THE PREVENTION OF WOUND CONTAMINATION

GENERAL CONSIDERATIONS

There is a greater opportunity for wound contamination in warfare because of the lapse of time after injury before definitive care can be given, the nature of the wounds, and the lack of facilities. Hence greater care must be exercised.

Even though a wound may already be infected, additional contamination can cause serious harm.

The contaminating organisms include:

A. Primary contaminants:— which are carried into the wound at the time of injury. These include the gas forming anaerobes and the bacillus of tetanus (see sections on tetanus and gas gangrene), and

B. Secondary Contaminants:— the most important of which are:
   1) Streptococci of Group A (Lancefield-Hare).
   2) Staphylococci (coagulase positive).

Other bacteria are also found, but these are relatively infrequent and of far less importance.

THE PATHOGENIC STAPHYLOCOCCI AND STREPTOCOCCI ARE USUALLY NOT INTRODUCED INTO WOUNDS AT THE TIME OF INFILCTION, BUT THEY GET IN LATTER: EVEN WHEN THESE WOUNDS ARE COVERED WITH PLASTER, SUCH INFECTIONS ARE DANGEROUS. THEY CAUSE SERIOUS DELAY IN WOUND HEALING AND MAY EVEN CAUSE FATALITIES.

The normal habitat of these bacteria

They are rarely if ever found to reside naturally on objects which normally cause injuries. They are seldom present on the patient's skin or clothing. They are found as follows:

Coagulase positive staphylococci — in the nose and throat, especially the nose.

Group A Streptococci — in the nasopharynx.

The contamination occurs mainly when the wounds are exposed during:

1) First aid
2) Definitive surgical care
3) Dressings

SOURCES OF WOUND INFECTION

1) From the upper respiratory tract of those attending the wound and of the patient during exposure of the wound.
2) From already infected wounds in other patients, by:
   a) Attendants' hands.
   b) Unsterile instruments and accessories used in the treatment of the wounds.
   c) Dressings contaminated by poor technique.

IN PATIENTS WITH ALREADY INFECTED WOUNDS THESE ORGANISMS ARE FOUND ON THE SKIN, BEDDING, DRESSINGS, AND IN THE AIR ABOUT THE FLOOR OF THE BED. IF THE FLOOR HAS BEEN SWEEP AND THE BEDDING AGITATED IN SUCH A CASE, THE AIR ABOUT ADJACENT BEDS MAY CONTAIN CONTAMINATED DUST WHICH MAY DROP INTO THE WOUNDS, IF THEY ARE DRESSED AT SUCH A TIME.

Individuals with upper respiratory or sinus infections are especially apt to cause wound infection.

Infections of wounds with a specific organism may spread through a ward like wildfire.
PREVENTION OF WOUND CONTAMINATION - Requires rigid asepsis and dressing technique at all times. This technique must be observed at all times from the time first aid is given to the time when the patient is discharged. It is as follows:

1) Masking of patient and attendants at all times when the wound is handled (even during first aid). The mask must cover the nose.
2) Adequate heat sterilization of all instruments and other accessories (sutures, dressings, etc.) before use and after use on infected cases.
3) Use of sterile gloves by the surgeon (these should be considered unsterile after they have been put on).
4) Rigid asepsis.
5) Good dressing technique:
   a) Dress with sterile forceps.
   b) Avoid touching dressings or patient's skin or bed with fingers.
   c) Do dressings when the air is quiet and has been so for several hours (not after floor has been swept, etc.)
   d) Avoid agitating dressings and bed clothes. Ward quiet and no milling about.
   e) Avoid many spectators.
   f) Segregate clean from infected cases:
      Different wards if possible.
      Wide bed spacing.
      Curtains.
   g) Segregate patients with upper respiratory infections. Ward personnel with such infections should not attend wounds.
   h) Reduce number of dressings to a minimum. Keep wound covered at all times. No peeking to see how it is getting on.
   i) After first aid dressing is applied - do not remove it until definitive care is given. Are justified in examining such a case only in the presence of grave complications such as hemorrhage or sucking wounds of the chest, and only if you have an adequate set-up for aseptic care.

Note: Always consider bed clothes as contaminated. Common operating room blankets are bad. Patient not to go to the operating room with his own bed clothes.

DISPOSE OF USED DRESSING CAREFULLY.
Sources of wound contaminants and their control.

The surgeon and the bacteriologist must work together to solve this phase of wound infection.

Surgeon must not accept wound infection as inevitable.

Minor infections as well as major infection are important, almost equal to avoid major. Modern surgery especially since specialties (nerve and tendon, plastic, craniocerebral, genitourinary, gastrointestinal etc.) built up on strict asepsis and respect for tissues.

In war many factors are at work which tend to increase the amount of infection.

Breakdown of technic because of excitement.

Great amount of tissue damage.

