"It is more dangerous to be in a hospital ward than on the battlefield at Waterloo," remarked Sir James Simpson when referring to wound sepsis in hospitals.

"It may seem a strange principle to enunciate as the very first requirement in a hospital that it should do the sick no harm," wrote Florence Nightingale some ten years later. Although hundred years later Sir James Simpson's statement is no longer tenable, we are far from attaining Florence Nightingale's principle, particularly in the realms of Hospital Infection.

Hospital Infection, or Cross Infection, or Added Infection, may be defined as a clinical or bacteriological infection which has been acquired from the hospital environment. It is therefore possible, and important to appreciate, to have an overt (clinical) or symptomless (bacteriological) infection. Hospitals are ideal places for acquiring organisms. The patients are usually of lowered resistance; there is a high concentration of people loaded with organisms; and as the ratio of patients to ward staff is always high, the possibility of transmission is greatly increased.

An important first step would be to examine the prevalency of Hospital Infection. This requires
quantitative data of which there is a serious shortage (Lowbury 1957), due partly to ignorance but mainly to lack of interest in high places. Bacteriologists, when eventually consulted, often find a considerable basal level of hospital infection has been going on for some time. Much of the information which is available concerns Staphylococcal cross infections, because it has recently become of major clinical importance, but other pathogenic organisms, particularly the spore bearing, are of importance too.

Cross infection is found in Maternity Units. Cunliffe (1957) reported a 75-100% cross infection of babies' noses by the end of the first week. This varies from unit to unit, and from month to month in any one unit. It has proved difficult to assess the clinical importance of this cross infection because septic complications arise after discharge and are not reported to the hospital.

Wounds provide an admirable medium for bacterial survival. This problem was particularly important during war time. Wounds are often left untreated for sufficient time to become heavily infected. In peacetime less importance has been stressed, because the usual hospital techniques greatly reduce the fatalities.
However, spore bearing organisms can still cause havoc. This may be particularly important nowadays when hospitals are undergoing reconstruction and setting free dust which may contain spores originating from organisms many years ago. There is some evidence of a rise in wound infection in some hospitals. Howe (1954) reports a rise in major post operative sepsis of wounds after clean operations in Massachusetts Memorial Hospital:

<table>
<thead>
<tr>
<th>Year</th>
<th>1949</th>
<th>1950</th>
<th>1951</th>
<th>1952</th>
<th>1953</th>
</tr>
</thead>
<tbody>
<tr>
<td>%</td>
<td>.6%</td>
<td>1.1%</td>
<td>1.4%</td>
<td>1.7%</td>
<td>2.35%</td>
</tr>
</tbody>
</table>

Gillespie (1957) reported a 41% infection of abdominal wounds in a Bristol hospital. 76% of these were found to have their source in the ward.

Shooter (1957) complains of the relatively small amount known about cross infection in medical wards compared to surgical, save perhaps that it is going on, and particularly in staphylococcal outbreaks which aren't controlled by usual medical and nursing practices. Staphylococcal cross infection is high in respiratory disease. Stuart and Harris (1953) report that in an influenza epidemic 20% of the patients had staphylococci in their sputum, whereas usually it was 4%. Robertson and Whitehead (1958) have shown the prognosis of patients with influenza who sustain this added infection is much worse. There is little information on the role of
cross infection of respiratory disease in non-epidemic periods.

During a 14-day period in April 1959 as a Student Clerk on a male medical ward of 24 beds, I recorded 5 cases of chest infection, who, after their arrival in hospital were cross-infected with staphylococcus aureus coagulase (+ve) (i.e. on admission their sputum was staphylococcus free). One died and another was rapidly declining into irreversible toxaemia. All patients showed a sudden arrest in initial progress with the arrival of the staphylococcus in the sputum. [Urinary and Gastrointestinal cross infection has also been reported in medical and surgical wards.]

From this brief survey of the prevalence of hospital infection it is clear that it is a problem. Many regard it of no real significance, because they consider 95% is reversible with antibiotics. But in fact it may well be accounting for a bigger death roll than is yet appreciated. It may well be possible for a patient's defence mechanism to be operating at a maximum against a primary invader. The arrival of a secondary invader, which classically may not be regarded as being highly pathogenic, may cause a slight diversion of the defence mechanism, and so lose control of the primary organism sufficiently for it to produce a severe relapse and even death. There is a natural
tendency for doctors to regard only overt (clinical) manifestation of Hospital Infection as of any importance, but a high percentage of bacteriological infection reveals the presence of an avenue down which highly virulent organisms may come, and efforts should be made to close this pathway. Rubbo (1948) related the increased bed occupancy due to sepsis of wounds after radical mastectomy. He showed that between the years 1945-1947 at least 5% of these required 10 extra days because of sepsis. He calculated a loss in a 500 bed hospital of 2,500 patient days, or the economic wastage for all surgical cases in a metropolitan area of the size of Melbourne, of £15,000 a year, assuming the very low cost of £1-10-0 per day.

The ultimate aim is adequate prevention and treatment. But to discuss these it is necessary to study where and how hospital infection arises.
The hospitalised patient provides four major targets for attacking organisms:

(a) The Respiratory System,
(b) The Intestinal System,
(c) The Genito-urinary System,
and (d) Areas denuded of their protective coverings, such as wounds, burns inoculation sites and damaged mucous membranes.

Organisms may reach these sites by ingestion, air transmission or direct contact. Each target is perhaps predominantly infected by a specific mode of spread.

The main avenue down which organisms reach the respiratory system is the air. Lister (1867) stressed the importance of microbes in the air, and so developed his carbolic spray. Airborne infection, however, was later forgotten under the strong influence of the German School of Von Bergman (1894), and the French School of Graucher (1900) and Hutinel (1894), who propounded that direct contact was more important. Not until Wells (1936) observed that bacteria, dispersed into the air after coughing, could be carried long distances in the air currents, were Lister's beliefs confirmed. Since then Crosby and Wright (1951) Garrod (1944), Rubbo (1948) AND MANY OTHERS have shown that dust carries bacteria. Anderson and Sheppard (1959) have cultured staphylococci from wool fibres from bed blankets. Wright, Cruikshank and Gunn (1944) claimed that dust was of
great significance in Clinical cross infection. Other workers have not been able to verify their findings.

