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Editorial

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Mini review section – Mucormycosis, also known as black fungus, it is a serious fungal infection, usually in people who are immunocompromised. It is a systemic fungal infection caused by members of the class Zygomycetes, order Mucorales. They have been isolated in the laboratories as contaminants for a long time. However, changing host environments are causing their emergence as potential pathogenic organisms leading to high morbidity and very high and quick mortality.

Current Trends section – Wound microbiology may be considered a complex and sometimes misunderstood area in clinical medicine, not least because a wound provides an environment in which the microbial ecosystem is very dynamic and unstable. The majority of dermal wounds are colonized with aerobic and anaerobic microorganisms, often referred to as the "indigenous" or normal micro biota that originate predominantly from mucosal surfaces such as those of the oral cavity and gut.

In Profile Scientist – Charles Louis Alphonse Laveran (18 June 1845 – 18 May 1922) was a French physician who won the Nobel Prize in Physiology or Medicine in 1907 for his discoveries of parasitic protozoans as causative agents of infectious diseases such as malaria and trypanosomiasis.

Bug of the month – *Cryptosporidium* is a microscopic parasite that causes the diarrheal disease cryptosporidiosis. Both the parasite and the disease are commonly known as "Crypto." There are many species of *Cryptosporidium* that infect animals, some of which also infect humans. The parasite is protected by an outer shell that allows it to survive outside the body for long periods of time and makes it very tolerant to chlorine disinfection. While this parasite can be spread in several different ways, water (drinking water and recreational water) is the most common way to spread the parasite.

Did You Know? – Since the Covid-19 pandemic began last year, face masks and other personal protective equipment have become essential for health care workers. Disposable N95 masks have been in especially high demand to help prevent the spread of SARS-CoV-2, the virus that causes Covid-19. "Perhaps unsurprisingly, the approaches that incorporate reusable aspects stand to have not only the greatest cost savings, but also significant reduction in waste," The study also found that fully reusable silicone N95 masks could offer an even greater reduction in waste. They are now working on developing such masks, which are not yet commercially available.

Best Practices – Confronted with worldwide evidence of substantial public health harm due to inadequate patient safety, the World Health Assembly (WHA) in 2002 adopted a resolution urging countries to strengthen the safety of health care and monitoring systems. The resolution also requested that WHO take a lead in setting global norms and standards and supporting country efforts in preparing patient safety policies and practices.

So go on, enjoy reading & don't forget to give us your valuable inputs & feedback.

BioShields 💿

HYGIENE SCIENCES

An Overview of Mucormycosis

Mucormycosis, also known as black fungus, it is a serious fungal infection, usually in people who are immunocompromised. It is a systemic fungal infection caused by members of the class Zygomycetes, order Mucorales. It is seen in patients debilitated by immune or metabolic disorders. Mucorales are ubiquitous fungi and are commonly found in decaying organic matter. They have been isolated in the laboratories as contaminants for a long time. However, changing host environments are causing their emergence as potential pathogenic organisms leading to high morbidity and very high and quick mortality.

Introduction:

Mucormycosis, a serious angioinvasive infection caused by common filamentous fungi, that is, mucormycetes, constitutes the third most common invasive fungal infection following aspergillosis and candidiasis. The disease can be transmitted by inhalation of spores or by direct inoculation of the spores into disrupted skin or mucosa. The etiologic agents can cause infections with high mortality in immunocompromised, mainly diabetic patients.

The class Zygomycetes consists of aseptate hyaline molds that reproduce by both sexual and asexual means. These fungi are ubiquitous in soil and decaying vegetation. Five genera in the order Mucorales are responsible for disease in humans: *Rhizopus, Mucor, Absidia*, and rarely, *Saksenaea* and *Cunninghamella*.

Patients contracting this infection uniformly suffer from predisposing conditions: acidosis, uncontrolled diabetes mellitus, leukemia, lymphoma, AIDS, severe malnourishment, severe burns, cytotoxic therapy, and immune suppression from corticosteroid use. It has also been observed in patients with chronic renal failure, liver problems, and dialysis patients on deferoxamine therapy.

Mucormycosis is a disease difficult to diagnose in clinical and laboratory settings. A high index of suspicion on the clinician side with the need to send an appropriate sample to the laboratory is a prerequisite. It is not easy to isolate and maintain in the laboratory since their poorly septate hyphae can lose the vital cytoplasm at the least manipulation. Moreover, the awareness among microbiologists is still low, with most isolates classified as "*Mucor* or *Rhizopus*" without further identification. Newer species are recognized (*Apophysomyces, Mucor irregularis*), new areas of isolation have been identified (isolated renal mucormycosis), and mucormycosis has broadened its host range (involving both immunocompetent and immunocompromised).

Mucorales are ubiquitous fungi and are commonly found in decaying organic matter. They have been isolated in the laboratories as contaminants for a long time. However, changing host environments are causing their emergence as potential pathogenic organisms leading to high morbidity and very high and quick mortality. The disease caused by them has been reported all over the world and although associated with immunocompromization states like carcinomas or immunosuppressive therapy, it also affects a newer range of susceptible hosts like diabetics, neutropenics, patients on desferroxamine therapy, etc.

Risk factors are different (being more common in diabetics, neutropenics than AIDS patients), and it is difficult to treat (not responding to the most common antifungal drugs used, i.e., azoles with posaconazole and isavuconazole being the exception). The disease warrants the efficient training of clinicians and surgeons in dealing with this fungus which is angioinvasive and often the hyphae have invaded deep into the healthy tissue before debridement is done. A wide area of healthy tissue needs to be debrided to get rid of the fungus from the surroundings of the wound.

Presently, Phylum Glomeromycota is divided into four subphyla: Mucoromycotina, Entomophthoromycotina, Kickxellales and Zoopagomycotina (elevating the orders Mucorales and Entomophthorales to a subphylum status). The mucormycetes (previously Zygomycetes) belong to the order Mucorales and involve 6 main families Syncephalastraceae (Genus Syncephalastrum), Saksenaeaceae (genera Saksenaea and Apophysomyces) Cunninghamellaceae (genus Cunninghamella), Mucoraceae (genera Mucor, Rhizopus, Rhizomucor and Actinomucor), Thamnidiaceae (Cokeromyces) and Lichtheimiaceae (genus Lichtheimia). About 70% to 80% of cases of mucormycosis are caused by fungi belonging to the genera Rhizopus, Mucor and Lichtheimia. The disease is commonly acquired by inhalation of sporangiospores. Other routes include ingestion of spores, direct implantation into injured skin, and trauma with contaminated soil, intravenous or intramuscular injections. The most common

manifestation of the disease is rhino-orbito-cerebral (ROC) mucormycosis, mostly seen in diabetics.

There are no known predispositions based on age, race, or sex. Most cases are acute surgical emergencies, though a few chronic, indolent forms have been reported with signs and symptoms developing over a 4-week period. The primary sites of invasion are the paranasal sinuses, lungs, skin, and the GI tract.