Delay in patient arriving at hospital.

Many contacts of patient with potential carriers.

Need for speed.

Facility of facilities and equipment.

Difficulty in caring for patient after operation.

Great numbers of young inexperienced surgeons.

Gradual heightening of bacterial virulence as war goes on.

Gradual lowering of patients resistance as war drags on.

Infection of wounds is due to specific bacterial invaders.

These invaders have certain definite reservoirs or sources.

These reservoirs must be controlled.
Dressing of antiseptics into wounds will no more solve the problem than will the malairial problem be solved by quinine or the syphilis one by arsenic.

Control measures must be directed against specific organisms - e.g., the specific spirochete of lues, the malairial organism and its vector, the organisms of wound infection. To strike out blindly by filling a wound up with chemicals and neglect to prevent the organisms from getting in is not intelligent.

The surgeon must disabuse his mind of the idea that simply because a wound contains bacteria that a few more will do no harm, or that because it is infected it cannot be harmed by additional bacteria.

WHAT ARE THE ORGANISMS WHICH CAUSE WOUND INFECTION?

Streptococci of group A (Lancefield-Hare)

Hare 1376 infections due to strep. found that 1307 of them were due to group A strep. (Also known as Strep. pyogenes, and as beta hem. strep.)

Staphylococci - coagulase positive.

Most but possibly not all staphylococcal infections are due to coagulase positive staphylococci.

Gas forming anerobes - (See section on gas gangrene)

Tetanus " " " tetanus

Other bacteria - form a very minor percentage of wound infections, and their source and modes of transmission are not as yet traced. Probably they are carried in much the same manner as the two above named, viz. Strep. A. and coagulase positive Staph.
WHEN DO THESE ORGANISMS GET INTO WOUNDS?

They rarely get in at the time of injury except as follows:

- Mouth bites
- Injuries from operating knives of infected cases.
- Tonsillar stumps.
- Safety pin injuries from infected cases.

These wounds directly implant into wound organisms already acclimated to human tissues, they are immediately invasive, and of extreme virulence. They are rarely found in wounds examined immediately after injury.

Wounds are found to contain many bacteria which have been introduced at time of injury:

<table>
<thead>
<tr>
<th>Sas</th>
<th>Dimitza and Gatscher</th>
<th>Sviridov</th>
<th>Pulaski, Melency and Spaeth</th>
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<tbody>
<tr>
<td></td>
<td>found almost 100% to</td>
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<td>be contaminated, but</td>
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<td>since virulence tests</td>
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<td>were not performed</td>
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<td>nificant.</td>
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Where the contaminating organisms have been typed

Strep. A are extremely rare as primary contaminant, and coagulase positive staph is low.

- Here 355 wounds - found no group A strep.
- 4 " showed them later, and 2 of these became infected.
- 10% of wounds showed coag.pos.staph. immediately
- 35% " " " " later.

Ewing, Scott and Geiger found no group A. strep.

immediately, but in 2 later. % wounds 5 only)
They are found to be more often present in older wounds than in younger wounds:

Fleming and Porteous 1919-20% of compound fractures contained hemolytic streptococci when arriving at base.

90% contained them after a week at the base.

Spencer, working for the Medical Research Council England, has found a higher incidence of strep.pyogenes and coagulase positive staph. in older wounds than in early wounds.

The following figures are taken from his tables and only a few of the recorded organisms are here noted.

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>31 air raid wds.</th>
<th>15 plastered under 48hrs.</th>
<th>21 plastered wounds over 48 hrs &amp; 3 weeks.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td># %</td>
<td># %</td>
<td># %</td>
</tr>
<tr>
<td>Strep. pyo.</td>
<td>2 0.65%</td>
<td>5 33.5%</td>
<td>20 70.0%</td>
</tr>
<tr>
<td>Staph.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coag. positive</td>
<td>2 0.065%</td>
<td>9 70.0%</td>
<td>19 70.0%</td>
</tr>
<tr>
<td>Coag. neg.</td>
<td>13 42.0%</td>
<td>5 33.5%</td>
<td>7 24.0%</td>
</tr>
<tr>
<td>E. Coli</td>
<td>6 19.0%</td>
<td>4 30.0%</td>
<td>15 51.0%</td>
</tr>
<tr>
<td>Cl. Welchii</td>
<td>3 25.3%</td>
<td>1 7.7%</td>
<td>2 6.7%</td>
</tr>
</tbody>
</table>

Miles et all sum up their evidence:
And another table recording results as to lapse of days between wounding and swabbing.

1-3 days 4-12 days 13-40 days.