Whether coughing plays an important part in airborne sources is rather doubtful. It has been shown that large droplets from a cough can travel 2-3', and from a sneeze 6-8'. By placing culture plates 1" from the mouth it has been shown that the majority of organisms fall down the 45° line. Direct transmission, therefore, seems unlikely as people rarely proximate to 1" of each other. Furthermore, Dugid (1946) has observed that, of the organisms collected from controlled coughing experiments, haemolytic Streptococci were found in only 39 out of 87 patients, and that these represented only 10% of the total number of colonies on the plates. The real importance of large droplets may, perhaps, have been revealed by the work of Hare (1940) and Hare and Mackenzie (1946) who have shown that $\frac{1}{3}$ of the large droplets after a sneeze fall onto the clothes immediately below the mouth. This $\frac{1}{3}$ represents a far greater danger because of the comparatively small area into which it has fallen. The hands, which are the natural tools of hospital infection, can then easily distribute them far and wide.

Small droplet nuclei (0.05 - .1 mm. in diameter) are also formed after coughing. These minute particles can be blown like whisps of smoke for long distances. They are believed to be important in the transmission of such virus
infections as measles, chicken pox and small pox.

In addition to droplet nuclei, organisms may reach the respiratory system from inanimate objects like books and toys etc. (these are termed formites). Harries (1935) has also shown that Nurse's hands may also help in spreading respiratory infection in the Ward.

The intestinal system may become cross infected by the ingestion of organisms. This may result from contaminated food, fingers, clothing, formites and anything which is liable to come in contact with the patients mouth.

The commonest forms of these infections are acute gastro-enteritis and Sonne dysentery, being more common in Childrens Wards. Taylor et al (1949) have located a certain serological strain of E.Coli which may be responsible for much of the infantile diarrhoea which has often been fatal. These organisms have been found in the dust, bedding, formites and even the hands of the Nurses who had just "scrubbed up" (Rogers 1951). Williams (1956) stressed the rapidity with which these organisms spread when he recorded an outbreak which, in a matter of hours, had become widespread.

Staphylococci which are usually resistant to the common antibiotics, and which seem to be highly virulent, are beginning to make a more widespread appearance in intestinal infections. Cook (1957) first recorded the importance of the association between the Staphylococcal and diarrhoea.
Frisby (1957) regarded staphylococcal diarrhoea as the major cause of death after partial gastrectomy in Oxford. Webster (1958) has shown that this condition is quite often there all the time, but is not noticed because it does not reach epidemic magnitudes. He recorded 5 fatal cases out of 8 studied in 4 months. These patients were harbouring intranasal gastric tubes or post used packs following abdominal surgery. Webster emphasises that the organisms had probably been pushed down into a bowel which had received preoperative antibiotics and was adynamic.

Salmonella has many times been the pathogenic organisms. Contaminated food has usually been the major source of the organisms. Food can be contaminated by members of the staff in the kitchens and on the wards. Inadequate storage allows flies or vermin to infect the food. Faulty sterilisation techniques have been a cause of cross-infection in bottle-fed babies. Convalescent carriers have been shown to be able to initiate a fresh outbreak of Salmonella and Shigella cross infection.

Hospital induced urinary infections are very common. Perhaps the catheter is the chief source of organisms, particularly, if it is left in situ for some time. Gynaecological surgery is often followed by a urinary infection. McLeod (1958) has produced evidence which incriminates dirty ward urine bottles. These urinary infections are usually
of no more consequence than of inconvenience to the patient. However, I have seen a patient die with an acute rheumatic endocarditis following catheterisation. Blood cultures showed a profuse growth of the hospital's resistant staphylococci.

Areas denuded of their natural antibacterial barriers are ideal targets for bacterial invasion. Wounds and burns are perhaps the most common examples today. Streptococci and Staphylococci are the most frequent invaders, but there is evidence of an increasing incidence of Proteus Vulgaris and Pseudomonas pyocyaneus. Perhaps the major single spreading factor in this form of cross infection is direct contact which may occur due to self (endogenous) infection or from somebody else, either a member of staff or a visitor. Such a person is known as a Carrier. Carriers may be either temporary or permanent. Thus a member of staff may transfer organisms to a clean wound from another infected wound by a careless technique, or the member of staff may carry the organisms on his body permanently. It has been shown that the anterior nares, throat and recently the skin of the perineal region can carry a high concentration of organisms. The former is perhaps most important because it is more commonly infected, more heavily infected and is a region which often comes into contact with fingers etc. The anterior nares is believed to contain glands which produce
secretions which are ideal for certain bacterial growth. There is considerable evidence to show that these carriers contaminate their environment and, perhaps, increase the incidence of cross infection. Hamburgher et al (1945 a & b) Robertson (1958), Hare (1957) and Williams (195) regard the nasal carrier as the principal source of resistant staphylococci in hospitals. Barber and Burton (1956), Munro and Markham (1958) have shown that Carriers with superficial lesions have also played their part in other cases of cross infection of wounds. Williams (195) while studying reported hospital epidemics between 1954-57 observed, "that it is striking how often a single individual is the cause of all the misery".

Wounds infection may occur in the Theatre or the Ward. In the Theatre it has been shown that contaminated air is an important factor. Robinson, McLeod and Downie (1940) reported 2 post operative tetanus cases in which dust had reached the Theatre from building operations. Sevitt (1953) reported a case of gas gangrene from dust in the Theatre. Bourdillon et al (1951) have shown that these are 3 major factors which increase the bacterial air count in Theatre, (a) Patient's entry, (b) Removal of Blankets, plasters, splints and etc. (c) The Air drawn in from the rest of the hospital. The surgeons and their staff have also been incriminated. Devenish and Miles (1939) first
reported cases due to leaky gloves and wet arms. The risk is even greater when any member of the Surgical Team is a Carrier.

Self infection may prove to be a bigger problem than is yet appreciated. Robertson (1958) has produced figures which show that patients with a positive staphylococci skin culture before operation have a higher incidence of post operatic infection.

