Clean air symposium—Part I

Clean air in the operating room

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The idea that airborne bacteria might be a common source of infection of surgical wounds in the operating room is as old or even older than Lister, and remains still a hypothesis not universally accepted. In this contribution I shall concentrate on the evidence for believing that airborne infection in the operating room is common; but I shall touch only briefly on the technical details of operating in clean air.

One difficulty in accepting the idea of airborne infection relates to the whole bacteriological concept of "airborne bacteria" which covers a much wider field than infection in the operating room, including as it does the epidemiology of many infectious diseases. It is generally agreed that living bacteria, in contrast to spores, are rarely present in the air by themselves, tuberculosis being one of the few for which experimental evidence is available. For this reason the insistence on filtration of air to submicron size, which is a strong selling-point of air engineers in the clean-rooms of industry, evokes no great sympathy from the bacteriologist. Postoperative infection by spore-bearing organisms, for which submicron filtration might be needed, is so rare

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in orthopaedic surgery as to be negligible, as evidenced in my own series of 85 infections in 5,000 operations where one case only was due to spore-bearing organism (*Bacillus cereus*), and even here there were good grounds for believing that it was not acquired at the time of operation.

On the other hand, few people would contest the idea that wounds might very well become infected by particles of dust, or by airborne epithelial scales, falling into the open wound and emanating from persons in an operating room. On a dust particle 10μ in diameter bacteria may exist in considerable numbers, and particles of this size are able to settle quickly from the air. What I shall describe of my experiences with airborne infection in the operating room relates to the very simple concept of *dust-free* air produced by filtration only in the 1- to 2-μ range and not to filtration at 0.3μ with the HEPA filter.

The requirements of industry for the absence of particles of any kind in the environment are much more exacting than is necessary in an operating room where the air is often heavily laden with particles of dust generated by sterile fabrics. Particle counting in the operating room, although a convenient and quick procedure, is of doubtful value in relation to asepsis in operating rooms and must inevitably increase the costs, quite unnecessarily, of any installation using it as a guide towards air sterility.

In the theory of airborne infection in the operating room a crucial point revolves round the "dose response" to bacterial inoculation. It remains still a matter of very grave doubt as to how many organisms are required to cause a wound infection. In the face of experimental work such as that of Elek and Conen[1] it would seem impossible to accept airborne infection in the operating room, because these workers found it necessary to inject several millions of organisms from a mixed culture of *Staphylococcus aureus* obtained from different human sources, before a subcutaneous collection of pus could be produced in human volunteers. Even in the presence of a foreign body, such as a silk stitch, it was found necessary to inject organisms to the tune of many thousands before infection supervened.

Nevertheless, a small number of careful studies are in the literature of staphylococcal infections of surgical wounds which have been proved, by phage tests, to have come from persons identified in the operating room as disseminating these typed cocci. Thus Payne[2] identified an anesthetist responsible for causing 33 infections in the operating room. That one person could shed so many infected particles that, when diluted in the volume of air of the whole of the operating room, more than perhaps one infected particle could fall into the wound would seem improbable. It can therefore be taken as proved that extremely small inoculations from the air in the operating room *can* cause human wound infections in surgical practice, even though this does not show how frequently this mode of infection is responsible for ordinary postoperative infections.

In airborne infection it must be fairly obvious that it is the statistical element of pure chance which determines whether an organism enters an open wound from the air. In total hip replacement the target area for viable deposition is probably much smaller

than the target presented by the area of the whole wound. In an extensive hip exposure, the soft parts may be just as capable of destroying bacteria deposited from the air as any other large surgical exposure such as a radical mastectomy, but the "bull's eye" of the target is the foreign body which in this operation has inside it a cavity containing an ideal culture medium, at blood heat, incapable of exchanging fluid with the defense mechanism of the body. In such a case no temperature or other systemic reaction need follow the operation; the wound can heal normally and the patient may leave the hospital with organisms, often considered to be nonpathogenic, spreading on the surface of the implant in soft tissues whose vitality is depressed by contact with the foreign body.

When self-curing acrylic cement has been used there may be a zone of tissue, perhaps 500μ in thickness, whose vitality is seriously, although only temporarily, prejudiced by the thermal and chemical trauma produced by the cement. We thus have circumstances ideal for the survival of a type of low-grade infection with which many bacteriologists until recently have not been familiar. For most bacteriologists infection in operating rooms is a matter relating most often to a specific organism, because usually they are called in only when epidemics of operating room infection have occurred. But in total hip replacement we are not concerned with epidemics but with a constant small infection rate year in year out, and we are concerned with a wide spectrum of wholly unrelated types of organism in which the *S. aureus* was incriminated in only about 50% of my own cases.

