MAY, 1952

THE ULSTER MEDICAL JOURNAL

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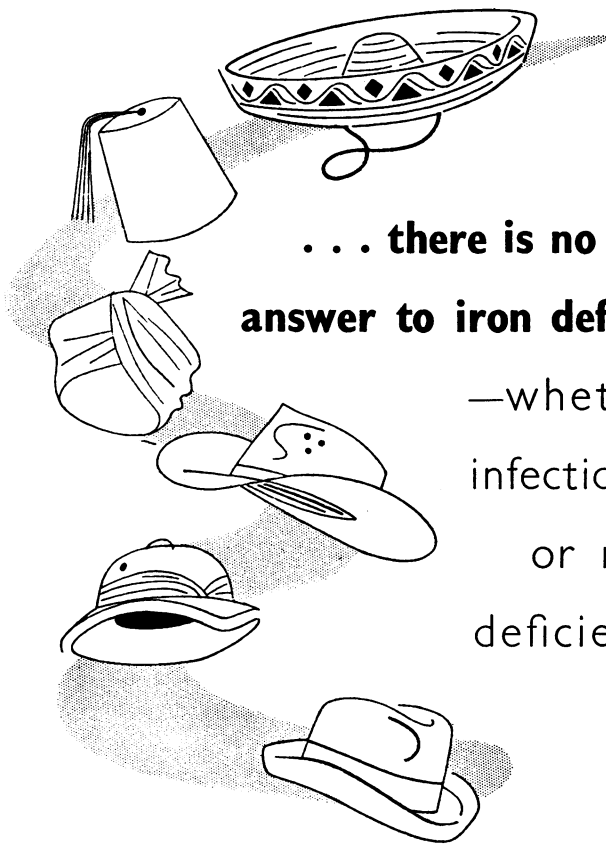

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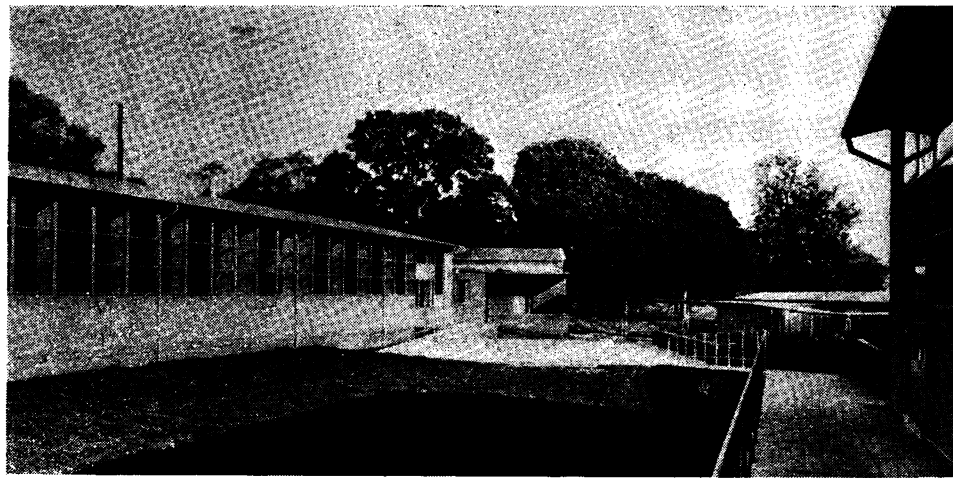

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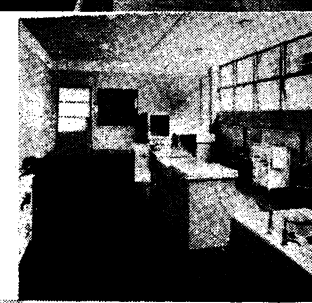

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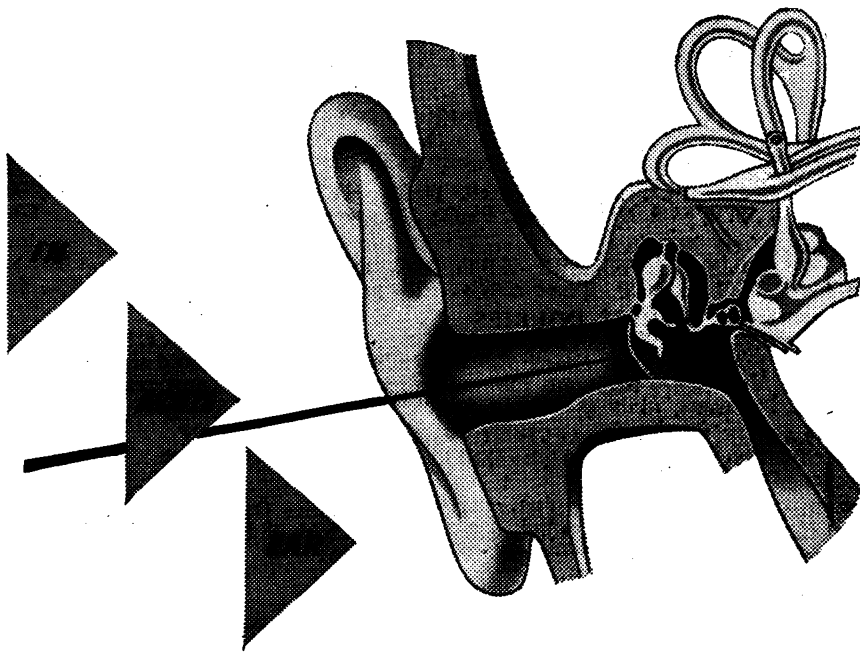

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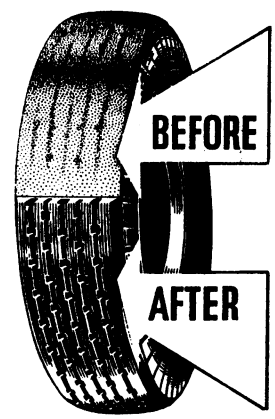

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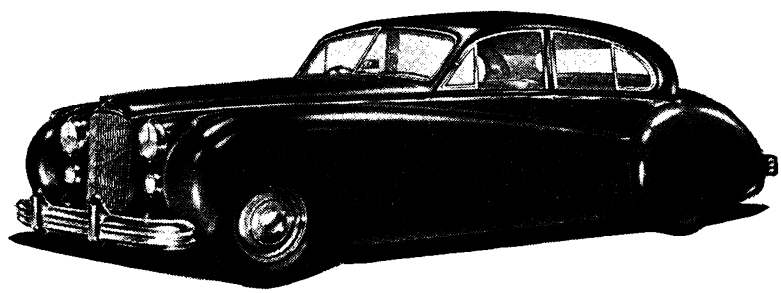

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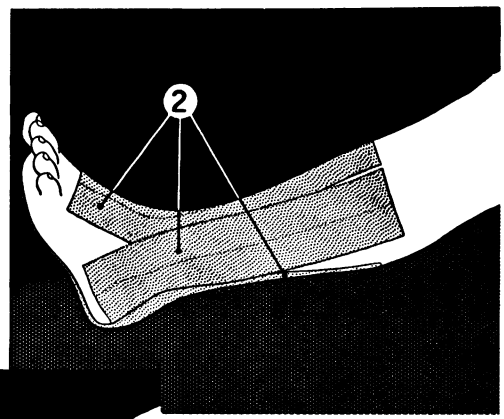

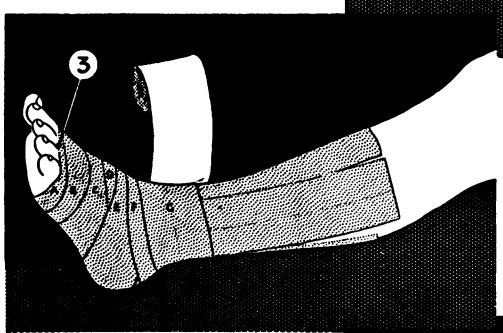

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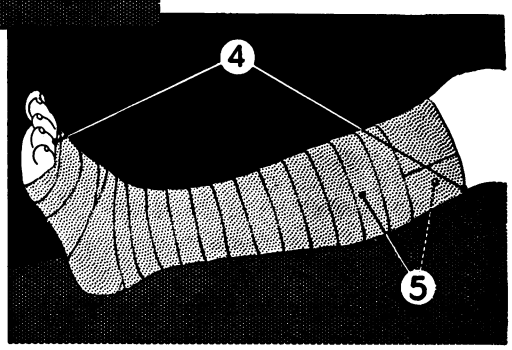

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CONTENTS

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THE
ULSTER MEDICAL JOURNAL

PUBLISHED ON BEHALF OF THE ULSTER MEDICAL SOCIETY

Vol. XXI 1st MAY, 1952 No. 1

Sources, Pathways and Prevention of Infection in Tuberculosis*

By V. D. ALLISON, M.D., D.P.H.
Director of Laboratory Services, Northern Ireland Hospitals Authority

DURING the present century there has been a rapid decline in most of the common infectious diseases in Great Britain. Measles and whooping-cough, almost alone, have not shared in this decline, and have so far resisted efforts at control, while poliomyelitis, for many years prevalent in Scandinavia, U.S.A., and Australia, has come to our shores to offer us a further challenge. For much of the decline in the other infectious diseases, medical research and the public health services can claim credit. Cholera disappeared with the development of a water carriage system for sewage and the provision of piped supplies of drinking water, and the widening extension of these amenities has had much to do with the great decrease in the incidence of outbreaks and cases of typhoid and paratyphoid fevers. On the other hand, dysentery of the Sonne type—fortunately a mild disease as a rule—is now practically endemic in Great Britain. All these, it will be noted, are diseases which have, as their portal of entry to the human body, the digestive system—they must be swallowed in food or drink. When we turn to infectious diseases which enter the human body via the respiratory system, the outstanding successes as regards prevention and control are those resulting from vaccination against smallpox and immunization against diphtheria. Smallpox is imported from abroad, from the Middle or Far East, and increased speed of travel has complicated the prevention of its entry into Great Britain, by allowing occasional cases to arrive while still incubating the disease. The answer to this is a well-vaccinated community. When I was at the Ministry of Health in London it was customary for the medical officers to be revaccinated every time there was a case or outbreak of smallpox involving personal investigation — thus certain medical officers might be revaccinated at intervals of a few weeks or a few months

*Opening lecture at the N.A.P.T. (N.I. Branch) Refresher Course, 1951.

as occasion demanded. If the phenomenal decrease in the incidence of diphtheria during the last ten years is to continue, and if diphtheria is to disappear from our islands, it will only result from a highly immunized population, and the continued immunization of all children during the first year of life. The great decrease in the incidence of the disease means that with fewer cases, there are fewer carriers, and less opportunity for natural immunization, which has accounted for much immunity in the past. Thus fewer contacts with the bacillus of diphtheria would mean higher susceptibility among the non-immunized, and possible disaster, if the proportion of immunized persons was to fall, for the diphtheria bacillus is like the poor—always with us—and has lost none of its sting. The great decrease in the severity and mortality of scarlet fever, which has been continuous since the beginning of the century, cannot be placed to the credit of the medical profession or the public health services. This is a natural phenomenon for which there is at present no explanation. The incidence of the disease remains high, although it has been robbed of most of its former terrors.

In the light of this decline in incidence and severity of the common infectious diseases in this country, it is but natural that more and more attention is being paid to the continued high incidence and mortality from tuberculosis. Our knowledge is still deficient regarding several factors in the ætiology of this disease, and few reliable records are available even for the morbidity rate. We are apt to forget that in England and Wales some four hundred people a week still die of tuberculosis. If an epidemic of one of the acute infectious fevers killed on this scale, it would arouse considerable disquiet, and intensive investigation.

During the past twenty years a great amount of valuable research has been carried out, both in Great Britain and in U.S.A., into the sources and paths of spread of some of the acute infectious diseases, in particular scarlet fever and the enteric fevers—typhoid and paratyphoid B. These investigations were made possible, first, by the comparative ease and speed with which the bacteria causing these diseases can be grown in the laboratory, and second, by the use of methods which enable the bacteria causing one disease to be identified and subdivided into numerous types. For example, the typhoid bacillus comprises more than twenty different types, and the streptococcus which causes scarlet fever can be subdivided into more than forty different types. These fine distinctions enable the investigator frequently to trace the source of an outbreak of scarlet fever or typhoid fever, and to show the paths by which it has been spread from source to victim. Furthermore, these diseases are acute infections with short incubation periods, and occur not infrequently as explosive outbreaks involving many persons over a comparatively short period of time. Similar investigations of the pattern of spread of tuberculosis are beset with difficulties. The tubercle bacillus is very fastidious as regards the laboratory media on which it will grow, and it is at present largely grown on a solid media prepared from fresh hen eggs and special salts. Specimens of sputum submitted to the laboratory for culture of tubercle bacilli are usually teeming with other bacteria. These must first be killed, as they multiply on laboratory media from thirty to forty times as rapidly

as the tubercle bacillus, and would outgrow it and use up all the nutriment. Methods are available for getting rid of unwanted bacteria, but we are again thwarted by the extremely slow rate of multiplication of the tubercle bacillus, which requires upwards of three or four weeks to produce visible growth, as against eighteen hours for average bacteria. The tubercle bacillus infecting man belongs to two types, the human and the bovine, and again special methods must be used to differentiate them. Tuberculosis is a slow, insidious disease, with a long incubation period of many weeks or months, and occurs as epidemics only with extreme rarity. Even in that most susceptible of all animals to tuberculosis, the guinea pig, which can be infected with as few as two tubercle bacilli, it may be from four to six or eight weeks before the infection can be diagnosed, while in the meantime the diagnosis of the infection in the patient must often remain in doubt. This indicates some of the disadvantages which hamper laboratory investigations on tuberculosis.

The portals of entry by which infections gain access to the body are four in number—inhalation, ingestion, implantation and inoculation.

INHALATION infections include those which enter via the air passages or respiratory tract and cause such infections as tonsillitis, scarlet fever, diphtheria, measles, chickenpox, whooping-cough, pneumonia and *pulmonary tuberculosis.*

INGESTION infections are those which are conveyed in food or drink and infect via the intestinal tract, such as infantile gastro-enteritis, dysentery, typhoid and paratyphoid fevers, food-poisoning and *bovine tuberculosis* from infected non-pasteurised milk.

IMPLANTATION—Under this heading are classified infections caused by the placing or dropping of infective germs on susceptible parts of the body, such as infection of wounds or burns, most cases of puerperal sepsis, pemphigus, "sticky eye" and ringworm in infants and children. It is extremely rarely that tuberculosis is conveyed by this route.

INOCULATION infections include those conveyed as the result of improper technique for the sterilization, use and care of syringes, resulting in cases or outbreaks of infection occurring at the inoculation site. These have caused severe and sometimes fatal infections with hæmolytic streptococci and staphylococci by intracutaneous, subcutaneous or intramuscular injections, and severe hepatitis with jaundice transmitted from patient to patient by intravenous inoculation via syringe or needle. Under the same heading may be included the causative agents of diseases such as malaria and yellow fever inoculated in the bite of the mosquito, epidemic typhus fever inoculated in the bite of the louse, and bubonic plague inoculated in the bite of the rat flea. I know of three outbreaks of *tuberculosis* due to transmission of the infection via syringe and needle to the unfortunate victims. One occurred in Germany about twenty years ago, one in Southern Ireland shortly before the second World War, and the third in Scotland during the War.

There are, therefore, at least three routes by which the tubercle bacillus may enter the body—by inhalation, by ingestion and by inoculation—and they lie in that order

of importance, with inhalation far surpassing in frequency the other two routes, and inoculation a very rare mode of entry, but always to be guarded against by those who have to use syringes.

The sources of infection with the tubercle bacillus are two in number—man, and milk from tuberculous cows, causing human and bovine tuberculosis respectively. I was once asked whether tuberculosis can be contracted by eating meat from an infected animal. There does not appear to be a single recorded instance of the disease being transmitted in this way. Tubercle bacilli occur in lungs, liver, kidneys, intestines, glands and udder, and not usually in muscle or fat. Moreover, the tubercle bacillus is very easily killed by heat, and little or no beef is eaten without cooking; even the process of smoking beef or ham is claimed to kill the germ. There is also an additional safeguard in the veterinary inspection and stringent regulations against the sale of meat from diseased animals.

As the human type of tubercle bacillus plays the predominant part in causing infection, let us consider first some of the knowledge which has accumulated regarding the methods of spread. But first there are certain fundamental biological facts, of which we must take cognizance. To survive, any parasite must as a rule find a way of getting out of the first victim it infects, and gaining access to another before the first victim dies or is completely cured. Unlike most bacteria which cause infections of the respiratory tract, such as scarlet fever, diphtheria and pneumonia, and which may regularly be cultivated from the nose or throat, the tubercle bacillus does not settle down in these sites, and when it reaches the lungs it is usually surrounded and sealed off from the outside air by blood and tissue cells. In the great majority of infections the story ends with the death of the organisms leaving only a small but permanent lifelong scar in the lungs. This is proved by the fact that post-mortem findings show that upwards of ninety per cent. of the population have at some time during their lives been infected with the tubercle bacillus. This is evidence not only of the wide dispersal of the tubercle bacillus, but of the general high resistance of the population to infection, due, in part, perhaps to repeated contacts with small doses of the organisms over generations. How different when for the first time the tubercle bacillus reaches primitive peoples, too often alas! via the civilized white man, when it spreads as a pestilence. Robert Louis Stevenson, himself a victim, tells of a tribe of fine physique in the Marquesa Islands, which was reduced by the ravages of tuberculosis in the space of a year from some three hundred souls to a pair of lone survivors.

Even if the tubercle bacillus becomes established in the lungs, it remains cut off from the outside air until the inflammation it has set up breaks down the enclosing wall of tissue and enables it to reach the air passages and be coughed out. In tuberculosis, it is the open lesion or lung ulcer that pours the organisms into the sputum and thus releases them for circulation in the patient's environment, a process that may continue for years and subject his most intimate contacts to heavy risk of infection. If the infection has not progressed too far or too rapidly, treatment may help the damaged tissues again to wall off the areas

of infection; the victim then ceases to disperse tubercle bacilli in his environment, and may be able to resume normal activities without being a danger to his contacts.

What actually happens when a patient with open pulmonary tuberculosis coughs or sneezes? Much of the information available is based on detailed investigation of what happens when patients suffering from acute respiratory infections such as scarlet fever or acute tonsillitis cough or sneeze, because the bacteria causing these infections can readily be cultured, identified and counted in the laboratory. During talking, coughing and sneezing, tiny moist droplets containing bacteria are expelled from the mouth or nose for distances up to six feet, according to the size of the droplets, and the violence of projection. Large droplets have a curved downward trajectory, and settle fairly rapidly in front of the person who coughs, on his personal clothing, bedclothes, floor, in fact on everything within a range of a few feet, infecting persons and objects in his close vicinity. Sneezing, according to the vigour of the sneeze and other factors, expels from 5,000 to 100,000 droplets. The number of droplets expelled in a cough can be counted in hundreds compared with thousands in a sneeze. Spray from a cough leaves the mouth at a rate of about ten miles an hour, and is atomized to droplets which vary in size from 1/140 mm. to 1 mm. or more in diameter. A diameter of 1/10 to 1/5 mm. is generally accepted as the dividing line between the larger droplets and the smaller so-called droplet-nuclei. The droplets, being larger and heavier, settle quickly in the immediate vicinity of the patient. The droplet-nuclei, on the other hand, evaporate very quickly (in about half a second) to a diameter of about 1/200 mm. or less, and being very minute and light they tend to remain suspended in the air like cigarette smoke and get wafted about by air movement — such droplet-nuclei may thus carry infection for a considerable distance.

In cases of scarlet fever or diphtheria or whooping-cough, it is possible to count the number of droplets containing the infecting organism emitted by a cough. This is done by holding a culture plate about three inches in front of the mouth during a cough. Mainly because of the slow growth of tubercle bacilli and of the presence of many other quick-growing organisms in the cough spray from a patient with open tuberculosis, this method is not practicable as a means of estimating the number of tubercle bacilli emitted in a cough spray. However, another method has given results—by placing a thin glass plate, three inches square, in front of the patient's mouth it is possible to collect the droplets from a cough on the surface of the glass. The droplets on the glass can be stained, then counted under the miscroscope, measured, and the number of droplets containing tubercle bacilli with the number per droplet estimated. Using this method, Duguid (1946) found recently that ten out of twenty patients with open tuberculosis expelled droplets containing tubercle bacilli during a cough, and that 36 out of 410 (about 9 per cent.) droplets from 120 coughs contained tubercle bacilli in numbers varying from one to 40,000. Sneezing is most productive of

the dissemination of organisms in acute throat infections, due to the atomization of the streptococcus-containing saliva, but in pulmonary infection the cough may be far more productive of environmental contamination than the sneeze. This is due to the larger size of droplets emitted by a cough, and these settle rapidly in the vicinity of the patient.

If we consider the person at the receiving end of a cough in the vicinity of a patient with open tuberculosis, there are two points of importance. First, there is evidence that a high percentage of the droplets do not contain tubercle bacilli. Second, recent work by Boyland and his colleagues (1947) on nasal filtration of airborne droplets showed that few particles or droplets larger than 1/200 mm. in diameter penetrate the nose of man in normal nasal breathing, and a spray intended to reach the lung should have droplets smaller than this or be administered by mouth breathing. These findings suggest that the risks of infection by inhalation of droplets from a tuberculous patient by someone in the vicinity, while still present, are not so great as appear at first sight. This is because of the rapid settling of the larger infected droplets, because a majority of the droplets are not infected, and because in normal nasal breathing only the smallest droplets can penetrate to the lungs. The moral, therefore, is to breathe through the nose.

There is almost overwhelming evidence that tuberculosis is usually an airborne disease, but there are many who believe that contact, direct or indirect, may also play a part in the transmission of infection. What evidence is there to support this view? Tubercle bacilli were found in saliva from the tip of the tongue in twenty-nine out of fifty cases of pulmonary tuberculosis by Nield and Dunkley (1909). More recently, Duguid (1946) found tubercle bacilli in the saliva from lips and gums at the front of the mouth in ten out of twenty tuberculous patients before coughing. There is no evidence to suggest that the organisms multiply in these sites, and indeed conditions in the nose and mouth are not favourable for the growth of the tubercle bacillus, but the positive findings suggest that a risk of infection from kissing tuberculous patients, though probably remote, cannot be ruled out.

Numerous workers have reported finding tubercle bacilli on the hands of tuberculous patients. Graziani (Rosenau, loc. cit., 1908) found tubercle bacilli on his own hands after shaking hands with tuberculous patients. Similar results have recently been described in U.S.A. (Hamburger, 1946) by the isolation of haemolytic streptococci from the hands of persons suffering from scarlet fever and from the hands of persons with whom they have shaken hands. These results are undoubtedly due to contamination of the hands by blowing the nose and handling the nose and mouth, and are possible factors contributing to the spread of infection. Their significance in this relation has not been assessed, but they should not be over-emphasized. In Germany, Ostermann (1908) recovered tubercle bacilli from the hands of seven out of fourteen phthisical patients and from one attendant, but he obtained them only four times from forty-two children living in tuberculous families.

Spray from the untrapped cough releases infected droplets into the air and several workers have recovered tubercle bacilli from the air of rooms or wards in which phthisical patients were being nursed. Klein (1893) infected guinea-pigs with tuberculosis by exposing them in the vent shaft extracting air from the wards of Brompton Hospital, but more recent attempts to repeat such tests were not successful. Other workers failed to find tubercle bacilli in swabs from the noses of physicians and nurses attending phthisical patients, although they found them in the noses of the patients themselves. Other things being equal, does not this suggest the importance of prolonged or repeated close contacts with cases of open tuberculosis as against the occasional or intermittent contact? It must be admitted, however, that the inhalation of a single large dose of tubercle bacilli on one occasion may act as the spark to start an infection.

It will be recollected that the larger droplets emitted by a cough, and those most likely to be heavily infected, settle rapidly on the personal clothes, on all objects in front of and below the level of the mouth, and on the floor. What is the ultimate fate of these droplets, and what is their significance when infected with tubercle bacilli? It needs no medical knowledge or experiment to know that the droplets of mucus, whether or not they contain pus cells or bacteria, dry fairly rapidly—our own handkerchiefs tell us that. Lange (1926) in Germany found that tuberculosis sputum sprayed on various surfaces, in droplets similar in size to those expelled in coughing, was sufficiently dried in an hour or less to become detached from handkerchiefs, clothing, bedding and solid surfaces as a fine dust which might remain suspended in the air for at least one hour. A large proportion of guinea-pigs exposed to inhalation of such dust developed primary lung lesions. There is, however, general agreement that in dust from dried droplets containing tubercle bacilli, a large proportion of the bacilli die off either in the process of drying or after a short period in the dry state. An experiment by Tytler (1940) indicated that the proportion of tubercle bacilli remaining alive in sputum after two days' drying was probably of the order of one to five per cent. of the numbers originally present in the sputum. Lange's experiments (1926) showed that for at least twenty-four hours after drying, the sputum may be highly infective for guinea-pigs, but the infectivity decreased after one or two days' exposure to diffuse sunlight, and after six days the dry sputum was no longer infective in doses from 100 to 1,000 times as great as that sufficient to infect immediately after drying. In the dark, infectivity was retained longer, but eighteen days after drying one hundred times the original infective dose was required.

Many attempts have been made by numerous investigators to isolate tubercle bacilli from dust under natural conditions. Cornet (1885) in Germany examined 147 samples of dust collected from hospital wards, dispensaries, private houses, streets, etc. He failed to find tubercle bacilli in streets or in places not occupied by tuberculous patients, and even in the environment of phthisical patients the germs were, as a rule, found only in the dust from rooms occupied by careless patients. On the other hand, tubercle bacilli have been found in dust from railway

carriages in Germany and from tramcars in U.S.A. In England, Coates (1901), by inoculation experiments, was able to demonstrate the presence of tubercle bacilli in sixty-six per cent. of specimens of dust from fourteen rooms occupied by tuberculous patients. On the whole, therefore, it appears that virulent tubercle bacilli are quite commonly present in the dust of rooms occupied by tuberculous patients, but especially in the environment of patients who are careless in the disposal of sputum, in coughing, and careless of personal hygiene. The source of the dust is the handkerchief, clothing, bedclothes, all objects in the patient's vicinity, and the floor.

Recently some experiments on the rôle of handkerchiefs in the transfer of respiratory infections have been carried out by the Common Cold Research Unit of the Medical Research Council in the Harvard Hospital, Salisbury (Dumbell, Lovelock and Lowbury, 1948). Sterile cotton handkerchiefs issued to volunteers in the Common Cold Unit were collected after two days' use, and dried in a quiet dust-free atmosphere at 70°F. The handkerchiefs were then shaken in a sealed room, and the number of bacteria-carrying particles obtained from them was estimated. It was found that the average number of bacteria-carrying particles obtained per handkerchief by manual shaking was 14,720, and by mechanical agitation 60,300. The mean count of bacteria-carrying particles from 211 handkerchiefs shaken for thirty seconds was 68,000. Probably ninety-eight per cent. or so of the organisms found in these dust particles were common non-pathogenic bacteria occurring in the nose, mouth, throat, and on the skin, but we must not lose sight of the significance of these findings in relation to the spread of tubercle bacilli from dry sputum-infected handkerchiefs. The authors of this investigation conclude by saying "the use of the handkerchief is probably the most important single action, except bed-making, in the contamination of the air with micro-organisms from the respiratory tract". Brown and I (Allison and Brown, 1937) have on repeated occasions isolated the streptococcus causing tonsillitis and scarlet fever from handkerchiefs of convalescent patients in scarlet fever wards, and also from dust in the pocket in which the handkerchief was kept, as well as from crockery and utensils used by the patients after a meal, and from the surface of the table at which they sat.

