**Notes from the 48th Annual Meeting of the Infectious Diseases Society of America (IDSA): Vancouver, Canada, October 21-24, 2010**

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**11/10/10**

The meeting consisted of many simultaneous sessions and this summary pertains only to some of the sessions that I attended. I cross-checked many points for accuracy, but errors in the summary may occur and they are my responsibility. Drug doses should be confirmed from standard sources before use.

Joel Ernst, M.D., Director of the Division of Infectious Diseases of the Department of Medicine at NYU was the 2010 Annual Meeting Program Chair.

1. **Symposium: Clinical Controversaries in Infectious Diseases**

1. **Treatment of bone and joint infections by AW Karchmer (Harvard, Beth Israel-Deaconess) and AF Widmer (Basel, SW)**

An agreed upon take away point aside from the disagreements that this area often invokes, is one already commonly used in practice, that is, **when an infected orthopedic prosthesis must be left in place** and experience has taught us that such an infection can rarely be eradicated by antibiotics alone, that the infection and its sx/signs can sometimes/often be **suppressed by** the indefinite use of oral antibiotics aimed at the pathogen, especially when combined with rifampin, which has potentially good distribution properties and activity against organisms in biofilms that are on prostheses. (Such microbes in biofilms are in heavy density and probably give out quorum sensing chemical signals to promote survival that result in a state of “microbial persistence”—the physiological state proposed many years ago by Rene Dubos, before any knowledge of biofilms/quorum signaling, for microbes in a state of slow replication and metabolism and resulting persistence, typical of that which was known by Dubos to be manifest with syphilis and tuberculosis—and therefore for bacteria in that “persister state” to be physiologically relatively resistant to antibiotic action.)

1. **Severe C. difficile infection: Do we need to resort to fecal transplantation?**
2. **\*Pro—Johan Bakken, M.D., Ph.D. (U of Minnesota)**

*(I was able to fill in additional details of this talk by phoning Dr. Bakken after the conference.)*

 C. diff now is most common severe hospital associated infection and is associated with a 20% relapse rate. Definition of severe infection: WBC >15,000 and serum creatinine rising by 50%. Sometimes progressing to severe, complicated infection with hypotension, toxic megacolon, high lactate levels with mortality up to 58%. The severe infection is now often associated with the toxin hypersecreting strain, NAP-1/BI/027, which has a binary toxin and a deleted gene that controls toxin production. High mortality with complication of toxic megacolon; mortality falls with colectomy.

Fecal Bacteroides and other anaerobic species are important for maintaining gut bacterial competition that wards off C. diff colonization and infection (“colonization resistance”). With stool transplantation, the C. diff infected patient’s gut flora develops the bacterial flora of the donor within 2 weeks and the stool pattern of the patient with severe infection often returns to normal within 24H of the transplantation, sometimes taking another couple of days, but with quicker response and much cost savings compared to PO vancomycin treatment.

Dr. Bakken reports 90% treatment success rate and recurrence prevention with stool transplantation. Donor is preferably a spouse or other family member for aesthetic and safety reasons; Bakken no longer screens for enteric pathogens when donor is a household member, although screening is described in his 2009 Anaerobe paper (see below for citation). Donor stool is homogenized with saline in a dedicated Waring blender and filtered thru a coffee filter in the Micro Lab (see citation below to his Anaerobe paper for detailed recipe for stool prep and more)—then instilled into the patient thru a NG tube with tip placed in the proximal duodenum, confirmed by X-ray, or by instillation thru a colonoscope or by a retention enema--all work equally well, but when there is toxic megacolon the instillation should be thru NG tube to avoid danger of colonic perforation. Instillation from below may need to be repeated. Bakken states that stool transplantation should become “standard therapy” for severe C. diff infection until something better becomes available, such as a prepared stool free mixture of the proper anaerobic bacteria. He now suggests this method for severe C. diff infection even before trying PO vancomycin treatment. John Bartlett of Hopkins at the IDSA meeting also noted the quick (generally 24H) response with stool transplant treatment (see under the literature review).