<table>
<thead>
<tr>
<th>Pre-op.</th>
<th>Post-op.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin +ve 41</td>
<td>11 = 26.8%</td>
</tr>
<tr>
<td>Skin -ve 356</td>
<td>21 = 3.9%</td>
</tr>
</tbody>
</table>

That all these were self infections is not possible to surmise because Robertson does not mention whether the infections were of the same phage type as the host's skin. Williams (1951) has produced similar findings but with patients who were Nasal Carriers. He has shown that 11% of nasal carriers and only 2% of non-nasal carriers developed wound sepsis. Endogenous infection is often regarded as inevitable, but it is evident that many of the organisms on the patient are from the hospital environment. Goslings et al. (1958) recorded 57% of 587 patients carried coagulase +ve staphylococci on discharge from hospital. The organisms colonised the patient's noses in the first two weeks in hospital.

Surgical wards are perhaps more likely to be places of cross infection than Theatres because of the traditional
extra care that is taken in the latter. In the Ward the Carrier, particularly the nasal carrier, has been incriminated many times. This has been so particularly for staphylococcal cross infections. Faulty dressing techniques have always been regarded as of prime importance since the work of Fleming and Tytler (1923). Bed clothing has also been shown to play some part. Barber and Dutton (1956) were unable to control an epidemic until the blankets were replaced, or chemically treated. Colonies of staphylococci have been cultured from blanket fluff collected during bed making, when there is a raised air concentration.

Obstetric and Gynaecological wards have suffered greatly from cross infection. Colebrook (1935) estimated the death rate from puerperal sepsis was 1.8/100 births. Using Griffith typing of streptococci, he showed that the important causal agents were Nurses, Mid-wives and Doctors in attendance. This was very similar to Semmelweiss' finding almost 100 years before.

Maternity hospitals have been shown to house a high concentration of organisms, particularly the Staphylococci. Markham (1958) in his studies showed that organisms reached
their destination either by carriers (not usually nasal) or by dust. The lesions produced were conjunctivitis or boils, but could become more serious like osteomyelitis and pneumonia etc. (Gonococcal ophthalmia and vulvo-vaginitis are rare cross infections nowadays). A heavy nasal invasion of the infant may produce a breast abscess in the mother. Organisms reaching the milk could cause serious intestinal infections in the baby. Williams (1956) has suggested 3 epidemiological patterns in breast abscesses, (a) A single Source — thus only one phage type. This usually incriminates a Nurse. (b) Dust — usually find a single phage type in all babies, and (c) Numerous sources due to faulty aseptic techniques — a mixed phage type is found. Jellard (1957) recorded the importance of the umbilical cord as a source of infection. (She records a 70% infection by the 4th day of life).

Burns are quickly colonised by gram-ve organisms, but secondary invasion by Haemolytic streptococci and Staphylococcus aureus is very common. Cruikshank (1935) found Haemolytic Streptococci to be the most common secondary invader shortly after admission. The organisms arrive by dust
dust and direct contact. Lowbury (1957) considers that secondary invasion is nearly always due to cross infection from other burns. Infected burns impede recovery by causing sloughing of skin grafts and arresting natural granulation processes. They are also reservoirs of organisms for further cross infection.
PREVENTION.

Infection of a hospitalised patient depends on:

1. THE PRESENTATION OF ORGANISMS TO THE PATIENT.
2. THE PATHOGENICITY OF THE ORGANISMS.
3. THE PATIENT'S RESISTANCE TO THE ORGANISMS.

Prevention, therefore should be directed at these 3 principles.

PRESENTATION OF ORGANISMS.

It has been shown (vide supra) that a source of organisms and various vectors are necessary to present organisms to the patient. Many hospitals practice preventive measures, and it is therefore necessary to apply a systematic investigation designed to uncover any hidden source or vector. Williams (1957) has outlined 3 main investigations which are necessary but all must be based on an adequate record of infections:

(a) Epidemiological. Notes should be made of the relation of onset of infection to admission; the people who were in attendance. Whether the infection was explosive or gradual in onset. It should always be remembered that of 5 infected patients it is not necessary for all to have become infected in the same way.

(b) Bacteriological. Information is needed on the species of the organism and more definitive typing. Phage typing has been particularly useful for staphylococci.

(c) General Hygiene. An examination of Ward techniques, laundry, sterilisation etc. are all necessary to find a possible weakness in the system.

In the Theatre stress has been laid upon air contamination. Bourdillon and Colebrock (1946) have shown the importance of air sucked into the Theatre from the rest of the hospital by fans and heat. They emphasised the use of a +ve pressure ventilation system, in the form of filtered,
humidified warm air pumped in from the roof. They produced 10-20 changes of air per hour without causing a draft. If this stress is to be laid upon a stream of air passing downwards, then it is important to avoid counter currents in the form of talking, hot lights and excess movement by any member of the Surgical Team. Much of this would not be necessary if it were possible to get a centripetal current of air from the operating table.

Ultra Violet irradiation has been used particularly in Thoracic Surgery to reduce bacterial air concentration. As the surgical team must use special goggles, it has not been very popular.

Patients should not be brought into the Theatre with blankets or other articles of clothing from the Ward. Ideally there should be as little disturbance as possible on entering Theatre. This is best achieved by putting the patient on to the Operating Table outside of the Theatre - in the anaesthetic room. Some hospitals will allow the admission of a ward bed into the Theatre to transfer directly a patient in a serious condition. This is not necessary as the operating table could be pushed out of Theatre before the transfer is made.

Every precaution must be taken to ensure that all Theatre equipment is up to a high standard of sterility. Autoclaves should be repeatedly checked. Masks should be of the standard requirements (M.R.C. War Mem. No. 6. 1941). Penikett and Gorill (1958) have produced a very simple device which instantly records whether a glove is perforated or not. This apparatus should be available for all Theatres as these workers recorded that 21 out of 187 "sterilised" gloves had one or more holes in them. Gowns should be available in good numbers and preferably of a non wettable material.

Some surgeons regard the real value of efficient Theatre ventilation is to keep them cool. There is, however,
evidence that positive pressure ventilation not only reduces the air bacterial count but that of sepsis too. Shooter Taylor, Ellis and Ross (1956) have produced these results:

<table>
<thead>
<tr>
<th>Ventilation Type</th>
<th>Colonies/ft.$^3$</th>
<th>% Sepsis Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>-ve pressure</td>
<td>130</td>
<td>9</td>
</tr>
<tr>
<td>+ve pressure</td>
<td>45</td>
<td>1</td>
</tr>
</tbody>
</table>

Lowbury from Birmingham reported similar findings:

<table>
<thead>
<tr>
<th>Changed Air</th>
<th>Unchanged Air</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Sepsis</td>
<td></td>
</tr>
</tbody>
</table>

Bourdillon and Colebrook (1951) suggested that bacterial air counts should not exceed $10/ft.^3$ for normal resistant tissue and not greater than $2/ft.^3$ for burns, brain and other low resistant tissue. These "safety levels" are empirical and were not intended to be anything more than a guide. More work on this problem is needed, but it can be seen that efficient +ve pressure ventilation can have far more reaching effects than cooling the surgeon.

