response between two groups of guinea-pigs in good and bad condition injected with 5 c.cm. toxoid. The mean response six weeks after injection of the former group was 0.284 unit, and of the latter group 0.041 unit. This observation has also been fully established in relation to diphtheria immunization in animals. Secondly, during the last war Mr. Glenny had a horse which had been injected with tetanus toxin-antitoxin mixture. Some time later it received a larger dose of toxin than was usual for a horse undergoing immunization, but although it responded by a considerable increase in antitoxin it suddenly developed tetanus and died.

The inference is that an animal even in good condition may have quite a high titre of circulating antitoxin in its serum but still develop tetanus from toxin fixed before the rise in antitoxin occurred. Although in the case of the horse the toxin was given in one large dose, and in wounded men it would be produced much more slowly, we cannot say that a lethal dose might not be present in these patients before sufficient antitoxin had been produced to overcome it. The risk of inadequate response is perhaps greater in men suffering from severe shock than in healthy individuals. To ensure safety it was decided by the medical authorities of the British Army that all wounded men should receive a prophylactic dose of antitoxic serum, irrespective of whether or not they might have had active immunization with toxoid. Provision was thus made for persons who respond badly to immunization—either to injections of toxoid or to the stimuli of small doses of tetanus toxin absorbed from a wound.

The writers of recent leading articles have stated that toxoid which is given before receipt of a wound has a distinct advantage at the present time not only for the armed Forces but also for certain classes of civilians—e.g., fire brigades, A.R.P. workers, police officers, ambulance men, and perhaps medical and nursing staffs. The alternative is the administration of tetanus antitoxin as soon as possible after an injury has been received. The main disadvantage of relying solely on antitoxin prophylaxis is that the method becomes impracticable when wounded men cannot be rescued immediately; moreover, the injection of a foreign serum protects only for a few weeks, permits the occurrence of late tetanus, and involves the risk of serum sickness not only after the protecting dose of serum but to an even greater degree after any further dose which may be necessary during the next few months. Admittedly the introduction of new methods of concentration of horse serum has greatly reduced the risks of serum sickness, but the higher and more durable immunity which can be induced by toxoid is a potent argument in favour of its use for active immunization. At least 0.01 unit is attained by most persons after two well-spaced injections of toxoid, and much higher titres may follow suitable reinoculation. The special value of antitoxic serum is not to replace toxoid but to reinforce it by affording additional and immediate protection to those few persons whose response to specific stimuli may have been inadequate.

Ramon (1940) has recently advocated for the non-vaccinated wounded patient one injection of antitoxic serum and repeated injections of toxoid (combined active-passive prophylaxis). The method is also claimed to give a better chance of success in the treatment of tetanus.

Conclusions

1. Two doses of tetanus toxoid at an interval of six weeks induce useful amounts of circulating antitoxin. Wider spacing of the doses than six weeks gives better immunity, but is usually inconvenient administratively.

2. Women on our staff gave significantly higher titres than men immunized with two doses.

3. A third injection of 1 c.cm. given after a long interval—e.g., seven to nine months in our series—often produces a dramatic increase in circulating antitoxin, and more than counteracts any tendency of titres to drop below levels which might be considered safe. One year may be a more convenient interval for this “boosting” dose of toxoid.

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HOSPITAL INFECTION OF WAR WOUNDS *

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The Effect of Hospital Infection

From the experience gained in the last five months there emerges a strong impression of widespread hospital infection of wounds. In the greater number of cases the infection is not obvious, because sepsis is often added to existing sepsis. The significance of hospital infection is often missed; the striking cases, which are rare, are attributed to unavoidable accident, and the many exacerbations of illness, which are in fact accompanied by the appearance of a new infecting bacterium in the wound, are put down to “pocketing,” etc. Haemolytic streptococci produce the most severe infections. They are rarely present in the early hours of a wound (Colebrook, 1940). In a series of seventeen air-raid wounds sampled within forty-eight hours of injury a colony of haemolytic streptococci was obtained in one wound. Yet it is not unusual to find, as we have, that 70% of the wounds in a ward are infected with *Strep. pyogenes*. Only rarely does the acquired streptococcus kill (one such case is reported above). More frequently it produces cellulitis, erysipelas, extension of the wound, delayed healing, extensive scarring, and inhibition of successful skin grafting in apparently healthy granulating wounds. It is responsible, in our experience, for many sore throats in patients and personnel, occasional bouts of pyrexia and ill-health, and infection of nurses' fingers and hands.

