Infection in Britain Yesterday

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My yesterday is distanced from today not merely by half a century of time, but by a vast leap in technology, a revolution in biological thinking and perhaps a deep change in many aspects of culture.

In the middle 1930s, when I qualified, a resident in a fever hospital in London was something of a gentleman. He was called with tea at 8 a.m., by his personal maid. After a leisurely bath, in his private bathroom, he repaired towards 9 a.m. to the hospital laboratory. This was very well equipped. We had platinum loops and Bunsen burners, wire baskets for the Loeffler slopes, an incubator and microscopes. These were equipped with oil-immersion lenses, and I can still remember the thrill when we switched from high power to oil-immersion and saw, in what we regarded as the ultimate in definition, those graceful dotted rods which we recognised as KLBs, Klebs-Loeffler bacilli. We had, of course, examined the noses and throats of our patients on admission the day before and we knew that these KLBs confirmed our clinical diagnosis.

The Loeffler slopes being all examined, and the baskets returned to the incubator, the laboratory was closed for the day and we made our way to the dining-room where, provided the senior resident medical officer had already arrived, we broke our fast about 9.45 a.m., presided over by the senior dining-room maid. We then glanced through the *Morning Post* and *The Times,* and finally towards 10.45 a.m. we made our way to the wards where before lunch we might see between 100 and 150 patients suffering from that very common and very severe malady, diphtheria. This was infectious disease in 1938, and we accepted it as an everyday part of life, or of death, in the East End of London.

We were fairly skilful at making a clinical diagnosis of diphtheria. We had plenty of practice and we knew the danger to the patient of waiting for laboratory confirmation. Even when the laboratory test was negative we still often preferred our clinical diagnosis. We were, of course, sometimes wrong but we had seen so many children die because they were treated too late. We had seen many children die from cardiovascular collapse brought on in the first two weeks of illness by diphtheria toxin. We had seen others who survived that stage, only to die, weeks later, from pharyngeal or respiratory paralysis. Some survived even these complications, perhaps after weeks in a Drinker respirator or iron lung. If they did recover, and this was the bright side, they recovered completely, with none of the severe residual respiratory paralyses we were to see, years later, in our patients with poliomyelitis.

That was diphtheria as we saw it in the wards every day, year in, year out. Unfortunately, we sometimes saw it as a result of infection on the ward. Today we have a new Hospital Infection Society and a new *Journal of Hospital Infection.* But hospital infection is nothing new, and in my book for nurses on infectious diseases, published in 1946[1], I had a chapter on cross-infection: the diseases dealt with include diphtheria, measles, dysentery, gastroenteritis, chicken-pox, mumps, rubella, typhoid and scarlet fever. In my early days I think I had experience of cross-infection with all of these. But perhaps scarlet fever was the main one. It caused us more trouble than any other of the infectious diseases. I came to know well the intense purplish rash of toxic scarlet fever, for it was not uncommon, and in 1938 I myself got it a few days after opening a cervical abscess on the scarlet fever ward. I remember being delirious but I also remember how in my delirium I heard the medical superintendent say to the sister, 'Do his parents know?' Well, my parents did not know, but that was a lesson to me ever afterwards to be careful of what one said in front of patients, even delirious ones. I did not, however, get any complications, probably because, as a doctor, though a very junior one, I was nursed in a side ward and did not have to inhale streptococci from other scarlet fever patients.

The concept of scarlet fever as a streptococcal disease was only then being fully established. In 1884 Loeffler[2] had isolated streptococci from the throat, lymphatics and internal organs of patients dead from scarlet fever, but he could not decide whether they caused the disease. In 1905 Jochmann[3] reported that he could not isolate streptococci from the blood of patients who died from scarlet fever after only a few days of illness, but that he could nearly always isolate them from patients who died a week or two after the onset of the disease. He proclaimed that the early deaths were caused by scarlatinal poison, and that streptococci became operative only later and caused death by septic complications. Jochmann's views were accepted until the 1920s and were not, of course, very far wrong. (The Dicks published their work on the erythrogenic toxin only in the mid-twenties[4].) The primary illness of scarlet fever is caused by streptococci and their products, but the septic complications are caused by streptococci, often different streptococci. Later work[5-7] made it clear that many of the complications and so-called relapses of scarlet fever were caused by streptococci of a different type from that present on the patient's throat on admission, i.e. by cross-infection.