Clinical symptoms, signs, and pathological findings are similar in mucormycosis, regardless of etiology. These fungi show a predilection for arterial invasion, causing extensive emboli and necrosis of surrounding tissues. Vein and lymphatic invasion can occur later in the course of the infection. The acidotic, hyperglycemic environment existing in patients with ketoacidotic diabetes mellitus particularly favors the growth of *Rhizopus*. It is thought that diabetic and immunocompromised patients lack normal phagocytic activity on their nasal and oral mucosal surfaces. This allows proliferation of fungus, which does not occur in people with intact phagocytic activity, and the fungus spreads via the blood vessels.

There are 5 forms of the disease:

- Rhinocerebral. This is the most common form, usually seen in patients with ketoacidotic diabetes mellitus. This form presents with sinusitis, facial and eye pain, proptosis, progressing to signs of orbital structure involvement. Necrotic tissue can be seen on the nasal turbinates, septum, and palate. This may look like a black eschar. Intracranial involvement develops as the fungus progresses through either the ophthalmic artery, the superior fissure, or the cribiform plate.
- **Pulmonary**. This is most frequently seen in patients with neutropenia, such as those with leukemia or lymphoma. This form presents with fever, dypsnea, and possible hemoptysis.
- **GI tract.** This form is seen in severely malnourished patients, particularly in kwashiorkor, and has been seen in patients with amoebic colitis and typhoid. The stomach, ileum, and colon are usually involved, mimicking intra-abdominal abcess.

- **Cutaneous.** This form can follow minor trauma, insect bites, wounds, burns, and use of non-sterile dressings. Necrotic lesions occur on the epidermis that are painful and hardened, usually with a blackened central area These lesions can progress into the dermis and even muscle.
- **Disseminated.** Dissemination can occur, mainly from the pulmonary form, to the heart, brain, bones, kidney, and bladder. Dialysis patients on deferoxamine therapy are predisposed to this form.

Diagnosis

Rapid diagnosis and initiation of therapy is critical due to the acute, fulminate nature of the infection. Diagnosis of mucormycosis rests upon the presence of predisposing conditions, signs and symptoms of disease, observation of fungal elements of specific morphology in histological sections, and direct smears of material, and, to a lesser extent, culture results. There are no reliable serological methods for diagnosis at present.

Direct examination in 10% KOH of scrapings from the upper turbinates, aspirated sinus material, sputum, and biopsy material can be valuable. The presence of thick-walled, aseptate, and refractile hyphae 6 to 15 μ m in diameter, with some hyphae being swollen and distorted, is indicative of the presence of Mucorales fungi.

Histological sections show acute suppurative inflammation with focal areas of granulomatous inflammation. There are aseptate hyphae 6 to 50 μ m in diameter, branching at 90°. The hyphae invade the adjacent blood vessel walls, producing thrombosis and infarction, but rarely disseminate through the vessels. Staining with Grocott-Gomori methenamine silver is best, though periodic acid-Schiff and hematoxylin & eosin (H&E) stains (Cat No. 207080190250) can be used. Diagnosis is frequently made from tissue sections.

Differentiation from *Aspergillus* and *Candida* must be made on histological section. *Aspergillus* and *Candida* do not take H&E stain. *Aspergillus* has septate, narrow, acutely branching hyphae with smooth, parallel walls. *Candida* has septate, narrow hyphae in tissue, with club-shaped pseudohyphae and yeast forms present.

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Mini Review

A culture result, by itself, is not diagnostic of infection, since Mucorales fungi are common in the environment. Culture is ideally done on biopsy specimens. Exudates and necrotic tissue contain few viable organisms; thus the inoculum from these specimens must be heavy. Zygomycetes do not survive for more than a few hours at refrigerator temperatures, so if culture is delayed, storage in Stuart's bacteriological transport media (Cat No. 201190360500) at room temperature is recommended. Sabouraud's dextrose agar (Cat No. 201190090500) or brain-heart infusion agar (Cat No. 201020230500) are most commonly used for isolation. Media containing cycloheximide must be avoided, as it inhibits the Zygomycetes. Antibacterial agents, such as chloramphenicol and polymyxin B, can be used to prevent bacterial overgrowth. Cultures should be set at 25°C and 37°C aerobically and incubated for 2 to 5 days. Other media good for inducing sporulation necessary for speciation are potato dextrose malt agar, Czapek solution agar, and hay infusion agar.

Colonies produce fluffy white, gray, or brown hyphae filling the culture container within 24 to 96 hours. The hyphae are coarse and dotted with brown or black sporangia. It is impossible to distinguish the genera based on colony morphology, as they appear similar.

Fig 1: Five-day-old, grayish and very wooly colony of Mucor sp. on Sabouraud's dextrose agar, 25°C; the reverse of the colony appears pale yellow (Upper panel).



Identification of genera is based on the presence of aseptate hyphae, the structure of the sporangiophore, and the presence and position of rhizoids relative to the sporangiophores. Lactophenol cotton blue (Cat. No. 207120390100) can be used to better visualize microscopic structures. Identification to the genus level is not very difficult and can be of great value to the physician in a critical situation. Speciation is difficult and is best left to a reference laboratory experienced in fungal identification. See **Fig 2** for structures.



Fig 2: Fungal Structures of the Order Mucorales.

- *Rhizopus* sp. are the most often recovered organisms from specimens. They exhibit unbranched sporangiophores that occur singly or in groups at nodes, directly above the rhizoids. The nodes are connected by stolons. The sporangia are dark walled and spherical. Species predominantly recovered are *R. oryzae* and *R. arrhizus*.
- Mucor sp. are the next most recovered organism from specimens. This genus demonstrates aerial unbranched and branched sporangiophores arising randomly from mycelia. No rhizoids are present. The sporangia are large and spherical. The main species recovered are M. circinelloides, M. ramosissimus, and M. javanicus.
- *Absidia* sp. demonstrates branching sporangiophores arising from nodes between rhizoids. The sporangia are pyreform. Species predominantly recovered are *A. ramose* and *A. corymbifera*.
- *Cuminghamella bertholletiae* and *Saksenaea vasiformis* have been isolated on rare occasions from clinical cases. They have unique microscopic morphologies. Species of *Synchephalastrum*, isolated as contaminants, are of interest since they can be mistaken for *Aspergillus* on casual inspection.

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Typical microscopic morphologies of these genera are shown in **Fig 3**







Fig 4: Scanning electron micrograph of *Mucor* sp. highlighting characteristic sporangium and hypha (bar = $10 \,\mu$ m).

Treatment

Once diagnosis has been established, correction of hypoxia, acidosis, hyperglycemia, and electrolytic imbalance needs to be undertaken. Steroids, antimetabolites, and immunosuppressive drugs should be discontinued, if possible. Aggressive surgical debridement is usually undertaken, along with high dose intravenous amphotericin B therapy (5mg/kg IV daily). Treatment is continued until remission is achieved. Liposomal amphotericin B may be more effective and less toxic. Resistance to amphotericin B has been observed with prolonged therapy. Local irrigation and packing to aid delivery of amphotericin B to necrotic and poorly perfused tissues has been tried as an adjunct to therapy. This could help prevent disfiguring surgery.