Even among patients treated with closed plasters, which are thought to reduce the spread of infection in the tissues, those infected with streptococci are apparently worse off than others. The following table, based on the clinical

* Concluding an Interim Report to the War Wounds Committee of the Medical Research Council. The first part appeared last week at page 855.

and bacteriological examination of twenty-nine patients with limb wounds treated with closed plasters, supports this view.

General State of Patient	No of Patients	No. Infected with Haemolytic Streptococci
Definitely ill	6	4
Fairly persistent pyrexia	4	4
Periodic pyrexia	7	6
Well throughout	12	6
	17	14

Only a half of those who were well throughout had streptococcal infection, whereas streptococci were in the wounds of 82.5% of the ill patients. The other infecting organisms, including *Clostridia*, *Proteus*, coliform bacilli, *Staph. aureus*, *Ps. pyocyanea*, and diphtheroids, were distributed in an entirely random manner throughout the twenty-nine wounds.

It is sometimes argued that patients are as a rule immune to the kind of bacteria introduced into wounds during treatment and that the incidence of *serious* hospital infection is so low that the effort needed to reduce it further would be incommensurate with the small effect achieved. Apart from the fact that morbid hospital infection is commoner than is generally supposed, there are two answers to this argument. First, though the incidence of serious infection were as low as 5%, no one patient suffering severely from a preventable infection is likely to be benefited by the thought that nineteen others have escaped it. Secondly, though the infecting bacteria may establish themselves in the wounds and throats of the immune without observable ill effect, the risk to which a susceptible patient is exposed increases greatly with the establishment of each carrier; and even in the relatively immune the acquisition of a streptococcus will almost certainly prejudice, for example, the healing of an extensive burn or the chances of a successful plastic operation.

Sources of Hospital Wound Infection

Patients and Personnel.—*Strep. pyogenes* is carried in the throat and nose and *Staph. aureus* in the nose. The carrier rates in normal populations vary greatly. Up to 40% of hospital staff may carry *Staph. aureus* (for references see Gillespie *et al.*, 1939), and *Strep. pyogenes* is present in from 6 to 13% of the normal population (Straker *et al.*, 1939). Those carrying *Staph. aureus* on the skin are also potential sources of danger, but with adequate precautions none of these carriers need be excluded from dealing with patients (Devenish and Miles, 1939). Proper masks will prevent infected droplets reaching the wound, and proper care and use of the hands make a skin carrier safe. In this connexion, if the wearing of masks for ward dressings is confined (as it is in some hospitals) to those with known or manifest throat infections, only a small proportion of the potentially dangerous streptococcal carriers are made innocuous. Wright (1940), in the course of one year's throat swabbing of 551 children and their nursing staff in children's wards, found that in 75% of eighty-five nurses and children with haemolytic streptococci in their throats the infection was either latent or at any rate *preceded* any clinical manifestations. Masks should be worn the whole time during dressings or inspection, and by the patient himself unless he keeps his mouth shut during the proceeding.

Wound Flora.—Wounds are, of course, prolific sources of bacteria, whose variety is exemplified in Tables I and II (p. 856). Some of the wounds containing bacteria showed little sign of infection. It is not only the inflamed or purulent wound which is a source of potentially pathogenic bacteria; and bacteria which apparently do no harm to one patient may infect another severely.

The wound flora is not confined to the obviously soaked dressing or the obviously diseased part. The skin round the wound is often heavily contaminated. In all of five tests made of infected wounds bacteria similar to those in the wound were found in the skin of the patient up to two inches from its edge. In six other tests wound bacteria were found up to four inches from the wound edge; in three instances these included *Strep. pyogenes* of the same type as that in the wound. In two cases the streptococcus disappeared from the wound during treatment, while the skin remained infected; here the skin streptococci apparently were protected by the vaseline used in the dressings from the cleansing processes of the treatment. Wounds closed with plasters may be equally unsafe. Swabbings from the outside of eighteen plasters yielded moderate or profuse growths of bacteria. Five yielded *Strep. pyogenes*; in four the type of streptococcus outside was the same as that in the wound; in the fifth the outside and inside streptococci, though Group A, did not belong to any recognized type. Pyogenic staphylococci were even more abundant on the outside of many plasters. Some measure is needed to disinfect the outside of a plaster and to minimize the escape of pus from its ends and from cracks.