Acrylic cement and deep infection of implants

It is important that I should make adequate mention of the role of acrylic cement in changing the picture of infection in orthopaedic implants, because this symposium on operating room asepsis would not be taking place were it not for the use of acrylic cement in orthopaedic surgery. It is absolutely necessary that we should satisfy ourselves that infections following the use of acrylic cement are caused by bacteria and are not chemical or allergic rejections which mimic infection. The idea of allergic rejection has been advanced because of failure to grow organisms from implants in certain cases where otherwise bacterial infection would be the obvious explanation.

Important evidence against allergic or chemical rejection is that in my 14 years of experience with this cement since 1958, amounting to well over 6,000 cases followed for over 1 year, there has been no bilateral complication to give rise to a serious suspicion of allergy. Nearly 25% of our arthrosic patients eventually come back to have the second hip operated on, so that we have many hundreds of bilateral operations available for study. In my total series of 85 infected hips it is interesting that 20 of these (approximately 25%) are in patients in whom the opposite hip carries a completely successful implant. Nine patients had the infected hip operated on before the good hip, and nine had the infected hip operated on after the good hip; two were operated simultaneously, only one hip becoming infected.

Also against the possibility that acrylic cement might produce compli-

cations simulating bacterial infection, I advance the simple fact that as a result of taking measures against exogenous infection in the operating room the infection rate has fallen from about 7% in 1959 to 0.5% at the present moment. If acrylic cement were to have been the main cause of infection-like complications in 1959, 1960, and 1961, this fall in infection rate could not have occurred.

The fall of infections, following clean air precautions in the operating room, includes the so-called "sterile" infections and also the infections delayed 6 and 12 months before being recognized. If the "sterile infection" and the delayed infections were caused by acrylic cement and not by bacteria acquired in the operating room this fall in infection rate could not be explained.

In a number of cases of deep infection we have patch-tested patients for allergies to acrylic cement without encountering a positive reaction so far.

I am thus convinced, after long observation directed to this point, that radiological complications suggesting osteitis after the use of acrylic cement are caused by two things and two things only: bacterial infection and, or, mechanical loosening of the implant. To diagnose simple mechanical loosening of a cemented implant is always difficult, and to make the diagnosis with confidence demands that the radiological abnormality shall be confined to one of the two parts of the implant; that the sedimentation rate should be low; and that the conditions should have been in existence for perhaps 2 years without sepsis having been proved. Even so final proof that it was not infection is established only when surgical replacement with another dose of cement results in a long-lasting cure.

While I deny that acrylic cement is the cause of infection being attracted to an implant, one must accept the fact that bone reacts differently when infected in the presence of cement. This difference can perhaps be explained by different physical states of the interfaces between implants and living tissues.

In the case of a metal implant without cement, the abrupt change between the smooth surface of hard steel and the soft cancellous bone invites movement at the interface under loads which produce an element of shear at the interface. As a result of slight movement, a layer of fibrous tissue develops between the steel and the bone which matures into a dense load-bearing layer. If bacteria have gained access at the operation organisms can lurk with the metallic implant inside the tough fibrous sheath, and it is probably this sheath which prevents spread of bacteria into the surrounding cancellous bone. This situation is not infrequently proved in ordinary clinical practice by cultures of implants removed at secondary operations. Radiological changes may arouse no more comment other than that mechanical loosening of the implant in the medullary cavity of the femur is clearly evident. When reviewed with hindsight, infection having been proved by exploration, these cases usually show more extensive radiological loosening than when no infection is present, but the important feature is that the cortical bone never shows radiological changes suggesting osteitis of the kind, which almost invariably is seen when infection is present with cement.

In the case of a cemented prosthesis the condition of the interface between implant and cancellous bone is very special: the injection of cement into many hundreds, even thousands, of intratrabecular spaces produces fixation against shear, no matter in what direction the load acts on the interface. In the absence of relative motion the tendency to generate a fibrous intersection is suppressed, and a layer of tissue exists which often is so thin as to be a mere membrane or septum, and which presumably will offer little or no resistance to penetration by organisms should they have gained access at the operation. An example of such an interface between cement and bone, 6 weeks after a cemented implant, is seen in *Figure 1* at 7 years, and shows how astonishingly thin the membrane separating a fat-filled cancellous space from the cement surface can be.

It seems possible therefore to suggest that cement changes the reaction of bone to infection, making it radiologically manifest, but not necessarily increasing the incidence of infection. Also, when a cemented prosthesis is infected it appears to cause more pain than when uncemented, and may produce a late spontaneous sinus, which almost never occurs in the absence of cement. Both of these features relate to the manifestation of infection more than to its incidence. There are many patients with defective results walking about with Moore's prostheses without cement which are probably infected but, because this is not manifest, they are considered simply to be mechanically loose.

