated fluid bottles ranged from 24 to 47% in various lots. Growth curves for the organisms are shown in Figure 1. The fluids remained clear. Normal adult serum killed most *erwinia*, and polymorphonuclear leukocytes phagocytosed *erwinia* and *E. cloacae* *in vitro*. In a mouse model, the pathogens behaved no differently from control *E. coli*.

(3) Ann. Int. Med. 77:881-890, December, 1972.



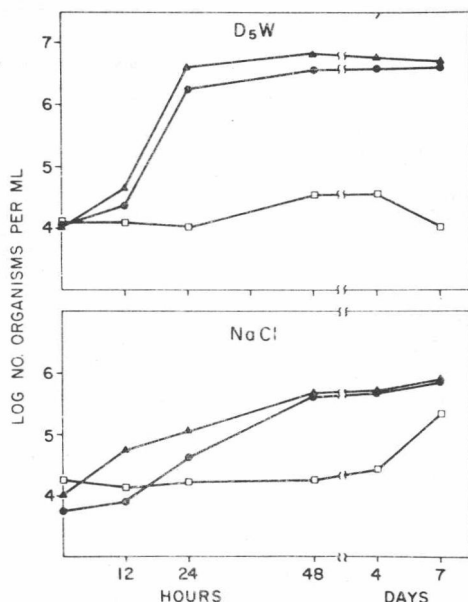


Fig. 1.—Growth curves of *erwinia* (solid circles), *E. cloacae* (solid triangles) and control *E. coli* (open squares) in Abbott 5% dextrose in water ( $D_5W$ ) and in Abbott normal saline ( $NaCl$ ). (Courtesy of Felts, S. K., et al.: Ann Int. Med. 77:881–890, December, 1972.)

The organisms involved in this epidemic were unusual blood stream pathogens. The space between the bottle cap and bottle neck was evidently contaminated when the bottles were sprayed with tap water after sterilization at the factory. The organisms involved were capable of persisting and multiplying in both 5% dextrose in water and in normal saline. The epidemic was characterized by persistent gram-negative sepsis, with repeatedly positive blood cultures despite apparently adequate antimicrobial therapy. For intravenous therapy likely to last several days, a new delivery set and needle should be used about every 24 hours.

► [Most alarming! While physicians have generally considered drugs for parenteral use and intravenous fluids sacrosanct, it is now clear that this is not necessarily the case. Further, despite the fact that this epidemic took place in a first-class university hospital with an active in-hospital epidemiologist and infectious disease team (my bias), it took 11 months to recognize the source. Even then it was that fact that the bacteria were uncommon blood stream pathogens that led to this detective work.

Abbott has been using the screw type bottle closure for 35 years for its intravenous fluids. The problem of "why troubles now?" has not been completely answered, but Center for Disease Control studies suggest that a recent change in the substance used in the liner of the bottle cap may have contributed. The old liner (gilsonite) was shown to have bacteriostatic properties against the strains involved, whereas the new one does not. As stated by the authors, "... it seems that an 'insignificant' change in packaging unknowingly increased the infection risk associated with use of the product." A new type of pliofilm packaging of smoked whitefish that "prolongs shelf life" probably contributed to the type E botulism epidemic reported from the same laboratory in 1964. Ah, technology!—Ed.]

**Fever and Bacteremia Associated with Hypertonic Saline Abortion.** With the liberalization of the New York State abortion law, instillation of intra-amniotic hypertonic saline has been the accepted form of abor-