When dressings, plasters, or bedclothes containing dried infected discharge are manipulated infected particles are showered into the air and contribute to the dust of the ward. A series of 154 plates exposed in the centre of a ward full of wounded soldiers grew the usual air organisms, and with the exception of a total of twelve colonies of *Staph. aureus* no pathogenic organisms were found. At no time was *Strep. pyogenes*, *Ps. pyocyanea*, or *Proteus* found on the plates thus exposed. But when plates were exposed on the beds of certain patients in the same ward while their wounds were dressed, pathogens were found on them. Fourteen patients were selected because their wounds contained "indicator" organisms like *Strep. pyogenes*. The three positive results obtained are summarized below.

Patient	Wound Swab	Pathogenic Organisms on the Air Plate
BB AC RW	Strep. 27 Strep. 4 Staph. aureus Staph. aureus	Strep. 27 (3 colonies) Strep. 4 (2 colonies) Staph. aureus (1 colony)

Similar tests in a theatre during the changing of plasters gave the following results:

Patient	Wound Swab	Pathogenic Organisms on the Air Plate
AS	Strep. Group C Staph. aureus Proteus	Strep. Group C (5 colonies)
WW	Staph. albus and aureus Strep. pyogenes Proteus	Proteus ++
WW	Strep. pyogenes Proteus Diphtheroid	Proteus ++

Bacterial contamination, often heavy, may be present in the absence of any visible soiling of the outer layers of gauze dressings. In five of six tests haemolytic streptococci and *Staph. aureus* similar to those in the wound have been isolated with ease from fingers that have lightly lifted the apparently unsoiled corners of a wound dressing.

That bedclothes are sometimes implicated is shown in a series of five tests, suggested by the work of van den Ende, Lush, and Edward (1940), in which plates were exposed on lockers during bed-making. The plates by one bed, used by a patient with a streptococcus Type 9 infection, yielded three colonies of *Strep. 9*. The patient was not

in the ward at the time; and he, apart from another patient in a bed 150 feet away, had the only Type 9 wound or throat infection in the whole ward at the time.

Channels of Hospital Wound Infection

In this section some of the means by which bacteria are transmitted to wounds will be reviewed. Some are self-evident and others may appear improbable. They are all of them suggested by observations made during the last four months.

Air.—In collaboration with Dr. C. H. Andrewes and his associates at the National Institute for Medical Research, a limited study was made of the air in wards and theatres. The bacterial content of the air in a ward is paralleled by the dust content. It is minimal during the early hours of the morning, and rises to well-defined maxima during sweeping and dusting, in dressing periods, and particularly during bed-making, when the number of colony-producing particles falling on to unit area of culture plates in unit time may be ten times that found in the quietest periods. In Fig. 2 are recorded

FIG. 2.—Colony counts of $3\frac{1}{2}$ in. blood-agar plates exposed during ward-sweeping and bed-making. Each column represents the count obtained during a five-minutes exposure. The block "S" represents ten minutes' sweeping; the block "B" five minutes' bed-making.

three short exposures during floor-sweeping and two during bed-making (summer weather, free window ventilation of ward). The rapid sedimentation of the disturbed bacteria is noteworthy, demonstrating that the institution of a relatively short quiet period in the ward before dressings are done will ensure a moderately bacteria-free air. Most of the bacteria are innocuous, and the pathogens do not appear on these plates except in the favourable circumstances described above in the sweeping and bed-making experiments. Nevertheless, pathogenic bacteria are present in the dust—for example, *Strep. pyogenes* is known to be present in the dust of puerperal fever and scarlet fever wards (White, 1936; Cruickshank, 1935; Allison and Brown, 1937; and it is obviously desirable, first, to minimize the production of infected dusts during the handling of contaminated bedclothes, dressings, and plasters, and, secondly, to keep down possibly infected dust, especially while wounds are being dressed.

The infectivity of dust-borne pathogenic bacteria for the human tissue is difficult to assess. It may be that air-borne *Strep. pyogenes*, at any rate, does not infect the wound directly, but that the small numbers of cocci in the air are concentrated in the upper respiratory tract until they are present in sufficient amount to set up a latent infection, and that the wound is infected by the relatively heavily charged droplet ejected from the streptococcus-infected throat. This danger can be met by the provision of masks for the staff during dressings. There is another process of concentration of air bacteria which must be mentioned. Hair-driers are used to accelerate the tanning of burns. A series of plates exposed to the air of a ward in the ordinary manner received about six bacteria a minute. Under the nozzle of a hair-drier operating in the same ward the average number falling

on the plate every minute was 170. The counts were not reduced when the heating element was in action: the amount of heat is apparently not sufficient to sterilize the air. Efficient filters on the machine impose too great a strain on the weak fan-motors and stop the draught. If induced air currents are to be employed for the rapid tanning of burns, machines discharging sterile air must be devised; or, alternatively, the ordinary hair-drier may be used if every possible precaution is taken to protect the air of the ward and theatre from contamination with pathogenic bacteria.