However, it is a very ill wind that blows nobody any good: perhaps it is fortunate for surgical science that acrylic cement does manifest infections so clearly, because it is futile to attempt the study of infection in the operating

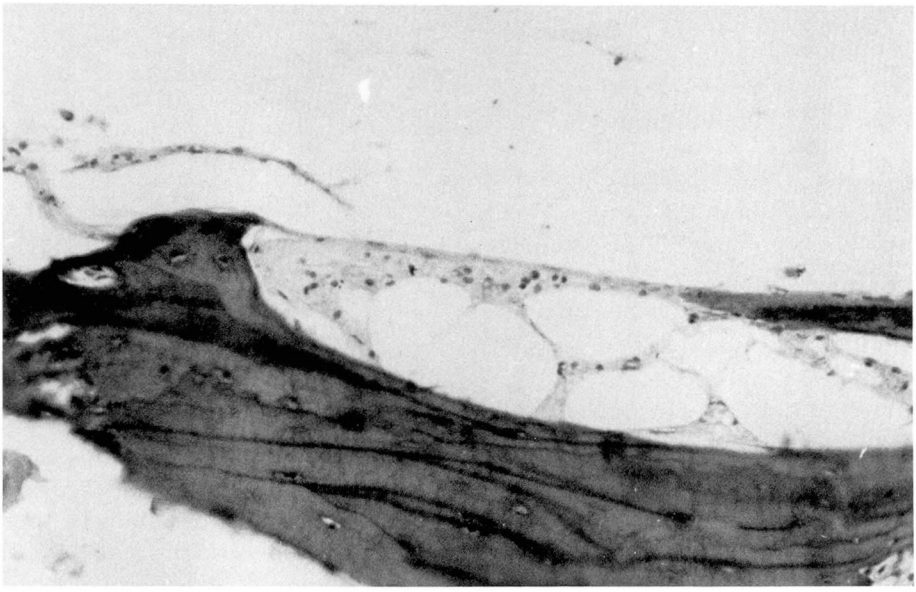

Fig. 1. Histology of endosteal surface of femur in contact with cement for 7 years. Note extremely thin membrane separating cement surface from fat in intratrabecular spaces.

room using as a test-object an operation which possesses high resistance to infection, or one in which infection cannot be detected. When cement is used in total hip replacement infection probably never can be overlooked if sufficient time (up to 3 years) is given for late manifestation.

Statistical evaluation of results

One of the great problems in studying postoperative wound infection relates to the significance of statistics. When the incidence of a complication is below 5% it is of paramount importance that the sizes of the samples should be carefully watched when preventive measures are being studied. To establish a fall from 3% to 1.5% at the 5% level of significance we need 780 observations.[3] When assessing infection rates below 1%, we are approaching a level where it might be impossible ever to mount a properly controlled trial. To establish a fall from 1% to 0.5% requires 2,600 observations and 2,600 controls. It is clearly impossible to perform 5,200 operations in a period of time short enough to avoid incurring new variables as surgical techniques advance every year. It is ironical that evidence against the argument can be accepted on quite small samples; thus in my own figures we accept infection rates at the level of 7% and 3% on samples as small as 100 and 200.

These considerations highlight the futility, of which we are all occasionally guilty, of comparing infection rates in tenths of 1%. At low levels of infection it is impossible to specify infection rates in units smaller than to the nearest 0.5% so that we must speak in terms of 1.5%, 1%, and 0.5%, rather than being tempted to use such figures as 0.9% or 0.4%.

Personal experience with infection in total hip replacement

Infection following total hip replacement has been a primary study since 1960 in my surgical unit where the numbers of these operations have been rising annually to a present level of about 1,200 per year or about 28 per week. These operations are, and have always been, performed according to a disciplined routine without individual variations by different members of the team. It is often suggested that special technical skill may be responsible for low infection rates, but the majority of the work in this unit (perhaps 60%) is performed by young men in training who have had very little previous practical experience of major surgery. While it is quite true that expert surgeons have lower infection rates than the inexpert when wounds are inoculated from the air by bacteria, our results show that if wounds are not inoculated by bacteria from the air, the work of relatively inexpert operators only rarely develops infection.

As was shown in a previous paper,[4] the infection rate in my service has fallen from the region of 7% in 1960 to about 0.5% in 1970, entirely as the result of precautions taken against infection in the operating room. I emphasize "precautions in the operating room," because no special precautions have ever been taken in this clinic against "cross infection" in the wards where new patients are often admitted to a bed near a patient already operated on who might have a superficial wound infection. The philosophy has always been that, for the duration of

the operation, it is possible to be 100% effective against all organisms brought to the operating room from the ward by chemical sterilization of the patient's skin, by efficient wound draping, and by a voluminous flow of clean air sweeping the operation site. Following operation the patient is returned to the ward with a sealed dressing, and this is not disturbed for 3 days, after which period it is supposed that any infection which might be acquired from the environment will be confined to the outer layers of the wound.