Endogenous infection may be prevented by thorough pre-operative lavage of the operational site. It is the usual practice to shave the hair off the day before the operation. The razor, particularly in female hands, can produce small abrasions which have a full 12-24 hours to become colonised before operation. These pockets of organisms may well escape the usual pre-operative lavage. (This is a suggestion and has no known clinical or experimental proof).

Theatres traditionally are places in the hospital where most attention is paid to the prevention of cross infection. It is remarkable, however, that alongside vigorous aseptic techniques lurk many mal-practices. Anaesthetists, in most hospitals visited, simply wear a cap, mask and gown
over their ordinary clothes. Many have a habit of leaving theatre for a few minutes "breather". In some hospitals visitors to the Theatre are not uncommon. If in galleries, they rarely have more than a mask on, and if on the theatre floor they tend to be similarly attired as the Anaesthetists. One wonders what Semmelweiss would say if he could see in 1959 a 4th year medical student, as part of his approved timetable, having spent an hour standing lift. away from a post mortem, leave and go up into a surgical Theatre in one of the eminent teaching hospitals in the world.

Prevention of cross-infection in the Wards is much less developed than in the Theatre. It should be founded upon decreasing the air pollution, controlling the handling of patients by contaminated hands or instruments and prevention of the ingestion of infected food.

In reducing air pollution particular attention has been taken to eliminate dust getting into the Ward, or the disturbing of that already in the Ward. Dark, dusty, and draughty corridors should not be linked with wards. Dust from bed clothes can be dangerous as it may be loaded with pathogenic organisms (Anderson and Sheppard 1959). In theory, therefore all attempts should be made to reduce circulating dust. Several methods may be employed:-

(a) Positive Pressure Ventilation.
(b) Abandon dry dusting, sweeping and floor polishing in the patient area. Oil has been used on the floor, and Lush and Edward (1940) reported a decreased bacterial air count.
(c) Bedding particularly blankets is often difficult to sterilise. Wool blankets are quickly destroyed if sterilised by heat. Barnard (1952) however, has used estyl pyridium bromin "Fixanol C." impregnation, and claims that blankets were not only sterilised but to some extent self disinfesting. Blowers and Wallace (1955) and Frisby (1957) have achieved similar success using a non-ionic detergent "Lissapol", and also a cationic detergent known as "Cirrasol". Another possibility would be to produce blankets which not only reduce.
the amount of fluff, but could be sterilised. Blowers (1957) reported three important advances:-

(1) The Synthetic polyester fibre (terylene) blanket which is good but expensive. (2) Cotton Turkish towelling which is also expensive and (3) Cellular cotton weave which is cheaper and more durable than wool.

There is no positive evidence that such measures on bedding produce a decrease in hospital infection. Wright, Cruikshank and Gunn (1944) have claimed a 54% reduction in streptococcal complications with measles, when floors and bedding were treated with oil. These figures seem conclusive but other investigators (Shooter 1958, Clark, Dalgleish and Gillespie (1954) have found no significant fall in staphylococcal cross-infections. Nevertheless, it would seem sensible to disinfect wool blankets in the meantime and to replace them gradually by blankets, such as the cellular cotton weave, which not only generates less dust, but can be boiled and cheaper. It should therefore be common practice to ensure that every patient gets a clean set of blankets and a plastic mattress and pillow cover.

(d) Ultra violet light is bacteriocidal, and has been used extensively in America. Hart (1941) reported success in thoracic surgery. Rosenstein (1948) reported similar success in the Ward when placing it across the open end of a cubical in “barrier nursing.” Many other such reports have been received, but there tends to be a general lack of convincing experimental details, and this is perhaps why it is not used extensively in this country.

(e) Spraying the air with chemicals such as hypochlorite, resorcinol, propylene, triethylene glycol, lactic acid and its derivatives has also been employed (Gudin (1942). They have fallen into disrepute, however, because of the difficulty in atomisation and maintenance of bacteriocidal concentrations, and with their relative inefficiency to deal with large dry carrying particles. Nevertheless Harris and Stokes (1945) have claimed some success using Glycols in a 3 year trial, but their work has not been verified by others.

(f) Isolation of patients with lesions liable to be sources of cross infection is an important method of preventing all modes of spread. Ideally separate wards are required with a special team which is not allowed to come in contact with other members of the hospital. Rocke Robertson (1958) has shown that such patients grouped together do no harm to each other or their attendants. Most hospitals in this country have no facilities for complete isolation but nevertheless barrier nursing should be done until it is available.
Prevention of the presentation of organism to the intestinal system is perhaps the easiest of all, because most is known of their mode of spread.

Kitchen hygiene is most important. Flies and vermin must be controlled. Staff should report immediately any injury which breaks the skin, particularly if septic. There should be efficient washing-up facilities, ample cold storage and general storage.

Great care should be taken in preparing babies' feeds. Ideally there should be a central milk kitchen, but as this is usually impossible, ward feed preparing should be carefully supervised. Bottles should be rinsed in cold water after use, then with hot detergent and rinsed again. They should always be boiled for 2-5 minutes before use.

If there is any indication that cross-infection is going on then a full investigation should be made. If no obvious sources are found then rectal swabs of all members of the staff and patients should be taken.

The problem of preventing organism reaching wounds etc. by direct contact is, perhaps, greater than any other, because it may well be the cause of the greater part of hospital infection.

A scheme must be developed which prevents members of staff carrying pathogenic organisms and putting them on to the patient. It is therefore first necessary to deal with the whole problem of carriers, although their importance is not restricted entirely to this mode of spread.