Mention of handkerchiefs as possible vehicles for spread of infection leads to the consideration of tuberculous patients' washing. It has already been pointed out that the patient with open tuberculosis infects his garments with the larger droplets when he coughs, and that sputum kept in the dark retains a gradually decreasing infectivity up to eighteen days after drying. While most hospitals and sanatoria make careful arrangements for the sterilization of bedding and linen used by patients with open tuberculosis, few, if any, hospitals undertake the laundering of the patients' personal linen. This is ordinarily taken home by the patients' relatives or friends and returned clean, and includes such articles as pyjamas, nightgowns, bed-jackets and possibly dressing gowns. All these articles are likely to be infected to a greater or lesser degree with dried sputum

containing tubercle bacilli, and yet, generally speaking, no advice or instruction is given to the relatives or friends on how to deal with this possibly infective material, which, by even gentle handling, will disperse particles of dried sputum into the surrounding air. Inquiries from relatives or friends visiting patients in sanatoria showed that while some sent the soiled linen direct to the laundry, more washed it at home. Some kept the patients' linen separate from the rest of the family laundry by washing it on a different day, and some soaked the linen in a disinfectant solution before washing.

It must be frankly admitted that we do not know what rôle infected linen plays in the spread of tuberculosis, but it is generally agreed that the possibility of infection from handling soiled laundry articles cannot be ruled out. I might perhaps quote the printed set of instructions given to patients and relatives in the Brompton Chest Hospital, London.

Recommendations to Tuberculous Patients for Dealing with Personal Clothing.

1. All such articles must be washed separately from those belonging to others.
2. No cooking utensils should be used for washing.
3. Handkerchiefs should be placed in a cotton bag and boiled for twenty minutes.
4. Other linen articles should be boiled for not less than ten minutes.
5. Woollen and other shrinkable articles should be aired, preferably out of doors, for twenty-four hours, and then washed in the ordinary way.
6. Suits and dresses should be aired for forty-eight hours, and then sent to the cleaners.

If these instructions are carried out, they should go far to prevent infected clothing taken home from being a source of danger to other members of the household, and the issue of advice such as this is worthy of extension. Unfortunately, in this climate airing out of doors may not be practicable for several months of the year, and airing indoors is fraught with risk, especially if the garments are hung over a line in a kitchen used mainly as a living room. If I were asked to comment on these instructions, I would add a further one :—

"No articles should be sent to the laundry until after they have been immersed in a disinfectant solution"—

The nature and concentration of the disinfectant to be used should be specified. It is not fair to subject laundry workers to such potential risk of infection. The only clue to the possible risk to laundresses handling articles infected with tubercle bacilli is given in figures published by the Registrar-General (1931). He showed that in male laundry workers the standard mortality rates from tuberculosis is low (70), and that in women laundresses over 34 years of age it is not significantly high (107), but in laundresses below 34 years of age the figure reaches 151. The clue is a slender one, but should not be disregarded. An alternative to present procedure, and one to be recommended if at all practicable, is the sterilization of the patients' personal laundry in the hospital before it is either handed to the relatives or sent to the hospital laundry.

Another problem which crops up from time to time, and which was put to me in 1944 by an After Care Committee on Tuberculosis in England, is the possibility of transmission of tuberculosis through books. Many workers have investigated the possibility of the transmission of some of the acute infective fevers via books, but I have only been able to find five reports on similar investigations into infection of books with tubercle bacilli, and in two of these carried out in Germany in 1903 and 1907 the conclusions do not carry conviction. Some more recent investigations, however, by Kenwood and Dove (1915), by Roodhouse Gloyne (Tytler et al., *loc. cit.* 1940), and by Smith in U.S.A. (1942), have enabled us to draw more definite conclusions, which may be summed up as follows—a book read by a phthisical patient may become contaminated by droplets from sputum, expelled during coughing or speaking. As the book is usually held within eighteen inches of the face and below the level of the mouth, the larger droplets, which fall rapidly, may readily contaminate it. It may also become infected by the practice of moistening a thumb or finger in the mouth in order to turn a page—a practice which should be as extinct as the dodo. [The *Lancet* in 1943 said this practice should be as obsolete as shuttle-kissing, but I learnt only recently, to my surprise, that in Northern Ireland at least, shuttle-kissing is still practised.] Kenwood and Dove gave small squares of paper to patients with open tuberculosis, to hold before the mouth while coughing, over a twenty-four hour period. The papers were then dried for forty-eight hours, washed, and the washings injected into guinea-pigs. Papers from eight out of fifteen patients produced tuberculosis. Similar squares of paper were given to six other patients, to be smeared twelve times with the thumb moistened with saliva. After two days' drying, washings from four out of the twelve papers infected guinea-pigs with tuberculosis. Roodhouse Gloyne pulped in sterile water a book which had been extensively used in tuberculosis wards, and the sediment from the washings infected one of two guinea-pigs with tuberculosis. Smith found that tubercle bacilli could be recovered alive from books and magazines for periods up to three and a half months after deposition. The organisms lived as long on paper as on glass and as long on printed as on blank paper. The period of ability to recover the organisms from books or magazines was dependent on the size of the dose initially deposited. Despite these data, we really do not know whether contaminated books or magazines can transmit tuberculosis. Lacking such proof, they must be regarded as possible vehicles of infection, although they are probably insignificant as compared with other agents already mentioned. It is difficult to carry out active sterilization without damage to books, and the use of ultra-violet light as a bactericide would involve exposure of every page of the book. There appears to be a general consensus of opinion that books contaminated or suspected to be contaminated with tubercle bacilli can be rendered safe and non-infectious by a quarantine period of a month. On the theory that a much used ragged book is likely to be an infected book, cheap books and soiled books should be destroyed. Books exposed to possible infection with tuberculosis should not be sold to second-hand dealers.

The last problem of this kind I wish to mention, is one about the potentialities of which we know little or nothing. It is the part played in the transmission of tuberculosis by articles manufactured by patients engaged in occupational therapy, especially articles made of leather, such as bags, purses, note-cases, and of wool or other fabric, such as bed-jackets, scarves, rugs, cushion covers, tapestry. I am not aware of any investigations having been carried out to determine whether such articles have ever been found contaminated with tubercle bacilli, but it is the practice in many sanatoria to expose all such manufactured articles to formalin-vapour before they are sold or given away. A working party set up by the Medical Research Council is at present investigating the value of exposure to formalin vapour for the disinfection of articles made by tuberculous patients in occupational therapy centres, and its report and recommendations will be awaited with interest. Formalin vapour is a very efficient disinfectant, but if one can argue from the formalin vapour disinfection of blankets and mattresses, simple diffusion of the vapour will not be sufficient, especially for articles of wool or other fabric. A preliminary vacuum in a closed chamber containing the articles will be necessary, so that when the formalin vapour is admitted it will penetrate and completely permeate the articles, leaving no enmeshed pockets of air to insulate areas of the articles from direct contact with the vapour.

Our most recent knowledge on non-pulmonary tuberculosis is based on the Report (1949) of a group investigation in England and Wales, planned by the Medical Research Council and carried out during 1943-45. The findings were based on the isolation and identification of tubercle bacilli from a total of 1,106 patients. The organisms were isolated from patients suffering from meningitis, infection of cervical glands, bones and joints, abdominal tuberculosis, genito-urinary tuberculosis, and other miscellaneous non-pulmonary sites. The figures for England showed that the bovine type of bacillus was the cause of approximately 28 per cent. of cases of tuberculous meningitis, 57 per cent. of cases of tuberculosis of cervical glands, 10 per cent. of bone and joint tuberculosis, 64 per cent. of abdominal tuberculosis and 20 per cent. of genito-urinary tuberculosis. In all groups the proportion of infections with bovine bacilli was higher in children than in adults, but in contrast with earlier findings, the highest proportion of infections of bovine type was in the 5-9 age group, instead of the 0-4 age group. This change is attributed to the gradual replacement of raw milk by heat-treated milk for infant feeding during the past twenty-five years or so—this has lessened the risk of infection with bovine type organisms during the first year or two of life. On the other hand, owing to the milk-in-schools scheme, school children are at greater relative risk of infection with bovine-type organisms, as many children now receive milk who would previously have had little or none on reaching school age. Most of the larger authorities provide a pasteurized or T.T. supply of milk to their schools, but it is true to say that there is still an appreciable proportion of schools (probably about 10 per cent.) in which raw milk is consumed under the scheme, especially in small towns and rural districts. On the basis of the findings, it is estimated that in England and Wales about 24 per

cent. of all cases of non-pulmonary tuberculosis were caused by infection with the bovine type of tubercle bacillus, and that in 1944 between 1,300 and 1,400 persons died from tuberculosis of bovine origin. Dr. Violet Reilly has recently (1950) pointed out that the incidence of bovine tuberculosis in humans in Northern Ireland has decreased very considerably during the last ten years. This is due largely to the high percentage of milk pasteurised, 98 per cent. in Belfast and over 90 per cent. in Northern Ireland, and also to the increase in the number of attested herds. I am informed that there is no bovine tuberculosis in Belfast, but during the last two years I have been surprised at the large number of cases of undulant fever in Northern Ireland which occur especially in rural areas, more in relation to the size of the population than occur in England and Wales—this is evidence that there is still a considerable amount of raw milk being drunk, especially in rural areas, as many people, especially children, may drink milk infected with *Br. abortus* for long periods without getting undulant fever.

I have tried to convey to you the main pathways by which the tubercle bacillus may be transmitted from one person to another, and some of the evidence available in support of the modes of infection. But, taking the broad view, one fact stands out broad and clear — as Geddes Smith of the Commonwealth Fund in U.S.A. expressed it in 1941 — "Tuberculosis spreads most dangerously in the close contacts of the family. Resistance breaks down before a mass attack, and the steady bombardment of each other by husbands and wives, parents and children, brothers and sisters, is the best of all mechanisms for perpetuating the disease. Taking all ages together, people exposed to tuberculosis in their own homes are roughly ten times as likely to fall sick of it as are people in the population at large."

In order to fit what has been said into the general concept of the causes of tuberculosis, let me quote from the Terry Lectures in U.S.A. for 1940, given by Professor Sigerist of John Hopkins University, taking as his subject "Medicine and Human Welfare" — "Bacteriology revealed external biological causes of diseases, and blinded by the great success of the new science, physicians were often inclined to overlook the social, economic and individual factors that are just as decisive in the genesis of disease. There is no tuberculosis without tubercle bacilli, but while most people are exposed to infection, very few actually develop the disease. A low standard of living can be as much responsible for the disease as the bacilli."

As regards the prevention of spread and the control of tuberculosis, it is unnecessary to detail the methods in current use for preventing the spread of infection by tubercle bacilli — the shielding of the cough with a handkerchief (the trite but true expression—"coughs and sneezes spread diseases" will not be forgotten), the use of paper handkerchiefs and methods for their disposal, the use of separate crockery, the methods of dealing with laundry and bed linen, the exclusion of children from the sick room, the value of soap and warm water not only for the hands but also for cleansing the bedroom of a phthisical patient,

the practice of dusting by damp dusting or by vacuum, *never* by dry dusting, and the value of sunlight, fresh air and ventilation. But, as that great American epidemiologist, Wade Hampton Frost, said in 1937: "While organization and weapons have been improved, the essential strategy of the attack against tuberculosis remains the same as it was fifty years ago. The main objective is still avoidance of exposure, and the strategy is that of a frontal assault on discoverable sources of infection. From the standpoint of prevention, curative treatment and measures designed to increase resistance to infection may be regarded as supplementary." Time alone will tell whether the widening use of B.C.G. vaccine will cause these views to be changed.

During the past fifty years, many diseases, mainly intestinal, have shown a great decrease both in incidence and in mortality. This has in considerable measure been due to the State which, through its Sanitary Authorities, has provided and continues to extend the provision of pure potable water supplies and the water carriage system for disposal of sewage. What the State can not do is to provide a pure supply of the air we breathe in our daily walks through life. That must in large measure depend on the individual, and in this respect each of us has a personal responsibility. Many of the infectious diseases to which the flesh is heir, still prevalent to-day and even on the increase, such as food poisoning and dysentery, are caused through human ignorance and carelessness. The essential root cause is lack or disregard of personal hygiene, which is responsible for a great deal of infectious disease, both intestinal and respiratory, including pulmonary tuberculosis. Suffice it to say, it is comparatively easy so to live as to prevent our own secretions from being inhaled or ingested by others. Personal cleanliness is less expensive than municipal cleanliness, and can be equally effective in preventing the spread of disease, and is within the reach of all. But to bring it into universal practice, there must be knowledge, and knowledge of this kind can be gained only by education. And it is on this note — the importance of education in personal hygiene — that I conclude — education in school, education in the home, education at parents' association meetings, in hospitals, and in large and small catering establishments. There is no doubt that the school is or should be one of the most important health institutions, helping the child to acquire early a natural habit of personal hygiene. To inculcate these tenets, the teachers must learn, appreciate and themselves practise health habits. In the home, this applies to the doctor and health visitors, in the hospital to the nursing and medical staff, and in the University and training school to the teaching and tutorial staff. Only by relentless efforts, by co-operation and by mutual understanding will we eventually achieve success in the fight against infectious disease, and tuberculosis is one of the "captains of the men of death" against which efforts must continue to be concentrated.

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REVIEW

BACTERIAL AND VIRUS DISEASES. Antisera, Toxoids, Vaccines and Tuberculins in Prophylaxis and Treatment. By H. J. Parish, M.D., F.R.C.P.E., D.Ph. 2nd ed. (Pp. viii + 204 illustrated. 10s. 6d.). Edinburgh: E. & S. Livingstone, 1951.

THIS book, intended as a working manual, is written from a very practical point of view and presents the practice of immunology in medical practice in a clear and lucid manner. It is sound in its principles, accurate in its facts, convenient in its size and eminently readable. It should be read by medical students, general practitioners, public health and laboratory workers rather than displayed on the library shelves of medical schools and public health departments.

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The Age Distribution of Tuberculosis Mortality in Northern Ireland

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INTRODUCTION.

IN a recent review of world tuberculosis mortality, McDougall (1950) demonstrated that mortality has declined in most of the countries which publish reasonably reliable statistics. Of twenty-nine countries considered, only in Portugal and Scotland were the crude death rates less in 1937-39 than in 1947-49. The average annual rates quoted for Northern Ireland, 91.2 and 66.7 per 100,000 of the population respectively, decreased during the decade by 27 per cent. — a relative trend more favourable than that of England and Wales (21 per cent. decrease), Eire (8 per cent. decrease) or Scotland (4 per cent. increase). The level of the crude death rate in Northern Ireland was exceeded in the British Isles only by that of Eire (115.2 per 100,000) in 1937-39, but in 1947-49 not only Eire (105.5 per 100,000) but also Scotland (74.1 per 100,000) had higher rates. However, these crude figures take no account of the age and sex constitutions of the populations concerned, and are consequently liable to be misleading as comparative measures of the efforts to combat the disease, and it should be noted that in most countries a small rise in the general mortality level occurred during the war years.

NORTHERN IRELAND MORTALITY.

In Table I the average annual death rates at specific ages are given for Northern Ireland for the three periods 1925-27, 1936-38 and 1947-49. Each sex is

TABLE I

Average annual number of deaths from all forms of tuberculosis per 100,000 of the population in specified age and sex groups—Northern Ireland, 1925-27, 1936-38 and 1947-49.

CALENDAR PERIOD	AGE GROUP IN YEARS.									
	0-4	5-14	15-24	25-34	35-44	45-54	55-64	65-74	75+	Total
	Male death rate per 100,000 of population.									
1925-27	135	44	187	214	189	179	133	69	25	142
1936-38	64	27	118	147	142	149	111	63	12	99
1947-49	54	15	67	81	98	128	124	87	40	73
	Female death rate per 100,000 of population.									
1925-27	118	70	263	266	183	140	108	41	19	160
1936-38	63	33	152	162	113	85	69	53	30	96
1947-49	39	20	92	93	72	60	49	52	18	60

shown separately and the rates are based on the registrations of all tuberculosis deaths in the three triennia, the age and sex distributions of the census populations of 1926 and 1937, and an estimated age and sex distribution of the population for 1948. The 1948 population data were provided by the Registrar-General for Northern Ireland (1950) for another purpose (Adams and Cheeseman, in press); the estimate of the age distribution is approximate and therefore the tuberculosis death rates for 1947-49 are less reliable than those of 1925-27 and 1936-38. It is unlikely that the approximations involved will effect the broad conclusions of this report, but they prevent the age specific rates in 1947-49 being used where precise levels of age specific mortality are required.

Table I can be read in two ways. Firstly, the rates can be considered along the rows of the table; for any one row the deaths all occurred in the same calendar period but among persons of different age. Thus for each calendar period the age group with the maximum mortality can be identified and the general form of the age mortality curve can be observed. To facilitate this type of examination table II and figure 1 have been prepared and are self explanatory. Secondly, the rates of table I can be read down the columns of the table; for any one column, all the deaths occur among persons of the same age but in different calendar periods. Thus the secular trend for each age group can be examined. To facilitate this aspect of the study table III and figure 2 are presented.

Had mortality rates been available for these age groups for a long continuous series of ten-year calendar periods, a third method of examination would have been possible—Greenwood (1936) gives the most lucid explanation of this procedure, sometimes described as a generation or cohort study. For this third purpose the rates would have been read diagonally from top left to bottom right and each diagonal series would have represented the tuberculosis mortality

TABLE II.

Death rates from all forms of tuberculosis for each calendar period in each age group as a percentage of the rate in all ages combined—Northern Ireland 1925-27, 1936-38 and 1947-49.

Calendar Period	Age Group in Years.									
	0-4	5-14	15-24	25-34	35-44	45-54	55-64	65-74	75+	Total
	Male death rates as percentages of rate at all ages.									
1925-27 -	95	31	132	151	133	126	94	69	18	100
1936-38 -	65	27	119	148	143	151	112	64	12	100
1947-49 -	74	21	92	111	134	175	170	119	55	100
	Female death rates as percentages of rate at all ages.									
1925-27 -	74	44	164	166	114	88	68	26	12	100
1936-38 -	66	34	158	169	118	89	72	55	31	100
1947-49 -	65	33	153	155	120	100	82	87	30	100

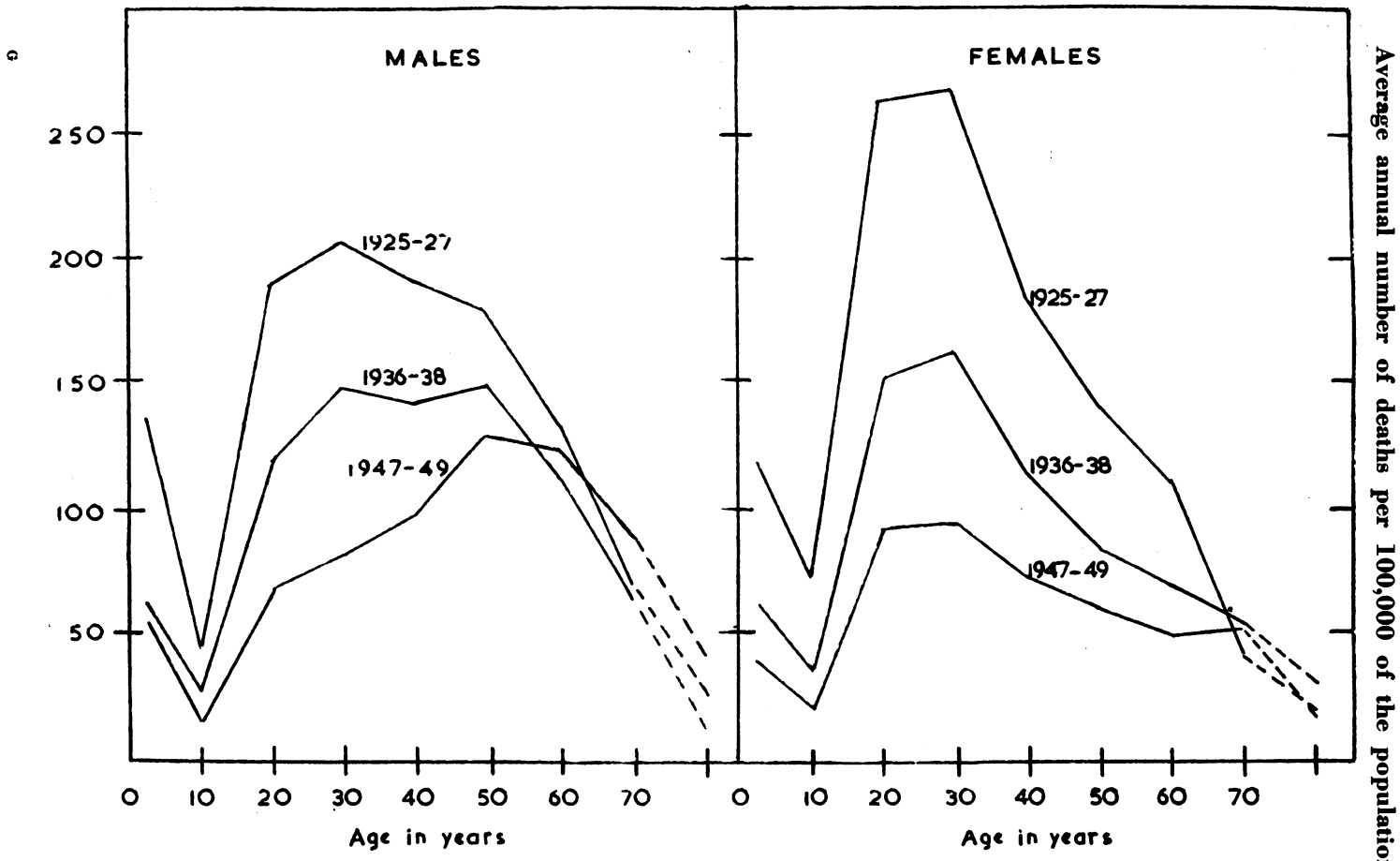

Fig.1. Mortality from all forms of tuberculosis by age and sex in Northern Ireland 1925-27, 1936-38 and 1947-49

rates experienced at different ages by a group of individuals all born within the same ten-year calendar period. Thus the subsequent mortality of persons born in a particular decade could have been traced throughout their life span. In fact, the limitation of the available data precludes this third operation in the present series, although, as will be seen, some further light can be thrown on the interpretation of the data if the principle is borne in mind.

Examination of the rows of table I shows that maximum male mortality in 1925-27 occurred in the age group 25-34 years; figure I and table II show that the mortality curve has a rounded effect being slightly skew towards the younger ages. In 1936-38, the highest male death rate actually occurred in the age group 45-54, but there was very little difference in the level of male mortality from ages 25 to 54 years. The result is a reasonably symmetrical curve with a plateau extending from ages 25 to 54. In 1947-49, the age group of maximum mortality was 45-54 and the rate at 55-64 was not much lower—the curve shows a gradual rise to the maximum between 45 and 64 and then falls away sharply. As table II shows, the rise to the maximum has tended to become relatively steeper, in terms of rates at young ages, as time has passed.

The age group of maximum female mortality has remained constant; in each of the three calendar periods it was 25-34, and in each the rate in the preceeding age group 15-24 was not much less than the maximum, which suggests that if the data were continuous for age the true maximum might be found about the age of 26 years. The relative shapes of the three curves are also very similar —a sharp rise to the maximum and a gradual decline thereafter. If more reliance could be placed on the rates of the last period one might be tempted, from table II, to infer a more gradual relative decline after the maximum than in other calendar periods, but one cannot be definite on this point.

TABLE III.

Death rates from all forms of tuberculosis for each age group (a) in 1936-38 and 1947-49 as a percentage of the rate in 1925-27, and (b) in 1947-49 as a percentage of the rate in 1936-38—Northern Ireland.

Calendar Period	Age Group in Years									
	0-4	5-14	15-24	25-34	35-44	45-54	55-64	65-74	75+	Total
	(a) Male death rates as percentages of 1925-27 rates.									
1936-38 -	47	61	63	69	75	83	83	91	48	70
1947-49 -	40	34	36	38	52	72	93	126	160	51
	(b) Male death rates as percentages of 1936-38 rates.									
1947-49 -	84	56	57	55	69	86	112	138	333	74
	(a) Female death rates as percentages of 1925-27 rates.									
1936-38 -	53	47	58	61	62	61	64	129	158	60
1947-49 -	33	29	35	35	39	43	45	127	95	38
	(b) Female death rates as percentages of 1936-38 rates.									
1947-49 -	62	61	61	57	64	71	71	98	60	63

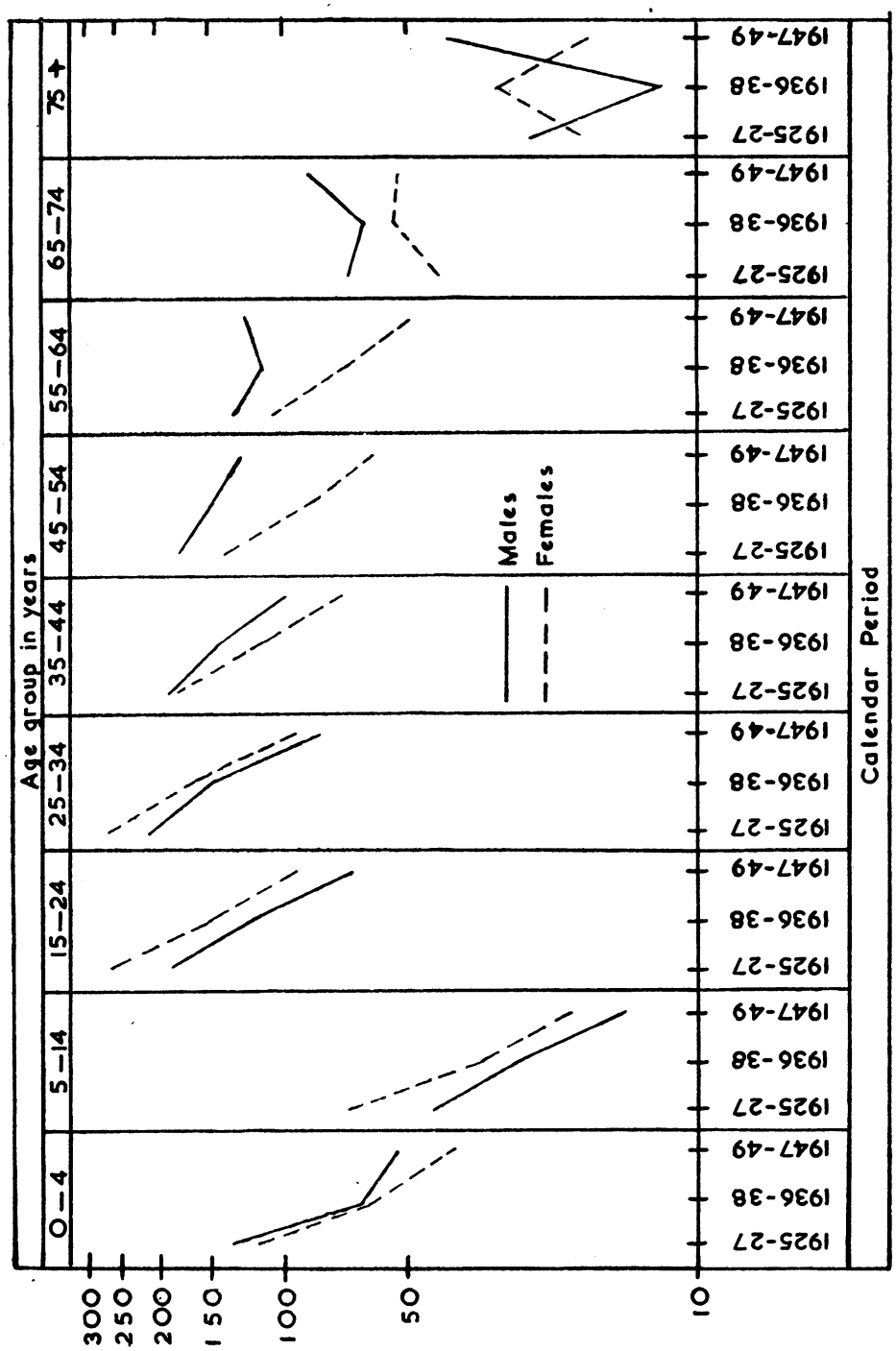

Fig.2. Mortality from all forms of tuberculosis by age and sex in Northern Ireland 1925-27, 1936-38, and 1947-49

Examining the columns of table I, reveals that below age 55 for men and 65 for women mortality was lower in 1947-49 than in 1936-38 and lower in 1936-38 than in 1925-27—the general trend below these ages has been favourable over the whole period reviewed. Above age 55 for men mortality was lower at all age groups in 1936-38 than in 1925-27 but higher in 1947-49 than in 1936-38. In fact, at ages over 65 the rates in 1947-49 were higher than in 1925-27. Above age 65 for women mortality was higher in the middle calendar period than in the first and lower in the third than the second (although at ages 65-74 this last difference was negligible).