Prognosis

The survival rate in patients with uncontrolled diabetes mellitus suffering from the rhinocerebral form is very grave. Patients with leukemia or lymphoma suffering from the pulmonary form usually die from the infection. The GI tract infection is usually diagnosed on autopsy. The overall mortality is high, usually 30% to 70%. Death usually results in 2 weeks if untreated or unsuccessfully treated. The survival rate lowers as the diagnosis to treatment interval increases. Seventy percent of survivors have permanent residual effects, including blindness, cranial nerve defects, and surgical disfigurement.

Wound Infection and Modern Biocides

Introduction

Wound microbiology may be considered a complex and sometimes misunderstood area in clinical medicine, not least because a wound provides an environment in which the microbial ecosystem is very dynamic and unstable. The human body contains an estimated 1014 microbial cells and these outnumber mammalian cells 10-fold. These micro biota are necessary for health but have the potential for causing disease given the opportunity. Infections occur when microorganisms overcome the host natural immune system and subsequent invasion and dissemination of microorganisms in viable tissue provoke a series of local and systemic host responses.

Wound Microbiology

The majority of dermal wounds are colonized with aerobic and anaerobic microorganisms, often referred to as the "indigenous" or normal micro biota that originate predominantly from mucosal surfaces such as those of the oral cavity and gut. These micro biota play an important role in preventing colonization by pathogens of significant virulence (colonization resistance). The role and significance of microorganisms in wound healing have been debated for many years. Some consider the microbial density to be critical in predicting wound healing and infection, while others consider the types of microorganisms to be of greater importance. However, these and other factors such as microbial synergy, the host immune response and the quality of tissue must be considered collectively in assessing the probability of infection. Whatever the outcome of these processes, wound microbiota are considered to be polymicrobial. The polymicrobial ecosystem of the wound is composed of a vast array of microorganisms which can be classified according to their nutritional and environmental requirements. One fundamental factor significant to wounds is the availability of oxygen which dictates which types of microbes can proliferate (Table 1). With acute and chronic wound infections, mixed populations of both aerobic and anaerobic microorganisms are commonly found. When anaerobes are evident, this is indicative of a more complex microenvironment in the wound. The existence of anaerobic bacteria in wounds may be significant but their presence is often overlooked as many standard laboratories do not routinely screen for them. Examples of common bacteria that have been isolated from chronic wounds may be seen in Table 2. However, the mere presence of these bacteria does not constitute an infected wound.

Table 1

Туре	Requirements
Obligate aerobe	Must have access to oxygen
Obligate anaerobe	Will only grow in the absence of oxygen
Facultative aerobe	An anaerobic organism will grow in the presence of oxygen
Facultative anaerobe	An organism that can grow in the presence and absence of oxygen

The age of a wound influences microbial composition and diversity, and the development of the microbial ecosystem can be divided into 3 phases. Phase I is predominately described as an aerobic process and the organisms most representative are classified as Gram-positive obligate aerobic or facultative anaerobic. This is an acute process. Phase II is transitional, occurring as the levels of oxygen are reduced by obligate aerobes, e.g. in poorly perfused tissue. This environment will encourage growth of anaerobic microbes, specifically obligate anaerobes. If such an environment persists, phase III may develop, reflected by a change in the predominant microbiota to a mixed microbial community favouring organisms that persist over time with less standard pathogenicity; key pathogenic features include enzymes and toxin production.

Historically, most cultures isolated from chronic wounds are based on the traditional culture methodology, either aerobic or anaerobic and have relied upon traditional methods of sampling and laboratory detection. Advanced technology now utilizes molecular techniques that allow for the identification of viable but non-culturable (VBNC) bacteria, that otherwise would remain undetected by traditional methods. This is a significant advance in wound microbiology. The significance of these VBNC organisms requires clarification specifically related to the area of bacterial synergy, which is known to be important in bacterial pathogenicity and in biofilm formation.

Wound Infection

The list of microbes associated with skin and soft tissue infections is growing. This list (Table 2) while not exhaustive, illustrates the complexity of the microbiology involved in wound management. Bacteria, specifically staphylococci, almost never appear as a single isolate in infected wounds as they are most often found in synergistic relationships with other bacteria.

In many wounds, when using culture techniques, the number of aerobic isolates recovered range from 1-8 with an average of 2.7 organisms per wound. However, when molecular techniques are used, significantly more bacteria are found to be present. Infected chronic wounds are biochemically and microbiologically complex with many deep wounds frequently hypoxic as a consequence of poor blood perfusion. This creates an ideal growth environment for microbes, including fastidious anaerobes that will proliferate as residual oxygen is consumed by obligate, facultative aerobic and anaerobic bacteria.

Table 2

Aerobes	Anaerobes
Acinetobacter baumanii	Bacteroides spp
Coliforms	Fusobacterium spp
Enterococcus faecalis	Peptostreptococcus
MRSA	Porphyromonas spp
Pseudomonas aeruginosa	Prevotella spp
Staphylococcus aureus	Veilonella spp
Staphylococcus epidermidis	
Streptococcus pyogenes	

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Bacterial species rarely exist in pure culture in wounds and as such, within a wound, the microbiology exists within a community structure. The complexes that occur within wounds are not clearly understood. A better understanding and knowledge base regarding bacterial interactions will be important in managing polymicrobial infected wounds. An example of a polymicrobial infected wound is considered to be a biofilm community. Biofilms which are considered by some to be associated with delayed wound healing are by definition sessile, and this stationary mode of growth will reduce the hazards which bacteria are accustomed to within the free floating or planktonic state.

Sampling Infected Wounds

It is important to remember that the quality of the laboratory report is dependent on the quality of the specimen and that simple cultures provide limited information. Additionally, if unrepresentative samples are obtained, unrepresentative reports will be generated. If a swab is taken, the specimen must be accompanied with significant clinical information, including specific anatomic site, classification of wound and prior or ongoing antibiotic therapy, and transported in appropriate media and processed within the recommended time frame. Recovery of true wound bacteria when bordered by skin flora is difficult as these are often classed as contaminates. Consequently, assessing the true microbiology of a wound infection does not have the same clarity as a sample recovered from sterile fluid such as blood or cerebrospinal fluid. Ideally, wound microbiology should only be interpreted in combination with the clinical diagnosis.

Biofilm Overview

Biofilms are found widely in nature and have been rigorously studied for many years. However, the study of biofilms in relation to health and in particular wounds is a relatively recent development. The National Institutes of Health (NIH) suggest that 80% of human infectious disease is caused by biofilm, usually manifesting as chronic infection. These chronic infections often viewed as benign are in fact insidious and progressive in nature and produce death tolls each year rivaling that of heart disease or cancer, yet clinicians appear to have developed an extremely passive relationship with biofilm disease including those implicated in wound infection. Most clinicians are familiar with planktonic bacteria as they are routinely cultured in the laboratory, challenged by antibiotics with sensitivity or resistance recorded and a treatment recommendation made. The problem with this approach is that chronic wound bacteria are quite different from their laboratory planktonic counterparts.