In patients with severe C. diff infection who require continual administration of antibiotics for serious infections that initiated the C. diff infection in the first place, Bakken suggests using antibiotics with lower rates of inducing C. diff (trying to avoid clindamycin, 2nd, 3rd generation cephalosporins, flouroquinolones, amp/clav, amoxicillin) and sometimes re-administering the stool transplant aliquot over a few days (the original homogenized stool material can be frozen and used for several instillations, if needed).

Private ID physicians have developed billing codes for its use in the US. In Denmark, a bacterial cocktail free of stool has been developed for treatment and efficacy is being evaluated. The recipe for preparing the stool for transplantation is contained in Dr. Bakken’s detailed paper: Bakken J Anaerobe 2009; **15**:285-89.

 *\*I found Dr. Bakken’s info and advice very helpful and sensible re: current mgt of this serious and common disease and in my opinion, it was the best practical clinical mgt. info that I heard at this year’s IDSA conference.*

1. **Con--Dale Gerding, M.D. (Hines VA and Loyola, Chicago)**

Dr. Gerding has a patent on and is working on the use of a non-toxin producing strain of C. diff which he believes will accomplish the same outcome as stool transplantation treatment. He also raises the question as to whether stool transplantation would have the same beneficial result with the continued use of antibiotics which is sometimes needed in patients who develop C. diff infection. In my later phone conversation with Dr. Bakken, he indicated that repeat instillations of the stool transplant material might be needed in such aforementioned circumstances, but gave no additional data on that point. Dr. Bakken in his rebuttal at the symposium suggested that, when available, the non-toxigenic C. diff will likely be expensive. Dr. Gerding noted that the use of IVIG as treatment for C. diff infection has had inconclusive results. Re prevention of recurrence of C. diff infection, Gerding discussed the use of tapering or pulse doses of vancomycin and the use of rifaximen for 2 weeks after the use of PO vanco for treatment, but noted that emergence of resistance to rifaximin has been encountered. He noted that use of the pareneteral form of vanco for PO use is less expensive than use of the PO formulation (but taste is bad). Probiotics, he stated, have no proven benefit for prevention. *[Note the literature review section with comments by John Bartlett re: an investigational antibiotic, fidaxomicin, which has promise for prevention of recurrence*.]

1. **Isolation for ESBL’s: Gowns plus gloves vs Gloves alone. Both opinions make valid points; I think no clear winner.**
2. **Pro Gowns plus gloves**—Anthony Harris (U of MD): High mortality from ESBL bacteremia; transmission demonstrated from both hands and inanimate objects; evidence for clonal spread in some hospitals and therefore implying patient to patient transmission; without both gowns and gloves study notes 15-20% visitors exit with the ESBL on board; hand hygiene often not adhered to; CDC suggests both gloves plus gowns.
3. **Gloves alone suggested**—Bob Weinstein (Stoger-old Cook County Hospital; Rush): advocates gloves plus alcohol hand hygiene (soap for hands after C. diff contact) for all patients plus chlorhexidine body washes for ESBL patients; Get out all unnecessary bladder catheters. No gowns for ESBL patients: concern for isolation of patients in contact isolation—fewer M.D. visits, more patient falls, fewer vital signs taken, more electrolyte abnormalities demonstrated.
4. **Symposium on Multi-Resistant Gram-negative Infections**

Gram negative bacilli are potential **“detoxifying machines” (**Lou Rice, Brown U) re: their emerging multiple mechanisms of antibiotic resistance (R), esp re: the numerous beta-lactamases that have been identified as coming from them. Some clinical strains of gram-negative rods are now called **pan-resistant**, when they are R to all available antibiotics.

1. **Background:**

Emergence of antibiotic R is primarily the result of the many years of **selective pressure** from the use of antibiotics which selects for emergence of R (mutated) strains that first exist as minor subpopulations among sensitive strains.