Reference to table III shows that relatively there are some inconsistencies in the rate of change of the mortality rates between different age groups and between the first and second half of the secular period reviewed. The general picture can be more easily appreciated from figure 2 where the mortality rates are plotted on a logarithmic scale, and the rates of change of the death rates can be compared by using the slopes of the lines.

After the age of 5 and where the rates declined over the whole calendar period, the male rates decreased slightly more rapidly in the second half of the calendar period than the first; there was very little difference in this aspect of the female mortality between the two secular phases, although a slight tendency can be seen towards a rather more favourable trend after 1935-39 — a tendency in converse to the male experience.

The general trend over the twenty-two years shows that the rate of reduction of male mortality is inversely associated with age—the lines become less steep as age advances—up to age 55 when the transition to an increasing rate begins. On the contrary, the rate of change of the female rates is remarkably constant while these rates are falling—the lines are roughly parallel—up to the early fifties when a slight tendency for a less favourable trend appears before the transition to an increasing mortality level after age 65.

It is of interest to note in passing that in each of the three periods the female rates exceed the male rates between the ages of 5 and 34; the reverse is true between the ages of 35 and 74. The sex ratio of the mortality (male/female) between the ages of 5 and 74 showed a general increasing tendency as age and secular time increased.

Mortality in Other Countries.

Springett (1950) has focussed attention on the apparent shift in the age of maximum mortality due to tuberculosis which has occurred in various countries. He calculated death rates in specific age groups for both men and women for a sequence of calendar periods and for a number of countries or towns and carried his analysis as far back into the past as the available statistics would permit. After comparing the age mortality curves between successive calendar periods he summarised his findings as follows :—

"Males of England and Wales and of Massachusetts show the most marked shift of the peak rate, from young adult life to an age about the end of working

life. In both countries the shift has been a slowly progressive one over a long period of time, and it is by no means certain that the limit has yet been reached. Males of Paris show only a slightly less marked shift of the peak, partly because the peak rate was already at age 35-44 years at the time when rates were first available; it is possible that if rates were available for a still earlier period a peak in even younger life would have been shown. In males of Danish towns the peak occurred late in life in 1890-99, the earliest period for which rates were available. More recently the peak has passed back to a younger age period. It it suggested that the fall began some years before 1890, in a similar way to that in England and Wales, and that the 1890-99 rates show the maximum shift of the peak. Males of Scotland have shown a shift of the peak more recently than males of England and Wales, and so far not to the same degree, but it is possible that the movement is not yet complete. Females of England and Wales and of Paris show a small shift of the peak rate to an older age period, followed by a greater movement back to an even younger age period than originally. Females of Denmark show the shift back to a younger age period from a peak at 35-44 years. In the same way as for males, it is suggested that this comparatively late peak of Danish females is the result of a change starting before 1890."

He goes on to instance the communities of which the available statistics failed to reveal any change in the age of maximum mortality. They were women of Scotland and Massachusetts and both sexes of Ireland, Norway and Sweden.

About the same time, Daniels (1949) and Daw (1950) also drew attention to the phenomenon in England and Wales and, before the war, Frost (1939) had commented on the change in the age mortality curve of males in Massachusetts in contrast to that of females, while Picken (1940) had shown similar results for England and Wales. Lancaster (1950) reported a change in the age of maximum male mortality in Australia from about 55 years in 1911-20 to about 70 years in 1941-45, while the female peak remained at age 20-30. In the case of the latter there was some evidence by 1941-45 of a secondary peak of mortality later in life, 60-70 years, in which group the death rate was nearly as great as in group 20-30.

Discussion.

It thus appears that, in so far as age is concerned, mass aspects of tuberculosis mortality are characterised by a tendency for the maximum age of mortality to move to higher ages; for the peak age for women to be less than that for men; and possibly for the maximum mortality after a shift to the higher ages to move back to young ages again. Over any given secular period these phenomena are not apparent in all communities—where the shift occurs in the male experience of one country it does not necessarily appear in the female. Further, the shift at any one moment of time appears to have reached different stages in different countries. This last point would be compatible with the hypothesis that tuberculosis is an endemic disease with epidemic waves (Heaf and Rusby, 1948) of different chronological origin in different countries.

Where the age of maximum mortality has moved from young adult to late middle life, as among the men of England and Wales, Massachusetts, Paris, Denmark, Scotland and Australia, there is evidence from the work of Frost, Daw, Picken, Lancaster and Springett that the high rates in late life are, in Frost's words, ". . . the residuals of higher rates in earlier life." Thus when the age specific death rates are examined in generations or cohorts and not in calendar periods, it is shown that the group of individuals who now have a high death rate belong to a generation, born some fifty years ago, who had a still higher rate in their twenties. Thus a consideration of the age mortality curves for generations reveals a consistent peak mortality in young adult life for successive generations—a peak which in most countries has become smaller as time has passed.

It has been shown that the Northern Ireland age mortality curves from tuberculosis have exhibited, in recent years, the features which have been characteristic of some other countries in the past. Most of the studies made elsewhere referred to events before 1941, although it is known that the shift of the male peak to higher ages has continued in England and Wales (Registrar-General for England and Wales, 1949), and the differential male and female experience at late ages in mortality is also observed in the limited morbidity data available in that country (Stocks, 1950).

Thus it would appear that the male shift of maximum mortality started in Northern Ireland rather later than in England and Wales, Massachusetts, Paris, Denmark, Scotland and Australia, and this fact might be used in support of Clarke's (1950) contention that the "tuberculosis epidemic" started later in Northern Ireland than in some other communities.

As the present data refer only to a limited period of some twenty years, it is not possible to trace the rates experienced by generations such as has been done in the cohort studies already mentioned. More detailed examination of the rates in table I, however, does show that the limited study that can be made along these lines gives results which are not at variance with the suggestion that in generations the maximum tuberculosis mortality is constantly experienced in the young adult period of life.

If the rates in ten-year age periods in table I are read diagonally downwards from left to right, then successive rates are roughly applicable to the same generation. For example, the male rates of 189 per 100,000 at age 35-44 in 1925-27, 149 per 100,000 at age 45-54 in 1936-38, and 124 per 100,000 at age 55-64 in 1947-49 refer to men born in 1881-92, 1882-93 and 1883-94 respectively. If it is assumed that the mortality experienced during the lives of men born in these overlapping periods is similar, then the three rates quoted give the tuberculosis mortality experienced by the generation of men born in approximately 1881-94 at the ages stated. Similar arguments apply to other diagonals, the generations becoming older as the table is read from left to right. The generation rates are thus available for a different thirty-year segment of each of six generations. The rates observed in these segments all show decreasing mortality

with increasing age except where the age groups in the range 15-34 are included. This is true of both sexes, and while it is not a proof that in successive generations maximum mortality always occurred in the young adult ages, it is at least compatible with such a hypothesis.

With the data for 1947-49 included there can be no doubt that the male shift in mortality has occurred; but it might be considered surprising that the tendency was not observed from pre-war rates alone—the change from the early peak in 1925-27 to the plateau of 1936-38 is highly suggestive. Springett's analysis for Ireland as a whole led him to conclude that no shift had occurred in the peak age in either sex and that the shape of the age mortality curves had not materially changed from 1871 to 1941. He did note ". . . a small increase in the rates for older age groups of males relative to those at age 25-34 years." If the Northern Ireland change in the shape of the curve for men was initiated during the late thirties and if no such change has begun in the males for the rest of Ireland, then this last observation of Springett's might well represent the sum total of the effect of the contribution of Northern Ireland to the mortality curve of all Irish males — the mortality rates of Eire, based on larger populations, would carry much greater weight in the combined rates than those of the North.

It is of interest to note that one of Springett's conclusions was that the shift of the age of maximum mortality tended to occur in both sexes in densely populated countries, not at all in sparsely populated countries, and in males alone when the density of population fell between the two extremes. The density of population which he quotes for Ireland (130 persons per square mile) is less than that of Massachusetts (514), Scotland (308), the Urban Districts of Paris, towns of Denmark and England and Wales, of which the combined density was 742 persons per square mile. It will be recalled that the shift in the peak of mortality did not occur in his Irish data; occurred in males only in Massachusetts and Scotland; and in both sexes in the other areas. The latest available density figures (Registrar-General for Northern Ireland, 1951) are 262 persons per square mile for Northern Ireland, 112 for Eire (the weighted average for the whole of Ireland being 137), 175 for Scotland, and 755 persons per square mile for England and Wales. The discrepancy of the Scottish figures is rather surprising, but whichever is taken as correct, the density for Northern Ireland is relatively more similar to Scotland than to the rest of Ireland. Consequently, the observation of the age shift in only male maximum mortality in Northern Ireland, an experience similar to that in Scotland, but not to that of the rest of Ireland, gives further support to Springett's thesis of an association between the occurrence of the age mortality phenomenon and density of population.

SUMMARY.

1. In common with most other countries, tuberculosis mortality in Northern Ireland has declined over the last twenty years, apart from a small increase in the war years.

2. Mortality has not fallen, however, at all ages and, moreover, where reduction in the rates has occurred, it has not been at a uniform rate at all ages.
3. Between 1925-27 and 1947-49 the peak age of male mortality has shifted from the young adult age groups to late middle life (roughly from about age 26 to age 55). Female mortality has in the same period maintained its maximum at about age 26.
4. It is suggested that the shift of maximum male mortality is the result of the so-called "generation effect" already reported from some other countries. Thus in a given age group and calendar period the mortality rates are dependent in part on the previous experience at earlier ages of the individuals concerned. The men now experiencing high rates of tuberculosis mortality in their fifties are the survivors of a generation which probably experienced still higher rates in young adult life. (The available data do not permit a precise conclusion on this point, but they are compatible with such a theory, which seems highly probable in the light of more precise work done elsewhere).
5. It is shown that the distribution of the age shift in males and not in females in Northern Ireland is to be expected, if density of population and experience of the shift are associated in the manner suggested elsewhere.

ACKNOWLEDGMENTS.

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Some Results of Tuberculin Testing of School Children in Northern Ireland

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As no figures were available concerning the tuberculin sensitivity in the general child population in Northern Ireland, it was decided to test as many as possible of the children in elementary schools in Belfast at the time of their first routine school medical examination during 1950. It had been intended to test the children during their first year of attendance at school, but it was much more convenient to obtain consent of parents in conjunction with arrangements for the first medical inspection.

Subsequently, when the Medical Research Council was asked for funds to assist in the enquiry, it was learned that a survey was taking place in England, in which school children in each age group up to 16 were being tested. Accordingly, the testing survey was extended to give comparable findings for children in Northern Ireland, by testing other children in Belfast and in country districts.

The findings are set out below. Part I refers to the young children in Belfast and Part II to the children in each age group in Belfast and in less urban districts in Northern Ireland.

Procedure.

In each instance the consent in writing of the parents was given before the child was tested.

Each parent was sent an explanatory form, and if consent was not given or no reply was received, a health visitor visited the home and tried to persuade the mother to agree; in this way the total proportion of consents was raised by about 10 per cent.

Technique.

The test method was that used by the Medical Research Council in a survey of tuberculin reactions in children in England. Each child was first given an inter-scapular patch test, using 60 per cent. old tuberculin jelly (A. and H.). An area just to one side of the spine was cleaned with acetone on cotton wool. It was then stroked lightly six times with a 1″ wide strip of grade 00 sand paper.

About ½″ of the jelly from the tube was then applied and the area covered with a small strip of adhesive tape and the test was read ninety-six hours later.

In the first 500 children a control test with A. and H. control jelly was given at the same time, but it was not used thereafter, as pseudo reactions did not occur.

This test was taken as positive if four or more vesicles or a single large vesicle occurred on an area of erythema.

All children whose patch test was doubtful or negative were given an intra-dermal test with 1/100 (1.0 mgms.) old tuberculin. A positive reaction was

*Assisted by a grant from the Medical Research Council.

TABLE I

Numbers of Young Belfast Children whose Parents were asked to Permit Tests.
Numbers of refusals, Uncompleted and Completed Tests.

Age	5		6		7		Total	
BOYS	No.	%	No.	%	No.	%	No.	%
Total in Survey	1366	100	2439	100	684	100	4489	100
No. of Refusals	181	13.25	377	15.46	135	19.74	693	15.44
No. Absent (Consenters)	177	12.96	317	13.00	107	15.64	601	13.39
Incompleted Tests	64	4.69	135	5.54	41	5.99	240	5.35
Total Tested	944	69.11	1610	66.01	401	58.63	2955	65.83
GIRLS								
Total in Survey	1395	100	2412	100	675	100	4482	100
No. of Refusals	194	13.91	387	16.04	142	21.04	723	16.13
No. Absent (Consenters)	202	14.48	324	13.43	99	14.67	625	13.94
Incompleted Tests	79	5.66	114	4.73	34	5.04	227	5.06
Total Tested	920	65.95	1587	65.80	400	59.25	2907	64.86

TABLE II

Results of Tuberculin Jelly and $\frac{1}{100}$ Introdermal Mantoux Tests on Young Belfast Children.

Age	5		6		7		Total	
BOYS	No.	%	No.	%	No.	%	No.	%
Total Tested	944	100	1610	100	401	100	2955	100
Positive to Jelly	212	22.46	345	21.43	94	23.44	651	22.03
Positive to $\frac{1}{100}$	35	3.71	71	4.41	10	2.49	116	3.93
Total Positive	247	26.17	416	25.84	104	25.93	767	25.96
GIRLS								
Total Tested	920	100	1587	100	400	100	2907	100
Positive to Jelly	212	23.04	315	19.85	92	23.00	619	21.29
Positive to $\frac{1}{100}$	33	3.59	42	2.65	9	2.25	84	2.89
Total Positive	245	26.63	357	22.51	101	25.25	703	24.18

TABLE III

Results of jelly and $\frac{1}{100}$ intradermal tuberculin tests in

Age	5		6		7		8		9	
BOYS	No.	%	No.	%	No.	%	No.	%	No.	%
Total Tested	188	100	142	100	177	100	167	100	162	100
Positive to Jelly	37	19.7	30	21.1	43	24.3	44	26.3	55	33.9
Positive to $\frac{1}{100}$	3	1.6	4	2.8	5	2.8	3	1.8	9	5.6
Total Positive	40	21.3	34	23.9	48	27.1	47	28.1	64	39.5
GIRLS										
Total Tested	177	100	183	100	181	100	156	100	140	100
Positive to Jelly	32	18.1	34	18.6	40	22.1	39	25.0	44	31.4
Positive to $\frac{1}{100}$	6	3.4	9	4.9	5	2.8	9	5.8	7	5.0
Total Positive	38	21.5	43	23.5	45	24.9	48	30.8	51	36.4

recorded when there was an area of palpable infiltration 5 ms. or more in diameter when the test was read at the end of 72 hours. A few marked reactions with areas of infiltration up to 25 ms. in diameter were encountered, but in no case was there any necrosis or ulceration.

PART I.

An attempt was made to test every child having his first routine medical examination in school during the year 1950. Table I shows the numbers and proportions of children tested and children not tested in the three age groups. It will be seen that absence from school either at the time intended for first testing or when a reading was required accounted for more than half of all the children not tested. To have returned to the schools to attempt to include such children would have entailed much additional time and considerable further upset of school routine.

It seems fruitless to speculate on the possible bias in the results due to the omission of about 35 per cent. of the children who were summoned for routine examination over the year.

There are about nine thousand children in each year age group in Belfast elementary schools. Depending on the time of year when they first attend a given

TABLE III

groups of Belfast children in age group 5 to 15 years.

10		11		12		13		14		15	
No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
...148	- 100	...143	- 100	...179	- 100	...153	- 100	... 57	- 100	...15	- 100
... 63	- 42.6	... 58	- 40.6	... 68	- 38.0	... 63	- 41.2	... 24	- 42.1	... 7	- 46.7
... 5	- 3.4	... 10	- 7.0	... 11	-...6.1	... 14	- 9.2	... 6	- 10.5	... 3	- 20.0
... 68	- 46.0	... 68	- 47.6	... 79	- 44.1	... 77	- 50.4	... 30	- 52.6	...10	- 66.7
...109	- 100	...142	- 100	...168	- 100	...163	- 100	...59	- 100	...10	- 100
... 49	- 45.0	... 50	- 35.2	... 49	- 29.2	... 45	- 27.6	... 12	- 20.3	... 1	- 10.0
... 12	- 11.1	... 11	- 7.7	... 23	- 13.7	... 26	- 16.0	... 13	- 22.0	... 2	- 20.0
... 61	- 56.0	... 61	- 42.9	... 72	- 42.9	... 71	- 43.6	... 25	- 42.3	... 3	- 30.0

school and whether they attend for examination in that year, their first school medical inspection (and in this year their test) may be while they are 5, 6 or 7 years of age.

Table II sets out the numbers and proportions giving positive reactions to the jelly test and to intradermal 1/100 O.T. by sex and age.

PART II.

In order to get figures comparable with the Medical Research Council data for England and Wales, children in each age group from 5-16 were tested in Belfast and in three provincial towns in selected schools where most of the children were country dwellers. These centres were Londonderry, Ballyclare (County Antrim), and Omagh (County Tyrone).

It was impossible to derive a satisfactory sample of the school populations for Part II of the investigation. Experience has shown that the proportion of acceptances is much higher if the parents of all the children in a school in an age group or class are asked to give permission than if isolated children in a school are picked out in a sample. In addition, the extra travelling involved in testing a random sample of the school population would have made the work extremely difficult. Finally, the upset to so many small schools which would have been

TABLE IV.

Results of jelly and $\frac{1}{100}$ intradermal tuberculin tests in schools children were

Age	5		6		7		8		9	
BOYS	No.	%	No.	%	No.	%	No.	%	No.	%
Total Tested	149	100	157	100	195	100	203	100	165	100
Positive to Jelly	31	20.8	45	28.7	62	31.8	75	37.0	63	38.2
Positive to $\frac{1}{100}$	6	4.0	5	3.2	9	4.6	16	7.9	15	9.1
Total Positive	37	24.8	50	31.8	71	36.4	91	44.8	78	47.3
GIRLS										
Total Tested	98	100	118	100	153	100	159	100	135	100
Positive to Jelly	23	23.5	30	25.4	49	32.0	53	33.3	62	45.9
Positive to $\frac{1}{100}$	6	6.1	3	2.5	11	7.2	10	6.3	8	5.9
Total Positive	29	29.6	33	28.0	60	39.2	63	39.6	70	51.9

occasioned by testing children chosen at random from County Education Department rolls would have interfered too much with school work.

Accordingly, what apeared to be representative schools (taking mostly country children) in the three centres were chosen and as many children as possible in each school were tested.

The children for the Belfast sample were derived by selecting what appeared to be representative schools without reference to previous testing of entrants. The same techniques were used as in Part I tests, but no records were kept of the proportions of refusals, as these were not required by the Medical Research Council. In each school the head teacher obtained as many consents as possible from the parents of his or her pupils.

The findings are set out in Tables III and IV.

An investigation such as was carried out would be impossible without the help of many people. The expense of the investigations was met by the Medical

TABLE IV.

in Ballyclare, Omagh, and Londonderry, where the majority of the country dwellers.

10		11		12		13		14		15	
No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
...181 -	100	...180 -	100	...166 -	100	...244 -	100	...188 -	100	...159 -	100
... 85 -	47.0	... 82 -	45.6	... 70 -	42.1	...121 -	49.6	...106 -	56.4	...90 -	56.6
... 13 -	7.2	... 10 -	5.6	... 15 -	9.0	... 16 -	6.6	... 6 -	3.2	...13 -	8.2
... 98 -	54.1	... 92 -	51.1	... 85 -	51.2	...137 -	56.1	...112 -	59.6	...103 -	64.8
...142 -	100	...141 -	100	...157 -	100	...138 -	100	... 90 -	100	...90 -	100
... 59 -	41.5	... 59 -	41.8	... 71 -	45.2	... 79 -	57.2	... 43 -	47.8	...46 -	51.1
... 7 -	4.9	... 18 -	12.8	... 7 -	4.5	... 6 -	4.3	... 6 -	6.7	... 3 -	3.3
... 66 -	46.5	... 77 -	54.6	... 78 -	49.7	... 85 -	61.6	... 49 -	54.4	... 49 -	54.4

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Dr. T. Fulton, Schools Medical Officer; Dr. J. Stuart Hawnt, Director of Education, Belfast, and the Headmasters of the schools in Belfast, who by their interest and assistance made the work in these schools possible.

Mr. A. C. Williams, of the Northern Ireland Ministry of Education; the Directors of Education of the Counties of Antrim and Tyrone and the City of Londonderry, selected typical schools in their areas, and the Headmasters of the schools chosen, were most helpful in obtaining parents' consents and in making the children available for testing.

Nurses M. M. McElroy and M. McCullagh assisted in the survey and visited many Belfast parents to get their consent, and Mr. J. D. Merrett, Department of Social and Preventive Medicine, The Queen's University of Belfast, helped with the records at all stages.

Retrolental Fibroplasia*

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HISTORY.

THIS abnormal condition which occurs in premature infants was first described by Terry in 1942. Terry described what is now known to be the final stage of the condition :—impairment of vision, nystagmus, squint, photophobia, microphthalmos, shallow anterior chanber and an opaque membrane behind the lens.

The early signs were not described until 1949, when Owens and Owens reported their observations. The first sign was dilatation and tortuosity of the fundal vessels (which was noticed from 4-12 weeks after birth); this progressed to retinal œdema, separation of the retina, and finally the formation of a retrolental membrane by organisation and fibrosis of the separated retina. Both eyes were usually affected, and spontaneous arrest was possible at any stage: indeed, different stages might be present in the two eyes.

In the U.S.A. the incidence has increased yearly since 1945, and retrolental fibroplasia is now said to account for one third to one half of blindness in young children. The incidence varies from area to area, and even from hospital to hospital in the same area.

In England the condition has been found in infants born as early as 1945, but few cases occurred before 1949. In most areas the incidence is increasing, and in some, the increase is alarming.

DIFFERENTIAL DIAGNOSIS.

Retrolental fibroplasia must be differentiated from a number of conditions :—

(1) *Persistent Hyaloid Vessels.* This condition is usually unilateral. It is present at birth, then disappears. Hæmorrhages and detachment of retina are not found.

(2) *Persistent Hyperplasia of Primary Vitreous.* This condition is usually unilateral and generally occurs in full-time infants. It can be detected shortly after birth (Reese).

(3) *Retinoblastoma.* This is unilateral, and often present at birth. The tumour has a yellowish tinge on ophthalmic examination, and there is X-Ray evidence of ocular calcification.

(4) *Congenital Cataract.* Cataract is usually unilateral and the opacity is *in* the lens.

(5) *Congenital Toxoplasmosis.* This is a differentiated by specific tests for toxoplasmosis.

*A lecture delivered to the Ulster Pediatric Society, 20th February, 1952.

(6) *Congenital Encephalo-Ophthalmic Dysplasia* (Krause). This syndrome includes neurological signs (microcephaly, hydrocephaly, cerebral dysplasia or mental retardation). It occurs also in full-time infants.

Early ophthalmic examination is of great assistance in the differential diagnosis of retrolental fibroplasia.

Possible Causes.

Because retrolental fibroplasia has only occurred during recent years, a pre-natal or congenital condition is unlikely to be the complete cause but it is impossible to exclude some pre-natal tendency to the condition which has been activated by one of the recent changes in post-natal care.

In fourteen Birmingham cases no relationship was found with any pre-natal condition and the preponderance of females and twins in this group of cases was similar to that for all surviving babies in the same weight group.

Possible post-natal causes include a better survival rate and recent changes in post-natal care. In addition, some new form of infection cannot be excluded.

The high incidence of cases in some areas cannot possibly be accounted for by the relatively small increase in survival rates. In the Birmingham Premature Baby Unit the majority of cases occurred during 1949, a year when survival rates were not at their highest.

Modern changes in the care of the premature baby which must be considered include :—

(i) Preliminary starvation period.

(ii) High-protein low-fat formulæ.

(iii) Variations in the type and administration of vitamin preparations.

(iv) Variations in the administration of iron.

(v) Increased administration of blood transfusions.

(vi) Use of new antibiotics.

(vii) Increased use of oxygen and different methods of administration.

In the Birmingham cases no relationship could be found with any of these factors except prolonged increased administration of oxygen.

According to Kinsey and Zacharias (1949), the incidence of the condition rose strikingly with increased number of days in oxygen. Was this because the child who is suffering from this complication requires more oxygen, or was the prolonged administration of oxygen a causative factor?

According to Szewczyk (1951), the whole picture can be explained by anoxia. When oxygen falls below the requirements of the fœtal retina, dilatation and tortuosity of the retina vessels occurs and new channels develop. If the need for oxygen is not met in this way, then œdema develops in the most fœtal (peripheral) parts of the retina, giving rise to greyish-white areas. If the retina uses all the oxygen available the venous walls will suffer, and transudations (accounting

for vitreous opacities) and hæmorrhages will occur. These lead to separation of the retina, the retina is pushed forward and atrophy and fibrosis occur.

If this condition were due to the administration of insufficient oxygen, the incidence would not be increasing with the modern tendency to use *more* oxygen. There is, however, the possibility that a *relative anoxia* is produced after acclimatising the infant to high oxygen tensions. In support of this theory, the following points can be made :—

(1) The condition does not arise while the infant is in an atmosphere with a high percentage of oxygen, but only after removal from such an atmosphere.

(2) In the U.S.A., oxygen beds were employed at an earlier date than in this country, and cases developed in the U.S.A. some years before they occurred over here.

(3) Nearly all cases (U.S.A. and England) occur in a few large centres which have special Premature Baby Units with facilities for the administration of oxygen.

(4) The occurrence of cases in this country has coincided either with the increased use of oxygen in established Premature Baby Units, or with the setting up of new units with full facilities for oxygen administration.