In studying this fall of infection since 1960, it is accepted that other factors in addition to clean air might have contributed, and an analysis of this has been published.[4] I shall select here only a few aspects for comment. The crux of my evidence turns on the fall in infection rate after June 1966. Before this date 1,080 total hip replacements had been performed in a prototype filtered air enclosure with turbulent flow at 130 air changes per hour resulting in an infection rate of 3%. After this date 909 operations were performed with less turbulent flow at 300 air changes per hour, and with an infection rate of 1.5%. The infection rate therefore fell by 50% over a number of operations which is statistically significant for a fall from 3% to 1.5%.

Unfortunately there are difficulties in specifying precisely the degree of air contamination in the enclosure ventilated at 130 air changes because as a result of turbulence the air was three or four times more contaminated on the periphery of the enclosure than in the vicinity of the wound. In order not to favor the theory of airborne infection, two separate estimates can be made, one with the air as clean as in the center of the enclosure (0.1 colonies per cubic foot) and one with air four times more dirty (0.4 colonies per cubic foot). If we accept the dirty figure as representing conditions round the open wound, we are forced to the conclusion that a fall in infection of 50% followed from improving air cleanliness from a level already 12.5 times cleaner than most good conventional operating rooms (if we accept five colonies per cubic foot for a good operating room).

Limitations of the role of airborne infection

If we accept that clean air caused a reduction in my infection rate from about 7% to 1.5%, this suggests that 10 years ago the primitive operating room in use at that time was responsible for 5.5% of the infections. This rate of infection was caused by air contamination averaging about 18 colonies per cubic foot.

Nevertheless it is an essential part of my observations and opinions regarding the role of clean air in the operating room that *clean air by itself* was unable to reduce the infection rate below 1.5%. The level of air cleanliness relating to this statement, from June 1966 until the introduction of the body exhaust gowns at the end of 1970, was in the range of 0 to 0.05 colonies per cubic foot of air (i.e., 0 to 1 colony in 20 cubic feet).

As far as contamination from the nose and throat or from the hair of the head is concerned, from the very earliest days of the enclosure (1961) this was controlled by a cloth hood completely investing the head and neck, combined with an aspirator applied to the mouth and nose to evacu-

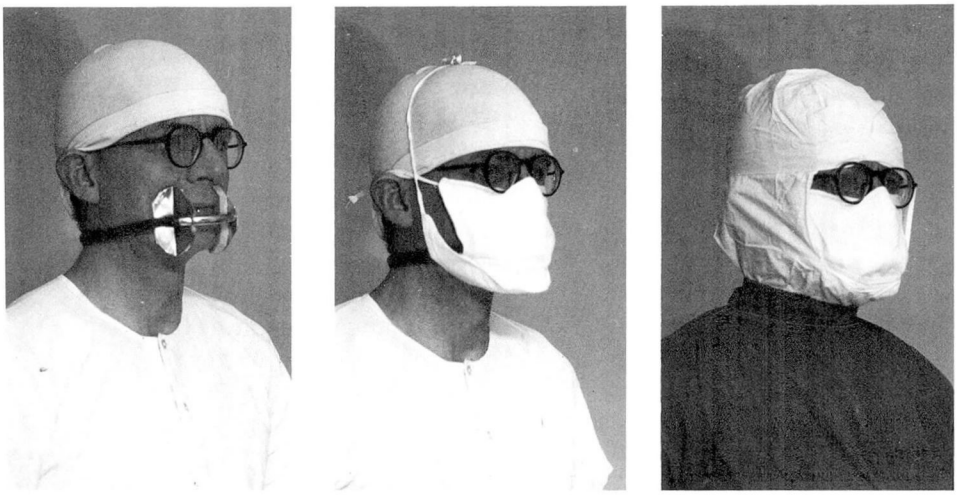

Fig. 2. Outmoded aspirator for nose and mouth. Note constriction at neck preventing body exhaust.

ate expired air (*Fig. 2*) but not evacuating the body.

This failure to get below a 1.5% infection rate led to a search for a route of infection which had been neglected, namely the permeability of the textile from which surgical gowns are made. The study of bacteria on the surface of a surgeon's gown at the end of a physically strenuous operation became valid as a result of operating in sterile air, and had this technique been available previously this route would undoubtedly have been tracked down many years ago.[5] The cotton textile known as "balloon cloth" was used for surgical gowns at this time and its porosity is seen in *Figure 3* where spaces up to 50μ are present.