Numerous types of R mechanisms among Gram negative bacilli: Most important are the **beta-lactamases** because they can inactivate the antibiotics of the most clinically effective and safe class of antibiotics. Many different types of gram neg beta-lactamases:

1. **“Broad spectrum beta-lactamases”:** serine center; plasmid mediated; inhibited by clavulanate, sulbactam, tazobactam. Inactivate pen, amp, ticarc, pip, 1st gen ceph. Possessed by many enterobacteriaceae, bacteroides, H. infl., GC.
2. **“Extended spectrum beta-lactamases” (ESBL’s):** serine center; usually plasmid based, but gene sometimes on chromosome. Spectrum of substrates greater than 1. above, brought about by one or a few point mutations in the genes that encode the enzymes noted in 1. above. Can also inactivate 2nd gen cephs (but not cephamycins like cefoxitin), 3rd gen cephs, aztreonam and sometimes 4th gen cephs such as cefipime, but not the carbapenems (imipenem, etc.), which are effective and preferred for Rx. Sometimes tricky to detect this R in Micro labs. Most common in Kebs and E.coli., esp now with CTX-M ST131. Major problem in hospitals with nosocomial spread.
3. **“Amp C type beta-lactamases”:** serine center; usually encoded on bacterial chromosome, but some genes have jumped on to plasmids. Frequent in Enterobacter, Serratia, Pseudomonas aeruginosa. Not inhibited by clavulanate, etc. Inactivates all beta-lactams except carbapenems and sometimes 4th gen cephs (cefipime). Enzyme may be present at low levels but then can be induced by substrates such as cephs; some strains have the enzymes stably de-repressed= always turned on at high level.
4. **“Serine based carbapenemases”:** geneusually on plasmids; can inactivate all beta-lactams, tho some do not inactivate 3rd/4th gen cephs; most important now and major concern is the KPC family (esp KPC-2 and 3, plasmid located and in many Kelbsiella pneumonia and some P. aeruginosa and Acinetobacter species—hydrolyzes all beta-lactams). May be difficult to detect in Micro lab using automated sens systems. Another type in this group-- the OXA (“oxacillinase”) serine centered carbapenemases found in some Acinetobacter, P. aeruginosa, Kleb, Enterobacter, Serratia. Not well inhibited by the clavulanate type beta-lac inhibitors.
5. **Metallo-carbapenemases:** Zinc centered. In all S. maltophilia, but now on some P. aeruginosa, Acinetobacter, Klebsiella and other enterobacteriaceae. Inactivates all beta-lactams. Gene on plasmid or chromosome. Not inhibited by clavulanate, etc. Prominent families now in this category; VIM and NDM-1. Can detect in lab with ordinary systems and also by the inhibition of the enzyme by an EDTA disc which chelates the Zn.

Aside from the beta-lactamases, some Gram neg rods are R to aminoglycosides by having enzymes that change the chemistry of those drugs making them inactive; have mutated outer membrane porins that prevent entry of beta-lactams, have pumps that expel a variety of antibiotics; P. aeruginosa can have outer membrane mutations that make them R to the polymyxins by preventing their binding to the bacterial target. One Gram negative strain may contain genes for multiple R mechanisms.