(5) The incidence has been reduced in Birmingham by limiting the administration of oxygen to cases showing cyanosis, and limiting the percentage of oxygen to the minimum required to keep the child pink : in other words, returning to the methods used before retrolental fibroplasia occurred. The use of oxygen has been limited in this way in two Birmingham Premature Baby Units (in one Unit for nearly two years and in the second for nearly a year and a half). Since the adoption of this practice, the survival rates have been maintained at the same level, but no further case of blindness has occurred. Two cases with early reversible changes occurred in one of the Units : they both returned to normal without treatment, and would not have been recognised if a routine weekly ophthalmic examination had not been carried out. It was interesting to note that both these cases required considerably larger amounts of oxygen than usual for their survival.

Against this theory it must be admitted that very occasionally an isolated case is reported, to which no oxygen has been given.

Administration of oxygen appears to have a very definite association with the development of retrolental fibroplasia, but more research is required to determine the method of causation and means of prevention. Accurate knowledge is of vital and urgent importance if this serious complication is to be prevented, and last October the Medical Research Council started an investigation into the possible causes of retrolental fibroplasia.

Treatment.

Even in the light of present knowledge, blindness due to this condition may be preventable if the obstetrician, pædiatrician and ophthalmic surgeon work together as a team.

(a) *Prevention.* Other factors being equal, the obstetrician should make every effort to prolong pregnancy until the infant weighs more than 4½ lbs. (i.e. 34-35 weeks gestation). The pædiatrician should only allow oxygen to be given to those infants who cannot maintain a good colour without it, and should keep the percentage of oxygen down to the lowest level which will maintain a pink colour. If prolonged administration or high concentrations are necessary, the infant should be weaned off the oxygen gradually and the fundi watched carefully.

(b) *Curative.* The ophthalmic surgeon should examine the fundi of all infants weighing 4½ lbs. or less at birth. The first examination should be made as soon as reasonably possible, and in all cases before the age of four weeks. Infants treated with oxygen should be examined within one week of stopping the administration of oxygen. Subsequent examinations should be made weekly.

Atropine ointment (1 per cent) should be used because drops may cause atropine poisoning. Good results are obtained with two applications 12 and 24 hours before the examinations.

If retinal œdema, vitreous opacities or hæmorrhages are seen, A.C.T.H. should be given (20 mg. twice daily for 7 days and repeated later if the condition progresses). Dilatation and tortuosity of the fundal vessels and pallor of the periphery of the retina are common findings in premature babies and do not require treatment unless later stages develop.

Szewczyk has suggested that infants who show early signs of retrolental fibroplasia might be cured by returning them to a high concentration of oxygen, but sufficient trial has not yet been given to this treatment.

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Neonatal Infection

With Particular Reference to Infection with Penicillin Resistant Staphylococci

By M. G. NELSON, M.D., M.R C.P., M.R C.P.I., D.T.M. & H.

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INFECTION among the new born babies of a maternity hospital nursery always gives rise to anxiety and engenders a feeling that all is not well with the nursery technique, although this may be quite unjustifiable. However, the problem is one which interests the medical and nursing staff and involves the bacteriologist in extensive investigations if any attempt is to be made to trace the infection to its source. It has been shown that infection during this period of the life of the infant is largely due to invasion by a pathogenic staphylococcus and usually manifests itself as conjunctivitis or infection of the skin. The skin lesion consists of pustules which have been referred to as contagious subepithelial dermatitis and rarely proceeds to pemphigus neonatorum.

In the Royal Maternity Hospital, Belfast, cases of skin infection of infants in the neonatal period, while not frequent, are persistent and continue despite a rigid aseptic technique. As these infections are considered to be due largely to hospital cross infection, the incidence, source and method of spread require to be carefully investigated before control can be effectively employed.

METHOD.

In the investigation of the carrier state, swabs for culture were collected from the hands, the throat and the anterior nares. Before specimens were collected from the hands and nares the swabs were first moistened in sterile 'Lemco' broth.

Swabs from all lesions and from carriers were planted on horse blood agar plates with a 'Lemco' broth base. The penicillin sensitivity was tested by the disc paper technique, using a five millimetre diameter filter paper disc and 0.02 ml. of penicillin solution containing 10 units/ml. After 18 hours culture at 37° C. the plates were examined for staphylococci. Coagulase production was determined by the slide test of Cadnes-Graves, Williams, Harper and Miles (1943) and the penicillin sensitivity of all coagulase positive staphylococci noted.

INCIDENCE OF NEONATAL INFECTION.

The incidence of neonatal infection over a six month period in 1950, in one unit of the maternity hospitals where the infants were nursed in the same general ward as the mothers was determined and it was found that neonatal infection occurred in 9.7 per cent. of the total number of live births recorded during the period of observation. Of eighty-four instances of neonatal infection 56 per cent were infections of the skin, 34 per cent were infections of the conjunctivæ and

the remainder was a variety of miscellaneous infections. Thus it can be seen that the majority of the infections noted in neonates involved the skin and conjunctival sac, with infections of the skin predominating. The criteria used for the diagnosis of infection of the skin were stringent so that even the smallest pustule was so classified, yet the overall incidence during the period investigated was only of the order of 10 per cent. Corner (1950) reported a neonatal infection rate in Bristol of 20-30 per cent, Barber, Hayhoe and Whitehead (1949) reported from London a rate of 14 per cent, and Roscoe (1949) recorded a 10 per cent incidence in Cambridge. A neonatal infection rate of 9.7 per cent therefore compares most favourably with that found in other centres where the same criteria of classification were employed.

In an attempt to discover whether localisation of infection was determined by sweating, with infection occurring mainly in the warmer parts of the body or by air and dust contamination of exposed parts, the site of the main lesions was noted. It was found that the body folds were mainly involved in six instances, the limbs in ten, the trunk in thirteen and the face and neck in twenty-seven. As is to be expected these results are equivocal because the air and dust contamination of the exposed parts of the body would also contaminate the bedclothes of the infant and hence the body. However, the absence of any preponderance of lesions in the body folds tends, in itself, to exclude excessive sweating as a major predisposing factor to infection and suggests a more general environmental contamination

Representative swabs taken from the lesions were examined bacteriologically (Table I). It was found that in 88 per cent the causal organism was a coagulase positive Staphylococcus pyogenes, but the most disturbing feature was that of the strains of Staph. pyogenes isolated from lesions in infants 67 per cent were penicillin resistant.

TABLE I.

BACTERIOLOGY OF NEONATAL INFECTIONS.

	Total		Coag. +ve Staphylococci		Penicillin Resistant
Eye - - - - -	31	...	29	...	22
Skin - - - - -	48	...	47	...	31
Miscellaneous - - -	5	...	0	...	0
	—	...	—	...	—
	84	...	76	...	53

These results again emphasise the well established fact that the Staph. pyogenes is the most important pathogenic organism in infection acquired during the neonatal period.

Treatment of the skin lesions and of the conjunctivitis consisted at first of the application of penicillin ointment to the cutaneous lesions or the installation of penicillin drops into the eyes, but the response to this therapy was disappointing because the majority of the strains of Staph. pyogenes isolated from these lesions

were resistant to penicillin when tested in vitro. The routine use of penicillin was then discontinued and the antibiotic or sulpha drug used was selected on the basis of the in vitro sensitivity of the casual organism to a range of therapeutic agents.

Pilot Survey.

In April, 1950, a pilot survey was carried out in the Royal Maternity Hospital in one ward where the infants were nursed together with the mothers in the same general ward to determine the staphylococcal carrier state among the nurses, attendants and patients. In an attempt to try and trace the source of infection the kind assistance of Dr. R. E. O. Williams of the Staphylococcal Reference Laboratory, Colindale, London, was obtained and he undertook to phage type all the strains isolated.

This survey showed that about 50 per cent of the nurses and mothers were carriers of potentially pathogenic staphylococci and of the 38 strains isolated 24 (or 63 per cent) were resistant to penicillin. All the strains of staphylococci isolated were subjected to phage typing and although many of the strains were untypeable, two traceable types were found and were distributed as shown in Table II.

TABLE II.

Distribution of Phage Typed Strains of Staphylococci.

	Cases		Carriers		
			Nurses		Mothers
52/52 A - - - - - -	6	...	9	...	3
47/54 - - - - - -	3	...	4	...	1
Miscellaneous or Untypeable - - -	11	...	25	...	14
	—	...	—	...	—
	20	...	38	...	18

Using the indicator strains it was obvious that the infection was not derived from one single chronic carrier among the staff or patients. On the contrary, there appeared to be a high carrier rate among all the ward inmates of many strains of stapyhlococci of differing phage type. This was associated with a heavy environmental contamination which was shown when dust was collected and swabs were taken from ward table tops, linen bins, wash-hand basins and various other sites in the ward. Air contamination was investigated by exposing 'settling plates' of blood agar in the ward at various times. Of some fifty specimens examined from the ward environment twenty showed the presence of coagulase positive staphylococci.

The Staphylococcal Carrier.

It has been recognised for some considerable time that potentially pathogenic staphylococci are frequently found in the anterior nares of otherwise normal healthy adults and some workers state that this carrier rate is of the order of

50 per cent (Hallman 1937; McFarlan 1938; Gillespie, Devenish and Cowan 1939). However, in any hospital community the carrier rate among the permanent staff or long stay patient is much higher than among the population at large and this is particularly true of the nursing staff (Miles, Williams and Clayton-Cooper 1944; Allison and Hobbs 1947).

To confirm these observations and to obtain information regarding the carrier state of Staph. pyogenes in this area among the general population and hospital inmates, a survey was made of a number of out-patients, taken as a rough sample of the local population, and of in-patients and staff of both the Royal Maternity Hospital and the Royal Victoria Hospital, Belfast.

TABLE III.

TOTAL STAPHYLOCOCCAL CARRIER RATE.

	Royal Victoria Hospital			Royal Maternity Hospital		
	Out-Patients	In-Patients	Nurses	Adult In-Patients	Babies	Nurses
Total	100	100	100	230	150	52
Carriers	31	47	62	98	92	30
Percentage	31	47	62	43	61	58

The results of the investigation into the carrier state are recorded in Table III from which it can be seen that the staphylococcal carrier rate is greater among hospital in-patients than among out-patients. The difference in incidence between these two groups is not large but is probably more significant than it appears when it is considered that the in-patient group is mixed and consists of many short stay patients or patients investigated shortly after hospitalisation, before they had time to acquire the ward flora.

The high carrier rate among the nursing staff of 58-62 per cent is the most significant finding and corroborates, if such were needed, the work of many others in this field. From this constant hospital source very heavy environmental contamination with Straph. pyogenes results so that there is a greatly increased risk of acquiring a staphylococcal infection in hospital. This risk is of course greater for patients with lowered resistance and constant exposure than it is for the hospital staff, and in hospital it is a very grave risk for infants with poorly developed powers of resistance to infection. As it will be shown that a high percentage of the strains of Staph. pyogenes isolated in hospitals are resistant to penicillin, any neonatal infection acquired in hospital will most likely prove not to be amenable to penicillin therapy.

Swabs were taken from the nose, throat and skin of the hands, of a group of patients and nursing staff of both the general and the maternity hospital. The results of the isolation of Staph. pyogenes from these sites is shown on Table IV which shows that the staphylococcal carrier is predominantly a nasal carrier and this fact is of considerable biological significance because a nasal carrier can so easily contaminate the environment.

TABLE IV.

Distribution of the Staphylococcus Among Carriers.

	Total Examined	Nose	Throat	Hands	Total Carrier State	Percentage Nasal Carriers
Maternity Hospital						
Nursery Staff - -	30	26	12	13	27	96
Ward Nurses - -	52	26	6	7	30	87
Mothers - -	58	28	4	7	33	85
General Hospital						
Out-Patients - -	100	22	1	13	31	71
In-Patients - -	100	43	12	28	47	91
Nurses - -	82	47	13	14	49	96
	—	—	—	—	—	—
	422	192	48	82	217	
	—	—	—	—	—	—
Average Percentage		46.1	11.6	19.8	52.4	88

The Staphylococcal Carrier State in the Neonatal Period.

During the course of this investigation swabs were collected from the anterior nares and skin of 150 newborn infants. Staph. pyogenes was isolated from 92 infants (61 per cent) and the majority (88 per cent) of these strains were isolated from the nose. Thus, it is seen that the carrier state in infants approximates to that found among the nursing staff and suggests that the infants are infected from the environment and are not directly infected from their mothers. This is in conformity with the views expressed by Allison and Hobbs (1947).

Concurrently with this investigation into the carrier state of staphylococci by infants, an attempt was made to determine the rapidity with which the infants in a maternity hospital acquired the flora of their environment by colonisation of the anterior nares. To this end swabs were taken at three day intervals from the noses of all infants born in this hospital and the isolation of Staph. pyogenes is recorded on Table V.

TABLE V.

Nasal Carrier Rate of Staphylococcus Pyogenes in Neonates.

Age in days - - -	0-	3-	6-	9-
	—	—	—	—
Total - - -	17	26	42	21
Staphylococcus pyogenes in nasal swabs - -	3	13	31	17
	—	—	—	—
Percentage carriers -	18	50	81	81

It can be readily seen that the newborn child, in hospital, rapidly acquires the staphylococcus from its heavily contaminated environment and that this risk

increases with the length of hospital stay, so that after one week some 80 per cent of infants have become carriers of a potentially pathogenic organism. In this connection the child is being used as a biological index of environmental contamination and it is probably true to say that if colonisation occurs in the nares at this alarming rate the skin and conjunctivæ are equally exposed to similar heavy contamination by a pathogenic organism which can readily invade the tissues of the infant if the normal biological bacterial barriers are temporarily lowered.

These facts require careful consideration by all ardent protogonists of institutional midwifery. The bacteriological data here presented would suggest that the shorter the post-partum period of hospitalisation the better from the point of view of the newborn infant—a practice which might do much to help the bed state in maternity hospitals.

Penicillin Resistant Staphylococci.

In chemotheraphy one of the most important problems is therapeutic failure due to the appearance of drug resistant micro-organisms. When penicillin was introduced it was considered that resistance to the drug would not prove a major problem in therapy. This hope has not been realized. Prior to the extensive use of penicillin only 12 per cent of the strains of staphylococci isolated were found to be naturally resistant to penicillin, Spink, Ferris and Vivino (1944), Bondi and Dietz (1945). The appearance of an increasing number of penicillin resistant strains was noted in hospital practice to which the use of the drug was, at first, confined. North and Christie (1945) recorded the incidence of penicillin resistant strains isolated in the same ward before and after the introduction of penicillin and showed a marked increase in resistant strains. Thus, of 128 strains of staphylococci isolated before the introduction of penicillin therapy in the ward, no strains were found to be resistant to this drug. Of the first 31 strains isolated after the drug had been used in the ward 17 strains were penicillin resistant.

It is the hospital population which is most likely to be exposed to penicillin therapy and has always shown the highest incidence of strains resistant to the drug. Many workers including Forbes (1949), Barber and Whitehead (1949), have shown that strains of staphylococci isolated from lesions contracted outside hospitals are much more likely to be penicillin sensitive than are strains isolated from similar lesions acquired in hospital. However, the more widespread use of the drug in general practice is likely to be associated with the appearance of more resistant micro-organisms among the population at large. This has indeed proved to be the case and reports from all parts of the world, where penicillin has been in general use, indicate that this increase in resistant strains is general in its geographical distribution. It is also quite independent of age, for resistant staphylococci were found in the nasopharynx of 55 per cent of newborn babies by Martyn (1949), in 30 per cent of healthy children by Beigelman and Rantz (1950), and in a varying percentage of healthy adults. This increase is not a static state

but is actually rising and Barber has reported in a number of papers a marked increase in penicillin resistant strains of staphylococci.

1946	.....	14 per cent
1947	.....	38 per cent
1948	.....	59 per cent

During the course of this investigation the penicillin sensitivity of the majority of the strains isolated was determined and the results are recorded in Table VI. From these results it can be seen that the incidence of penicillin resistant staphylococci among the general population (out-patients) was 11 per cent, while the hospital population showed a very much higher proportion of carriers of penicillin resistant strains (39-94 per cent). The higher incidence of penicillin resistant strains isolated from carriers among the in-patients and nurses of the general hospital than from patients and nurses of the maternity hospital is not easily explained. In both hospitals, however, more penicillin resistant strains were isolated from nurses than from adult in-patients in the same hospital.

TABLE VI.

PENICILLIN RESISTANCE OF STAPHYLOCOCCUS PYOGENES ISOLATED FROM CARRIERS.

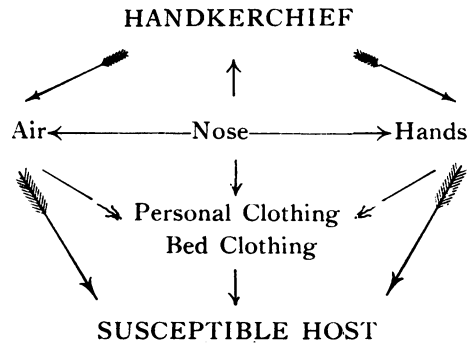
	Royal Victoria Hospital.			Royal Maternity Hospital		
	Out-Patients	In-Patients	Nurses	Babies	Adult In-Patients	Nurses
Total strains	36	83	55	47	38	42
Number resistant to penicillin	4	68	52	37	15	28
Percentage resistant	11	82	94	79	39	67

The high carrier rate of penicillin resistant staphylococci by the nursing staff is of considerable significance in the spread of penicillin resistant staphylococcal infections in hospitals and this must be regarded as the main source of hospital infection with these resistant organisms.

To explain the development of penicillin resistant forms of micro-organisms various theories have been propounded but the one most widely accepted is based on genetic principles. This theory postulates that in any large population of any penicillin sensitive strain of micro-organism there are a few, naturally occurring and relatively resistant variants. When such a population mixture is exposed to penicillin, the sensitive organisms are destroyed but the resistant forms not only survive but multiply. Among the progeny new variants appear which are resistant to higher concentrations of penicillin. This resistance is permanent but is not associated with any loss of invasiveness on the part of the micro-organism. We are therefore faced with a biological phenomenon of great importance to the host and one which poses a problem for penicillin therapy in the future. Fortunately aureomycin has so far proved to be a most effective antibiotic for penicillin-resistant staphyococci. It is left to the chemist to continue to produce antibiotics of wide range and greater effectiveness to circumvent this natural selection of resistant variants.

Method of Spread.

The main reservoir of pathogenic staphylococci is the anterior nares as has been shown by previous workers, Gillespie, Devenish and Cowan (1939); Miles, Williams and Clayton-Cooper (1944) and Williams (1946) and again demonstrated in this investigation where 88 per cent of carriers had Staph. pyogenes in their nasal swabs. From this source the method of spread has been shown by Hare and Mackenzie (1946) and others, to be largely indirect.



The nasal carrier is particularly likely to contaminate the environment, for Hamburger and Green (1946) have shown that the nasal carrier expels a hundred times as many pathogenic organisms into the air as a throat carrier who has negative nasal cultures. Millions of organisms are expelled by blowing the nose and the act of blowing the nose contaminates the handkerchief, the hands and the air. Once the hands are contaminated they infect everything they touch. Thus the great secondary "reservoir of infection" is created in the personal clothing and bedclothes of the patient which are readily contaminated by the hands or from the air. It is the major 'source' of air borne infection (Brown and Allison 1937).

The presence of Staph. pyogenes in the air and dust of the wards of the maternity hospital was amply demonstrated during this investigation by the presence of these organisms on 'settling plates' of blood agar exposed in the wards, in swabs collected from various sites and in various samples of dust.

Prevention of Neonatal Infection.

The control of the spread of infection in a nursery can conveniently be discussed under three headings viz., the source of infection, the method of spread, and the susceptible host. As the majority of infections is caused by the Staph. pyogenes the control will be largely orientated towards methods designed against the spread of this particular organism.

(1) *The Source.*—This is recognised to be a contaminated person, either a mother, a nurse, or other attendant, who infects the air or dust of the ward. This indirect type of infection is thought to be much more important than any direct transfer of organisms to the susceptible infant. As nurses have a very

high carrier rate of penicillin resistant Staph. pyogenes they must always regard themselves as a potential source of danger and must at all times take such steps as to minimise environmental contamination. This is of course inherent in their training but must be rigidly carried out in practice. Particular attention should be paid to the face mask which must be of approved design, and worn in the appropriate way. Slipshod methods in using a face mask are to be deplored, as they give a false sense of security. The other great personal sources of environmental contamination are the handkerchief and the hands. The staphylococcus is carried predominantly in the anterior nares where it can readily be dispensed into the atmosphere and particularly into the handkerchief which is always the most heavily contaminated article of personal apparel. From the handkerchief or the pocket the hands can be secondarily contaminated and thence organisms are conveyed to everything which is touched. Therefore attendants should at all times be most careful about hand hygiene, especially after use of a handkerchief.

However, these measures are purely negative and do not take into account the obvious source of possible infection from nasal carriers among the mothers in the ward. A positive step to eradicate all carriers in a ward unit would involve treatment designed to remove or greatly reduce the carrier rate among all the ward inmates including the nursing mothers. This presents many difficulties. It requires the topical application to the anterior nares of a cream containing an antibiotic. This antibiotic must be one to which the organism is sensitive and to which the organism does not rapidly develop resistance. The method requires conscientious treatment but despite rigid application the objective may be vitiated by changes in personnel necessitated by off-duty and emergencies. A preliminary trial of the value of the local daily application of cream containing aureomycin to the anterior nares was made on persistent nasal carriers of Staph. pyogenes among the laboratory staff with completely satisfactory results. The method was then given an extensive trial on one ward unit in the Royal Maternity Hospital where the infants were nursed in the general ward, for an eight week period using either 5 mgm. of aureomycin or 5 mgm. of chloromycetin in a base containing 5 per cent Promulsin wax and 10 per cent ground nut oil made into an emulsion with water. The interest of the nursing staff was first aroused and the co-operation of all the staff was excellent. The cream was applied night and morning to the anterior nares of all the nurses in the ward and to all the mothers. The infants were left untreated and used as a biological index of environmental contamination. The results were most discouraging although it was noted that *permanent* nursing staff who had been persistent nasal carriers showed negative swabs throughout the period of the trial.

The average figures of the ward carrier rate before and during the trial are shown (Table VII) together with the nasal carrier rate of Staph. pyogenes in a control untreated ward unit where the infants were nursed in a separate nursery. Therefore, for the untreated ward figures for the neonatal carrier rate cannot be included.

TABLE VII.

THERAPEUTIC TRIAL OF ANTIBIOTIC CREAM APPLIED TO THE NARES OF MOTHERS AND NURSES.

		Percentage Staphylococcal Carrier Rate		
		Ward not Treated		Ward Treated
Adults:	Control period - - - - -	59	...	64
	Test period - - - - -	35	...	40
Infants:	Control period - - - - -	—	...	72
	Test period - - - - -	—	...	65

It will be seen that there is a fall in the ward carrier rate but this occurred also concomitantly in the untreated ward and cannot therefore be regarded as due to the method of control under trial but due to general factors operative over the period surveyed. The carrier rate among the infants did not fall significantly, so that the environmental contamination appeared to be uncontrolled, probably because the method used did not reduce the general carrier rate in the ward below a critical level. These results were very disappointing and reflect the difficulties of the practical application on a wider scale of a successful but more easily controlled experiment on a few patients. It is interesting to note that no aureomycin or chloromycetin resistant strains of Staph. pyogenes were detected during the period of observation.

(2) *Method of Spread.*—Measures directed against the methods of spread of neonatal infection consist largely of control of the contaminated air and dust in the ward—i.e.

(a) *Dust Hygiene*
- Wet sweeping or vacuum cleaning
- Oiling of floors
- Oiling of blankets

(b) *Air Hygiene*
- Good cross ventilation
- Germicidal aerosols
- Ultra-violet light

Methods for dust suppression are already in use in the hospital concerned and include both wet sweeping and oiling of floors but the blankets are not treated. Ventilation is natural and no methods of air hygiene by the use of germicidal vapours or ultra-violet light are employed in the general wards.

The most popular germicidal aerosol at present in use is triethylene glycol vapour which has been extensively tried. However, Krugman and Ward (1951) reported after a prolonged trial of air sterilisation in an infant's ward by this method that although cross infections were statistically reduced the bacterial content of the air still remained high. This was probably due to the fact that the bactericidal properties of the aerosol are impeded by dust or dried dust-borne bacteria. These authors reported that the most significant reduction of

the bacterial content of the air was achieved by a combination of dust suppressive measures and the use of germicidal aerosols. Because of the disparity between the cross infection rate and bacterial content of the air, these authors were led to believe that air borne infection did not play the major role in cross infection in hospital—a view which is not widely held.

It has been known for some seventy-five years that ultra-violet rays will kill bacteria and more recent researches have established the effective wave length for bactericidal action to lie within the range 2,600-2,500 A.U. of the ultra-violet spectrum. Suitable lamps with a high emission around the effective range have been produced and extensively tried by various methods of which indirect or upper air irradiation has been the most popular. The results of this method of air sterilisation in hospital wards in America have been encouraging although conflicting reports have appeared. It is known that the germicidal action of ultra-violet light has its greatest effect in atmospheres free from dust and with low humidity and these environmental factors may affect the results obtained.

A trial of this method of air sterilisation was made. Settling plates were exposed in two wards of the nursery in four different sites and at four periods of one hour each, two during maximum and two during minimum periods of activity in the wards. From these a base line of general ward contamination was obtained. One ward was then fitted with ultra-violet lamps sited to irradiate the upper air and one ward was kept as a control. After a suitable working period the settling plates were again exposed in the same sites and for the same periods on two occasions and the results assessed. The results have shown a marked diminution of aerial contamination.

(3). *The Susceptible Host.*—It can be seen that the newborn infant nursed in the same general ward as the mother is exposed to the hazards of an environment heavily contaminated with pathogenic staphylococci. The nursing technique employed reduces the possibility of contact and droplet infection by ward attendants to a minimum and measures designed to prevent the contaminated air and dust of the ward from penetrating the delicate skin of the newborn baby are already in use. These largely consist of the application of a rigidly aseptic and non-traumatising technique. This has been reinforced in some centres by the application to the skin of the infant of an antiseptic lotion or barrier cream but this method has not achieved any popularity.

In actual practice, as Allison and Hobbs (1947) have shown, it is not easy to eradicate neonatal infection even by the application of the strictest aseptic precautions. This suggests, as the technical difficulties of adequate preventative measures in nurseries are so great, that it might be better to nurse mother and infant together in a small ward or cubicle. Whether this would completely solve the problem is doubtful, for Loosli, Smith, Cline and Nelson (1950) reported widespread dissemination of streptococci throughout the cubicilised wards of the Harriett Lane Home of the Johns Hopkins Hospital. The source of infection was a case of eczema infected with Streptococcus pyogenes and the spread occurred through contaminated air and dust despite the fact that air sterilisation procedures

using triethylene glycol were in use at the time. These authors were able to show that the efficient treatment of the focus of infection reduced the environmental contamination and the number of secondarily infected cases.

The statement of Coburn that the spread of infection among infants is uncontrollable in spite of strict aseptic precautions is most discouraging but emphasises the necessity for attacking the problem on a broad front with particular reference to control of air and dust contamination of the ward. It is also evident that the increasing numbers of penicillin resistant strains of the common infecting organism have not made the problem any easier.

Summary.

In one unit of a maternity hospital neonatal infection was of the order of 10 per cent of all live births and this infection consisted largely of lesions of the skin and conjunctivæ.

The causative organism responsible for the majority of neonatal infections was the coagulase positive Staph. pyogenes which was isolated from 88 per cent of the lesions and of these strains 67 per cent were resistant to penicillin.

A high staphylococcal carrier rate was found among the maternity hospital nursing staff (58 per cent) and this carrier state was persistent. A survey of the carrier state of nurses in the nearby general hospital showed that the carrier state was of the same order (62 per cent).