Fingers and Instruments.—The practice of lifting a series of loosened dressings with either fingers or forceps for inspection of a wound is dangerous unless the fingers or forceps are sterilized between each inspection. Often a rigorous antiseptic and aseptic ritual is observed for the actual dressing and a relaxed ritual employed for a teaching round or for procedures not immediately surgical. For instance, dressing forceps are used in a sterile manner by the surgeon inspecting plaster cases, while the porter entrusted with plaster removal carries the shears direct from case to case, merely wiping grossly purulent contaminations from the shears with bits of the dressing wool. On three occasions a profuse growth of *Proteus* was obtained from the blades of the shears after their use on a *Proteus*-infected plaster case. The proper cleansing of hands and sterilization of instruments are sometimes scamped in wards inadequately equipped with lavatory basins and sterilizers.

Dressings.—Clean dressings are frequently kept in a bag or drum and taken out with Cheatle's forceps; the handling of the forceps is often bacteriologically unintelligent, and the drum is often exposed to contamination. In one instance a patient with a pleural empyema was instructed to cough during the dressing of his wound, with the open drum of dressings immediately in the line of fire of the drainage tube. The practice of wrapping and sealing each dressing separately in a brown paper package or a towel before autoclaving has much to recommend it. The dressing is not opened until immediately before use; dressings of several sizes can be made up, and economy therefore effected, for selection of the appropriate size at the time of dressing is easy; and, finally, if the paper-wrapped dressings are sterilized in large loose bags the imperfect sterilization that sometimes occurs in the centre of tightly packed drums is avoided (Hayes, 1937). Wrapping of each dressing in cloth is less effective, for whereas new brown paper is impervious to dust-borne bacteria, open-weave towelling must be protected from the ward dust. The use of loose bags for the sterilization of unwrapped dressings is not recommended.

Baths.—Patients with open infected wounds often use the ordinary ward baths, sometimes as an integral part of their treatment. Cultures from the inside of ward baths used for wound cases have often yielded copious growths of *Staph. aureus* and many other bacteria. Haemolytic streptococci are not infrequently obtained as well. The bacterial flora of a bath is richest in the line of grease (from vaseline gauze and "tulle gras" dressings) left at the level of the water surface.

Bed-pans and urine bottles are possible vehicles of infection, especially in wounds of the thighs and buttocks. Other vehicles: foreign bodies removed from infected wounds might well be sterilized before being returned to their owners as souvenirs.

Stopping Hospital Infection at the Source

The remedies for some of the defects in the prophylaxis against hospital infections are obvious. At the risk of stressing the obvious, attention is drawn to the following points:

(a) Masks should be worn by all the staff concerned during dressing or inspection of wounds. The patient himself should not talk, or should be masked, during the dressing or inspection; all unauthorized spectators should be kept away. These points are easily observed during the formal visit of a surgeon. It is equally important to observe them at all times when wounds are handled or dressing is being readjusted. Many types of mask are inefficient. They should cover the nose, mouth, and chin with comfort (i.e., should measure about 6×8 in.), and

should contain a layer of impervious substance between at least two layers of gauze. Sheets of cellophane, grease-proof paper, or stiff brown paper may be used, slipped into masks which are made in the form of pockets. Masks must be sterilized after use, but once sterilized and dried it is sufficient to maintain them in a "socially clean" condition. Fresh cellophane or paper may be inserted before reissue; the sheet must not be allowed to crumple inside the pocket. The increased work for sterilizing and laundry services that proper masking entails may be offset by reducing the often unnecessary number of sterile towels used for the dressing of wounds in the wards.

(b) Wounds should be dressed with clean dry hands, all material handled with sterile instruments, and the instruments sterilized before further use. This technique demands large numbers of forceps or frequent boiling of instruments.* In this connexion it should be remembered that the orthodox fifteen to twenty minutes' boiling is designed to kill all but the most resistant bacterial spores. The great majority of pathogenic forms are killed after a few seconds in boiling water. We are indebted to Prof. Alexander Fleming for the following experimental results bearing on this point. Thick living cultures of *Strep. pyogenes*, enterococci, staphylococci, *Ps. pyocyanea*, and *Proteus vulgaris*, and a streptococcal pus, were each sealed in 0.75 mm. capillary tubes. Five to ten seconds in boiling water was sufficient to kill the bacteria. Spencer Wells forceps were contaminated with the cultures, or with the pus to which massive doses of streptococci, staphylococci, or *Proteus vulgaris* were added. The infecting material was in each case worked into the joints and the blade corrugations of the forceps. After the fluid was dry the forceps were closed and dipped in boiling water. A dip lasting five seconds was lethal in all tests.