1. **Treatment of MDR Gram-negative infections: No major breakthroughs—desperate need for new antibiotic classes.**
2. **Combinations of antibiotics:** No proven combinations. [Possibly for P. aeruginosa when the strain is sensitive to both an anti-Pseudomonas beta-lactam and an aminoglycoside, the two together in Rx may be more effective than the one—old data].
3. **Use of the polymyxins when there is not R (MIC>2 mcg/ml) to it:** Continuous infusion over 4h gives better PK than rapid infusions; nephrotoxicity common, but usually reversible. [Note difft doses for polymyxin B sulfate and colistin (polymyxin E) methane sulphonate—different hospitals have one or the other.] Emergence of R to these drugs is increasingly encountered especially after their prior use.
4. **For MDR Acinetobacter**, check sulbactam sensitivity with sulbactam E test strip (30-90% of strains are sensitive to sulbactam) and use amp/sulbactam when sulbactam sensitive and patient not allergic to pen, in high dosage (off label) of the amp/sulbactam: 24-36 gm per 24h (if patient has normal renal function) in severe infection-- and if polymyxin sensitive add that to the amp/sulbactam. No good data on usefulness of other combinations. Tigecycline sometimes active in vitro [but controversial re: clinical effectiveness—limited tissue levels achieved].
5. **Symposium on the Pneumococcus**

Much data presented—what follows is only a bit of that:

1. Recent studies indicate effectiveness of **PCV 7 vaccine**.
2. Relationship of more **heavily encapsulated strains of S. pneumo to higher mortality of invasive disease when that occurs, but lower likelihood of invasiveness**— [much of that known decades ago from studies of M. Finland and R. Austrian, classically with Type 3 and experimental studies of Barry Wood]—also **higher NP carrier rates in children with the more heavily encapsulated types compared to the less heavily encapsulated types.** (Weinberger DM et al.CID 2010;51:692-699)
3. Liise-anne Pirofski (Einstein/Montefiore) called my attention to look for a recent study—Zhang Z et al. JCI 2009;119:1899-1909.—Study of S.pneumo naiive **mice i**ndicating TLR-2 mediated activation of CD4 cells stimulating **macrophage mucosal clearance of primary S. pneumo NP carriage-**- in contrast to mice with previous colonization in which polys are the classic clearing phagocytes [which in humans is enhanced by presence of opsonizing substances, i.e. type specific antibody with or without activated complement].
4. Opinions of Dan Musher (Baylor; Houston VA), a recognized scholar of S. pneumo disease, re: **his evaluation of recent studies of treatment of invasive pneumococcal disease:**
5. **Increased effectiveness of treating pneumococcal pneumonia with ceftriaxone plus a macrolide,** such as azithromycin, the latter for its anti-inflammatory effects. [I strongly agree with this—several clinical studies suggest this, tho they are relatively small observational studies and not optimal in design. They are very suggestive for use of that combination and there is a plausible explanation for the benefit of the combination—but not because of any bacterial synergy against the growth of that pathogen; azithro and other macrolides are known to potently diminish macrophage cytokine release and to diminish other inflammatory mechanisms; azithro reaches high and prolonged concentrations in pulmonary alveolar macrophages and its anti-inflammatory effects presumably are available there in the lung. The morbidity/mortality of pneumococcal infection is mediated primarily by the inflammatory response of the host in contrast to that achieved by some other bacteria which also possess bacterial toxins that can directly injure vital organs. Osler recognized and was impressed by the “toxemia” in patients with pneumococcal pneumonia. Lewis Thomas in his wonderful “Lives of the Cell” (1974) essay wrote about how “We are mined”-- regarding the role of the host response contributing to morbidity/mortality in the pathogenesis of infectious diseases. Maxwell Finland often remarked about the extreme rarity for pneumococcal pneumonia to be associated with formation of a lung abscess, that is, for it to be necrotizing, except when it is rarely seen with Type 3 which is notorious for its heavy capsule.]
6. **Advocates use of activated Protein C in patients with pneumococcal sepsis when not contraindicated by bleeding diatheses.**
7. Steroids not helpful for S. pneumo pneumonia, but is helpful for S. pneumo meningitis
8. ?? role for statins in invasive S. pneumo disease [remains to be determined]
9. **Symposium: Viral hemorrhagic fevers**