We could not easily control this cross-infection in our large open wards, and within a year, in 1938, I saw almost every possible complication of scarlet fever—

mastoiditis, meningitis, cervical abscess, nephritis, arthritis and rheumatic endocarditis. We also had return cases—cases occurring in a house to which we had discharged a patient within the previous 28 days. The discharged patient had usually developed some evidence of persisting infection—a sore nose, swollen glands or a running ear. We had no sure way of preventing this. We sometimes insufflated a patient's nose with sulphonamide powder for four or five days before discharge, but we were somewhat scared of using these new-fangled drugs parenterally: the early ones like sulphapyridine were quite toxic. Later we found that sulphonamides did not eradicate streptococci from an inflamed throat.

Scarlet fever was common in 1938, 10,200 cases being admitted to the London fever hospitals. In the same year there were 7,268 admissions for diphtheria, and 13,644 for measles. Roughly 1,000 cases of each disease came to the Eastern Fever Hospital in that year. Two years ago the Eastern closed for infectious diseases.

So what happened? We can explain diphtheria. We had known about diphtheria immunisation and practised it to some small extent since the late 1920s, but in the early 1940s Sir Wilson Jameson, Chief Medical Officer at the Ministry of Health, launched a national campaign to get rid of this preventable disease. He conducted his campaign on the radio, with such success that within a few years diphtheria ceased to be an epidemic disease in this country. On one day in 1941 the hospitals in Liverpool had 800 cases, but when I took over in that city five years later we had one small ward of mainly doubtful or negative cases, and that ward was closed for diphtheria a year or two later.

Scarlet fever almost disappeared, too, and such cases as occurred were always mild. We saw no more toxic or septic cases, but for this decline there is no obvious explanation other than that the streptococcus seemed to have lost its epidemic thrust. Diseases do seem to be like empires, they do decline and fall; whether, unlike empires, they can rise again, I do not know. Was there some change in the social conditions in London between 1938 with its 10,000 admissions for scarlet fever and 1978 with possibly none at all, some change big enough to explain the disappearance of this severe and sometimes fatal disease? I find that hard to believe, although there may have been an improvement, nutritional perhaps, great enough to explain the fall in the severity of measles during that period. By 1938 we had long since achieved most of the sanitary improvements that had such an effect on some of the great epidemic diseases: on typhus, for example, which in 1846 killed nearly 6,000 people in Liverpool. Typhoid too had come under sanitary control, although in 1936 there were over 700 cases in the milk-borne outbreak in Bournemouth and a year later 300 in the water-borne outbreak in Croydon.

The improvement in hygiene had a different effect on another infectious disease, poliomyelitis. From 1947 onwards this was the epidemic scourge of the developed world. In the 1952-53 epidemic in Copenhagen cases were being admitted to the Blegdams Hospital at the rate of 50 patients a day. Altogether they had over 3000 cases and more than 300 of them had respiratory embarrassment. Doctors in fever hospitals had used iron lungs or tank respirators to treat children with diphtheritic respiratory paralysis: they formed perhaps the only body of doctors at all familiar with these machines, but it soon became obvious that there were not enough machines to go round. The same doctors were also expert at performing tracheotomies, for they had had ample experience in treating children with laryngeal diphtheria. Anaesthetists used positive pressure bag ventilation through a laryngeal tube every day during surgical operations but few would have felt that it could be continued indefinitely through a tracheostomy tube without risk to the patient. But when I visited the Blegdams Hospital I saw no fewer than 75 patients being ventilated in this way: final year students operated the ventilating bags, their text-books lying open before them on the patient's bed. We had nothing like this in Britain, but as a result of the Danish experience special treatment centres were set up and equipped in the infectious disease hospitals and it might well be said that it was from this intensive work with poliomyelitis patients in the Western world that the modern concept of intensive care emerged in the early sixties. Immunisation has almost eradicated poliomyelitis as an epidemic disease in that world, but unfortunately this is far from true in the developing world, where there is also very little that could be called intensive care.