It is eminently possible to prevent the genesis of a temporary carrier. This can be done by making quite sure that members of staff do not pick up organisms from other lesions. They may do this when dressing septic wounds, and it is important that a no touch technique is applied. It has long been known that organisms may be picked up by merely touching an infected patient's bed. This caused Groucher (1900).
and Hutinel (1894) to institute barrier nursing. Graucher considered netting round the bed to be quite adequate, where as Hutinel used 3 m. high partitions. These measures were designed to prevent physical touching, or to remind the staff not to touch. Barrier nursing is essentially as follows:—

1. Avoidance of air contamination by keeping doors shut.
2. Patient has his own thermometer, pulse glass and pencil for charting.
3. Nurses should work bare armed.
4. Nurses and doctors should have their own gowns which must be worn whenever the patient is attended. The gowns must only leave the cubicle for laundring.
5. Staff must wash hands before and after treatment.
6. Patients should not be visited by convalescent patients.
7. Each patient should have his own bed clothes, bathing facilities etc.

It has already been shown that permanent carriers of pathogenic organisms tend to infect their environment. (Hare 1957) has evidence that some carriers liberate staphylococci more heavily than others. This is a reflection on either their general habits or a heavy nasal carriage. There has been much scepticism as to whether carriers are of any real clinical importance. Evidence is meagre, but Williams (1959a) quotes figures from 2 epidemics:—

<table>
<thead>
<tr>
<th>% development of Sepsis.</th>
<th>Carrier Present</th>
<th>Carrier Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidemic.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.</td>
<td>26</td>
<td>2</td>
</tr>
<tr>
<td>B.</td>
<td>47</td>
<td>8</td>
</tr>
</tbody>
</table>

He concluded that the healthy nasal carriers are of major importance. Whether or not a carrier will produce an overt cross-infection depends on the pathogenicity of the organisms.

There are two major ways of dealing with the carrier. It is possible to assist greatly by exerting some physical control. Thus in the Theatre it has been suggested that ordinary clothes should never be allowed. Talking and
movement should be kept at a minimum. On the wards the no touch technique is vital where wounds and babies are concerned. Staff with septic lesions should not be allowed on the Ward. Above all, the staff must be educated to appreciate that the organisms they house can cause great harm. The problem of education however, will be dealt with later.

Chemotherapy has been used to control the carrier. As the most common and potent reservoir of staphylococcus aureus is believed to be the anterior nares, much work has been done using antibiotic nasal creams and examining the subsequent environmental bacterial concentration, and resulting hospital-infection. Gould (1957) reported that originally they used the common systemic antibiotics made in an oil and water emulsion and directly applied to the nares. It was applied several times a day for about ten days giving a total dose of 2-4G. In the majority of cases the nares were cleared for sometime after treatment (eg. 50% were totally clear for 1 month). There were several objections to the use of "Systemic" creams:-

(a) They increased the resistant Strains.
(b) If a patient became sensitised the drug could not be used again.
(c) They destroyed the natural flora.
(d) They tend to contaminate the environment with antibiotics.

Gould and Allan (1954) attempted to reduce some of the criticisms by using antibiotics which were not in common use. They found 1% "Hibitane" (Chlorhexidine) and .5% neomycin reasonably good. In this paper they reported that the incidence of hospital-infection was lowered while the carrier rate was low. (Rountree (1956) had similar findings in neonatal staphylococcal infections.) There was also a lowered incidence of autoinfection of carriers, and finally the environmental bacterial count was lowered.

Using these antibiotics only prevents resistance to the common systemic antibiotics; resistance to neomycin and
Chlorhexidine will develop, and it may be of vital importance when eventually they are required for systemic use. Thus carefree carrier "clean outs" are to be discouraged, and a list of indications for chemotherapeutic treatment of carriers is suggested:

1. If the carrier rate is very high at a particular time of the year.
2. If the carrier has a nasal discharge.
3. If there is a high incidence of cross-infection which can be traced to carriers.
4. In old people who are carriers and are liable to sustain a fatal auto-infection while in hospital suffering from a condition such as influenza.

Antibiotics used for this purpose should be withheld from systemic use. This should avoid many of the criticisms so far raised.

The development of aseptic dressing techniques, is essential in the successful treatment of wounds (M.R.C. War Mem. No. 6.1941). No detail is intended here, but several important factors are emphasised:

(a) Dressing should be started at least 1 hour after bed making, and no visitors should be allowed.
(b) Masks should be of a reasonable standard. (There are two types, a bacterial filter and deflector. The latter is probably more efficient and certainly more comfortable. They should have at least six layers of 40 threads/inch material. It should be snout shaped, and not worn for more than 2 hours).
(c) Hands must never touch the wounds. Gloves often give a false sense of security and should not be trusted.
(d) Each patient should be dealt with individually, and clean wounds should be dressed before known septic ones.
(e) Wounds should not be palpated too often, and should be kept as dry as possible and well covered. The skin around the wound should be treated with great care.
(f) There should be a team of at least two for all dressing procedures. Care should be exercised in disposal of soiled dressings. Instruments must be properly sterilised.
The standard of sterilisation in many hospitals appears to be low. Darmady, Hughes, Jones and Verdon (1959) examined 7 hospitals and found that of the equipment used on the Wards, which was supposed to be sterile, anything from 9-50% of it was in fact contaminated. In their work they have found that a small percentage of the contaminating organisms were in fact pathogenic. Some have tended to sit back with relief and regard this problem as of no clinical significance. The truth of the matter is that this disgraceful state of affairs appears of little significance purely because the majority of the population of hospital organisms are not pathogenic. There is, however, increasing evidence (vide infra) which suggests that there is a sharp rise in antibiotic resistant pathogenic organisms in the hospital environment. Hare (1959) has shown that this situation can be remedied if a thorough examination of the sterilisation techniques are made, and required improvements are made with full co-operation of all members of staff. Most sterilisation is done by a porter, who usually, does not understand the principles and may be careless. Ideally each hospital group should have a central sterilising supplies: this would eventually be more economical and safe. This, however, is not likely to come for some time, and it is therefore essential that technicians in charge of sterilisation appreciate the principles and importance of their work. Regular checks should be made by the hospital engineers to ensure that the sterilisers are operating efficiently.

Special attention has been given to the prevention of infection of burns. Lowbury (1957) has grouped 5 important factors which are necessary to maintain control:

1. Barrier Nursing.
2. "Non touch Technique".
3. Antibiotic Creams applied to the burn. (Erythromycin Cream was first used, and if resistant staphylococci appear then Novobiocin may be used.).
4. Controlled +ve pressure ventilation.
5. Proper covering of burns.