A biofilm is a complex community comprising a mixed population of different microorganisms.

It is typified by the secretion of extracellular polymeric substance (EPS), a glue that protects the bacteria and holds the community together. The EPS matrix protects the individual bacteria from environmental stresses, scavenges nutrients from the environment and provides shelter for the unique heterogeneous micro-niches inside the biofilm. The biofilm microcolony achieves a critical density of bacteria (a quorum) through the release of signaling molecules and permits differentiation into a true biofilm society. This complex system of quorum-sensing molecules is tightly controlled and suggests that biofilm is most appropriately thought of as an organism composed of billions of

individual cells and specialized structures.

Reproduction is carried out by the biofilm breaking down portions of itself and releasing fragments which contain cells incased in matrix material. These detachment fragments have the ability to attach to a suitable surface, become metabolically active, and reform a biofilm community. The biofilm community also forms secondary structures, including mushroom-type projections off the surface, water channels and extensions. These structures allow nutrient inflow and waste outflow throughout the biofilm.

Bibliography

Allen MJ, Morby AP, White GF (2004) Cooperativity in the binding of the cationic biocide polyhexamethylene biguanide to nucleic acids. Biochem Biophys Res Commun **318**(2): 397–404

Ansorg RA, Azem T, Fabry WH, Rath PM (2002) Influence of mucin on the activity of the antiseptic Lavasept against Staphylococcus aureus. Chemotherapy **48**(3): 129–33

Bergin SM, Wraight P (2006) Silver based wound dressings and topical agents for treating diabetic foot ulcers. CochraneDatabase Syst Rev Issue 1 Art. No.: CD005082. DOI:10.1002/14651858.CD005082.pub2

Best Practice Statement (2011) The Use of Topical AntimicrobialAgents in Wound Management. Wounds UK, London. Availableat: http://www. wounds-uk.com/best-practicestatements/bestpractice-statement-the-use-of-topicalantisepticantimicrobialagents-in-wound-management- 2ndedition

Brennan SS, Leaper DJ (1985) The effects of antiseptics on the healing wound: a study using the rabbit ear chamber. Br J Surg 72(10): 780–2

Broxton P, Woodcock PM, Heatley F, Gilbert P (1984a) Interaction of some polyhexamethylene biguanides and membrane phospholipids in Escherichia coli. J Appl Bacteriol 57(1): 115–24

Burd A, Kwok CH, Hung SC, Chan HS, Gu H, Lam WK, Huang L (2007) A comparative study of the cytotoxicity of silver-based dressings in monolayer cell, tissue explant and animal models.Wound Repair Regen **15**(1): 94–104

Cazzaniga A, Serralta V, Davis S, Orr R, Eaglstein W, Mertz PM (2002) The effect of an antimicrobial gauze dressing impregnated with 0.2-percent polyhexamethylene biguanide as a barrier to prevent Pseudomonas aeruginosa wound invasion. Wounds 14(5): 169–76

Chaby G, Senet P, Vaneau M, Martel P, Gauillaume JC et al (2007) Dressings for acute and chronic wounds: a systematic review. Arch Dermatol **143**(10): 1297–304

Clarke S (2003) The silver solution. Journal of Community Nursing **1**7(1): 25–8

Cookson BD (2000) Methicillin-resistant Staphylococcus aureus in the community: new battlefronts, or are the battles lost? Infect Control Hosp Epidemiol **21**(6): 398–403

Cooper RA (2004) A review of the evidence for the use of topical antimicrobial agents in wound care. WorldWide Wounds. Available online at: http://tinyurl.com/76m6ctg

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Current Trends

Cooper RA (2007) Iodine revisited. Int Wound J 4(2): 124-37

Cooper RA, Halas E, Molan PC (2002b) The efficacy of honey in inhibiting strains of Pseudomonas aeruginosa from infected burns. J Burn Care Rehabil **23**(6): 366–70

Cooper RA, Molan PC, Harding KG (2002a) The sensitivity to honey of Gram-positive cocci of clinical significance isolated from wounds. J Appl Microbiol **93**(5): 857–63

Daeschlein G, Assadian O, Bruck JC, Meinl C, Kramer A et al (2007) Feasibility and clinical applicability of polihexanide for treatment of second-degree burn wounds. Skin Pharmacol Physiol **20**(6): 292–6

Davies A, Bentley M, Field BS (1968) Comparison of the action of vantocil, cetrimide and chlorhexidine on Escherichia coli and its spheroplasts and the protoplasts of Gram-positive bacteria. J Appl Bacteriol **31**(4): 448–61

Davies A, Field BS (1969) Action of biguanides, phenols and detergents on Escherichia coli and its sphereoplasts. J Appl Bacteriol 32(2): 233–43

Demling R, DeSanti L (2001) The role of silver in wound healing: part 1. Effects of silver on wound management. Wounds: A Compendium of Clinical Research and Practice **13**(1 Suppl A): 4–15

Denyer J (2009a) Epidermal bullosa and silver absorption in paediatrics. Presentation at Wounds UK Conference, 11 November 2009, Harrogate

Denyer J (2009b) Management of the infant with epidermolysis bullosa. Infant 5(6): 168–71

Dissemond J, Gerber V, Kramer A, Riepe G, Strohal R et al (2010) A practice-oriented expert recommendation for treatment of critically colonised and locally infected wounds using polihexanide. J Tissue Viability 19(3): 106–15

Dow G (2001) Infection in chronic wounds. In: Krasner DL, Rodeheaver, GT, Sibbald RG eds. Chronic wound care: a clinical source book for healthcare professionals (3rd edn). HMP Communications Wayne, PA: 385–356

Dowsett C, Edwards-Jones V, Davies S (2004) Infection control for wound bed preparation. Br J Community Nurs 9(9s): 12–17

Eberlein Th, Wild Th (2008) Approaches to therapy and an outlook on improving the clinical data situation, with a special focus on the polihexanide-containing Suprasorb X + PHMB Hydrobalance dressing. EWMA conference extended abstract. Available online at: http://tinyurl.com/cfs6rak

Edwards R, Harding KG (2004) Bacteria and wound healing. Curr Opin Infect Dis 17(2): 91–6

European Wound Management Association (2006) Position Document: Management of wound infection. MEP Ltd, London Galitz C, Hämmerle G, Signer M, Traber J, Eberlein T et al (2009) Polyhexanide versus silver wound dressings: first interim results of a controlled, randomized, prospective, multicentric study. Poster presentation. European Wound Management Association (EWMA) Conference, Helsinki. Available at: http://tinyurl.com/737n6go

Gilbert P (2006) Avoiding the resistance pitfall in infection control. Does the use of antiseptic products contribute to the spread of antibiotic resistance? Ostomy Wound Manage **52**(10A *Suppl*): 1S–3S

Harbs N, Siebert J (2007) In vitro efficacy of octenidine and polihexanide against biofilms composed of Pseudomonas aeruginosa. GMS Krankenhaushyg Interdiszpl **2**(2): Doc.45 (20071228)