Pathogenic bacteria that form heat-resistant spores play little part in hospital infection. We have been able to find only one instance of added infection by such bacteria. This was by *Cl. welchii*, which in the tissues is a reluctant spore-former. Where there is any shortage of instruments or sterilizers, therefore, it is better to give everything a brief sterilization than to take risks with some instruments and thoroughly sterilize others. Two minutes in boiling 1.5 to 2% sodium carbonate should be sufficient, *provided that all visible organic material* (pus, blood, etc.) *is removed before immersion*. The addition of the soda not only prevents rusting of the instruments but adds enormously to the lethal power of the boiling water (von Jettmar, 1938).

Dressings and plasters, especially those obviously contaminated with dried discharge, should be removed with the minimum disturbance, and perhaps damped before removal. Small dressings may be discarded into 2% lysol; large dressings should be completely wrapped in towels or sheets for removal to the incinerator, the towel being subsequently steeped in lysol. There is a tendency to regard the bandage and outer layers of a dressing, even of an infected wound, as "sterile enough" to replace over the renewed under-dressings. It is important, however, to regard all the layers of the dressing as sources of infection for other patients, and none should be used again until they have been properly sterilized.

* The following dressing equipment is recommended for a fifty-bed surgical ward. The figures in brackets refer to the numbers required for a fifty-bed ward for plastic surgery, where larger and more frequent dressings are undertaken. Dissecting forceps, 12 (18); Cheate's forceps, 3 (6); bowl forceps, to be kept by sterilizer, 1; sharp-pointed stitch scissors, 2 (with 1 in store for each 100 beds, for replacement during sharpening); sinus forceps, 1; probes, 2; one sterilizer for instruments, large enough to take Cheate's forceps (12×4×4 in.), and one bowl sterilizer, 12 in. deep and 24 in. long; lidded buckets, or "binettes," 2.

The same considerations apply to contaminated sheets and blankets. It is not an overstatement to say that blankets used by a patient with an infected wound may be as dangerous to another patient as blankets from a case of scarlet fever: the same kind of pathogenic streptococci may be present on blankets in both cases, and precautions against their dissemination should be taken. For example, the communal "theatre trolley" blankets in which a patient is wrapped for operation may become infected by one patient and so infect another. The alternative of carrying a patient to the theatre in his own blanket is equally dangerous, since the air of the theatre may become contaminated with streptococci or other pathogens. The remedy for this sort of danger is the provision of clean sheets or towelling for each patient going to the theatre that will protect the theatre blankets, both when the patient is on the trolley and when he is on the operation table. The sheet should be discarded for sterilization when the patient returns to his bed.

Blocking the Channels of Infection

Dust.—The relative importance of contact and air-borne infection of wounds is unknown, since few observations have as yet been possible on ward communities in which all the numerous channels of contact and droplet infection have been stopped. Nevertheless it is urged that dust be regarded as a potential source of infection, and treated with respect (for references see Wells and Wells, 1936; Hart, 1937; Ives and Hirshfeld, 1938). A great deal of ward dust and bacteria may be kept out of the air by applying spindle oil to the floors (van den Ende, Lush, and Edward, 1940), and it may soon be possible to treat blankets, which are prolific sources of dust, with a similar substance and render them less dusty. Wards should, so far as is possible, be dusted with damp cloths and swept with vacuum cleaners that have an efficient dust trap (some makes are very poor in this respect) or with mops impregnated with spindle oil. Adequate through ventilation is probably the most important factor in minimizing air-borne infection, but more direct evidence on this point awaits observation during winter months in black-out conditions. Ideally, each ward should have attached to it a small dressing theatre, to which all beds are removed for the treatment of wounds. But, wherever the dressings are done, every effort should be made to reduce the air dust for at least one hour before dressings. Wounds should be exposed to the air as little as possible. During unavoidable delays in the treatment of an exposed wound it should be covered with a sterile towel.