Much of this work was pioneered by Colebrook (1944)
Cross-infection in obstetric units was first really attacked by Colebrook (1935). He stressed several important principles:

(a) The danger of infected medical attendants, particularly with an upper respiratory tract infection.
(b) All attendants must use rubber gloves and masks.
(c) Avoidance of intravaginal manipulations when the membranes were ruptured.
(d) Use of a good disinfectant (Chlorxylenol) as a douche and in a cream for maintenance of sterility of the hands.
(e) Immediately complete isolation of a patient contracting puerperal sepsis.

In 1954 Colebrook was able to report that epidemics of puerperal sepsis had almost been eliminated and the incidence of the disease itself had greatly decreased. Antibiotics had, of course, played an important part.

Maternity units must develop special precautions. Infants are quickly colonised (Vide Supra). Hexochlorophene and Hibitane soaps have been used, but there is no evidence to show that they are of any value. The infant's umbilicus should be painted with triple dye or dusted with Hexachlorophene. Napkins should be handled with care, and dropped into 1: 160 Izal or Jeyes Fluid. They should always be boiled. Special attention should be made to avoid overcrowding. Antibiotics should be given only when absolutely necessary. Some Australian hospitals have ensured that mothers only handle their child. This has eliminated direct contact transmission from nurses. The results have been encouraging and ought to be given a careful trial in this country.

It is important for the sake of the hospital treasurer and medical staff's co-operation to examine what results if any have been produced by the many precautions suggested. The non-touch technique in dressing wounds has proved to be successful.- (Williams 1959a)
Other workers have similar findings. Logue et al (1945) Williams et al (1945).

Rocke Robertson (1958) has made a valuable contribution in Canada: He emphasises that half hearted attempts rarely produce lasting results. However, when he instituted a rigorous preventive scheme, (based on a non-touch dressing technique, efficient +ve pressure ventilation in Theatres and Ward, efficient sterilisation isolation wards and restriction of carriers, there was a dramatic fall in what appeared to be a rising incidence of post-operative wound sepsis:

\[ \text{Graph} \]

Gillespie et al (1957) have shown that barrier nursing, chemotherapy of nasal carriers and the regular treatment of blankets reduced the concentration of hospital...
organisms:

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<tr>
<th>April</th>
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<th>June</th>
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<td>Type 80</td>
<td>4</td>
<td>10</td>
<td>8</td>
<td>4</td>
<td>2</td>
<td>1</td>
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<tr>
<td>And others</td>
<td>2</td>
<td>5</td>
<td>2</td>
<td>2</td>
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**Treatment.**

Colebrook et al (1944) observed that when full preventive techniques to burns, infected with *Haemolytic streptococci* were applied, a 70-90% cross-infection fell to 30-50%.

Gillespie, Simpson and Tozer (1958) have found that the rate of acquisition of staphylococci by babies and also the sepsis rate fell when hibitane cream was used on the nurses' hands after washing, hexachlorophane dusting powder on the babies' umbilicus at each nappy change.

It may well be important to locate possible pools of organisms outside hospital which visitors or members of staff off duty can bring into the hospital. Places where people are crowded together would seem obvious places to watch. Buses could perhaps be important. (I have recorded in March, 1959, a 32% higher rate of coughing in the top deck of an Edinburgh Corporation bus in 20 counts of each deck. This might have something to do with the smoking upstairs). Telephones or money may play a part in bringing organisms into a hospital. Certainly it would be wise to check all visitors entering a ward and to be absolutely sure that new admissions were not a potent source of organisms. A study should be made of the effects of particular members of staff, such as the matron, the physiotherapists and porters, who tend to go into many wards each day.

Preventive measures are ultimately carried out by the hospital staff. It is therefore essential that they should be educated in how and why they should take special care. Education however, would seem to be not enough. One cannot
expect a nurse or a junior doctor to be very strict in aseptic techniques, if their seniors do not set an example. It is sometimes common place to see senior surgeons roaming the wards in their theatre attire, or to go from bed to bed inspecting wounds without washing hands. It never ceases to amaze me that some authorities do not object when Sisters keep cats on the Ward.

While working in hospital in April, 1959, I spent some considerable time interviewing nurses. I have attempted to analyse the results, but from the outset it is emphasised that the numbers interviewed were small. Therefore few definite conclusions can be drawn. After some time had been taken to gain the Nurse's confidence, the question was put as follows:

"Do you report immediately any upper respiratory tract infection such as a cold, or a sore throat, and any septic lesion such as a septic cut or a boil".

The answers were:

<table>
<thead>
<tr>
<th>NO</th>
<th>YES</th>
<th>DON'T KNOW</th>
</tr>
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<tbody>
<tr>
<td>27</td>
<td>1</td>
<td>7</td>
</tr>
</tbody>
</table>

Six of the "don't knows" were P.T.S. Nurses who had never been confronted with the problem. The other "don't know" was a first year nurse. Six out of seven 1st year nurses said no, and 10 out of 10 2nd and 3rd year nurses said "No". The one "Yes" was a Sister.

In the discussion which followed it was evident that all the Nurses had been educated moderately well about the problem. They were in fact disobeying orders when they withheld information, and they knew it. There were two main reasons for not reporting the infection:-(a) If a nurse is off work about a certain number of days in her course, she is liable to be withdrawn from her P.T.S. group when they sit finals - she has to wait until the next time. Thus she finds herself in a new class without her old friends, and a step backwards in the struggle for seniority - a much coveted
prize in the profession. (b) There appeared to be some strife between Sick Bay Sister, who appeared in their eyes to look on such reports as acts of malingering. (It seems that nurses 30 years ago had more stamina and character!). These findings were very common in six other hospitals visited.

In the M.R.C. report (No. 11) on hospital infection, there was a recommendation that nurses did not lose any pay when put off work by infections. The problem nowadays seems to be a little more subtle, but no less human and important. It seems to me that it is wrong to deny a girl the right to sit an examination, because she has missed 2-3 days more than she is allowed, on the grounds that she has not had her proper training. All she may have missed are a series of temperature and bedpan rounds. Perhaps a little more liberal imagination is needed here.

THE PATHOGENICITY OF THE ORGANISMS.