Strep. hemo. 8.7% 13.5% 20.0%
Staph. sur. 50.0% 50.0% 88.5%
Cl. Welchii 34.7% 11.1% 90.0%

Miles et all sum up their evidence or hospital infection under three headings:
1. Wound flora is found to change in groups of wounds at different stages:

<table>
<thead>
<tr>
<th>108 Dunkirk wounds</th>
<th>49 Air raid wds.</th>
</tr>
</thead>
<tbody>
<tr>
<td>(late wds.)</td>
<td>(Early wds.)</td>
</tr>
<tr>
<td>Strep. hemo</td>
<td>31.4% 14.3%</td>
</tr>
<tr>
<td>Staph. sur.</td>
<td>54.3% 55.1%</td>
</tr>
<tr>
<td>Cl. Welchii</td>
<td>22.8% 4.1%</td>
</tr>
</tbody>
</table>
2. Wounds sampled at intervals will show additional contaminating organisms.
   a. 17 of 20 g.s.w. received from France (Dunkirk) became contaminated with additional bacteria. 
      Staph. aur. in 9 cases. 
      Strep. pyo. in 8 cases. 
      Aerog. spores in 3 cases. 
      Other bact. in 10 cases. 
      In 7 of these 166 cases appearance of new bacteria was assoc. with symptoms. 
      Fresh contaminants appeared between 3rd and 27th day.

   b. 10 of 37 air raid wounds became infected with additional bacteria between the 3rd and 18th day.
      Micrococi appeared in 5 cases
      Strep. pyo. in 4 cases
      Diphtheroids in 3 cases
      Coliforms in 3 cases
      Staph. aur. in 2 cases
      Other bact. in 2 cases

   c. 6 of 24 wounds encased in plaster were found to harbor new organisms ate plaster changes.
      6 wounds acquired 1 new species.
      4
      1
      3
      1
      1

  Summing up a, b, and c they say that of 74 wounds studied 43 or 57% acquired new organisms.

3. Definitely identifiable bacteria have been shown to spread through wards.
a. *E. proteus* found in three wounds after plaster change in the same theater.

b. Unusual type of *strep.* A 23/11 was found in three patients in the same ward.

c. Ward infections by typed *hemo. strep.* whose distribution was known.

One group of 5 men infected with *strep.*

One group of three " " " 4

Two patients infected with group 11 by o.r. nurse who carried this organism in her throat.

In connection with the spread of bacterial strain thru a surgical ward an experience of Colebrook (EMJ, '41,11/22, p 743.) Flying officer with severe fatal burn infected with type 11 *strep*., sulfanilamide resistant. Followed by 13 cases with same drug-resistant bacteria in infected burns, unsuccessful skin grafts, etc.

Spooner (EMJ, 11/22 '41, p743) in his hospital 65% of infections due to *hemo. strep*., of these 1/10th cross inf. Describes 2 epidemics: 1 with sulfas-resistant *strep.* involving 15 wds and 3 throats, another with type 4 *strep.* in 5 burns (Foci) 4 wds, and 19 throats.

Hare has shown similar spread of identifiable bacteria in wards under study at Toronto.

**Therefor it would seem justified to conclude that pathogenic bacteria (group A *strep.* and coag-positive *staph.*) do not often get into wounds at the time of infliction but get in later, even when these wounds not are covered with plaster. It is in accordance with known facts to plead that these bacteria lie dormant in wounds only to become culturable later, such is not the usual behavior of these bacteria, while it is true that they may not often cause fatal infection, that they may cause no symptoms or lead to cellulitis, delay in healing or disturbance does not detract from their importance.**
TO ANSWER THE QUESTION AS TO HOW THESE BACTERIA GET INTO WOUNDS WE MUST FIRST KNOW WHERE THEY ARE FOUND IN NATURE.

WHERE ARE THESE BACTERIA FOUND IN NATURE?

Coagulase positive staphylococci

Found in 5% of normal skins according to some, on 20% according to others. Have found in 10% of wounds immed. and in 35% later.

Found in nose and throat (esp. the nose) in 43.4% Gillespie et al.

32% Smith.

Group A streptococci

Nasopharynx 7% Hare. (20% carry hemo. strep. but not all group A.)

6-13% Straker.

Skin of hands Colebrooke about 4%

Hare none unless strep. carrier (u.r.i.)

On and about patients with

Upper resp. infection

Hands

Drieff,

Droplets

Infected wounds

(Incl. burns

Cruickshank

(Incl. puer.

Cruickshank

White

Skin

Bedding

Dressings, even outer

Skin about wds.

Air about

Floor about bed

Scarlet fever wards

Wards with pts with U.R.I.
These bacteria are rarely found

In ordinary dust and dirt (Aher

In air of city or country (Hare Schaefer 1935, no path.

bacteria in air, not typed.

Skin of hands of normal person (Hare v.s.

Skin of legs (Hare

Perianal skin in parturient women (Cruickshank 160 pts.