Instruments.—These have been mentioned above; it is sufficient to stress here the necessity for sterilizing instruments and material used by plasterers and similar attendants. Needles used for injection may carry infection. A rinse in spirit is not enough to resterilize a needle: all needles should be boiled.

Baths.—The cleaning methods in common use have been found ineffective. Even hard scrubbing with diluted disinfectant, such as lysol, does not remove either the grease or the bacteria. Nor does "boiling" water poured from a kettle over the bath surface. Ether soap is likewise ineffective. The method of cleaning adopted must satisfy four requirements: it must remove grease and remove or kill bacteria; it must be reasonably quick and easy; and it must not be expensive. Two methods tested have been found to fulfil these requirements. The first consists in scrubbing with two ounces of undiluted lysol. A mop may be used, or, if rubber gauntlets are worn, a cloth is probably better. *Lysol must not be splashed on to the hands of the cleaner*, and the bath must be very thoroughly rinsed after cleaning.

The second method is to remove the gréase with a cheap domestic cleaning powder, and to disinfect with 1/10 or 1/5 lysol. It is possible to combine the two by mixing two or three ounces of the powder with five or six ounces of dilute lysol in the dry bath, and then rubbing the mixture thoroughly over the entire surface of the bath by hand with a cloth, for a period of not less than two minutes. A mop is unsuitable. *A rubber gauntlet should be worn to protect the cleaner*, and the bath must be thoroughly rinsed afterwards. It may be objected that the use of a cleaning powder will wear the enamel. Since most hospital baths inspected have been found already chipped, and since no method short of heat will sterilize a chipped patch, a general wearing of bath enamel more rapid than usual will be an advantage, for it should lead to more frequent re-enamelling. Arm baths may be cleaned in the same way. Those with chipped enamel, or those metal baths made in two pieces with an uncleanable angle between them, should be discarded.

Whatever cleaning method is used it must be thoroughly applied, and the cleaner's responsibilities clearly explained to her.

Personnel: General Considerations.—The lapses in prophylaxis recorded above were for the most part unforeseen failures in a carefully devised and rigorously imposed routine, and such lapses may be expected at one time or another in most surgical wards and theatres. The fact that some hospital infections are not attended by striking clinical signs should not lead to an underestimation of their importance. If channels of hospital infection are left open, sooner or later serious infections will pass through them.

Usually the routine of asepsis and antisepsis is adequately observed in the operation theatre. But so firmly is this routine associated with the discipline of a theatre that the one is relaxed automatically with the other; manipulations are made in a ward or casualty department with one-tenth the care devoted to the same manipulations in the theatre. A similar association of the routine with the presence of surgeons themselves leads to its relaxation in a quite indefensible manner when patients with open wounds are handled by porters (plasterers, barbers, etc.) or by the staff of non-surgical departments (e.g., x-ray or massage). There is a tendency, perhaps unconscious, to regard all sepsis as having the same significance, and consequently to relax precautions when dressing a series of septic wounds, on the grounds that transfer of septic matter from one septic wound to another will be of little consequence.

Careful prophylaxis must be associated with the wound itself and the routine be ordered to one end—namely, that bacteria can neither escape from nor get into the wound or its dressings. This simple aim, together with a recognition of reservoirs of potential infection in personnel, should ensure a proper prophylaxis. The imposition of this idea will not be easy, for until the staff (and patients) are educated they will feel that the observance of such prophylaxis will restrict the work of a ward to an intolerable degree. The biggest burden of education will fall upon the senior nursing staff, who will add to their duties those of a bacteriological policeman, with powers of summary jurisdiction. We suggest, for consideration, the appointment in a hospital of a person of sufficient authority and seniority, from either the qualified or the nursing staff, whose sole occupation would be the supervision of aseptic and antiseptic measures in the hospital and the training of nursing staff in such measures and in their bacteriological significance. A constant watch is needed; too often carefully imposed precautions are wasted. Examples of common sources of danger are the patient who persists in inspecting his own

wound; the attendance at a wound dressing of a crowd of high-spirited convalescent soldiers; or the nurse who, in fear of the authorities' displeasure, uses an antiseptic gargle immediately before a throat-swabbing designed to trace the source of an infection.

Ritualized precautions are dangerous because their observance is considered sufficient to meet all contingencies, and because in certain circumstances they are useless or even harmful. The precautions should be subject to modification at the decision of the surgeon, and constantly be put to bacteriological test. But if they are allowed to degenerate into ritual the principles upon which they are based will be forgotten, and any change that must be made will be greeted with bewilderment or annoyance.