The ultimate pathogenicity of the organisms in modern terms depends upon its:

1. Invasiveness.
2. Toxicity.
3. Susceptibility to antibiotics.

There is increasing evidence of a rising hospital population of resistant organisms to many of the common antibiotics and it is perhaps time to consider other methods of control.

Organisms will settle down in any niche which offers enough room, and other conditions which it needs for existence, such as food and moisture etc. Important work is needed therefore on the essential biological characteristics of the pathogenic organisms. It should be possible theoretically, to fill the bacterial space with a non pathogenic variety. These would exclude the pathogenic variety by sheer numbers, as long as they could grow equally well in the niche. Thus one could envisage hospital bacteriological farms producing
non pathogenic organisms which could be regularly supplied to carriers' noses and the general hospital environment. It would seem impossible to eliminate completely all the pathogenic organisms, because the rate at which they mutate always leaves a few surviving. The suggestion, is therefore, that the tables be turned on these resistant organisms, which have often gained a foot-hold by simply moving into an area cleared of other organisms by an antibiotic, and maintained all available spaces with non pathogenic organisms.

Special care would be necessary after treatment with an antibiotic for some other condition. It is possible that the antibiotic might cause the non pathogenic organism to mutate into pathogenic varieties, as Hoffstadt and Youmans (1932), Pinner and Voldritch (1932) and Barber (1955) have recorded results which suggest that pathogenic and non-pathogenic staphylococci all stem from the one sources. They have shown all permutations and combinations in relation to the production of pigments, lysozyme, coagulase and other toxic substances.

THE PATIENT'S RESISTANCE

The patient's resistance to a pathogenic organism depends on many known and unknown factors of which there are at least three:-

(a) The ability to prevent invasion.
(b) The ability to neutralise toxic products.
and (c) The ability to attenuate or kill the invading organisms by either producing antibodies which agglutinate, lyse, precipitate or opsonise the organisms, or by increasing the number and activity of the phagocytes.

It would therefore be a highly desirable state of affairs to
assist prevention, by increasing the patient's resistance. This may perhaps be done by boosting these known factors.

Malnutrition causes an increased sensitivity to the tubercle bacillus and staphylococci (Dubos, Smith and Schaedler (1955). One imagines it producing an impairment of the anti-bacterial barriers, skin and mucous membranes. Cannon (1943) has shown that rabbits on a low protein intake had a decreased ability to produce agglutinins. There is considerable evidence (Ssacharoff 1928) to suggest that infants at an early age depend upon the gamma-globulins transmitted from their mother in addition to those generated in early life. Later this resistance to infection drops until the gamma-globulin operates efficiently.

Without going into the complexities of antibody formation, it is possible to postulate ways and means of using it to assist the prevention of common hospital infections. Kellner et al (1958) have shown that Group A haemolytic streptococci produce a D.P. Nase. This acts as an antigen, and specific antibodies are produced (the concentration of which rises after a streptococcal infection). These workers have also produced a strain of organisms which produce D.P. Nase (and were not pathogenic), but nevertheless stimulated the production of the specific antibody to D.P. Nase. Similar possibilities have been shown by Emmat and Cole (1955) who have cultured a streptococcus which produces hyaluronidase. This was extracted and given to the rabbit in increasing doses so that it gradually produced an antihyaluronidase. Tacking (1955) has been able to produce an antipenicillinase in rabbits. Johanovsky (1958) observed high antileucocidin titres in post natal women with staphylococcal lesions. Immunisation of mothers during pregnancy with an anatoxin adsorbed on aluminium hydroxide increased the antitoxic and antileucocidin titres in both mother and child; an antileucocidin titre of greater than 2 units/ml. appeared substantially to protect against infection. It therefore seems possible in the future to isolate toxins of hospital pathogens, and immunise
patients before admission — with perhaps a second dose the day before admission. This of course could not be applied to acute admission. Prevention could also be envisaged, theoretically, by giving the patient on admission white cells which were already producing a high concentration of antibodies. This would involve taking blood before admission, and in vitro subjecting it to the hospital organism's antigens. The antibody producing cells would be galvanised into activity, and all daughter cells would be antibody producers too. These cells would then be put back into the same patient on admission. There is yet no way of keeping lymphocytes etc. alive for more than a few hours in vitro, but this problem is on the verge of solution.

Administration of specific antitoxins is a much more difficult problem because organisms like staphylococcus seem to produce many toxins and in various combinations.

There is some evidence of an endocrine control of antibody production, Cortisone assists their formation. Adrenaline enhances infection (Miles 1955). The exact nature of this is unknown, but may be due to a lack of local vascular reaction to invading organisms. A rather more abstract hypothesis, but nevertheless possible mechanism, is that organisms thrive best when the patient has a high blood sugar; diabetics are certainly more prone to infections. Cockrill (1955) has observed that rabbits recover from a staphylococcal infection if they are given insulin. Thus perhaps a low blood sugar is advisable in prevention of hospital infection.

Long (1955) suggested that a certain degree of immunity may well depend on the balance of cortisone and insulin in the body. This major balance can be effected by other endocrine secretions particularly thyroxine which tends to decrease the patient's resistance.

An intact intestinal flora may well prove to be of great importance in intestinal hospital infection. The easiest method of prevention is to avoid the use of unnecessary antibiotics.
Phagocytosis is a natural method of removing and perhaps killing organisms. It would, therefore, be of great importance in prevention to increase the number of phagocytes or at least avoid their disappearance. The latter has become of great importance with the more common use in hospitals of drugs which cause agranulocytosis. It would therefore be unwise to give these drugs to patients in a ward where the carrier rate and general ward contamination was high.

Pollock and Victor (1955) observed that rabbit's phagocytes didn't ingest bacteria until they had made contact with them a certain number of times. They reduced this number by washing the bacteria with dog's R.B.Cs. They concluded that a substance from the R.B.Cs. sensitised the bacteria. It may perhaps be possible to extract this substance and inject it into patients. There is some evidence that the B.C.G. for tuberculosis has a tendency to increase the activity of phagocytes.

Prevention of hospital infection is obviously better than cure, but treatment of infection, whether hospital or otherwise in origin, will always be necessary, and as the form of treatment is becoming increasingly important it is purposed to discuss the problem now.
TREATMENT.