I could go on and on. I was taught that the best treatment for a patient with acute lobar pneumonia was good nursing and fresh cool air playing over the patient's face; patients' beds were wheeled over to the open ward windows each morning. We had no specific therapy. I can well remember my sister suffering from lobar pneumonia and how early in the morning on the ninth day of her illness she had a drenching sweat which marked the onset of the crisis and the beginning of recovery. In many other diseases too we had no specific treatment. In my 1946 book I wrote of tuberculous meningitis that 'as the disease accelerates the child shows every sign of grave meningitic involvement: his condition becomes extremely distressing and death comes as a relief from suffering'. The only drug we used was morphia. I also wrote, 'Diet is of supreme importance in the treatment of typhoid fever. Alcohol is occasionally useful, especially if the patient is used to alcohol', a statement which is still true even after the discovery of many antibiotics. Of rubella I wrote, 'Recent work tends to show that if a woman suffers from rubella in the early months of pregnancy, the resulting child may show some deformity. It is too early to say whether this is indeed the case, but such figures as are available do suggest that there is some connection'. It gives me some satisfaction that in those early days I expressed myself with such commendable caution. On the treatment of scarlet fever with antitoxin I was not over-enthusiastic. 'It should be remembered', I wrote, 'that serum treatment is very expensive: an average dose may cost as much as one guinea so that it should not be used on a lavish scale unless there are very good reasons for it'. When I wrote that, I was a medical superintendent, in complete administrative control of my hospital, and obviously very cost-conscious.

There are many famous names in the history of infectious disease in this country, such as Maurice Mitman, E. H. R. Harries and Stanley Banks. They worked full-time behind high walls inside large fever hospitals, isolated sometimes almost as much as their patients. They were known as 'fever merchants', a term which included not a little of affection and a great deal of admiration. I was too junior, I think, ever to earn that title, but it has been a pleasure to recall something of the atmosphere of that era.

This article is based on a paper read at the Conference on Infection in Britain Today held at the Royal College of Physicians in November 1980.

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Book Review

Topics in Therapeutics 6. Edited by H. F. Woods. Pitman Medical, Tunbridge Wells, 1980. 204 pages. Price £10.50.

This books consists of the papers delivered at the Topics in Therapeutics Conference held at the College in November 1979. Most are single author contributions from clinical pharmacologists and, like its predecessors, the volume provides a number of short and well-written reviews on selected topics, with a bias towards subjects that emphasise the scientific basis of drug therapy.

It has been said that one of the main differences separating man from the lower primates is the avidity with which he consumes drugs, and this book starts appropriately with a section dealing with the patterns and problems of prescribing in the NHS. It is no surprise to learn of the continuing popularity of psychotropic agents and, in particular, the wide acceptance of the benzodiazepines. Wade's survey of prescribing practice shows that BNF preparations are seldom used, but would fulfil 91 per cent of prescriptions without loss to the patient and at a saving of 25 per cent of the drug bill.

The second section is two chapters devoted to self-monitoring of therapy. Sönksen's contribution on blood glucose monitoring in the home is a model of informed excellence. The simple expedient of allowing patients to regulate their own diabetic control is both popular and cost-effective. Raftery's companion chapter demonstrates the feasibility of blood pressure monitoring by the hypertensive patient, but leaves a few unanswered questions about its value.

The next section deals with the metabolic consequences of therapy, including drug-induced diabetes, and concludes with a useful, if waspish, review by Alberti on drugs and the interpretation of laboratory biochemical tests. This identifies a real need for closer communication between physicians (the clinical cuckoos) and pathologists (the wise sparrows), and the extent of the problem is illustrated by the number of drugs that interfere with common laboratory tests. It provokes the thought that routine biochemical screening of asymptomatic people should be abandoned.

The fourth section on drug metabolism is occasionally repetitious in detail and over-involved, but it repays attention, for much of clinical interest emerges.

The management of hypertension in pregnancy confirms the value of methyldopa but makes little mention of hydrallazine (considered by some authorities to be the drug of first choice). This chapter is partnered by one on the safety of drugs during lactation, which refers to six conflicting references on the excretion of warfarin in breast milk. This is now known to be negligible, but the example justifies the criticism that this area of therapeutics requires more direct research.

The sixth section provides a wealth of information on nutritional aspects of therapy. The chapter on diet and drugs is of particular clinical relevance, and Lee's review of parenteral nutrition is of great topical interest, though the expense of correcting nutritional deficiency in the critically ill by this means is mind-boggling for, apart from the cost of nutrients, the laboratory sparrows should earn massive overtime dealing with a hefty paragraph of recommended daily monitoring requests. Hockaday writes competently about the use of gel-forming fibre in diabetic dietary management—a use unfortunately limited by the unpalatability of present guar mixtures.

The last section on selected topics was, perhaps, the most rewarding, for it demonstrated quite superbly how the scientific approach to pharmacology in medicine can illuminate the practice of therapeutics to the advantage of the patient. Though it would be invidious to select a best buy from amongst these excellent offerings, Herrington's chapter was a model of style and a most interesting contribution to the biology of depression.

I enjoyed reading this book and benefited from it, and though my copy bore a few physical scars of hasty assembly the lapses are minor and the references are up-to-date.

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