THEREFORE IT SEEMS THAT THESE BACTERIA ARE RARELY IF
EVER FOUND TO RESIDE NATURALLY ON OBJECTS WHICH NORMALY
CAUSE INJURIES, THAT THEY ARE Seldom PRESENT ON THE
PATIENT'S SKIN OR CLOTHING, AND EARLIER WE SHOWED THAT
THEY ARE Seldom PRESENT IN THE WOUNDS IF THESE ARE CARE-
FULLY CULTURED. HENCE THEY MUST GET IN LATER AND IT IS
LOGICAL TO ASSUME THAT THEY COME FROM THE SOURCES LISTED
ABOVE. THEY ARE CARRIED INTO THE WOUNDS BY SOMEONE
BY DROPLETS FINGERS OR UNSTERILE DRESSINGS OR INSTRUMENTS,
OR FALL IN FROM THE AIR WHICH HAS BEEN CONTAMINATED BY
A SOURCE.

HOW DO THESE BACTERIA GET INTO WOUNDS?

Carried in by humans:

Bypasses in aseptic technic

Routine is poor

Routine is not observed

Droplet contamination from nose and throat

Hands not protected by gloves

Glove punctures
Unsterile dressings
Unsterile instruments
Hasty chemical sterilization
Inadequate heat sterilization
No sterilization

Poor dressing technic
Finger dressing
Dressings exposed

Unsterile ligatures or sutures.
Sinus infection in operating surgeon
Dressing or bandage scissors

Air borne in air about patients with infected wounds especially during agitation e.g. bed making. (Scarlet and puerperal wards) Burns, Bath tubs, bedpans and urinals.
Hair dryers for drying burns under tunic treatment.

WHEN DO THE BACTERIA GET INTO WOUNDS?

At time of first aid if this hasty and careless.

Each time wound is examined if aseptic precaution not taken, masks not used etc.

At time of operation, not so much from unsterile instruments as from people e.g. surgeon, nurses, orderlies, patient himself if not masked.

Talking or breathing over wounds
Postoperative dressing if careless

Dressing with "bare finger" technic.

The hospital infections of impetigo and puerperal sepsis, are the modern counterparts of severe epidemics of hana, gangrene, erysipelas etc (Holmes, Semmilweis, Pirogoff) Wound sepsis today is due to same causes which are better controlled but still not perfectly so and control measures tend to break down due to haste and carelessness which may attend war surgery.
HOW MAY THESE BACTERIA BE PREVENTED FROM GETTING INTO WOUNDS?

We should be guided by intelligence and not ritual.

The safeguards of asepsis must go thru the hospital and not stop in the operating room. Wards, x-ray room, physiotherapy, etc.

The responsibility rests on all the staff and not just the surgeon, he however must constantly evaluate tech. and procedures with reference to transfer.

Remember that no wound is so bad that it cannot be made worse.

Peace time technic may not be adequate for war time, perhaps instead of fewer precautions we should take more and be more particular about wound exposure. Badly damaged tissue, wounds seen late and patients who may be exhausted from exposure may withstand bacterial invasion very poorly.

Drugs may help but nothing can excuse us from taking all possible precautions to prevent contamination.

Sources must be controlled:

- Masking at all times
- First aid
- Emergency care
- Operation
- Dressings
  (Mask patient also!)

Good dressing technic

- Press with sterile forceps
- Avoid touching dressings or patients skin, or bed with fingers.
- Do dressings when air is quiet and has been so for several hours.
- Avoid agitating dressings, and bed clothes.
- Ward quiet and no milling about
- Avoid many spectators

* Gone are (should be) the days when are gaily laid bare for the round of inspection by the big white chief and his retinue of maskless followers.*

Avoiding exposure of clean wounds near infected ones. Any wound not healed must be looked upon as a potential source even if there are no symptoms.

Proper sterilization of everything that will come into contact with wounds or has done so.

Dressings and instruments
Blankets and bedclothes are contaminated
Common o.r. blankets bad.
Pt. not to go to o.r with his own bedclothes
Dressings should be carefully disposed of

Air sterilization methods to date do not seem
 to have become accepted. Even if used we must
still observe a careful aseptic technic.
Oiling of floors, and bed clothes may help.

**Protect the wound at all times:**
Covered at all times.
No peeking
Segregate clean from infected cases
different wards if possible
wide bed spacing
curtains
Reduce number of dressings and thus number of ex-
posures.

An infected wound may be contaminated with other bacteria,
and these may be more virulent than those already pre-
sent or may establish symbiotic relationship.

After the first aid dressing has been applied it should
not be removed until definitive care can be given.
Nothing is to be gained by peeking or tampering. Only
in such grave complications as hemorrhage or sucking
wounds of the chest are we justified in looking at wounds
with inadequate precautions for aseptic care.

Even if the definitive care which may be given is most
minimal this is **no** excuse for haphazard acre and dis-
regard of asepsis.