Hansson C (1998) The effects of cadexomer iodine paste in the treatment of venous leg ulcers compared with hydrocolloid dressing and paraffin gauze dressing. Cadexomer Iodine Study Group. Int J Dermatol **37**(5): 390–6

Hunt TK, Hopf HW (1997) Wound healing and wound infection. What surgeons and anaesthesiologists can do. Surg Clin North Am 77(3): 587–606

Ikeda T, Ledwith A, Bamford CH, Hann RA (1984) Interaction of a polymeric biguanide biocide with phospholipid membranes. Biochim Biophys Acta 769(1): 57–66

Ikeda T, Tazuke S, Watanabe M (1983) Interaction of biologically active molecules with phospholipid membranes. I. Fluorescence depolarization studies on the effect of polymeric biocide bearing biguanide groups in the main chain. Biochim Biophys Acta 735(3): 380–6

Karayil S, Deshpande SD, Koppikar GV (1998) Effect of honey on multidrug resistant organisms and its synergistic action with three common antibiotics. J Postgrad Med 44(4): 93–6

In Profile

Charles Louis Alphonse Laveran



Charles Louis Alphonse Laveran (18 June 1845 - 18 May 1922) was a French physician who won the Nobel Prize in Physiology or Medicine in 1907 for his discoveries of parasitic protozoans as causative agents of infectious diseases such as malaria and trypanosomiasis.

Charles Louis Alphonse Laveran (18 June 1845 – 18 May 1922) was a French physician who won the Nobel Prize in Physiology or Medicine in 1907 for his discoveries of parasitic protozoans as causative agents of infectious diseases such as malaria and trypanosomiasis. Following his father, Louis Théodore Laveran, he took up military medicine as his profession. He obtained his medical degree from University of Strasbourg in 1867.

At the outbreak of the Franco-Prussian War in 1870, he joined the French Army. At the age of 29 he became Chair of Military Diseases and Epidemics at the École de Val-de-Grâce. At the end of his tenure in 1878 he worked in Algeria, where he made his major achievements. He discovered that the protozoan parasite *Plasmodium* was responsible for malaria, and that *Trypanosoma* caused trypanosomiasis or African sleeping sickness. In 1894 he returned to France to serve in various military health services. In 1896 he joined Pasteur Institute as Chief of the Honorary Service, from where he received the Nobel Prize. He donated half of his Nobel prize money to establish the Laboratory of Tropical Medicine at the Pasteur Institute. In 1908, he founded the Société de Pathologie Exotique. Laveran was elected to French Academy of Sciences in 1893, and was conferred Commander of the National Order of the Legion of Honour in 1912.

Early life and education

Alphonse Laveran was born at Boulevard Saint-Michel in Paris, to parents Louis Théodore Laveran and Marie-Louise Anselme Guénard de la Tour Laveran. He was an only son with one sister. His family was in a military environment. His father was an army doctor and a Professor of military medicine at the École de Valde-Grâce. His mother was the daughter of an army commander. At a young age his family went to Algeria to accompany his father's service. He was educated in Paris, and completed his higher education from Collège Saint Barbe and later from the Lycée Louis-le-Grand. Following his father he chose military medicine and entered the Public Health School at Strasbourg in 1863. In 1866 he became a resident medical student in the Strasbourg civil hospitals. In 1867 he submitted a thesis on the regeneration of nerves and earned his medical degree from the University of Strasbourg.

Career

Laveran was Medical Assistant-Major of the French Army at the time of Franco-Prussian War. He was posted to Metz, where the French were eventually defeated and the place occupied by Germans. He was sent to work at Lille hospital and then to the St Martin Hospital (now St Martin's House) in Paris. In 1874 he qualified a competitive examination by which he was appointed to the Chair of Military Diseases and Epidemics at the École de Val-de-Grâce, a position his father had occupied. His tenure ended in 1878 and he was sent to Algeria, where he remained until 1883. From 1884 to 1889 he was Professor of Military Hygiene at the École de Val-de-Grâce. In 1894 he was appointed Chief Medical Officer of the military hospital at Lille and then Director of Health Services of the 11th Army Corps at Nantes. By then he was promoted to the rank of Principal Medical Officer of the First Class. In 1896 he entered the Pasteur Institute as Chief of the Honorary Service to pursue a full-time research on tropical diseases.[4]

Discoveries

In 1880, while working in the military hospital in Constantine, Algeria, he discovered that the cause of malaria is a protozoan, after observing the parasites in a blood smear taken from a patient who had just died of malaria. He found the causative organism to be a protozoan which he named *Oscillaria malariae*, but later renamed *Plasmodium*. This was the first time that protozoans were shown to be a cause of disease of any kind. The discovery was therefore a validation of the germ theory of diseases.^[8]

Laveran later worked on the trypanosomes, particularly sleeping sickness, and showed once again that protozoans were responsible for the disease.

Awards and honours

Laveran was awarded the Bréant Prize (*Prix Bréant*) of the French Academy of Sciences in 1889 and the Edward Jenner Medal of the Royal Society of Medicine in 1902 for his discovery of the malarial parasite. He received the Nobel Prize in Physiology or Medicine in 1907. He gave half the Prize for foundation of the Laboratory of Tropical Medicine at the Pasteur Institute. In 1908 he founded the Société de pathologie exotique, over which he presided for 12 years. He was elected to membership in the French Academy of Sciences in 1893, and was conferred Commander of the National Order of the Legion of Honour in 1912. He was Honorary Director of the Pasteur Institute in 1915 on his 70th birthday. He was elected President of the French Academy of Medicine in 1920. His work was commemorated philatelically on a stamp issued by Algeria in 1954.

Personal life and death

Laveran married Sophie Marie Pidancet in 1885. They had no children. In 1922 he suffered from an unspecified illness for some months and died in Paris. He is interred in the Cimetière du Montparnasse in Paris. He was an atheist.

Recognition

Laveran's name features on the Frieze of the London School of Hygiene & Tropical Medicine. Twenty-three names of public health and tropical medicine pioneers were chosen to feature on the School building in Keppel Street when it was constructed in 1926.

Jokes

Now A Days!! Position Of Husband Is Like A Split A.C, No Matter How Loud He Is Outside, But Inside The House He Is Designed To Remain Silent, Cool & Controlled By.....

A Woman Is Standing Looking In The Bedroom Mirror... She Is Not Happy With What She Sees And Says To Her Husband, "I Feel Horrible; I Look Old, Fat And Ugly... I Really Need You To Pay Me A Compliment." The Husband Replies, "Your Eyesight's Damn Near Perfect."

Husband Throwing Knives On Wifes Picture. All Were Missing The Target! Suddenly He Received Call From Her "Hi, Wat Ru Doin?" His Honest Reply, "MISSING U"

After Massive Demand From All Husbands... A New App Called, "Fear" Is Launched In IPHONE 7 You Just Say, "Wife" And It Immediately Closes All Websites, Hides All Chats, Shuts Down All Games, Hide All Special Folders And Deletes Chat History! And Best Above All, It Puts Your Wife's Photograph As A Wallpaper.