The staphylococcal carrier state of adult in-patients was 43 per cent in the maternity hospital and 45 per cent in the general hospital. The incidence was lower among adult out-patients, 31 per cent.

As has been previously demonstrated, the staphylococcal carrier is most often a nasal carrier and in this series 88 per cent of carriers showed the presence of Staphylococcus pyogenes in the anterior nares.

The main reservoir of infection is from staphylococci in the anterior nares of such a high proportion of the nursing staff and to a lesser extent the in-patients of the ward. This is responsible for a heavy environmental contamination of the hospital ward, so that all newborn infants rapidly become carriers of the prevalent organism and could thus be used as an indirect index of environmental contamination. Direct evidence of general ward contamination is afforded by the isolation of coagulase positive staphylococci from various sites in the ward.

Prevention is discussed from the points of view of control of the source, the method of spread and the susceptible host.

My thanks are due to the medical and nursing staffs of both the Royal Maternity and Royal Victoria Hospitals, Belfast, for their very willing co-operation and to my clinical pathologists and technicians for much assistance during this investigation.

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REVIEW

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MANY readers of this Journal are familiar with Doctor Coope's valuable work on Diseases of the Lungs, but here is another aspect of his scholarship. In the foreword Sir Russell Brain reminds us that an anthology is a collection of flowers, and one cannot recall a more varied and beautiful bouquet than is here presented. The sources are indeed so numerous that one wonders at the collector's range of travel and catholicity of taste; and every bloom is worthy of inclusion. There are quotations from Saint Theresa and George Bernard Shaw, from Byron and Bachya Ibn Pakudah, from Rabelais and John Lyle . . . and when you read the book we believe that you will never say "Now, why on earth did he put that in?" Each quotation seems to be worthy of a place in the book which will live as long as mankind has any need for doctors or doctors any love for their patients. R. M.

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The Second International Poliomyelitis Conference

A Report by

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THE Second International Poliomyelitis Conference was held in Copenhagen in September, 1951. It was sponsored jointly by the National Foundation for Infantile Paralysis of the United States of America and the Danish National Association for Infantile Paralysis.

The National Foundation was founded by the late Franklin D. Roosevelt, who himself had suffered from this disease. In 1934, when Mr. Roosevelt was President of the United States, dances to raise funds for the organisation were held on his birthday, 30th January, in all parts of the country. An annual fund-raising appeal is conducted during the first weeks in January yearly called "The March of Dimes." Some seventy million individuals subscribe to this fund and in the current year the voluntary subscriptions totalled thirty million dollars. During the years of its existence the National Foundation and its twenty-eight hundred local chapters in all parts of the States have been devoted to the programme of

1. Providing financial assistance for care of poliomyelitis patients needing aid, regardless of race, colour or creed.
2. Financing of the most extensive voluntary research programme ever levelled at a single disease.
3. Financing education of professional personnel.

These are incorporated in the organisation's basic credo "To lead, direct and unify the fight against Infantile Paralysis."

The problem of poliomyelitis in the United States is a growing one. In the three years 1939 to 1941 there were some 26,000 cases. In the same period 1942 to 1944 there were 35,000 cases. In 1945 to 1947 there were 50,000. In the three years 1948 to 1950 there were 104,000 cases of the disease.

The original International Poliomyelitis Conference was held in New York in 1948. Its success warranted the occasion of the second Conference. This second Conference was attended by six hundred delegates representing thirty-seven Nations. The Conference was opened by the Rector of the University of Copenhagen, in the Ceremonial Hall of the University in the presence of the Queen of Denmark. Thereafter for five days the scientific sessions were held in the Medical Anatomy Institute of the University. The papers at these sessions were delivered in English, French, Spanish or German. There was a simultaneous interpretation of each address into the three other languages through the interpretation mechanisms of the type used by the United Nations.

The first scientific session was devoted to discussion of Virus and its interaction with the Host Cell. A paper on "External Factors Influencing Virus" by Horsfall, of the Rockefeller Institute of New York, made it clear that, for the present there is no immediate therapy for poliomyelitis. Work reveals that it may be possible to interrupt the multiplication of virus within the host cells, but this is unlikely to be of clinical application for some time.

Enders, working at the Children's Hospital, Boston, reported that it was now possible to cultivate poliomyelitis virus on non-nervous tissue. Hitherto the only means available for the recovery of virus from diseased tissues was by animal inoculation. Cultures of several human tissues have been studied. Among these are embryonic brain, intestine, skin, muscle, kidney, heart and liver. Of these embryonic tissues, skin muscle and brain have proved most suitable for routine experimental work. On account of the difficulty of obtaining a suitable supply of embryonic tissue, mature tissues have been investigated. These tissues include foreskin, uterus, kidney and testis removed at operation. Of these, uterus and kidney tissue have, so far, been most used. Apart from human tissues, virus multiplication has been sought in tissue of whole chick embryos, chick embryonic brain, whole mouse embryo, embryonic mouse brain, adult mouse brain, mixed beef embryonic tissue, and adult rabbit testis.

Apart from the three standard strains of poliomyelitis virus, namely, Brunhilde, Lansing and Leon, additional strains isolated from stools have been examined. For the typing of these viruses, immune sera prepared in rhesus monkeys have been used. In the culture of virus certain types of tissue cells are attacked and destroyed. Fibroblasts appear to be the first to be affected and are the best index of viral multiplication. It is possible to prevent these cellular changes by the addition to the culture of the type-specific anti-serum. For example, if Lansing type serum is added to the culture just before inoculation with Lansing virus, cell injury is inhibited or greatly retarded. Inhibition by serum is type-specific and this affords evidence that the virus is directly or indirectly responsible for the injury observed. It will provide a means of typing specifically an unknown virus or anti-serum. The detection of virus by tissue culture would appear to be at least as sensitive as by monkey inoculation. It is possible that strains of virus hitherto unrecognised because of low pathogenicity for monkeys may be revealed. The time required for isolation and culture of a strain by this method will probably not exceed that required by animal inoculation technique. It has also been shown that this culture of virus is followed by diminution of its pathogenicity for lower animals. This fact encourages the hope that further culture will reveal variants whose diminished virulence for man might allow of their use as specific immunising agents against poliomyelitis.

Bodian, from the Poliomyelitis Laboratory of Johns Hopkins University, discussed virus and host factors determining the nature and severity of lesions and of clinical manifestations. He recalled that virus strains differ greatly in their capacity to injure or destroy nervous tissue. The severity of lesions and their

symptoms may be reduced due to partial immunity from previous paralytic or non-paralytic infection and individual hosts vary considerably in response to similar infective doses. Work suggests that the inflammatory reaction as it develops may be complexly determined; its origin is dependent on the specific reaction of the virus with nerve cells.

Bowden, working at the Royal Free Hospital School of Medicine, London, on studies of skeletal muscle in poliomyelitis, produced evidence to suggest that hypertrophy of the normal muscle fibres plays no significant part in the recovery from paralysis within the first six months. There is, however, evidence that the recovery of muscle in the experimental animal is due, to some extent, to the extension of the residual motor units.

Daldorf, working in the New York State Laboratories, discussed the isolation and properties of the Coxsackie viruses. These viruses derived their name from the town, Coxsackie, where they were first isolated in 1947. They were recovered from the fæces of two children recently ill with paralytic poliomyelitis. The intervening years revealed two main groups of this virus, with several sub-types. A distinguishing feature of the Coxsackie viruses is that they are pathogenic only for suckling mice. A second feature is the nature of the experimental disease. Mice infected with group A strains develop flaccid paralysis. On the other hand, mice infected with group B strains have tremors and spasms as well as weakness. Hepatitis and myocarditis have occurred in experimental animals. Differentiation of types is made by the neutralisation test using serum prepared in large mice and adult hamsters by injection into them of living virus. Complement fixation tests have confirmed the results of neutralization tests. Diagnosis of human infection by serological methods is cumbersome. It is safer to rely for diagnosis on the isolation of virus. The physical properties of the Coxsackie viruses are similar to those of poliomyelitis virus. Both are small and stable. Specimens from patients remain infective for years when stored in dry ice. Infective tissue may be preserved indefinitely in 50 per cent glycerol. Poliomyelitis virus is usually inactivated at a lower temperature than Coxsackie virus.

Coxsackie viruses have by now been isolated in many parts of the United States, Canada, Sweden, Denmark, France, Israel and Australia. They have usually been found in patients with symptoms of poliomyelitis. It is now well known that they also occur in association with other conditions. In New York during the past four years summer outbreaks of poliomyelitis have been of mixed variety. The presence of Coxsackie virus has been demonstrated in specimens that also yielded poliomyelitis virus. Clinical records suggest that Coxsackie virus is more frequently associated with less severe poliomyelitis.

Curnen, of Yale University, discussed the immunology, epidemiology, and clinical aspects of Coxsackie virus infection. Type specific immunity is demonstrable in mice born of immunised mothers and in chimpanzees. Antibody responses to Coxsackie viruses in man were investigated in six laboratory workers who contracted infection while working with these agents. Each patient developed neutralising and complement fixing antibodies following

infection. There is no evidence available to show whether or not immunity in man to any of the Coxsackie viruses confers protection against any of the poliomyelitis viruses.

Viruses of the Coxsackie group have been found in fæces, pharyngeal swabbings, cerebro-spinal fluid and blood in human cases. Various types of Coxsackie virus have been recovered from sewage and from flies. Present information suggests the same seasonal incidence as the poliomyelitis virus, that is, mainly during the warmer months. There appears to be identical incidence between the two sexes. Infection may occur at any time of life. The spread of Coxsackie viruses appears to be by similar means to that of poliomyelitis. The viruses may be present in pharyngeal secretions for days and may be excreted in the fæces for periods of many weeks. At present, it appears that man himself is the main source, reservoir and conveyor of human infection, and from him infection may be spread by contamination of water or food, as well as by direct contact.

Clinically it does not seem possible to distinguish some infections with Coxsackie virus from non-paralytic poliomyelitis. Findings in the cerebro-spinal fluid are similar. Evidence is accumulating that viruses of this group may be the cause of epidemic myalgia, pleurodynia or Bornholm disease. Twelve patients, whose illnesses resembled Bornholm disease and who showed evidence of infection by strains of Coxsackie virus had fever, eleven of these had chest pain, eight had headache, six had abdominal pain, four had stiff neck, three had nausea, two had limb pains, two had weakness, two had hyperæsthesia and one had vomiting and sore throat.

Coxsackie viruses have been recovered from patients complaining of sore throat who had vesicular and aphthous lesions on the faucial pillars. This condition now styled Herpangina, resembled strongly the condition formerly described as herpetic sore throat. It is probable that the virus may yet be recovered from many of the similar febrile conditions.

Debré, of Paris, discussed the differential diagnosis in paralytic poliomyelitis. He reviewed 509 cases in the Paris Hospital for Sick Children with a diagnosis poliomyelitis between 1947 and 1950. The diagnosis in 71, or 14 per cent of these had to be revised. Of these, 26 had non-neurological affections and of these 26, eleven had acute arthritis, four osteomyelitis and two injuries.

Of the revised diagnoses forty-five were other types of neurological affections; of these, 36 had radiculo-neuritis with Guillain-Barré syndrome. Debré suggests that the physician tends to overlook poliomyelitis during the initial phase, and he often tends to accept it too lightly when he is confronted with the sudden onset of paralysis. In attempting clinically to distinguish radiculo-neuritis from poliomyelitis, he stresses the absence of an invasion period in radiculo-neuritis; the gradual onset of paralysis often found in this condition as distinct from the sudden onset in poliomyelitis; the days or weeks of the phase of extension of the paralysis in contrast with the brief period in poliomyelitis. The diffuse symmetrical type of paralysis without complete loss of power in radiculo-neuritis

differs from the localised irregular but intense paralysis of poliomyelitis, and the diffuse abolition of reflexes in radiculo-neuritis contrasts with the localised abolition in poliomyelitis. There is occasional disturbance of sensation in radiculo-neuritis, sequelæ are absent and the outcome of radiculo-neuritis is invariably favourable in contrast with that of poliomyelitis.

Smadell, from the Department of Virus Diseases of the Army Medical Services School, Washington, discussed the laboratory aspects of differential diagnosis in acute poliomyelitis. During the initial febrile episode of the diphasic type of poliomyelitis, the spinal fluid may show increased cellular content or increased proteins. In 90 per cent of the cases abnormalities appear in the fluid with the onset of involvement of the central nervous system. Some of the remaining 10 per cent would show deviations from normality if subsequent examinations of spinal fluid were undertaken. The cells are mainly lymphocytes after the first day or so of nerve involvement and number usually between 12 and 100 per cubic millimetre. They rarely reach a level of 1,000, which would be suggestive of lymphocytic chorio-meningitis. The fluid protein is moderately elevated, but remains well below the level usually found in the Guillain-Barré syndrome. Occasionally a slightly elevated protein content is the only abnormality in the fluid from classical cases of paralytic poliomyelitis. There has been consistent failure to isolate the virus from spinal fluid. Recovery of the virus from nasopharynx or stools is as yet too difficult to be of clinical value. A demonstration of development of neutralising antibodies has no practical application in poliomyelitis and the complement fixation test is not yet developed beyond the experimental stage.

Non-paralytic cases tend to occur more frequently than paralytic cases. It is these non-paralytic cases which present the difficult problems in differential diagnosis, indeed most of them are erroneously diagnosed as aseptic meningitis or labelled pyrexia of unknown origin.

The virus of lymphocytic chorio-meningitis is the casual agent in approximately 10 per cent of patients presenting the syndrome of acute aseptic meningitis. In the laboratory diagnosis of this condition two serological procedures are available; they are complement fixation and neutralisation tests. The complement fixation bodies appear about the third week and persist for a few weeks or months before disappearing.

One of the occasional manifestations, especially in adults, of infection with mumps virus, is aseptic meningitis. The diagnosis is often facilitated by the history of exposure to this infection within the material incubation period. In patients presenting purely neurological symptoms and signs the laboratory is of help in diagnosis by the demonstration of complement fixing antibodies which appear during the latter half of the first week and increase during the next few weeks. These antibodies persist for an appreciable length of time.

The virus of herpes simplex is now regarded as a frequent cause of meningitis and even encephalitis. The infection is so common that most human beings will have some neutralising antibodies to this virus. In fatal cases the diagnosis

is confirmed by a post mortem finding of characteristic lesions in the nervous tissue and the recovery of the herpes virus from the brain tissue. In non-fatal cases the diagnosis may be made by the demonstration of the absence of specific antibodies for herpes virus in the acute phase serum, together with the appearance of these antibodies in the convalescent serum, and isolation of the virus from spinal fluid. Cases of meningitis or encephalitis due to herpes simplex comprise about 5 per cent of the total aseptic meningitis.

The probable diagnosis of those forms of meningitis due to infective mononucleosis is reached by demonstration of a positive Paul-Bunnell reaction in the patient's serum. The total and differential white blood cell count would supplement this diagnosis.

Infection with leptospira of Weil's disease or with leptospira canicola may be present in the guise of aseptic meningitis with fluid findings identical with those of virus infection. In either case a diagnosis is reached by the demonstration of specific antibodies in the serum of a convalescent patient previously without these antibodies.

The diagnosis of the condition from early bacterial meningitis, especially tuberculous or influenzal, is sugested by a demonstration of lowered sugar content of the cerebrospinal fluid in the bacterial infection as distinct from the normal sugar in virus infection.

Casals, from the Rockefeller Institute, described a complement fixation test for poliomyelitis. A specific antigen has been prepared which reacts with sera from different animal species, including mouse, rhesus monkey, chimpanzee, and man. In nearly all human cases of poliomyelitis a positive complement fixation occurs but some individuals with no history of poliomyelitis also react positively. Thus the range of practical application of this complement fixation test is as yet undetermined.

Salk, from the University of Pittsburg, discussed immunological classification of poliomyelitis viruses. Poliomyelitis virus was first discovered as long ago as 1909. In 1931 a second variety was identified and in 1949 the existence of a third variety was confirmed. These three varieties are known respectively as Brunhilde, Lansing and Leon. One hundred strains were available for typing. Of these, 85 were of Brunhilde type; 12 were of Lansing type and 3 of Leon. These strains that were examined were selected because they were available rather than for any other consideration. The results of this typing are therefore to be interpreted with caution; 68 strains were obtained from paralysed patients, 26 were from non-paralysed individuals, and the origin of the other six strains is uncertain; 34 of the strains were from fatal cases. For the present it would appear that only these three antigenic types of poliomyelitis virus need be considered.

Wilson, of Ann Arbor, discussed the management of respiratory insufficiency. He listed the three variations of respiratory failure, any one, two, or all of which may appear in the same patient. Firstly, respiratory failure due to paralysis of the primary respiratory muscles, the intercostals and the diaphragm. This type

is treated symptomatically by use of a cabinet respirator. Secondly, respiratory failure due to pharyngeal paralysis, where secretions or vomit continually interrupt the air-way. In this form treatment is less simple, but can be effective and life-saving. Thirdly, respiratory failure due to central disturbances difficult to localize. The respiratory, vaso-motor, and vagal centres of the brain are probably all involved to some extent. In this variety, treatment is much less effective. Wilson has always advocated the early use of the respirator in respiratory paralysis to avoid fatigue. Thereafter, careful observation and management of the patient is required to avoid the "respirator-fast" condition; that is, inability to do without the respirator. The search continues for an apparatus which will allow the patient in the respirator to cough. A difficult problem for patients in these machines is to adapt themselves by submitting their own rhythm to that of the mechanical apparatus. This embarrassment is common to patients with pharyngeal paralysis or with bronchitis or pneumonia, and in those with disturbances of the respiratory centres. The recent advance in this direction has been the production of a so-called electro-phrenic respirator at the Harvard school. Here, direct electrical stimuli are sensitively controlled and intermittently applied to either the exposed phrenic nerve or to the skin over the phrenic nerve. This has been effective in producing a rhythmical contraction of the diaphragm, and seems to inhibit the patient's own respiratory efforts when these are abnormal and ineffective. Unfortunately it appears that this apparatus will not be generally applicable to patients with respiratory difficulty in poliomyelitis, but it promises to be life-saving in selected cases.

The nursing of patients enclosed in a cabinet respirator has always been difficult. Chest respirators have been developed which fit either all around the thorax and the torso or only over the anterior surface of the chest and abdomen, making contact closure along the sides. Their action is most efficient upon the diaphragm, but they have a minor effect as substitutes for the intercostal muscles. These devices allow access to the body at all times and even allow the patient to sit up. They are useful during the weaning of a patient from a cabinet respirator, for the prolonged care of moderately paralysed patients, and especially in a tracheotomy case.

The rocking bed is a development of the old technique for artificial respiration by rocking a man on a plank across a barrel so that the abdominal contents by their weight alternately push and pull on the diaphragm and so produce a tidal movement of air. The rocking bed is of help in weaning a patient from a cabinet respirator.

The value of intermittent positive pressure over the mouth and nose is well known to anæsthetists. It is undoubtedly useful for short periods to allow of nursing attention to the remainder of the body, but it has the outstanding disadvantage of denying access to the upper respiratory tract. A blister or dome on the front of the tank respirator by which intermittent positive pressure can be applied enables the same nursing procedures to be carried out, without actually

contacting the patient's face with a mask. In this way it has obvious advantages.

The chief measures in nursing the patient with pharyngeal paralysis are postural drainage and aspirating or suction apparatus. In some patients neither procedure is sufficiently effective, and tracheotomy may be the only solution to by-pass the obstruction. Where good nursing facilities are available, tracheotomy can be avoided in most cases of pharyngeal paralysis. Recovery from pharyngeal paralysis is usually rapid and often complete. Tracheotomy is the only treatment available for the rare laryngeal abductor paralysis.

Seddon, of London, discussed reconstructive surgery of the upper limb. He provoked some discussion by his statement that "provided that good treatment has been given throughout, it may safely be assumed that no muscle remaining completely paralysed after six months will show any useful recovery." This statement was not uniformly accepted. Arthrodesis, being irrevocable, should not be lightly undertaken. He stressed the importance of pressing on with tendon and muscle operations as quickly as possible after the state of permanent paralysis has been reached, as some degree of restoration of function will improve the patient's chance of occupation and diversion.

Cobb, of New York, discussed the correction of scoliosis. He stressed that every poliomyelitis patient should have the spine checked for possible scoliosis whenever examined for any orthopædic condition, and at least every six months until sixteen years of age or a minimum of ten years from onset. Some poliomyelitis curves will increase to a severe degree in spite of all forms of treatment. He suggests that massive and mature spine fusion is the only method at present known of obtaining the significant amount of correction in a poliomyelitis scoliosis, and some which are increasing can be stopped only by spine fusion. This fusion should not be done too early; the optimum age is often 14 to 16 years.

Bennett, from Georgia Warm Springs Foundation, described the role of physical medicine in poliomyelitis. Modern treatment of the after-effects of poliomyelitis is based on a series of treatment steps ending only when the patient has recovered maximum function and capacity. The treatment guides the patient through the phase of acute illness, through the months necessary to restore co-ordinated strength, and assists him to make an adequate adjustment to his ultimate physical handicap.

Howe, from the Department of Epidemiology of Johns Hopkins University, reported upon antibodies and immunity to poliomyelitis. It is possible to identify a high serum antibody titre with immunity in the chimpanzee. The minimum level which secures immunity has not yet been ascertained. It is possible to produce active immunity in the chimpanzee by oral administration of vaccine but the application of this method to humans is not without risk. The disease produces natural immunity at a cost of approximately one paralytic accident in one hundred immunizations. It is true that the risk of death or severe disability increases with age and there is no doubt that poliomyelitis is now affecting a

relatively older age group than it did years ago. Should this advance into older age groups continue, it may become worth the risk to attempt vaccination of humans.

Ritchie-Russell, of Oxford, drew attention to factors which influence the clinical course of poliomyelitis. There appear to be at least two groups of such factors. Firstly, the group operating several days before there is any sign of infection, which may conceivably give the virus special facilities for reaching one part of the nervous system such as may occur after tonsillectomy, local injury or possibly injection. Secondly, factors of which physical activity seems most important, operating after the major illness has begun and virus has entered the nervous system. The association between tonsillectomy or local injury in poliomyelitis is firmly established. The position with regard to injections, especially immunising injections, is not so uniformly accepted.

Physical activity of any kind soon after the onset of the illness has an unbelievably harmful effect in some patients. In practice complete rest is advisable but often this is taken too late and no subsequent measure can limit the spread of paralysis. Complete rest and adequate sedation are desirable at this stage. The possibility of the harmful effects of a frightening, and sometimes exhausting ambulance journey to hospital has to be considered.

Bradford-Hill, from the London School of Tropical Medicine, reviewed inoculation procedures as a provoking factor in poliomyelitis. Evidence has accumulated that inoculation procedures may play a small part in the production of paralytic attacks of the disease. The evidence consists largely of the observation that in recently inoculated children the limb of the injection is the site of paralysis more frequently than the un-inoculated limbs and more frequently than is the case in other children not recently inoculated. It has been suggested that the effective reason in inoculation may be to localise the paralysis in the limb of injection in a child already incubating poliomyelitis. There is some evidence that the combined diphtheria and pertussis vaccine may be more liable to cause paralysis than diphtheria, and the pertussis vaccine alone has been blamed too. Questions very difficult to answer have been raised by these observations. For the present it appears desirable to suspend immunization during the months of prevalence of poliomyelitis, and at other times to give the antigens subcutaneously, rather than intramuscularly.

Gear, of the South African Institute of National Research, reviewed the extra-human sources of poliomyelitis. Recent epidemiological evidence suggests that there are, at least, one hundred non-paralytic or silent infections of poliomyelitis for every recognised case with paralysis. It is not surprising then to find large amounts of virus in the sewage of affected cities and this is probably a true reflection of the incidence of infection in the particular community. Investigation of sewage disposal indicates that the virus may persist in sewage for a long time and survive certain stages of sewage purification. It is probable that the virus is present in the final effluent of most sewage works during an epidemic. These effluents may contaminate the rivers and streams into which they flow, and, in

turn, may contaminate water supplies. These theoretical possibilities were examined during a recent epidemic of poliomyelitis in Johannesburg. As a result there was little evidence forthcoming to incriminate a properly designed and supervised sewage purification plant in the spread of poliomyelitis. No outbreak of poliomyelitis has, with certainty, been traced to contamination of vegetables or fruit by sewage, although the circumstances in a few outbreaks have been suspicious.

Evidence has been produced to incriminate water supplies in spreading poliomyelitis and several times the virus has been demonstrated in polluted streams. Epidemiological studies have not confirmed that infection of water supplies has been responsible for any widespread epidemic of poliomyelitis. The evidence attributing epidemics to swimming-bath water is open to doubt. There is less doubt, however, that the strenuous exercise in swimming may have the effect of precipitating a paralytic attack in individuals who would otherwise have had a non-paralytic attack.

Various species of flies have been shown to be infected with the virus and these flies may be responsible for the transmission and the maintenance of infection in a region. Campaigns against flies have not been found to lessen the incidence of poliomyelitis. There are several diseases in animals resembling poliomyelitis in man, but no animal reservoir of the virus of human poliomyelitis has yet been found.

Modern hygiene and sanitation greatly diminish the chances of an infant being infected with the virus of poliomyelitis in the early years of life. For lack of early immunising infections communities advanced in hygiene are liable to epidemics of poliomyelitis. On the other hand, in primitive communities where the infection is endemic and where circumstances such as the prevalence of flies and the pollution of water favour its wide spread, most infants are infected early in life. Such communities are not liable to epidemics of poliomyelitis. Extra human sources probably play an important part in spreading the infection amongst people who live under relatively primitive conditions.

Francis, from Ann Arbor, described the distribution of poliomyelitis virus in a community. Formerly stool culture was used for this investigation, but latterly the presence of Lansing antibodies was regarded as indicative of infection. The irregularities in distribution of cases of frank disease in a large-scale geographic area or in the more limited confines of the city have been emphasised repeatedly. The impression has been maintained that poliomyelitis infection is widely and uniformly distributed through the general population, with certain differences between urban and rural populations.

Using the presence of virus in stools as an index, there is a growing number of reports illustrating the frequency with which infection involves the apparently healthy members of the family of a recognised case. Although it is particularly high among children—up to 90 per cent, a considerable number of adult members may also be affected. Probably the common circumstance is that a good proportion of the group becomes infected almost simultaneously, for when specimens

are obtained promptly after the disease is recognised, the virus has already become established in the household contacts of the patient. It strongly suggests a common source of infection of the group, and that out of it a case of paralytic poliomyelitis develops, rather than indicating that the case is responsible for the infection of the others. Multiple cases in families occur most frequently within three or four days of each other, and unless a short incubation period is uniformly postulated the intervals would represent differences in the incubation period. It is emphasised that the majority of the persons in these family groups from whom virus is recovered are not passive transient carriers, but are true cases of subclinical infection in whom virus is propagating and who generally continue to excrete virus for three to eight weeks. In an effort to estimate the amount of virus excreted, it was found that one one-hundred-thousandth part of a gramme of stool was infective for a monkey.

In investigating the contacts, household and extra-household, of a frank case of poliomyelitis, it was found that 74 per cent of the household contacts were excreting virus as compared with 39 per cent of the extra-household contacts. This suggests that the virus is not evenly disseminated, but tends to be selective with the highest concentration in those families in which the frank cases of disease arise. Similar studies in boys' camps demonstrated that intimate groups are rapidly infected while adjacent ones, using the same eating and recreational facilities, may remain uninvolved. These interpretations seem to emphasise that clinical, especially paralytic poliomyelitis, is the accident of, or is incidental to the virus infection.