Following recognition of the permeability of gowns we improvised measures by using double thickness aprons while the body exhaust or negative pressure gown was being developed. In the years 1969 and 1970, relating to about 2,100 operations, the infection rate fell to its present level of about 0.5% estimated at not less than 1 year after the operation. This fall is in the presence of the same air cleanliness as regards the volume and filtration of air entering the enclosure as introduced in June 1966, so that the improvement must be attributed to simple measures to render the front of the surgeon's clothing impermeable combined possibly with an improved method of wound closure. The improved method of wound closure related to the use of plastic foam compression pads, used with deep tension sutures, to prevent hematoma formation in the fat (*Fig. 4*).

My inability to get below a 1.5% infection rate by clean air alone makes it difficult to explain claims for infection rates below 1% for this same operation performed in conventional operating rooms. I can only suggest that a more extended follow-up may bring to light a greater number of late infections than originally suspected. I cannot believe that success in a conventional operating room is to be attributed to antibiotic prophylaxis, in view of evidence that to disturb

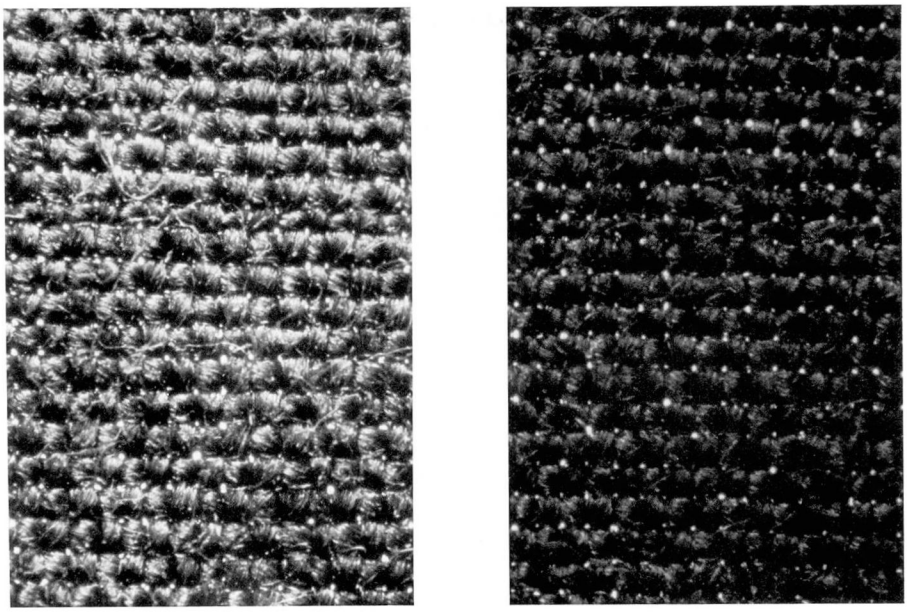

Fig. 3. Fine texture "balloon cloth" often used for surgical operating gowns. Left side of picture by incident light; right side with addition of transmitted light to show 50μ apertures in the weave.

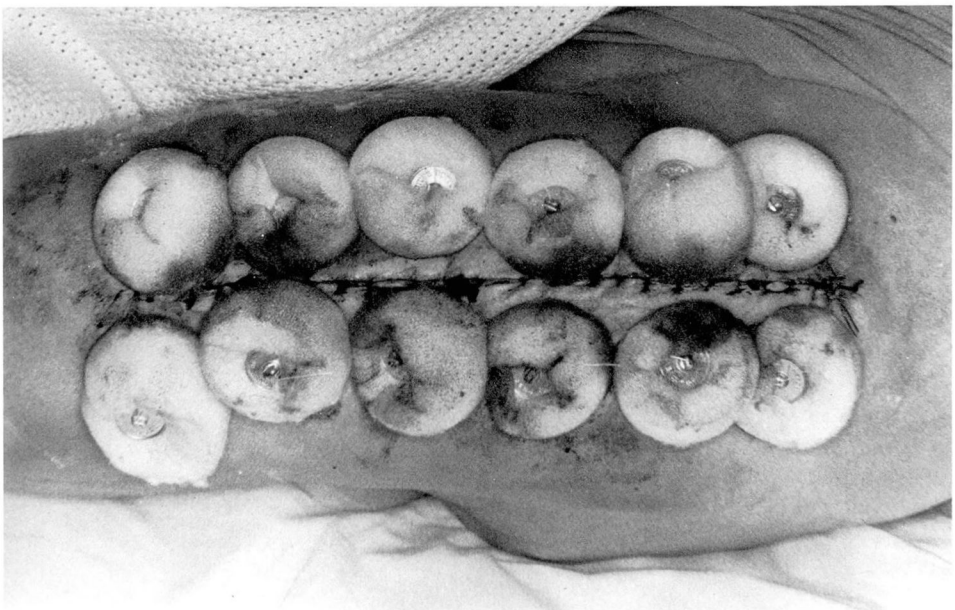

Fig. 4. Foam pressure pads to control superficial wound hematomata.