A Wife Treats Hubby By Taking Him To A Lap Dance Club For His Birthday.. At The Club: Doorman Says: Hi Jim How R You? Wife Asks: How Does He Know You? Jim Says: Oh Dear, I Play Football With Him Inside Barman Says: The Usual Jim? Jim Says To Wife: Before You Say Anything, He's On The Darts Team In My Local Next A Lap Dancer Says: Hi Jim Do You Crave Special Again? The Wife Storms Out Dragging Jim With Her & Jumps Into A Taxi.. Driver Says "Hey Jimmy Boy, You Picked Up An Ugly One This Time.." Jim's Funeral Is On Sunday

Doctor: Madam, Your Husband Needs Rest And Peace So Here Are Some Sleeping Pills. Wife: Doc, When Should I Give Them To Him? Doctor: They Are For You.!!

Wife: You Always Carry My Photo In Your Handbag To The Office. Why?

Darling : When There Is A Problem, No Matter How Impossible, I Look At Your Picture And The Problem Disappears.

Wife: You See, How Miraculous And Powerful I Am For You?

Darling : Yes, I See Your Picture And Say To Myself, "What Other Problem Can There Be Greater Than This One?

Bug of the Month

HYGIENE SCIENCES

Cryptosporidium

Cryptosporidium is a microscopic parasite that causes the diarrheal disease cryptosporidiosis. Both the parasite and the disease are commonly known as "Crypto."

There are many species of *Cryptosporidium* that infect animals, some of which also infect humans. The parasite is protected by an outer shell that allows it to survive outside the body for long periods of time and makes it very tolerant to chlorine disinfection.

While this parasite can be spread in several different ways, water (drinking water and recreational water) is the most common way to spread the parasite. *Cryptosporidium* is a leading cause of waterborne disease among humans in the United States.

Illness & Symptoms

Symptoms of cryptosporidiosis generally begin 2 to 10 days (average 7 days) after becoming infected with the parasite. The most common symptom of cryptosporidiosis is **watery diarrhea**. Symptoms include:

- Watery diarrhea
- Stomach cramps or pain
- Dehydration
- Nausea
- Vomiting
- Fever
- Weight loss

Some people with Crypto will have no symptoms at all.

Symptoms usually last about 1 to 2 weeks (with a range of a few days to 4 or more weeks) in persons with healthy immune systems. Occasionally, people may experience a recurrence of symptoms after a brief period of recovery before the illness ends. Symptoms can come and go for up to 30 days.

While the small intestine is the site most commonly affected, in immunocompromised persons *Cryptosporidium* infections could possibly affect other areas of the digestive tract or the respiratory tract.

People with weakened immune systems may develop serious, chronic, and sometimes fatal illness. Examples of people with weakened immune systems include:

- people with HIV/AIDS;
- those with inherited diseases that affect the immune system; and
- cancer and transplant patients who are taking certain immunosuppressive drugs.

The risk of developing severe disease may differ depending on each person's degree of immune suppression.

Sources of Infection & Risk Factors

Crypto lives in the intestine of infected humans or animals. An infected person or animal sheds *Cryptosporidium* parasites in the stool. Millions of Crypto parasites can be released in a bowel movement from an infected human or animal. Shedding begins when the symptoms begin and can last for weeks after the symptoms (e.g., diarrhea) stop. You can become infected after accidentally swallowing the parasite. Crypto may be found in soil, food, water, or surfaces that have been contaminated with the feces from infected humans or animals. Crypto is not spread by contact with blood. Crypto can be spread by:

- Putting something in your mouth or accidentally swallowing something that has come in contact with the stool of a person or animal infected with Crypto.
- Swallowing recreational water contaminated with Crypto. Recreational water can be contaminated with sewage or feces from humans or animals.
- Swallowing water or beverages contaminated by stool from infected humans or animals.
- Eating uncooked food contaminated with Crypto. All fruits and vegetables you plan to eat raw should be thoroughly washed with uncontaminated water.
- Touching your mouth with contaminated hands. Hands can become contaminated through a variety of activities, such as:
 - o touching surfaces (e.g., toys, bathroom fixtures, changing tables, diaper pails) that have been contaminated by stool from an infected person,
 - o changing diapers,
 - o caring for an infected person, and
 - o handling an infected animal such as a cow or calf.

People with greater exposure to contaminated materials are more at risk for infection 1,2, such as:

- Children who attend childcare centers, including diaperaged children
- Childcare workers
- Parents of infected children
- Older adults (ages 75 years and older)
- People who take care of other people with cryptosporidiosis
- International travelers
- Backpackers, hikers, and campers who drink unfiltered, untreated water
- People who drink from untreated shallow, unprotected wells

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HYGIENE SCIENCES

Bug of the Month

- People, including swimmers, who swallow water from contaminated sources
- People who handle infected cattle
- People exposed to human feces through sexual contact

Contaminated water may include water that has not been boiled or filtered, as well as contaminated recreational water sources. Several community-wide outbreaks of cryptosporidiosis have been linked to drinking municipal water or recreational water contaminated with *Cryptosporidium*.

Cryptosporidium parasites are found in every region of the United States and throughout the world. Travelers to developing countries may be at greater risk for infection because of poorer water treatment and food sanitation, but cryptosporidiosis occurs worldwide. In the United States, an estimated 748,000 cases of cryptosporidiosis occur each year³.

Once infected, people with decreased immunity are most at risk for severe disease. The risk of developing severe disease may differ depending on each person's degree of immune suppression.

Diagnosis & Detection

Diagnosis of cryptosporidiosis is made by examination of stool samples. Because detection of *Cryptosporidium* can be difficult, patients may be asked to submit several stool samples over several days. Most often, stool specimens are examined microscopically using different techniques (e.g., acid-fast staining, direct fluorescent antibody [DFA], and/or enzyme immunoassays for detection of *Cryptosporidium* sp. antigens).

Molecular methods (e.g., polymerase chain reaction – PCR) are increasingly used in reference diagnostic labs, since they can be used to identify *Cryptosporidium* at the species level. Tests for *Cryptosporidium* are not routinely done in most laboratories; therefore, healthcare providers should specifically request testing for this parasite.

A new study calculates the waste generated by N95 usage and suggests possible ways to reduce it

Since the Covid-19 pandemic began last year, face masks and other personal protective equipment have become essential for health care workers. Disposable N95 masks have been in especially high demand to help prevent the spread of SARS-CoV-2, the virus that causes Covid-19.

All of those masks carry both financial and environmental costs. The Covid-19 pandemic is estimated to generate up to 7,200 tons of medical waste every day, much of which is disposable masks. And even as the pandemic slows down in some parts of the world, health care workers are expected to continue wearing masks most of the time.

That toll could be dramatically cut by adopting reusable masks, according to a new study from MIT that has calculated the financial and environmental cost of several different mask usage scenarios. Decontaminating regular N95 masks so that health care workers can wear them for more than one day drops costs and environmental waste by at least 75 percent, compared to using a new mask for every encounter with a patient.

"Perhaps unsurprisingly, the approaches that incorporate reusable aspects stand to have not only the greatest cost savings, but also significant reduction in waste," The study also found that fully reusable silicone N95 masks could offer an even greater reduction in waste. They are now working on developing such masks, which are not yet commercially available.