Paul, winding up the Conference, reviewed the present concept of this disease, originally regarded as sporadic, as now that of a highly contagious condition. The disease in which the original picture was thought to be limited to acute paralysis, is now regarded as one in which only one in a hundred or so of those infected becomes paralysed. The condition originally thought to be rare is now regarded as extremely common and one which has been a periodic scourge in Western Europe, the United States and Australia.

The Conference revealed the enormous amount of work which is being done on the virology and pathology of the disease. It was disappointing from the therapist's point of view to realise that there is still no specific remedy for the condition. It was interesting to hear it suggested that the increased susceptibility to, or the severity of paralytic poliomyelitis in the following conditions might be related to the adrenal cortical stimulation which accompanies each one; pregnancy in women and mice, excessive fatigue during the incubation period in monkeys and man, and probably following the surgical procedure of tonsillectomy in man and monkeys, and possibly following other operations which appear to result in similar incidences of severe poliomyelitis.

There is much encouragement to be derived from the results obtained by well-directed physiotherapy in paralytic cases. There is much to be said for planning towards adequate physiotherapy for this condition.

A better concept of the epidemiology of the disease and a better appreciation

of the proportion of immunes in the community may lessen the terrors of the condition.

The discovery that poliomyelitis virus can be grown in non-nervous tissues opens a very wide field. The attenuation of the virus by successive transfer in tissue cultures provides hope of the production of non-virulent strains of the virus, and these may ultimately be used to provide an immunising agent for human beings. Therein lie grounds for hope that at least the end of the beginning has been reached in the study of the epidemiology of the disease.

The opportunity to attend this Conference was provided by a grant from the Northern Ireland Hospitals Authority.

REVIEW

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Traumatic Dislocation of the Shoulder Joint

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Paper read before the Ulster Medical Society in April, 1951

TRAUMATIC dislocation of the shoulder joint is not a simple injury. It is followed by complications in about 50 per cent of cases, which delay recovery by months or which may even produce permanent loss of function of the joint.

INTRODUCTION.

It is interesting to recall some historical aspects of the condition, as it seems clear that man must have suffered from the dislocation for countless ages before he finally embarked on his experiment of civilization. It is quite certain that in Hippocratic times the lesion was well known and its treatment well established. Whether the section in the Hippocratic Corpus was the work of Hippocrates himself or of others is unknown, but the passages on shoulder dislocation have become classical and immortalised by the Hippocratic method of reduction. The author of this section recognised anterior luxations, but doubted the observations of his contemporaries who described superior and lateral forms. Reduction was effected by traction on the arm, with counter-traction in the axilla by the hand, heel or shoulder of the operator or his assistant. For more resistant cases an apparatus was devised and traction applied mechanically.

Recurrent dislocation was also recognised and was treated by placing the cautery over the front of the axilla, allowing it to burn through skin, fat and down to the tendons of the joint. This was done with the knowledge that the scarring which resulted would contract and shorten those structures, the looseness of which permitted the recurrent luxations. Over 2,000 years have passed, and yet Watson-Jones re-stated this ancient belief only a few years ago when he suggested that, in treating recurrent dislocation, operative techniques were relatively unimportant, provided they were sufficiently traumatic and sufficiently bloody to produce strong fibrous tissue where it was specially needed. From Hippocratic times onwards, we read of shoulder luxations in the writings of Paul of Ægina, Roger of Palermo, Avincenna and others, but, up to the middle of the nineteenth century, treatment varied in no essential way from the Hippocratic method. Surgeons did, however, vie with one another in producing more and more elaborate windlasses, racks and apparatus generally to effect reduction of the dislocations. The illustrations of apparatus for this purpose from the works of Paré, de Cruse, Videus and Scultetus published in the seventeenth century,

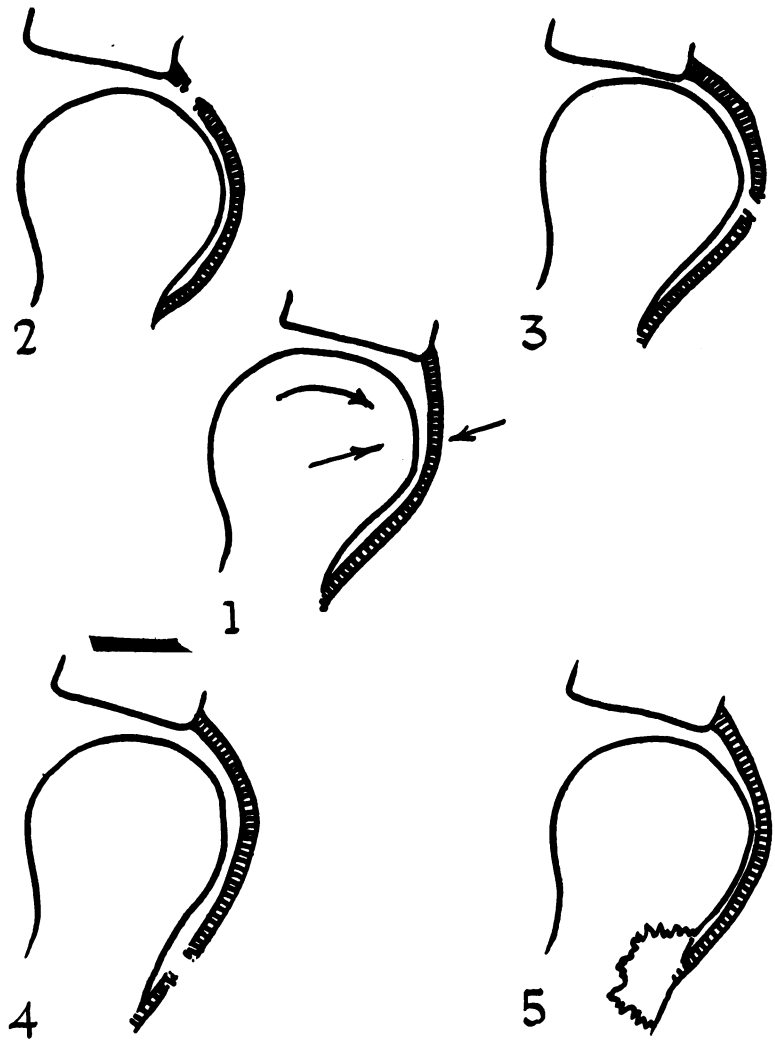
make one admire the fortitude of the sufferers and raise doubts as to which was the worst, the dislocation or its treatment.

In the nineteenth century, surgeons began to consider the possibility of effecting reduction with minimal trauma, as they had become impressed by the irreparable damage which mechanical enthusiasm was inflicting on joint tissues and indeed on other structures far removed the joint. One of these was Theodore Kocher of Berne who, in 1870, described a method of reduction based on the principle of tiring out the muscle holding the humeral head in its dislocated position, i.e., the subscapularis, by slow gradual external rotation of the joint. For long the Kocher method remained the standard practice in every country, but in recent years surgeons have returned to simple traction — counter-traction methods and general anæsthesia now make the reduction a simple procedure. But has the march of time resulted in better end results and, admitting that our present methods of reduction are less painful and less traumatic than those of our forebears, is it the trauma of dislocation which is the cause of post-reduction disability, or must we revise our standards of the technique of reduction?

In an attempt to answer these questions I have analysed all the cases of shoulder dislocation treated in the Royal Victoria Hospital Fracture Service between 1943-1949 (257 cases). I have been able to trace 147, and their review forms the basis of this paper. There were 92 males and 55 females. The left shoulder was affected in 72 cases and the right in 75. The average age was 46 years, the youngest being 18 and the oldest 81. There were no deaths in the series. In those cases which recovered without complications, functional return was rapid, and within five weeks, on an average, a full range of shoulder movement had taken place. Most cases returned to full work a few weeks later. This was the rule in 78 cases or 53 per cent, but in the remainder, 69 cases or 47 per cent, there were complications which caused disability often for many months or even led, in a small number, to permanent uselessness of the joint.

The Dislocation.

It was often difficult to be certain, from the patient's history, of the exact mechanism of the dislocation. After all, the shoulder dislocates at an unguarded moment, the patient falls, and the first thing he knows is that his shoulder is painful and he cannot use his arm. He is immediately concerned with relief from pain and has no scientific regard for the exact position his arm occupied at the moment of injury. In general it was considered that 75 per cent resulted from falls on the shoulder, elbow or hand, i.e., violence by pulsion, 20 per cent from sudden traction on the arm, and 5 per cent from muscular violence. In surgical text books it is still customary to divide shoulder dislocations into subglenoid, subcoracoid, subclavicular, subspinous and luxatio-erecta types. There is no practical point in this, since almost all traumatic dislocations are of anterior type, the posterior type is a rarity, and luxatio-erecta is such a curiosity that many surgeons of mature years have never seen a case.



Diagrams representing the shoulder seen from above. The anterior part of the capsule is shown and its relationship to the humerus and to the front of the glenoid.

Fig. 1—A normal shoulder showing the capsular mechanism by which the head is prevented from forward displacement on the glenoid.

Fig. 2—Showing the common type of tear in the capsule. When it heals functional return is full, but when it does not heal the ground is laid for recurrent dislocation.

Fig. 3—Showing the type of capsular lesion which causes a "frozen shoulder."

Fig. 4—Showing the type of capsular lesion which gives rise to a "ruptured supraspinatus syndrome."

Fig. 5—Showing the "pull-off" by the capsule of the greater tubercle of the humerus. This demonstrates that fractures of the great tubercle are due to traction from the pull of the capsule.

In the present series, 145 cases were anterior and only two were posterior. It is therefore with anterior dislocation that we are concerned, and the subglenoid, subcoracoid and subclavicular groups are only positional variations of the same injury, being dependent on the degree of tearing of the shoulder capsule and the violence of the dislocating force.

The Capsular Lesion.

The head of the humerus is normally prevented from forward displacement by the "buffering mechanism" of the anterior part of the capsule and its reinforcing gleno-humeral ligaments. Along with this the glenoid is slightly deepened by the presence of the glenoidal labrum. Above, the capsule, in the adult, is fused with the tendon of the supra-spinatus and the upper parts of the tendons of the infraspinatus and subscapularis to form the musculotendinous cuff of the shoulder. Degenerative changes occur in this cuff as age advances so that minor injuries can cause it to rupture in those of advancing years. The function of the cuff is to tense the head of the humerus against the glenoid during shoulder movements, so that a stable "hinge" can be presented to the deltoid to effect elevation of the arm at the shoulder joint.

The medial part of the "buffer" is the capsular attachment to the glenoid, glenoidal labrum and the neck of the scapula. In external rotation, the front of the capsule gradually becomes taut and forces the humeral head backwards on the glenoid. When all capsular slack has been taken up, any further movement of external rotation or of hyperextension will cause acute strain to the anterior part of the capsule and eventual rupture of it, either in its substance, or more commonly at or close to its medial or lateral bony attachments. Only when this has occurred can anterior dislocation of the shoulder take place. It it therefore suggested that the complications which follow shoulder dislocation are due to the capsular lesion — and the site of the lesion pre-determines the specific complication — or else result from the pressure of the humeral head, after it has escaped from the joint, on the neuro-vascular structures in the axilla.

The capsule tears most commonly at its glenoidal attachment and it is firmly believed by the author that the rapid return of function, without apparent complication, in any particular case proves that the capsular lesion was at the glenoidal attachment and nowhere else. Tears in other parts of the capsule or the humeral attachment invariably give rise to complications; a glance at the accompanying diagrams shows the co-relation between the complications and the different sites of the capsular lesion.

Reduction of the Dislocation.

Most of the cases were reduced by manipulation under anæsthesia within twelve hours of the accident. The methods used appeared to have no bearing at all on the end results and any way all were reduced without difficulty. There were, however, 14 exceptions to this rule :—

5 cases—were reduced, 16, 17, 20, 21 and 25 days respectively after the accident by manipulation under anæsthesia, and all recovered without complications.

2 cases—were reduced 10 days after the accident by manipulation but were complicated by deltoid paralysis, though eventually there was full recovery of function in both.

2 cases—were left unreduced as they presented 10 and 11 months respectively after the accident. Both gave good, though not perfect, results and there was no pain.

5 cases—were reduced by open operation from 1 to 4 months after the injury, manipulation having failed. All recovered without complications.

In all these cases of late reduction, whether by manipulation or operation, it it noteworthy that no serious complication was seen. Considerable force was usually necessary and this fact alone supports my belief that the complications which follow shoulder dislocations are due to the dislocating force and not to the reduction, i.e., the complications are already present when the surgeon first sees the case, though he may not recognise them at the time.

Early and gentle reduction by manipulation, and always under anæsthesia, will give the patient the best possible chance and, if the reduction is followed by a period of complete rest for several weeks to allow healing of the capsular lesion, there will be no grounds for recrimination should a perfect result not take place. Rough or rapid manipulation, or manipulation without anæsthesia, should be things of the past—the capsule is already torn and the patient in pain; don't rupture it further, and most certainly do not cause the patient additional suffering.

COMPLICATIONS.

Recurrent Dislocation	...	13 cases
"Frozen Shoulder"	...	14 ,,
"Ruptured Supraspinatus"	...	7 ,,
Fractures of the Great Tubercle	...	16 ,,
Nerve Lesions	...	14 ,,
Other Complications:—	...	5 ,,
Fractured Acromion ... 1		
Colles' Fracture ... 1		
Fracture Neck of Humerus 3		

No vascular complications were seen in this series.

Recurrent Dislocation.—This complication occurred in 9 per cent of the series or 13 cases. With the exception of one female case, all were males and all were under the age of 30 years at the time of the first dislocation. In the entire series of 147 cases, only 38 were under the age of 30 years and so one-third of all cases under 30 years became recurrent. This is a big percentage and worthy of note and thought. The least number of re-dislocations was 3, the greatest 34, and the average 12, before patients sought advice. Some authors believe that

a special injury must have been sustained at the first dislocation to lay the ground for recurrence, and yet in this series all the common types of injury were found—fall on the hand, fall on the shoulder, a twisted arm, swinging a motor car starting handle, etc.

I believe that the cause of recurrent dislocation is simply incomplete healing, or healing with deficiency, of the tear of the anterior capsule at or close to its glenoidal attachment. If this is true it is wrong to encourage activity early in an apparently uncomplicated dislocation after reduction, for it would be unreasonable to expect the glenoidal humeral attachment to heal in a few days. Rather, complete rest to the shoulder should be insisted on for several weeks and this should be specially stressed in that group most liable to recurrent dislocation, namely, males under the age of 30 years. I believe that if this suggestion were put into rigid practice the number of recurrent dislocations would be greatly reduced in any future review of this subject.

If a weakly healed or unhealed glenoidal capsular lesion results from the primary dislocation, any simple extension or external rotational strain is sufficient in the future to allow the humeral head to slip forwards over the front of the glenoidal rim, thereby producing a further dislocation. Once recurrence starts it becomes easier each time for re-dislocation to take place, since each displacement further stretches the already stretched glenoidal part of the anterior capsule.

In 1870 Broca and Hartmann described fully the changes in the shoulder joint in recurrent dislocation. In 1932, Bankhart drew surgeons' attention once again to these changes and with so much force that many to-day refer to the "Bankhart Lesion." Four changes were described by Broca and Hartmann and by Bankhart.

1. Detachment of the glenoidal labrum from the glenoidal rim.
2. Arthritis localised to the antero-inferior aspect of the glenoid.
3. A hatchet-shaped humeral head, due to deficiency of the postero-lateral aspect of the head or its articular surface.
4. Cartilaginous loose bodies in the joint.

All these abnormalities are unimportant compared to the primary lesion of non-healing of the glenoidal part of the anterior capsule, for, after successful repair of it by operation, the other changes still persist and yet the function of the joint returns to normal or near normal in most cases. They are the effects of dislocation, either originally or as the result of re-dislocation, not the cause of recurrences, and all have been seen in this present review in cases with fully functioning shoulder joints where no recurrence of the primary dislocation had occurred.

Many dozens of operative procedures have been devised for the cure of this complication and all have had their successes and, of course, their failures. Those who believe that detachment of the glenoidal labrum is the important cause of recurrence will employ the Bankhart technique, and, after opening the shoulder joint from in front, will replace the labrum and hold it in position on the glenoidal rim by sutures or staples. Those who subscribe to a weakly healed or unhealed glenoidal capsular lesion will carry out the Putti-Platt operation and repair the

deficiency by "vest over pants" overlap of the glenoidal capsule and the subscapularis muscle close to the glenoid. In the twelve recurrent cases of this series, no treatment was advised in three as symptoms were not disabling and recurrences infrequent. Surgery was performed in the other nine. Three of these had already been operated on by the techniques of Nicola, Henderson and Clairmont respectively, but without success—they were cured by further operation. The Bankhart operation was carried out in three cases and in the remainder the Putti-Platt technique was employed. All were successful and the average loss of movement afterwards was minimal for full elevation, but external rotation was usually considerably reduced. All the cases were able to return to full pre-accident duties.

My own reaction to these results is that almost any surgical technique will be followed by success if the operation produces strong fibrous tissue at the junction of the anterior capsule and the glenoid rim and effectively and permanently limits full external rotation of the shoulder joint.

"Frozen Shoulder."—This complication was seen in fourteen cases or 10 per cent and there were roughly twice as many females as males. The term implies complete stiffness of the shoulder following an injury, the underlying cause of which is a capsulitis or inflammatory reaction in the damaged capsule starting in the neighbourhood of the tear and rapidly spreading to other parts. The condition is slow to settle down and anytime up to 12 months may elapse before shoulder movement returns to normal. In a few cases, permanent, though incomplete, loss of full shoulder movement is the end result.

The symptoms produced are pain and loss of shoulder movement. The pain is often continuous to begin with and worse at night, but later it is usually only felt on attempted movement. It is diffuse over the shoulder in the early stages, but in the later stages it is felt at or about the insertion of the deltoid muscle. The shoulder stiffness for some time is protective since it relaxes under anæsthesia, but later, when the inflammatory process has abated, it is due to adhesions in and around the capsule and is therefore still present under anæsthesia.

In the early stages of this complication the shoulder is irritable and demands rest in its treatment by supporting the shoulder with the arm in a sling, though in the later stages return of function can be hastened by active exercises aided, if need be, by manipulations of the joint under anæsthesia. It is difficult to know when to start movement though the result of examination under anæsthesia is a fairly accurate guide. It must be stressed that for a while nothing can be done to hasten recovery; indeed it is perfectly clear that well-meaning, though ill-advised, attempts at treatment by physiotherapy often make the patient's discomfort much worse and may well prolong the acute inflammatory phase in the shoulder capsule by weeks or even months.

In the 14 cases of this series, 11 made full functional recoveries in 10 months with extremes of 6 months (the shortest time) and 16 months (the longest time). Three cases were left with permanent stiffness of the shoulder amounting to a restriction of full shoulder movements by about 20 per cent.

Ruptured Supraspinatus.—Seven cases suffered this complication, i.e., 5 per cent of the series. Six were females and one male, and all were over 60 years of age, i.e., were well into the years where degenerative changes in the musculotendinous cuff of the capsule are to be expected in the normal course of events. All were treated conservatively by rest in an abduction splint for many weeks and, with one exception, the end results were extremely poor, almost complete loss of shoulder movement being the rule.

Possibly the term "ruptured supraspinatus" is a misleading one since it is clear that the tear is seldom confined to the supraspinatus alone but practically always extends into the subscapularis in front and the infraspinatus behind. Indeed in some cases, as recent operations have revealed, the "rupture' may be a complete avulsion of the entire upper and front part of the capsule from its bony attachment to the humerus. The condition should be thought of at once in any patient who, after reduction of the dislocation, is quite unable to elevate the arm whilst the deltoid can be felt contracting under the examiner's hand. If the patient is unable to hold the arm in the elevated position when so placed by the examiner and it drops uselessly to the side, then the diagnosis is certain. This is an excellent test and the "drop-arm" sign is diagnostic of this unfortunate complication.

So poor have been the results of conservatism that nowadays operative treatment is advised in every case showing no return of the power of elevation after a few weeks of conservative measures. This consists in excision of the acromion process, opening the subdeltoid bursa and suturing the torn capsule back to the humerus. A period of five weeks' rest follows and then active exercises are started and encouraged by hot packs and assisted movements.

Fractures of the Greater Tubercle of the Humerus.—Sixteen cases or roughly 11 per cent showed this complication. Three-quarters of them involved the superior facet only, and the other quarter the whole tubercle. This complication is to be looked upon as a traction fracture, the bony fragment being pulled away from the humerus by the taut capsule. Most of these cases showed little or no displacement after the reduction of the dislocation, but several, on pre-reduction films, showed quite a marked displacement. It is evident that a pre-reduction X-ray is necessary in all cases to be satisfied of the question of separation of any fracture of the great tubercle.

Most cases were treated with the arm supported in a sling with an axillary muff holding the humerus in slight abduction. Those with wide displacement in the pre-reduction X-rays were treated with the arm in an abduction shoulder splint for three to five weeks. In this connection it may be worthy of note that, should a splint be thought necessary, it is inadvisable to apply it straight away, as there is a very decided risk of re-dislocation taking place within the first few days of reduction. Active exercises were started within five weeks of the injury and in all cases recovery of movement was full. The period of incapacity usually lasted from four to five months as shoulder stiffness was present in most cases after the fracture had united.

Other Fractures.—In this series there was one case of fracture of the acromion process, one case of Colles' fracture, and three cases of fracture of the neck of the humerus (3 per cent). These last cases were really examples of dislocation fractures. All occurred in elderly females, all were reduced by manipulation but all gave poor shoulders from a functional point of view, as the head of the humerus was in each case reduced upside-down so that early traumatic osteo-arthritis developed.

Nerve Lesions.—Nerve complications occurred in 14 cases or 10 per cent of the series. Two were lesions of the posterior cord of the brachial plexus with clinical evidence of a dropped wrist. The other 12 were lesions of the circumflex nerve producing a paralysis of the deltoid muscle.

There is nothing to be learnt from their analysis, as recovery was full in most with return of nerve function in about ten weeks, provided "a frozen shoulder" was not an additional factor. Some difficulty was experienced by young doctors in hospital in being sure whether they were dealing with deltoid paralysis or rupture of the supraspinatus. There should be no difficulty at all, since in ruptures of the supraspinaus, as previously pointed out, the deltoid can be felt contracting firmly under the examiner's hand when arm elevation is attempted even though no movement is in point effected. On the other hand, with circumflex nerve paralysis, no matter how hard the patient tries to elevate the arm, no contraction of the deltoid can be felt by the examining hand.

The two cases of dropped wrist were simply treated by support to the wrist in a cock-up splint and passive movements daily to prevent stiffness of the fingers and wrist joints.

All the cases of deltoid paralysis were treated by support to the shoulder in an abduction splint. I now wonder whether this is really necessary or whether the splint has any real influence on the recovery of function of the deltoid. For long it has been taught that a paralysed muscle should not be allowed to be overstretched and those using an abduction splint feel that they are relaxing the deltoid muscle and preventing it being stretched during the recovery of the nerve lesion. The position of rest for the arm, however, is not with the arm at right angles at the shoulder but it is with the arm at the side. I now do not believe that the use of the abduction splint for a paralysed deltoid muscle has any other effect than to cause extreme discomfort to the patient.

About half the cases of deltoid paralysis were treated by various physiotherapeutic measures including galvanic stimulation, and the other half received no treatment at all. Those treated by physiotherapy did not seem to recover function any earlier or any more completely than those cases left to their own devices.

Conclusions.

1. Given gentleness of reduction of dislocations of the shoulder under anæsthesia, the complications are the result of the injury and not of its treatment.

2. Complications occur in almost half the cases, the main factor concerned in their production being the capsular lesion.
3. Recurrent dislocation occurs in 30 per cent of shoulder luxations under the age of 30 years, but it can be prevented or at least minimised by ensuring that sufficient rest is given the shoulder after reduction, so as to allow the capsular lesion time to heal.
4. Rupture of the shoulder "cuff" is the most serious complication of all and, as conservative treatment gives such poor results, operative repair should be advised in all cases.
5. Fractures of the greater tubercle, nerve lesions and capsulitis ("frozen shoulder") delay recovery by many months. Time and patience are needed in their management, but the ultimate outlook is good.

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The Emeritus Regius Professor of Physics in the University of Glasgow has considerably enlarged his book, which consists of short biographical sketches of medical men who have distinguished themselves in other ways than in the practice of medicine. Here are soldiers and scientists, poets, peers, politicians and philanthropists, ambassadors, administrators, actors and aeronauts, saints and criminals, several pirates and at least one Rosicrucian. This is indeed a fascinating volume. R. M.

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Pneumonia in Childhood

Treatment by Combined Penicillin and Sulphamezathine

By ROBERT J. YOUNG, M.D., M.R.C.P.(LONDON).

Royal Belfast Hospital for Sick Children

AFTER the neonatal period pneumonia and bronchitis rank as the principal killing diseases until the age of 4 years. Reviewing 178 cases of pneumonia in children of all ages treated with sulphapyridine Gaisford (1940) recorded an overall mortality rate of 8.4 per cent, but in those under six months of age there were 5 deaths out of a total of 11 cases, giving a mortality rate of 45 per cent. Smellie (1949) recorded a mortality rate of 28 per cent from bronchitis and pneumonia in infants under one year of age for the period 1946-1948. More recently Holzel and Wolman (1950) recorded 28 deaths among 76 infants (36 per cent) under six months of age. Over this age there were five deaths in 118 cases (4 per cent). In the present series there were no deaths in 45 cases, 12 of which were under six months of age. These results are extremely gratifying and it is considered that the series is worth recording.

CLINICAL DATA IN CHILDREN UNDER SIX MONTHS.

Pneumonia in the first six months of life causes most concern and in all published series this period has by far the highest mortality figures.

Predisposing Causes.—An analysis of the twelve cases in this age group shows the important part played by lesions of the upper respiratory tract in the ætiology of pneumonia in infants. In five cases coryza immediately preceded the pneumonia. In one case influenza in the parents was followed by pneumonia in their infant. Pneumonia developed in one child who had just recovered from an abscess on the buttock and in another child whose resistance had been lowered by gastro-enteritis four weeks previously. Two cases were from families in which the home conditions were deplorable.

Symptoms and Signs. — In one case the onset was by loss of appetite and vomiting. A cough, dry or loose, was the initial symptom in all other cases. This was followed in two or three days by increased respiratory rate. In most cases the alæ nasi and other accessory muscles of respiration were in action. In eleven cases crepitations or râles were audible in the chest. Six out of the twelve cases showed definite cyanosis. No child was apyrexial on admission to hospital and the admission temperature varied between 101°F and 105°F. Convulsions occurred in only one case.

The duration of symptoms before treatment was three days in eight cases, two days in two cases and one day in two cases.

The time taken for the pyrexia to subside following the commencement of therapy varied from two to seven days, the average time being five days.

Excepting one premature infant, who had been bottle fed and who relapsed on the eleventh and again on the twenty-ninth day but finally recovered, the duration of stay in hospital varied from eight to fourteen days.

X-Ray Findings.—The lung fields were normal in five cases and definite areas of consolidation were seen in only four cases. "An increase in vascular pattern at the bases," "severe bronchitis," and "hilar enlargement" were each seen in one case.

Tuberculin Tests.—A complete Mantoux series was done in eight children, and in all the tests were negative.

Clinical Data in Children Over Six Months.