Most alarming are infections caused by **Marburg and Ebola viruses**, which are filoviruses, single-strand, negative sense RNA viruses, recognizable on EM of tissues by their filamentous shape. Infections can be acquired in rain forest areas of central sub-Saharan Africa. Ultimate reservoir in fruit bats with potential transmission to non-human primates, antelopes and humans. High human mortality. Ebola virus has 5 species of which the Zaire species is the worst with 80-90% mortality. Human to human transmission by droplets from body fluids. With intubation an aerosol is possible. Essential: personal protective equipment must be worn with barrier nursing care for involved health care workers who are at substantial risk. Morbidity/mortality presumably mediated by cytokine “storm” released by macrophages and other cells causing damage, esp to endothelial cells, with vast array of symptoms and signs. In early stage the sx/signs are non-specific (fever, myalgia, headache) and later mimic a “septic syndrome”, sometimes with bleeding from various sites. Dx confirmed at US CDC lab (BSL-4 type) with traditional and molecular methods. Diff Dx: especially malaria and “sepsis”. Death, if it is to occur, often in second week of illness with multiple organ failure and shock. Cases acquired in Africa may show up here (very rare so far). Hospital lab equipment may become contaminated. No magic bullet yet for Rx—sepsis protocols followed, including use of activated Protein C. Exp’tal Rx: interference RNA’s, a phosphodiamidate morphino oligonucleotide, macrolide as an anti-inflammatory agent, statins (?). Need a vaccine. Virus may persist for weeks in “protected” sites such as in semen and sexual transmission has occurred.

1. **Treatment Dilemmas in Immunocompromised Hosts**
2. **Mgt of Febrile Neutropenia in the era of MDR bacterial pathogens**

**Allison Freifeld (U of Nebraska).** *Good talk but nothing new.*

Emphasized the need to appreciate the underlying cause of the neutropenia in analyzing the risk of the neutropenia. Early paper (Bodey G Ann Int Med 1964) on neutropenia and infection risk included mostly patients with acute leukemia and prolonged neutropenia and we now know that is a relatively high risk group for Gram neg bacteremia with hi mortality -50%, but risk lower with other causes of neutropenia such as with solid tumors with low tumor burden and chemotherapy. The empiric antibiotic regimens used to diminish chances of sepsis for low and hi risk neutropenic patients can differ, but for all mono-therapy is suggested to begin with, especially with an antibiotic active vs P. aeruginosa and other gram neg rods. For hospitalized patients that is often an iv anti-Pseudomonas penicillin. Don’t start vanco at the beginning; add if isolate MRSA. Add vanco, dapto or linezolid if signs deep tissue infection. Risks for MDR Gram neg rod bacteremia: prior flouroquinolones, beta-lactams, health care association, hematologic malignancy, stem cell transplant, deep tissue involvement, lung and CNS infections. Use of polymyxin early depends on local epi.

1. **Immune reconstitution syndrome (IRS) in transplant patients. Nina Singh (U of Pitt)**

 IRS is an antigen driven Th1 immune response. In AIDs patients she emphasized the high rate (17%) of IRS with Crypt. infection and indicated that crypt has mitogen as well as an antigen effect in promoting IRS (no data given). In crypt-induced IRS in transplant patients the focus of the IRS is often in the brain where there is latent infection and need imaging to find it-high mortality (20%). No good biomarkers—occasionally there is hypercalcemia, presumably resulting from activated macrophages producing Vit D.

Also IRS often found with invasive Aspergillus infection in patients with hematologic malignancy. Treatment of IRS with corticosteroids when there is organ dysfunction, but not advised in presence of Hep C. Sometimes TNF inhibitors used to treat IRS. Potential role of statins(?)

1. **Symposium on Invasive Fungal Infections by a panel from the Mycoses Study Group (MSG): Lindsey Baden (Brigham), John Wingard (U of Florida), Tom Walsh (Cornell), Peter Pappas (U of Alabama), Neil Clancy (U of Pitt), Claudio Viscolli ( Genova, Italy), J Peter Donnelly (Netherlands)**

They **focused on mold infections- invasive aspergillus and mucor infections.**

1. For aspergillus infections the serum and especially the BAL **galactomannan assay (GM)** results are helpful for diagnosis, but some controversy on the cut-off points that should be used to suggest the infection. For now the cut-off suggested is =/> 0.5 , though some experts are of the opinion that it should higher (such as =/> 1.5). Pip/tazo (Zosyn) antibiotic therapy can cause false positives in the GM assay which can persist for a few days after the last use.