bacterial ecology can sometimes precipitate infection with organisms which otherwise might not have proliferated, and most particularly the favoring of gram-negative organisms resulting from the suppression of gram-positives. The complexity of antagonisms between different bacteria inhabiting the skin is a field only just becoming realized.[6]

Blood-borne infection

With our present infection rate at about 0.5% the question of blood-borne infection obviously raises itself as an explanation of this hard-core not yielding to techniques directed against exogenous infection. Of one thing I am certain, and that is that septicemic infection is a definite condition and is quite a different matter from bacteremic infection *if such a thing occurs at all.* In my total series of infections four cases have been excluded because they were quite clearly septicemic, appearing after a period of normal convalescence with high temperature and toxemia. Whether a symptomless bacteremia can be responsible for some of the residual 0.5% of infections currently being experienced is a question which must remain open.

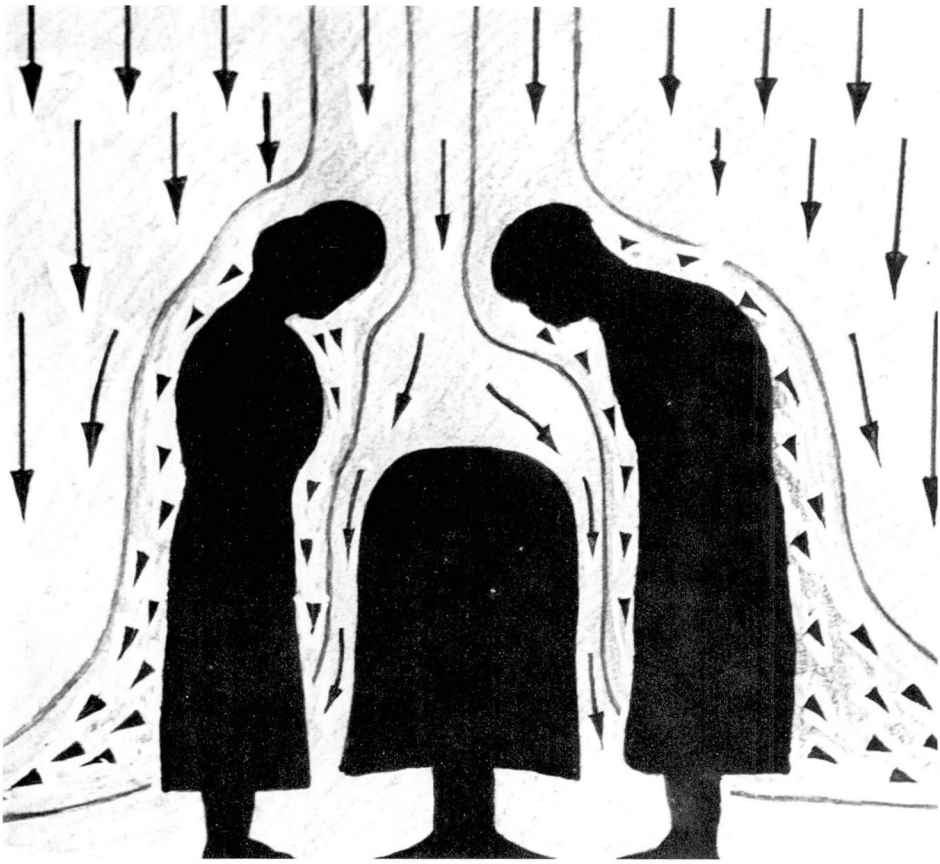

Fig. 5. Theoretical process whereby laminar flow sweeps away infected dust particles emitted by operating team and insulates open wound in a clean air current.

Laminar flow systems

It would appear to be inherent in the concept of perfect laminar flow that the lateral pressure of moving columns of air prevents turbulence at the sides of an object and causes the air to cling to the object and follow its contours. It is hoped that a situation is created where the laminar flow creates a barrier of clean air between the wound and the surgeon (*Fig. 5*).

This idealized system is seriously prejudiced when the surgical team comes in close contact with the operating table and commences to agitate arms over the wound (*Fig. 6*). Movements of the hands can attain speeds of 600 feet per minute compared with the 50 to 100 feet per minute of the airflow in laminar systems. This situation is unattractive when one considers the cost of laminar flow designed for perfection and the fact that it is impossible to use an overhead light of the type which has been developed specially for surgery over many years.

The body exhaust system

In this system it is possible to evacuate all infected dust particles at source; which source is the bodies of the surgical team. It is thus possible to pre-

Fig. 6. Practical effect of manual movements near open wound with obstruction to air flow by proximity of bodies of surgical team to the operating table.