Conclusions

From observations made mainly in two hospitals we conclude that hospital infection of war wounds is far more common than is generally suspected, and is at least as frequent as other investigators of the problem have insisted on in the past. There is no reason to suppose that other hospitals differ greatly in this respect from those with which we were concerned. This statement is made with no derogatory intention, for most hospital infection is recognizable only by detailed bacteriological investigation; it is not clinically recognizable, because it either produces little ill effect or merely adds to existing illness. Nevertheless the barely recognizable infections must materially add to the burden both of the patient and of those who treat him. An average decrease of three days in each wounded patient's stay in hospital through the elimination of added infection would represent a marked economy of emergency medical services. Dramatic or fatal effects, in either patients or personnel, that are obviously due to hospital infection are happily less frequent. They cannot, however, be avoided unless steps are taken to eradicate both the "latent" and the manifest hospital infections. We have attempted to present a general picture of the sources of infection and its modes of spread and to indicate the simpler means by which the latter may be overcome.

To this end close co-operation of hospital staff and bacteriologist is required. The bacteriologists of this investigation have benefited enormously from the generous submission of the hospital staff to what was of necessity bacteriological "snooping." Such snooping would be far from fruitful in an institution if the discovery of hospital infection were regarded as a sign of gross inefficiency. Carelessness and ignorance undoubtedly contribute to hospital infections, not least on the part of the patients themselves. But in the complex hospital environment there is so much opportunity for hospital infections that they cannot be regarded as entirely avoidable. They are the common concern of surgeon, nursing staff, and pathologist. Lapses are occasions for examination, not for the distribution of blame.

We are aware that we have not demonstrated anything new, though our examples are up to date. Nor are the measures advocated new, except some of those concerned with minimizing dust. But we insist that they should be applied with intelligent appreciation of the risks of hospital infection. The patient at present is not well enough protected by the automatic rituals of asepsis and antisepsis.

In summary, we advocate: (1) the application of aseptic and antiseptic measures to *all* manipulations of wounds, and not only to those made in the sanctity of the operating theatre; (2) the recognition of potential reservoirs of infection in the hospital environment, and the reduction of their menace so far as is possible.

Note.—Throughout the paper "Staph. aureus" refers to coagulase-positive streptococci; coagulase-negative Gram-positive cocci are labelled "Staph. albus" or "micrococci."

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TREATMENT OF BRONCHOPNEUMONIA UNDER THE AGE OF ONE YEAR

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Bronchopneumonia under the age of 1 year is generally considered to be a particularly severe disease, carrying a high mortality rate—up to 90% (Moncrieff, 1934). At the same time, unless complicating one of the acute exanthemata or an acute gastro-enteritis, it cannot be regarded as a common disease, and it is difficult to collect enough cases treated in hospital to enable conclusions to be drawn as to the efficacy of methods of therapy. During the severe winter of 1939-40 a number of infants suffering from various types of pneumonia came into hospital under my care, and of these I have selected those in whom the prognosis should have been poor—i.e., infants in the first year of life with extensive and widely disseminated patches of consolidation. The following is a brief summary of each case.

Case Reports

Case 1.—Female aged 4 weeks, admitted December 13, 1939. Severe bronchopneumonia following acute bronchitis, with consolidation of all zones of right lung and middle and lower zones of left. Maximum temperature 103° F., pulse 146, respirations 90. *Treatment:* Oxygen tent fourteen days; sulphapyridine 0.125 gramme four-hourly, total 5½ grammes; subcutaneous drip saline four days. *Feeds:* Two-hourly alternating breast milk and half-strength normal saline. In hospital twenty-four days.

Case 2.—Male aged 3 months, admitted January 9, 1940. Severe bronchopneumonia secondary to common cold. General condition poor. Temperature 99° F., pulse 160, respirations 72; maximum respirations 110. *Treatment:* Subcutaneous drip saline twenty-four hours; oxygen tent one week; sulphapyridine 0.25 gramme four-hourly, total 6 grammes. *Feeds:* Two-hourly, alternating humanized dried milk and half-strength normal saline. In hospital fourteen days.

Case 3.—Male aged 4 weeks, admitted January 20, 1940. Common cold followed by moderately severe bronchopneumonia. Consolidation of left middle and lower zones and right lower zone. General condition poor, with marked cyanosis. Temperature 100.4° F., pulse 200, respirations about 40 and irregular; maximum respirations 74. *Treatment:* Subcutaneous drip saline three and a half days; oxygen tent ten days; sulphapyridine 0.125 gramme four-hourly, total 3 grammes. *Feeds:* Two-hourly, alternating humanized dried

milk and half-strength normal saline. Convalescence complicated by abscess of chest wall with slow healing by granulation. In hospital eleven weeks.