 2. **Beta-glucan** (fungal cell wall component) serum assays are sometimes useful for suggesting the presence of invasive fungal infections in general with cut off of =/>80 pg/ml.

3**. Appreciate that immunosuppressed patients with fungal infections have mixtures of fungal pathogens in 10-15% of cases.**

4. In lung transplant patients the invasive aspergillus infection is usually a late onset infection with a lower mortality than the typically early onset infection in those with hematological malignancy, in whom there is more angioinvasion and higher GM levels.

5. **Diagnosis:** With invasive pulmonary aspergillus infection when there is a combination of a typical clinical/radiological picture: (1) dense, well circumscribed lesion(s) with or without halo sign (2) air-crescent sign (3) cavity (4) sometimes wedge-shaped infiltrates and segmental or lobar consolidation. Plus a positive GM assay, the panel suggests that there is no need for a biopsy. If the GM assay is negative they suggest need for biopsy. With non- pulmonary syndromes suggesting aspergillus infection they suggest pursuing more aggressive diagnostics such as biopsy.

1. **Multiple warnings:** In 1/3 of cases of proven invasive pulmonary aspergillus infection the radiological picture is indistinguishable from bacterial bronchopneumonia. In children there are fewer classic pulmonary lesions and often only a bronchopneumonia picture. Sometimes P. aeruginosa produces an angioinvasive pneumonia that appears radiologically the same as the classic picture of invasive aspergillus pulmonary infection. Can have a mixture of disseminated or local aspergillus together with a mucor infection.
2. For dx of invasive fungal infections in vitro hybridization techniques plus immunochemistry are probably more effective than immunohistochemistry alone. [**What is the Gold standard for evaluating a diagnostic modality,** since these molds often fail to grow in culture even when seen histologically in tissue?]
3. **Review of recent literature**

I note only a few items of the many presented by Michael Lederman (Case Western), Joel Ernst (NYU) and John Bartlett (Johns Hopkins):

1. **Differences in the way two monkey species handle chronic infection by immunodeficiency viruses:** Estes J et al. J PLoS Pathogens 2010;6:e1001052. African Sooty Mangabey-high viral load, rare CD4 depletion, no OI’s, low level of immune activation, no LPS translocation from gut, low level of CCR5 expression on CD4 cells. In contrast-African rhesus monkey-high viral load associated with CD4 depletion, OI’s and death, major immune activation associated with damaged colonic mucosa and mucosal bacterial translocation, including LPS which is found in lymph nodes. Bacterial translocation from damaged gut in immunodeficiency virus infected monkey is associated with major immune activation which is capable of major damage to cell mediated immunity. [Helpful recent reviews re HIV/AIDS immunopathogenesis, including role of damage to gut mucosa: Douek DC et al. Annu Rev Med 2009; 60:471-84 and Paiardini M et al. AIDS Rev 2008; 10:36-46.]

2. **Importance of HIV transmission exclusively utilizing the CCR5 receptor on host cells** and yet paradoxically endocervical cells have both CCR5 and CCR4 receptors (more CCR4)—only R5 viruses transmitted to them.

3. **HIV vaccine development continues to be a problem:** viral antigen needed for promotion of development of crucial neutralizing Ab is protected from immune cells by glycosylation as well as being subject to mutational escape. Potentially important finding from NIH coming from extensive bioengineering of the HIV envelope protein: the **isolation of a potent broadly neutralizing Ab (VRC 01)**—the corresponding viral antigen may prove important as a vaccine immunogen for prevention of infection.