Fig. 7. Semiturbulent air flow combined with body exhaust system. Turbulence not sufficient to permit currents rising from the floor. Perfect overhead surgical lighting possible.

serve asepsis in air which is mildly turbulent (*Fig. 7*). A conventional overhead lamp can be used and vigorous movements of the surgeon's arms do not threaten the sterility. It is easily possible to produce a bulk movement of slightly turbulent air without air from below knee level rising to table height, as we have proved repeatedly with smoke tests.

The body exhaust system,* or negative pressure gown, is made of a textile which is impermeable to bacteria and water, and almost impermeable to air. As well as extracting the infected dust particles the exhaust system operates as a cooling system without which it would be intolerable to wear. The conventional operating gown is tolerable because body heat, equivalent to that given off by a 150 watt electric light, is lost in convection curves rising from the gown and passing out through the open textile weave. Also cooling follows from the "bellows action" of a gown as the surgeon draws air in and during certain movements expels it in others in significant gusts. The cur-

* *The body exhaust system is patented and available from Codman and Shurtleff, Boston, Mass.; and James Howarth Ltd., Farnworth, England.*

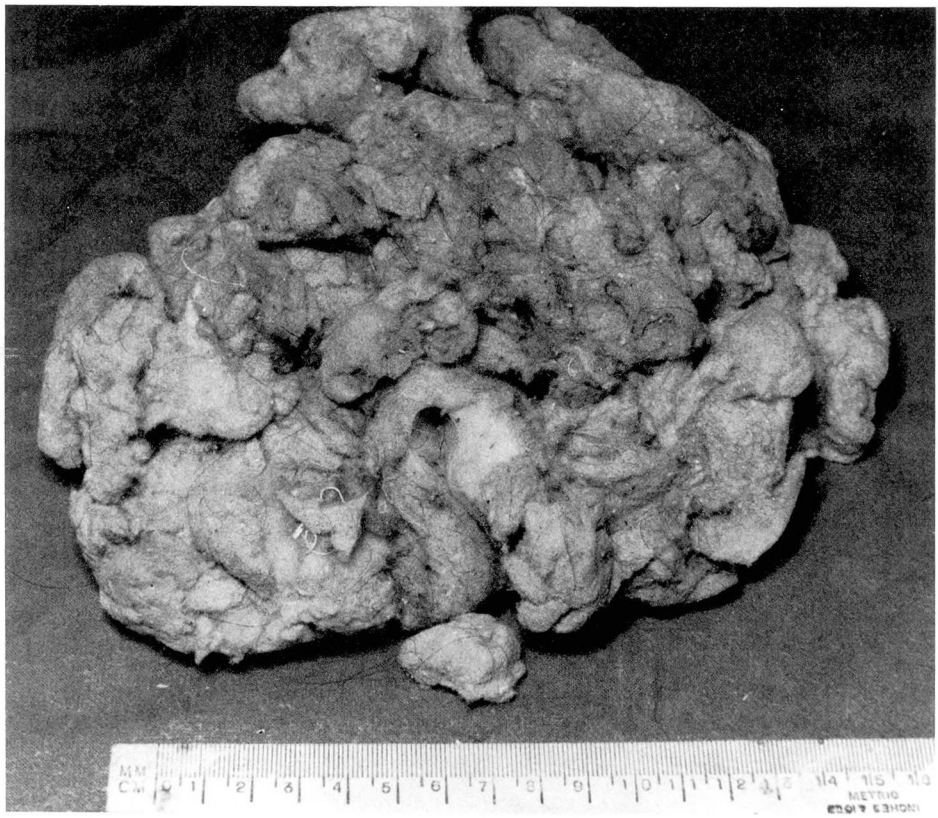

Fig. 8. Debris weighing 50 g extracted from body exhaust fan from four persons in the surgical team over about 800 total hip replacements. This constitutes only a fraction of total amount emitted.

rents of hot air escaping from a surgeon's gown can be shown by cinematography using the Schlieren technique, which depends on differences in the optical refractive indices of cold and warm air.

A dramatic illustration of the body exhaust system is the amount of debris accumulating in the exhaust fan from the bodies of the four persons in the surgical team. Debris weighing nearly 50 g consisting of lint from the gowns and underwear and epithelial scales (*Fig. 8*) represents only a fraction of what was emitted by four persons in the course of 800 total hip operations. It would seem that each person during the course of one operation may lose from 10 to 20 mg of debris including a number of hairs from the head. Presumably this will go into the general environment under conventional operating conditions. Close inspection of this debris shows it to be loaded with human hair (*Fig. 9*).