Reduce and reuse

In the early stages of the Covid-19 pandemic, N95 masks were in short supply. At many hospitals, health care workers were forced to wear one mask for a full day, instead of switching to a new one for each patient they saw. Later on, some hospitals, including MGH and Brigham and Women's Hospital in Boston, began using decontamination systems that use hydrogen peroxide vapor to sterilize masks. This allows one mask to be worn for a few days.

Last year, They began developing a reusable N95 mask that is made of silicone rubber and contains an N95 filter that can be either discarded or sterilized after use. The masks are designed so they can be sterilized with heat or bleach and reused many times.

Our vision was that if we had a reusable system, we could reduce the cost. "The majority of disposable masks also have a significant environmental impact, and they take a very long time to degrade. During a pandemic, there's a priority to protect people from the virus, and certainly that remains a priority, but for the longer term, we have to catch up and do the right thing, and strongly consider and minimize the potential negative impact on the environment."

Throughout the pandemic, hospitals in the United States have been using different mask strategies, based on availability of N95 masks and access to decontamination systems. The MIT team decided to model the impacts of several different scenarios, which encompassed usage patterns before and during the pandemic, including: one N95 mask per patient encounter; one N95 mask per day; reuse of N95 masks using ultraviolet decontamination; reuse of N95 masks using hydrogen peroxide sterilization; and one surgical mask per day.

They also modeled the potential cost and waste generated by the reusable silicone mask that they are now developing, which could be used with either disposable or reusable N95 filters.

According to their analysis, if every health care worker in the United States used a new N95 mask for each patient they encountered during the first six months of the pandemic, the total number of masks required would be about 7.4 billion, at a cost of \$6.4 billion. This would lead to 84 million kilograms of waste (the equivalent of 252 Boeing 747 airplanes).

They also found that any of the reusable mask strategies would lead to a significant reduction in cost and in waste generated. If each health care worker were able to reuse N95 masks that were decontaminated with hydrogen peroxide or ultraviolet light, costs would drop to \$1.4 billion to \$1.7 billion over six months, and 13 million to 18 million kilograms of waste would result (the equivalent of 39 to 56 747s).

Those numbers could potentially be reduced even further with a reusable, silicone N95 mask, especially if the filters were also reusable. The researchers estimated that over six months, this type of mask could reduce costs to \$18 million and waste to 1.6 million kilograms (about 2.5 747s).

"Masks are here to stay for the foreseeable future, so it's critical that we incorporate sustainability into their use, as well as the use of other disposable personal protective equipment that contribute to medical waste," Chu says.

Environmental burden

The data the researchers used for this study were gathered during the first six months of the pandemic in the United States (late March 2020 to late September 2020). Their calculations are based on the total number of health care workers in the United States, the number of Covid-19 patients at the time, and the length of hospital stay per patient, among other factors. Their calculations do not include any data on mask usage by the general public.

"Our focus here was on health care workers, so it's likely an underrepresentation of the total cost and environmental burden," While vaccination has helped to reduce the spread of Covid-19, we believes health care workers will likely continue to wear masks for the foreseeable future, to protect against not only Covid-19 but also other respiratory diseases such as influenza.

He and others have started a company called Teal Bio that is now working on further refining and testing their reusable silicone mask and developing methods for mass manufacturing it. They plan to seek regulatory approval for the mask later this year.

While cost and environmental impact are important factors to consider, the effectiveness of the masks also needs to be a priority. "Ultimately, we want the systems to protect us, so it's important to appreciate whether the decontamination system is compromising the filtering capacity or not," he says. "Whatever you're using, you want to make sure you're using something that's going to protect you and others." JOURNAL OF HYGIENE SCIENCES

WHO GUIDELINES FOR SAFE SURGERY

Confronted with worldwide evidence of substantial public health harm due to inadequate patient safety, the World Health Assembly (WHA) in 2002 adopted a resolution urging countries to strengthen the safety of health care and monitoring systems. The resolution also requested that WHO take a lead in setting global norms and standards and supporting country efforts in preparing patient safety policies and practices. In May 2004, the WHA approved the creation of an international alliance to improve patient safety globally; WHO Patient Safety was launched the following October. For the first time, heads of agencies, policy-makers and patient groups from around the world came together to advance attainment of the goal of "First, do no harm" and to reduce the adverse consequences of unsafe health care. The purpose of WHO Patient Safety is to facilitate patient safety policy and practice. It is concentrating its actions on focused safety campaigns called Global Patient Safety Challenges, coordinating Patients for Patient Safety, developing a standard taxonomy, designing tools for research policy and assessment, identifying solutions for patient safety, and developing reporting and learning initiatives aimed at producing 'best practice' guidelines. Together these efforts could save millions of lives by improving basic health care and halting the diversion of resources from other productive uses. The Global Patient Safety Challenge, brings together the expertise of specialists to improve the safety of care. The area chosen for the first Challenge in 2005–2006, was infection associated with health care. This campaign established simple, clear standards for hand hygiene, an educational campaign and WHO's first Guidelines on Hand Hygiene in Health Care. The problem area selected for the second Global Patient Safety Challenge, in 2007–2008, was the safety of surgical care.

The groundwork for the project began in autumn 2006 and included an international consultation meeting held in January 2007 attended by experts from around the world. Following this meeting, expert working groups were created to systematically review the available scientific evidence, to write the guidelines document and to facilitate discussion among the working group members in order to formulate the recommendations. A steering group consisting of the Programme Lead, project team members and the chairs of the four working groups, signed off on the content and recommendations in the guidelines document. The guidelines were pilot tested in each of the six WHO regions—an essential part of the Challenge—to obtain local information on the resources required to comply with the recommendations and information on the feasibility, validity, reliability and cost–effectiveness of the interventions.

Ten Essential Objectives for Safe Surgery

Surgical care is complex and involves dozens of steps which must be optimized for individual patients. In order to minimize unnecessary loss of life and serious complications, operating teams have 10 basic, essential objectives in any surgical case, which the WHO safe surgery guidelines support.

- 1. The team will operate on the correct patient at the correct site.
- 2. The team will use methods known to prevent harm from administration of anaesthetics, while protecting the patient from pain.
- 3. The team will recognize and effectively prepare for life threatening loss of airway or respiratory function.
- 4. The team will recognize and effectively prepare for risk of high blood loss.
- 5. The team will avoid inducing an allergic or adverse drug reaction for which the patient is known to be at significant risk.
- 6. The team will consistently use methods known to minimize the risk for surgical site infection.
- 7. The team will prevent inadvertent retention of instruments and sponges in surgical wounds.
- 8. The team will secure and accurately identify all surgical specimens.
- 9. The team will effectively communicate and exchange critical information for the safe conduct of the operation.
- 10. Hospitals and public health systems will establish routine surveillance of surgical capacity, volume and results.