From six months of age to one year there was little change in the natural history of pneumonia from that described above, except that listlessness and anorexia often preceded a cough. Two out of the four cases in this age group vomited occasionally. One child had a history of convulsions in infancy, but in spite of this there were no muscular twitchings or convulsions during the course of a pneumonia in which there was a high grade pyrexia. One child exhibited the typical features of mongolism and, true to type, it had always been "chesty" and had a ventricular septal defect. The youngest age at which antral infection played a part in lower respiratory tract infection was in a child of nine months in whom X-ray sinuses showed "infection of both antra especially right." Seven days after admission this child was apyrexial, the chest was clear clinically and there were no symptoms. Twenty-four hours later the temperature rose to 103°F and coarse crepitations were audible on the right side of the chest. In a further twenty-four hours crepitations and bronchial breathing were audible at the left base. X-ray of chest at this stage showed "Consolidation at left base and well marked bronchitis on right side." The infection finally responded to streptomycin $\frac{1}{8}$G. twice daily for three weeks. This child was in hospital for thirty-five days.

Over the age of one year the symptoms and signs of pneumonia are well known and only a few points of special interest need be mentioned. Coryza was still occasionally the precursor of pneumonia. A child aged one year and eight months suffered from recurrent head colds, was anæmic (Hb. 65 per cent), and had had a recent attack of influenza. Another child had left otitis externa and bilateral blepharitis and was in a debilitated condition just before the onset of pneumonia. In several cases influenza in a parent was followed by penumonia in the child.

One unusual case conformed to the debatable entity described as rheumatic pneumonia which occurs in just over 1 per cent of cases of acute rheumatism. This type of pneumonia is characteristed by a rather silent course. There is no chill, breathing is usually not embarrassed and cough may be absent or trivial. The physical signs include dulness to percussion, bronchial breathing and crepitations. A characteristic feature of these signs is their transient and sometimes migratory nature. A child aged eight years was admitted to hospital with a history of having had pain in the left wrist five days before admission. The pain

subsequently flitted to both knees and then to both hips. There were no chest symptoms. On admission the child was pale and thin, pyrexial, sweating, flushed and dyspnœic. Heart sounds were soft, there was an apical systolic murmur and a tachycardia but no appreciable cardiac enlargement. Bronchial breathing and crepitations were distinctly audible at the base of the right lung. An X-ray of chest (25. 9. 50) showed "Prominent hilar shadows and some inflammatory changes at right base suggesting a resolving pneumonia." In one week this opacity was less marked and had completely disappeared in a further week. The blood sedimentation rate (Westergren) was 65 mm. in the first hour. An E.C.G. on 29. 9. 50 showed "lowish voltage limb leads. PR intervals all of 0.2 seconds. Some prolongation of QT interval. Would fit in with an acute rheumatic state."

Diagnosis.

The rapid respiratory rate and the working of the alæ nasi and other accessory muscles of respiration, with or without cyanosis, suffice for the diagnosis in an infant. In general the younger the infant the less important are the physical signs, though adventitious sounds were present in eleven of the twelve cases under the age of six months.

In an older child the diagnosis may not be so obvious, but a hot dry skin and dyspnœa are strongly suggestive, and a cyanotic tinge is common, but not invariable. Careful auscultation is required in the child with no obvious respiratory distress or cyanosis, and then the diagnosis depends upon the proper appraisement of minimal symptoms and signs. Pneumonia in infancy and childhood may have an insidious onset. Cough is then the commonest presenting symptom but malaise, vomiting, diarrhœa, or a convulsion may herald the infection. In any ill child the possibility of pneumonia should be kept in mind. Crepitations, râles, or a small patch of bronchial breathing are the usual confirmatory signs.

X-ray appearances showed pneumonic patches of consolidation in twenty-five out of forty-five cases. Large opacities may clear radiologically in one week.

Prognosis and Assessment of Response.

The great recuperative powers of children and prompt and modern therapy justify an optimistic immediate and remote prognosis. It is now accepted that the leucocyte count has no prognostic significance.

Using the treatment detailed below in the 0-6 months age group (12 cases) immediate response was good in four cases and fair in seven. In one case there was no improvement, but later the illness responded to streptomycin. In the 6 months-1 year group the immediate response was good in two, fair in one and one responded only later and to streptomycin. In the 1-2 years groups (9 cases) immediate response was good in five and fair in four cases. In the 2-5 years age group (10 cases) immediate response was good in eight, and fair in two. In the 5-10 years age group (10 cases) there was a good immediate response in all cases except the case of rheumatic pneumonia.

Treatment.

Prophylaxis.—Detailed histories taken in this series confirmed that overcrowding and an inadequate diet predisposed to respiratory infection. Adults with coryza or influenza should avoid close contact with children. Where contact is unavoidable some form of mask should be worn. An adequately balanced diet in which emphasis is placed on the protective foods is the most important ancillary aid to the prevention of these infections. The protective foods are dairy-foods (milk, cheese, cream, butter, eggs and meat) and garden-produce (green-leaf vegetables, carrots, tomatoes and oranges). The catarrhal child with a second-rate respiratory mucosa should be given double the ordinary dose of cod-liver oil. Mackay et al (1946) found that anæmia was associated with an increased morbidity rate and concluded that the anæmia was nutritional and correctable by iron therapy. Green vegetables and some proprietary breakfast foods (corn flakes, whole bran, shredded wheat) are good sources of iron and during childhood some of these foods should be given daily.

Curative. — Combined penicillin and sulphamezathine therapy in the dosage shown below was used. Streptomycin was used in cases failing to respond to these drugs.

Treatment Scheme.

Age	0-6 months	6 mths.-1 year	1-2 years	2-5 years	5-12 years
Penicillin	1 c.c. Distaquaine twice daily for five days for all age groups.				
Sulphamezathine	Initial dose 0.5 G. and 0.25 G. four hourly for 5 days	Initial dose 0.5 G. Repeat in 6 hours and 0.25 G. four hourly for 5 days	Initial dose 0.75 G. and 0.5 G. four hourly for 5 days	Initial dose 1 G. and 0.5 G. four hourly for 5 days	Initial dose 2 G. and 1 G. four hourly for 5 days
Streptomycin	$\frac{1}{8}$ G. 12 hourly for 5 days	$\frac{1}{8}$ G. 12 hourly for 5 days	$\frac{1}{8}$ G. 12 hourly for 5 days	$\frac{1}{4}$ G. 12 hourly for 5 days	1 G. daily for 5 days

Oxygen therapy by means of an oxygen tent should be given to all cyanosed infants.

"Estopen," a new chemical derivative of penicillin, is reputed to have an exceptional affinity for the lungs. The aqueous suspension is administered by intramuscular injection. So far as the writer's experience goes it is not more effective than the scheme outlined above.

In cases failing to respond to penicillin and sulphamezathine aureomycin would now be used, though, because of the tendency of this drug to induce nausea and vomiting, it will probably give way to terramycin when the latter becomes more readily available. Using oral terramycin Swift (personal com-

munication) successfully treated fifteen children with lobar or broncho-pneumonia. "Subsidence of acute signs was rapid and fever disappeared in one to two days. An infant of nine months having failed to improve with penicillin and aureomycin, responded immediately to terramycin (50 mg./lb.)." Swift recommends an initial loading dose of 50 mg./lb. and a maintenance dose of 25mg./lb. of body weight six hourly for a period of four to eight days.

Summary.

A survey of all cases of pneumonia admitted to a ward at the Royal Belfast Hospital for Sick Children between 1st October, 1950, and 31st April, 1951, suggests that combined sulphamezathine and penicillin therapy can effect an improvement in the morbidity and mortality figures of pneumonia in infancy and childhood. Forty-five cases were successfully treated. There were no complications. A recurrence of pyrexia does not indicate a relapse provided that the child's general condition is improving. Recurrence of pyrexia with a deterioration in the child's condition should lead to a suspicion of lung abscess, empyema or otitis media.

These cases were under the care of Dr. T. Howard Crozier, to whom I am greatly indebted for permission to publish and helpful criticism. Thanks are due to Dr. Douglas Boyd for the reports on X-rays and to the Editor for advice on the preparation of this paper.

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REVIEW

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Senile Mumps With Suppuration

By J. H. Adams, M.D., and R. W. M. Strain, B.Sc., M.D., M.R.C.P.I.
Clifton House, Belfast

Mumps in the aged is a rarity, and so is parotid suppuration in this condition at any time.

The two patients about to be described were old women occupying beds which faced each other at the end of a long ward. One case became evident six days after the other.

They were nursed in the same ward behind screens, and there were no other cases among the other occupants of the room, all of whom were of about the same advanced age.

1.—Miss I. R., aged 80.

In August, 1951, this old lady developed acute left-sided cardiac failure with pulmonary œdema. Always of a depressive type, during this attack she became almost unmanageable. Under prolonged and profound sedation with morphine and hyoscine supplemented by the barbiturates, she made an unexpected recovery, but remained apathetic.

On 16th October she developed a painful swelling under the right jaw, and within a few days had the full collar swelling of mumps. Chloromycetin was given from the second day, but was discontinued after three days as there appeared to be no response to it. The parotitis subsequently appeared to be resolving in the usual way on the right side, but on the left it became very massive. She was then put on penicillin. She was unable to shut the right eye. The mass became fluctuant, and an abscess began to point behind the left ear. There appeared to be several loculi, and radial incisions were made into the anterior part of the gland. From the pus which drained freely from all of these a staphylococcus aureus was cultured. At this time her urinary diastase was 100 units, but she had no abdominal pain and the fæcal fat analysis was normal. The abscess gradually resolved. She did not develop any salivary fistula, and was once more able to shut the left eye normally. Nine days after the formation of the abscess her urinary diastase had fallen to 8 units. Exhaustion and debility were considerable at the end of this illness, and she died without any further cardiac manifestations on 17th November.

2.—Mrs. McG., aged 84.

This old lady had a hemi-plegia of several years' standing, and was confined to a cot bed on account of her tendency to fall out of an ordinary one. Cerebration was slow and her conversation reiterative.

Six days after the onset of mumps in the first case this patient developed a

typical attack. This ran a perfectly normal course. She had chloromycetin for four days. Resolution was complete and there were no complications.

She subsequently developed a further slow cerebral thrombosis from which she eventually died. There was nothing to suggest that her mumps had anything to do with the onset of her terminal illness.

While suppuration in epidemic parotitis is uncommon, it seems most likely that if it is going to occur, it will be in cases such as those described, where old age and its accompanying debility must be considerable factors in the predisposition to secondary infection.

REVIEW

A SYNOPSIS OF OPHTHALMOLOGY. By J. L. C. Martin-Doyle, M.R.C.S., L.R.C.P., D.O. (Oxon). (Pp. 246. 20s.). Bristol: Wright & Sons Ltd., 1951.

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I like his apt quotations here and there in the book, which is clearly written and well printed. It is a pity no coloured illustrations of fundi, etc., have been found possible as they are a tremendous help to students.

There are some points of treatment with which one might disagree but these are of a minor nature.

To sum up, one might say the book comes under the aids to ophthalmology series.

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PROCEEDINGS, 1950-1951

President: **Dr. H. Hilton Stewart.** ***Secretary:*** **Dr. J. H. D. Millar**

Friday, 28th October, 1950, at Claremont Street Hospital:

Miss Mitchell introduced Miss Van Thal, F.C.S.T., who gave a paper on speech disorders. Speech was defined as "the utterance of sound patterns composed of vowels and consonants." The physiology of speech was discussed in some detail, namely, the integration of respiration, phonation, resonance and articulation to produce the syllable, which constitutes the unit of speech.

The pronunciation of a word such as SEPTEMBER requires rapid alteration between activity and rest of the vocal muscles. "S," "P," and "T" are pronounced without voice, the rest of the sounds with voice. In dysarthria, dysarthrophonia and in cluttering such movements are not carried out in an adequate manner nor in the right tempo.

Stammering was referred to briefly to stress that there is still too little known about its etiology, and that this word is in use for a variety of disorders of communication with symptoms in common.

Friday, 10th November, 1950, at Claremont Street Hospital:

Dr. Henry Yellowlees read a paper entitled "The Psychiatric Witness," and discussed his report to the Royal Commission on Capital Punishment. He defined the McNaughton rules, and while he agreed they were unsatisfactory, no reasonable substitute had been found despite criticism and many commissions. In their favour it must be said that they worked, but that a fair and broad interpretation was required by the Judge. This occurred in 95 per cent of trials. He considered that in murder trials there should be three separate hearings—the first being to decide whether the accused was fit to plead, the second whether he was guilty, and the third the degree of responsibility. The first decision was an entirely medical problem. He deplored the situation which arose when contrary evidence was given by doctors for the Crown and defence. He thought that the system at present in use in Scotland and in certain states in the U.S.A. was a good one. Here the sanity of the accused is considered by a board of doctors who report directly to the Judge.

An interesting discussion followed. Dr. Lothian and Dr. Robinson were the main speakers.

Annual General Meeting,
Friday, 22nd December, 1950, at Claremont Street Hospital:

Dr. H. Shepherd read a paper on the radiological diagnosis of spinal cord tumours.

(A) STRAIGHT RADIOGRAPHY.

1. Importance of oblique projections in the cervical area to show the intervertebral foramina and pedicles.
2. *Bone erosion.*
   (a) *Pedicles.*
   Importance of the normal range of interpedicular distances measured by Elsberg and Dyke.
   Changes in pedicles vary from cortical erosion to complete destruction.
   (b) Laminæ.
   (c) Posterior surface of body—"scalloping."
   (d) Extent of bone erosion depends on size of tumour.
   (e) Predilection of extradural neurofibromata to grow through and erode Intervertebral foramina.
3. *Paravertebral Shadow*—deformed by extravertebral growths and by tumours growing through the thoracic intervertebral foramina.
4. *Calcification*—seen in meningiomata and prolapsed disc lesions.
5. *Scoliosis and Kyphosis*—no connection with presence of tumour, except congenital arachnoid cysts, which cause a marked kyphosis.

(B) MYELOGRAPHY.

1. Contrast used—Ethyliodophenyl—undecylate (Pantopaque. Myodil). 3-5 ccs. used—5 ccs. preferred for examination above level of conus.
2. Preference for lumbar injection, unless a cauda equina tumour is suspected. Cisternal injection also used if it is desired to know the upper level of a tumour.
3. Important to examine in both prone and supine positions with horizontal lateral "shoots" at any suspicious level.
4. Characteristic defect produced by extradural, extramedullary and intramedullary lesions.
   (a) *Extradural.*
      1. Irregular transverse lower margin.
      2. Similar appearance with addition of "step" on opposite side to lesion.
      3. Displacement of contrast away from tumour.
   (b) *Extramedullary.*
   Sharply defined concave margin, with displacement of cord away from tumour mass, and contrast tending to pass on opposite side to tumour.
   (c) *Intramedullary.*
   Irregular deep concave margin, with evidence of cord expansion, and contrast tending to pass on both sides.
   (d) Differentiation between tonsillar herniation and high cervical tumour. Herniation produced a characteristic biconcave defect while tumour shows a concave margin to the contrast column.

Friday, 19th January, 1951, at Purdysburn Hospital:

Dr. C. B. Robinson read a paper on Leucotomy—a follow-up of sixty cases.

From October, 1948, to June, 1950, sixty cases were treated at Purdysburn Hospital by prefrontal leucotomy. The blind lateral approach following the standard method of Freeman and Watts was carried out in each case and the maximum cut possible was made as close to the tip of the anterior horn of the ventricle as possible.

Of the sixty cases treated forty-one were of Schizophrenia—Dementia Præcox, seven of Paraphrenia, eight of Affective Psychosis, two of Paranoia, one of Obsessive-Compulsive Neurosis, and one of Arteriosclerotic Dementia with Paranoid traits.

There were three deaths in the series, giving a mortality rate of 5 per cent. Two cases died from post-operative pneumonia and one from blood clot in the aqueduct.

Thirty cases showed mental improvement of whom nineteen were discharged, but two have since returned to hospital care. Thirteen of those discharged are working, the occupation varying from full household duties to a coppersmith in the shipyards. Thirteen other patients have shown improvement in behaviour without mental change.

The vast majority of these patients were chronic mental hospital cases whose prognosis was poor and possibility of discharge nil.

Nine clinical cases were shown ranging from a female selected for operation showing great tension, apprehension and fear, to three of the discharged and working post-leucotomy patients.

An interesting discussion followed.

Mr. Calvert stressed the risks involved in elderly patients submitted to operation. Dr. Dorothy Gardiner emphasised the good results in paraphrenics. Dr. Hilton Stewart brought up the question of pain appreciation in intercurrent affections. Dr. O'Malley discussed the aspect of criminal responsibility in non-psychotics following the operation. Dr. Allison brought up the question concerning the patient's post-operative gain in weight and the question of peptic ulcer in association with operation. Dr. Mulligan stressed the value of the operation for patients who were difficult to manage. Dr. Millar discussed the possibility of a second operation where the first had failed and also the effects on coincidental hypertension.

Friday, 23rd February, 1951, at Claremont Street Hospital:

Dr. Desmond Curran read a paper on "Murderers." He said that he had seen 17 since the end of the War — 7 for the defence and 10 in the statutory enquiry. He stated that 70 people commit murder per year — of these almost half commit suicide, one-seventh are unfit to plead, a quarter are guilty but insane, and one-fifth are hanged in England and Wales.

In his experience and that of other psychiatrists, no murderer is depressed or

upset by the experience; in fact, they sleep well and gain weight. All profess an amnesia for the crime and none showed any remorse for the victim. All but one, in his experience, cheated at cards when playing with the warders.

He then enlarged on certain cases and discussed the value of the E.E.G. in some cases, and stated that it might mislead and prejudice the court too much. The incidence of epilepsy, he stated, was lower in Broadmoor than in the general population. He also could not agree that there should be different rules for epilepsy in murder than for other crimes.

Friday, 30th March, 1951, at Claremont Street Hospital:

Dr. Mulligan showed an interesting case :—

C.McM., aged 21 : *Railway Fireman* : *Single.*

Admitted Holywell Hospital 25th October, 1950. Certified patient, being transferred from Crumlin Road Jail, where he had been awaiting trial for breaking a shop window and stealing jewellery.

On admission he was very dull and slow, disorientated for time, was unable to give much account of himself, his recent memory was very poor and his attention was difficult to hold. He had left-sided anæsthesia and diminution of left visual field. He had left-sided paresis involving the face, left arm and leg.

Previous History—25.10.48 fell from tender of an engine, was admitted to Royal Victoria Hospital, where he remained six weeks. On discharge from Royal Victoria Hospital he had weakness of left leg, but there were no neurological signs and air encephalogram was normal. Following his accident patient complained of nervousness, intense headaches, dizzy turns, irritability and poor memory.

In view of his history of accident, his paresis and apparent mental changes he was transferred to Claremont Street Hospital, and later to Royal Victoria Hospital for further neurological investigation, as he was suspected of having a subdural hæmatoma. E.E.G., X-ray of skull, lumbar puncture, blood investigation and C.S.F. investigation all gave normal results.

In the Royal Victoria Hospital he complained that the devil was talking to him and that he seemed to be in a dark tunnel with the devil. He was diagnosed schizophrenia and re-admitted to Holywell Hospital on 27th January, 1951.

Investigation under Kemithal and hypnosis revealed :—

(a) Complete memory for his accident.

(b) Memory of breaking the shop window.

(c) Being locked in the coal-house at the age of 9.

(d) Fear of being a cripple as the result of his accident.

Further information obtained :—He was an illegitimate child, and had been adopted by foster-parents at the age of two weeks. At the age of five he had greatly resented a nephew, then a baby, receiving the attention of everyone in the house and had broken the baby's rattle in his resentment. Later in tempers

he had broken plates, and in these incidents gained gratification and attention.

His real mother had had her left arm amputated and patient feared that he might lose his left arm. A foster-brother suffers from epilepsy, has left-sided paresis, and at home is regarded as a cripple and receives much attention.

Following this investigation and explanation patient's paresis disappeared, anæsthesia disappeared and patient was able to use his left arm and leg in a normal manner and showed marked improvement in his general mental attitude.

FURTHER INVESTIGATIONS.

Fields of vision perimetry showed hysterical type restriction of visual fields. Intelligence quotient 104. Performance Ability 81 I.Q.

PHYSICAL FINDINGS.

Diminished knee and ankle jerk left side. Hypotonia left arm and leg, some difficulty in pointing out left side of his body, altered sensation over left forehead and upper cheek. Specimens of patient's writing before and after treatment and his attempts at Milhill Vocabulary Test were exhibited.

This case was presented as a case of gross hysterical overlay following his accident in 1948. His breaking the shop window was an attempt to gain sympathy and attention, his hallucinations were of hysterical nature and a return of his experience as a child, his aggressiveness in the Hospital was regarded as being a return of his infantile aggressiveness.

Friday, 4th May, 1951, at Holywell Hospital:

Dr. Smith demonstrated three cases of Huntington's chorea, two from one family, and on family trees showed the presence of this disease in other members of these families, and in previous generations.

In a brief survey of the early American literature he drew attention to the comparative frequency of this condition in certain districts of the New World, and how the origin of these cases had later been traced to a few settlers who had immigrated from the East Anglian district of England in the seventeenth century. He mentioned that cases were still to be found in this and in neighbouring regions of England, and that there would appear to be a similar pocket of the disease in the Portglenone district of County Antrim, where both families of his cases had originated.

Before showing the cases Dr. Smith dealt briefly with the physical and mental symptoms of Huntington's chorea, mentioning certain features of the involuntary movements, speech, gait and facial expression. He was of the opinion that mental symptoms ocurred early in the disease and he drew attention to the frequent occurrence of outbursts of violence in the early stages of the disease, and to increased sexual desire, which often led to aggressive sexual behaviour.

This interesting paper was discussed at some length by Drs. Weir, Mulligan, O'Malley, Millar and Robinson.

Dr. Mulligan demonstrated a case of Encephalitis. A man aged 21, a joiner, was admitted to Holywell Hospital on 20th July, 1950, from the Royal Victoria Hospital.

There were no serious illnesses and no history suggestive of schizophrenia predisposition. In April, 1950, the patient fell ill with "flu" and then became listless, dull and lethargic. Ten days later he became confused, feverish, and later had severe headache with vomiting. In May, 1950, he became delirious and was transferred to Whiteabbey Chest Hospital as possible tuberculous meningitis. Cerebral spinal fluid investigations failed to confirm the diagnosis, and he was transferred to Royal Victoria Hospital on 29th May, 1950.

He was then very confused, disorientated and confabulated freely; he had stiffness of neck, blurring of optic discs, generalised increase of tone in muscles, but no paresis. He was doubly incontinent and later appeared to be in a catatonic state. He was given benzedrine mgm.X and within twenty minutes developed status epilepticus which was with difficulty controlled by paraldehyde. Following this he became elated and noisy and had to be transferred to Holywell Hospital. At first he was noisy, confused and appeared to be indifferent to the feelings of others. He smeared himself with fæces, exposed himself, sang at the top of his voice and required to be fed.

Gradually he improved, was more co-operative, less confused and had control of his habits and conduct. Steady progress was maintained till at time of clinical demonstration patient was able to work in the joiners' shop and had little behaviour disorder.

On Demonstration—He is still childish, euphoric, emotionally labile, cannot concentrate easily, and shows marked loss of ability to perform practical tests.

E.E.G. showed a focus of theta waves in right temporo-occipital area.

Summary.—A case of organic type mental changes following encephalitis, possibly of virus origin.

An interesting discussion followed.

REVIEWS

THE COST OF HEALTH. By Ffrangcon Robert, M.D. (Pp. 193 + 7. 16s.). London: Turnstile Press. 1952.

EVERY now and then in the whirligig of life it is necessary for humanity to stop and ask itself where it is going. This is true not only of mankind in general, but also of the professions and particularly, since it holds a peculiarly intimate position, of the medical profession. Though the publishers commend this book to the layman it is of even more interest and indeed of fundamental importance to the members of our profession. In the last few years we have seen and taken part in a revolution in medicine. The nation has taken notice of what we have to contribute to its concept of the Welfare State, and in a flurry of Parliamentary activity, of B.M.A. conferences, of arguments which on the whole have been concerned more with economics than with professional status or ideology, the new state of medical nationalisation has been born. The book deals with the consequences of that act. The fallacy of so-called planning on schemes of such magnitude is exposed. The £170,000,000 estimate of Beveridge in 1942, the £230,000,000 estimate of Bevan in 1948, have in the experience of three years been found to be mere guesses, and Parliament has had finally to limit the expenditure on health to £400,000,000. In other words, medicine is rationed, and it is only within the framework of that ration that diagnosis, treatment and medical advance can be perpetuated. The humanitarian outlook of the doctor in the past has not been questioned, but now it becomes necessary to strip aside emotionalism and wishful thinking and to re-orientate the aims of the profession. Where money is scarce is it better to spend it prolonging life through palliation but not cure of the degenerative diseases, or to maintain the general health of the community by providing better houses and a reasonable standard of living? For the doctor it is a bitter choice. "We are rapidly approaching the time when humanitarian demands can be satisfied only by inroads upon our resources prejudicial to our standard of living, when the claims of physical health will conflict with the claims of economic health." All this is largely the result of the advance of medical science. Since the discovery of insulin there has been an ever-increasing cost of ill-health. The advance of medicine as a science has resulted not in a simplification of disease but in an ever-growing complexity. The "complete" doctor of the last century has become subdivided into an ever-growing number of specialists, each speciality in turn conceiving the need for more and more technicians, until now in Addenbrooke's hospital every two patients require the services of three full-time people. Is all this technical help really necessary? If technicians on a lower scale of salary do so much of the essential work are so many doctors required? If they are required is it essential that there should be such a differential rate of pay? These are problems to which the medical profession must give its mind, and its future depends largely upon how they are solved. No one likes ill-health, but when medicine is nationalised the economic value of the doctor to the state becomes a matter of importance, an importance which is linked with the productive capacity of the state and hence related to the number of strikes, the will to work of its people, and to the exercise of restraint and unselfishness by every individual citizen. There is no limit to the possible advance of medicine. The abolition of disease is an unattainable objective. Into this paradox has been thrust the inescapable logic of a limited expenditure. What is to be the solution for the medical profession? Some solution must be found if our traditions and ideology are to remain inviolate.

Every doctor must read this book. Medical organisations must consider the problems posed by it. Analytical as it is the answer to its questions has not been found. J. H. B.

EMBRYOLOGY OF THE PIG. By Bradley M. Patten. Third edition. (Pp. i-xiii, 1-352. 35s.). George Allen & Unwin Ltd., London.

THIS new edition of Professor Patten's book appears to have taken three years to cross the Atlantic, as it was published in Toronto in 1948. A book, the second edition of which has been reprinted ten times in sixteen years, and has now been re-edited, deserves a quicker transit than this. The book is designed as a guide to the laboratory study of developing mammalian embryos, and particular emphasis is placed on the critical stages of emergence and differentiation of the organ systems. The original, well-proved layout has been retained, but, owing to enlargement of the page width, certain illustrations have been enlarged for the benefit of the student. The text has not been materially lengthened, and it has been revised and improved where possible. The recent work by Amoroso (1950) on the changes undergone by the chorion and uterine epithelium of the pig during the course of pregnancy, renders the older ideas on the microscopic classification of placentæ inadequate, and a much fuller account of the placenta than is given here is necessary for a proper appreciation of this important organ, and will doubtless be given in future editions. The histogenesis of tissues and organs, except for cartilage and bone, has been omitted on the grounds of lack of space or because such changes can easily be demonstrated on laboratory material. It is felt that a short account of the major histological changes in the various organs would add considerably to the value of the book as a whole. It is unfortunate that the term "Gill (branchial) arches" is used when writing of the mandibular, hyoid, and the other visceral arches. It is inaccurate and most misleading to the student of comparative anatomy. A welcome note of caution in accepting too easily the prevalent ideas on the mechanism of closure of the ductus arteriosus is struck in the account of the development of the cardiovascular system, in which it is pointed out that no revolutionary changes in the load on the circulation occur at birth.