Case 4.—Female aged 6 months, admitted February 1, 1940. Common cold followed by severe bronchopneumonia, with consolidation of right and left upper zones and right lower zone. Temperature 103.4° F., pulse 200, respirations 100 and irregular. *Treatment:* Oxygen tent; subcutaneous drip saline; sulphapyridine 0.25 gramme four-hourly. *Feeds:* Two-hourly, alternating boiled cow's milk and half-strength normal saline. Died in convulsions ten hours after admission.

Case 5.—Male aged 5 months, admitted February 9, 1940. Moderately severe bronchopneumonia complicating gastro-enteritis; marked dehydration. Maximum temperature 106° F., pulse 220 and irregular, respirations 124 and irregular. *Treatment:* Oxygen tent; sulphapyridine 0.25 gramme four-hourly; subcutaneous drip saline. *Feeds:* Two-hourly, alternating humanized dried milk and half-strength normal saline. Increasing cyanosis and death three days after admission.

Case 6.—Female aged 11 months, admitted February 22, 1940. Severe bronchopneumonia following a common cold, with consolidation affecting whole of left lung and lower zone of right. Infant showed typical features of cretinism, and, despite severity of pulmonary lesions, temperature, pulse, and respirations remained at a comparatively low level throughout the disease. *Treatment:* Oxygen tent ten days; subcutaneous drip saline forty-eight hours; sulphapyridine 0.25 gramme four-hourly, total 7 grammes. *Feeds:* Two-hourly, alternating boiled cow's milk and half-strength normal saline; feeding was very difficult. The pulmonary consolidation resolved and cretinism responded to thyroideum B.P., so that the infant was weaned and given a normal mixed diet. Unfortunately she later developed another cold, which was again complicated by bronchopneumonia, and this proved fatal. This case has previously been reported (Gill, 1940b).

Case 7.—Female aged 10 months, admitted February 29, 1940. Severe bronchopneumonia following a common cold. Consolidation of both lower zones and left upper zone. Temperature 104.4° F., pulse 188, respirations 68; maximum respirations 112. *Treatment:* Oxygen tent; sulphapyridine, initial dose 0.5 gramme, then 0.25 gramme four-hourly, total 4 grammes. *Feeds:* Two-hourly, alternating boiled cow's milk and half-strength normal saline. Condition complicated on third day by right pleural effusion, which was aspirated and found to be heavily blood-stained, but sterile on culture. Further sulphapyridine given intramuscularly, 1 gramme four-hourly; total 6 grammes. Relapse on fifth day and fatal termination with hyperpyrexia.

Case 8.—Male aged 8 months, admitted April 24, 1940. Common cold, followed by diarrhoea and later bronchopneumonia, affecting especially the right upper and middle zones. Temperature 101° F., pulse 136, respirations 64. *Treatment:* Oxygen tent forty-eight hours; sulphapyridine 0.5 gramme, then 0.25 gramme four-hourly, total 3½ grammes. *Feeds:* Two-hourly, alternating boiled cow's milk and half-strength normal saline. In hospital twenty-three days.

Case 9.—Male aged 11 months, admitted April 25, 1940. Acute bronchitis followed by severe bronchopneumonia, with consolidation of left middle and lower zones and right lower zone. Temperature 103° F., pulse 188, respirations 128. *Treatment:* Oxygen tent ten days; sulphapyridine 0.5 gramme, then 0.25 gramme four-hourly, total 6½ grammes. *Feeds:* Two-hourly, alternating boiled cow's milk and half-strength normal saline. Complicated on eighth day by right pleural effusion, which was aspirated and found to be heavily blood-stained, but sterile on culture. In hospital four weeks.

Case 10.—Male aged 6 months, admitted April 28, 1940. Moderately severe bronchopneumonia following acute bronchitis. Temperature 105° F., pulse 160, respirations 48; maximum respirations 60. *Treatment:* Oxygen tent twenty-four hours; sulphapyridine 0.5 gramme, then 0.25 gramme four-hourly, total 2½ grammes. *Feeds:* Two-hourly, alternating boiled cow's milk and half-strength normal saline. In hospital nineteen days.

Case 11.—Male aged 5 weeks, admitted May 18, 1940. Severe bronchopneumonia complicating gastro-enteritis, with consolidation of right upper and left middle and lower zones.