Guide to infrastructure, supplies, and anaesthesia standards at three levels of healthcare facilities

Level 1 - Small hospital or health centre (Should meet at least 'highly recommended' anaesthesia standards)	Level 2 - District or provincial hospital (Should meet at least 'highly recommended' and 'recommended' anaesthesia standards)	Level 3 - Referral hospital (Should meet at least 'highly recommended', 'recommended' and' suggested' anaesthesia standards)
 Rural hospital or health centre with small number of beds (or urban location in an extremely disadvantaged area); sparsely equipped operating room for 'minor' procedures Provides emergency measures in the treatment of 90–95% of trauma and obstetrics cases (excluding caesarean section) Referral of other patients (for example, obstructed labor, bowel obstruction) for further management at a higher level 	 District or provincial hospital (e.g. with 100–300 beds) and adequately equipped major and minor operating rooms Short-term treatment of 95–99% of major life-threatening conditions 	 A referral hospital with 300–1000 or more beds and basic intensive care facilities. Treatment aims are the same as for level 2, with the addition of: Ventilation in operating room and intensive care unit Prolonged endotracheal intubation Throacic trauma care Homodynamic and inotropic treatment Basic intensive care unit patient management and monitoring for up to 1 week: all types of cases, but possibly with limited provision for: Multi-organ system failure Hemodialysis Complex neurological and cardiac surgery Prolonged respiratory failure Metabolic care monitoring
Essential Procedures	Essential Procedures	Essential Procedures
 Normal delivery Uterine evacuation Circumcision Hydrocele reduction, incision and drainage Wound suturing Control of haemorrhage with pressure dressings Debridement and dressing of wounds Temporary reduction of factures Cleaning or sterilization of open and closed fractures Chest drainage (possibly) Abscess drainage 	 Same as level 1 with the following additions: Caesarean section Laparotomy (usually not for bowel obstruction) Amputation Hernia repair Tubal ligation Closed fracture treatment and application of plaster of Paris Acute open orthopaedic surgery, e.g., internal fixation of fractures Eye operations, including cataract extraction Removal of foreign bodies e.g., in the airways Emergency ventilation and airway management for referred patients such as those with chest and head injuries 	Same as level 2 with the following additions: • Facial and intracranial surgery • Bowel surgery • Paediatric and neonatal surgery • Thoracic surgery • Major eye surgery • Major gynaecological surgery, e.g., vesico-vaginal repair
Personnel	Personnel	Personnel
 Paramedical staff or anaesthetic officer (including on-the-job training) who may have other duties as well Nurse-midwife 	 One or more trained anaesthetists District medical officers, senior clinical officers, nurses, midwives Visiting specialists, resident surgeon, obstetrician or gynaecologist 	• Clinical officers and specialists in anaesthesia and surgery
Drugs	Drugs	Drugs
 Ketamine 50 mg/ml injection Lidocaine 1% or 2% Diazepam 5 mg/ml injection, 2 ml or midazolam 1 mg/ml injection, 5 ml 	 Same as level 1, but also: Thiopental 500 mg/g powder or propofol Suxamethonium bromide 500 mg powder 	 Same as level 2 with the following additions: Propofol Nitrous oxide

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Best Practices

 Pethidine 50 mg/ ml injection, 2 ml Morphine 10 mg/ml, 1 ml Epinephrine (adrenaline) 1 mg Atropine 0.6 mg/ml Appropriate inhalation anaesthetic if vaporizer available 	 Pancuronium Neostigmine 2.5 mg injection Ether, halothane or other inhalation anaesthetics Lidocaine 5% heavy spinal solution, 2 ml Bupivacaine 0.5% heavy or plain, 4 ml Hydralazine 20 mg injection Frusemide 20 mg injection Dextrose 50% 20 ml injection Aminophylline 250 mg injection Ephedrine 30/50 mg ampoules Hydrocortisone (?) Nitrous oxide 	 Various modern neuromuscular blocking agents Various Modern Inhalation anaesthetics Various inotropic agents Various intravenous antiarrhythmic agents Nitroglycerine for infusion Calcium chloride 10% 10 ml injection Potassium chloride 20% 10 ml injection for infusion
Equipment: Capital Outlay	Equipment: Capital Outlay	Equipment: Capital Outlay
 Adult and paediatric self-inflating breathing bags with masks Foot-powered suction Stethoscope, sphygmomanometer, thermometer Pulse Oximeter Oxygen concentrator or tank oxygen and a drawover vaporizer with hoses Laryngoscopes, bougies 	Complete anaesthesia, resuscitation and airway management systems including: Reliable oxygen sources Vaporizer(s) Hoses and valves Bellows or bag to inflate lungs Face masks (sizes 00–5) Work surface and storage Paediatric anaesthesia system Oxygen supply failure alarm; oxygen analyser Adult and paediatric resuscitator sets Pulse oximeter, spare probes, adult and paediatric* Capnograph* Defibrillator (one per operating suite or intensive care unit)* Electrocardiograph monitor* Laryngoscope, Macintosh blades 1–3(4) Oxygen concentrator(s) (cylinder) Foot or electric suction Intravenous pressure infusor bag Adult and paediatric resuscitator sets Magill forceps (adult and child), intubation stylet or bougie Spinal needles 25G Nerve stimulator Automatic non-invasive blood pressure monitor	 Same as level 2 with these additions (per each per operating room or intensive care unit bed, except where stated): Electrocardiograph monitor* Anaesthesia ventilator, reliable electric power source with manual override Infusion pumps (two per bed) Pressure bag for intravenous infusion Electric or pneumatic suction Oxygen analyser* Thermometer (temperature probe*) Electric overhead heater Infant incubator Laryngeal mask, airways sizes 2, 3, 4 (three sets per operating room) Intubating bougies, adult and child (one set per operating room) Anaesthetic agent (gas and vapour) analyser Depth of anaesthesia monitors are being increasingly recommended for cases at high risk of awareness but are not standard in many countries. * It is preferable to combine these monitoring modalities in one unit.
Equipment: Disposable	Equipment: Disposable	Equipment: Disposable
 Examination Gloves Intravenous infusion and drug injection equipment Suction catheter size 16FG Airway support equipment, including airways and tracheal tubes Oral and nasal airways 	 Electrocardiograph electrodes Intravenous equipment (minimum fluids: normal saline, Ringer lactate and dextrose 5%) Paediatric giving sets Suction catheter size 16FG Suction gloves sizes 6–8 Nasogastric tubes sizes 10–16 FG Oral airways sizes 000–4 Tracheal tubes sizes 3–8.5 mm Spinal needles sizes 22 G and 25G batteries size C. 	 Same as level 2 with these conditions: Ventilator circuits Yankauer suckers Giving sets for intravenous infusion pumps Disposables for suction machines Disposables for capnography, oxygen an alyser in accordance with manufacturer's specifications Sampling lines Water traps Connectors Filters and fuel cells.

In Focus

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The Microbiology of Milk and Milk Products. Wound Infection and Modern Biocides

In Profile

Howard Martin Temin.

Bug of the Month Cyclospora cayetanensis.

Gut bacteria influence brain development.

WHO Guidelines for Safe Surgery (Part II).

Nakts @



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BioShields 🔘 Applying Science In Disinfection