The general production of the illustrations and letterpress is excellent, and a useful bibliography is appended at the end of the text. Misprints are few, but 'potency' for 'patency' of the foramen ovale (p. 261), and 'maker' for 'makes' (p. 96) are two of the slips which have been noticed. The book can be recommended unhesitatingly to the medical or science student studying vertebrate embryology in the laboratory, and it is to be hoped that these will be increased in number as more and better facilities are made available in the anatomy departments on this side of the Atlantic.

E. C. Amoroso: "The Essential Parenchyma of the Pig's Placenta"; Communication to Anatomical Society, November, 1950; 1950. W. R. M. M.

ORAL AND DENTAL DISEASES. By Hubert H. Stones, M.D., M.D.S., F.D.S.R.C.S.Eng. Second edition. (Pp. 1012+xix; 959 figures. £5. 0s. 0d.) Edinburgh: E. & S. Livingstone, 1951.

THE first edition of this book was published in 1948 and the appearance of a second edition so soon afterwards is an indication of its popularity. In general form and make-up the new edition resembles its predecessor, but it has been extensively revised and several chapters have been almost completely re-written. An examination of the references appended to each chapter reveals that many of them are as recent as 1949 and 1950, a few 1951 — showing that this edition is not a reprint of the original with minor alterations, but that the author has, to a large extent, produced a new text. There is a completely new section on diseases of the nervous system and muscles. The whole work is a balanced review of the extensive field of oral pathology and the many references make it a source book for the recent literature. The treatment of the subject matter is systematic, clear and concise, and the whole work is profusely illustrated, many of the figures being in colour. This book has already become the accepted text-book for dental students in Great Britain and thoroughly deserves its popularity. The medical practitioner should find much to help him, particularly in the discussion on oral sepsis in relation to systemic disease and in the accounts of stomatitis and allied diseases of the oral mucosa. P. J. S.

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MODERN TREATMENT YEAR BOOK, 1951. Edited by Sir Cecil Wakeley. (Pp. viii + 356, Plates 31. 17s. 6d.). London: Baillière, Tindall & Cox. 1951.

THIS volume is an attractively produced collection of thirty-nine chapters on a wide variety of subjects written by a well balanced team of experts under the general editorship of Sir Cecil Wakeley. The team consists of members from the main London hospitals and from many widely scattered Provincial hospitals. Consequently a broad cross-sectional view of modern treatment is provided.

Of general interest the article on streptomycin gives accurate guidance of its value and limitations specially in application to tuberculosis. The chapter concerning occupational therapy provides a welcome introduction to an increasingly important aspect of treatment. Three chapters are devoted to tuberculosis relating to B.C.G. inoculation, mass-radiography, antibiotic and surgical treatment. The value of the chest clinic in diagnosis and supervision of selected cases is emphasised.

Cancer in various areas is referred to in four chapters, including breast and uterus with special reference to the tragedy of its incidence in the stomach. Prostatic carcinoma is considered under the aspects of prostatectomy, testicular evisceration and hormone therapy.

Peptic ulcer treatment by vagotomy and the problem of perforation are considered in two chapters. Cardio-vascular problems of hypertension and emergencies are covered in two valuable chapters, though one cannot agree with the prohibition of intravenous mersalyl. Apart from tuberculosis, two chapters are occupied with differential diagnosis and emergencies in respiratory conditions and provide real help.

Gynæcology is represented by chapters on prolapse and cancer of the uterus. Skin diseases are discussed regarding recent advances in treatment with special reference to Psoriasis. Pædiatrics is well provided with four chapters, that on emergencies being specially valuable. Two useful chapters concern diabetic emergencies and management of the diabetic child. Chapters on plastic surgery, E.N.T. infections and anæmia indicate possibilities and expected results of modern therapy.

The largest section of eight chapters deals with a heterogeneous collection of congenital and acquired orthopædic problems. This proportion of the whole appears rather generous, specially in view of the fact that the treatments are all hospital procedures. If it does emphasise early diagnosis it may be justified.

Generally the material is good, advice is clear and its rationale explained. The keen general practitioner specially will be well advised to buy and digest this book. J. S. M.

DISEASES OF THE NOSE, THROAT AND EAR. By I. Simpson Hall, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E. Fifth edition. (Pp. xii + 463 + figs. 82. 18s.). Edinburgh: E. & S. Livingstone, 1952.

THIS is entitled a handbook for students and practitioners, and admirably attains this object. The fifth edition is definitely the best up-to-date book of its kind to-day. The whole subject is condensed into a book of reasonable size for the student or practitioner. The paper and print are of the first quality, and the illustrations are good. There are no bibliographies.

Each section begins with a short description of the anatomy and physiology of the part.

In the description of the blood supply of the nose, the ethmoidal vessels are omitted. There is a good description of the technique of opening a quinsy, but no guide is given as to when to open it. The presenting features of naso-pharyngeal malignant growths do not include enlargement of the upper deep cervical glands. The use of streptomycin might have been mentioned in the treatment of pain in tuberculous laryngitis. Great stress is rightly placed on the necessity of having a competent laryngologist examine every case of hoarseness of three weeks' duration, which is not showing improvement.

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GYNÆCOLOGICAL ENDOCRINOLOGY FOR THE PRACTITIONER. By P. M. F. Bishop, D.M.(Oxon). Second edition. (Pp. 132. 12s.). Edinburgh: E. & S. Livingstone. 1951.

THIS book of 132 pages is a compact, interesting and easily read review of the very difficult and complicated subject of gynæcological Endocrinology.

The book is primarily for general practitioners but many consultants and M.R.C.O.G. examination candidates can read the book and profit thereby. Dr. Bishop is a leading authority on endocrinology in Guy's Hospital Medical School, the Chelsea Hospital and the Postgraduate Medical School of London.

In this edition many alterations have been made and much of the book has been re-written and brought up to date—very necessary in dealing with such a changeable subject as endocrinology.

A chapter on steroid chemistry has been introduced, and in spite of the frightening nature of the carbon ring diagrams the author has succeeded in simplifying for the uninitiated a very difficult branch of chemistry. This chapter, like all the others, is self-contained and can therefore be utterly ignored without interrupting the general theme of the book.

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The book opens with a brief review of the commoner causes of death at the different ages of childhood, and ends with chapters on infant feeding and an appendix containing a short Formulary. The intervening sections cover most of the diseases likely to be encountered by general practitioners. The majority of the matter contained therein is in accordance with the current opinions of pædiatricians, but there are a few rather misleading statements, and some omissions.

Atresia of the œsophagus is an anomaly which can now be treated with a high chance of survival if diagnosed early, and mention might have been made of the signs leading to the diagnosis. Among the causes of recurrent abdominal pain, one is surprised to find no mention of non-tuberculous mesenteric adenitis, which is probably responsible for some 50 per cent of such cases. Dr. Williamson rightly stresses the value of ante-natal rhesus typing of mothers, and the early detection of antibodies in the hope of ensuring early diagnosis and treatment of affected infants, but does not mention the Coombs' test which is more valuable than mere rhesus testing of the infant. A recent survey has confirmed his impression that Cæsarian section or premature induction of labour increases the mortality in these infants.

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W. A. B. C.

AN ATLAS OF GENERAL AFFECTIONS OF THE SKELETON. By Sir Thomas Fairbank, D.S.O., O.B.E., M.S., Hon. M.Ch. (Orth.), F.R.C.S. (Pp. viii + 411. Figs. 510. 55s.). Edinburgh: E. & S. Livingstone. 1951.

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The general plan of the book is that of discussion of each entity with useful, but occasionally uncritical, reference to the literature followed by case histories abundantly illustrated and selected from the author's own material. The range and variety of the case material is remarkable and the author is to be congratulated on the success of his collecting interest in this field. This book will prove useful to the surgeon, to the post-graduate student and is not without value to the physician and pathologist. Once again E. & S. Livingstone earn our congratulations for a beautiful production.

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In the final papers Marson gives preliminary results of investigations showing unsuspected alterations in adrenal functions in a series of elderly patients.

These papers should interest many people besides those in the medical profession whose work brings them constantly in contact with the social and medical problems of old age.

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The book is very easy to read, the diagrams and illustrations are plentiful and most helpful. A separate chapter (xxxiv) deals quite effectively with physical therapy, and it would have been better to have concentrated this form of treatment in this chapter rather than to limit it to a line or two where relevant at the end of most of the chapters elsewhere, e.g., on page 655. "General measures are appropriate, and physiotherapy, although it has no special application, is helpful when the joints become stiff. Surely physiotherapy is to prevent such joints becoming stiff. Again on page 721—"In physiotherapy of the shoulder joint, radiant heat, massage and movements to the neck and shoulder, usually given three times a week for six weeks. If no improvement has taken place at the end of that time, the treatment is stopped." Very few cases respond within this period, and it is fairly common to have many of these shoulder cases attending the Department of Physical Medicine long after the six weeks have elapsed.

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CLINICAL PRACTICE IN INFECTIOUS DISEASES. By E. H. R. Harries, M.D., F.R.C.P., and M. Mitman, M.D., F.R.C.P., with the collaboration of Ian Taylor M.D., M.R.C.P. Fourth edition. (Pp. 698. 30s.). Edinburgh: E. & S. Livingstone. 1951.

THE fourth edition of this work, first printed in 1940, enhances the position enjoyed by its earlier editions.

The presentation of the current teaching of infectious diseases is commendable for its orderliness. Following chapters upon notification, infection, transmission, diagnosis and epidemiology the individual diseases are arranged in groups with common features.

Each disease is adequately catered for and an extensive bibliography follows each chapter. A synopsis of the contents appears at the opening of every chapter. The bacteriology and laboratory diagnostic measures are adequately treated and the description of symptoms and clinical signs is excellent.

Fashions in the treatment of acute infectious conditions change with such irregularity in style and detail that authors of textbooks must find the position embarrassing. It must be nigh impossible to prescribe treatment of some acute conditions acceptable to all the critics.

This book should prove of great help to teacher, consultant, practitioner and public health worker. For postgraduate students it is first class but for the undergraduate its size may relegate it to a work for reference only. To those students who can afford the time it is warmly to be commended.

F. F. K.

STATISTICS FOR MEDICAL AND OTHER BIOLOGICAL STUDENTS. By L. Bernstein, B.Sc., M.R.C.S., L.R.C.P., and M. Weatherall, M.A., D.M., B.Sc. (Pp. 180+xii; figs. 17. 18s.). Edinburgh: E. & S. Livingstone, 1952.

THIS book is based on a course of instruction given to first year preclinical medical students. The first nine chapters are very clearly written and should present no difficulty to beginners. The subject is introduced by a discussion on scientific method and an explanation of elementary probability problems. The reader who is unfamiliar with algebraic notation should not be disheartened by the volume of algebraic symbols in the chapter on probability, these are fully explained and a grasp of this chapter will greatly facilitate the appreciation of what follows. Frequency distributions, averages, measures of scatter, sampling problems and a study of chance differences, which make up the remainder of the first nine chapters, are dealt with very fully and are illustrated by good numerical examples.

Regression, correlation, transformations and the analysis of variance are described, in the next four chapters, mainly from theoretical aspects; in fact the authors stress that these descriptions are not intended to give detailed working instructions. In my opinion, much of the potential value of the book is lost in these sections by this limitation. Numerical examples, of the high standard set by the earlier chapters, would have helped through these more advanced sections of the book those students who have difficulty with algebraic notation. The last chapters on experimental design, the conduct of therapeutic experiments and the interpretation of observations should be of considerable use to beginners in the field of medical research.

It is never easy to decide how much of a subject should be included in an elementary text book and therefore it is probably a matter of personal choice that no demonstration of the life table technique in the comparison of survival rates is included. Also with the ground prepared by the chapter on probability a short explanation of the exact treatment of a fourfold table might have been included in the sections on Chi^2.

A few detailed points require comment. On page 58 it is said that "The arithmetic mean is the value which makes the sum of the deviations from it as small as possible"; the sum of the deviations of individual observations from their mean is always zero, it is the sum of the squared deviations which is a minimum. On page 81 it is said of the difference between the means of two samples that it "should be small compared with its own standard error if the two samples

CLINICAL PRACTICE IN INFECTIOUS DISEASES. By E. H. R. Harries, M.D., F.R.C.P., and M. Mitman, M.D., F.R.C.P., with the collaboration of Ian Taylor M.D., M.R.C.P. Fourth edition. (Pp. 698. 30s.). Edinburgh: E. & S. Livingstone. 1951.

THE fourth edition of this work, first printed in 1940, enhances the position enjoyed by its earlier editions.

The presentation of the current teaching of infectious diseases is commendable for its orderliness. Following chapters upon notification, infection, transmission, diagnosis and epidemiology the individual diseases are arranged in groups with common features.

Each disease is adequately catered for and an extensive bibliography follows each chapter. A synopsis of the contents appears at the opening of every chapter. The bacteriology and laboratory diagnostic measures are adequately treated and the description of symptoms and clinical signs is excellent.

Fashions in the treatment of acute infectious conditions change with such irregularity in style and detail that authors of textbooks must find the position embarrassing. It must be nigh impossible to prescribe treatment of some acute conditions acceptable to all the critics.

This book should prove of great help to teacher, consultant, practitioner and public health worker. For postgraduate students it is first class but for the undergraduate its size may relegate it to a work for reference only. To those students who can afford the time it is warmly to be commended.

F. F. K.

STATISTICS FOR MEDICAL AND OTHER BIOLOGICAL STUDENTS. By L. Bernstein, B.Sc., M.R.C.S., L.R.C.P., and M. Weatherall, M.A., D.M., B.Sc. (Pp. 180+xii; figs. 17. 18s.). Edinburgh: E. & S. Livingstone, 1952.

THIS book is based on a course of instruction given to first year preclinical medical students. The first nine chapters are very clearly written and should present no difficulty to beginners. The subject is introduced by a discussion on scientific method and an explanation of elementary probability problems. The reader who is unfamiliar with algebraic notation should not be disheartened by the volume of algebraic symbols in the chapter on probability, these are fully explained and a grasp of this chapter will greatly facilitate the appreciation of what follows. Frequency distributions, averages, measures of scatter, sampling problems and a study of chance differences, which make up the remainder of the first nine chapters, are dealt with very fully and are illustrated by good numerical examples.

Regression, correlation, transformations and the analysis of variance are described, in the next four chapters, mainly from theoretical aspects; in fact the authors stress that these descriptions are not intended to give detailed working instructions. In my opinion, much of the potential value of the book is lost in these sections by this limitation. Numerical examples, of the high standard set by the earlier chapters, would have helped through these more advanced sections of the book those students who have difficulty with algebraic notation. The last chapters on experimental design, the conduct of therapeutic experiments and the interpretation of observations should be of considerable use to beginners in the field of medical research.

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The reviewer has never met Miss Powell but it is obvious, on reading between the lines, that she has been both an orthopædic nurse and a physiotherapist and has decided, after a trial of both activities, that of the two orthopædic nursing holds price of place, and how right she is.

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It is perhaps a pity that no bibliography is provided. As the author suggests, references are easily obtained from the larger textbooks, but these books often fail to indicate those papers most likely to be of value to those practitioners for whom this book is intended.

This book can be recommended to all requiring a readable and informative introduction to the virus diseases. J. E. M.

STEROID HORMONES AND TUMOURS. By Alexander Lipschutz. (Pp. 310; fig. 111. 46s. 6d.). Baltimore: Williams & Wilkins Co.

This book represents a summary of the experimental work upon which Lipschutz and his associates have been engaged since 1936. All too often new drugs are introduced into the clinical armamentarium without the clinician gaining the necessary understanding of their true value and limitations. The author has set out to find out the mechanism by which the steroids may play a part in the dynamics of cancer. Perforce his work has been entirely on animals, and he is the first to recognise that the results are not transferable in their entirety to man, but they point the way to investigations in the human subject. His work appears to establish the dangers of continuous œstrogenic therapy. "The fundamental problem of therapeutics with œstrogens is not searching for a 'less toxic' or 'less tumorigenic' compound, but establishing those timing conditions of administration which guarantee discontinuous action of the œstrogen." With appropriate timing, all œstrogens are harmless. Without regard to timing, all œstrogens may be harmful. The introduction of steroid treatment for prostatic carcinoma renders an understanding of their action in the human more than ever important. They are potent weapons, involved in many of the endocrine relationships, and their physiological balance should only be disturbed with understanding.

MEDICAL BOTANY. By Alexander Nelson, Ph.D., D.Sc., F.R.S.E. (Pp. xii + 544; 13 figs., 16 plates. 30s.). Edinburgh: E. & S. Livingstone, 1951.

This is described as a hand-book for medical men and all who are concerned in the use of plants: nutritionists, dieticians, pharmacists and veterinarians. It presents much information, some of which will be useful to medical men, and requires for its understanding only a small knowledge of academic botany. It is not a textbook of botany for medical students and the author has written a text book for that purpose. The author suggests, however, that if, in the first year course, the amount of academic material was lessened, time could be found for the introduction of some of the topics discussed. If the syllabus could be so arranged, the material presented here is much more likely to be of interest and profit to students than much that is at present taught.

The author first considers the composition of vegetable foods in general. He then describes the foodstuffs derived from various grains, seeds and vegetables and something of their properties and preparation. Plants as the source of drugs receive attention, but most of the information, if required, can be found in text books of materia medica. References by which the information given on a specific problem might be supplemented are not included.

We join with the author in hoping that doctors and members of the public will find interest and profit in reading here something of how botany, by its impact on our health and well-being, enters into our daily lives. We doubt if the book can find much place in the overcrowded syllabus and library of the medical student.

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This is described as a hand-book for medical men and all who are concerned in the use of plants: nutritionists, dieticians, pharmacists and veterinarians. It presents much information, some of which will be useful to medical men, and requires for its understanding only a small knowledge of academic botany. It is not a textbook of botany for medical students and the author has written a text book for that purpose. The author suggests, however, that if, in the first year course, the amount of academic material was lessened, time could be found for the introduction of some of the topics discussed. If the syllabus could be so arranged, the material presented here is much more likely to be of interest and profit to students than much that is at present taught.

The author first considers the composition of vegetable foods in general. He then describes the foodstuffs derived from various grains, seeds and vegetables and something of their properties and preparation. Plants as the source of drugs receive attention, but most of the information, if required, can be found in text books of materia medica. References by which the information given on a specific problem might be supplemented are not included.

We join with the author in hoping that doctors and members of the public will find interest and profit in reading here something of how botany, by its impact on our health and well-being, enters into our daily lives. We doubt if the book can find much place in the overcrowded syllabus and library of the medical student.

with virus disease and its treatment. This book attempts to remedy this. Obviously it cannot be a comprehensive textbook; much has had to be omitted, and some will doubtless disagree with what has been selected. Description of the special techniques for virus study and of the virus agents themselves may appear inadequate, but the book is concerned with virus diseases and not with the organisms alone. Almost all human virus diseases are referred to and the descriptions of the commoner diseases usually include accounts of their main clinical features, of their diagnosis by laboratory methods and of their epidemiology and sometimes of salient features of their pathology. The order in which these aspects are discussed varies, and, while this avoids a stereotyped presentation, it is not always easy to find the desired information.

It is perhaps a pity that no bibliography is provided. As the author suggests, references are easily obtained from the larger textbooks, but these books often fail to indicate those papers most likely to be of value to those practitioners for whom this book is intended.

This book can be recommended to all requiring a readable and informative introduction to the virus diseases. J. E. M.

STEROID HORMONES AND TUMOURS. By Alexander Lipschutz. (Pp. 310; fig. 111. 46s. 6d.). Baltimore: Williams & Wilkins Co.

This book represents a summary of the experimental work upon which Lipschutz and his associates have been engaged since 1936. All too often new drugs are introduced into the clinical armamentarium without the clinician gaining the necessary understanding of their true value and limitations. The author has set out to find out the mechanism by which the steroids may play a part in the dynamics of cancer. Perforce his work has been entirely on animals, and he is the first to recognise that the results are not transferable in their entirety to man, but they point the way to investigations in the human subject. His work appears to establish the dangers of continuous œstrogenic therapy. "The fundamental problem of therapeutics with œstrogens is not searching for a 'less toxic' or 'less tumorigenic' compound, but establishing those timing conditions of administration which guarantee discontinuous action of the œstrogen." With appropriate timing, all œstrogens are harmless. Without regard to timing, all œstrogens may be harmful. The introduction of steroid treatment for prostatic carcinoma renders an understanding of their action in the human more than ever important. They are potent weapons, involved in many of the endocrine relationships, and their physiological balance should only be disturbed with understanding.

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A PRACTICAL HANDBOOK OF MIDWIFERY AND GYNÆCOLOGY. By W. F. T. Haultain, O.B.E., M.C., B.A., M.B., B.Ch., F.R.C.P.Ed., F.R.C.S.Ed., F.R.C.O.G., and Clifford Kennedy, M.B., Ch.B., F.R.C.S.Ed., F.R.C.O.G. Fourth ed. (Pp. 412+x; figs. 47. 24s.). Edinburgh: E. & S. Livingstone, 1951.

THIS is the fourth edition of a well known book from the Edinburgh School which compresses the subjects of Obstetrics and Gynæcology into 401 pages. It is concise, presents the orthodox principles in an easily assimilable form and yet notes the more recent changes in the two subjects in a way that will attract the attention of the reader. It is a book which would be valuable to the student for quick revision rather than as a text book from which to prepare for an examination. Where so much has been covered in such an excellent way some criticism is inevitable.

It is surely not in keeping with the modern requirements to state that a normal case "need only be examined three times before her confinement," the first examination being advised "preferably as soon as a period has been missed" and the second at 35 weeks. It is difficult to see how the doctor is going to recognise an abnormality between 6 weeks and 35 weeks without examinations in the interval.

The statement on page 45—"The urine may also be tested for sugar, but this is not nearly so important as the test for albumen, as 40 per cent of pregnant women have sugar in their urine at some time during their pregnancy and its presence in the great majority of cases is of no importance," is dangerous advice. Surely the urine should be tested for sugar and if this is present some investigations should be carried out to prove that it is of no importance.

The rules for the management of normal labour in domiciliary practice are excellent and of great value to the student or practitioner, but is the advice given under the management of the second stage wise? Here it is stated "If the head is not entering the pelvis well try Walchers position."

The section on placenta prævia has been rewritten and it is gratifying to see the stress laid on omitting a vaginal examination except when the patient is in hospital prepared for any necessary operative interference.

One would, however, question the advice regarding the use of Hegar's dilators for dilatation of the cervix to permit palpation of the placenta. The fœtal mortality is surely not worst in the fourth degree (central) placenta prævia as in this degree Cæsarean section is the usual method of treatment.

The treatment of asphyxia livida is scarcely in accord with modern teaching apart from the first sentence "Remove inhaled fluids and mucus from the mouth and upper air passages." The methods of artificial respiration mentioned are rarely if ever used to-day.

The section of the book devoted to gynæcology is excellent and gives the student a clear clinical picture and well considered therapeutics. It does seem, however, out of proportion to devote so much space to the operation of uterine suspension with minute operative details.

In view of the attention paid to the cure of stress incontinence nowadays it might have been advisable to warn the student and practitioner that the recently adopted operations are not always successful, and that even in skilled hands are associated with certain risks and complications.

The book is short, readable and, considering its scope, fulfils its purpose excellently.

C. H. G. M.

FOOD AND NUTRITION. By E. W. H. Cruickshank, M.D., D.Sc., Ph.D., M.R.C.P. Second edition. (Pp. xii + 443; figs. 51; tables 78, 30s.). Edinburgh: E. & S. Livingstone, 1951.

THIS is a readable, authoritative and well documented survey of the science of nutrition. After a brief, but informative introduction on food economics and the evolution of human diets the author illustrates the present inequality of food distribution in the world to-day. The precarious position of our own economy is evident, but some may think that the problems raised by over population, especially in the Far East, are not sufficiently emphasised. About one-sixth of the book presents in detail the changes in British diet before, during and after the War. The author reminds us that "no diet, which is characterised by monotony, difficult planning, uncertain shopping and is not enlivened by a freedom of choice, can be regarded as wholly adequate, no matter how perfectly it may meet the required standards for calories and nutrients." After a clear presentation of the energy requirements of the body the contributions of proteins, fats and carbohydrates, minerals and vitamins are individually reviewed in a reliable and entirely up-to-date discussion. The properties of various foodstuffs such as bread, milk, protein and fat-rich foods, vegetables, fruit and nuts are then described. Many, whose interest is not entirely scientific, will find much to interest them here and in the chapter on the dehydration and preservation of foods. These chapters and those on dietary standards and planning, diet in dental caries and on appraisal of nutritional status should be read by anyone who would presume to advise people in normal health concerning diet. The book is not concerned with diet for specific disease conditions, but all medical men who require an authoritative survey of the modern science of nutrition, and of recent contributions which have been made to it, often by non-medical workers, should read this book.

A HANDBOOK OF SURGERY. By R. C. B. Ledlie & M. Harmer. (Pp. viii+536, figs. 56. 21s. net). London: Baillière, Tindall & Cox. 1951.

MODERN surgery is such a gigantic subject that it would seem a well-nigh impossible task to write a reasonably comprehensive account in a little over 500 pages — this, however, is what Messrs. Ledlie & Harmer have achieved. All branches of surgery, with the exception of ophthalmics, are covered. The result is a little book which will be of the greatest value both to the student and to the general practitioner.

There are 56 drawings which are clear and effective. Photographs and X-ray reproductions are excluded. Inevitably in such a book discussion of alternative methods of treatment is limited, and in places this leads to dogmatism. Generally the result is orthodox, but most surgeons would quarrel with the statement, referring to carcinoma of the stomach, that "In practice, for a neoplasm not confined to the pylorus this means a *total gastrectomy* (author's italics), the spleen being also removed. . . ."

The whole work is up to date and at least touches on most recent advances. It is, however, disappointing to find no mention in the chapter on cerebral surgery of intra-cranial aneurysms which form so large and rewarding a part of modern neurosurgery. The section dealing with fractures is closely based on the well known work of Sir Reginald Watson-Jones, to whom acknowledgment is made.

There is very little to criticize and this little book can be strongly recommended to students and practitioners. T. K.

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INTRODUCTION TO CLINICAL NEUROLOGY. By Gordon Holmes, M.D., F.R.S. Second edition. (Pp. vii+189. 12s. 6d.). Edinburgh: E. & S. Livingstone, 1952.

THE author of this book requires no introduction. His original intention in writing it was to discuss the nature and significance of the symptoms and signs seen in nervous disease in terms of the underlying structures and functions involved. How well he has succeeded is illustrated by the appearance of a second edition this year to which some new material has been added. A wholly admirable work, it may be commended unreservedly to all students and especially post-graduates who wish to look beyond the mere formal or systematic description of nervous diseases and study how symptoms and signs arise. In attempting this difficult task few men can be in a better position than the author to assess conflicting doctrines and to interpret the progress which has been made in the understanding of nervous functioning. Considering the range of subjects treated: the motor systems, muscle tone and co-ordination; convulsions and involuntary movements; sensation; the visual system; postural reactions and the vestibular system; speech, agnosia and apraxia; the autonomic system, the book is remarkably compact, comprising as it does not more than 186 pages of text. This conciseness of expression has entailed exercise of the fine judgment possessed by all good teachers in deciding how much to leave out and how much to include, and, of course, the exclusion of any vestige of superfluity.

R. S. A.