4. **Review of M Liu et al. JCI 2009; 120:1914-1924: mouse model of Rhizopus infection (mucormycosis)** showing that hyperglycemia and free iron (both increased in patients with DKA who are also at increased risk for mucormycocis) up regulate a host endothelial protein receptor (GRP78) for Rhizopus attachment. Acidosis displaces chelated iron and the free iron upregulates the protein binder. Blocking the binder with antibody to it helps salvage mice. Suggests plausible mechanism for the long known clinical association of uncontrolled DM, esp ketoacidosis, with mucormycosis.

5. **Data of B. Hahn suggests origin of P. falciparum malaria in West African gorillas.**

6. **C. difficile infection reviewed by John Bartlett (Johns Hopkins)**

 a) fidaxomicin seems better than po vanco in preventing recurrence of C. diff. infection—probably no better for treatment. [It is an investigational drug that is a macrocyclic antibiotic that inhibits RNA polymerase]

 b) monoclonal Ab effective in treatment

 c) Fecal transplant very effective in treatment of C. diff infection and often gives quick results by returning normal stool pattern within 24H. See symposium labeled “Controversies in clinical ID” for more details on this.

 7. **Much research re the** **human microbiome**—90% of our body’s cells are microbial; most have never been cultured or named as species (data from nucleic acid analysis of stools and other colonized surfaces). Metabolic syndrome can be transplanted by stool flora from one mouse to another—[that is, the human endogenous flora is not just in a passive resident status—it contributes to our biology in ways we are just learning about, not only competing for a niche on our surfaces and thereby preventing colonization by potential pathogens, but also participating in various immunological and metabolic activities-perhaps good or bad.]

8. **Hemaglutinin of** **2009 H1NI influenza virus** similar in structure to that of the reconstructed 1918 Influenza virus

9. **MRSA toxins**—controversial data on significance of the **PVL toxin** in infections of humans—the toxin is injurious to human and rabbit leukocytes, but less so to those of mice; therefore, significance for interpretation of animal model studies. **S. aureus alpha hemolysin as a toxin** seems to be impt in human skin infections.

1. **Some of the named lectures**
2. **Maxwell Finland Lecture by Gerald Friedland (Yale). Convergent Epidemics of TB, HIV and Drug Resistant TB**

I found this talk inspiring—focused on the work of his group in an impoverished community of South Africa. It demonstrated that emphasis on simple and sensible management & control maneuvers by a dedicated staff, including that of the population involved, can make effective diagnoses, initiate good therapy and decrease transmission. One small, but potent example: open the windows (and continuously monitor that) in the clinic and hospital to markedly increase air exchanges to decrease TB transmission—and there was a lot more discussed.

1. **Edward Kass Lecture by Richard Wenzel (Med Coll of Va). The Convergence of Infection Control and Antibiotic Resistant Pathogens**

Much epidemiology data reviewed with historical background. He favors the ‘horizontal” (i.e., multiple species) elimination of potential pathogens which may be carried by hospitalized patients using chlorhexidine bathing and “vertical” (one species) elimination of nasal MRSA only in high risk patients (such as pre-major surgery) with nasal mupiricin. [20-40% 0f persons are nasal carriers of S. aureus; some, but not all, studies show that ridding nasal carriage decreases subsequent surgical site infections; some S. aureus are R to mupiricin; there is an E test strip to test for mupiricin R.] Wenzel noted a study demonstrating that the nasal colonization site for MRSA is in the anterior vestibule hair follicles [other studies indicate that S. aureus adheres to nasal mucus; probably both are correct.] Another study, Wenzel notes, indicates that S. epidermidis antagonizes nasal colonization by S. aureus.

1. **John Enders Lecture by Don Ganem (UCSF). Can we do better than “I think you have a virus”? Advances in viral diagnostics and discovery.**

A detailed review of the amazing availability and rapid emergence of sophisticated machines for the molecular diagnoses of the pathogens, especially viruses, that cause infectious diseases.