Essentially, the direct treatment of infection is to kill or attenuate the pathogenic organisms either directly or indirectly, or to neutralise the toxins. Chemotherapeutic agents have for some years now fulfilled at least some of these requirements for most infections. Colebrook et al (1942) reported Haemolytic streptococci which were resistant to sulphonamide. From this time it has become increasingly evident that there is arising population of organisms, of many species, with a resistance to one or many of the common antibiotics. Clarke, Dalgleish and Gillespie (1952) reported a strain of staphylococcus aureus which was resistant to six antibiotics. Meleney and Johnson (1953) have reported the percentage resistant staphylococci from wounds in a New York Hospital's S.O.P.D.:

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<tr>
<td>%</td>
<td>17.8%</td>
<td>27.5%</td>
<td>43.5%</td>
<td>43%</td>
<td>31%</td>
<td>22.3%</td>
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(The fall in 1952 was believed to be due to the arrival of bacitracin and neomycin).

Studies have been conducted also in London hospitals they record the percentage resistant staphylococci (Williams 1959b):

<table>
<thead>
<tr>
<th>Year</th>
<th>1946</th>
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<th>1949</th>
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<tbody>
<tr>
<td>%</td>
<td>14</td>
<td>31</td>
<td>37</td>
<td>73</td>
<td>80</td>
<td>81</td>
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</tbody>
</table>

The origin of resistant organisms is perhaps most understood in staphylococcus. The present high prevalence of penicillin resistant staphylococci is associated with a destruction of the sensitive organisms with antibiotics.

Another view is that the resistant strains arise by mutation from sensitive strains under the influence of the antibiotic. If this were so one would expect the distribution
of resistant strains to be the same as found in sensitive strains - the majority in groups 1 & 2. Most resistant strains, however, are phage group 3. Barber and Whitehead (1949) have shown that group 3 sensitive staphylococci tend to become resistant to penicillin more readily than the other groups. Gould (1955) has shown that a change of phage type can occur in vitro, so that phage group 3 penicillinase producing organisms can be produced from groups 1 & 2. If this occurs in vivo it also indicates that organisms can develop resistance within the individual during inadequate chemotherapy. This would be an important step forward, as resistant organisms are more commonly believed to be acquired by secondary invasion.

The effect of this rising population of resistant strains should have a quantitative effect on hospital infection. Unfortunately there are no absolute figures which show a corresponding rise in sepsis rate. There are some figures which show an increase in the percentage of epidemics due to staphylococcus 80. (williamson 1950 - 1956).

<table>
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<tr>
<th>Year</th>
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<td></td>
<td>13</td>
<td>32</td>
<td>30</td>
<td>61</td>
<td>69</td>
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These figures do not indicate a rise in sepsis generally, but merely a rise in the population of this particular strain of staphylococcus, and perhaps a greater capacity for epidemic spread.

It is also possible that these resistant organisms may be more virulent. Rountree (1953) and Freeman (1955) isolated a strain of staphylococcus by phage typing known as staph. 80. They were particularly impressed with its above average virulence, as it produced severe lesions throughout Australia. Anderson and Williams (1956); Gillespie and Adler (1957) and others have also isolated an unusually severe staphylococcal infection with a similar phage type. Rosental
and Faber (1955) have shown that penicillin resistant streptococci were inclined to produce hyaluronidase. Much of the increased virulence may be apparent in that never before has there been such a high concentration of these pathogenic strains in the patient's environment.

Weisener and Stuck (1955) have some evidence that penicillin depresses the natural immunity reactions of the patient.

There is no clear way out of this dilemma, Garrod (1955), however, has put forward certain ideas which seem to be acceptable. He suggests that there are certain conditions which will respond well to specific antibiotics:

1. Those of Haemolytic Streptococci and Lobar pneumonia, for Penicillin, and Typhoid Fever or Chloramphenical.
2. Those caused by a single organism, but its resistance varies, such as boils, carbuncles and other staphylococcal infections.
3. Those caused by a variety of organisms such as bronchopneumonia, bronchitis, endocarditis, urinary infections, wound sepsis and puerperal fever.

Garrod emphasises that in the treatment of lesions which are staphylococcal, or non-specific it is vital, as far as possible, to obtain laboratory sensitivities. If the patient is acutely ill then a rule of thumb method may be necessary—sulphonamides for urinary infections or penicillin for bronchopneumonia and puerperal sepsis.

Antibiotics have been as much as 95% misused in the U.S.A. (Jawetz 1954). Some of this has been for unnecessary prophylaxis. Garrod suggested that it is quite unnecessary for any clean operation; Operations on the mouth, throat or stomach; in normal labour; for simple wounds. But it is permissible for preparation for a lobectomy or colon surgery; for long-term prophylaxis of rheumatic fever; for dental extraction and tonsillectomy in rheumatic subjects; Labour with manual interference and extensive contaminated traumatic wounds.
Prevention of mutation can be best done by giving an adequate concentration of the antibiotic, and by giving two antibiotics together. Synergism of antibiotics is a well known factor, for instance bacitracin and erythromycin (Chabbert and Veron. 1955). Cortizone has been used successfully with chloramphenical in the treatment of typhoid fever (Angioni 1955).

The influx of antibiotics has relatively pushed the use of antitoxin into the background. It may well be that an intensive study of the latter would be extremely valuable, because it would provide a way to neutralise the toxins without interfering with the organisms and so avoid resistance, and provide another weapon, so relieving the over-worked antibiotics. However, the discovery of 6-amino-penicillanic acid may greatly relieve this problem. (Bachelor, Doyle, Naylor and Rollinson. 1959).

In conclusion it would seem that in dealing with hospital-infection members of staff must make themselves alive to their personal responsibilities. Each hospital should have a control-of-infection-committee, headed by a respected senior doctor, which ensures that all wards keep records of infection, and that never tires in looking for improvements. It is also important that the doctors of tomorrow receive more instruction than they do, and that the authorities of the teaching hospitals see that they can practice what they preach.

The ministry of health must inspire the treasury to give more money for improvements such as Engineers, efficient ventilation, isolation blocks, more nurses and bacteriologists. This would all tend to raise the morale and the standard of asepsis and antisepsis. Thus perhaps in 5 years the government would have saved the money expended.

Patients putting their lives in the hands of the profession, are entitled to be protected from any avoidable
ignorance and indifference; the time has not yet come when we can feel sure that they have such protection. Florence Nightingale's principle is therefore still a dream.
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