Slit sampler tests show a remarkable degree of sterility using the body exhaust system, even when the level of sampling is below knee level of the surgical team, and even in an enclosure which does not pretend to produce strict laminar flow. The slit sampler plates (*Fig. 10*) compare conditions in our "clean air" enclosure,

using the body exhaust system with semiturbulent air, with five colonies per cubic foot in our anaesthetic room, a degree of contamination often regarded as acceptable for a modern operating room.

The design of the body exhaust clothing as I have evolved it has taken into consideration simplicity, the possibility of low cost, and comfort for the wearer, in addition to the obvious requirement of efficiency in achieving its purpose.

The gown, of a washable cotton

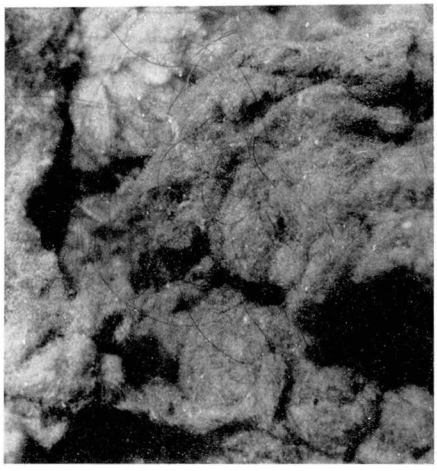

Fig. 9. Close view of debris showing admixture of human hair in the cotton debris worn off the inside of the gowns and wearers' underwear.

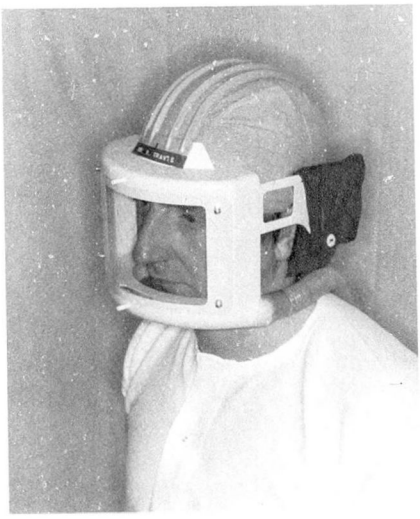

Fig. 11. Headpiece with transparent window and suction hoses.

Fig. 10. Slit sampler plates. Each 7 inches (17.5 cm) in diameter and rotating in 1 hour at 3 cfm. Taken 12 inches (30.48 cm) from ground in semiturbulent enclosure with body exhaust = 0.01 colonies/cubic foot. Plate from anaesthetic room represents five colonies/cubic foot.

material almost impermeable to air, incorporates a hood in such a manner that the gown hangs from the summit of the wearer's head and, having no constrictions at the neck or waist, encourages warm air from the body to rise into the triangular spaces existing between the shoulders and the sides of the head. A lightweight plastic headpiece, carrying a transparent window and the internal openings of the suction hose, forms the base on which the gown rests and registers with the window aperture (*Figs. 11 and 12*).

Air is evacuated from a central suction line at a rate varying according to the comfort of the surgeon from 15 cfm to 35 cfm. At the higher rates of suction there is considerable wind noise, though it is surprising how soon the wearer can ignore this and at its worst it is less than the noise experienced in a private airplane. Some wearers find it necessary to use an electronic intercommunication system but for well-rehearsed routine surgery little or no conversation is needed, and we at Wrightington never feel the need for this additional complication.

A feature of this form of clothing is its effect on the physicial well-being of the surgeon. In place of the sensa-

Fig. 12. Gown in position. Note absence of constriction at neck and waist.

Fig. 13. Experimental bacterial filter in suction line.

tion of complete enervation, like the effect of a Turkish bath, customary after several hours operating with the rebreathing of humid expired air in a conventional face-mask, this system is almost exhilarating, akin to activity in the open air. Not only is sweating of the hands inside rubber gloves reduced, but mental concentration is improved, and the surgeon is in a better position towards the end of a long operating session to cope with unexpected surgical complications should they arise.

In *Figure 13* is shown an experiment where a specially constructed air filter, with initially a sterile interior, was inserted in the suction line during the course of an actual surgical operation. Between 2,000 and 6,000 colony forming particles were recovered per hour.

Conclusion

1. Criticisms of the concept of airborne infection in operating rooms and the dosage of bacteria needed to cause a wound infection are discussed.

2. Factors are reviewed exonerating acrylic cement as a cause of infection.

3. Personal statistics are offered in reducing an infection rate from 7% to 0.5% using impermeable clothing.

4. It is believed that by itself clean air cannot be guaranteed to lower the infection rate below 1.5% in total hip replacement using cement.

5. No explanation can be offered for reports of infection rates below 1%, for this operation performed in conventional operating rooms and using cement.

6. Details of the body exhaust system are described.

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